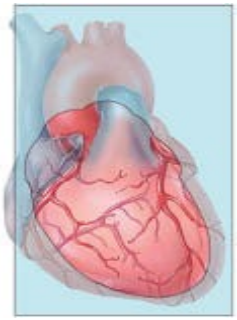


ANATOMY AND PHYSIOLOGY OF CARDIOVASCULAR SYSTEM

Course Name: Anatomy and Physiology 2

Course Code: 0521215

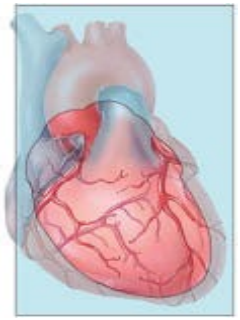
Lecturer: Ms. Asma El-Shara'. MPH

Faculty Of Pharmacy,

Philadelphia University-Jordan

جامعة فيلادلفيا

Philadelphia University



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Anatomy of the Heart

- ***OBJECTIVES***

- Describe the location of the heart.
- Describe the structure of the pericardium and the heart wall.
- Discuss the external and internal anatomy of the chambers of the heart.
- Relate the thickness of the chambers of the heart to their functions.

Location of the Heart

- The heart is located in the mediastinum, with two-thirds of its mass to the left of the midline.

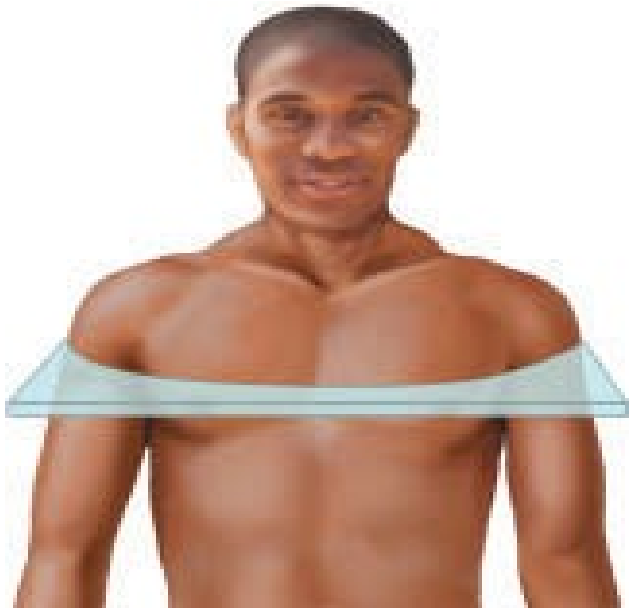
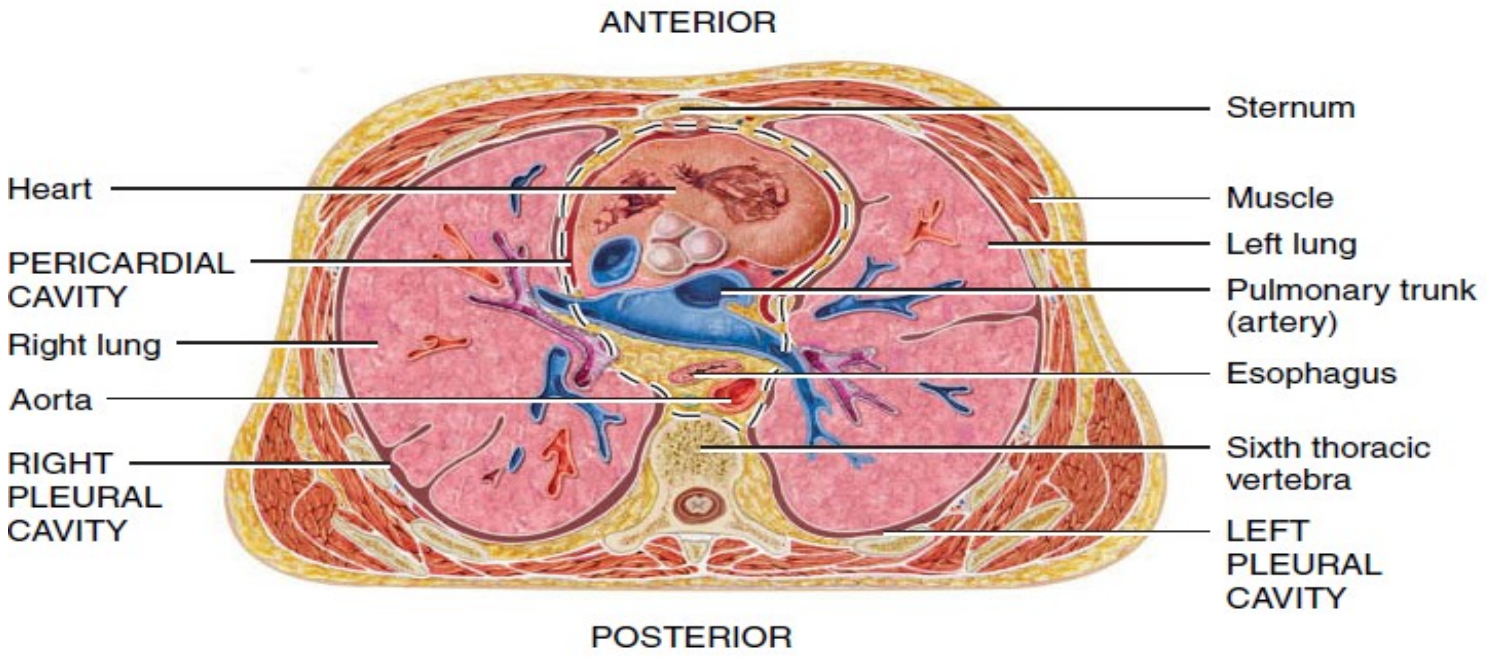


Figure 20.1

Location of the Heart

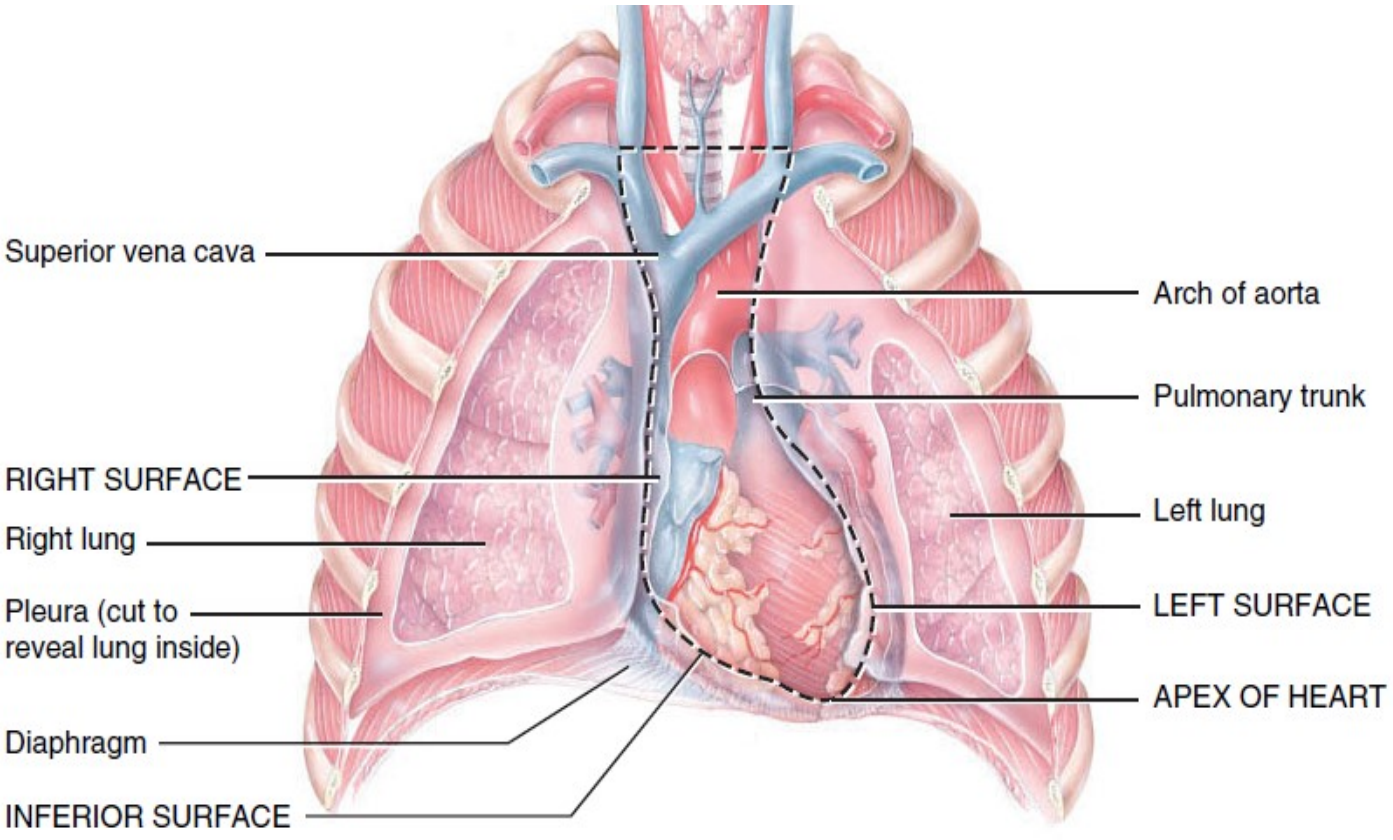
- The heart is located in the mediastinum, with two-thirds of its mass to the left of the midline.



(a) Inferior view of transverse section of thoracic cavity showing the heart in the mediastinum

Figure 20.1a

Location of the Heart



(b) Anterior view of the heart in the thoracic cavity

Figure 20.1b

Location of the Heart (continued)

- For all its might, the **heart** is relatively small, roughly the same size (but not the same shape) as your closed fist. It is about 12 cm (5 in.) long, 9 cm (3.5 in.) wide at its broadest point, and 6 cm (2.5 in.) thick, with an average mass of 250 g (8 oz) in adult females and 300 g (10 oz) in adult males.

Location of the Heart (continued)

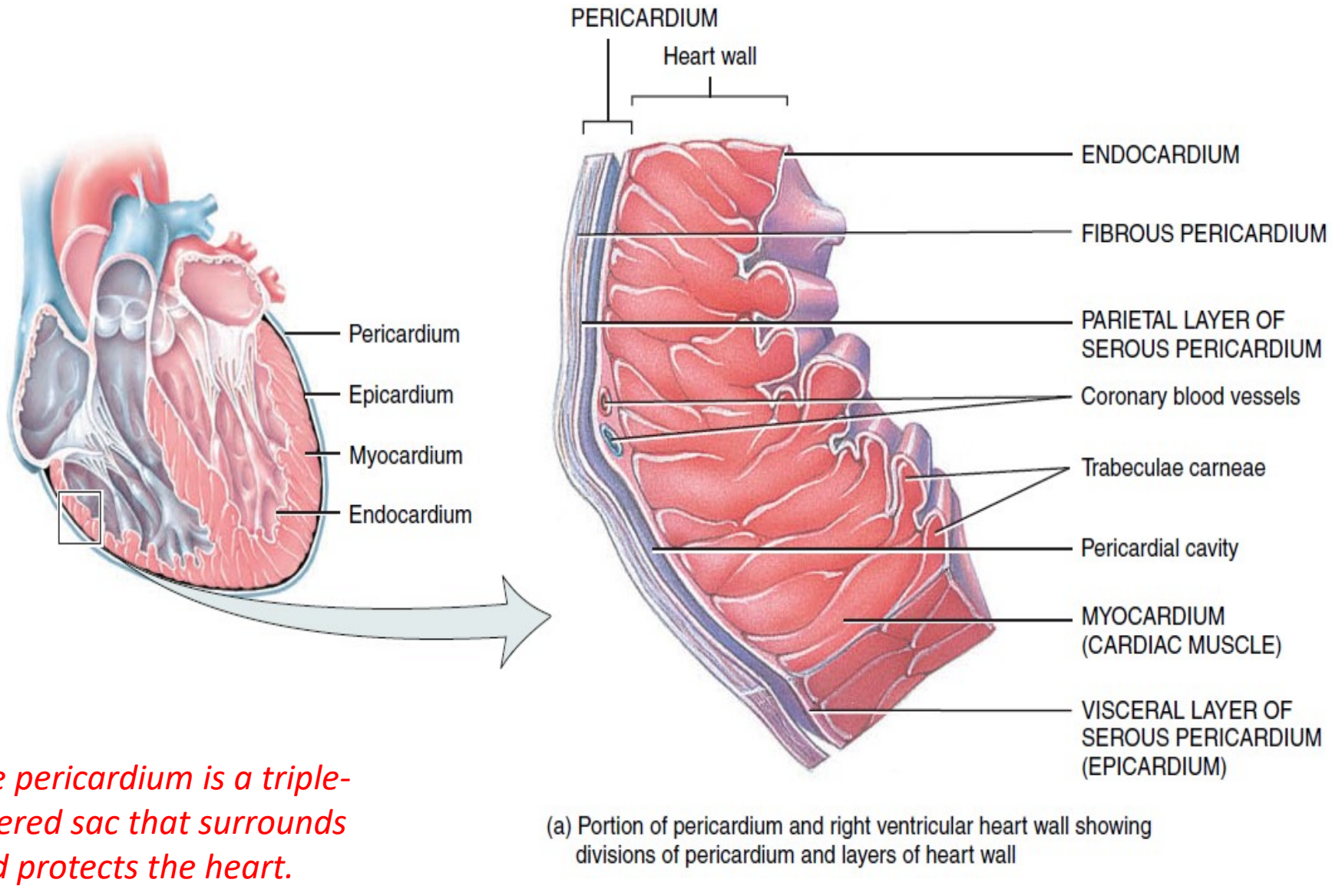
- The heart rests on the diaphragm, near the midline of the thoracic cavity. Recall that the midline is an imaginary vertical line that divides the body into unequal left and right sides. The heart lies in the **mediastinum** (me»-de»-as-TI»-num), an anatomical region that extends from the sternum to the vertebral column, from the first rib to the diaphragm, and between the lungs (Figure 20.1a).
- About two-thirds of the mass of the heart lies to the left of the body's midline (Figure 20.1b). You can visualize the heart as a cone lying on its side. The pointed **apex** is formed by the tip of the left ventricle (a lower chamber of the heart) and rests on the diaphragm. It is directed anteriorly, inferiorly, and to the left.
- The **base** of the heart is opposite the apex and is its posterior aspect. It is formed by the atria (upper chambers) of the heart, mostly the left atrium (see Figure 20.3c).

Location of the Heart (continued)

In addition to the apex and base, the heart has several distinct surfaces.

- The **anterior surface** is deep to the sternum and ribs.
- The **inferior surface** is the part of the heart between the apex and right surface and rests mostly on the diaphragm (Figure 20.1b).
- The **right surface** faces the right lung and extends from the inferior surface to the base.
- The **left surface** faces the left lung and extends from the base to the apex.

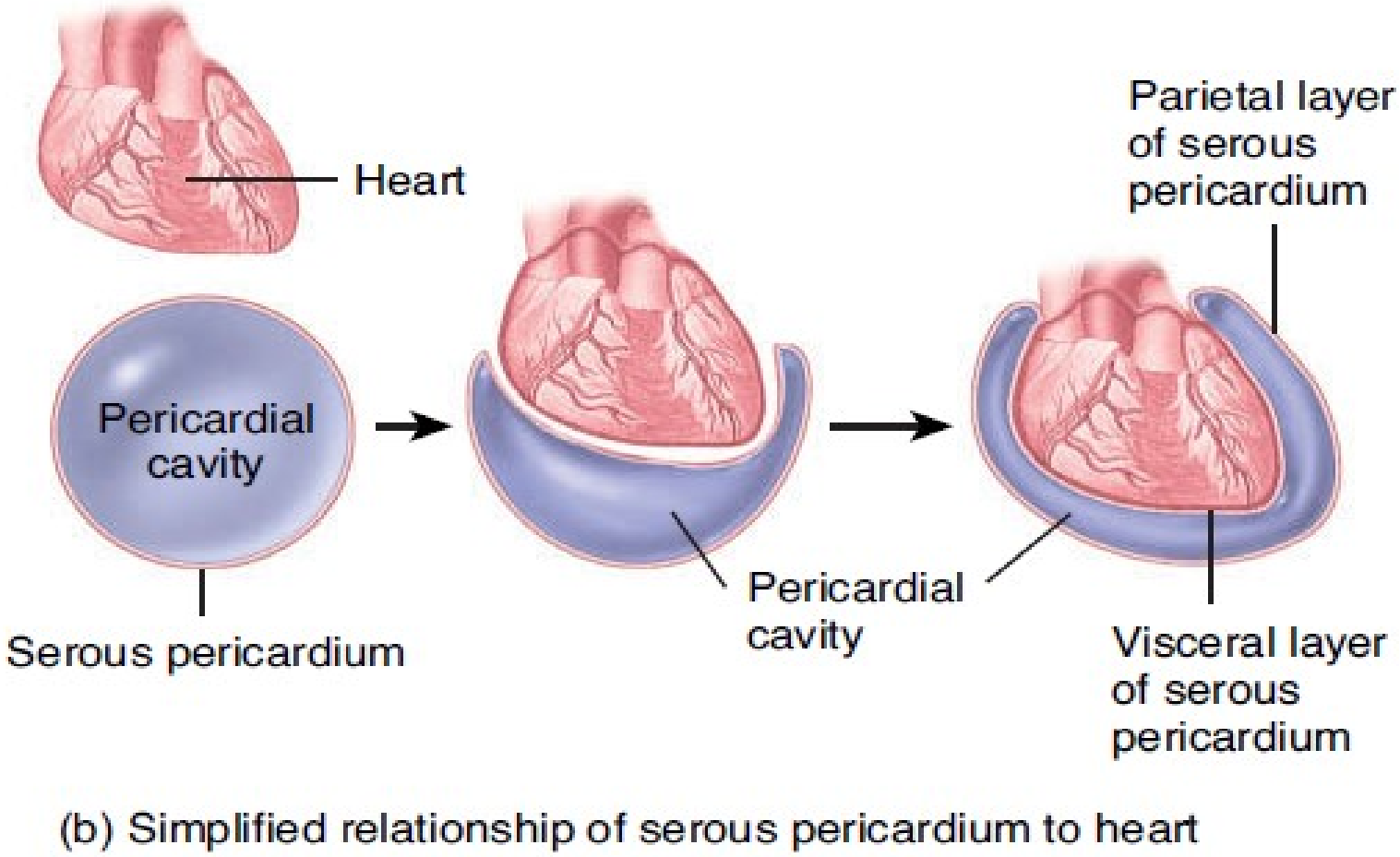
PERICARDIUM



(a) Portion of pericardium and right ventricular heart wall showing divisions of pericardium and layers of heart wall

The pericardium is a triple-layered sac that surrounds and protects the heart.

The pericardium is a triple-layered sac that surrounds and protects the heart.



(b) Simplified relationship of serous pericardium to heart

PERICARDIUM (continued)

The membrane that surrounds and protects the heart is the **pericardium** (per-i-KAR-de»-um; *peri-* around).

It confines the heart to its position in the mediastinum, while allowing sufficient freedom of movement for vigorous and rapid contraction.

The pericardium consists of two main parts:

(1) the fibrous pericardium

(2) the serous pericardium

a- The outer **parietal layer of the serous pericardium** is fused to the fibrous pericardium.

b- The inner **visceral layer of the serous pericardium**, which is also called the **epicardium** *(is one of the layers of the heart wall and adheres tightly to the surface of the heart).*

Layers of the Heart Wall

- The epicardium (external layer)
- The myocardium (middle layer)
- The endocardium (inner layer)

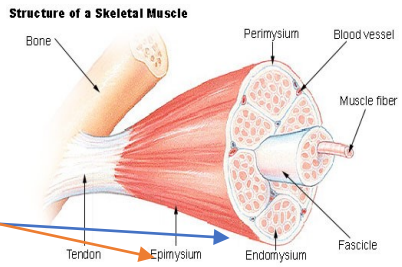
Layers of the Heart Wall
1- The epicardium (external layer)

- Composed of two tissue layers. The outermost, as you just learned, is called the *visceral layer of the serous pericardium.* This thin, transparent outer layer of the heart wall is composed of mesothelium. Beneath the mesothelium is a variable layer of delicate fibroelastic tissue and adipose tissue. The **adipose** tissue predominates and becomes thickest over the ventricular surfaces, where it houses the major coronary and cardiac vessels of the heart. The amount of fat varies from person to person, corresponds to the general extent of body fat in an individual, and typically increases with age.

- The epicardium imparts a smooth, slippery texture to the outermost surface of the heart. The epicardium contains blood vessels, lymphatics, and vessels that supply the myocardium.

Layers of the Heart Wall
2- The myocardium (middle layer)

- It is responsible for the pumping action of the heart and is **composed of cardiac muscle tissue**.
- It makes up approximately 95% of the heart wall.
- The muscle fibers (cells), like those of striated skeletal muscle tissue, are wrapped and bundled with connective tissue sheaths composed of endomysium and perimysium.
- The cardiac muscle fibers are organized in bundles that swirl diagonally around the heart and generate the strong pumping actions of the heart (Figure 20.2c). Although it is striated like skeletal muscle, recall that cardiac muscle is involuntary like smooth muscle.



Layers of the Heart Wall
2- The myocardium (middle layer)

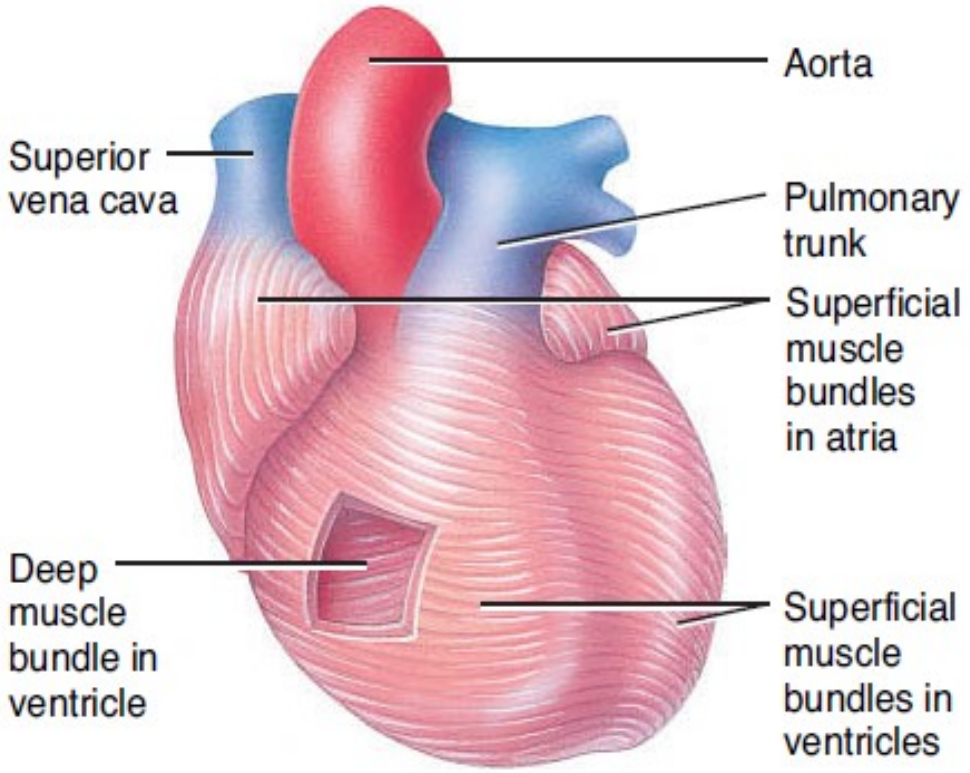


Figure 20.2c (c) Cardiac muscle bundles of myocardium

Layers of the Heart Wall
3-The endocardium (inner layer)

- It is a thin layer of endothelium overlying a thin layer of connective tissue.
- It provides a smooth lining for the chambers of the heart and covers the valves of the heart.
- The smooth endothelial lining minimizes the surface friction as blood passes through the heart.
- The endocardium is continuous with the endothelial lining of the large blood vessels attached to the heart.

CHAMBERS OF THE HEART

- **The heart has four chambers.**

- The two superior receiving chambers are the **atria** (entry halls or chambers)

- The two inferior pumping chambers are the **ventricles** (little bellies).

