



# **Physiology and Anatomy of the Heart**

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# Disclosure

- The material and the illustrations are adopted from the textbook **“Human Anatomy and Physiology / Ninth edition/ Eliane N. Marieb 2013”**

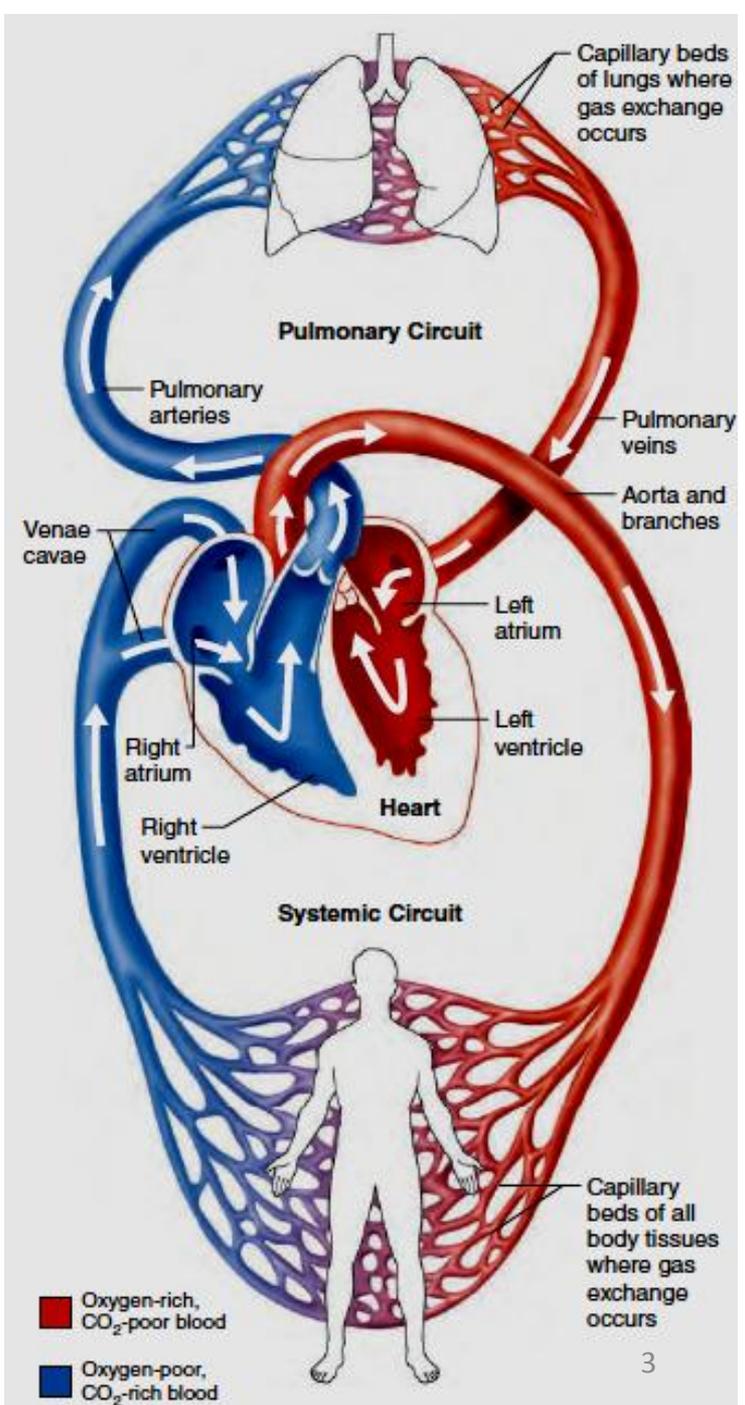
# The systemic and pulmonary circuits

- **Pulmonary circuit**

- The right side of the heart is the pulmonary circuit pump.
- It pumps blood through the lungs, where the blood picks up oxygen and dumps carbon dioxide.

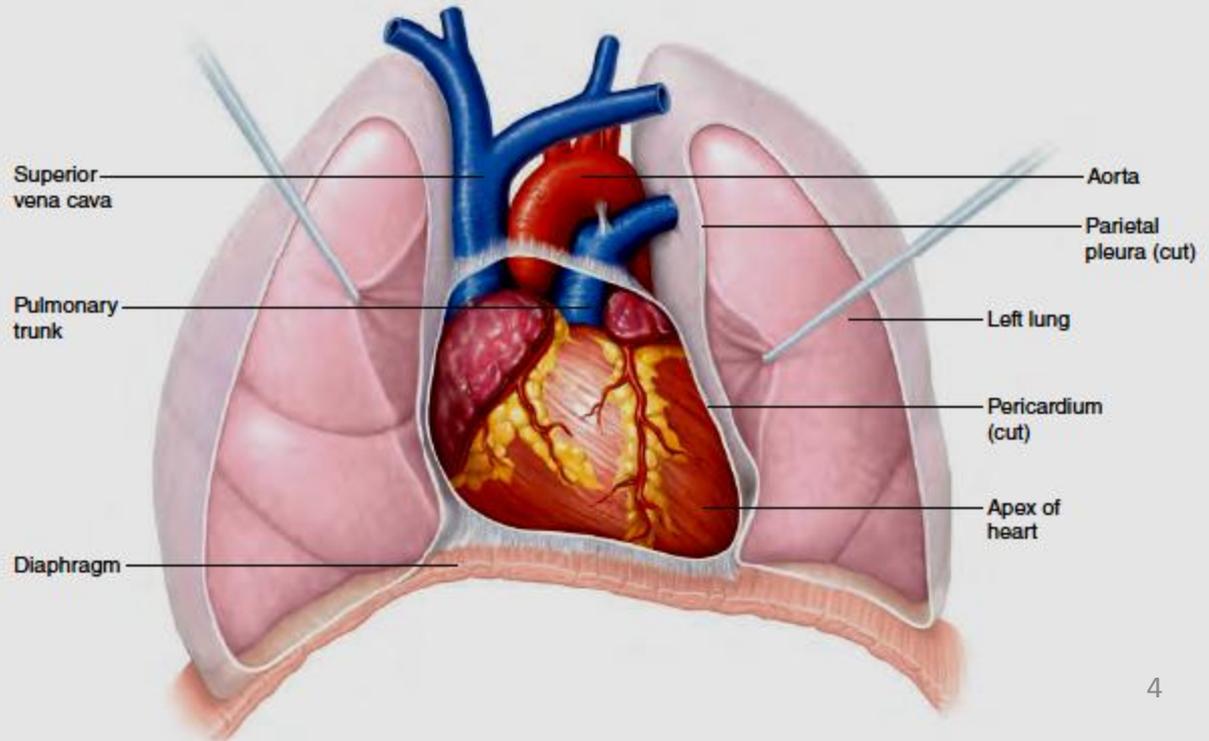
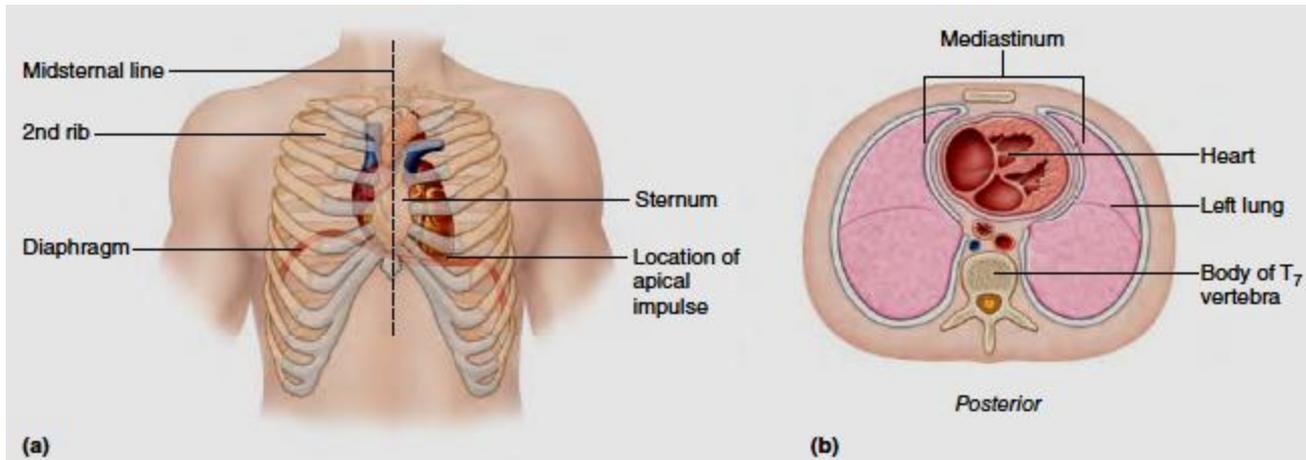
- **Systemic circuit**

- The left side of the heart is the systemic circuit pump.
- It pumps blood through the body's tissues, supplying them with oxygen and nutrients and removing carbon dioxide.



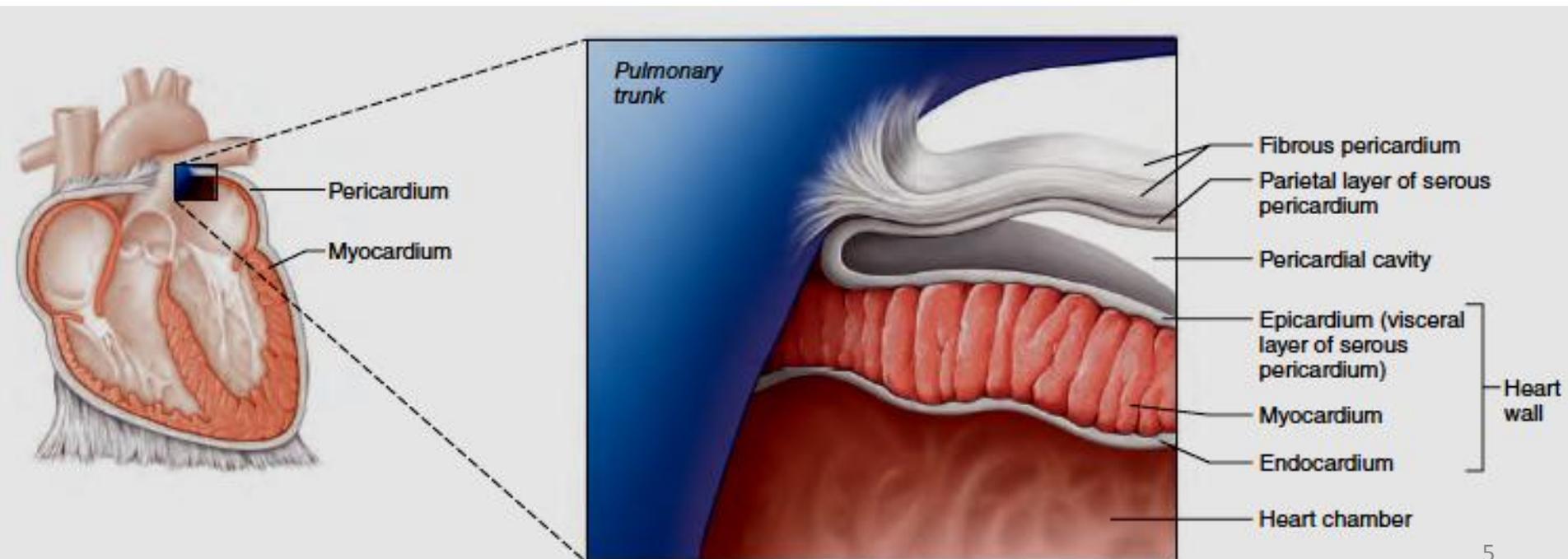
# Location of the heart in the mediastinum.

- The human heart is about the size of a clenched fist
- It is located within the mediastinum of the thorax



# The pericardial layers and layers of the heart wall

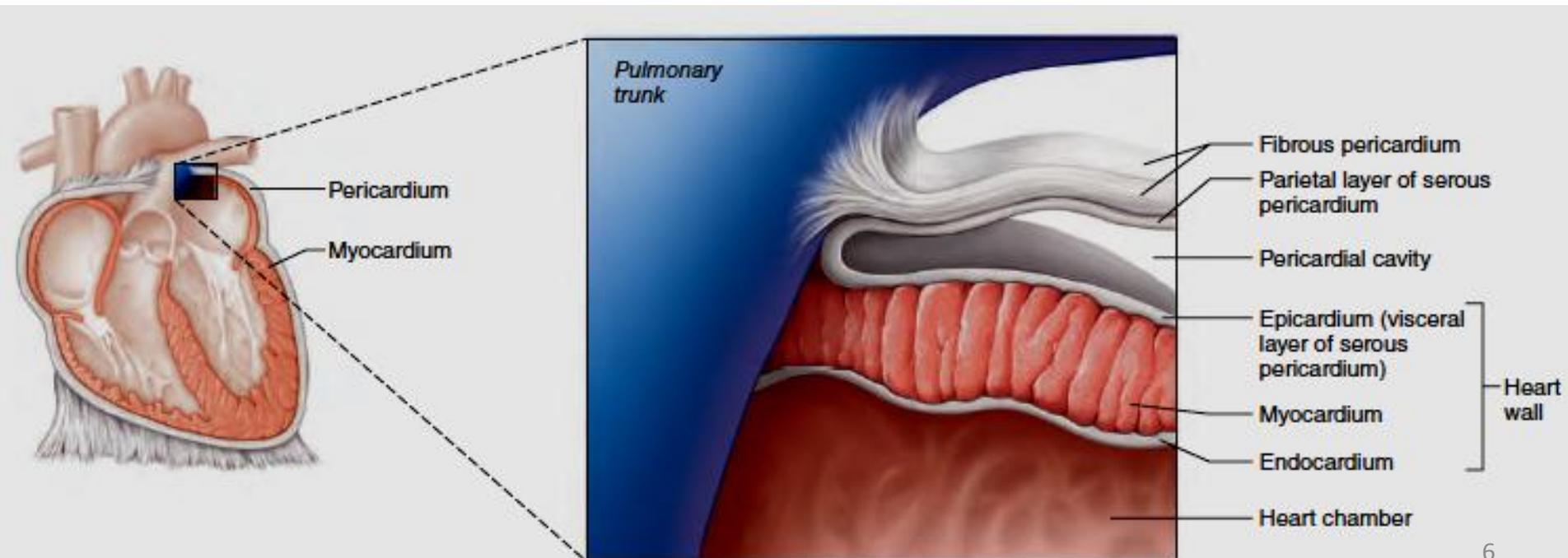
- The heart is enclosed within a double sac made up of the outer fibrous pericardium and the inner serous pericardium
- The pericardial cavity between the serous layers contains lubricating serous fluid.



