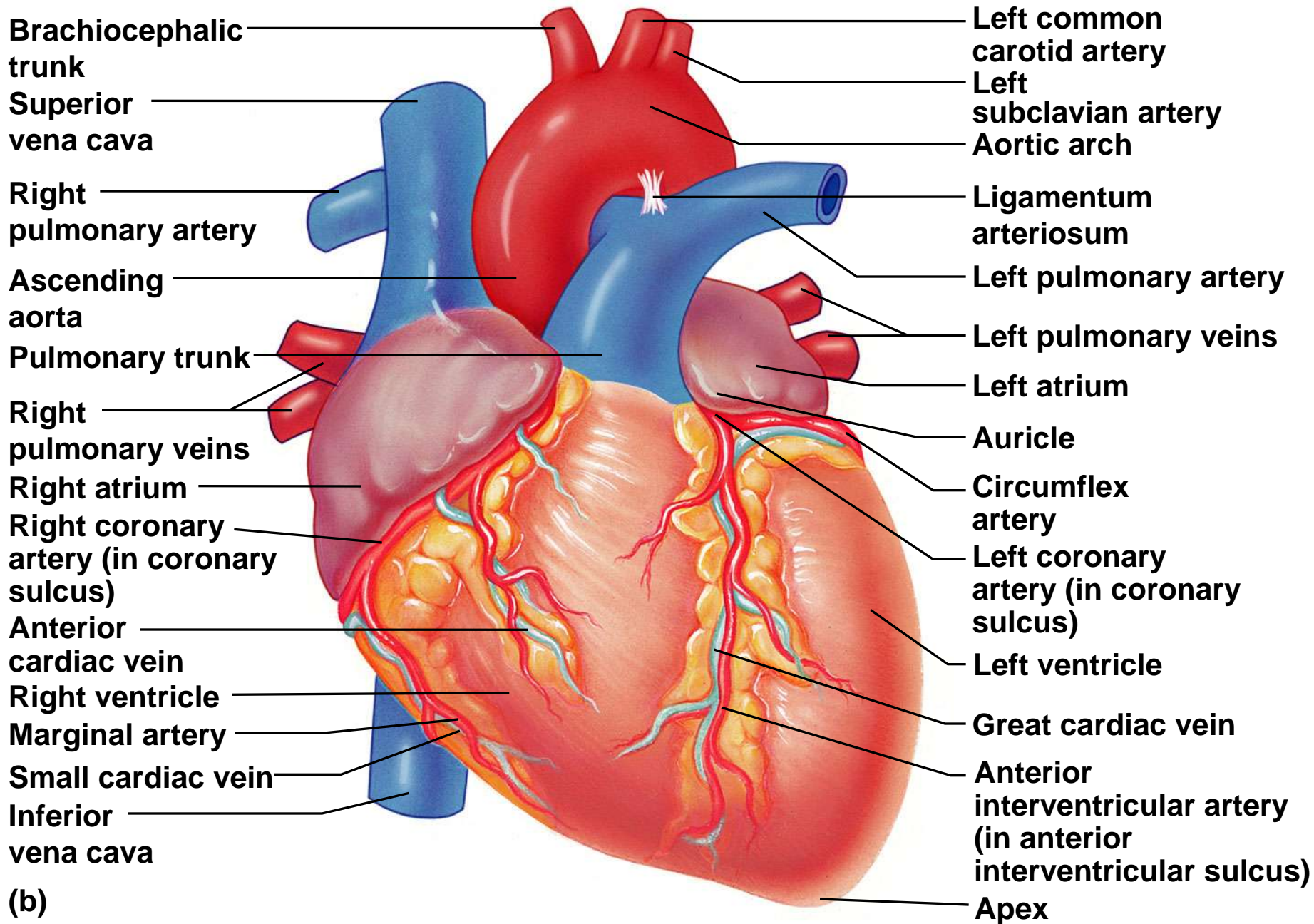

THE HEART

Dr. Ali Ebneshahidi

Functions is of the heart & blood vessels

- 1. the heart is an essential pumping organ in the cardiovascular system where the **right heart** pumps deoxygenated blood (returned from body tissues) to the lungs for gas exchange, while the **left heart** pumps oxygenated blood (returned from the lungs) to tissues cells for sustaining cellular respiration.