1- Right Atrium

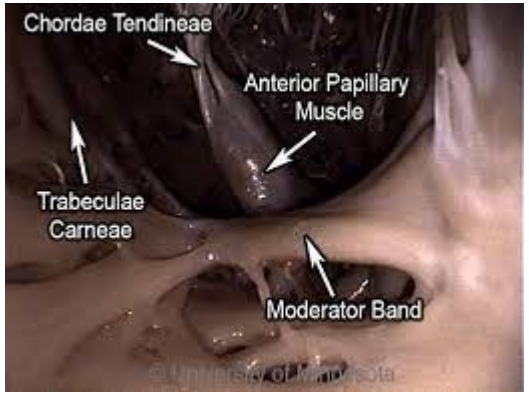
- The **right atrium** forms the right surface of the heart and receives blood from three veins: the *superior vena cava, inferior vena cava,* and *coronary sinus* (Figure 20.4a). (Veins always carry blood toward the heart.)
- Between the right atrium and left atrium is a thin partition called the **interatrial septum** (*inter-* between; *septum* a dividing wall or partition). *A prominent feature of this septum is an oval depression called the **fossa ovalis**, the remnant of the foramen ovale, an opening in the interatrial septum of the fetal heart that normally closes soon after birth (see Figure 21.30).*
- Blood passes from the right atrium into the right ventricle through a valve that is called the **tricuspid valve** because it consists of three **cusps** or leaflets (Figure 20.4a). It is also called the *right atrioventricular valve*. The valves of the heart are composed of dense connective tissue covered by endocardium.

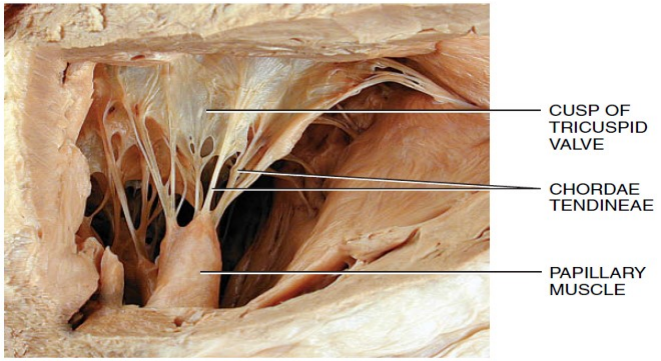
2- Right Ventricle

- The **right ventricle** is about 4–5 mm (0.16–0.2 in.) in average thickness and forms most of the anterior surface of the heart.
- Internally, the right ventricle is separated from the left ventricle by a partition called the **interventricular septum**.
- Blood passes from the right ventricle through the **pulmonary valve** (*pulmonary semilunar valve*) into a large artery called the *pulmonary trunk,* which divides into right and left *pulmonary arteries* and carries blood to the lungs. Arteries always take blood away from the heart (a mnemonic to help you: artery away).

2- Right Ventricle (continued)

- The inside of the right ventricle contains a series of ridges formed by raised bundles of cardiac muscle fibers called **trabeculae carneae** (tra-BEK-u»-le» KAR-ne»-e»; *trabeculae* little beams; *carneae* fleshy; see Figure 20.2a).
- Some of the trabeculae carneae convey part of the conduction system of the heart,
- The cusps of the tricuspid valve are connected to tendonlike cords, the **chordae tendineae** (KOR-de» ten-DIN-e»-e»; *chord-* cord; *tend-* tendon), which in turn are connected to cone-shaped trabeculae carneae called **papillary muscles** (*papill-* nipple).





(c) Tricuspid valve open

3- Left Atrium

- The **left atrium** is about the same thickness as the right atrium and forms most of the base of the heart (Figure 20.4a). It receives blood from the lungs through four *pulmonary veins*.
- Blood passes from the left atrium into the left ventricle through the **bicuspid (mitral) valve**, which, as its name implies, has two cusps.
- The term *mitral* refers to the resemblance of the bicuspid valve to a bishop's miter (hat), which is two-sided. It is also called the *left atrioventricular valve*.

4- Left Ventricle

- The **left ventricle** is the thickest chamber of the heart, averaging 10–15 mm (0.4–0.6 in.), and forms the apex of the heart (see Figure 20.1b).
- Like the right ventricle, the left ventricle contains trabeculae carneae and has chordae tendineae that anchor the cusps of the bicuspid valve to papillary muscles.
- Blood passes from the left ventricle through the **aortic valve** *(aortic semilunar valve)* into the *ascending aorta* (*aorte* to suspend, because the aorta once was believed to lift up the heart). Some of the blood in the aorta flows into the *coronary arteries,* which branch from the ascending aorta and carry blood to the heart wall.
- The remainder of the blood passes into the *arch of the aorta* and *descending aorta (thoracic aorta* and *abdominal aorta)*. Branches of the arch of the aorta and descending aorta carry blood throughout the body.

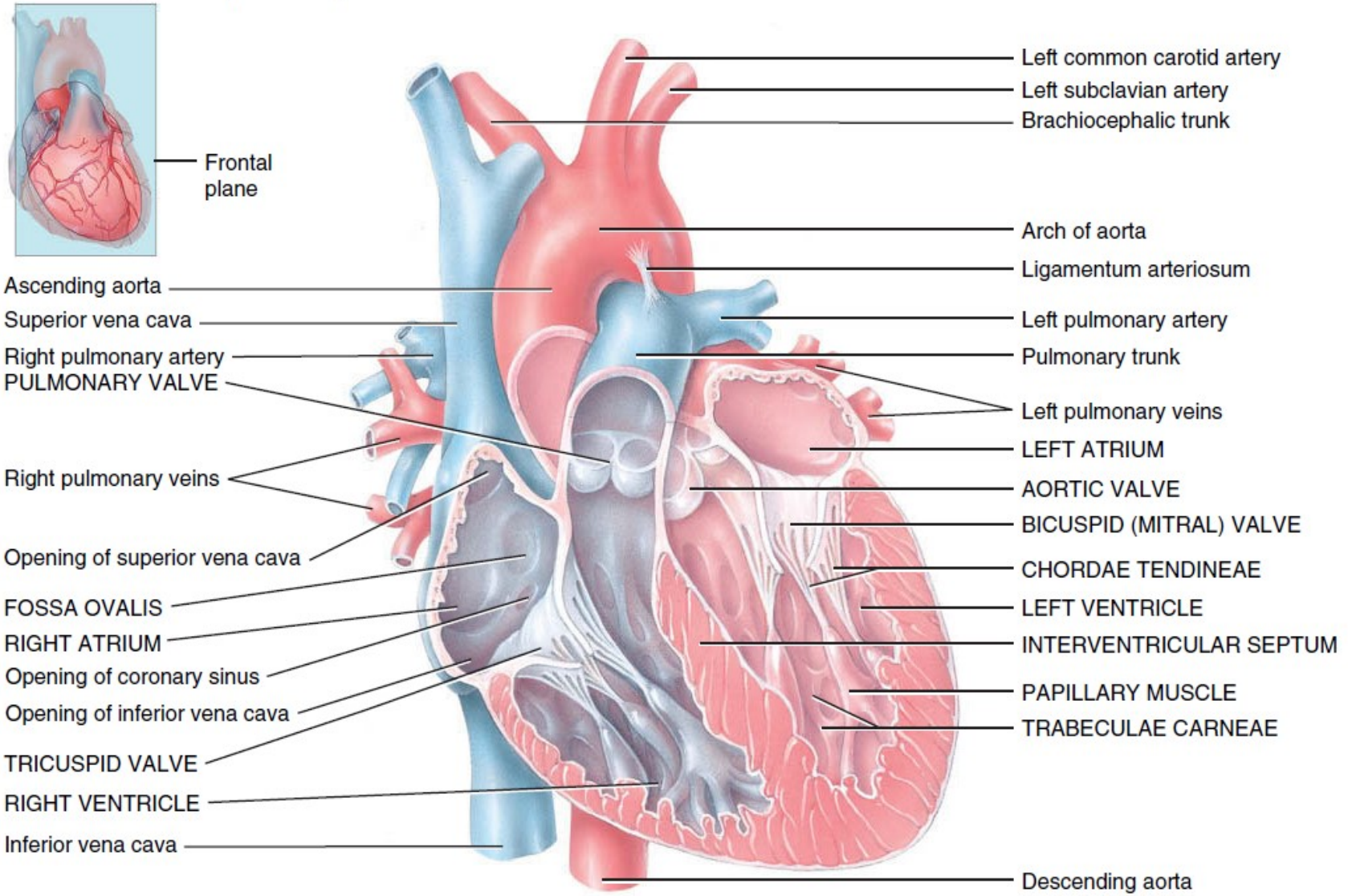
4- Left Ventricle (continued)

- During fetal life, a temporary blood vessel, called the *ductus arteriosus,* shunts blood from the pulmonary trunk into the aorta.

- Hence, only a small amount of blood enters the nonfunctioning fetal lungs (see Figure 21.30). The ductus arteriosus normally closes shortly after birth, leaving a remnant known as the **ligamentum arteriosum**, which connects the arch of the aorta and pulmonary trunk (Figure 20.4a).

Figure 20.4 Structure of the heart: internal anatomy.

Blood flows into the right atrium through the superior vena cava, inferior vena cava, and coronary sinus and into the left atrium through four pulmonary veins.



(a) Anterior view of frontal section showing internal anatomy

Fibrous Skeleton of the Heart

- The fibrous skeleton consists of FOUR dense connective tissue RINGS that surround the valves of the heart, fuse with one another, and merge with the interventricular septum.

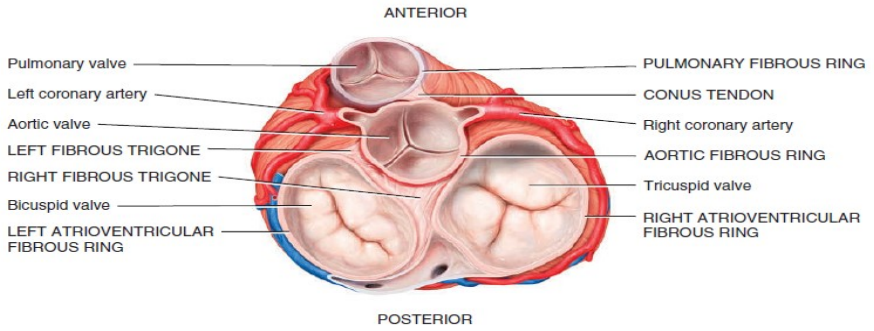
FUNCTION:

1- Forming a structural foundation for the heart valves.

2- Prevents overstretching of the valves as blood passes through them.

3- Serves as a point of insertion for bundles of cardiac muscle fibers

4- Acts as an electrical insulator between the atria and ventricles.



HEART VALVES AND CIRCULATION OF BLOOD

Describe the structure and function of the valves of the heart.

Outline the flow of blood through the chambers of the heart and through the systemic and pulmonary circulations.

Discuss the coronary circulation.

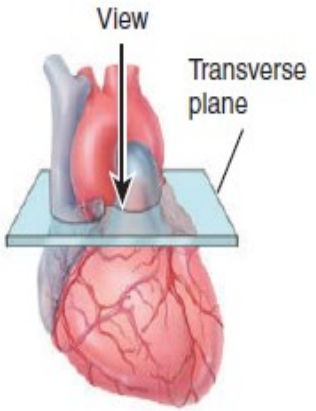
Introduction

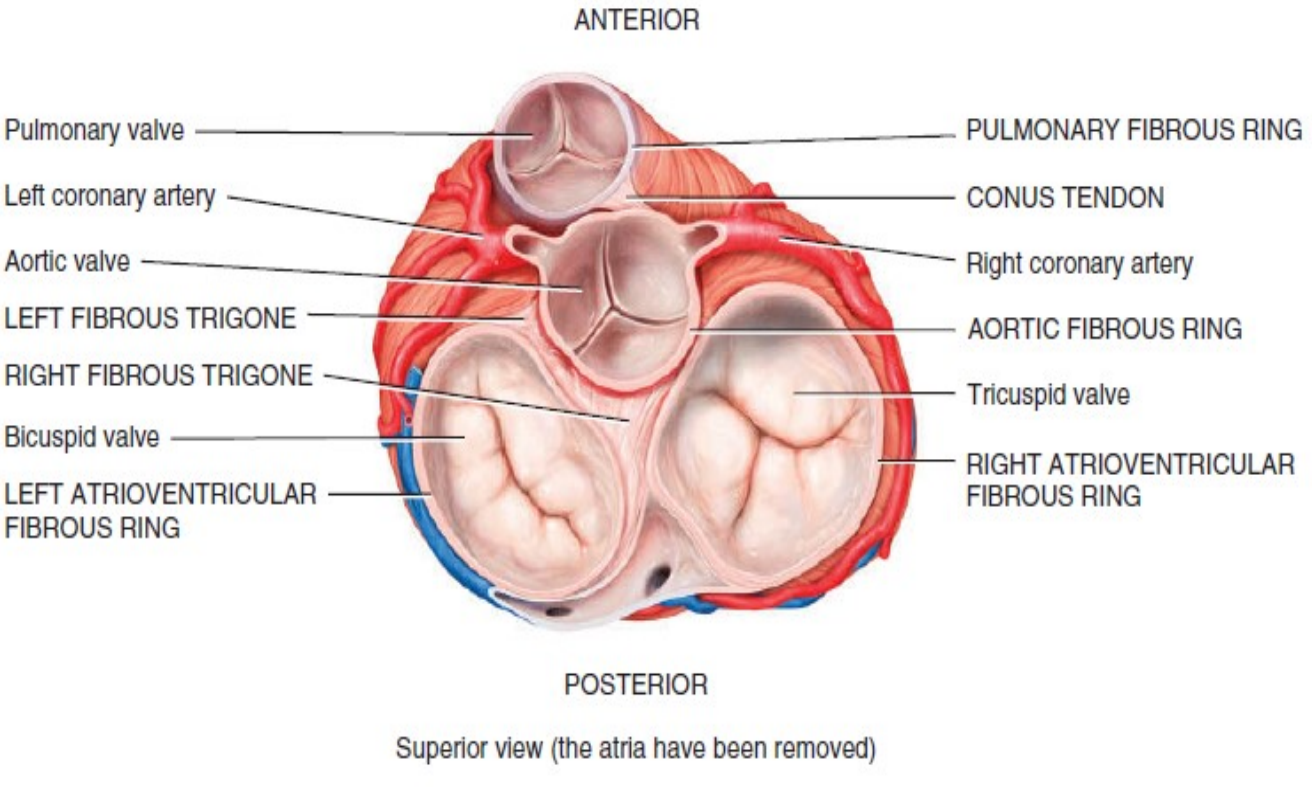
As each chamber of the heart contracts, it pushes a volume of blood into a ventricle or out of the heart into an artery.

Valves open and close in response to *pressure changes* as the heart contracts and relaxes.

Each of the four valves helps ensure the **oneway** flow of blood by opening to let blood through and then closing to prevent its backflow.

First: Heart Valves





Superior view (the atria have been removed)

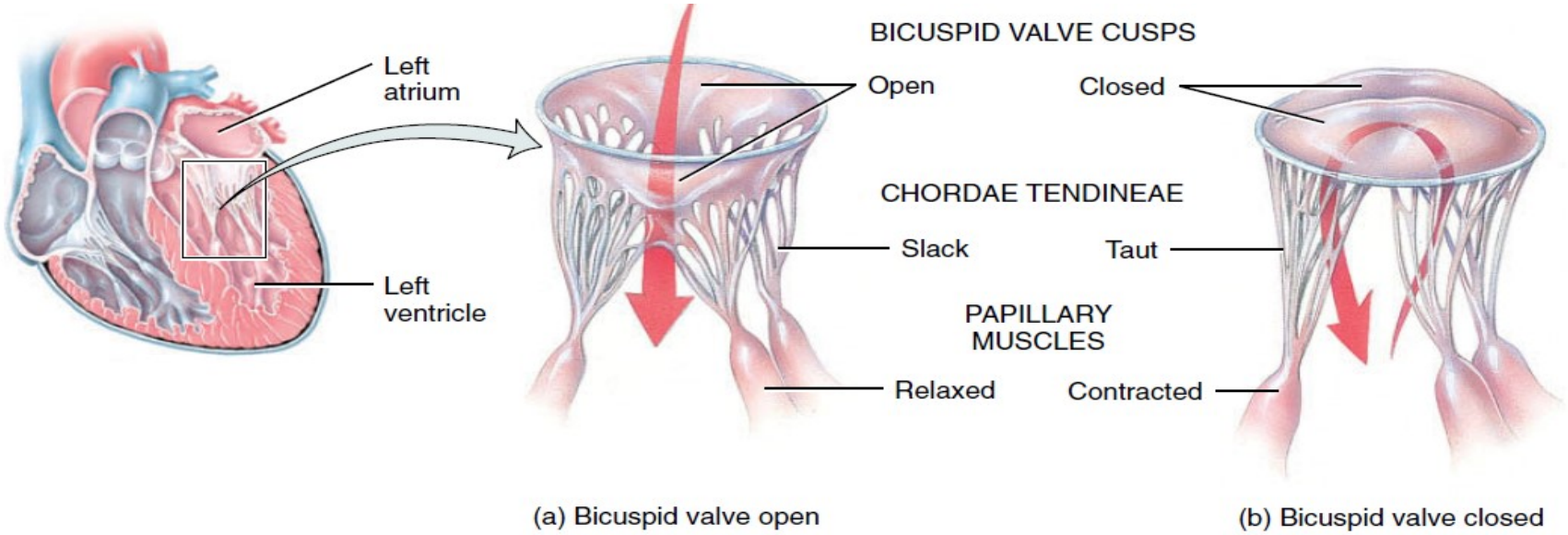
Operation of the Valves
Terms

1-Atrioventricular (AV) valves :
A-Tricuspid valve
B- Bicuspid valve (Mitral valve)

2- Semilunar (SL) valves:
A- Aortic Valve
B- Pulmonary valve

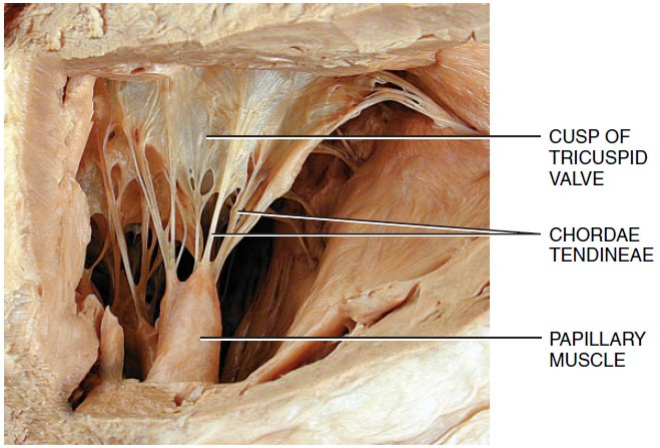
- The papillary muscles
- The chordae tendineae

Operation of the Valves

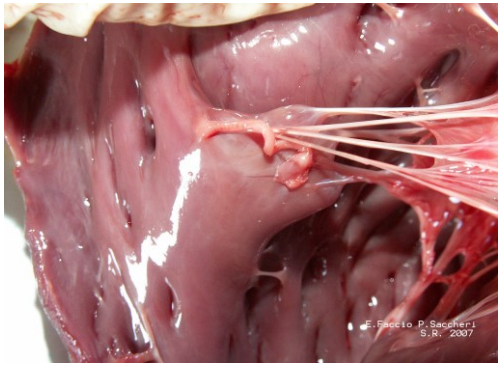


(a) Bicuspid valve open

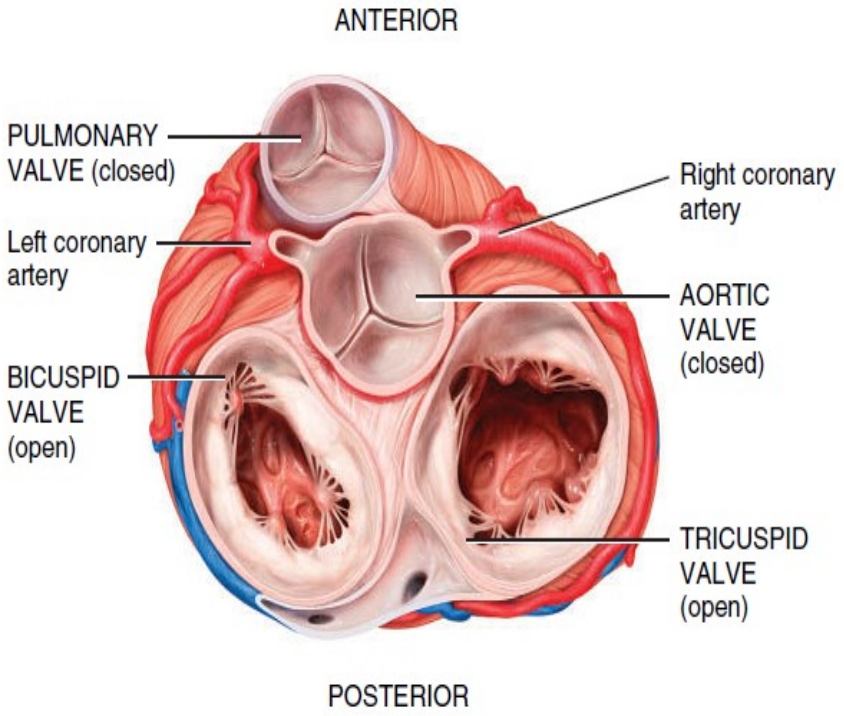
(b) Bicuspid valve closed



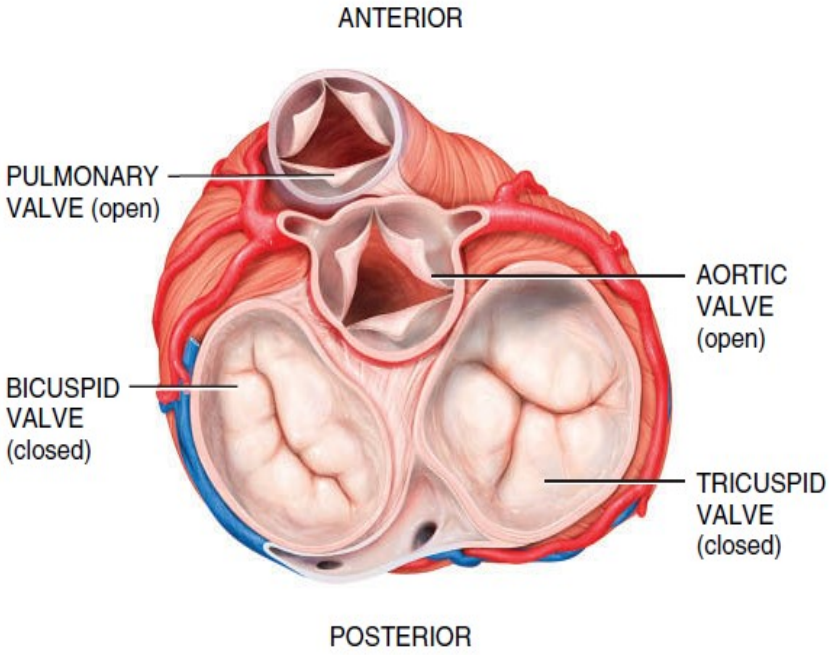
(c) Tricuspid valve open



Operation of the Valves (continued)



(d) Superior view with atria removed: pulmonary and aortic valves closed, bicuspid and tricuspid valves open



(e) Superior view with atria removed: pulmonary and aortic valves open, bicuspid and tricuspid valves closed

1- Operation of the Atrioventricular Valves

- *Because they are located between an atrium and a ventricle, the tricuspid and bicuspid valves are termed* ***atrioventricular (AV) valves****.*
- **When an AV valve is open**, the rounded ends of the cusps project into the ventricle. → **When the ventricles are relaxed**, the papillary muscles are relaxed, the chordae tendineae are slack, and blood moves from a higher pressure in the atria to a lower pressure in the ventricles through open AV valves (Figure 20.6a, d).
- **When the ventricles contract,** the pressure of the blood drives the cusps upward until their edges meet and **close** the opening (Figure 20.6b, e). At the same time, the papillary muscles contract, which pulls on and tightens the chordae tendineae. This prevents the valve cusps from everting (opening into the atria) in response to the high ventricular pressure.
- If the AV valves or chordae tendineae are damaged, blood may regurgitate (flow back) into the atria when the ventricles contract.