# The pericardial layers and layers of the heart wall

**Layers of the heart wall, from the interior out, are:**

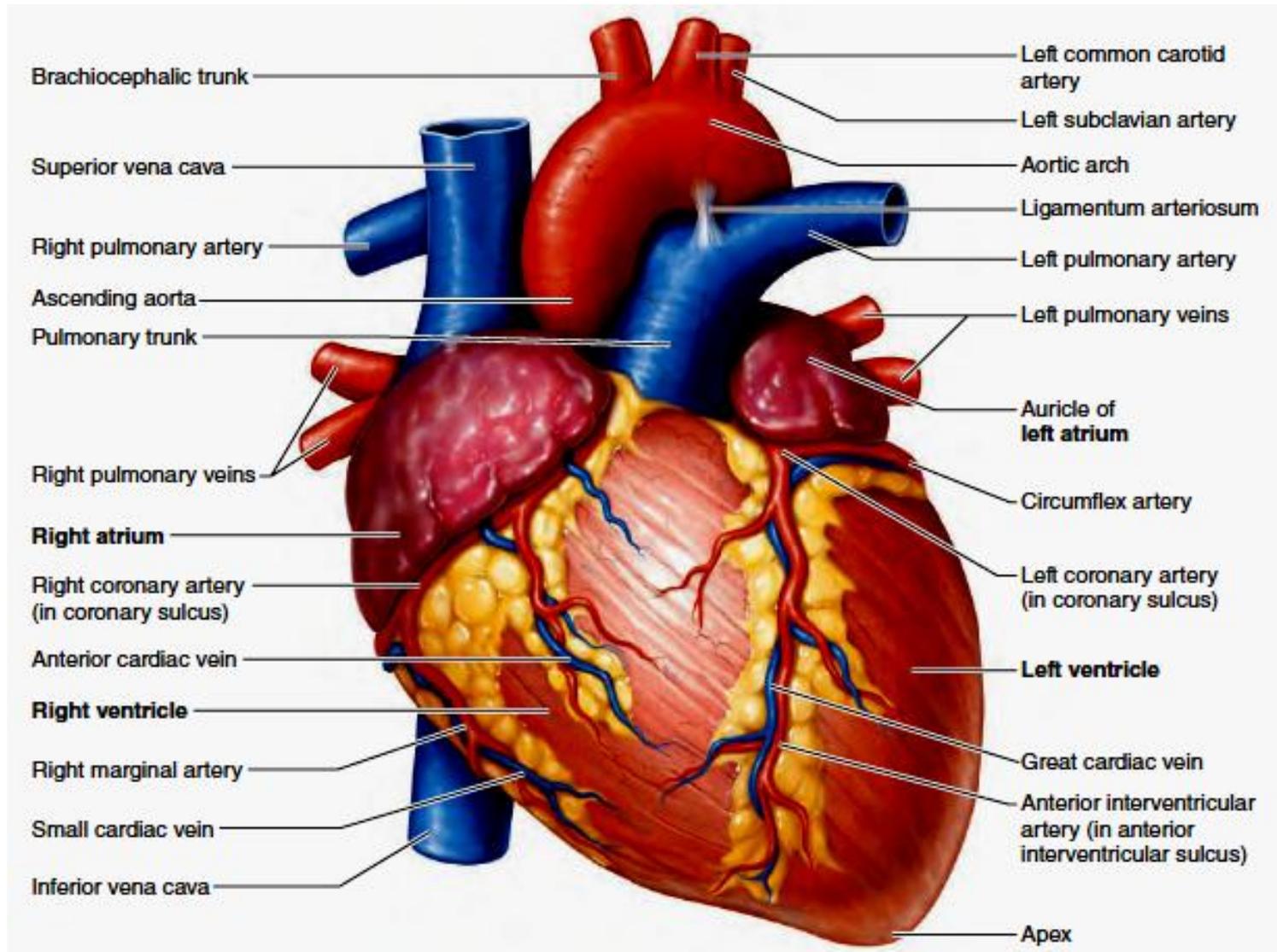
- Endocardium
- Myocardium (reinforced by a fibrous cardiac skeleton)
- Epicardium (visceral layer of the serous pericardium)



# Chambers and Associated Great Vessels

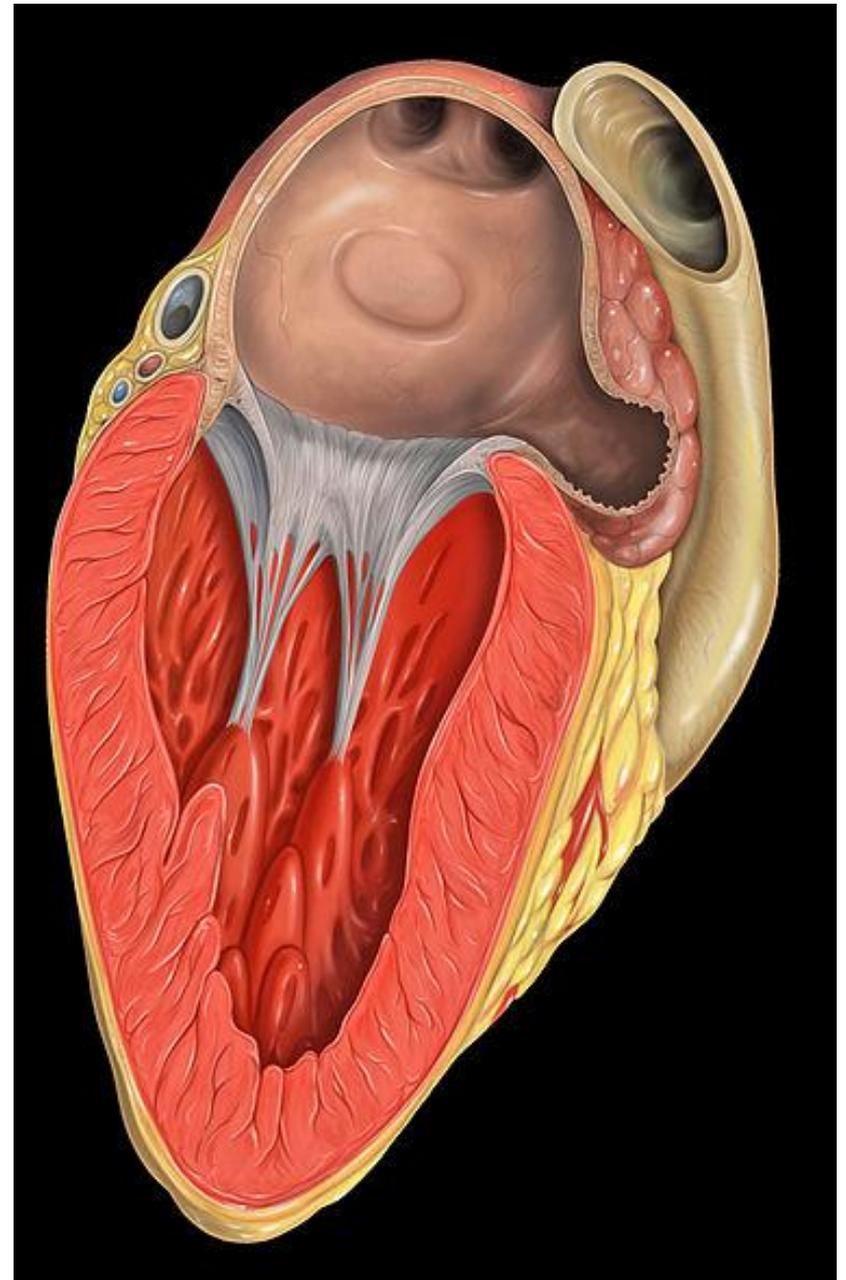
- The heart has two superior atria and two inferior ventricles.
- Functionally, the heart is a double pump.
- **Entering the right atrium are:**
  - The superior vena cava
  - The inferior vena cava
  - The coronary sinus
- Four pulmonary veins enter the left atrium
- The right ventricle discharges blood into the pulmonary trunk
- The left ventricle pumps blood into the aorta.

# Gross anatomy of the heart



**Anterior view** vessels transporting oxygen-rich blood are red; those transporting oxygen-poor blood are blue.

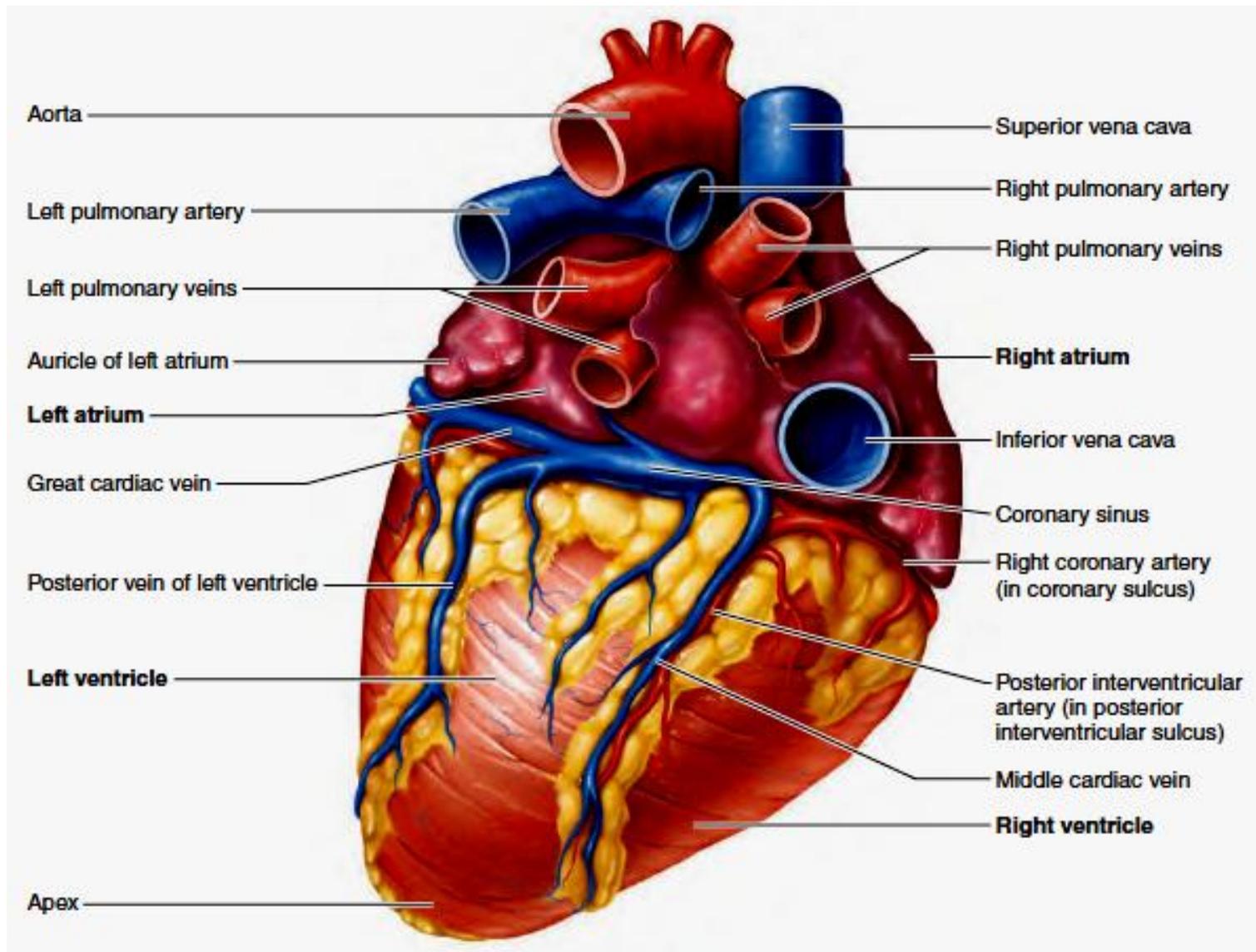
# Cardiac atria versus auricle (appendages)