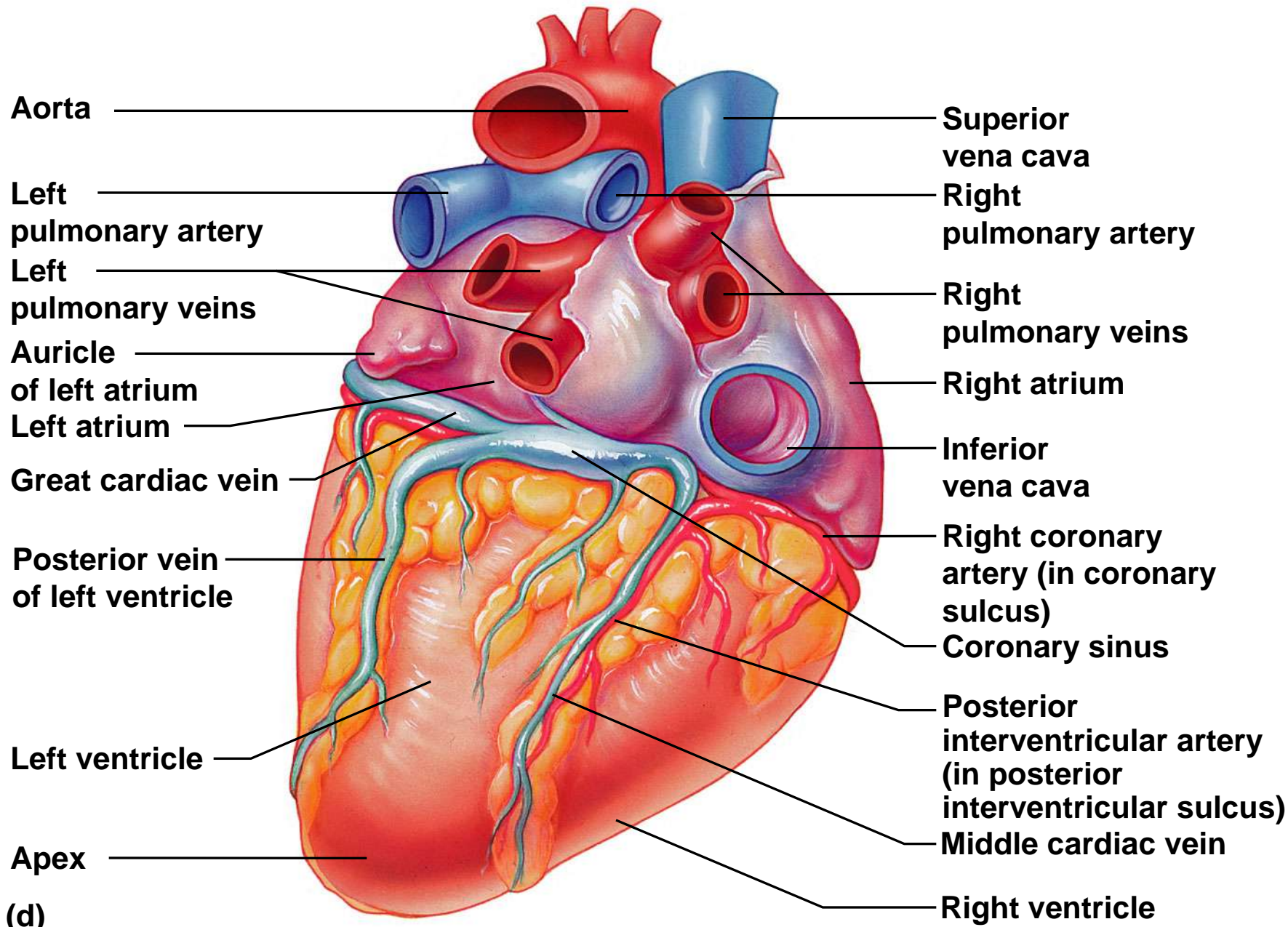
- 2. Attached to the heart is blood vessels that transport blood in various circulation pathways- **pulmonary blood vessels** transport blood between the heart and the lungs, and **systemic blood vessels** transport blood between the heart and body tissues.



Heart chambers

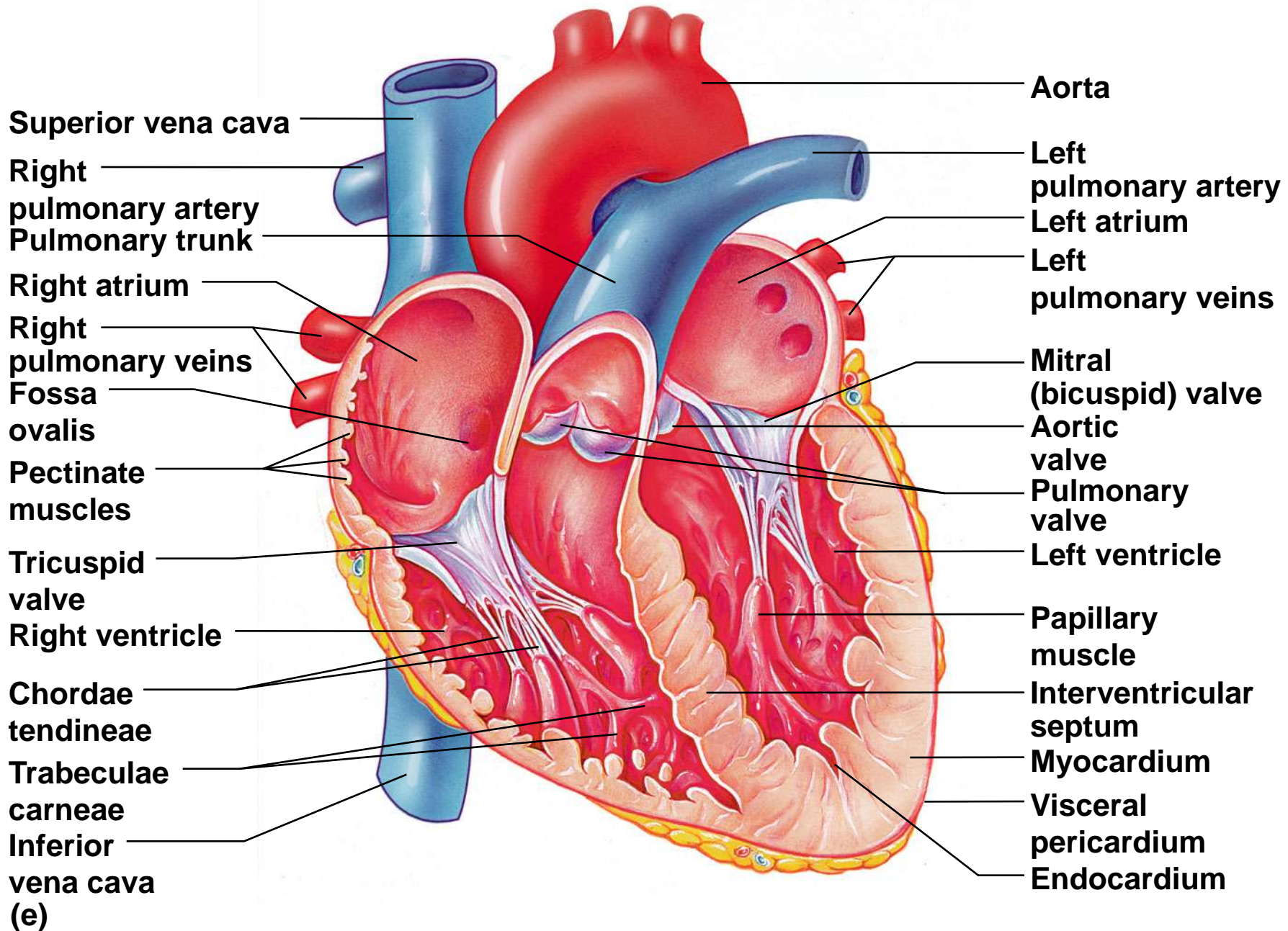
- hollow cavities within the heart for containing blood.
- Two smaller chambers called **atrium** are near the base, and two larger chambers called **ventricle** are close to the apex.
- **Right atrium (RA)** after receiving deoxygenated blood from body tissues through the superior and inferior vena cava, pumps the blood into the **right ventricle (RV)** via the right atria ventricular orifice. **RV** then pumps the blood to the lungs for gas exchange, through the pulmonary trunk and arteries.
- **Left atrium (LA)** after receiving oxygenated blood from the lungs through the pulmonary veins, pumps the blood into the **left ventricle (LV)** via the left atria ventricular orifice.