2- Operation of the Semilunar Valves

General information:

- The aortic and pulmonary valves are known as the **semilunar (SL) valves** (sem-e»-LOO-nar; *semi-* half; *-lunar* moon-shaped) because they are made up of three crescent moon–shaped cusps (Figure 20.6d).
- Each cusp attaches to the arterial wall by its convex outer margin.
- The SL valves allow ejection of blood from the heart into arteries but prevent backflow of blood into the ventricles. The free borders of the cusps project into the lumen of the artery.

2- Operation of the Semilunar Valves (continued-1)

- **When the ventricles contract,** pressure builds up within the chambers. The semilunar valves open when pressure in the ventricles exceeds the pressure in the arteries, permitting ejection of blood from the ventricles into the pulmonary trunk and aorta (Figure 20.6e).

- **As the ventricles relax,** blood starts to flow back toward the heart. This backflowing blood fills the valve cusps, which causes the free edges of the semilunar valves to contact each other tightly and close the opening between the ventricle and artery (Figure 20.6d).

2- Operation of the Semilunar Valves (continued-2)

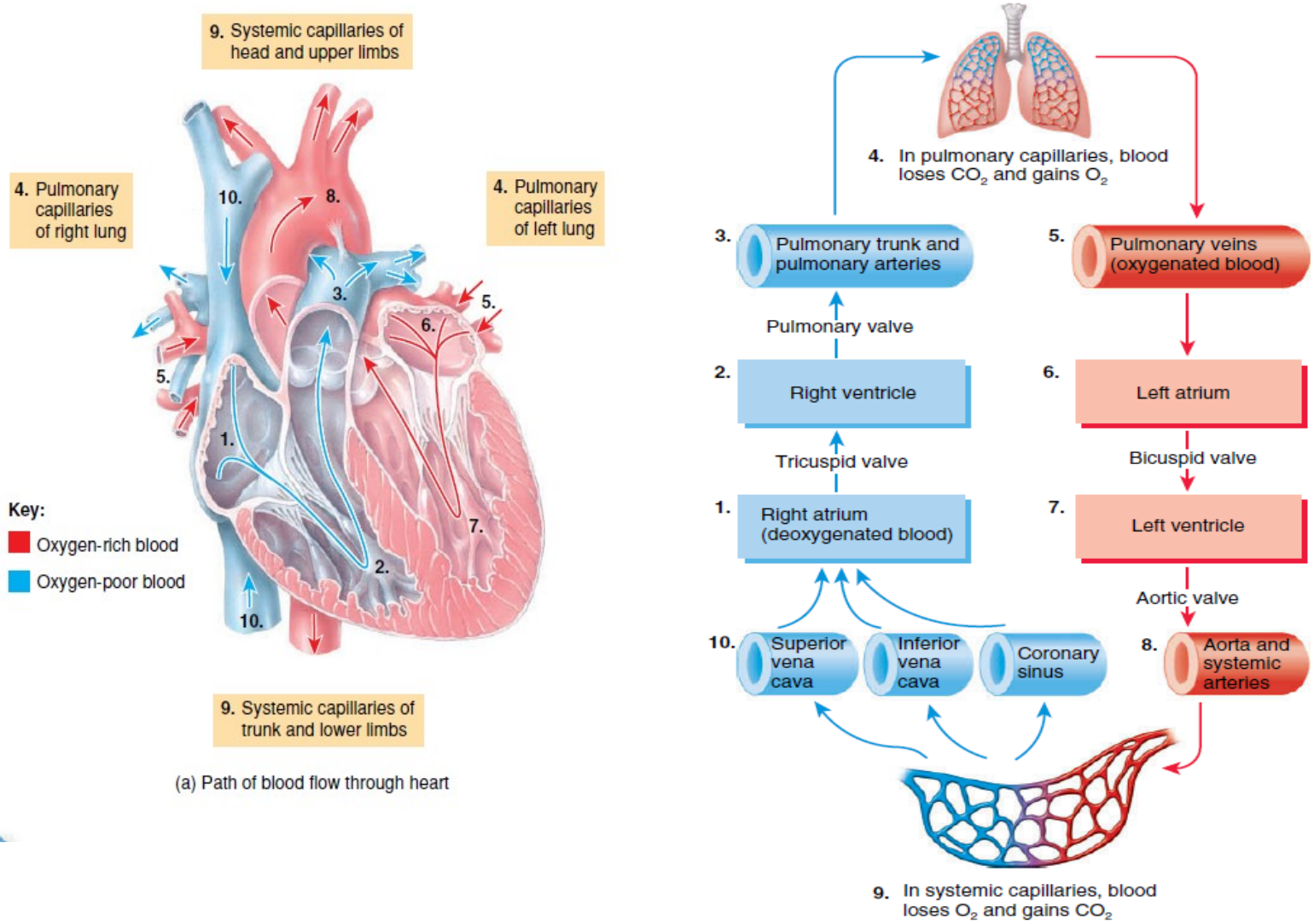
- Surprisingly perhaps, *there are no valves guarding the junctions between the venae cavae and the right atrium or the pulmonary veins and the left atrium*. As the atria contract, a small amount of blood does flow backward from the atria into these vessels. However, backflow is minimized by a different mechanism; as the atrial muscle contracts, it compresses and nearly collapses the weak walls of the venous entry points.

Second: Systemic and Pulmonary Circulations

- In postnatal (after birth) circulation, the heart pumps blood into two closed circuits with each beat—**systemic circulation** and **pulmonary circulation** (*pulmon-* lung) (Figure 20.7).

- The two circuits are arranged in series: The output of one becomes the input of the other.

Figure 20.7 Systemic and pulmonary circulations



Second: Systemic and Pulmonary Circulations

- **The left side of the heart is the pump for systemic circulation;** it receives bright red *oxygenated* (oxygen-rich) *blood* from the lungs.
- The left ventricle ejects blood into the *aorta* (Figure 20.7).
- From the aorta, the blood divides into separate streams, entering progressively smaller *systemic arteries* that carry it to all organs throughout the body—except for the air sacs (alveoli) of the lungs, which are supplied by the pulmonary circulation.
- In systemic tissues, arteries give rise to smaller-diameter *arterioles,* which finally lead into extensive beds of *systemic capillaries.* Exchange of nutrients and gases occurs across the thin capillary walls. Blood unloads O2 (oxygen) and picks up CO2 (carbon dioxide). In most cases, blood flows through only one capillary and then enters a *systemic venule.* Venules carry *deoxygenated* (oxygen-poor) *blood* away from tissues and merge to form larger *systemic veins.* Ultimately the blood flows back to the right atrium.

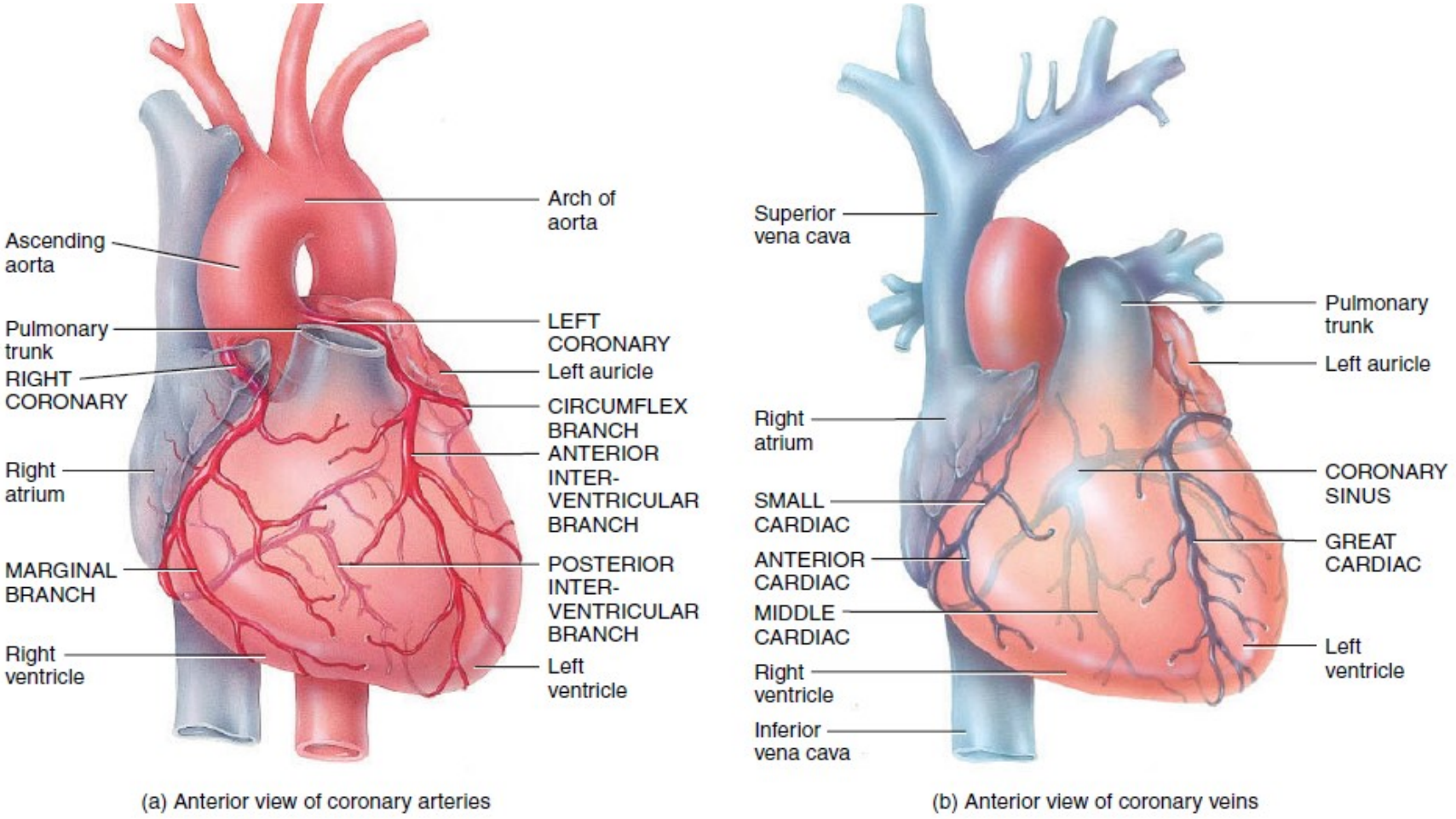
Second: Systemic and Pulmonary Circulations
(continued)

- **The right side of the heart is the pump for pulmonary circulation;** it receives all of the dark-red deoxygenated blood returning from the systemic circulation.
- Blood ejected from the right ventricle flows into the *pulmonary trunk,* which branches into *pulmonary arteries* that carry blood to the right and left lungs.
- In pulmonary capillaries, blood unloads CO2, which is exhaled, and picks up O2 from inhaled air.
- The freshly oxygenated blood then flows into pulmonary veins and returns to the left atrium.

Third: Coronary Circulation

- *Nutrients are not able to diffuse quickly enough from blood in the chambers of the heart to supply all layers of cells that make up the heart wall.*
- For this reason, the myocardium has its own network of blood vessels, the **coronary circulation** or *cardiac circulation* (*coron-* crown).
- The **coronary arteries** branch from the ascending aorta and encircle the heart like a crown encircles the head (Figure 20.8a).
- While the heart is contracting, little blood flows in the coronary arteries because they are squeezed shut.
- When the heart relaxes, however, the high pressure of blood in the aorta propels blood through the coronary arteries, into capillaries, and then into **coronary veins** (Figure 20.8b).

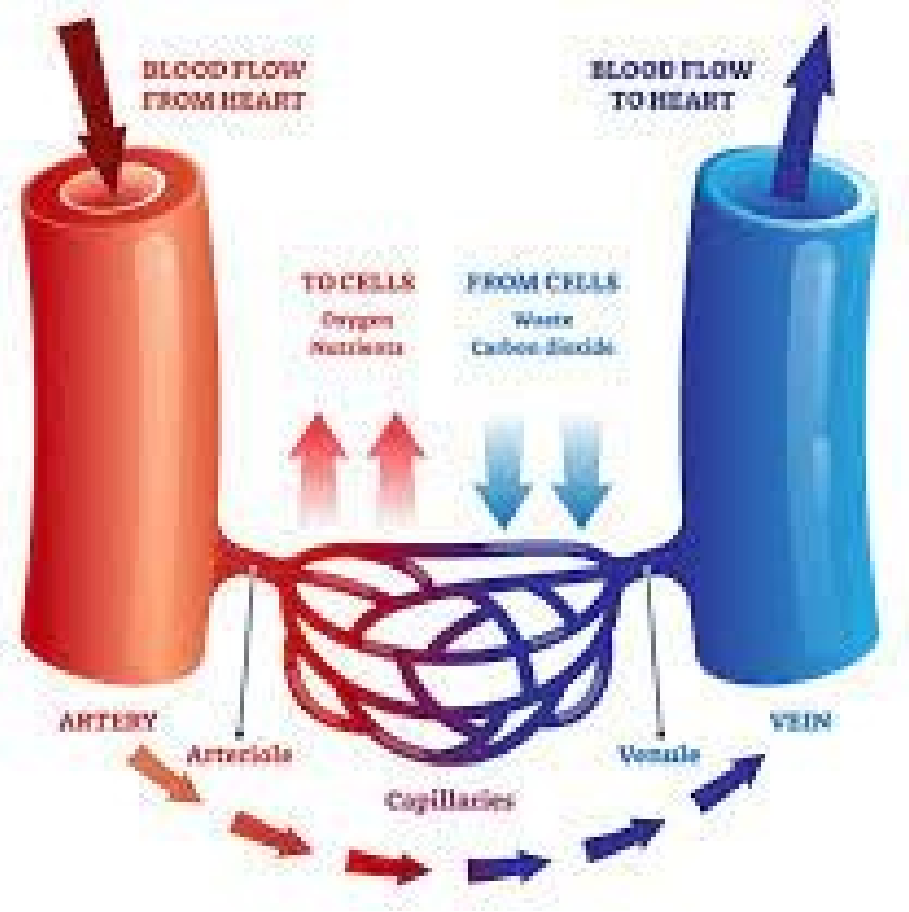
Figure 20.8 The coronary circulation. The views of the heart from the anterior aspect in (a) and (b) are drawn as if the heart were transparent to reveal blood vessels on the posterior aspect.



(a) Anterior view of coronary arteries

(b) Anterior view of coronary veins

Blood Vessels

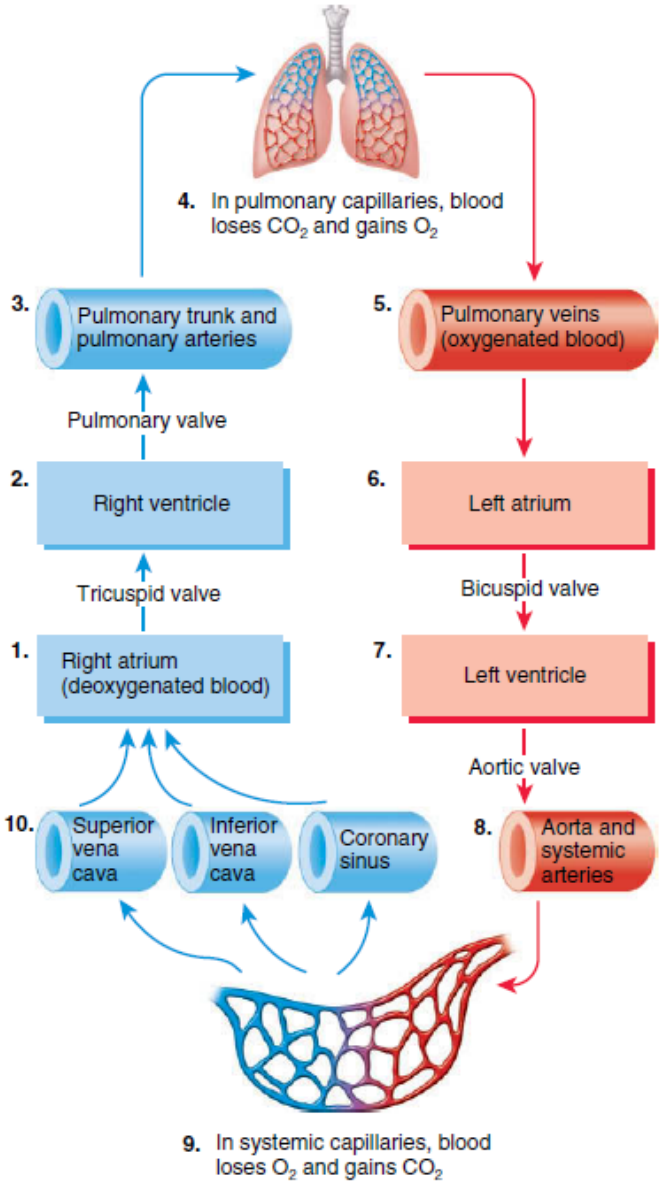


Structure and Function of Blood Vessels

The five main types of blood vessels are:

1. **Arteries** (AR-ter-e»z; *ar-* air; *-ter-* to carry) carry blood *away from the heart* to other organs. Large, elastic arteries leave the heart and divide into medium-sized, muscular arteries that branch out into the various regions of the body. Medium-sized arteries then divide into small arteries, which in turn divide into still smaller arteries called **arterioles**.
2. **Arterioles** (ar-TE» R-e»-o» ls), As the arterioles enter a tissue, they branch into numerous tiny vessels called **capillaries**
3. **Capillaries**, (KAP-i-lar-e»z hairlike). The thin walls of capillaries allow the exchange of substances between the blood and body tissues. Groups of capillaries within a tissue reunite to form small veins called **venules.**
4. **Venules**, (VENu»ls little veins). These in turn merge to form progressively larger blood vessels called veins.
5. **Veins** (VA» NZ) are the blood vessels that convey blood from the tissues *back to the heart.* (see Figure 20.7).

Figure 20.7: Systemic and pulmonary circulations.



(b) Path of blood flow through systemic and pulmonary circulations

Basic Structure of a Blood Vessel

- The wall of a blood vessel consists of three layers, or ***tunics***, of different tissues:

1- An epithelial inner lining→ tunica interna (intima)

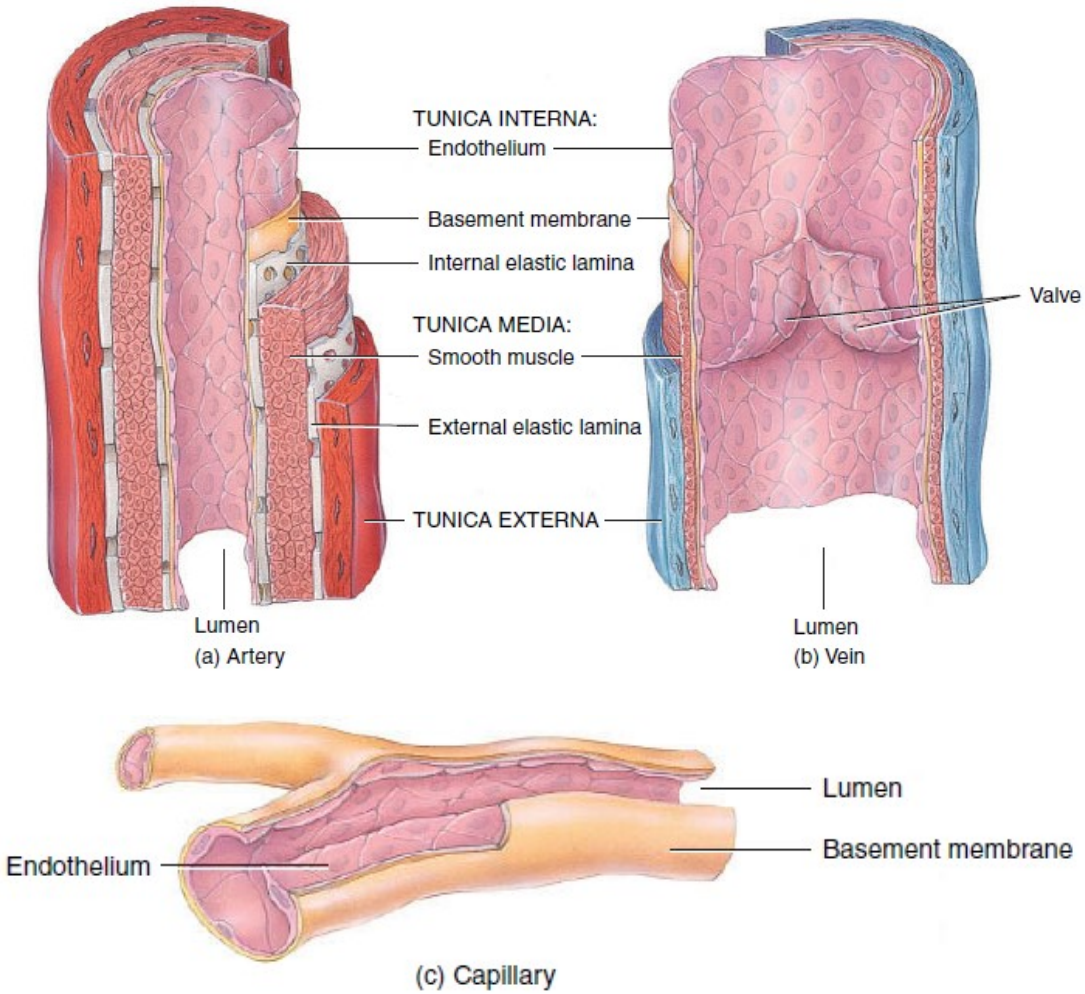
2- A middle layer consisting of smooth muscle and elastic connective tissue → tunica media

3- A connective tissue outer covering→ tunica externa (adventitia)

- **Modifications** of this basic design account for the five types of blood vessels and the structural and functional differences among the various vessel types → *Always remember that structural variations correlate to the differences in function that occur throughout the cardiovascular system.*

Figure 21.1 Comparative structure of blood vessels. The capillary (c) is enlarged relative to the artery (a) and vein (b).

Arteries carry blood from the heart to tissues; veins carry blood from tissues to the heart



1- Arteries

- The wall of an artery has the three layers of a typical blood vessel, but has a thick muscular-to-elastic tunica media (Figure 21.1a). Due to their plentiful elastic fibers, arteries normally have high *compliance*, which means that their walls stretch easily or expand without tearing in response to a small increase in pressure.