**Left atrial appendages (heart's left auricle)**

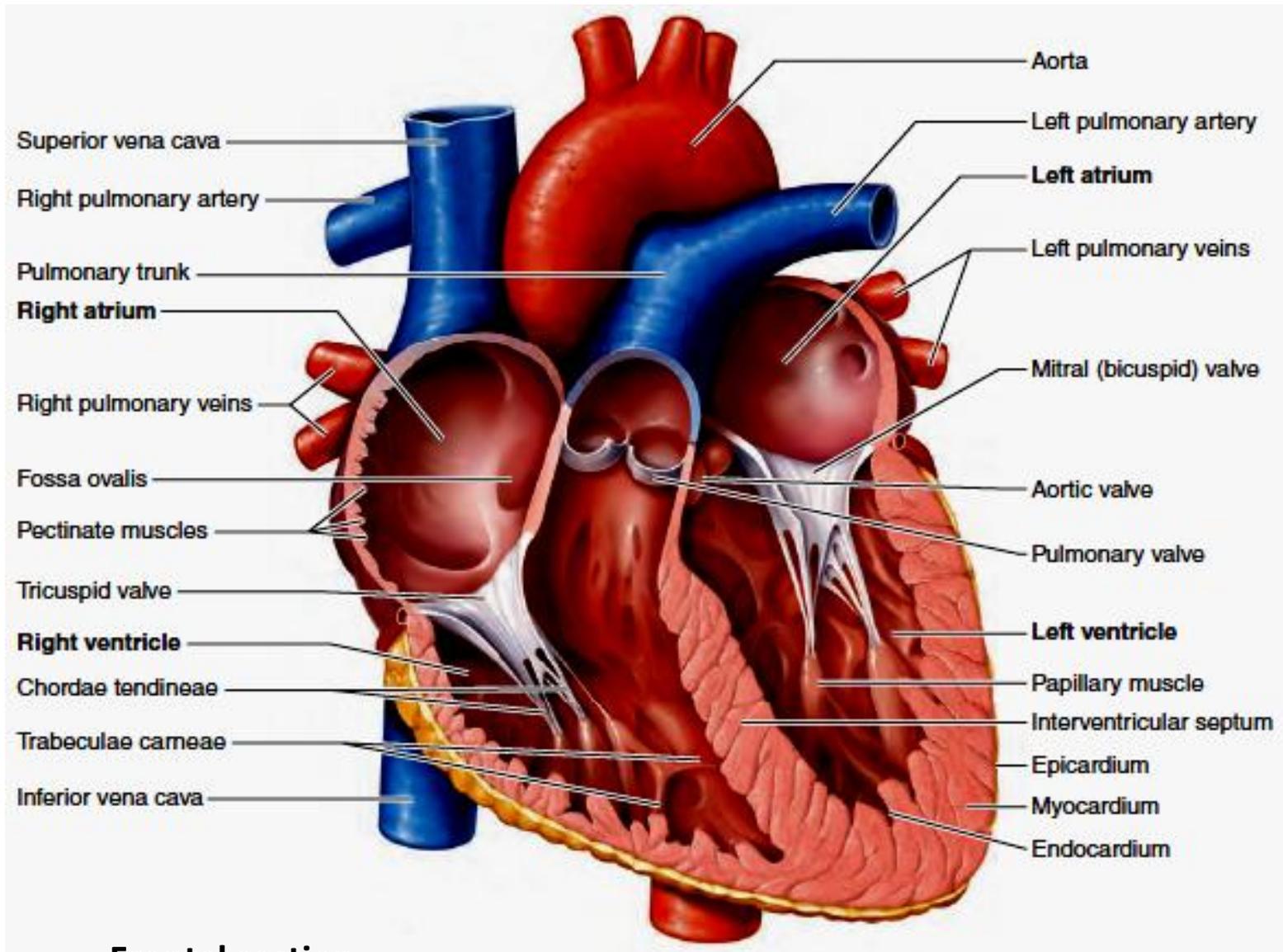
*Patrick J. Lynch, medical illustrator*

# Gross anatomy of the heart



Posterior surface view

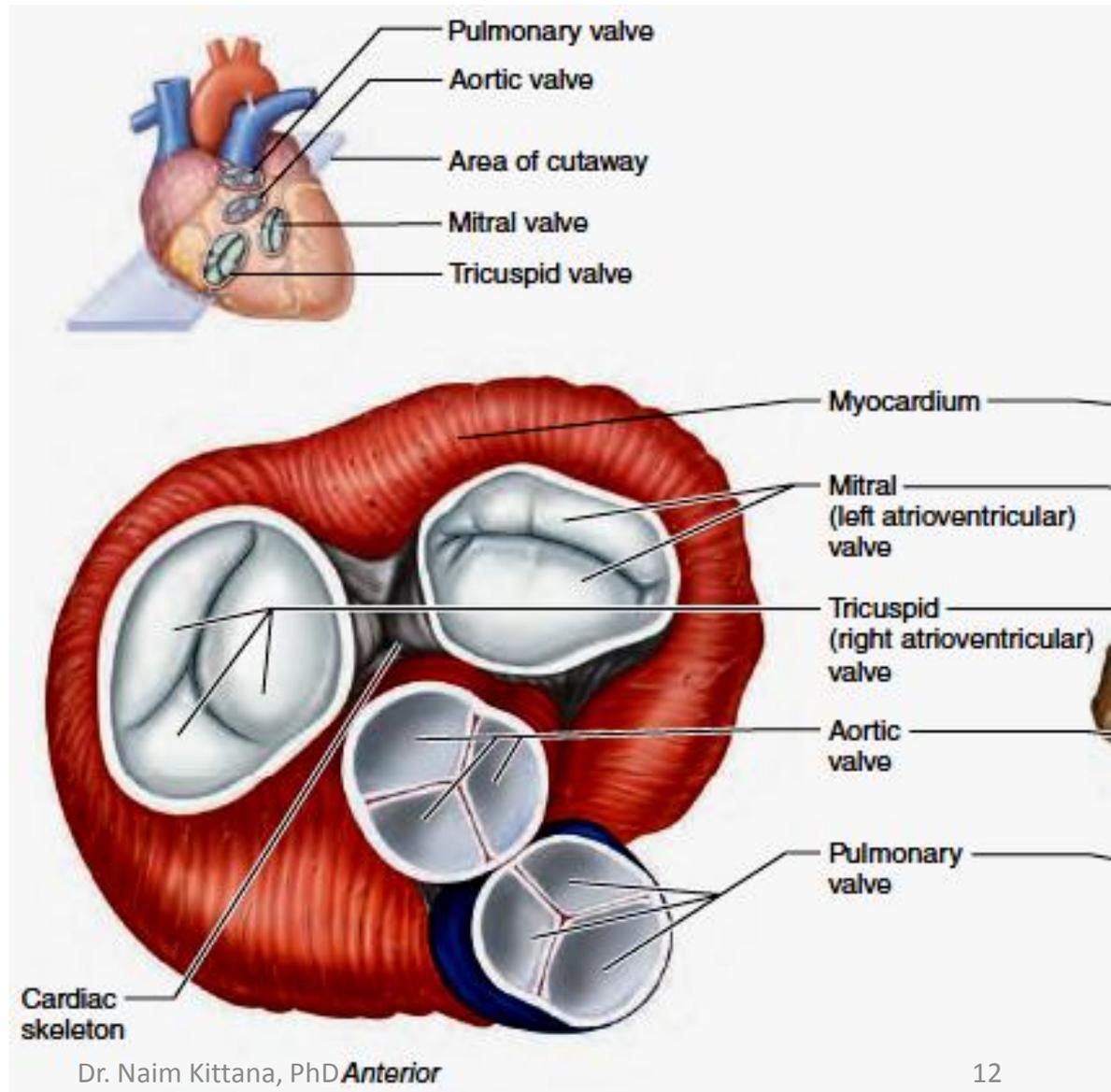
# Gross anatomy of the heart



**Frontal section**

# Heart Valves

- The atrioventricular (AV) valves (tricuspid and mitral) prevent backflow into the atria when the ventricles are contracting
- The semilunar (SL) valves (pulmonary and aortic) prevent backflow into the ventricles when the ventricles are relaxing.

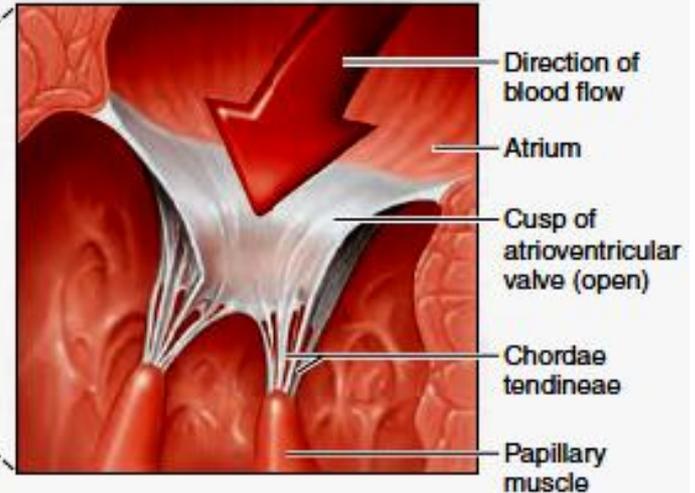
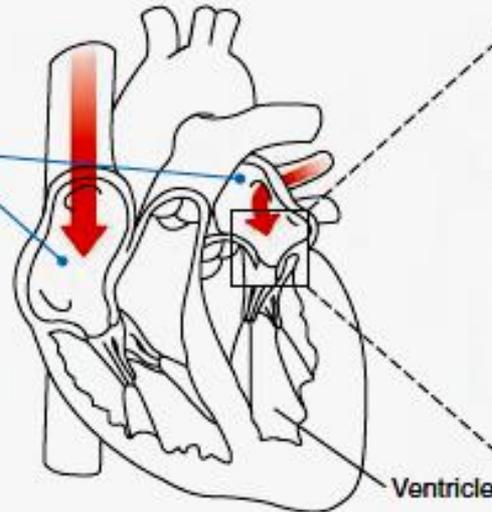


# The atrioventricular (AV) valves.

① Blood returning to the heart fills atria, pressing against the AV valves. The increased pressure forces AV valves open.

② As ventricles fill, AV valve flaps hang limply into ventricles.

③ Atria contract, forcing additional blood into ventricles.

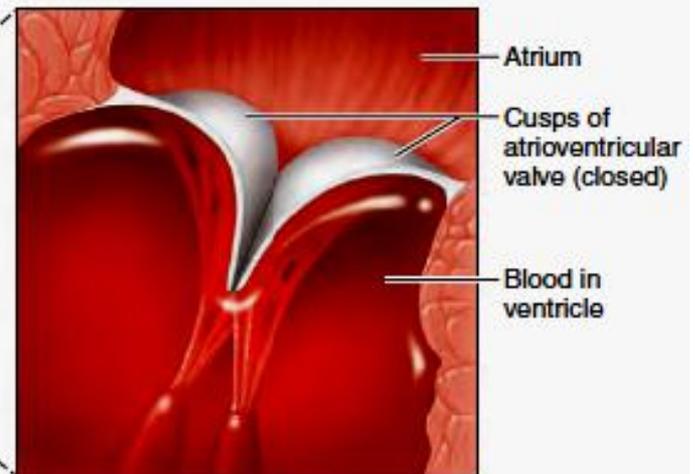
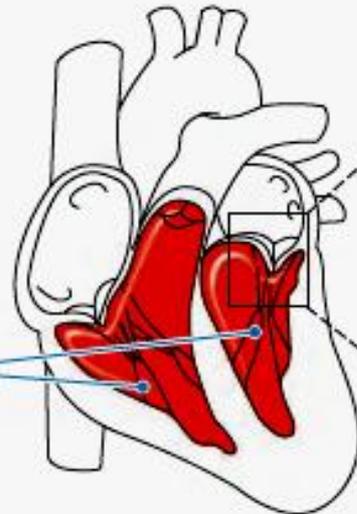


**(a) AV valves open; atrial pressure greater than ventricular pressure**

① Ventricles contract, forcing blood against AV valve cusps.

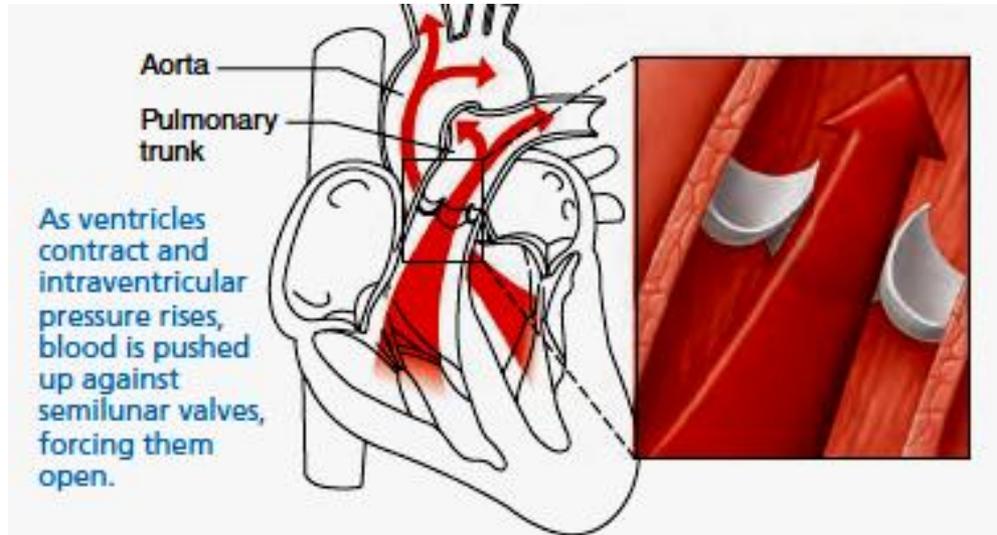
② AV valves close.

③ Papillary muscles contract and chordae tendineae tighten, preventing valve flaps from everting into atria.

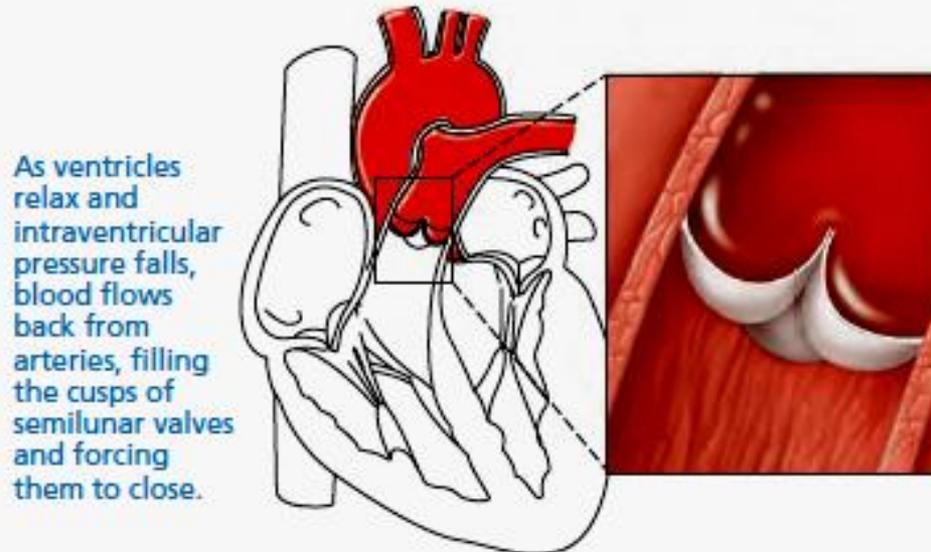


**(b) AV valves closed; atrial pressure less than ventricular pressure**

# The semilunar (SL) valves



**(a) Semilunar valves open**



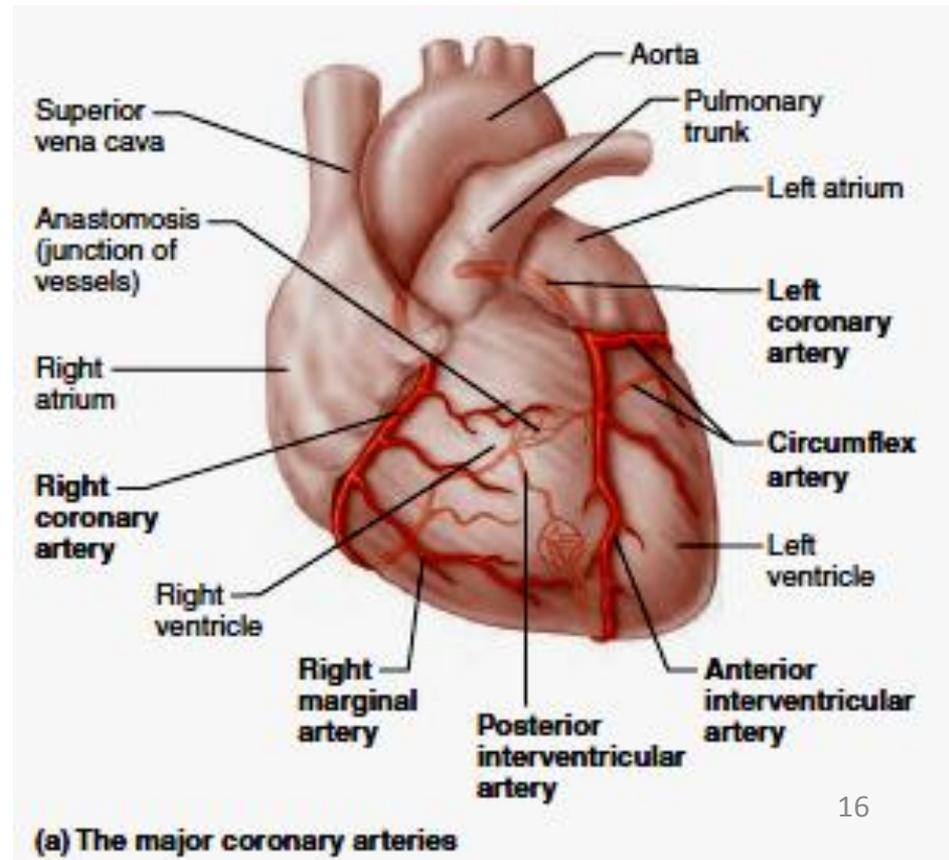
**(b) Semilunar valves closed**

# Pathway of Blood Through the Heart

- Oxygen-poor systemic blood enters the right atrium, passes into the right ventricle, through the pulmonary trunk to the lungs, and back to the left atrium via the pulmonary veins.
- Oxygenated blood entering the left atrium from the lungs flows into the left ventricle and then into the aorta, which provides the functional supply of all body organs.
- Systemic veins return the oxygen-depleted blood to the right atrium.