-
- LV then pumps the blood to the body tissues for supplying oxygen to every body cell, through the aorta.
 - RA and LA are separated by a central heart wall called **interatrial septum**, while RV and LV are separated by **interventricular septum**.
 - LV has a thicker myocardium layer (for stronger contractions) and contains rough ridges called **trabeculae carneae** (for containing a larger blood volume in exercising conditions).
 - Note: **Coronary arteries** (from the first branching of aorta) supply oxygenated blood to the cardiac muscle.



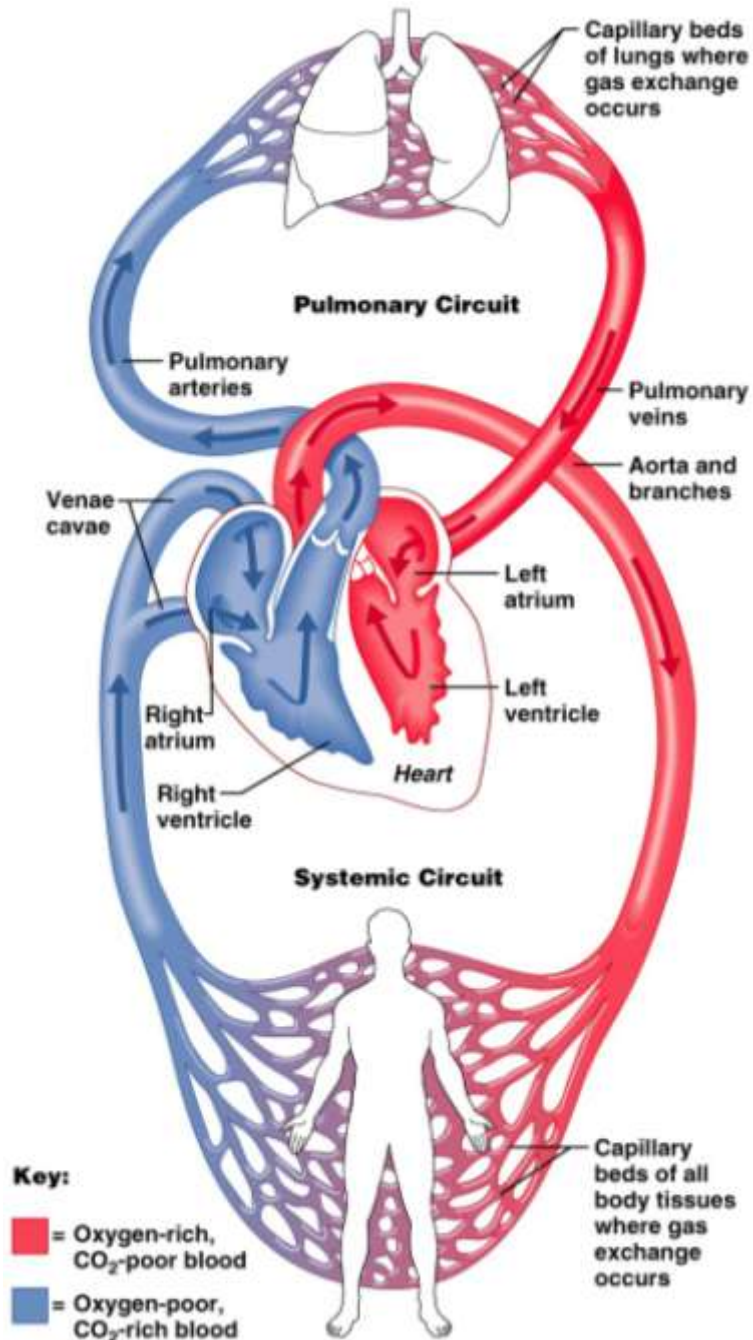
Heart Valves

- Two heart valves located between atria and ventricles are called **atrioventricular valves (AV valves)** which include the **tricuspid valve** between **RA** and **RV** , and **bicuspid valve** (or mitral valve) between **LA** and **LV**.
- Two heart valves located at the exiting arteries are called **semilunar valves (SL valves)** which include the **pulmonic semilunar valve** at the base of pulmonary trunk, and the **aortic semilunar valve** at the base of aorta.
- Each AV valve consists of **cusps** (extensions of endocardium), **chordae tendineae**, and **papillary muscles** (the latter two are designed to prevent eversion of the cusps into the atria).
- AV valves prevent backflow into atria, while SL valves prevent backflow into ventricles.