Types of Arteries

1-Elastic Arteries

2-Muscular Arteries

Types of Arteries-1

1-Elastic Arteries

- **Elastic arteries** are the largest arteries in the body
- These vessels are characterized by well defined internal and external elastic laminae, along with a thick tunica media that is dominated by elastic fibers, called the **elastic lamellae**
- Elastic arteries include the two major trunks that exit the heart (the aorta and the pulmonary trunk), along with the aorta's major initial branches, such as the brachiocephalic, subclavian, common carotid, and common iliac arteries.
- Elastic arteries perform an important function: They help propel blood onward while the ventricles are relaxing. As blood is ejected from the heart into elastic arteries, their walls stretch, easily accommodating the surge of blood. As they stretch, the elastic fibers momentarily store mechanical energy, functioning as a **pressure reservoir**

Types of Arteries- 2

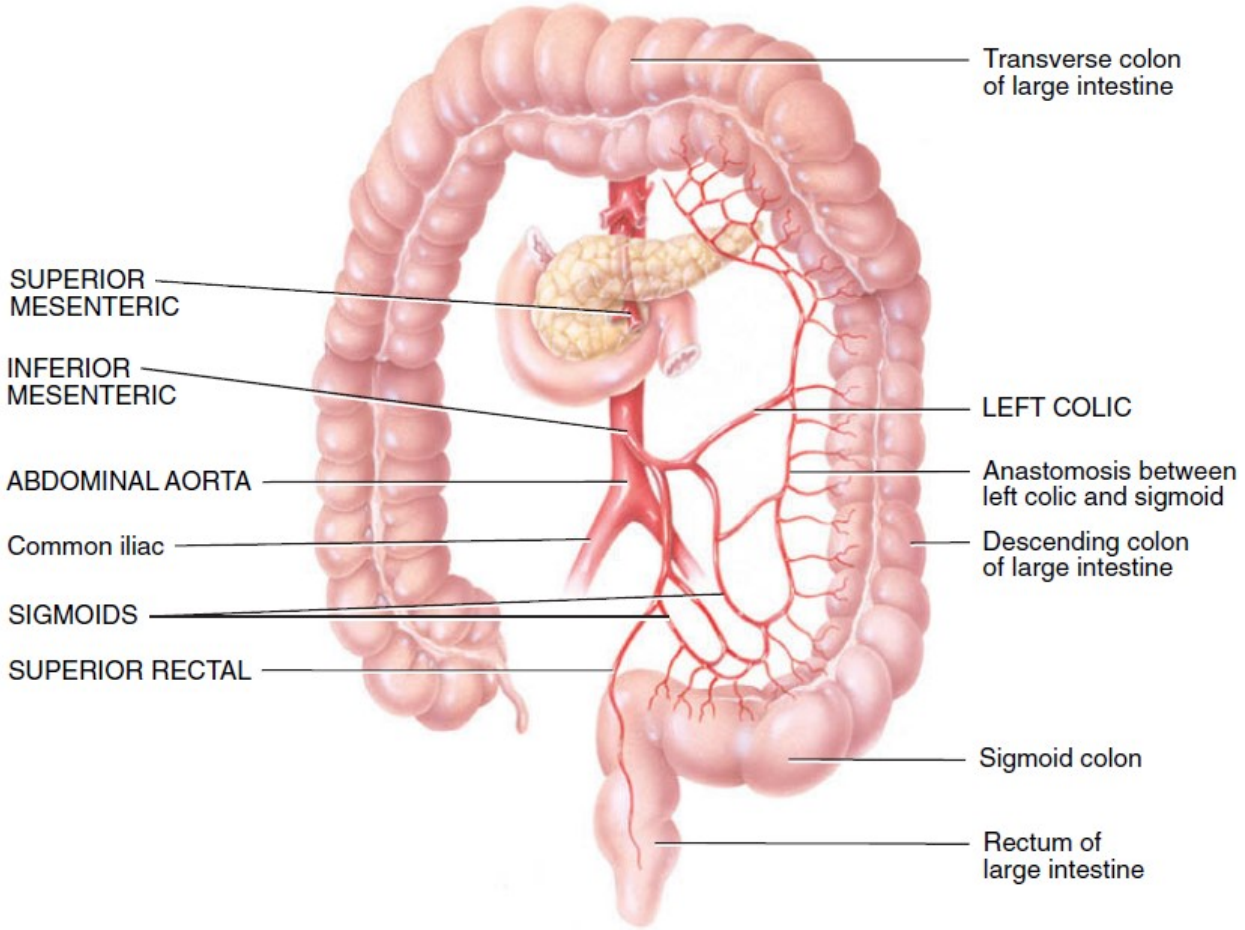
2-Muscular Arteries

- Medium-sized arteries are called **muscular arteries** because their tunica media contains more smooth muscle and fewer elastic fibers than elastic arteries.
- Thus, muscular arteries are capable of greater vasoconstriction and vasodilation to adjust the rate of blood flow.
- Muscular arteries span a range of sizes from the pencil-sized femoral and axillary arteries to string-sized arteries that enter organs→ Examples include the brachial artery in the arm and radial artery in the forearm.
- Because the muscular arteries continue to branch and ultimately distribute blood to each of the various organs, they are called **distributing arteries**.
- Because of the reduced amount of elastic tissue in the walls of muscular arteries, these vessels do not have the ability to recoil and help propel the blood like the elastic arteries. Instead, the thick, muscular tunica media is primarily responsible for the functions of the muscular arteries. The ability of the muscle to contract and maintain a state of partial contraction is referred to as *vascular tone*. Vascular tone stiffens the vessel wall and is important in maintaining vessel pressure and efficient blood flow.

Anastomoses ????

- Most parts of the body receive blood from branches of more than one artery, and where two or more arteries supply the same region, they usually connect.
- These connections, called **anastomoses** (a-nas-to»-MO» -se»s), provide **alternate routes**, called **collateral circulation**, for blood to reach a particular organ or tissue.
- The myocardium contains many anastomoses that connect branches of a given coronary artery or extend between branches of different coronary arteries. They provide detours for arterial blood if a main route becomes obstructed. Thus, heart muscle may receive sufficient oxygen even if one of its coronary arteries is partially blocked.
- Anastomoses may also occur between veins and between arterioles and venules. Arteries that do not anastomose are known as **end arteries**.
- Obstruction of an end artery interrupts the blood supply to a whole segment of an organ, producing necrosis (death) of that segment. Alternative blood routes may also be provided by non-anastomosing vessels that supply the same region of the body.

Anastomoses

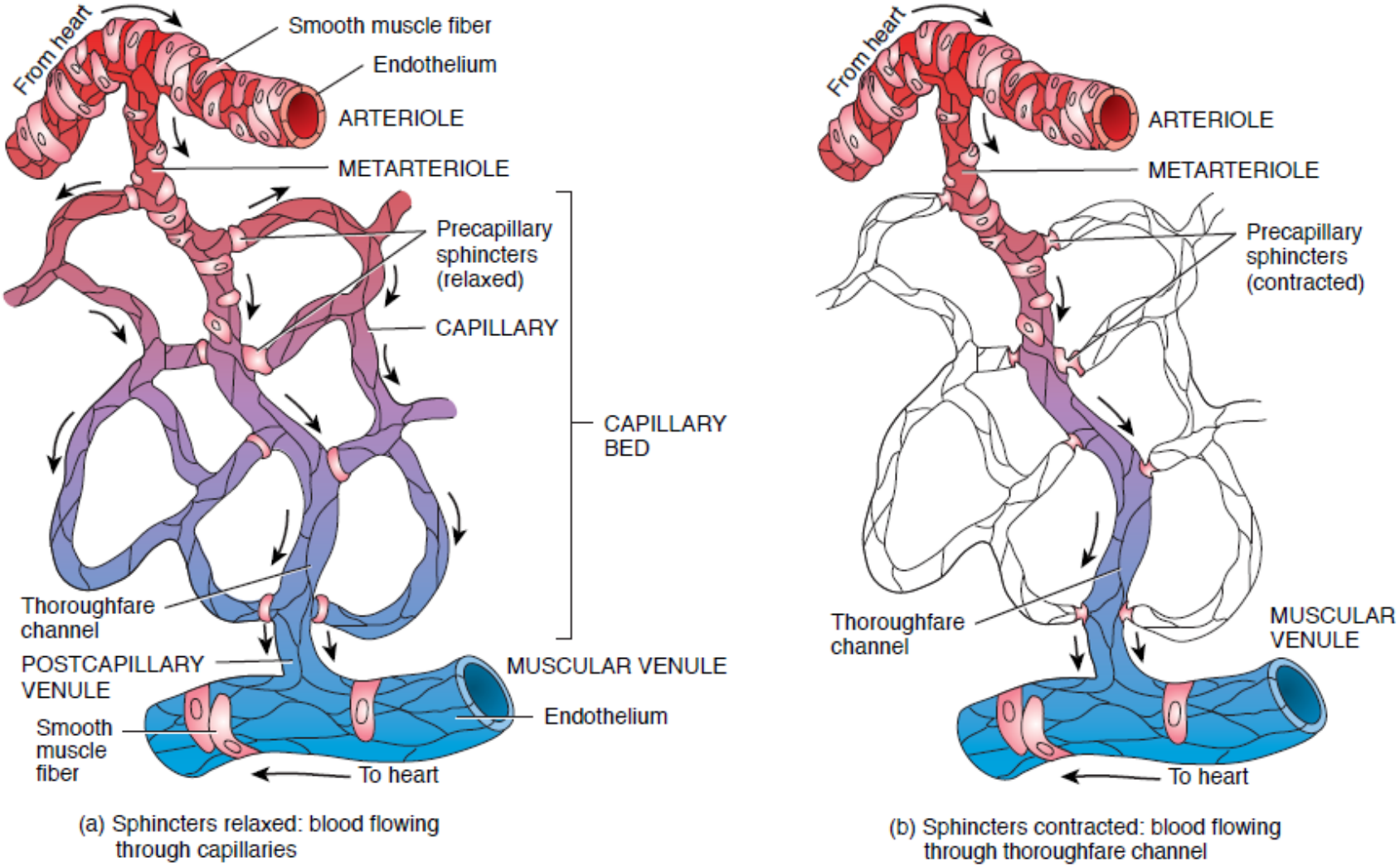


(c) Anterior view of inferior mesenteric artery and its branches

2- Arterioles

- Literally meaning small arteries, **arterioles** are abundant microscopic vessels that regulate the flow of blood into the capillary networks of the body's tissues
- The terminal end of the arteriole, the region called the **metarteriole** (met-ar-TE»R-e»-o» l; *meta* after), tapers toward the capillary junction. At the metarteriole–capillary junction, the distalmost muscle cell forms the **precapillary sphincter** (SFINGK-ter to bind tight), which monitors the blood flow into the capillary; the other muscle cells in the arteriole regulate the resistance (opposition) to blood flow (see Figure 21.3).
- The tunica externa of the arteriole consists of areolar connective tissue containing abundant unmyelinated sympathetic nerves. This sympathetic nerve supply, along with the actions of local chemical mediators, can alter the diameter of arterioles and thus vary the rate of blood flow and resistance through these vessels. Arterioles play a key role in regulating blood flow from arteries into capillaries by regulating **resistance,** the opposition to blood flow due to friction between blood and the walls of blood vessels.
- Because of this they are known as *resistance vessels*.

Figure 21.3 Arterioles, capillaries, and venules. Precapillary sphincters regulate the flow of blood through capillary beds.
→ In capillaries, nutrients, gases, and wastes are exchanged between the blood and interstitial fluid.



3- Capillaries

- **Capillaries**, the smallest of blood vessels, have diameters of 5–10 m, and form the U-turns that connect the arterial outflow to the venous return (Figure 21.3). Since red blood cells have a diameter of 8 m, they must often fold on themselves in order to pass single file through the lumens of these vessels. Capillaries form an extensive network, approximately 20 billion in number, of short (hundreds of micrometers in length), branched, interconnecting vessels that course among the individual cells of the body.
- This network forms an enormous surface area to make contact with the body's cells. The flow of blood from a metarteriole through capillaries and into a **postcapillary venule** (venule that receives blood from a capillary) is called the **microcirculation** (*micro* small) of the body.
- The primary function of capillaries is the exchange of substances between the blood and interstitial fluid. Because of this, these thin-walled vessels are referred to as *exchange vessels*.

Capillaries –continued

- The structure of capillaries is well suited to their function as exchange vessels because they lack both a tunica media and a tunica externa.
- Because capillary walls are composed of only a single layer of endothelial cells (see Figure 21.1e) and a basement membrane, a substance in the blood must pass through just one cell layer to reach the interstitial fluid and tissue cells.
- Exchange of materials occurs only through the walls of capillaries and the beginning of venules; the walls of arteries, arterioles, most venules, and veins present too thick a barrier.
- Capillaries form extensive branching networks that increase the surface area available for rapid exchange of materials.
- In most tissues, blood flows through only a small part of the capillary network when metabolic needs are low. However, when a tissue is active, such as contracting muscle, the entire capillary network fills with blood. Throughout the body, capillaries function as part of a **capillary bed** (Figure 21.3), a network of 10–100 capillaries that arises from a single metarteriole.

4- Venules

- Unlike their thick-walled arterial counterparts, **venules** and veins have thin walls that do not readily maintain their shape.
- Venules drain the capillary blood and begin the return flow of blood back toward the heart (see Figure 21.3).
- Venules that initially receive blood from capillaries are called **postcapillary venules**. They are the smallest venules, measuring 10 m to 50 m in diameter, and have loosely organized intercellular junctions (the weakest endothelial contacts encountered along the entire vascular tree) and thus are very porous.
- They function as significant sites of exchange of nutrients and wastes and white blood cell emigration, and for this reason form part of the microcirculatory exchange unit along with the capillaries.
- As the postcapillary venules move away from capillaries, they acquire one or two layers of circularly arranged smooth muscle cells.
- These **muscular venules** (50 m to 200 m) have thicker walls across which exchanges with the interstitial fluid can no longer occur.
- The thin walls of the postcapillary and muscular venules are the most distensible elements of the vascular system; this allows them to expand and serve as excellent reservoirs for accumulating large volumes of blood.
- Blood volume increases of 360% have been measured in the postcapillary and muscular venules.

5- Veins

- While **veins** do show structural changes as they increase in size from small to medium to large, the structural changes are not as distinct as they are in arteries.
- Veins, in general, have very thin walls relative to their total diameter (average thickness is less than one-tenth of the vessel diameter). They range in size from 0.5 mm in diameter for small veins to 3 cm in the large superior and interior venae cavae entering the heart.
- Although veins are composed of essentially the same three layers as arteries, the relative thicknesses of the layers are different.
- Veins lack the internal or external elastic laminae found in arteries
- They are distensible enough to adapt to variations in the volume and pressure of blood passing through them, but are not designed to withstand high pressure.
- The lumen of a vein is larger than that of a comparable artery, and veins often appear collapsed (flattened) when sectioned.

Veins -2

- The pumping action of the heart is a major factor in moving venous blood back to the heart.
- The contraction of skeletal muscles in the lower limbs also helps boost venous return to the heart (see Figure 21.9). The average blood pressure in veins is considerably lower than in arteries. The difference in pressure can be noticed when blood flows from a cut vessel. Blood leaves a cut vein in an even, slow flow but spurts rapidly from a cut artery.
- Most of the structural differences between arteries and veins reflect this pressure difference. For example, the walls of veins are not as strong as those of arteries.
- Many veins, especially those in the limbs, also contain **valves**, thin folds of tunica interna that form flaplike cusps. The valve cusps project into the lumen, pointing toward the heart (Figure 21.5).

Veins -3

- The low blood pressure in veins allows blood returning to the heart to slow and even back up; the valves aid in venous return by preventing the backflow of blood.

- A **vascular** (*venous*) **sinus** is a vein with a thin endothelial wall that has no smooth muscle to alter its diameter. In a vascular sinus, the surrounding dense connective tissue replaces the tunica media and tunica externa in providing support. For example, dural venous sinuses, which are supported by the dura mater, convey deoxygenated blood from the brain to the heart. Another example of a vascular sinus is the coronary sinus of the heart.

Blood Distribution

- The largest portion of your blood volume at rest—about 64%—is in systemic veins and venules. Systemic arteries and arterioles hold about 13% of the blood volume, systemic capillaries hold about 7%, pulmonary blood vessels hold about 9%, and the heart holds about 7%.
- Because systemic veins and venules contain a large percentage of the blood volume, they function as **blood reservoirs** from which blood can be diverted quickly if the need arises.
- For example, during increased muscular activity, the cardiovascular center in the brain stem sends a larger number of sympathetic impulses to veins. The result is *venoconstriction*, constriction of veins, which reduces the volume of blood in reservoirs and allows a greater blood volume to flow to skeletal muscles, where it is needed most.
- A similar mechanism operates in cases of hemorrhage, when blood volume and pressure decrease; in this case, venoconstriction helps counteract the drop in blood pressure. Among the principal blood reservoirs are the veins of the abdominal organs (especially the liver and spleen) and the veins of the skin.

Distinguishing Features of Blood Vessels-1

BLOOD VESSEL	SIZE	TUNICA INTERNA	TUNICA MEDIA	TUNICA EXTERNA	FUNCTION
Elastic arteries	Largest arteries in the body.	Well-defined internal elastic lamina.	Thick and dominated by elastic fibers; well-defined external elastic lamina.	Thinner than tunica media.	Conduct blood from heart to muscular arteries.
Muscular arteries	Medium-sized arteries.	Well-defined internal elastic lamina.	Thick and dominated by smooth muscle; thin external elastic lamina.	Thicker than Tunica media.	Distribute blood to arterioles.

Distinguishing Features of Blood Vessels-2

BLOOD VESSEL	SIZE	TUNICA INTERNA	TUNICA MEDIA	TUNICA EXTERNA	FUNCTION
Arterioles	Microscopic (15–300 m In diameter).	Thin with a fenestrated internal elastic lamina that disappears distally.	One or two layers of circularly oriented smooth muscle; distalmost smooth muscle cell forms A precapillary sphincter.	Loose collagenous connective tissue and sympathetic nerves.	Deliver blood to capillaries and help regulate blood flow from arteries to capillaries.
Capillaries	Microscopic; smallest blood vessels (5–10 m in diameter).	Endotheliu m and basement membrane.	None.	None.	Permit exchange of nutrients and wastes between blood and interstitial fluid; distribute blood to postcapillary venules.

Distinguishing Features of Blood Vessels- 3

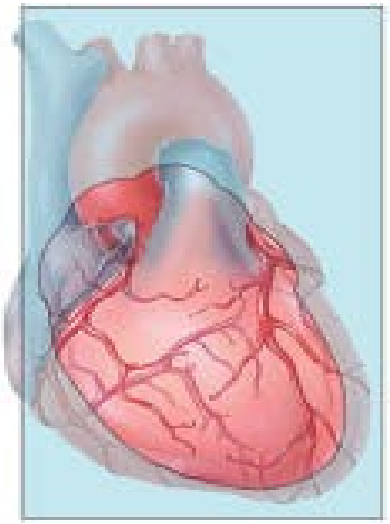
BLOOD VESSEL	SIZE	TUNICA INTERNA	TUNICA MEDIA	TUNICA EXTERNA	FUNCTION
Postcapillary venules	Microscopic (10–50 m in diameter).	Endothelium and basement membrane.	None.	Sparse.	Pass blood into muscular venules; permit exchange of nutrients and wastes between blood and interstitial fluid and function in white blood Cell emigration.

Distinguishing Features of Blood Vessels- 4

BLOOD VESSEL	SIZE	TUNICA INTERNA	TUNICA MEDIA	TUNICA EXTERNA	FUNCTION
Muscular venules	Microscopic (50–200 m in diameter).	Endothelium and basement membrane.	One or two layers of circularly oriented smooth muscle.	Sparse.	Pass blood into vein; act as reservoirs for accumulating large volumes of blood (along with postcapillary venules).

Distinguishing Features of Blood Vessels- 5

BLOOD VESSEL	SIZE	TUNICA INTERNA	TUNICA MEDIA	TUNICA EXTERNA	FUNCTION
Veins	Range from 0.5 mm to 3 cm in diameter.	Endothelium and basement membrane; no internal elastic lamina; contain valves; lumen much larger than in accompanying artery.	Much thinner than In arteries; no external elastic lamina.	Thickest of the three layers.	Return blood to heart, facilitated by valves in limb veins.



PHYSIOLOGY OF CARDIOVASCULAR SYSTEM

Course Name: Anatomy and Physiology 2

Course Code: 0521215

Lecturer: Ms. Asma El-Shara'. MPH

Faculty Of Pharmacy,

Philadelphia University-Jordan

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Philadelphia University

MAIN FUNCTIONS OF THE CIRCULATORY SYSTEM

1. 1. Transport and distribute essential substances to the tissues.
2. Remove metabolic byproducts.
3. Adjustment of oxygen and nutrient supply in different physiologic states.
4. Regulation of body temperature.
5. Endocrine function by releasing atrial natriuretic peptide (ANP)→ ANP is a powerful vasodilator released by atrial myocytes in response to high blood pressure.

The Cardiac Conduction System

OBJECTIVES

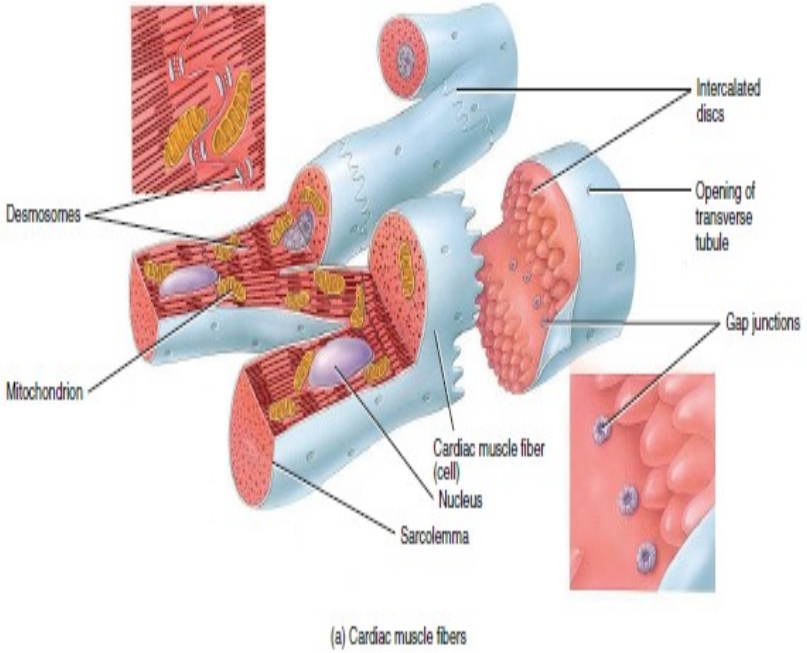
- Describe the structural and functional characteristics of cardiac muscle tissue and the cardiac conduction system.
- Explain how an action potential occurs in cardiac contractile fibers.
- Describe the electrical events of a normal electrocardiogram (ECG).

The Heart Histology

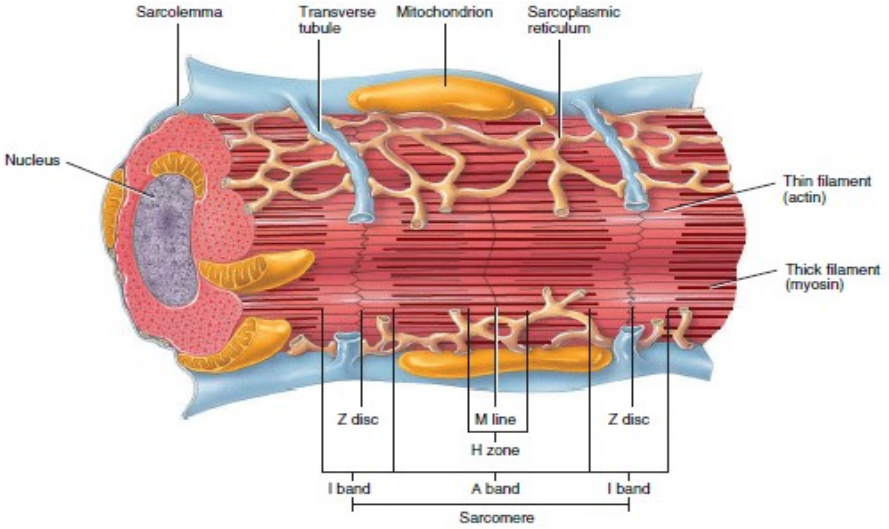
Anatomical Properties of Cardiac Muscle Fibers

- Involuntary
- Branched and interdigitated
- Cell-cell cohesion forming what is called intercalated disk
- Contain one nucleus
- Contain many mitochondria
- Striated

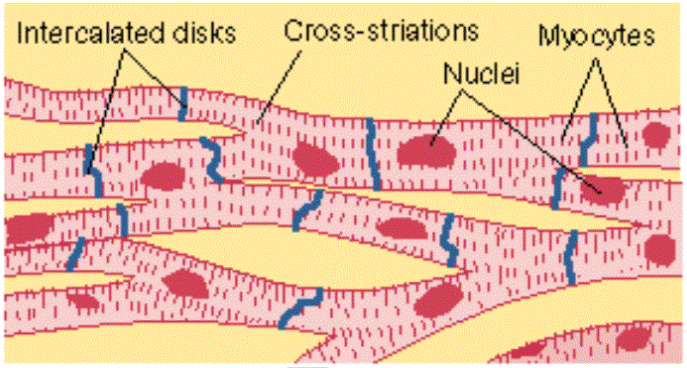
Histology of cardiac muscle tissue



(a) Cardiac muscle fibers



(b) Arrangement of components in a cardiac muscle fiber



Note: The sarcoplasmic reticulum of cardiac muscle fibers is somewhat smaller than the SR of skeletal muscle fibers. As a result, cardiac muscle has a smaller intracellular reserve of Ca^{2+}.

Histology of cardiac muscle tissue (continued-1)

- The ends of cardiac muscle fibers connect to neighboring fibers by irregular transverse thickenings of the sarcolemma called **intercalated discs** (in-TER-ka-la»t-ed; *intercalat-* to insert between).

The Membrane junctions:

- The discs contain **desmosomes**, which hold the fibers together, and **gap junctions**, which allow muscle action potentials to conduct from one muscle fiber to its neighbors.

- ***Gap junctions allow the entire myocardium of the atria or the ventricles to contract as a single, coordinated unit.***

Histology of cardiac muscle tissue (continued-2)

- Specialized conduction system facilitates and coordinates transmission of electrical excitation from the atria to the ventricles to ensure synchronization between atrial and ventricular pumping.