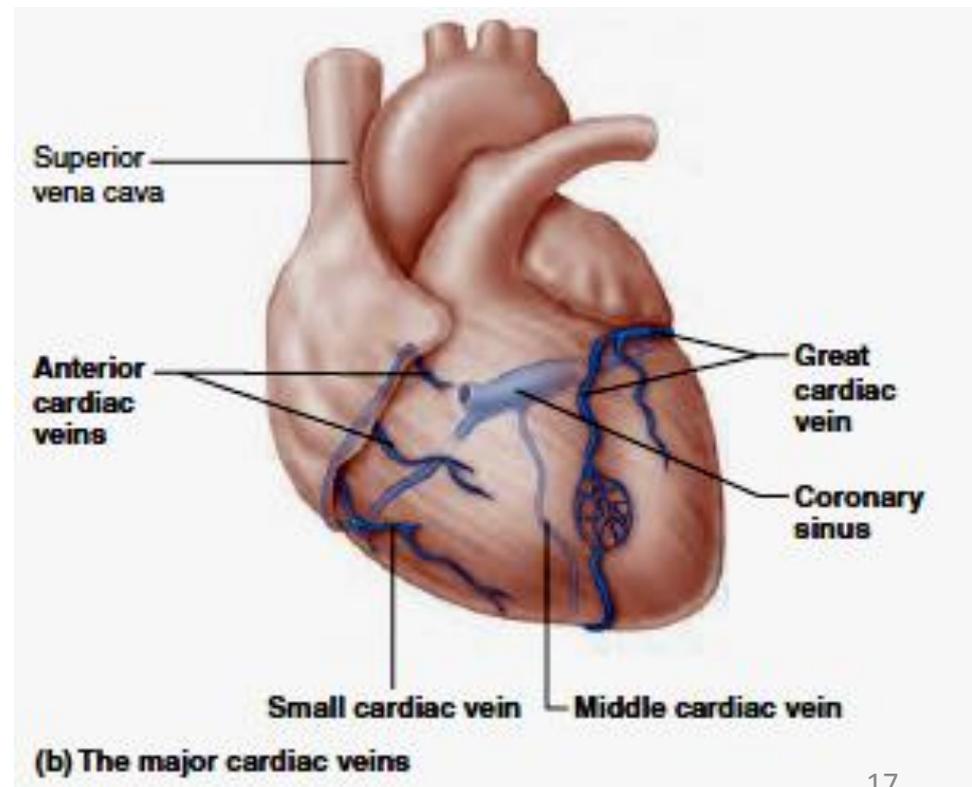
# Coronary Circulation

- The right and left coronary arteries branch from the aorta and via their main branches (anterior and posterior interventricular, right marginal, and circumflex arteries) supply the heart itself.

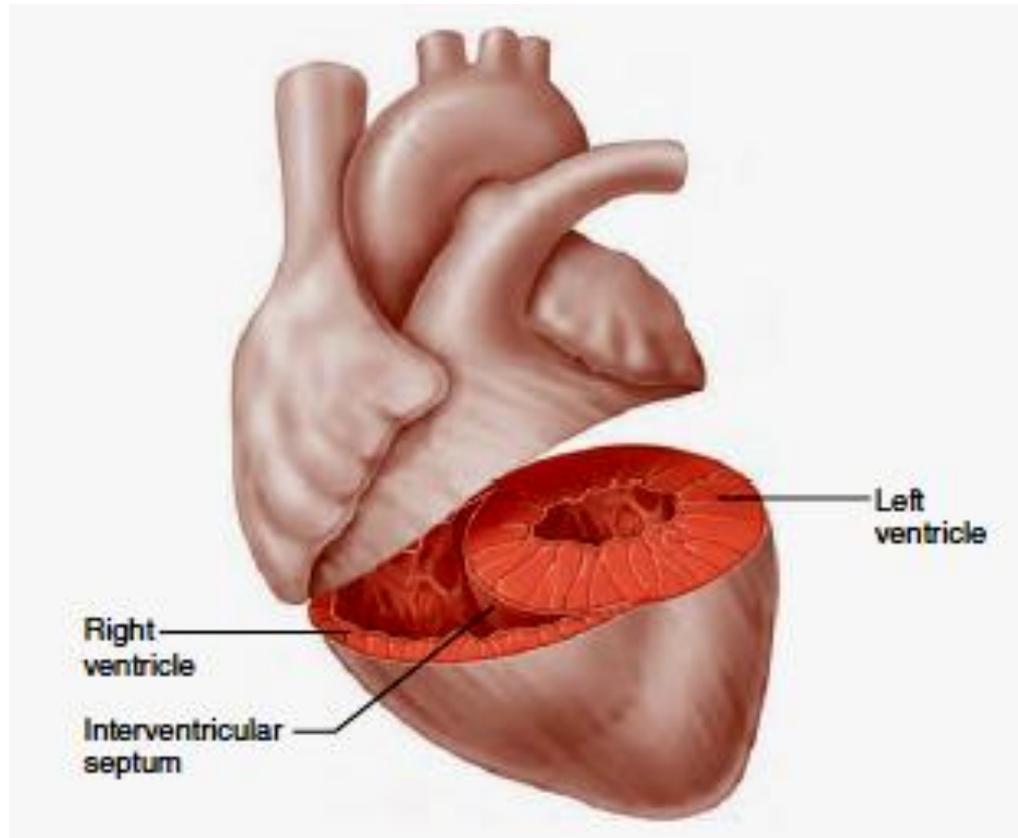


# Coronary Circulation

- Venous blood, collected by the cardiac veins (great, middle, and small), empties into the coronary sinus.
- Blood delivery to the myocardium occurs during heart relaxation.



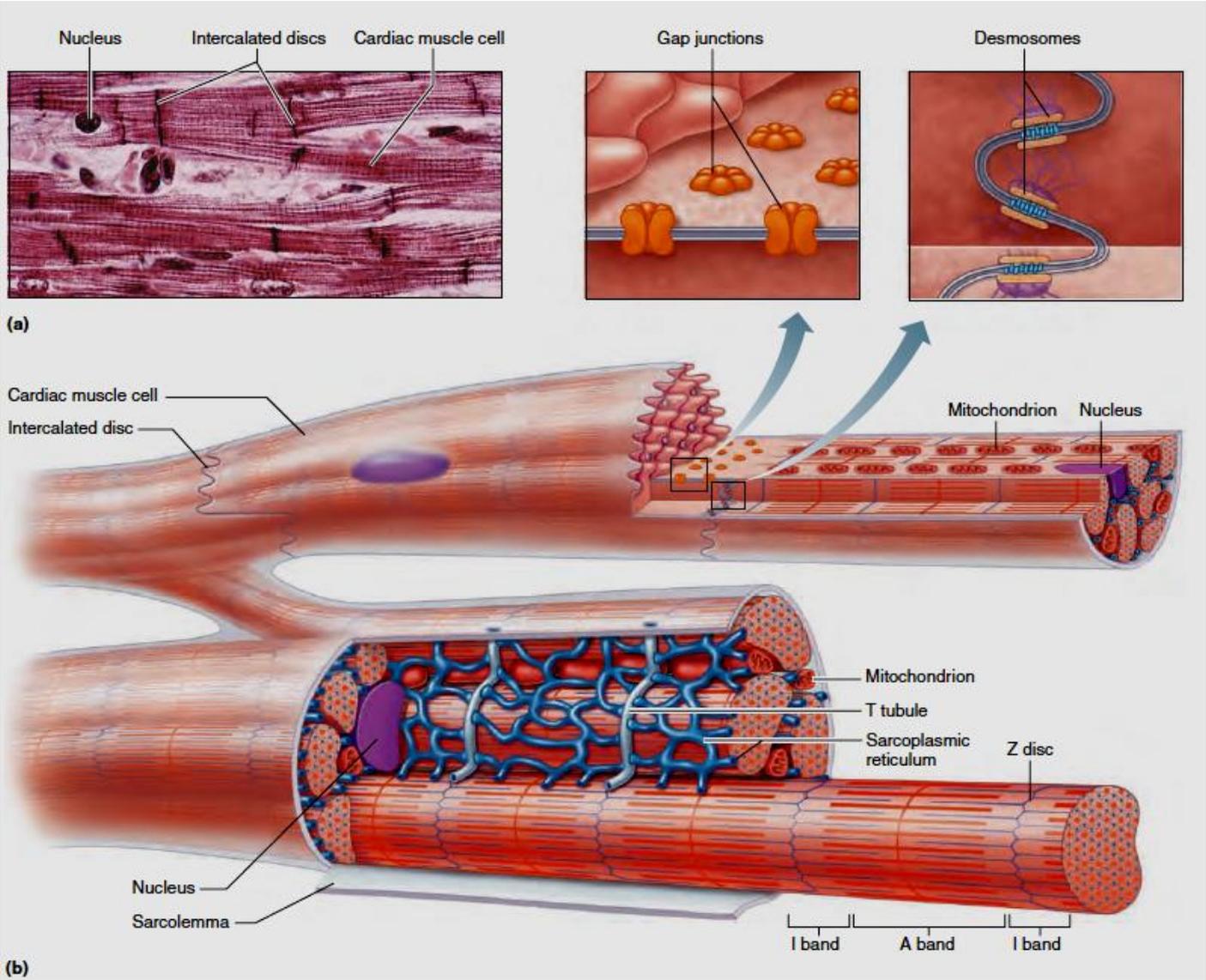
# Anatomical differences between the right and left ventricles



# Microscopic anatomy of cardiac muscle

- Cardiac muscle cells are branching, striated, generally uninucleate cells.
- They contain myofibrils consisting of typical sarcomeres.
- The myocardium behaves as a functional syncytium because of electrical coupling provided by gap junctions.

# Microscopic anatomy of cardiac muscle

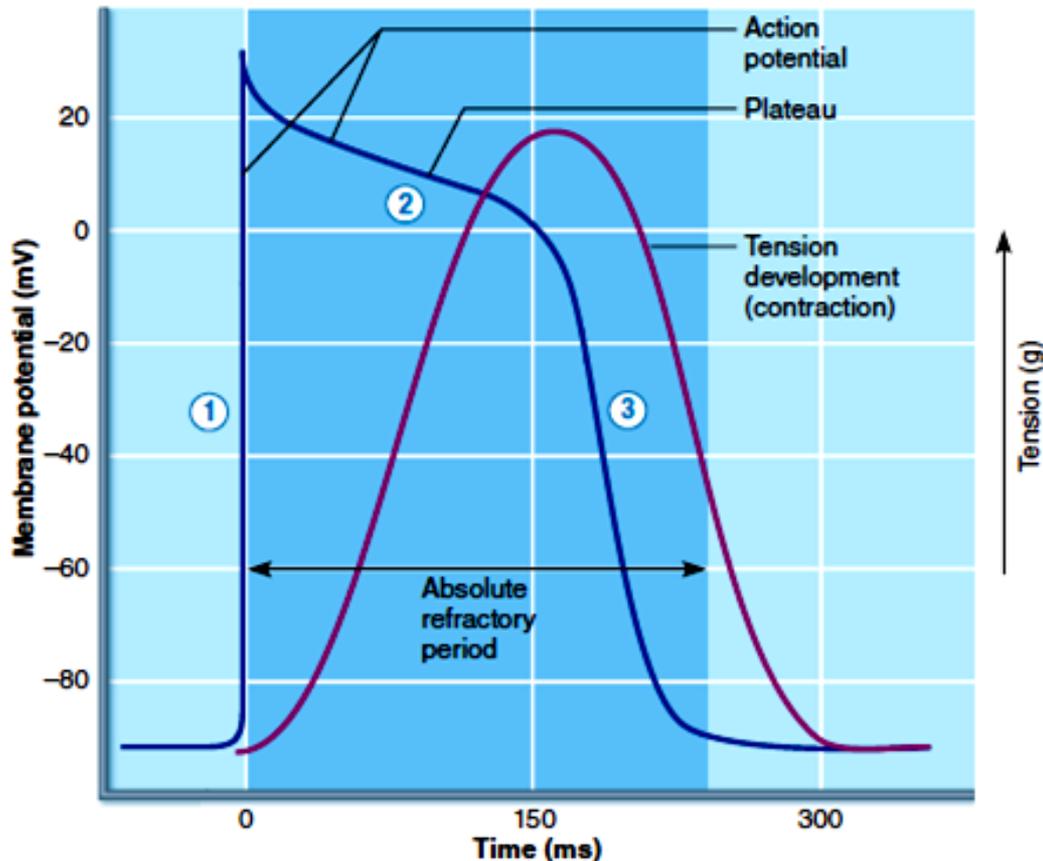


- Like skeletal muscle, cardiac muscle is striated and contracts by the sliding filament mechanism.
- However, in contrast to the long, cylindrical, multinucleate skeletal muscle fibers, cardiac cells are short, fat, branched, and interconnected.
- Each fiber contains one or at most two large, pale, *centrally located nuclei*

- Intercalated discs containing desmosomes and gap junctions connect adjacent cardiac cells.
- The desmosomes prevent adjacent cells from separating during contraction
- The gap junctions allow ions to pass from cell to cell, transmitting current across the entire heart.
- Because gap junctions electrically couple cardiac cells, the myocardium behaves as a single coordinated unit

- Large mitochondria account for 25–35% of the volume of cardiac cells (compared with only 2% in skeletal muscle), a characteristic that makes cardiac cells highly resistant to fatigue

# Mechanism and Events of Contraction



- 1. Depolarization:** is due to  $\text{Na}^+$  influx through fast voltage-gated  $\text{Na}^+$  channels. A positive feedback cycle rapidly opens many  $\text{Na}^+$  channels, reversing the membrane potential. Channel inactivation ends this phase.
- 2. Plateau phase:** is due to  $\text{Ca}^{2+}$  influx through slow  $\text{Ca}^{2+}$  channels. This keeps the cell depolarized because few  $\text{K}^+$  channels are open
- 3. Repolarization:** is due to  $\text{Ca}^{2+}$  channels inactivating and  $\text{K}^+$  channels opening. This allows  $\text{K}^+$  efflux, which brings the membrane potential back to its resting voltage.