Circulation Pathways:

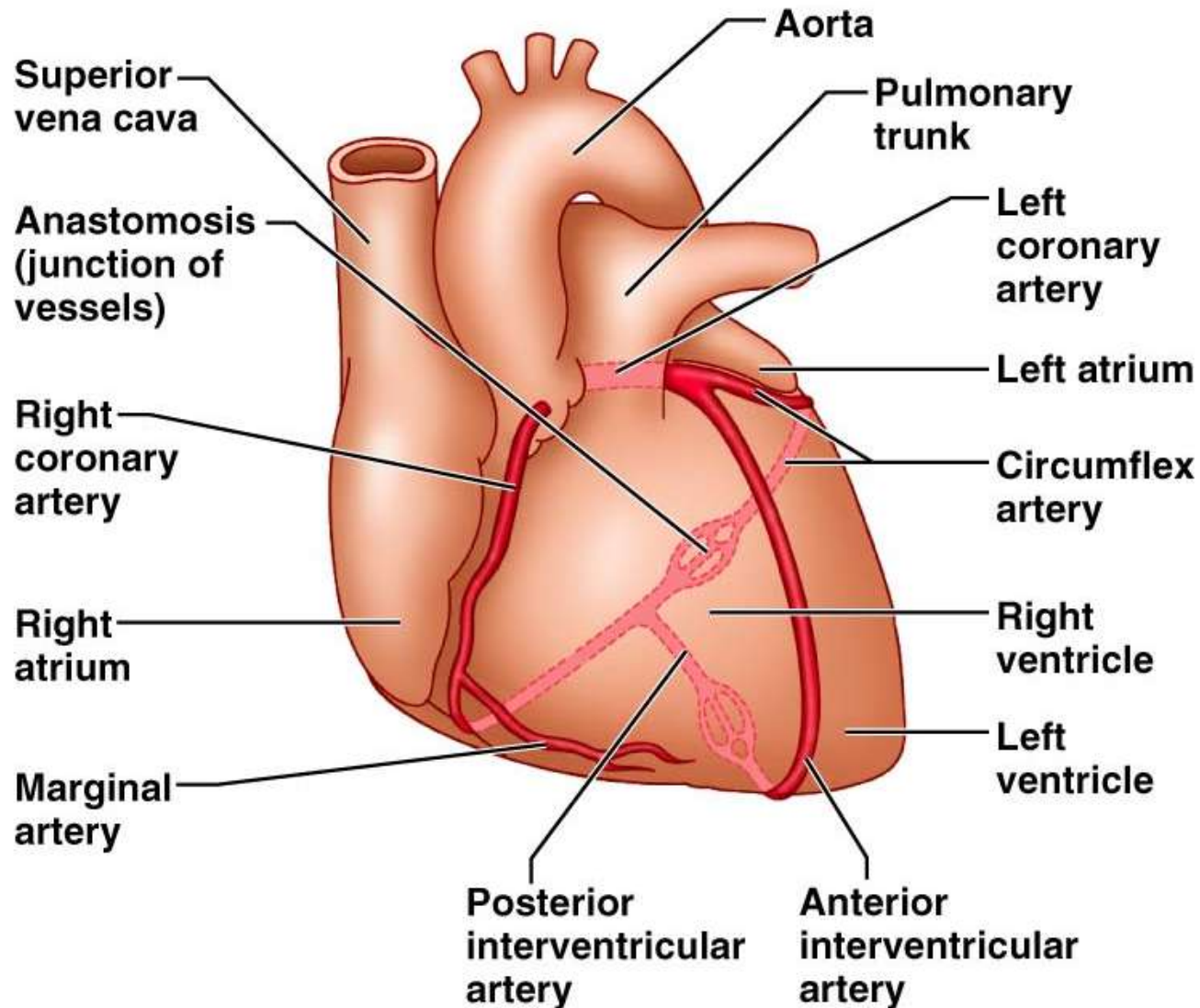
- **Pulmonary circuit** allows **deoxygenated blood** to be transported into the lungs for gas exchange, so that oxygenated blood can once again flow into the left heart
- **Deoxygenated blood** from body tissues → superior & inferior vena cava → RA → tricuspid valve → RV → pulmonary SL valve → pulmonary arteries → lungs (gas exchange occurs) → oxygenated blood travels in pulmonary veins → LA → bicuspid valve → LV.