- The atria and ventricles each secrete a hormone involved in regulation of blood pressure… act on kidney

Autorhythmic Fibers: The Conduction System

- An inherent and rhythmical electrical activity is the reason for the heart's lifelong beat.
- The source of this electrical activity is a network of specialized cardiac muscle fibers called **autorhythmic fibers** (aw-to»-RITH-mik; *auto-* self) because they are self-excitable.
- Autorhythmic fibers repeatedly generate action potentials that trigger heart contractions. They continue to stimulate a heart to beat even after it is removed from the body—for example, to be transplanted into another person—and all of its nerves have been cut.

Autorhythmic Fibers: The Conduction System (continued-1)

- During embryonic development, only about 1% of the cardiac muscle fibers become autorhythmic fibers; these relatively rare fibers have **two important functions:**

1. They act as a **pacemaker**, setting the rhythm of electrical excitation that causes contraction of the heart.

2. They form the **cardiac conduction system**, a network of specialized cardiac muscle fibers that provide a path for each cycle of cardiac excitation to progress through the heart. The conduction system ensures that cardiac chambers become stimulated to contract in a coordinated manner, which makes the heart an effective pump.

Autorhythmic Fibers: The Conduction System (continued-2)

Cardiac action potentials propagate through the conduction system in the following sequence (Figure 20.10a):

FIRST:

- ✓Cardiac excitation normally begins in the **sinoatrial (SA) node**, located in the right atrial wall just inferior and lateral to the opening of the superior vena cava.
- ✓SA node cells do not have a stable resting potential. Rather, they repeatedly depolarize to threshold spontaneously.
- ✓The spontaneous depolarization is a **pacemaker potential**. When the pacemaker potential reaches threshold, it triggers an action potential (Figure 20.10b).
- ✓Each action potential from the SA node propagates throughout both atria via gap junctions in the intercalated discs of atrial muscle fibers.
- ✓Following the action potential, the two atria contract at the same time.

SECOND

- ✓By conducting along atrial muscle fibers, the action potential reaches the **atrioventricular (AV) node**, located in the interatrial septum, just anterior to the opening of the coronary sinus (Figure 20.10a).
- ✓At the AV node, the action potential slows considerably as a result of various differences in cell structure in the AV node.
- ✓This delay provides time for the atria to empty their blood into the ventricles.

THIRD

- From the AV node, the action potential enters the **atrioventricular (AV) bundle** (also known as the *bundle of His*, pronounced HIZ).
- This bundle is the only site where action potentials can conduct from the atria to the ventricles. (Elsewhere, the fibrous skeleton of the heart electrically insulates the atria from the ventricles.)

FOURTH

- ✓After propagating through the AV bundle, the action potential enters both the **right** and **left bundle branches**.

- ✓The bundle branches extend through the interventricular septum toward the apex of the heart.

FIFTH

- ✓Finally, the large-diameter **Purkinje fibers** (pur-KIN-je») rapidly conduct the action potential beginning at the apex of the heart upward to the remainder of the ventricular myocardium.

- ✓Then the ventricles contract, pushing the blood upward toward the semilunar valves.

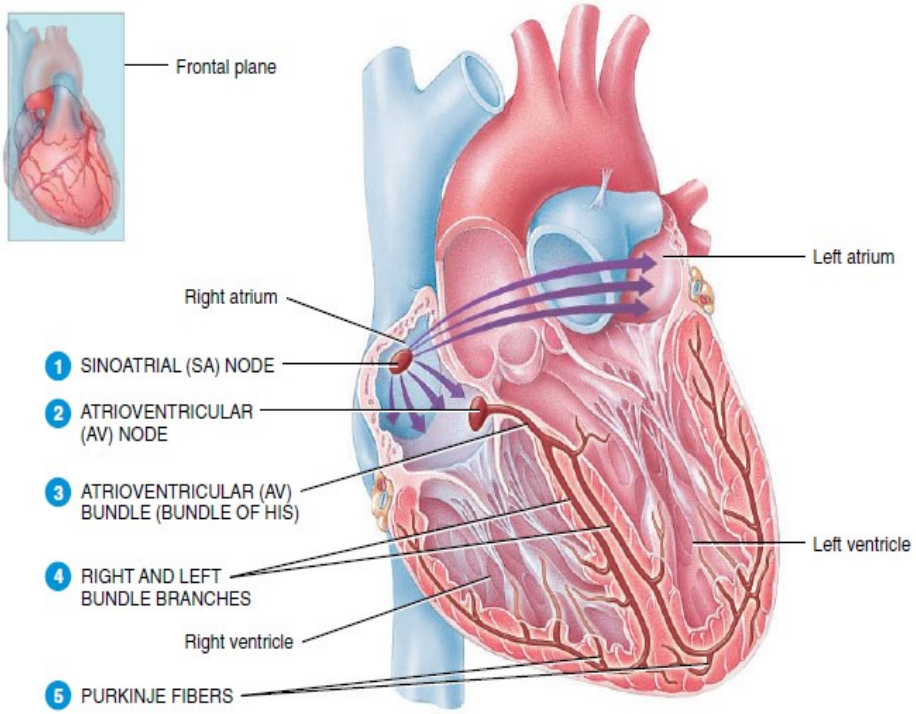
- On their own, autorhythmic fibers in the SA node would initiate an action potential about every 0.6 second, or 100 times per minute. Thus, the SA node sets the rhythm for contraction of the heart—it is the *natural pacemaker*. This rate is faster than that of any other autorhythmic fibers.

- Because action potentials from the SA node spread through the conduction system and stimulate other areas before the other areas are able to generate an action potential at their own, slower rate, the SA node acts as the natural pacemaker of the heart.

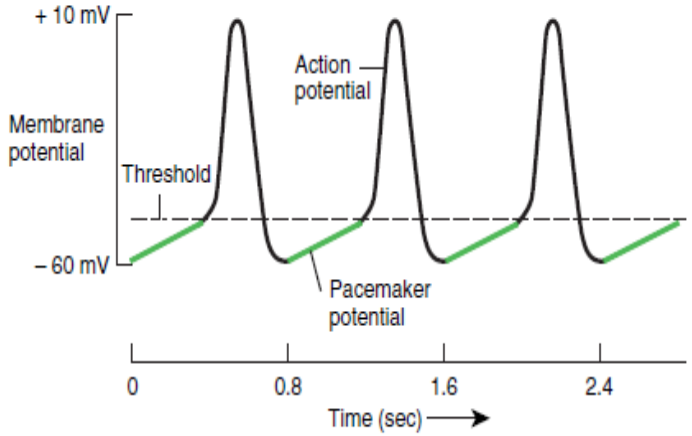
- Nerve impulses from the autonomic nervous system (ANS) and blood-borne hormones (such as epinephrine) *modify the timing and strength* of each heartbeat, but they *do not establish the fundamental rhythm*.

- In a person at rest, for example, acetylcholine released by the parasympathetic division of the ANS slows SA node pacing to about every 0.8 second or 75 action potentials per minute (Figure 20.10b).

Figure 20.10 The conduction system of the heart. Autorhythmic fibers in the SA node, located in the right atrial wall (a), act as the heart's pacemaker, initiating cardiac action potentials (b) that cause contraction of the heart's chambers.

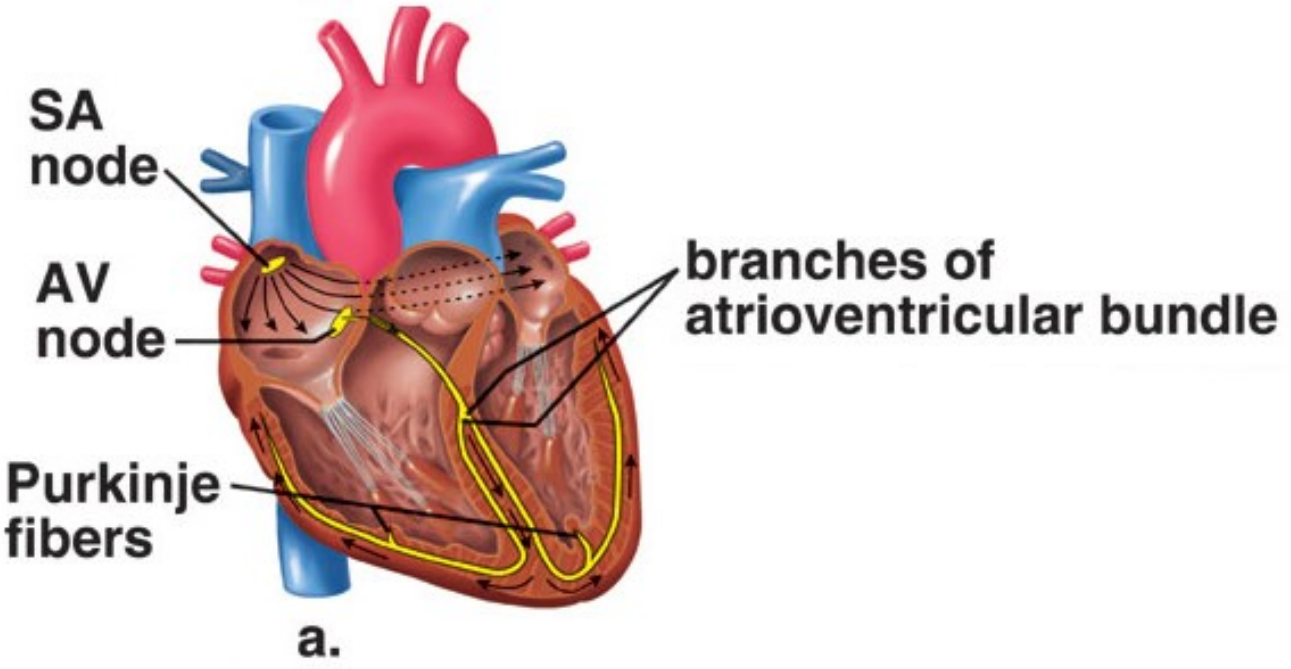


(a) Anterior view of frontal section



(b) Pacemaker potentials (green) and action potentials (black) in autorhythmic fibers of SA node

Conduction system of the heart



- The rate for the SA node is 70–80
- The rate for the AV node is 40–60
- The rate for the Bundle of His and Purkinje fibers is 20–40

Action Potential and Contraction of Contractile Fibers

The action potential initiated by the SA node travels along the conduction system and spreads out to excite the "working" atrial and ventricular muscle fibers, called **contractile fibers**. An action potential occurs in a contractile fiber as follows

Figure 20.11 Action potential in a ventricular contractile fiber. The resting membrane potential is about 90 mV *(A long refractory period prevents tetanus in cardiac muscle fibers.)*

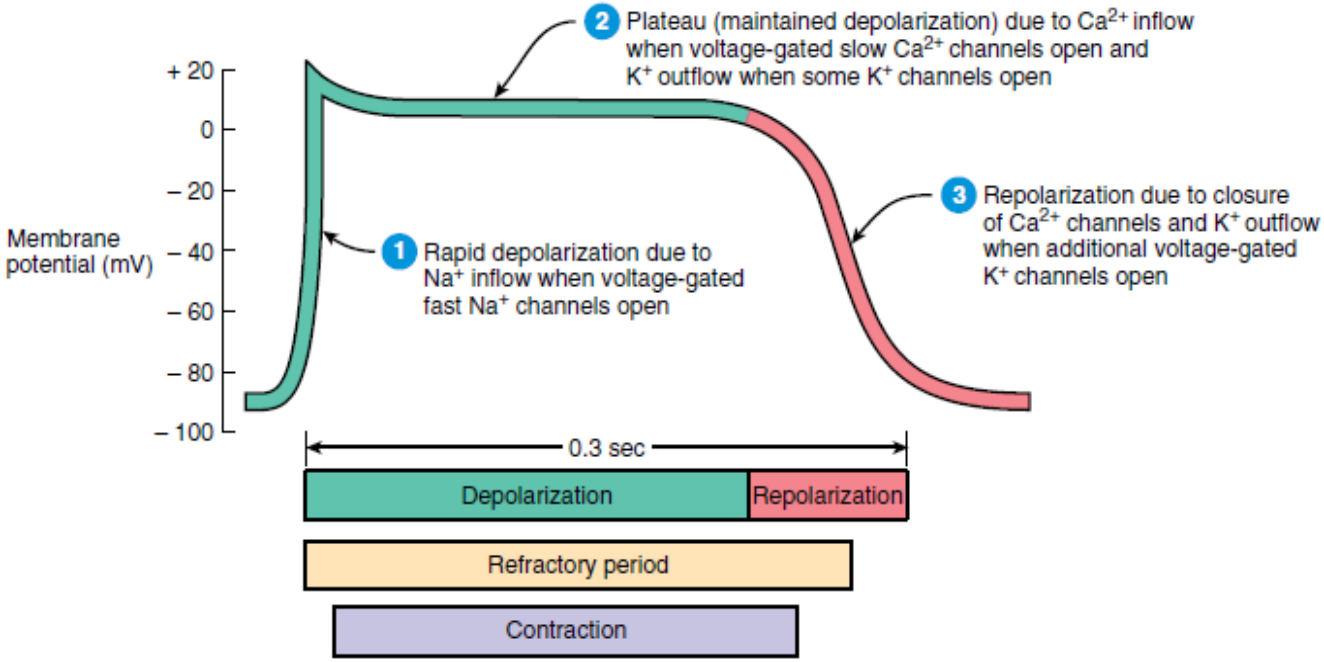
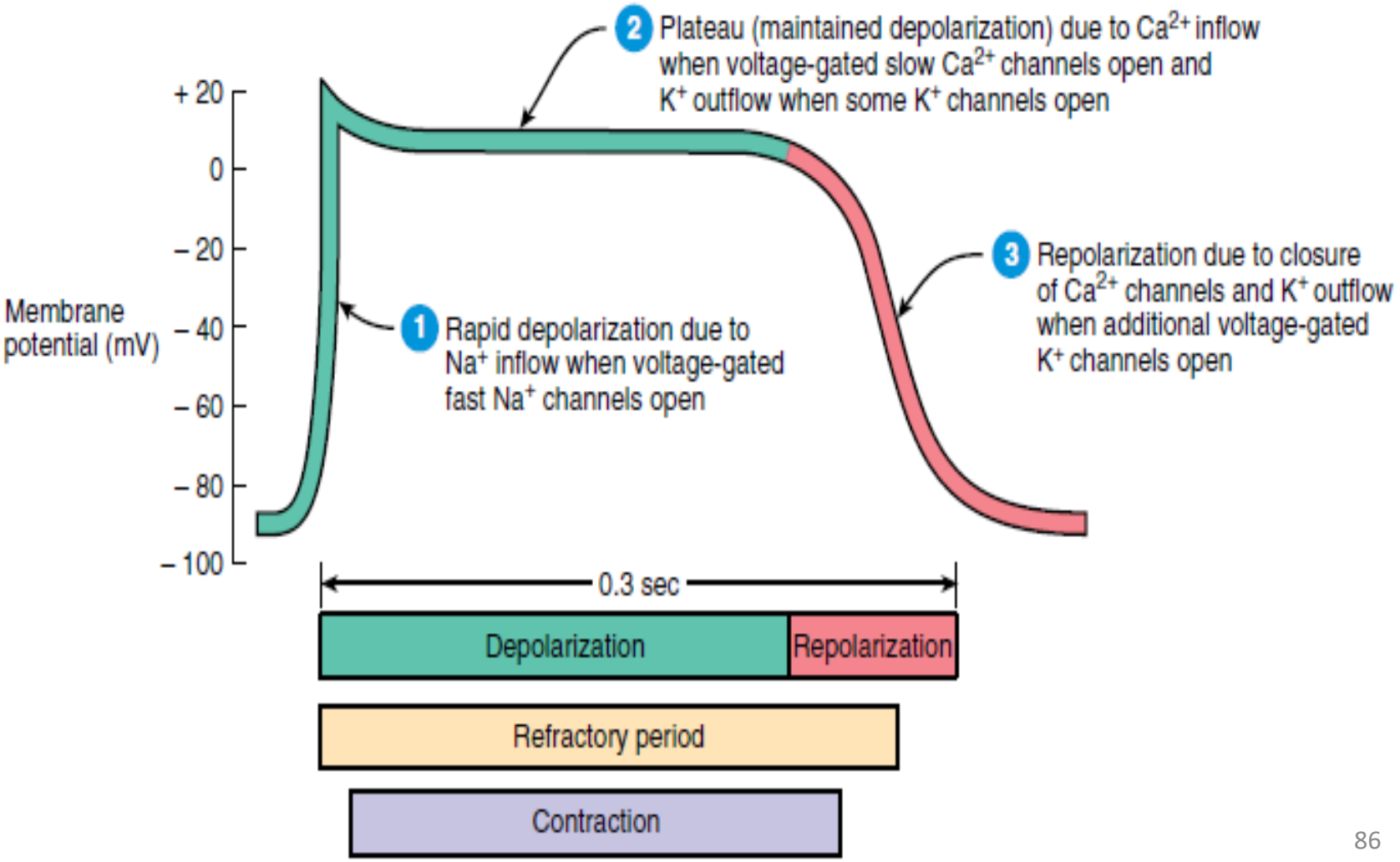


Figure 20.11 Action potential in a ventricular contractile fiber. The resting membrane potential is about 90 mV *(A long refractory period prevents tetanus in cardiac muscle fibers.)*



The action potential of cardiac contractile cells shows a characteristic plateau.

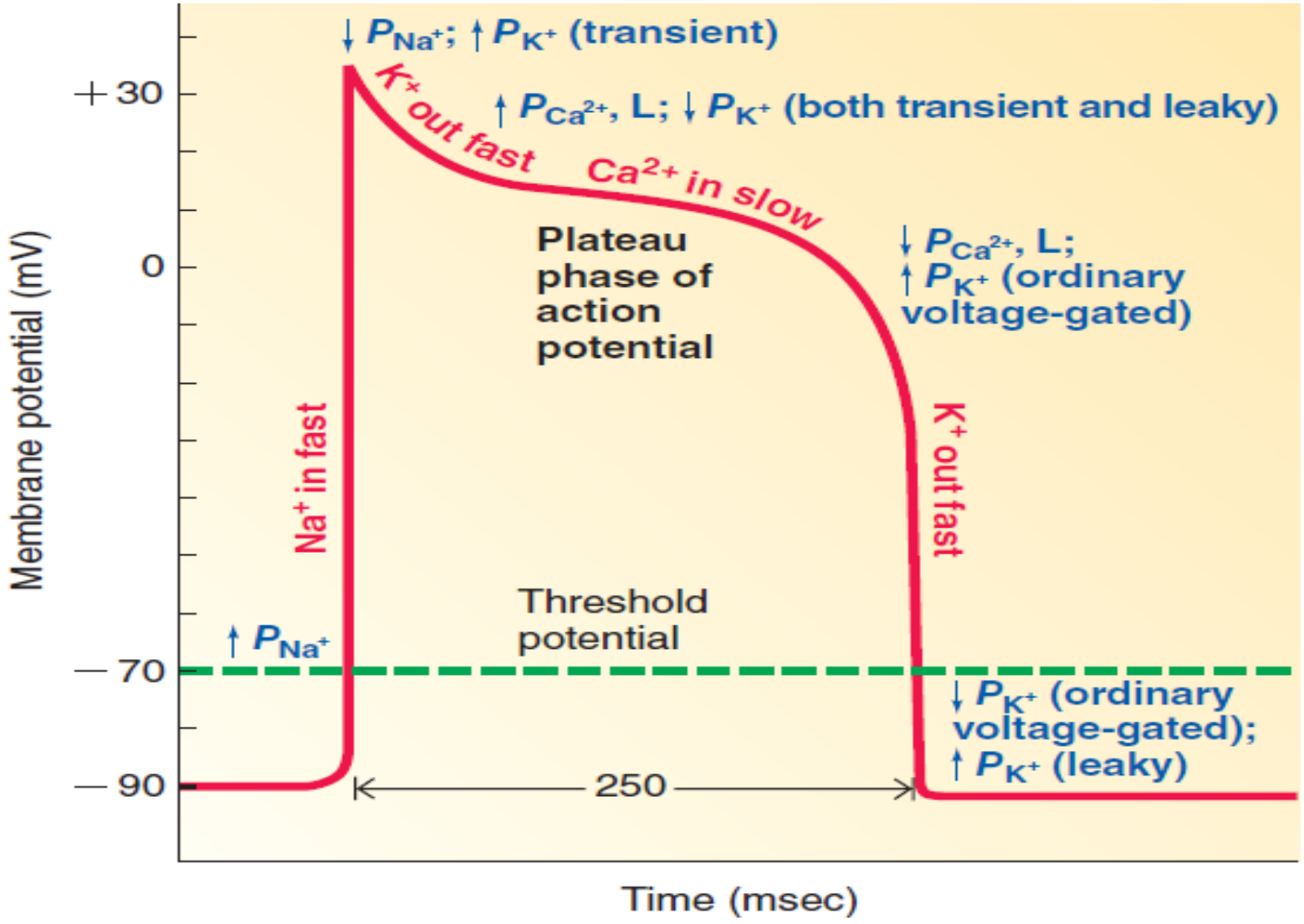


Figure 9-10 Action potential in cardiac contractile cells. The action poten

Action Potential and Contraction of Contractile Fibers (Continued)

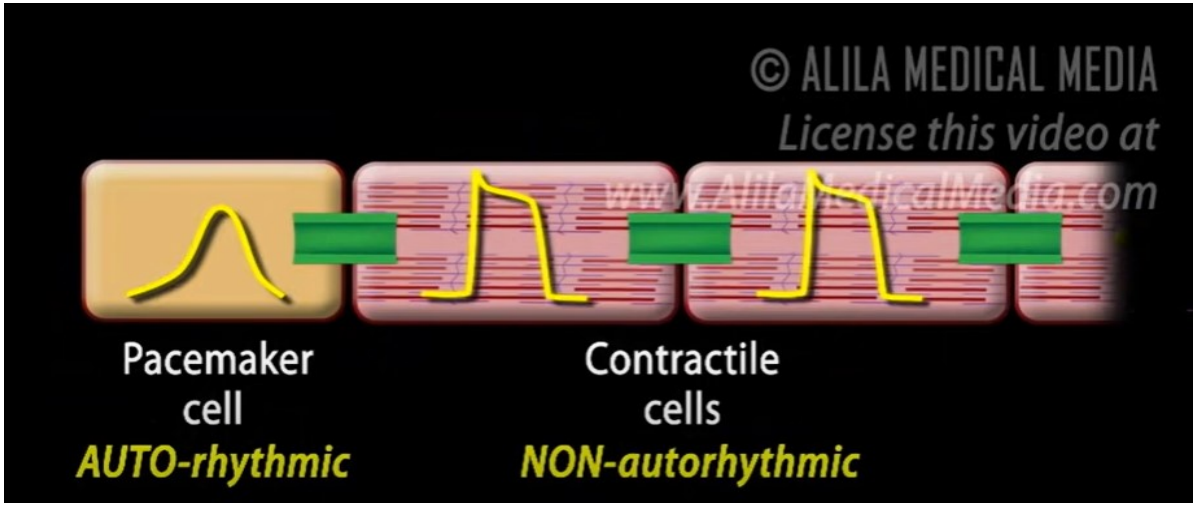
- The action potential initiated by the SA node travels along the conduction system and spreads out to excite the "working" atrial and ventricular muscle fibers, called **contractile fibers**. An action potential occurs in a contractile fiber as follows (Figure 20.11):

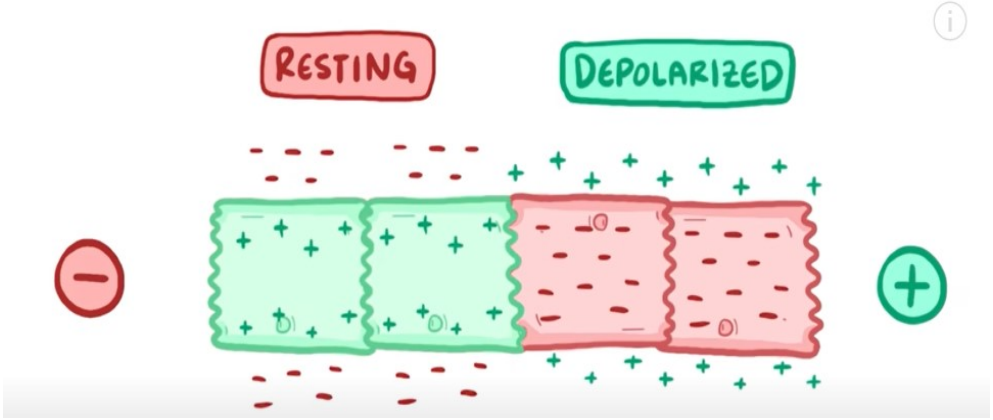
1- Depolarization.