# Fundamental differences between heart muscle and skeletal muscle contractile tissues

- **Means of stimulation:**

- Each skeletal muscle fiber must be stimulated to contract by a nerve ending, but some cardiac muscle cells are self-excitabile.
- These cells can initiate not only their own depolarization, but that of the rest of the heart as well, in a spontaneous and rhythmic way (automaticity) or (autorhythmicity)

# Fundamental differences between heart muscle and skeletal muscle contractile tissues

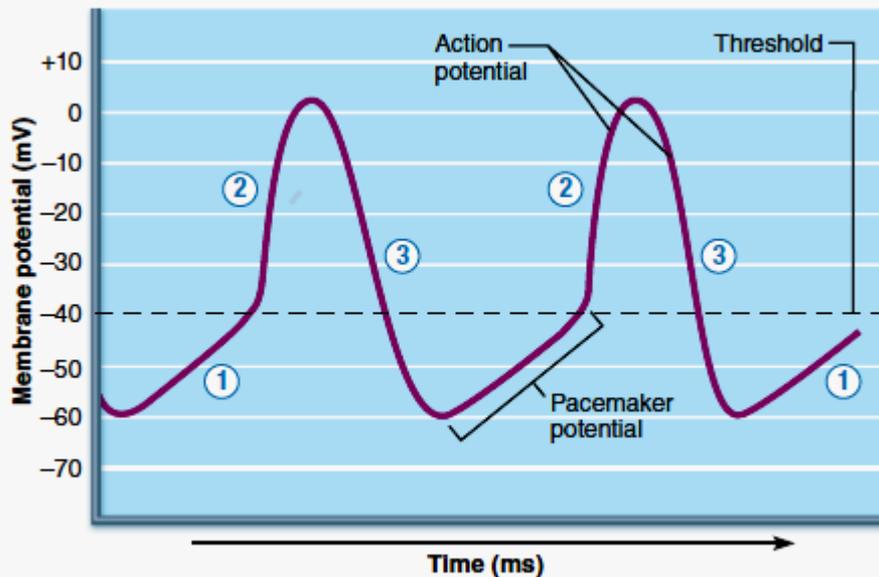
- **Organ versus motor unit contraction:**
  - In skeletal muscle, impulses do not spread from cell to cell. Only muscle fibers that are individually stimulated by nerve fibers contract.
  - in cardiac muscle, either all fibers in the heart contract as a unit or the heart doesn't contract at all.
  - This coordinated action occurs due to the electrical coupling via intercalated discs

# Fundamental differences between heart muscle and skeletal muscle contractile tissues

- **Length of absolute refractory period:**
  - The cardiac refractory period is normally longer

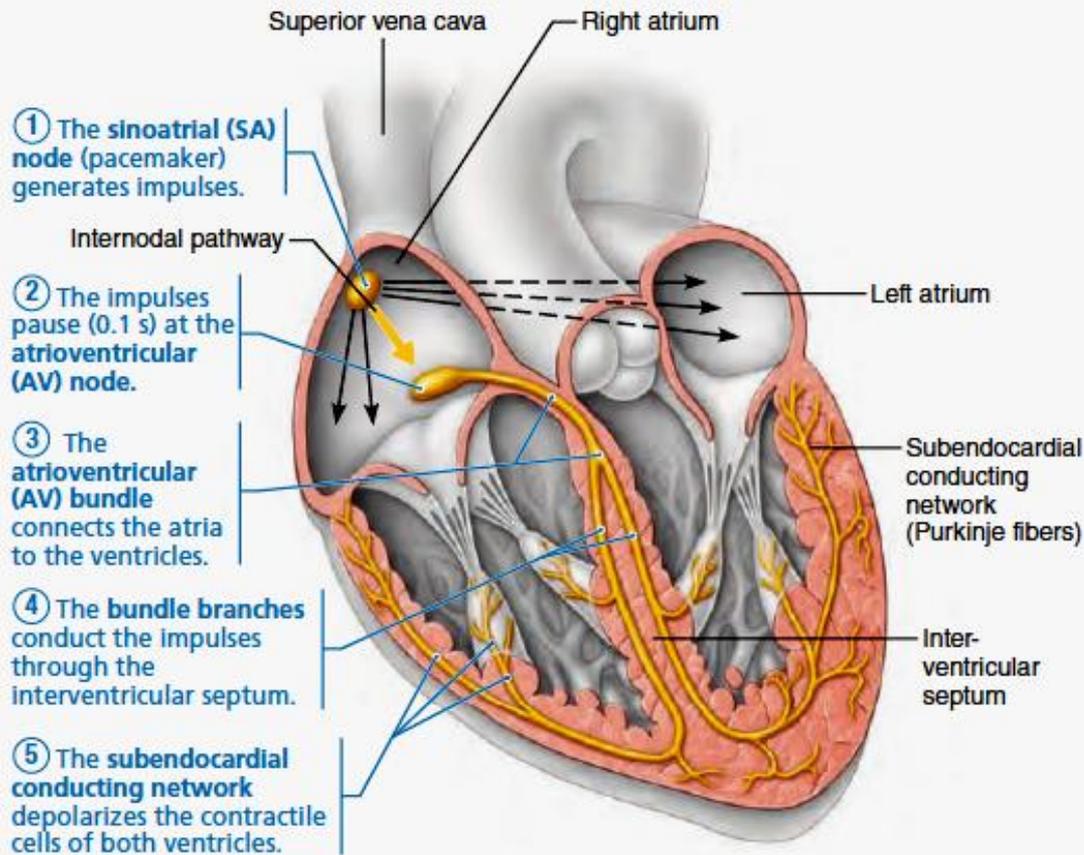
# Pacemaker and action potentials of pacemaker cells in the heart

- **Cardiac pacemaker cells (or autorhythmic cells)** making up the intrinsic conduction system have an unstable resting potential that continuously depolarizes, drifting slowly toward threshold.

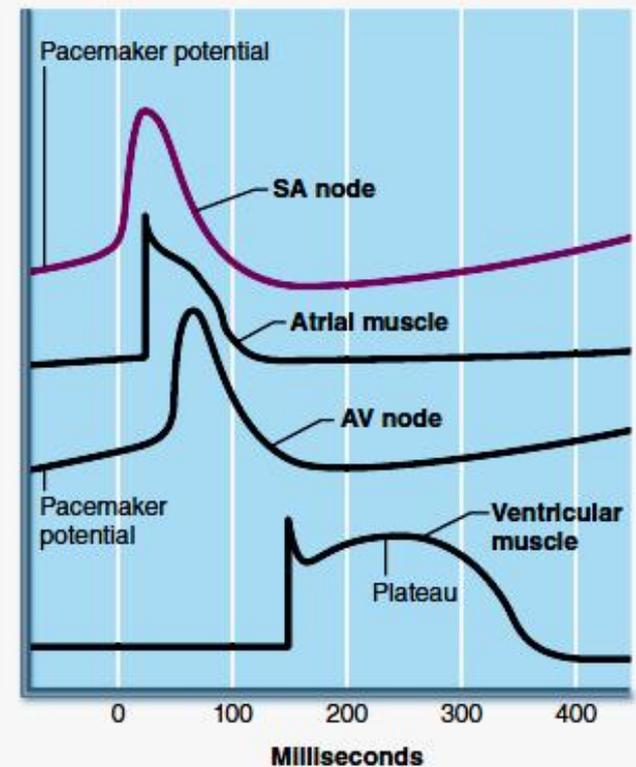


1. **Pacemaker potential:** This slow depolarization is due to both opening of  $\text{Na}^+$  channels and closing of  $\text{K}^+$  channels.
2. **Depolarization:** The action potential begins when the pacemaker potential reaches threshold. Depolarization is due to  $\text{Ca}^{2+}$  influx through  $\text{Ca}^{2+}$  channels.
3. **Repolarization:** is due to  $\text{Ca}^{2+}$  channels inactivating and  $\text{K}^+$  channels opening. This allows  $\text{K}^+$  efflux, which brings the membrane potential back to its most negative voltage.

# Intrinsic cardiac conduction system and action potential succession during one heartbeat



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



(b) Comparison of action potential shape at various locations

# Sinoatrial (SA) node

- located in the right atrial wall, just inferior to the entrance of the superior vena cava.
- The SA node typically generates impulses about 75 times every minute.
- The SA node sets the pace for the heart as a whole because no other region of the conduction system or the myocardium has a faster depolarization rate

# Atrioventricular (AV) node

- From the SA node, the depolarization wave spreads via gap junctions throughout the atria and via the internodal pathway to the AV node
- AV node is located in the inferior portion of the interatrial septum immediately above the tricuspid valve.
- At the AV node, the impulse is delayed for about 0.1 s, allowing the atria to respond and complete their contraction before the ventricles contract.
- Once through the AV node, the signaling impulse passes rapidly through the rest of the system.

# Atrioventricular (AV) bundle

- From the AV node, the impulse sweeps to the atrioventricular bundle (also called **the bundle of His**) in the superior part of the interventricular septum.
- Although the atria and ventricles abut each other, they are not connected by gap junctions.
- The AV bundle is the only electrical connection between them.
- The fibrous cardiac skeleton is nonconducting and insulates the rest of the AV junction.

# Right and left bundle branches

- The AV bundle persists only briefly before splitting into two pathways
- The right and left bundle branches, which course along the interventricular septum toward the heart apex

# Subendocardial conducting network

- Also called Purkinje fibers
- Essentially long strands of barrel-shaped cells with few myofibrils
- They complete the pathway through the interventricular septum, penetrate into the heart apex, and then turn superiorly into the ventricular walls.

# Subendocardial conducting network

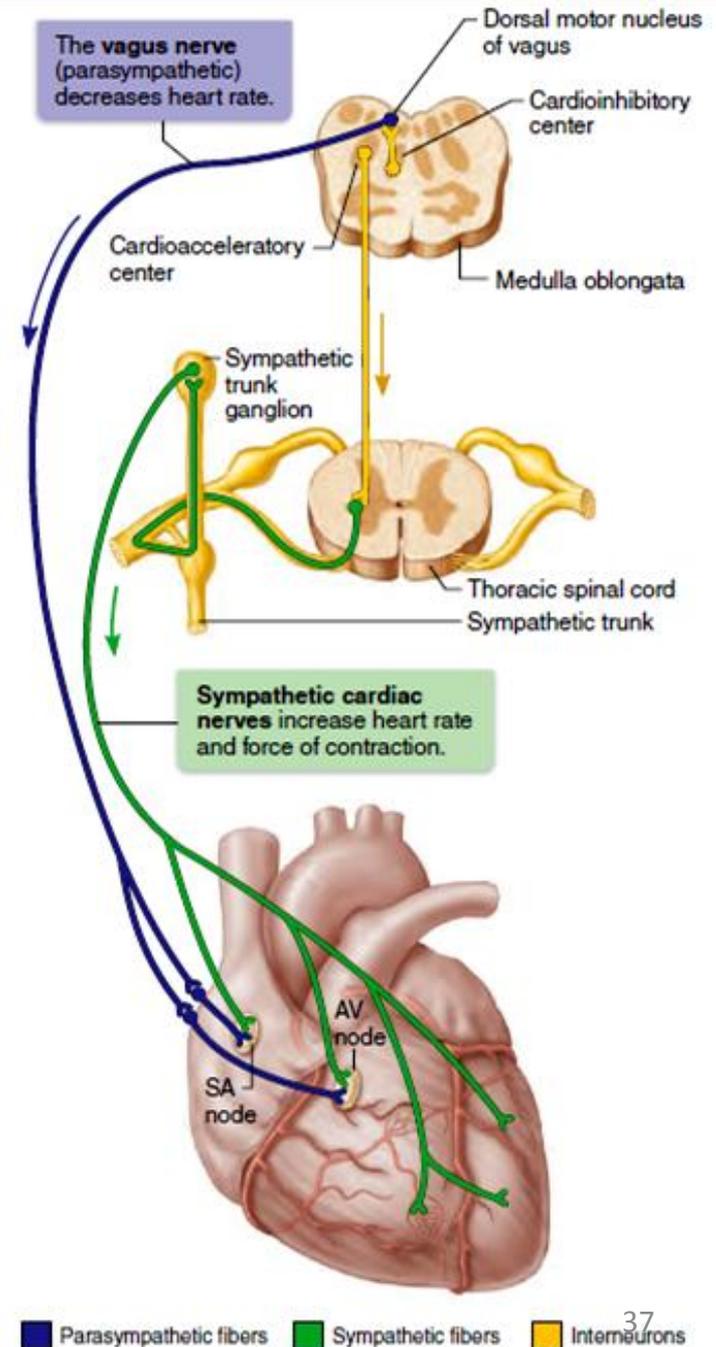
- The bundle branches excite the septal cells, but the bulk of ventricular depolarization depends on the large fibers of the conducting network and, ultimately, on cell-to-cell transmission of the impulse via gap junctions between the ventricular muscle cells.
- Because the left ventricle is much larger than the right, the subendocardial conducting network is more elaborate in that side of the heart.