Coronary circuit

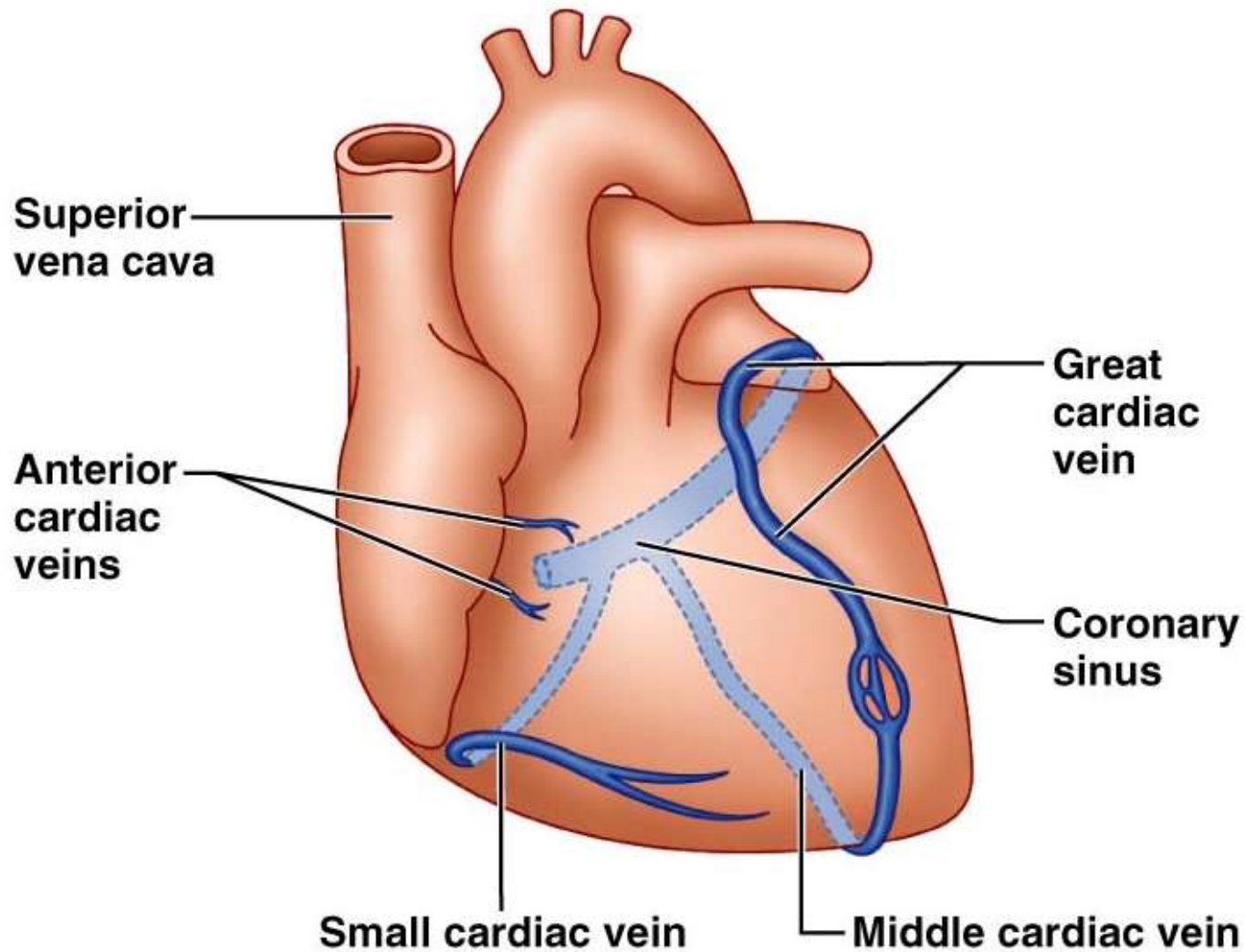
- **Coronary circuit** allows **oxygenated blood** to be delivered to cardiac muscle cells in the heart wall, and its deoxygenated blood is drained back to the RA –
- **Oxygenated blood** in LV → aortic SL valve → aorta → coronary arteries → arterioles → capillaries in myocardium (gas exchange occurs) → **deoxygenated blood** travels into venules → cardiac veins → coronary sinus → RA.

Coronary Circulation: Arterial Supply



(a)

Coronary Circulation: Venous Supply



(b)

Systemic Circuit

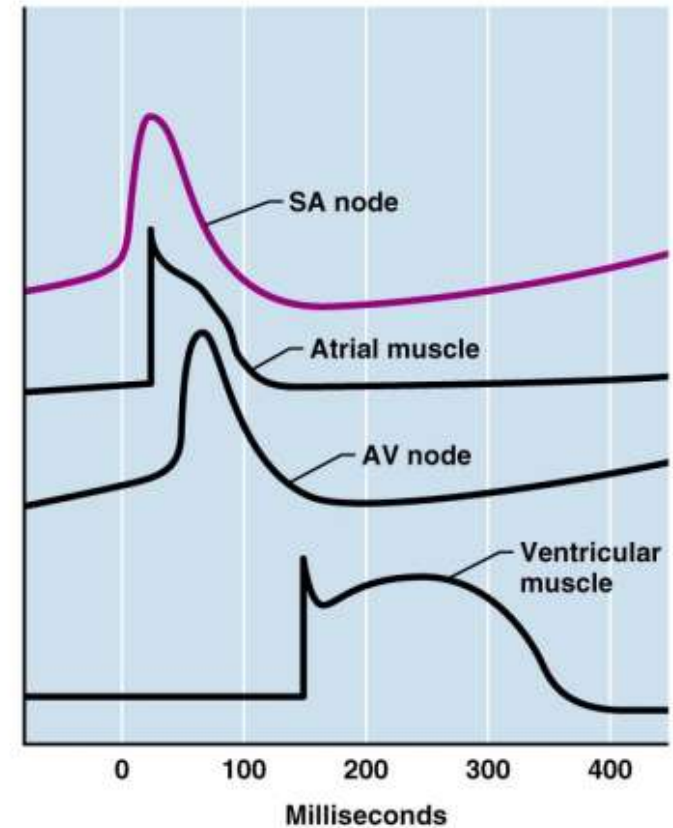
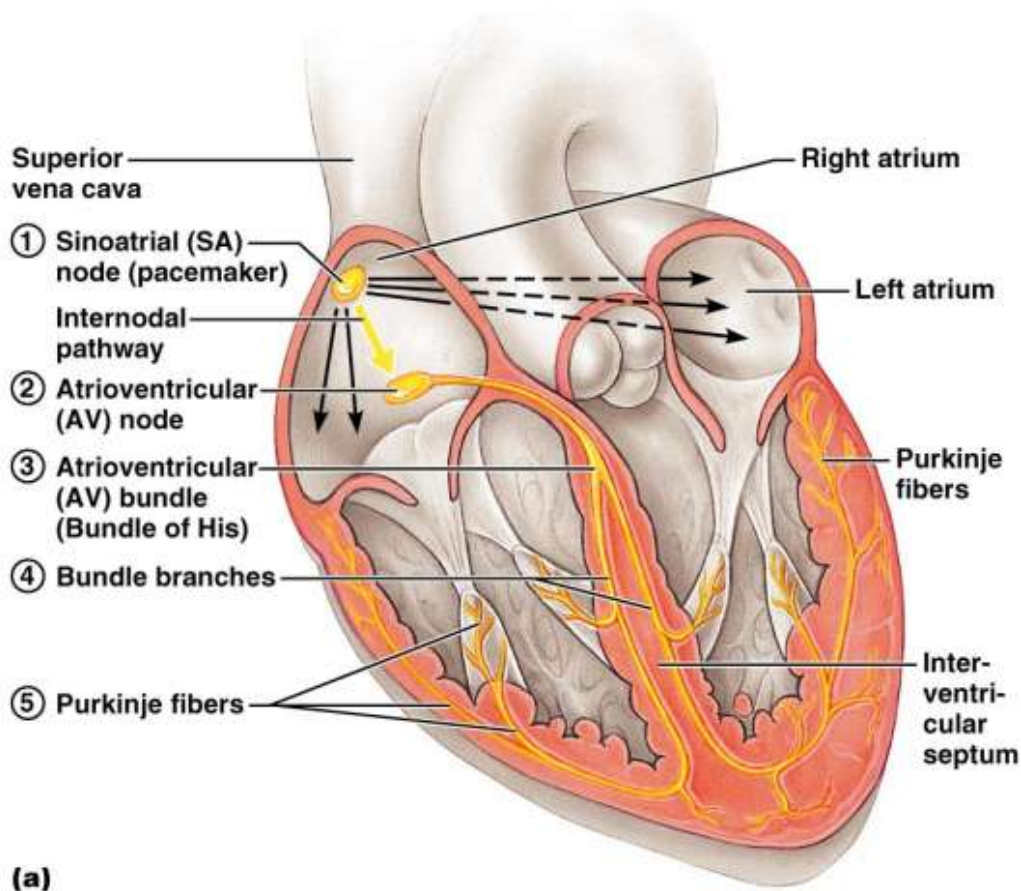
- **Systemic circuit** allows **oxygenated blood** from the left heart to be delivered to tissue cells through arteries and arterioles , and **deoxygenated blood** is transported back to the right heart through veins and venules.
- **Oxygenated blood** in LV → aortic SL valve → aorta → arteries → arterioles → capillaries in tissues (gas exchange occurs) → **deoxygenated blood** travels in venules → veins → superior & inferior vena cava → RA.