2- Plateau.

3- Repolarization.

Action Potential Difference between autorythmic fiber and contractile myocytes





First… Depolarization

- Unlike autorhythmic fibers, contractile fibers have a stable resting membrane potential that is close to -90 mV.
- When a contractile fiber is brought to threshold by an action potential from neighboring fibers, its **voltage-gated fast Na channels** open. These sodium ion channels are referred to as "fast" because they open very rapidly in response to a threshold-level depolarization.
- Opening of these channels allows Na^+ inflow because the cytosol of contractile fibers is electrically more negative than interstitial fluid and Na^+ concentration is higher in interstitial fluid.
- Inflow of Na^+ down the electrochemical gradient produces a **rapid depolarization** (de»-po»-lar-i-ZA» -shun).
- Within a few milliseconds, the fast Na channels automatically inactivate and Na inflow decreases.

Second… Plateau (1)

- The next phase of an action potential in a contractile fiber is the **plateau**, a period of maintained depolarization.
- It is due in part to opening of **voltage-gated slow Ca^{2+} channels** in the sarcolemma. When these channels open, calcium ions move from the interstitial fluid (which has a higher Ca^{2+} concentration) into the cytosol. This inflow of Ca^{2+} causes even more Ca^{2+} to pour out of the sarcoplasmic reticulum into the cytosol through additional Ca^{2+} channels in the sarcoplasmic reticulum membrane. The increased Ca^{2+} concentration in the cytosol ultimately triggers contraction.
- Several different types of **voltage-gated K^+ channels** are also found in the sarcolemma of a contractile fiber.

Second… Plateau (2)

- Just before the plateau phase begins, some of these K^+ channels open, allowing potassium ions to leave the contractile fiber. Therefore, depolarization is sustained during the plateau phase because Ca^{2+} inflow just balances K^+ outflow.

- The plateau phase lasts for about 0.25 sec, and the membrane potential of the contractile fiber is close to 0 mV.

- By comparison, depolarization in a neuron or skeletal muscle fiber is much briefer, about 1 msec (0.001 sec), because it lacks a plateau phase.

Third… Repolarization

- The recovery of the resting membrane potential during the **repolarization** (re»-po»-lar-i-ZA»-shun) phase of a cardiac action potential resembles that in other excitable cells. After a delay (which is particularly prolonged in cardiac muscle), additional voltage-gated K channels open.

- Outflow of K^+ restores the negative resting membrane potential (90 mV). At the same time, the calcium channels in the sarcolemma and the sarcoplasmic reticulum are closing, which also contributes to repolarization.

Similarity to skeletal muscle!!

- The mechanism of contraction is similar in cardiac and skeletal muscle: The electrical activity (action potential) leads to the mechanical response (contraction) after a short delay. As Ca^{2+} concentration rises inside a contractile fiber, Ca^{2+} binds to the regulatory protein troponin, which allows the actin and myosin filaments to begin sliding past one another, and tension starts to develop.

- Substances that alter the movement of Ca^{2+} through slow Ca^{2+} channels influence the strength of heart contractions. Epinephrine, for example, increases contraction force by enhancing Ca^{2+} flow into the cytosol.

What is a Refractory Period?

- In muscle, the **refractory period** (re-FRAK-to-re») is the time interval during which a second contraction cannot be triggered.

- The refractory period of a cardiac muscle fiber lasts longer than the contraction itself (Figure 20.11). As a result, another contraction cannot begin until relaxation is well under way. For this reason, tetanus (maintained contraction) cannot occur in cardiac muscle as it can in skeletal muscle. The advantage is apparent if you consider how the ventricles work. Their pumping function depends on alternating contraction (when they eject blood) and relaxation (when they refill). If heart muscle could undergo tetanus, blood flow would cease.

Electrocardiogram Introduction

- As action potentials propagate through the heart, they generate electrical currents that can be detected at the surface of the body.
- An **electrocardiogram** (e-lek-tro»-KAR-de»-o»-gram), abbreviated either ECG or EKG (from the German word *Elektrokardiogram*), is a recording of these electrical signals.
- The ECG is a composite record of action potentials produced by all of the heart muscle fibers during each heartbeat. The instrument used to record the changes is an **electrocardiograph**.

Electrocardiogram (continued-1)

The electrocardiograph amplifies the heart's electrical signals and produces 12 different tracings from different combinations of limb and chest leads. Each limb and chest electrode records slightly different electrical activity because of the difference in its position relative to the heart.

By comparing these records with one another and with normal records, it is possible to determine:

(1) if the conducting pathway is abnormal,

(2) if the heart is enlarged,

(3) if certain regions of the heart are damaged, and

(4) the cause of chest pain.

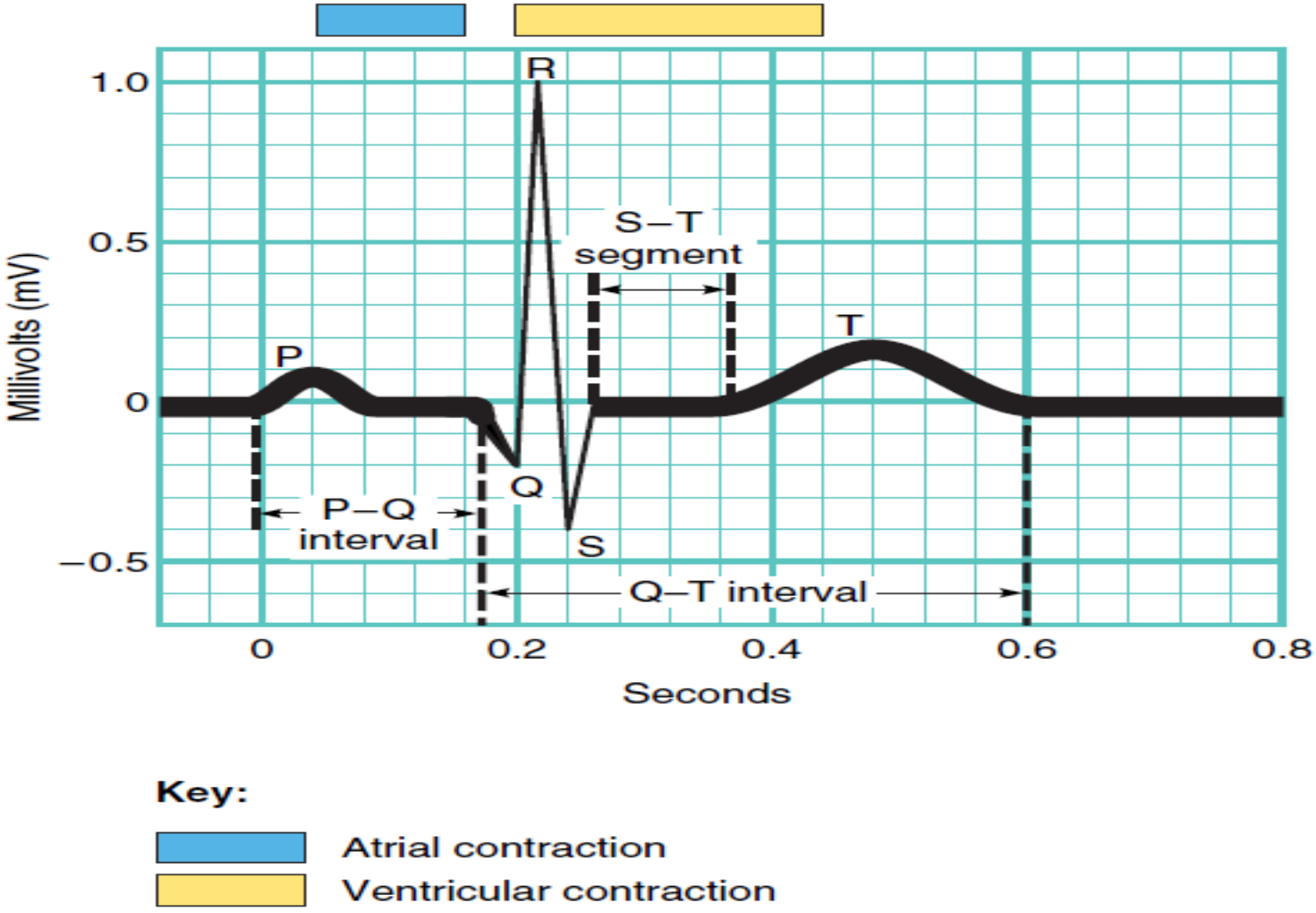
Electrocardiogram (continued-2)

- The components of a normal ECG waves:

1- P wave → atrial depolarization

2- QRS complex → ventricular depolarization
→ onset of ventricular contraction

3- T wave → ventricular repolarization
→ just before ventricles start to relax



So… where is the Atrial repolarization?
Atrial repolarization is usually not visible → masked by larger QRS complex

Analysis of an ECG

Analysis of an ECG also involves measuring the time spans between waves, which are called **intervals** or *segments*

The size of the waves can provide clues to abnormalities.

Examples of abnormalities:

- Larger P waves indicate enlargement of an atrium;
- An enlarged Q wave may indicate a myocardial infarction;
- An enlarged R wave generally indicates enlarged ventricles.
- The T wave is flatter than normal when the heart muscle is receiving insufficient oxygen
- The T wave may be elevated in hyperkalemia (high blood K^+ level), in coronary artery disease.

Analysis of an ECG: (P-Q interval)

- The **P–Q interval** is the time from the beginning of the P wave to the beginning of the QRS complex.
- It represents the conduction time from the beginning of atrial excitation to the beginning of ventricular excitation.
- The P–Q interval is the time required for the action potential to travel through the atria, atrioventricular node, and the remaining fibers of the conduction system.
- ***Examples of abnormalities:***

As the action potential is forced to detour around scar tissue caused by disorders such as coronary artery disease and rheumatic fever, the P–Q interval lengthens.

Analysis of an ECG: (S–T segment)

The **S–T segment**, which begins at the end of the S wave and ends at the beginning of the T wave, represents the time when the ventricular contractile fibers are depolarized during the plateau phase of the action potential.

Examples of abnormalities:

1- The S–T segment is elevated (above the baseline) in acute myocardial infarction

2- The S–T segment is depressed (below the baseline) when the heart muscle receives insufficient oxygen.

Analysis of an ECG: (Q–T interval)

- The **Q–T interval** extends from the start of the QRS complex to the end of the T wave.

- It is the time from the beginning of ventricular depolarization to the end of ventricular repolarization.

Examples of abnormalities:

The Q–T interval may be lengthened by myocardial damage, myocardial ischemia (decreased blood flow), or conduction abnormalities.

Correlation of ECG Waves with Atrial and Ventricular Systole

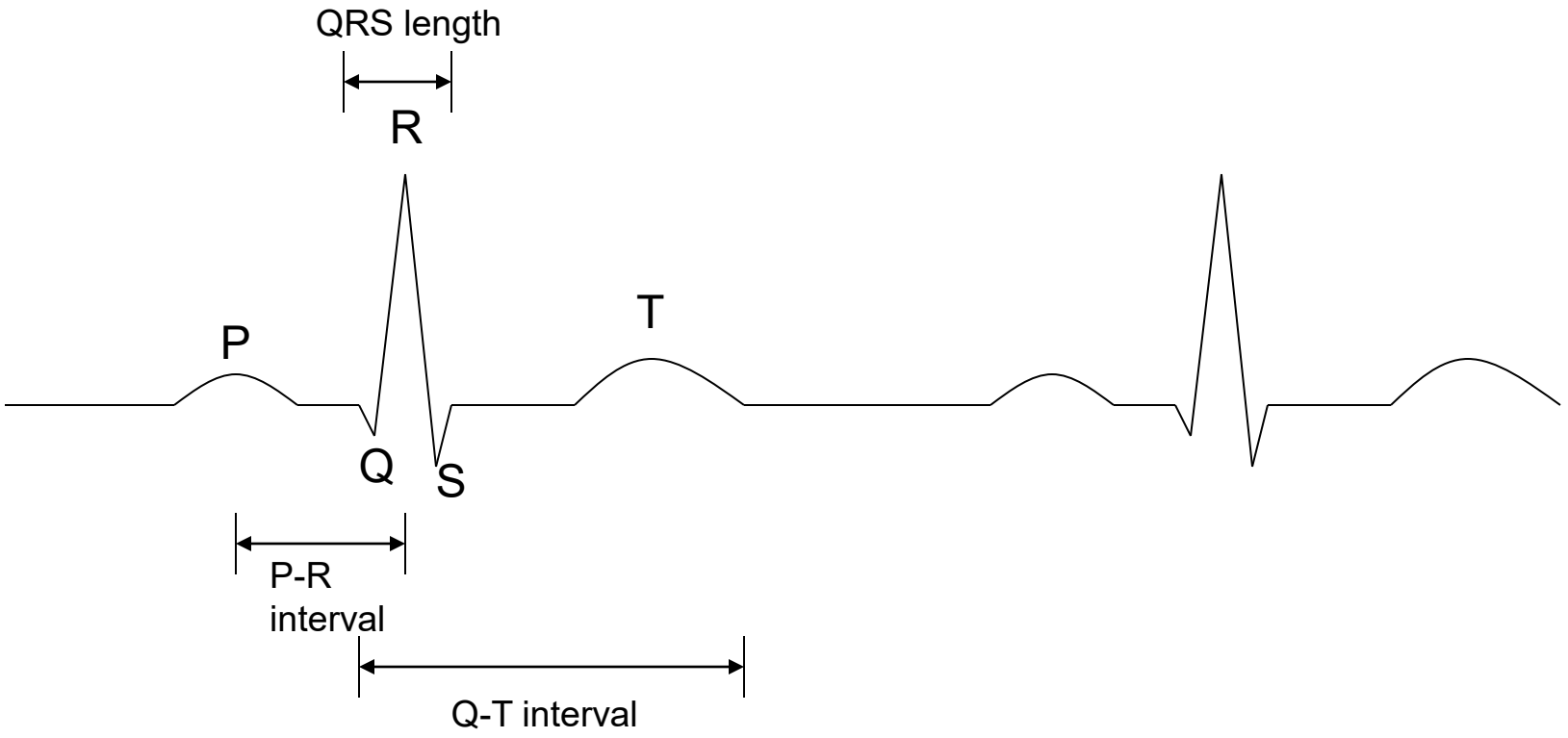
- The atria and ventricles depolarize and then contract at different times because the conduction system routes cardiac action potentials along a specific pathway.

- The term **systole** (SIS-to¯-le» contraction) refers to the phase of contraction

- The term **diastole** (dı»-AS-to¯-le» dilation or expansion); refers to phase of relaxation.

- The ECG waves predict the timing of atrial and ventricular systole and diastole. At a heart rate of 75 beats per minute, the timing is as shown in (Figure 20.13) and in the following slide.

ECG Waves and Intervals



Normal: PR interval: 0.12-0.2 sec
QRS length: <0.10 sec
QT interval: 0.3-0.4 sec

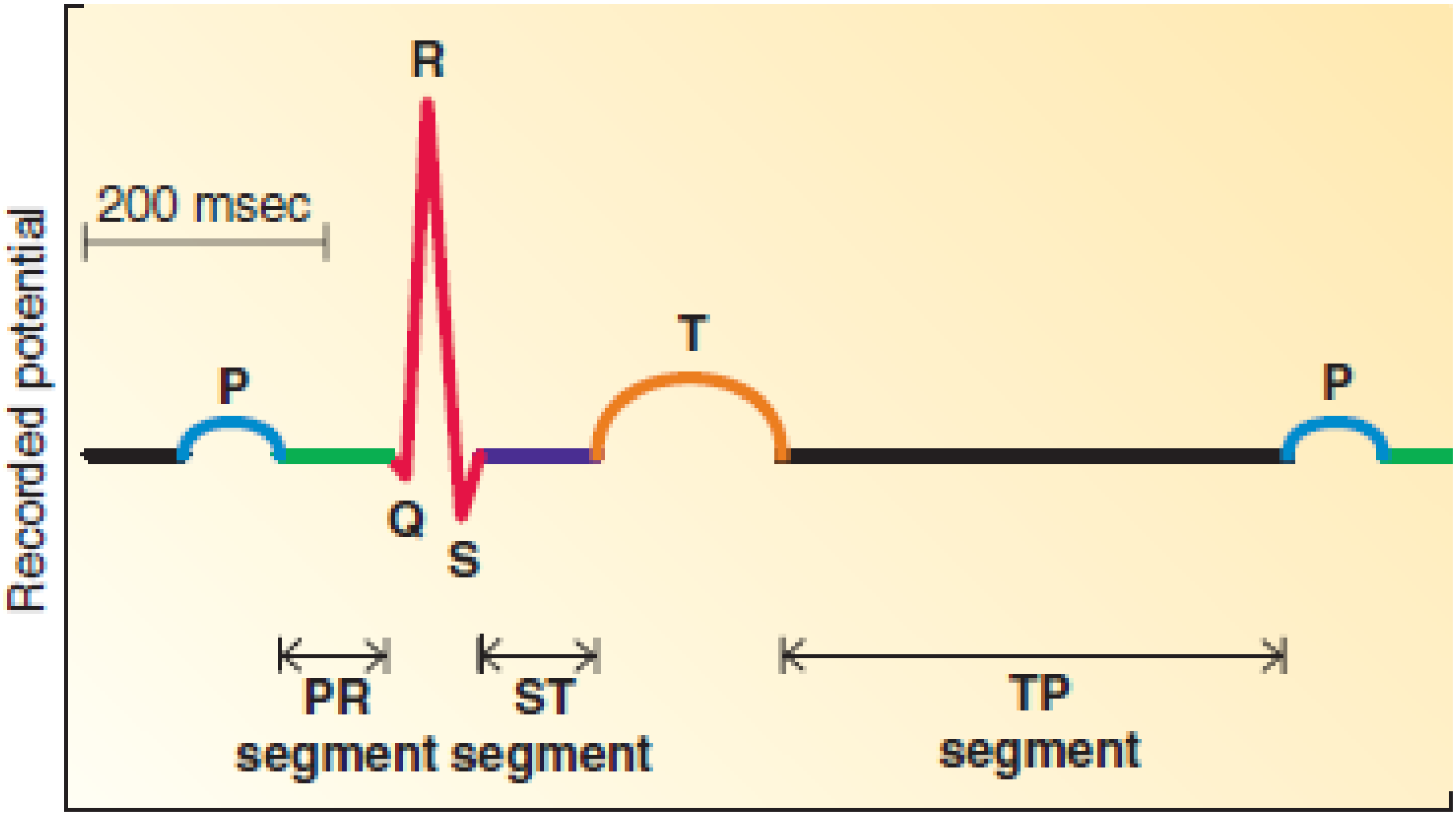
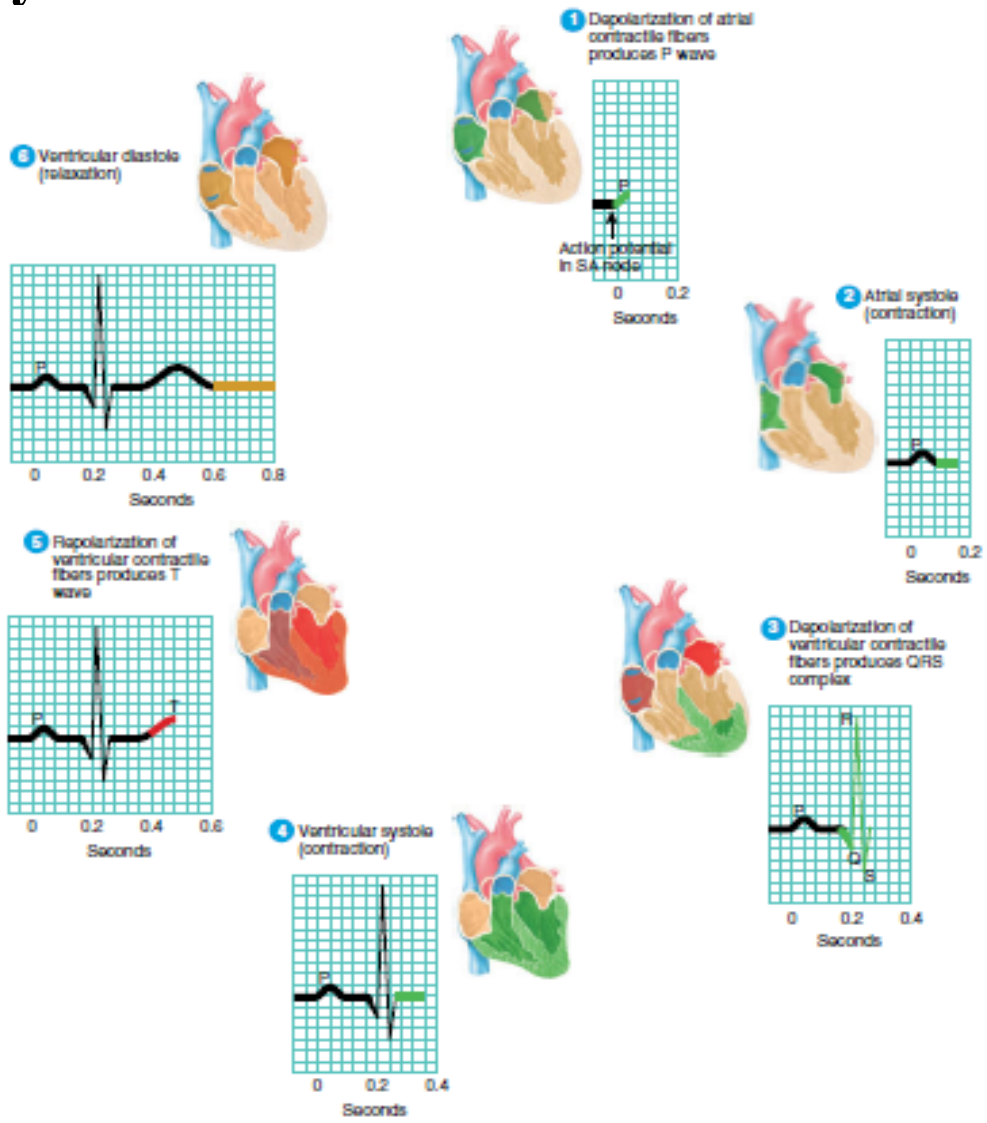
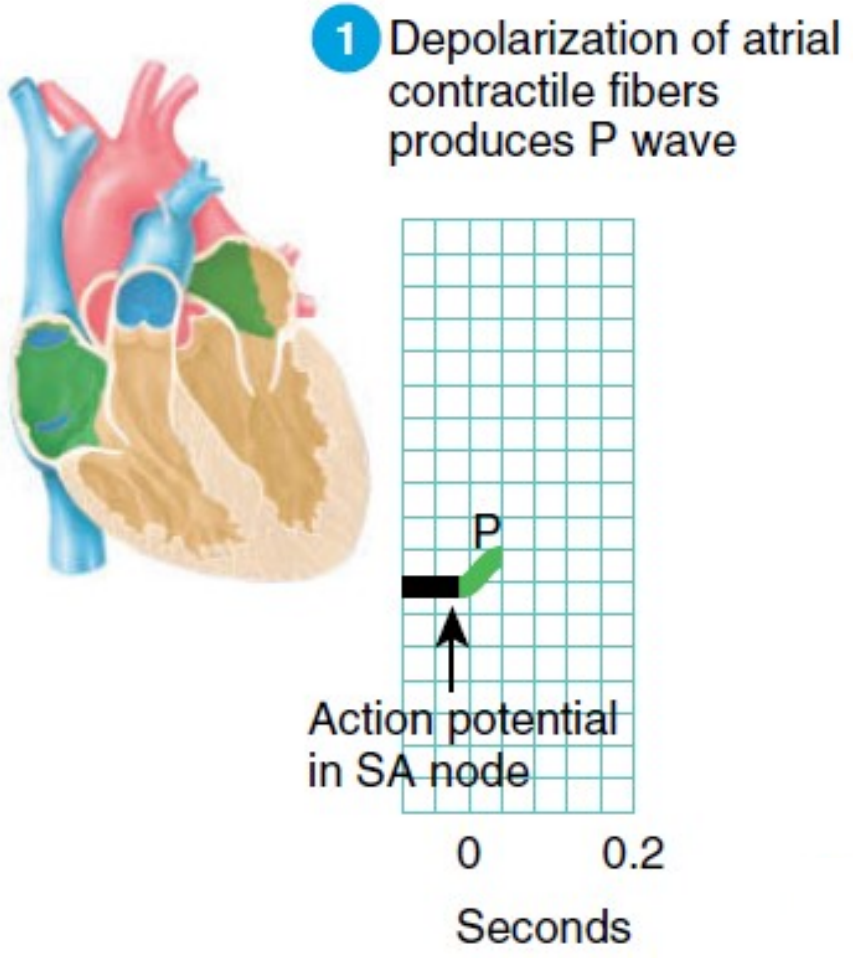