# Modifying the Basic Rhythm: Extrinsic Innervation of the Heart

- The intrinsic conduction system sets the basic heart rate, but this can be modified by the autonomic nervous system
- The sympathetic nervous system (the “accelerator”) increases both the rate and the force of heartbeat.
- The parasympathetic activation (the “brakes”) slows the heart

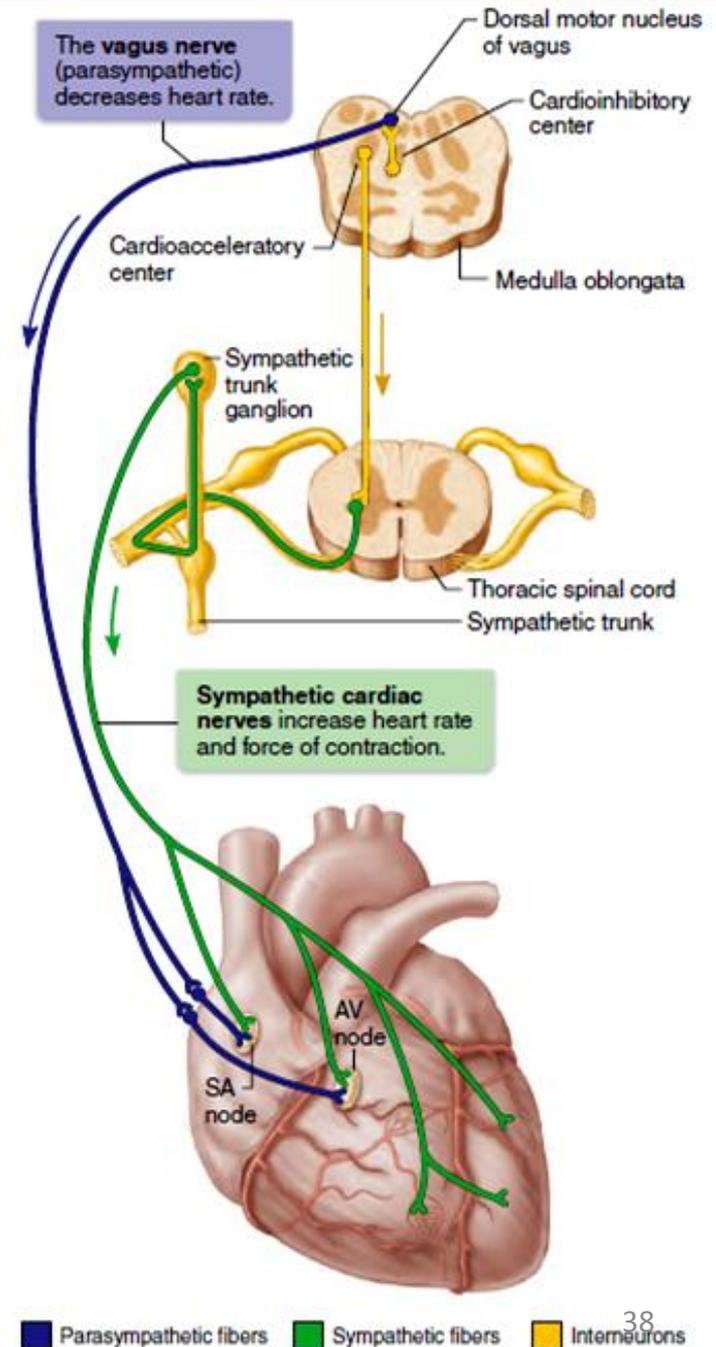
# Sympathetic Innervation:

- The cardiac centers are located in the medulla oblongata.
- The cardioacceleratory center projects preganglionic neurons that synapse with postganglionic neurons in the cervical and upper thoracic sympathetic trunk
- From there, postganglionic fibers run through the cardiac plexus to the heart where they innervate: the SA and AV nodes, heart muscle, and coronary arteries.

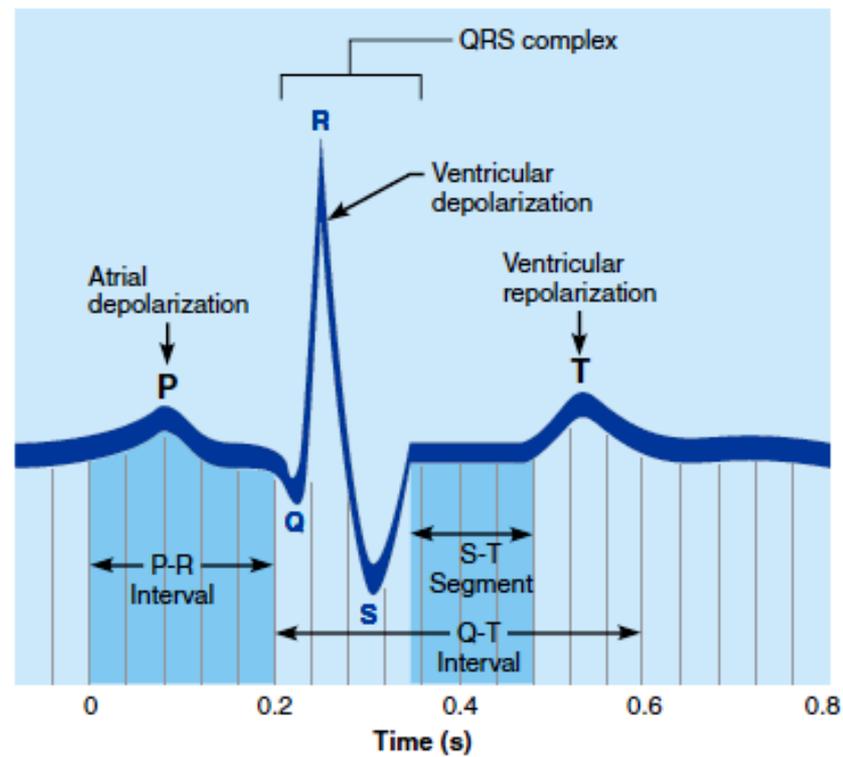
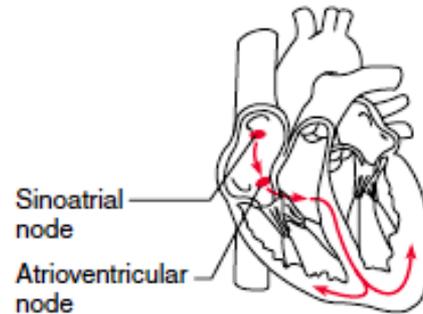


# Parasympathetic Innervation:

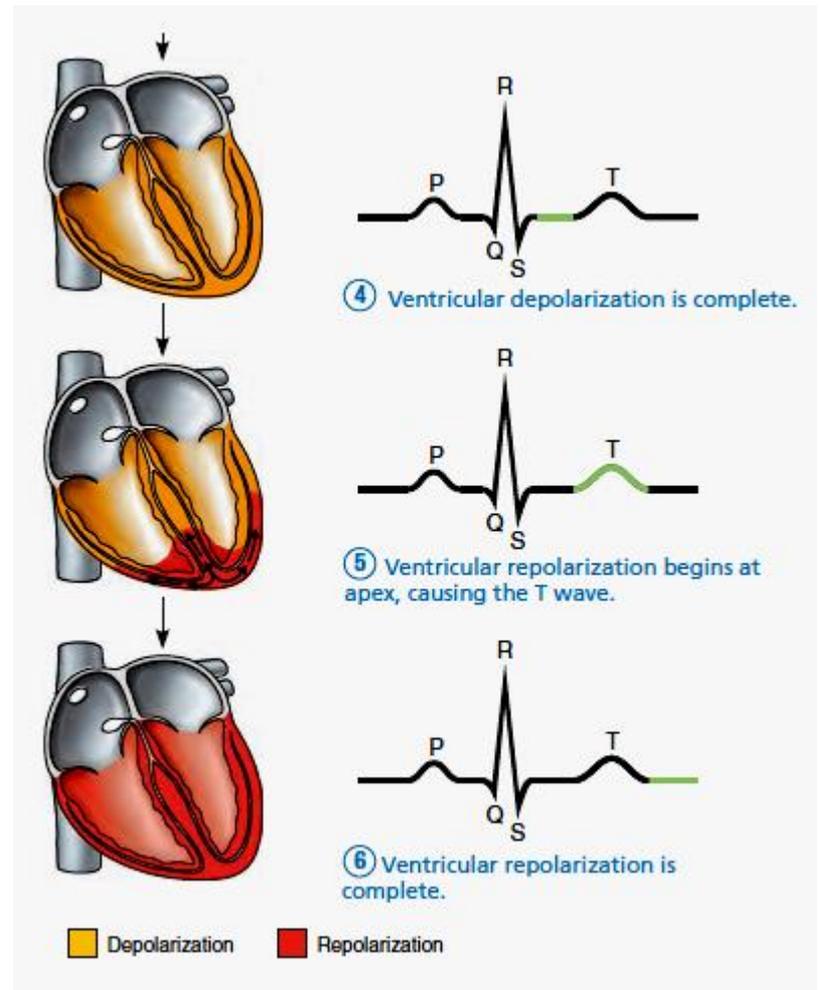
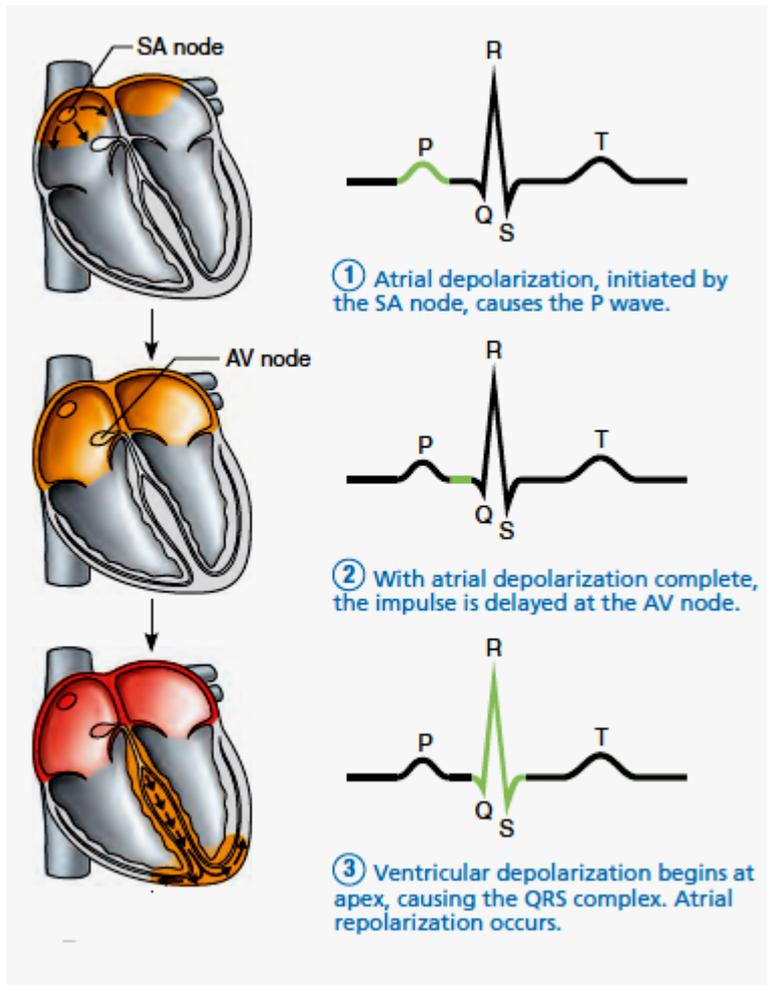
- The cardioinhibitory center sends impulses to the parasympathetic dorsal vagus nucleus in the medulla, which in turn sends inhibitory impulses to the heart via branches of the Vagus nerves.
- Most parasympathetic postganglionic motor neurons lie in ganglia in the heart wall and their fibers project most heavily to the SA and AV nodes.



# Electrocardiography (ECG)

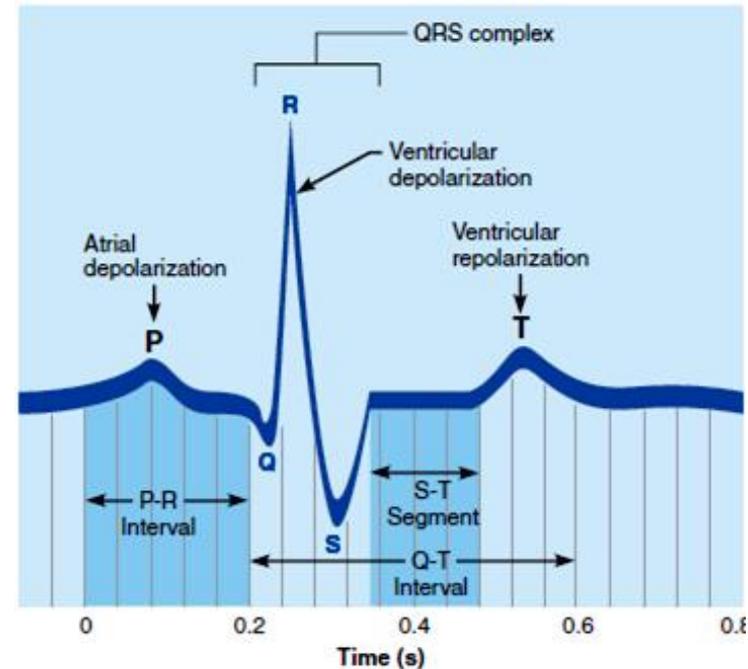


# Electrocardiography (ECG)



# Homeostatic Imbalance

- Enlarged R wave hints of enlarged ventricles
- S-T segment elevation or depression indicates cardiac ischemia
- Prolonged Q-T interval reveals a repolarization abnormality that increases the risk of ventricular arrhythmias



# Homeostatic Imbalance (Arrhythmias)

- **Arrhythmias:** irregular heart rhythms
- **Fibrillation:** condition of rapid and irregular or out-of-phase contractions in which control of heart rhythm is taken away from the SA node by rapid activity in other heart regions.
- Fibrillating ventricles are **useless** as pumps; and unless the heart is defibrillated quickly, circulation stops and brain death occurs.