-
- Cardiac cycles are mainly controlled by nerve impulse, while hormones only can influence the heart rate.
 - Two mechanisms to regulate cardiac cycles- **intrinsic control** (consists of pacemakers and a conduction system) and **extrinsic control** (consists of sympathetic and parasympathetic nerves, and hormones, that influence the pacemakers and affect the heart rate).

Impulse conducting system of the heart (Intrinsic control)

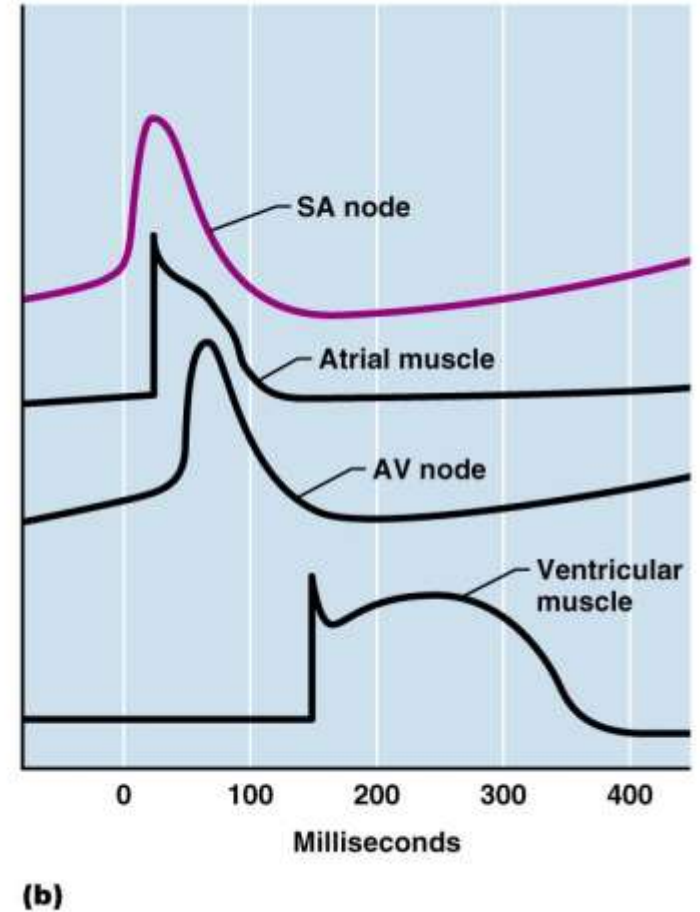
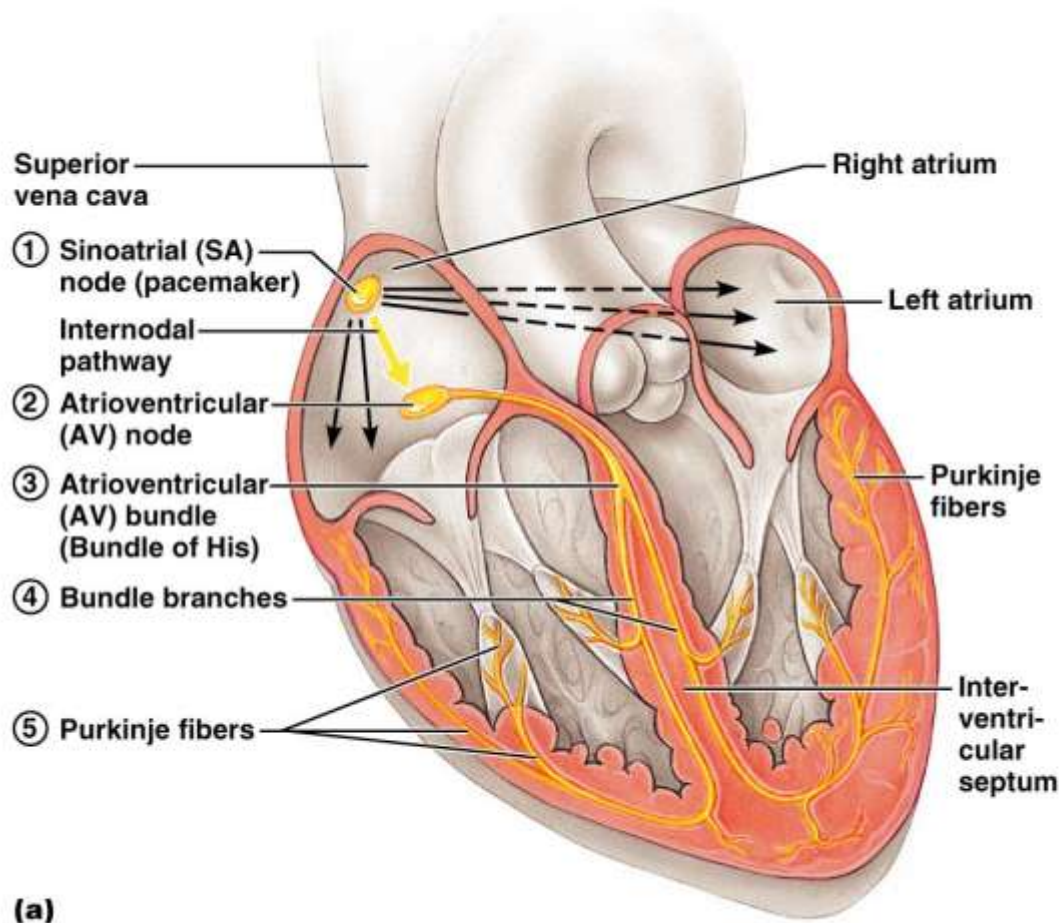
- Group of muscle cells specialized for conduction rather than contraction . Innervated by both the sympathetic and parasympathetic nervous system . Regulate cardiac rhythm and adapts cardiac output to the physiologic needs of the body .
- **Components :**
- 1.Sinoatrial node (pace maker) , cluster of excitable cells which sets cardiac rhythm . Located near the inlet of the superior vena cava .
- 2. Atrial syncytium , a mass of merging cells that act as a unit . Cardiac muscle fibers function like those of skeletal muscles , but the fibers connect in branching networks . Stimulation of any part of the network send impulses throughout the heart , which contracts as a unit .