R
200 msec
T
P
P
Q
S
Recorded potential
PR
segment
ST
segment
TP
segment

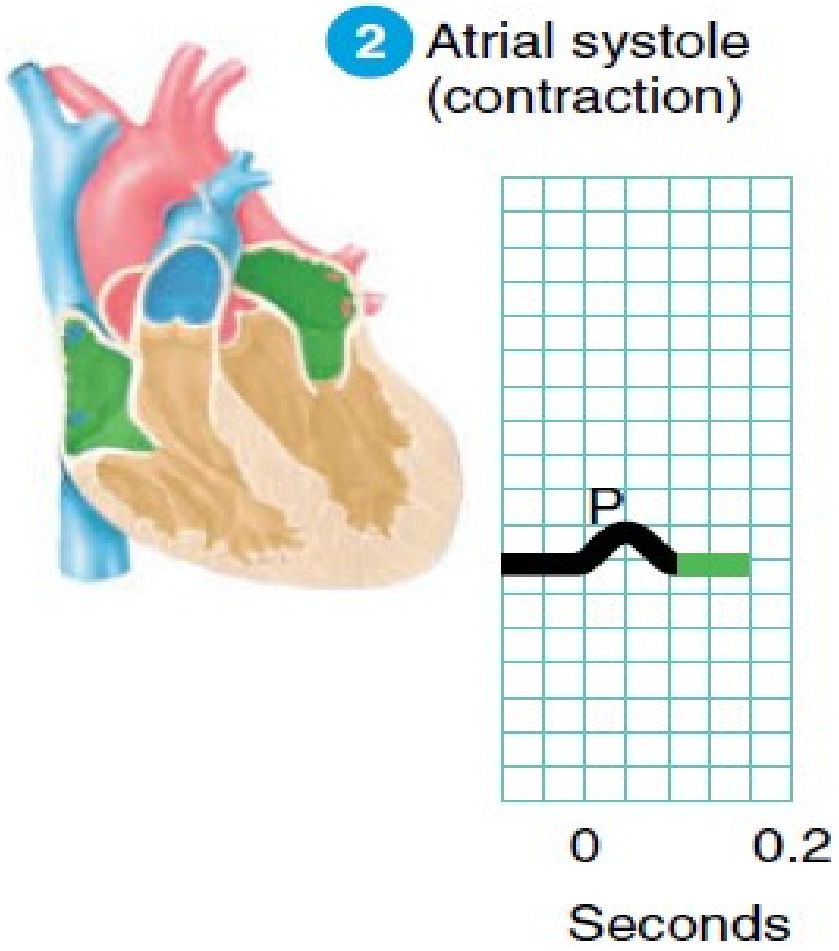
Figure 20.13 Timing and route of action potential depolarization and repolarization through the conduction system and myocardium



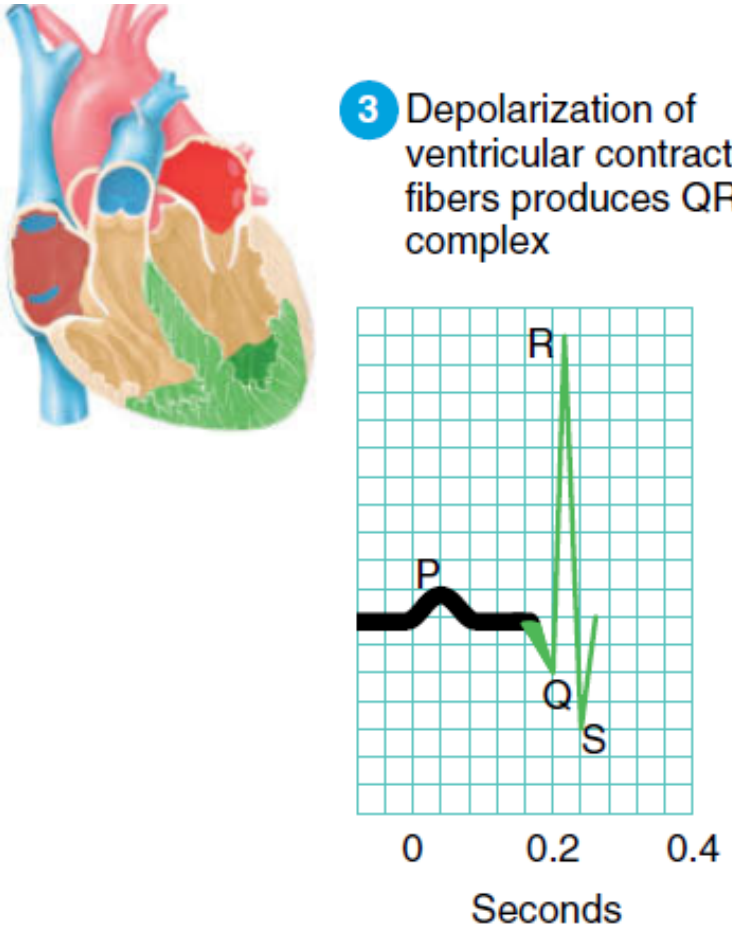
FIRST: Figure 20.13 **Timing and route of action potential depolarization and repolarization through the conduction system and myocardium**



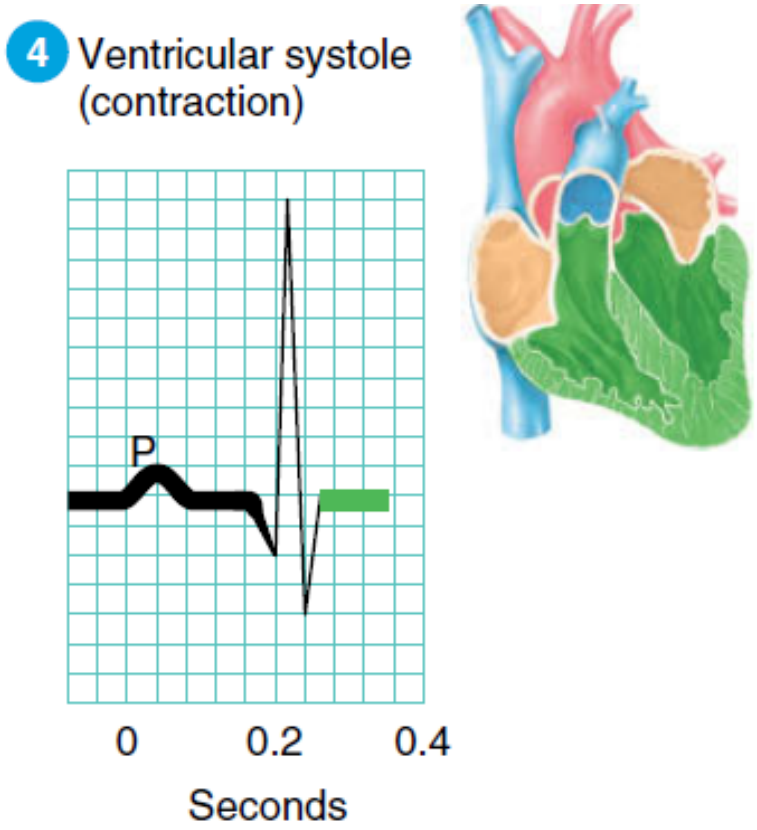
SECOND: Figure 20.13 **Timing and route of action potential depolarization and repolarization through the conduction system and myocardium**



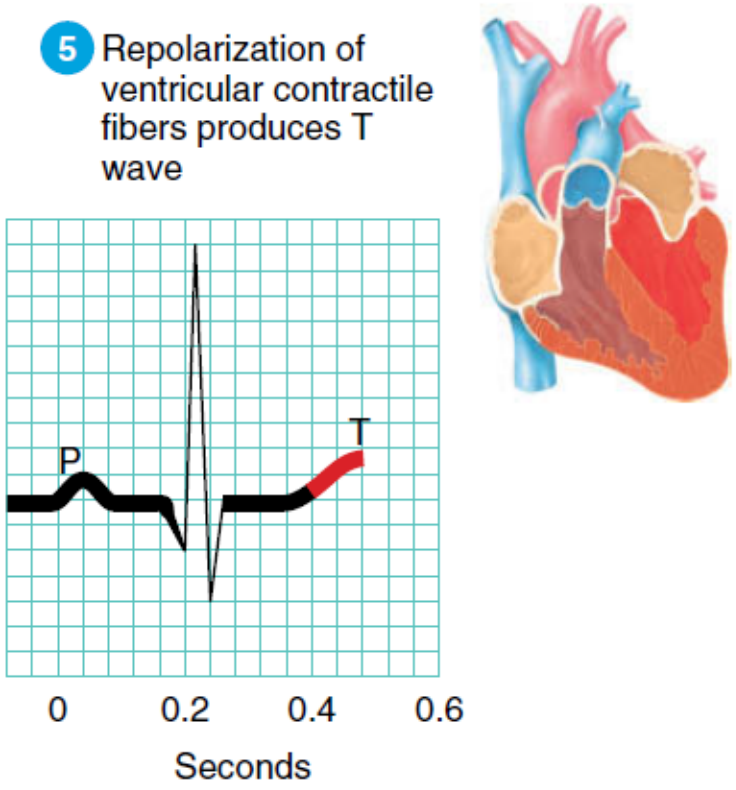
THIRD: Figure 20.13 **Timing and route of action potential depolarization and repolarization through the conduction system and myocardium**



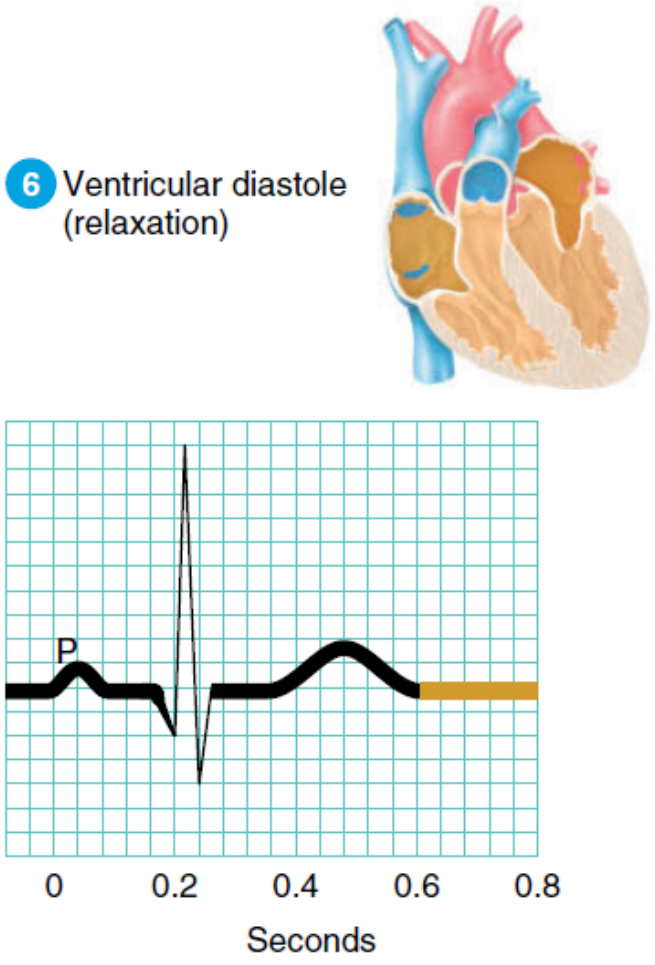
FOURTH: Figure 20.13 **Timing and route of action potential depolarization and repolarization through the conduction system and myocardium**



FIFTH: Figure 20.13 Timing and route of action potential depolarization and repolarization through the conduction system and myocardium



SIXTH: Figure 20.13 Timing and route of action potential depolarization and repolarization through the conduction system and myocardium



The Cardiac Cycle

- ***Atrial Systole***
- During **atrial systole**, which lasts about 0.1 sec, the atria are contracting. At the same time, the ventricles are relaxed.
- ***Ventricular Systole***
- During **ventricular systole**, which lasts about 0.3 sec, the ventricles are contracting. At the same time, the atria are relaxed in **atrial diastole**.
- ***Relaxation Period***
- During the **relaxation period**, which lasts about 0.4 sec, the atria and the ventricles are both relaxed. As the heart beats faster and faster, the relaxation period becomes shorter and shorter, whereas the durations of atrial systole and ventricular systole shorten only slightly.

Heart Sounds (Terminology)

- Auscultation is the act of listening to heart sounds
- Sound of heart valves closing: Four sounds but only two loud enough to be heard by stethoscope → (S1 and S2)

1- S1 = **lubb** = louder and a bit long, booming sound atrioventricular (AV) valves closing

2- S2 = **dupp** = shorter and not as loud as the first, sharp sound Semilunar (SL) valves closing

3- S3 blood turbulence during ventricular filling

4- S4 blood turbulence during atrial systole

Cardiac Output

- ✓The **heart rate**, also called the pulse, is the number of beats per minute.
- ✓→ Heart rate (HR) = 75 beats/min, one cycle requires 0.8 sec

- ✓A **pulse** is the rhythmic bulging of artery walls with each heartbeat.

- ✓The **stroke volume (SV)** is the amount of blood pumped in a single contraction, or blood volume ejected per beat from each ventricle that equals70 ml.

Cardiac Output (continued)

- ✓The **cardiac output (CO)** is the volume of blood pumped into the systemic circulation per minute and **depends on** both the heart rate and stroke volume
- ✓Cardiac output equals **stroke volume (SV)** times **heart rate (HR)**

$$CO = SV \times HR$$

- ✓ **Cardiac output** = stroke volume (SV) ml/beat ، heart rate (HR) beat/min= 70 X 75 = 5.25 L/min.

Regulation of Stroke Volume

- Three factors regulate stroke volume and ensure that the left and right ventricles pump equal volumes of blood:

(1) Preload, the degree of stretch on the heart before it contracts;

(2) Contractility, the forcefulness of contraction of individual ventricular muscle fibers;

(3) Afterload, the pressure that must be exceeded before ejection of blood from the ventricles can occur.

Regulation of Heart Rate

1-Autonomic Regulation of Heart Rate

2- Chemical Regulation of Heart Rate

3- Other Factors in Heart Rate Regulation

1-Autonomic Regulation of Heart Rate

- Nervous system regulation of the heart originates in the **cardiovascular center** in the ***medulla oblongata:***

1- **Proprioceptors** that are monitoring the position of limbs and muscles send nerve impulses at an increased frequency to the cardiovascular center.

2-Chemoreceptors, which monitor chemical changes in the blood,

3-Baroreceptors, which monitor the stretching of major arteries and veins caused by the pressure of the blood flowing through them.

Autonomic Regulation of Heart Rate (continued)

1- Sympathetic neurons extend from the medulla oblongata:

→**Cardiac accelerator nerves** extend out to the SA node, AV node, and most portions of the myocardium.

- Impulses in the cardiac accelerator nerves trigger the release of norepinephrine, which binds to beta-1 (β-1) receptors on cardiac muscle fibers. (1) speeds the rate of spontaneous depolarization, (2) enhances Ca^{2+} entry through the voltage-gated slow Ca^{2+} channels, thereby increasing contractility.

***2-** Parasympathetic nerve impulses reach the heart via the right and left vagus (X) nerves*

→ They release acetylcholine, which decreases heart rate by slowing the rate of spontaneous depolarization in autorhythmic fibers. As only a few vagal fibers innervate ventricular muscle, changes in parasympathetic activity have little effect on contractility of the ventricles

2- Chemical Regulation of Heart Rate

1. *Hormones.*

→ Epinephrine and norepinephrine

→ Hyperthyroidism

2. *Cations.*

Given that differences between intracellular and extracellular concentrations of several cations.

- **Elevated blood levels of K^+ or Na^+ <u>decrease</u> heart rate and contractility.**

→ Excess Na^+ blocks Ca^{2+} inflow during cardiac action potentials, thereby decreasing the force of contraction,

→ Excess K^+ blocks generation of action potentials.

- A <u>moderate</u> increase in interstitial (and thus intracellular) Ca^{2+} level speeds heart rate and strengthens the heartbeat.

3- Other Factors in Heart Rate Regulation

- ***Age*** (baby is likely to have a resting heart rate over 120 beats/min; declines with age).

- ***Gender*** (Adult females often have slightly higher resting heart rates than adult males).

- ***Physical fitness*** (physically fit person may even exhibit **bradycardia** a resting heart rate under 50 beats/min; as slowly beating heart is more energy efficient than one that beats more rapidly).

- ***Body temperature*** (causes the SA node to discharge impulses more quickly, thereby increasing heart rate. Decreased body temperature decreases heart rate and strength of contraction)

Hemodynamics: Factors Affecting Blood Flow

How the cardiac output becomes distributed into circulatory routes that serve various body tissues depends on two more factors:

(1) the *pressure difference* that drives the blood flow through a tissue

(2) the *resistance* to blood flow in specific blood vessels. Blood flows from regions of higher pressure to regions of lower pressure; the greater the pressure difference, the greater the blood flow. But the higher the resistance, the smaller the blood flow.

Blood Pressure

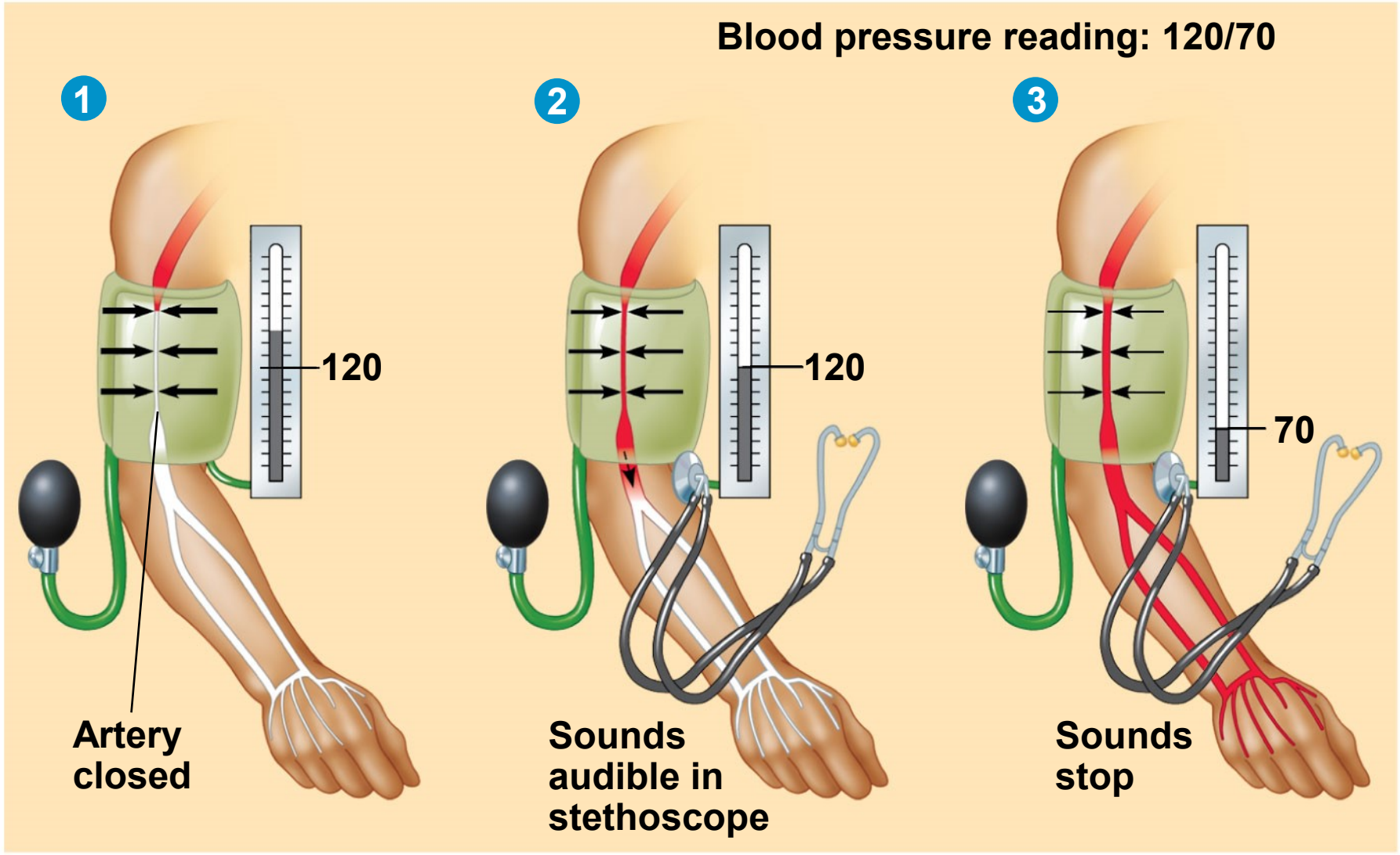
- Blood flows from areas of higher pressure to areas of lower pressure.

- **Blood pressure** is the pressure that blood exerts against the wall of a vessel.

- Blood pressure is generally measured for an artery in the arm at the same height as the heart.

- Blood pressure for a healthy 20 year old at rest is 120 mmHg at systole and 70 mmHg at diastole.

Sphygmomanometer (blood pressure measurement device)



Changes in Blood Pressure During the Cardiac Cycle

- **Systolic pressure** is the pressure in the arteries during ventricular systole; it is the highest pressure in the arteries
- **Diastolic pressure** is the pressure in the arteries during diastole; it is lower than systolic pressure
- **Pulse pressure** is the pressure difference between systolic and diastolic pressure.
- **Mean arterial pressure** is diastolic pressure + 1/3 pulse pressure.

Changes in Blood Pressure During the Cardiac Cycle (continued)

- **Blood pressure also depends on:**

We have already seen that **cardiac output** equals **heart rate** multiplied by **stroke volume**.

Another way to calculate cardiac output is to divide mean arterial pressure (MAP) by **resistance** (R): CO= MAP ÷ R.

By rearranging the terms of this equation, you can see that

$$MAP = CO \times R.$$

- If cardiac output rises due to an increase in stroke volume or heart rate, then the mean arterial pressure rises as long as resistance remains steady.
- Blood pressure also depends on the **total volume of blood** in the cardiovascular system.

Vascular Resistance

Vascular resistance is the opposition to blood flow due to friction between blood and the walls of blood vessels.

Vascular resistance depends on:

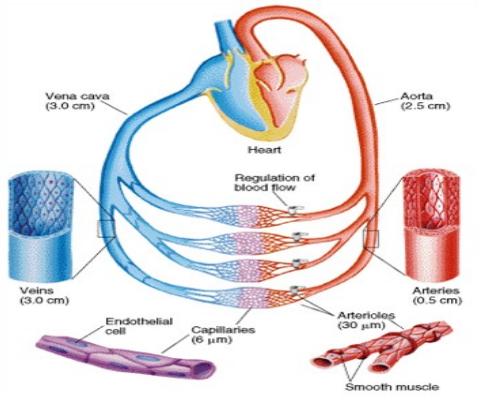
(1) Size of the blood vessel lumen→ (The smaller the diameter of the blood vessel, the greater the resistance it offers to blood flow)

(2) Blood viscosity→ (The viscosity (thickness) of blood depends mostly on the ratio of red blood cells to plasma (fluid) volume, and to a smaller extent on the concentration of proteins in plasma).

(3) Total blood vessel length

→(Resistance to blood flow through a vessel is directly proportional to the length of the blood vessel.

→ The longer a blood vessel, the greater the resistance).