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

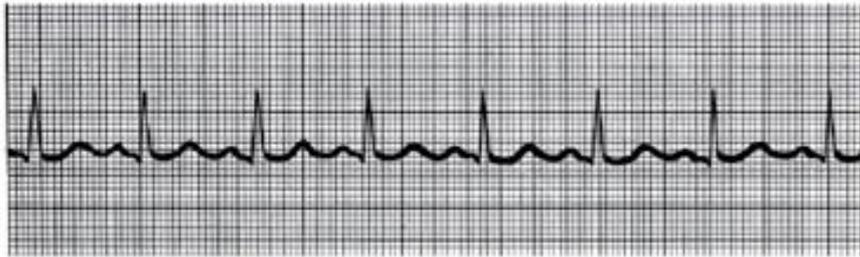
# Homeostatic Imbalance (Arrhythmias)

- **Defibrillation:** is accomplished by electrically shocking the heart, which interrupts its chaotic twitching by depolarizing the entire myocardium.
- It gives a chance for the SA node to takeover and re-function normally

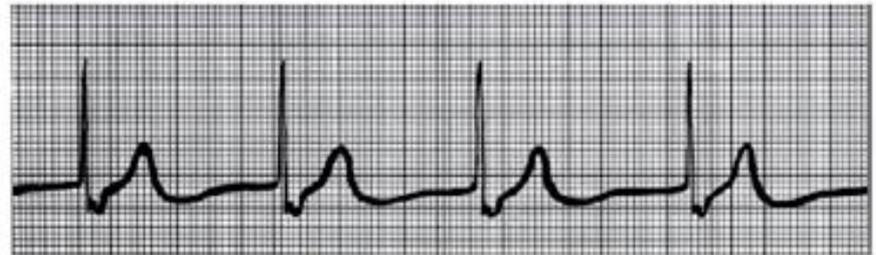
# Ectopic pacemakers

## Etiology of ectopic pacemakers generation:

- 1) **A defective SA node:** abnormal pacemaker, may appear and take over the pacing of heart rate, or the AV node may become the pacemaker (generates a junctional rhythm with 40 to 60 beats per minute), slower than sinus rhythm but still adequate to maintain circulation.



(a) Normal sinus rhythm.



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

# Ectopic pacemakers

## Etiology of ectopic pacemakers generation:

### 2) A small region of the heart becomes hyperexcitable:

Sometimes as a result of too much caffeine (several cups of coffee) or nicotine (excessive smoking)

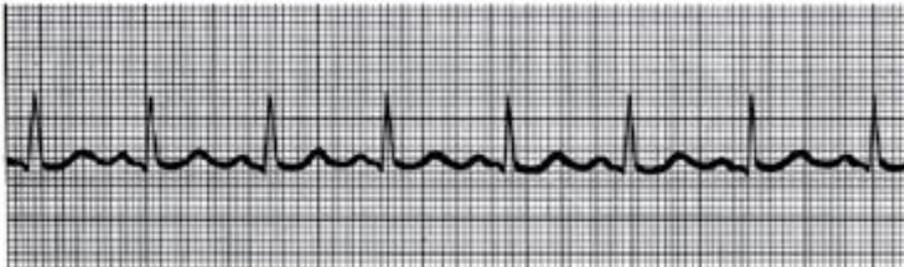
- SA node may be operating normally
- They generate impulses more quickly than the SA node
- This leads to a premature contraction or **extrasystole** before the SA node initiates the next contraction

# Heart block

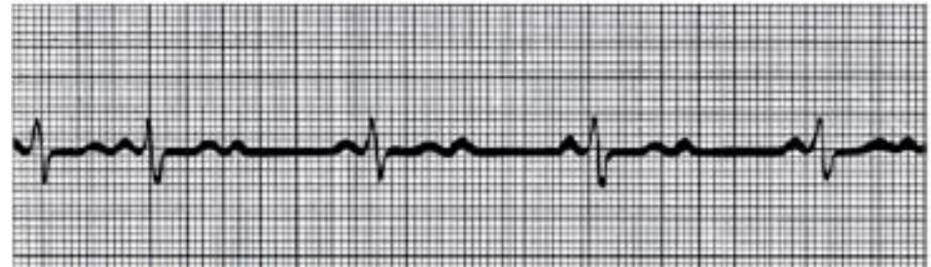
- The only route for impulse transmission from atria to ventricles is through the AV node.
- Thus any damage to the AV node interferes with the ability of the ventricles to receive pacing impulses, causing **heart block**.
- **In total heart block no impulses** get through and the ventricles beat at their intrinsic rate, which is too slow (about 30 times per minute) to maintain adequate circulation.

# Heart block

- **In partial heart block**, only some of the atrial impulses reach the ventricles.
- In both cases, artificial pacemakers are implanted to recouple the atria to the ventricles as necessary.

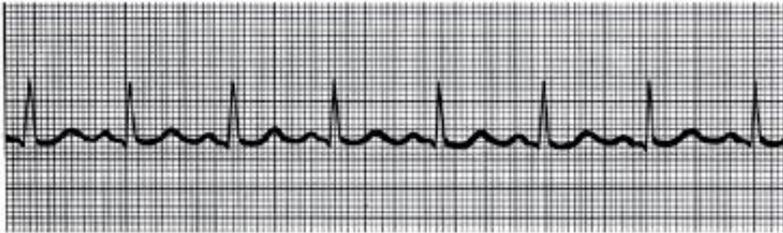


(a) Normal sinus rhythm.



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

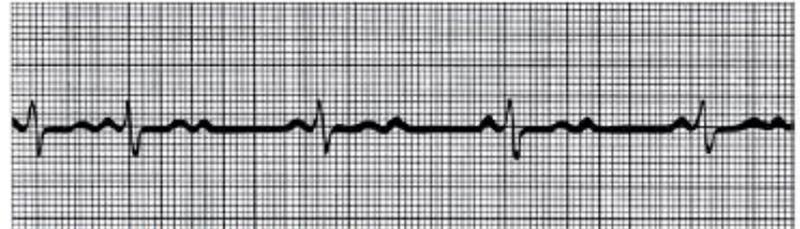
# Homeostatic Imbalance



(a) Normal sinus rhythm.



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



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

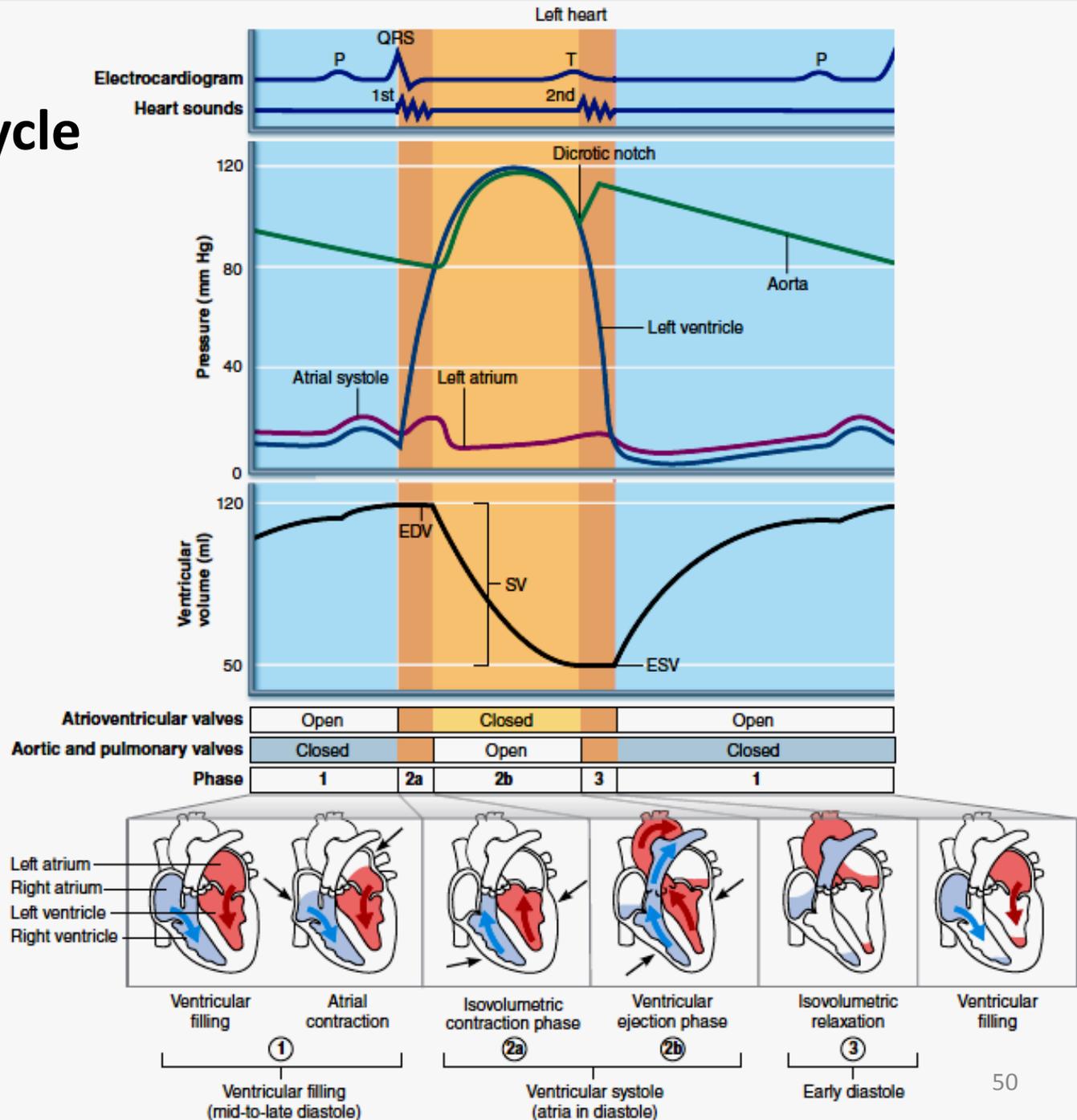


(d) Ventricular fibrillation. These chaotic, grossly irregular ECG deflections are seen in acute heart attack and electrical shock.

# Mechanical Events: The Cardiac Cycle

- **Systole:** periods of contraction
- **Diastole:** periods of relaxation
- **Cardiac cycle:** includes all events associated with the blood flow through the heart during one complete heartbeat
- Cardiac cycle is marked by a succession of pressure and blood volume changes in the heart

# The Cardiac cycle



## 1. Ventricular filling: mid-to-late diastole:

- Pressure in the heart is low
- Blood returning from the circulation is flowing passively through the atria and the open AV valves into the ventricles
- The aortic and pulmonary valves are closed
- More than 80% of ventricular filling occurs during this period

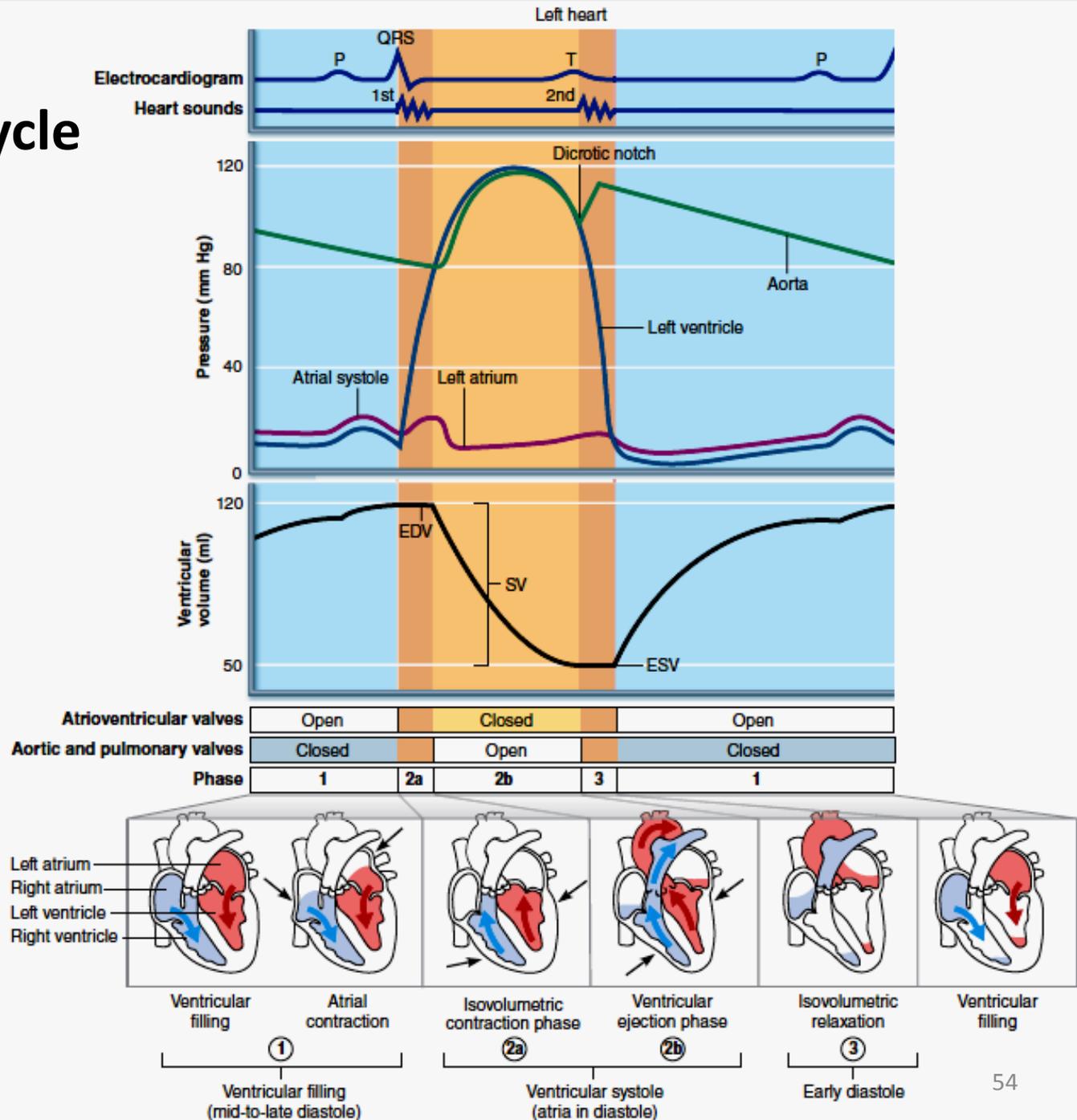
## 1. Ventricular filling: mid-to-late diastole:

- The AV valve flaps begin to drift toward the closed position.
- The remaining 20% is delivered to the ventricles when the atria contract toward the end of this phase (following P-wave).
- This causes a sudden slight rise in atrial pressure

## 1. Ventricular filling: mid-to-late diastole:

- At this point the ventricles are in the last part of their diastole and have the maximum volume of blood they will contain in the cycle, an amount called the **end diastolic volume (EDV)**.
- Then the atria relax and the ventricles depolarize (QRS complex).
- Atrial diastole persists through the rest of the cycle.