Impulse conduction



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-
- 3. Action potential spreads through atrial musculature .
 - 4. Junctional fibers – impulses travel from cell to cell by gap junctions .
 - 5. Atrioventricular node – group of excitable fibers located to the right of the upper portion of the interventricular septum , picks up the wave of depolarization of atria and relays it to :
 - 6. Bundle of His – nerve like group of excitable tissue which spreads action potential down the IV septum and soon divides into a left or right branch which run down the septum and on the apex split into :
 - 7. Purkinje fibers which carry the action potential concentrically to the ventricular musculature , running subendocardially .
 - ventricular syncytium .



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Functions of the cardiac conduction system

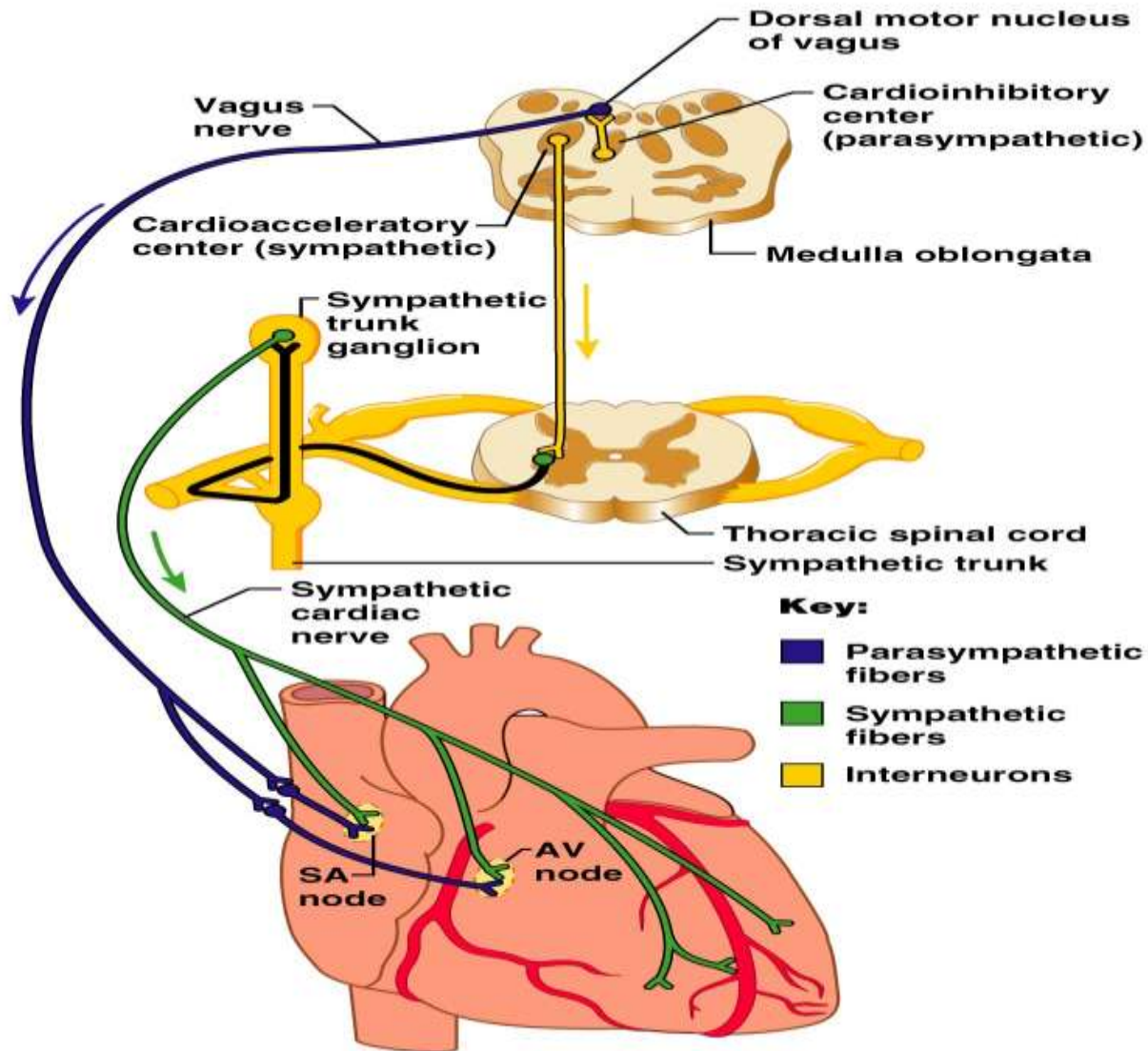
- 1. Causes both atria to contract simultaneously and generate enough hydrostatic pressure to open both AV valves so that blood flows to ventricles .
- 2. Causes the ventricles to contract a few milliseconds later , overcome the resistance of the semilunar valves and generate enough hydrostatic pressure to circulate the blood through the pulmonary and systemic pathways .
- 3. Adapts the heart to beat under different functional demands. Sympathetic nervous system increases rate and output , parasympathetic decreases .