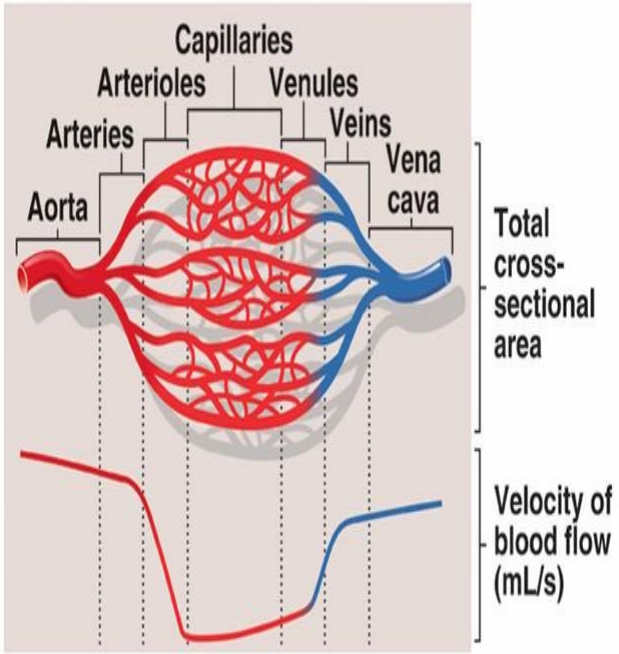
Blood Flow Velocity

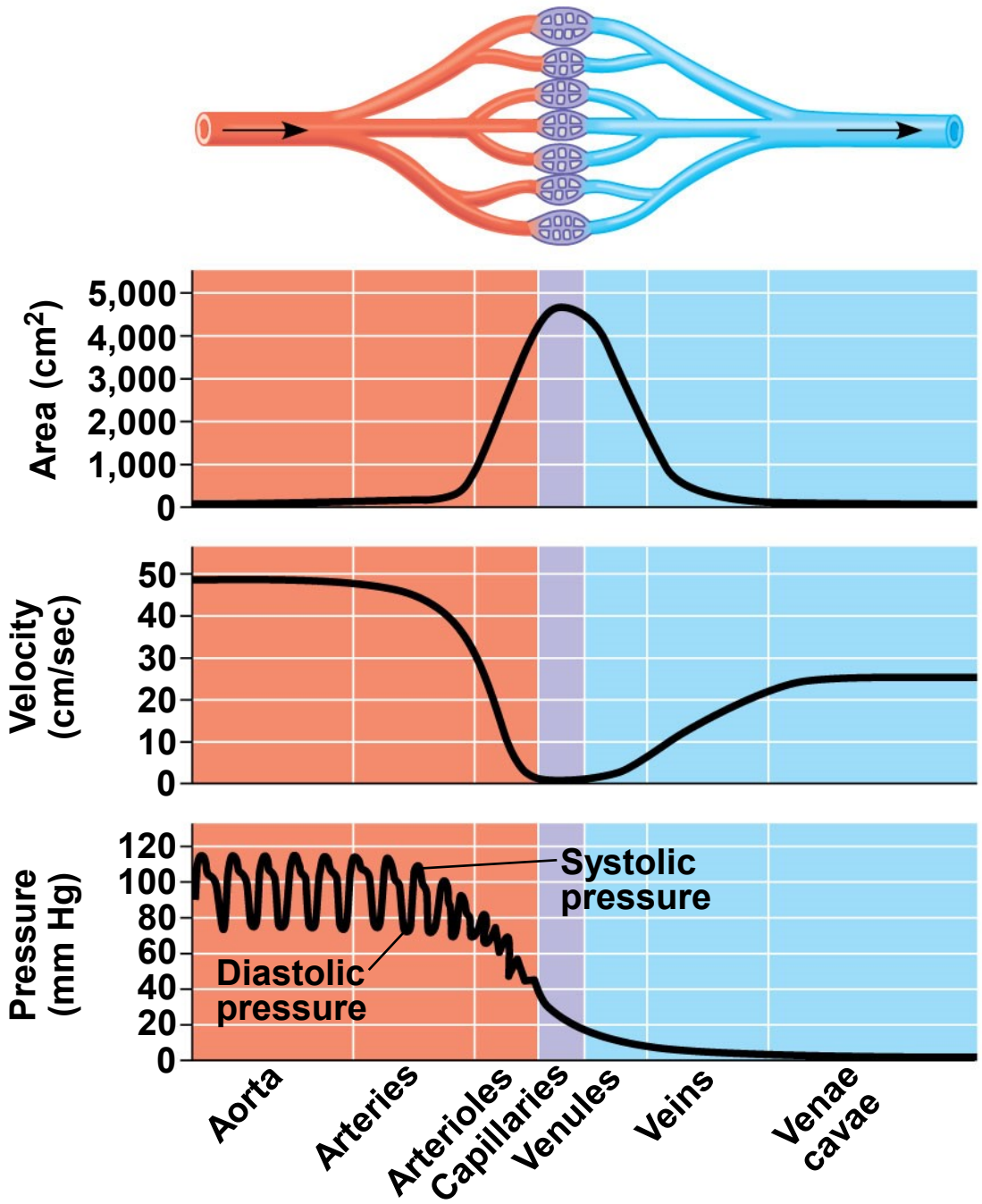
- Physical laws governing movement of fluids through pipes affect blood flow and blood pressure

- Velocity of blood flow is slowest in the capillary beds, as a result of the high resistance and large total cross-sectional area

→ **Systemic vascular resistance (SVR),** also known as *total peripheral resistance (TPR),* refers to all of the vascular resistances offered by systemic blood vessels.

- Blood flow in capillaries is necessarily slow for exchange of materials





Blood Flow in Veins

Venous blood flow is dependent upon:

a. Skeletal muscle contraction

b. Presence of valves in veins

c. Rhythmic contractions of smooth muscles in the wall of veins and venules.

d. Respiratory movements: the change in pressure during inhalation causes the vena cava and other veins to expand and fill with blood.

→ Compression of veins causes blood to move forward past a valve that then prevents it from returning backward.

Blood Flow in Veins (continued)

- When a person is standing, gravity helps pull the blood downward to the lower extremities. Without gravity, blood tends to remain closer to the heart.
- The force of gravity also makes it more difficult for the blood to flow upward to return to the heart and lungs for more oxygen.
- Our bodies have evolved to deal with the ever-present downward force of gravity; our leg muscles function as secondary pumps to help in the process of **venous return** *which is blood flow back to the heart, also referred to as cardiac input).*
- During walking or other leg movements, the muscles contract, forcing blood up through the veins of the calf toward the heart. The valves in the veins are arranged so that blood flows only in one direction. This mechanism effectively counteracts the force of gravity.

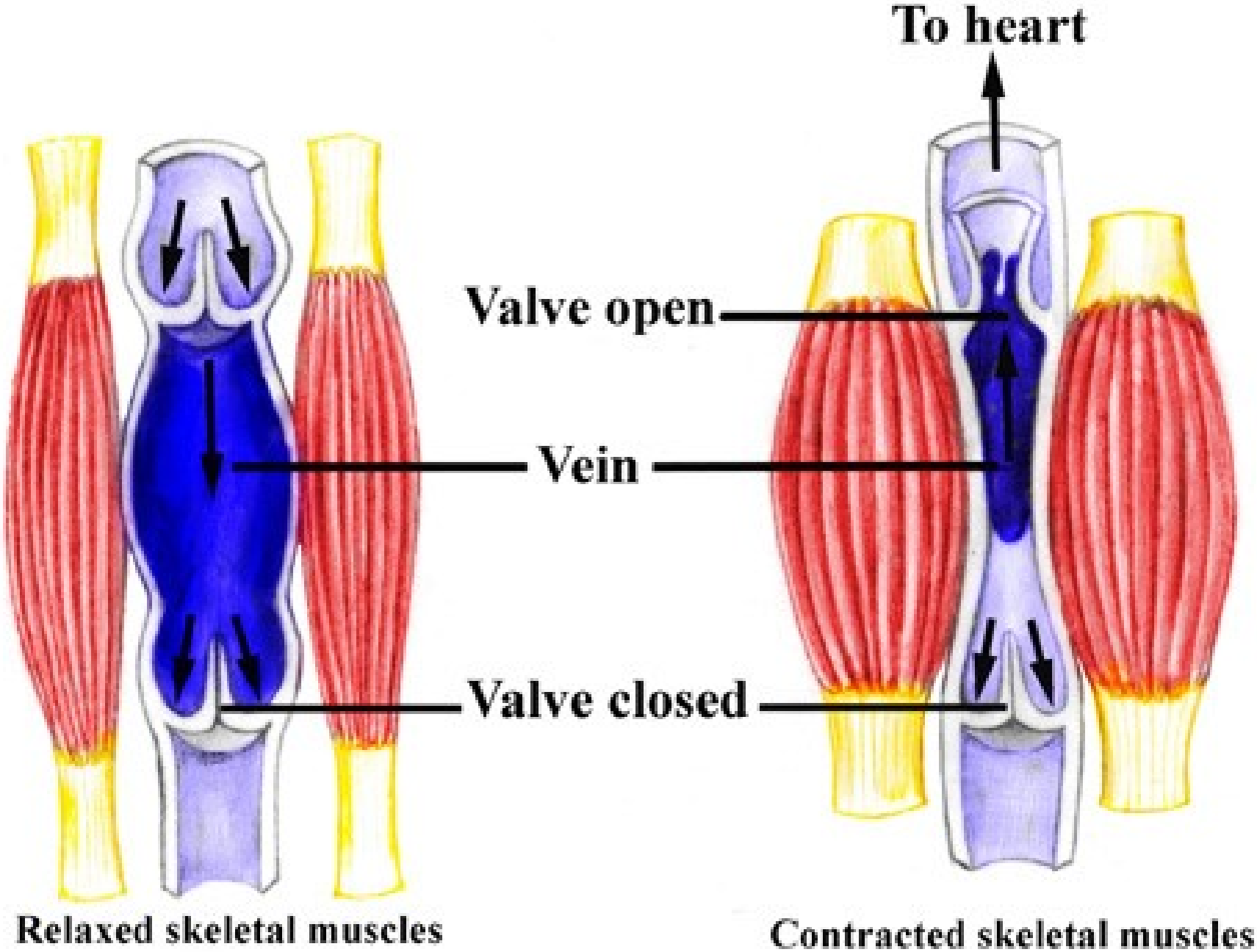
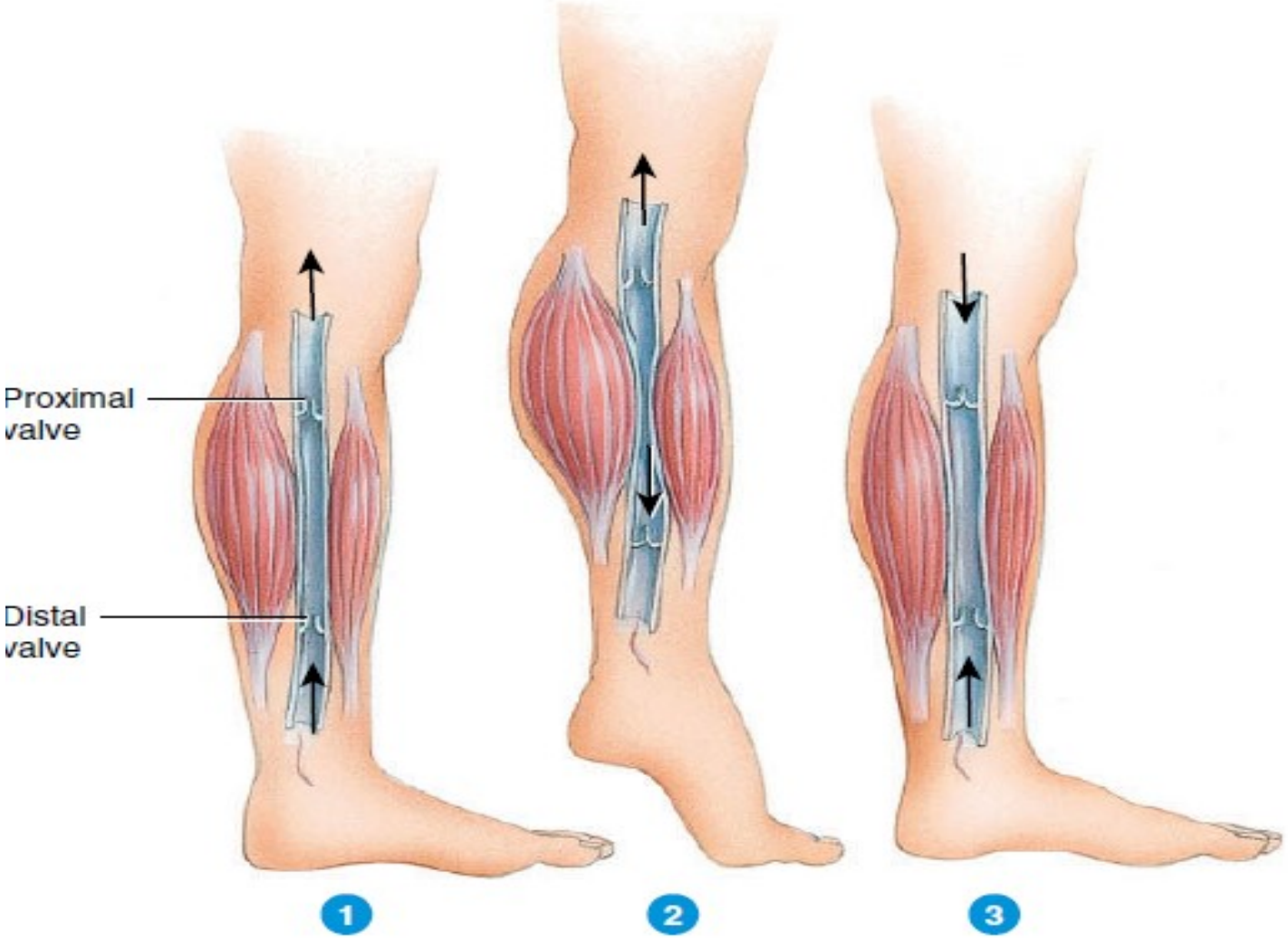

To heart
Valve open
Vein
Valve closed
Relaxed skeletal muscles
Contracted skeletal muscles

Figure 21.9 Action of the skeletal muscle pump in returning blood to the heart.

Milking refers to skeletal muscle contractions that drive venous blood toward the heart.



Control of Blood Pressure and Blood Flow → short term

The cardiovascular center receives input both from **higher brain regions** and from **sensory receptors**.

1-Nerve impulses descend from:

→The cerebral cortex

→Limbic system,

→ Hypothalamus to affect the cardiovascular center.

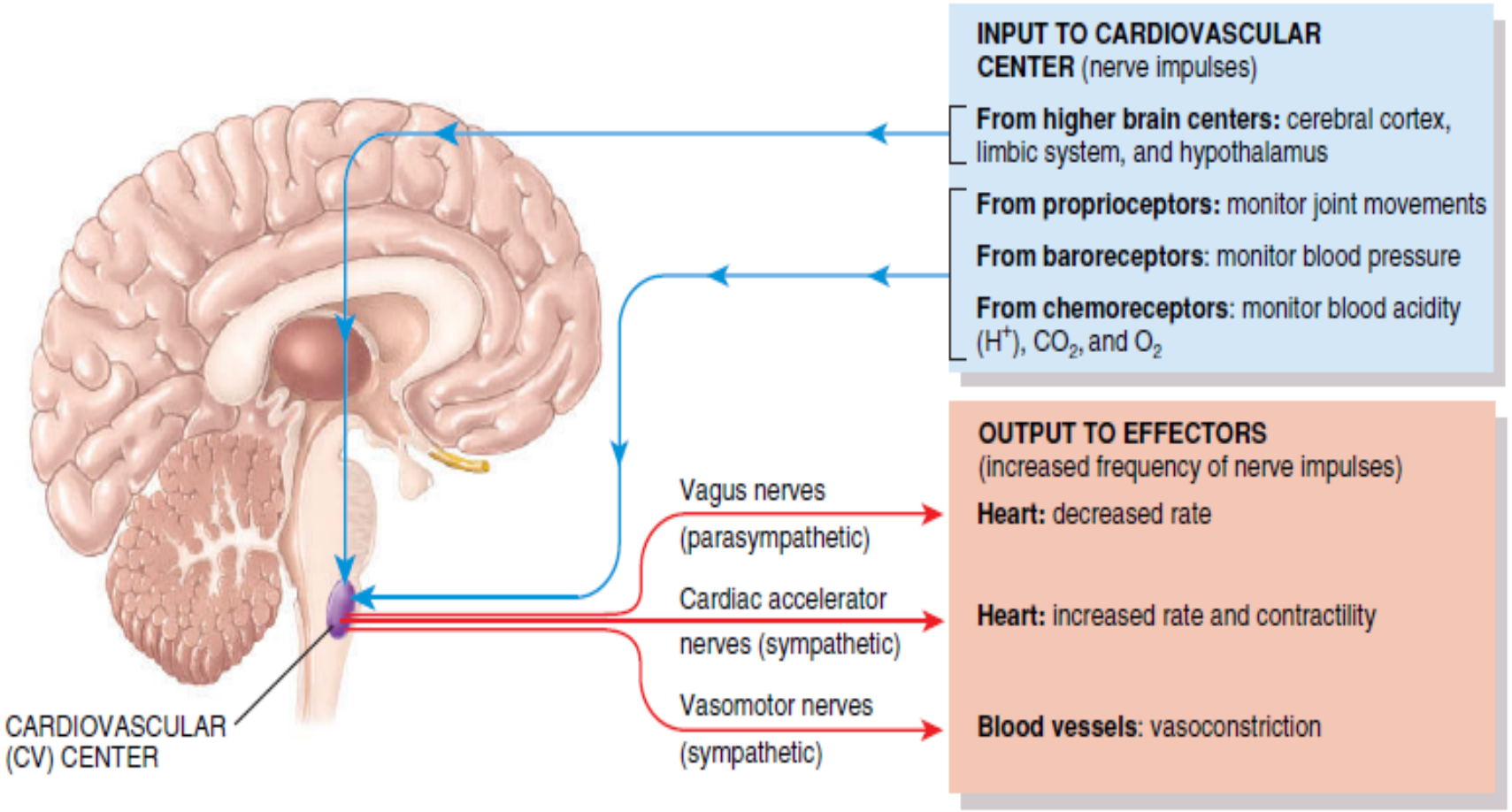
2- **Sensory receptors:** The three main types of sensory receptors that provide input to the cardiovascular center are:

→Proprioceptors (Carotid and aortic)

→Baroreceptors (carotid and aortic bodies)

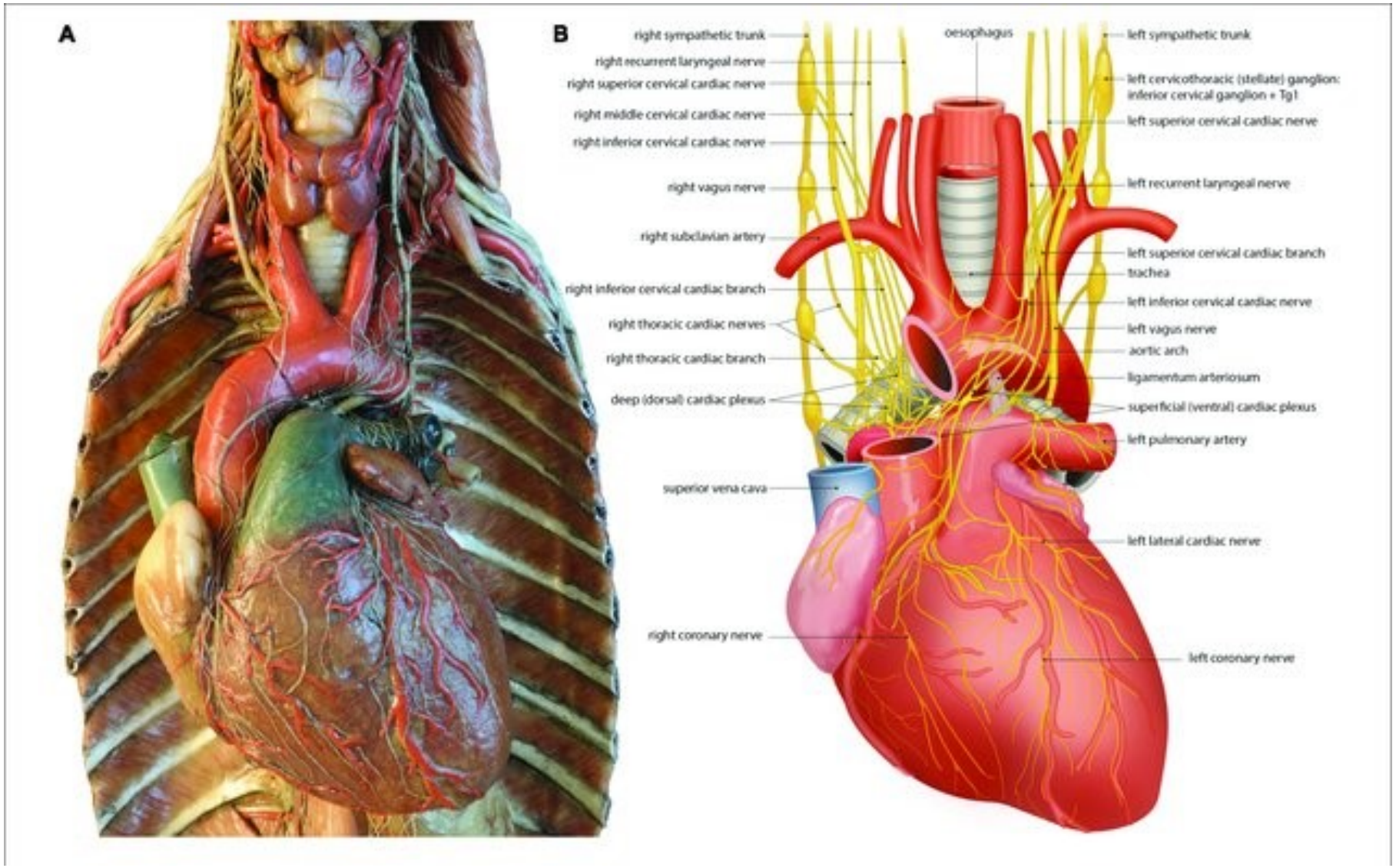
→Chemoreceptors (carotid and aortic bodies).

Location and function of the cardiovascular (CV) center in the medulla oblongata. The CV center receives input from higher brain centers, proprioceptors, baroreceptors, and chemoreceptors. Then, it provides output to the sympathetic and parasympathetic divisions of the autonomic nervous system (ANS).



Cardiac Plexus:

source:
https://www.researchgate.net/publication/354636140_Asymmetry_and_Heterogeneity_Part_and_Parcel_in_Cardiac_Autonomic_Innervation_and_Function/figures?lo=1



Hormonal Regulation of Blood Pressure→ Long-term Blood Pressure Regulation

Blood Pressure Regulation by Hormones

FACTOR INFLUENCING BLOOD PRESSURE	HORMONE	EFFECT ON BLOOD PRESSURE
CARDIAC OUTPUT		
Increased heart rate and contractility	Norepinephrine, epinephrine.	Increase.
SYSTEMIC VASCULAR RESISTANCE		
Vasoconstriction	Angiotensin II, antidiuretic hormone (ADH), norepinephrine,* epinephrine.†	Increase.
Vasodilation	Atrial natriuretic peptide (ANP), epinephrine,† nitric oxide.	Decrease.
BLOOD VOLUME		
Blood volume increase	Aldosterone, antidiuretic hormone.	Increase.
Blood volume decrease	Atrial natriuretic peptide.	Decrease.

*Acts at α_1 receptors in arterioles of abdomen and skin.
†Acts at β_2 receptors in arterioles of cardiac and skeletal muscle; norepinephrine has a much smaller vasodilating effect.

Autoregulation of Blood Flow

Two general types of stimuli cause autoregulatory changes in blood flow:

1. *Physical changes.* Warming promotes vasodilation, and cooling causes vasoconstriction. In addition, smooth muscle in arteriole walls exhibits a **myogenic response -** it contracts more forcefully when it is stretched and relaxes when stretching lessens. If, for example, blood flow through an arteriole decreases, stretching of the arteriole walls decreases. As a result, the smooth muscle relaxes and produces vasodilation, which increases blood flow.

2. *Vasodilating and vasoconstricting chemicals.* Several types of cells—including white blood cells, platelets, smooth muscle fibers, macrophages, and endothelial cells—release a wide variety of chemicals that alter blood-vessel diameter. **Vasodilating chemicals** released by metabolically active tissue cells include K, H, lactic acid (lactate), and adenosine (from ATP). Another important vasodilator released by endothelial cells is nitric oxide (NO). Tissue trauma or inflammation causes release of vasodilating kinins and histamine. **Vasoconstrictors** include thromboxane A2, superoxide radicals, serotonin (from platelets), and endothelins (from endothelial cells).

Factors Affecting Blood Pressure (BP)

- **Age**
- **Gender**
- **Exercise**
- **Smoking:** refrain from smoking at least 30 minutes before having a blood pressure measurement taken.
- **Stress**
- **Daytime and night time**