# The Cardiac cycle



## 2. Ventricular systole (atria in diastole).

- As the atria relax, the ventricles begin contracting.
- The ventricular pressure rises rapidly and sharply, closing the AV valves.
- The split second period when the ventricles are completely closed chambers and the blood volume in the chambers remains constant is the **isovolumetric contraction phase**

## 2. Ventricular systole (atria in diastole).

- Ventricular pressure continues to rise.
- When it finally exceeds the pressure in the large arteries, the isovolumetric stage ends as the SL valves are forced open and blood rushes from the ventricles into the aorta and pulmonary trunk.
- During this **ventricular ejection phase**, the pressure in the aorta normally reaches about 120 mm Hg

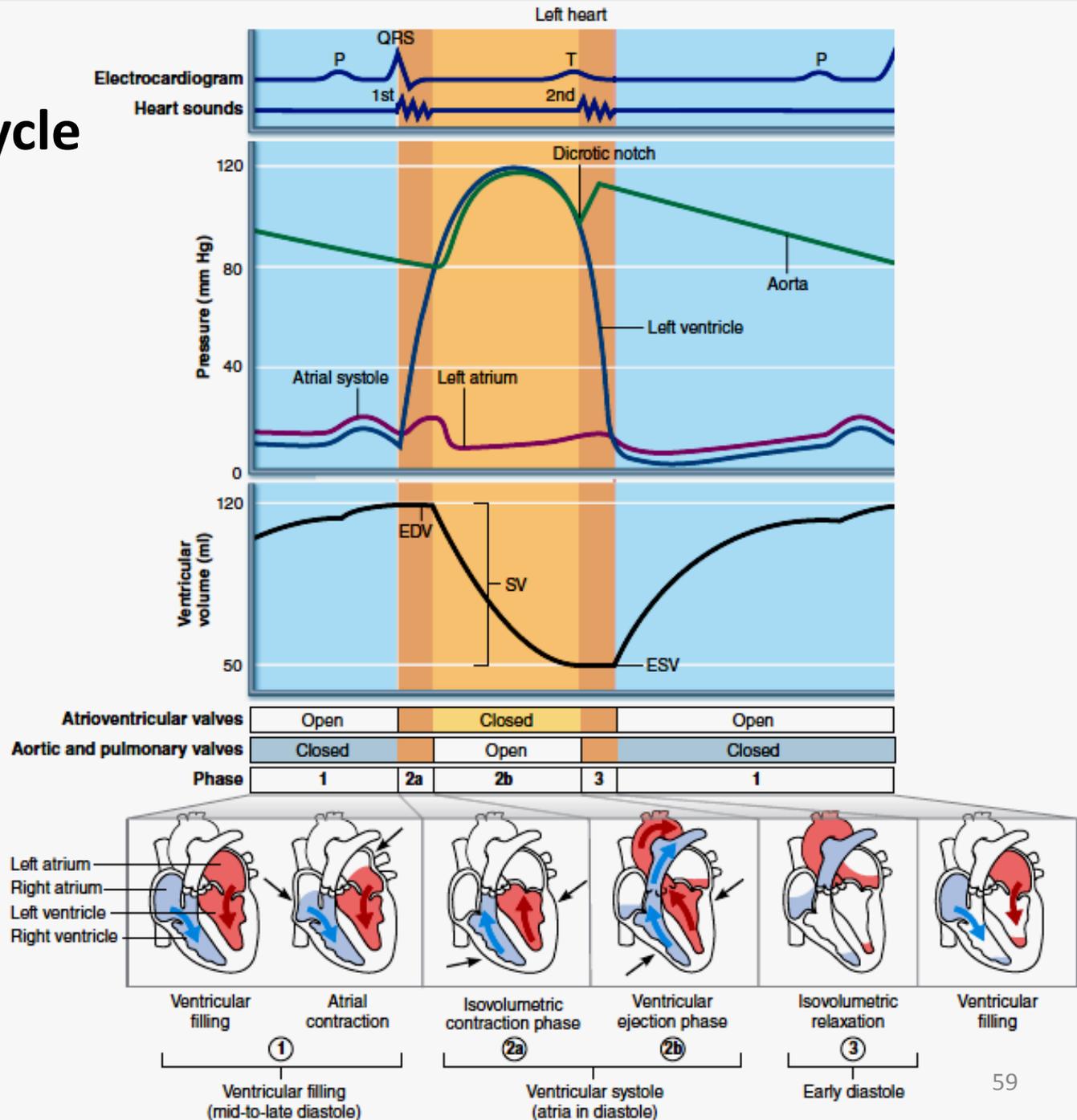
### **3. Isovolumetric relaxation: early diastole.**

- During this brief phase following the T wave, the ventricles relax.
- Ventricular pressure drops rapidly and blood in the aorta and pulmonary trunk flows back toward the heart, closing the SL valves.

### 3. Isovolumetric relaxation: early diastole.

- Therefore, the blood remaining in their chambers (**end systolic volume (ESV)**), is no longer compressed
- Closure of the aortic valve raises aortic pressure briefly as back flowing blood rebounds off the closed valve cusps.
- Once again the ventricles are totally closed chambers.

# The Cardiac cycle



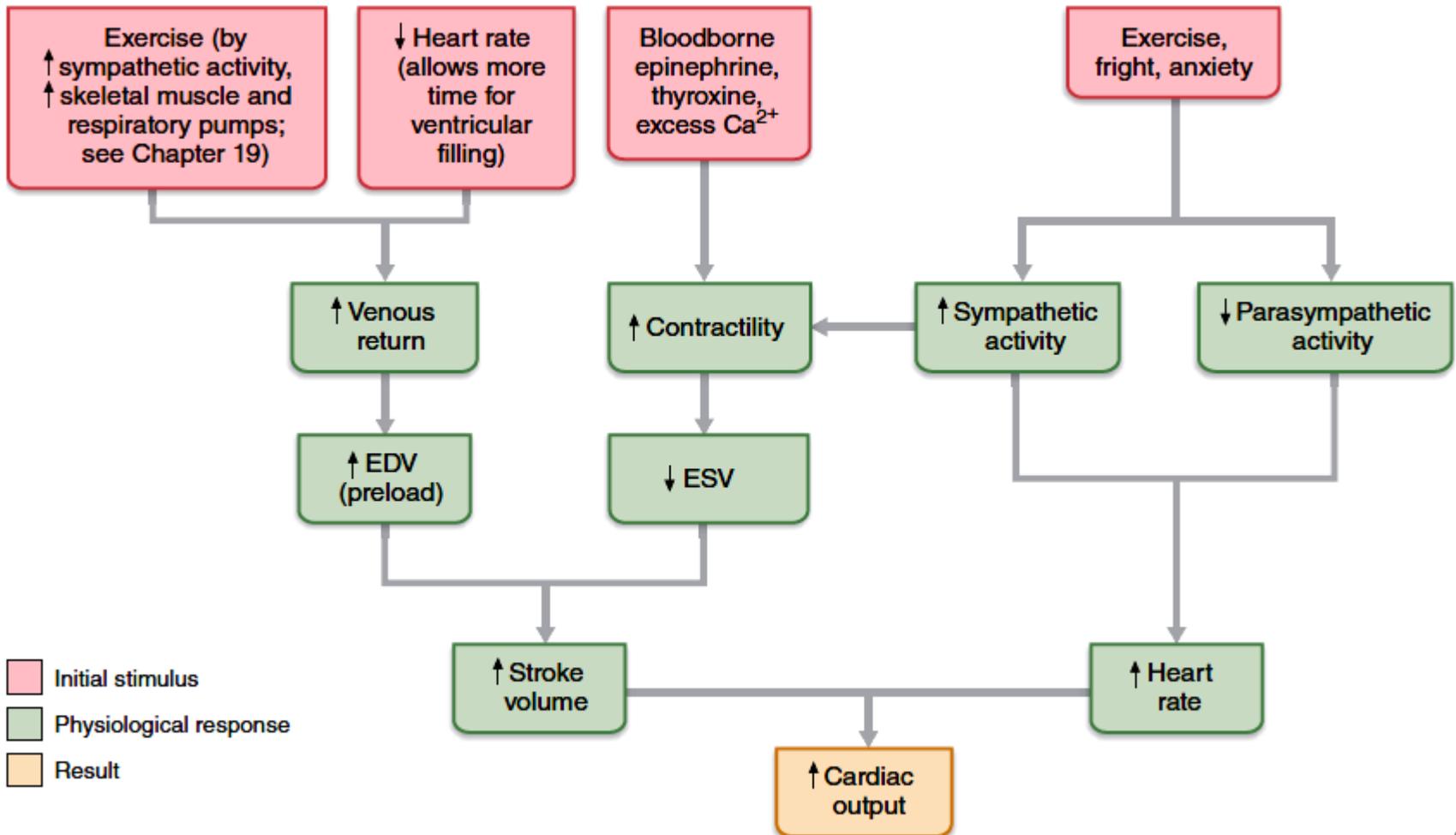
# Cardiac Output (CO)

- It is the amount of blood pumped out by each ventricle in 1 minute.
- $CO = HR \times SV$
- HR = Heart rate (normally 75 beat/min).
- SV = Stroke volume (normally 70 ml/beat).
- Stroke volume is defined as the volume of blood pumped out by one ventricle with each beat.
- In general, stroke volume correlates with the force of ventricular contraction

$$\begin{aligned} CO = HR \times SV &= \frac{75 \text{ beats}}{\text{min}} \times \frac{70 \text{ ml}}{\text{beat}} \\ &= \frac{5250 \text{ ml}}{\text{min}} = \frac{5.25 \text{ L}}{\text{min}} \end{aligned}$$

- The normal adult blood volume is about 5 L.
- The entire blood supply passes through each side of the heart once each minute.
- Cardiac reserve is the difference between resting and maximal CO.
- In nonathletic people, cardiac reserve is typically 4 to 5 times resting CO (20–25 L/min)
- CO in trained athletes during competition may reach 35 L/min (7 times resting CO)

# Factors involved in determining cardiac output



# Regulation of Stroke Volume

- Activation of the sympathetic nervous system increases heart rate and contractility
- Parasympathetic activation decreases heart rate but has little effect on contractility.
- At resting state, the effect of parasympathetic nervous system is dominant on the heart.
- Chemical regulation of the heart is effected by Hormones (epinephrine and thyroxine) and ions (particularly  $K^+$  and  $Ca^{2+}$ ).
- Ion imbalances severely impair heart activity

# Regulation of Stroke Volume

- **Preload:** Degree of Stretch of Heart Muscle The degree to which cardiac muscle cells are stretched just before they contract (also controls stroke volume)
- In normal heart, the higher the preload, the higher the stroke volume will be.
- This relationship between preload and stroke volume is called the **Frank-Starling law of the heart**
- Resting cardiac cells are normally shorter than optimal length. As a result, stretching cardiac cells can produce dramatic increases in contractile force.

# Regulation of Stroke Volume

- The most important factor stretching cardiac muscle is **venous return**, the amount of blood returning to the heart and distending its ventricles

# Regulation of Stroke Volume

- **Afterload: Back Pressure Exerted by Arterial Blood**
  - It is the pressure that the ventricles must overcome to eject blood.
  - It is the back pressure that arterial blood exerts on the aortic and pulmonary valves—about 80 mm Hg in the aorta and 10 mm Hg in the pulmonary trunk.

# Regulation of Stroke Volume

- **Afterload: Back Pressure Exerted by Arterial Blood**
  - In healthy individuals, afterload is not a major determinant of SV because it is relatively constant.
  - In people with hypertension, afterload is important because it reduces the ability of the ventricles to eject blood.
  - Consequently, more blood remains in the heart after systole, increasing ESV and reducing stroke volume

# Homeostatic Imbalance

- **Tachycardia: “heart hurry”** is an abnormally fast heart rate (more than 100 beats/min) that may result from:
  - Elevated body temperature
  - Stress
  - Certain drugs
  - Heart disease.
  - Persistent tachycardia is considered pathological because tachycardia occasionally promotes fibrillation.

# Homeostatic Imbalance

- **Bradycardia:** is a heart rate slower than 60 beats/min.  
It may result from:
  - Low body temperature
  - Certain drugs
  - Parasympathetic nervous activation

# Homeostatic Imbalance

- **Bradycardia:** is a heart rate slower than 60 beats/min.  
It may result from:
  - Low body temperature
  - Certain drugs
  - Parasympathetic nervous activation
- **Congestive heart failure (CHF):** the heart acts as an inefficient pump, due to myocardium weakness, as a result CO is inadequate to meet tissue needs

# Homeostatic Imbalance

- **Coronary atherosclerosis:**
  - Fatty buildup that clogs the coronary arteries
  - It impairs blood and oxygen delivery to cardiac cells.
  - The heart becomes increasingly hypoxic and begins to contract ineffectively

# Homeostatic Imbalance

- **Persistent high blood pressure:**

## Classification of Blood Pressure

	Systolic mm Hg		Diastolic mm Hg
<b>Normal</b>	<120	and	<80
<b>Pre-hypertensive</b>	120-139	or	80-90
<b>Stage I</b>	140-159	or	90-99
<b>Stage II</b>	≥160	or	≥100
<b>Hypertensive emergency</b>	≥180	or	≥120

- If afterload is chronically elevated, ESV rises and the myocardium hypertrophies.
- Eventually, the myocardium becomes progressively weaker

# Homeostatic Imbalance

- **Myocardial infarctions (MI):**
  - Following MIs (heart attacks) depresses pumping efficiency because noncontractile fibrous (scar) tissue replaces the dead heart cells
  - This can end up with heart failure

# Homeostatic Imbalance

- **Dilated cardiomyopathy (DCM):**
  - The ventricles stretch (dilate) and become flabby and the myocardium deteriorates

## **Some potential underlying causes include:**

- Drug toxicity (alcohol, cocaine, excess catecholamines, chemotherapeutic agents)
- Inflammation of the heart following an infection

# Homeostatic Imbalance

- **Pulmonary congestion**

- If the left side fails
- The right side continues to propel blood to the lungs, but the left side does not adequately eject the returning blood into the systemic circulation.
- Blood vessels in the lungs become engorged with blood, the pressure in them increases, and fluid leaks from the circulation into the lung tissue, causing pulmonary edema.
- If the congestion is untreated, the person suffocates