Extrinsic Control

- In extrinsic control, **pressure receptors** along the aorta and common carotid arteries detect changes in blood pressure and send nerve impulses to the **cardiac centers** in **medulla oblongata**, which in turn activates either **sympathetic nerves** (to increase heart rate and contractility) or **parasympathetic nerves** (to decrease heart rate and contractility). These nerves innervate the **SA** node, changing the basic rhythm in cardiac action.
- **Hormones** such as **epinephrine** and **norepinephrine** can also have the same stimulatory effect on the **SA** node.

Note: Emotion, stress, body temperature, ions (potassium, calcium), certain drugs, and alcohol can indirectly affect the intrinsic and extrinsic mechanisms.

Extrinsic Control



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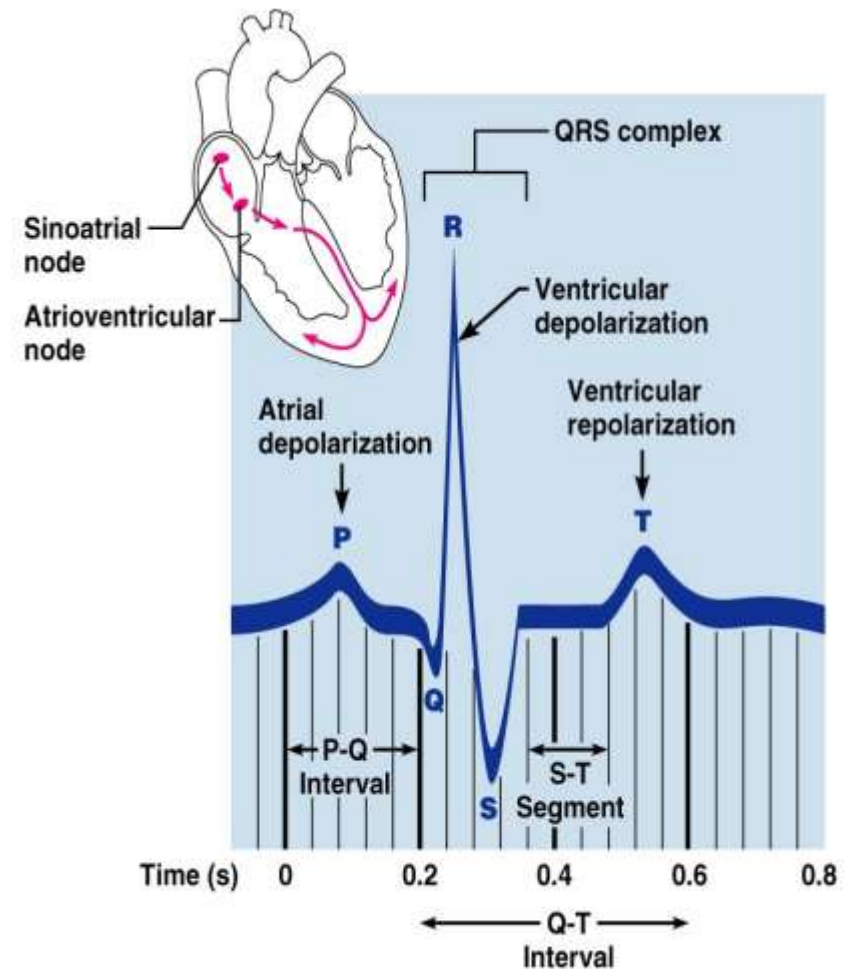
Cardiac Excitability & Contractility

- 1. The duration of the cardiac action potential is very long , lasting throughout contraction . Action potentials in cardiac muscle is 100 times longer than in skeletal muscles .
- 2. There are prolonged refractory periods .
- 3. Cardiac muscle contractions are always brief twitches . In skeletal muscles, contractions resulting from rapid repetitive stimulation can summate to provide sustained contraction. This can not happen in cardiac muscle because the long refractory period cancels any stimulus that occurs before the heart has a chance to relax .
- Relaxation between beats is essential for the heart to fill with blood , to be pumped at the next beat . Summation of contractions (tetanus) does not occur due to long refractory periods .
- Note Ca^{++} entry prolongs the period of depolarization .

-
- 4. Cardiac muscles are interconnected by gap junctions – the heart beat is All – or – None , and spreads .
 - 5. Cardiac muscles excites itself . Normally skeletal muscles will contract only if it receives a nerve impulse . Nerves that carry impulses to the heart influence the rate and strength of contraction , but they do not initiate the primitive heartbeat .
 - 6. Excitation for the heart beat arises from within the cardiac muscle itself ; **spontaneous depolarization** of the SA node cell which spread to other cells. Syncytium refers to cells that contract as a unit .
 - SA and AV node cells are pacemaker cells ; they have **intrinsic automaticity** characterized by spontaneous depolarization which perhaps may be due to a decreased membrane permeability to K^+ .

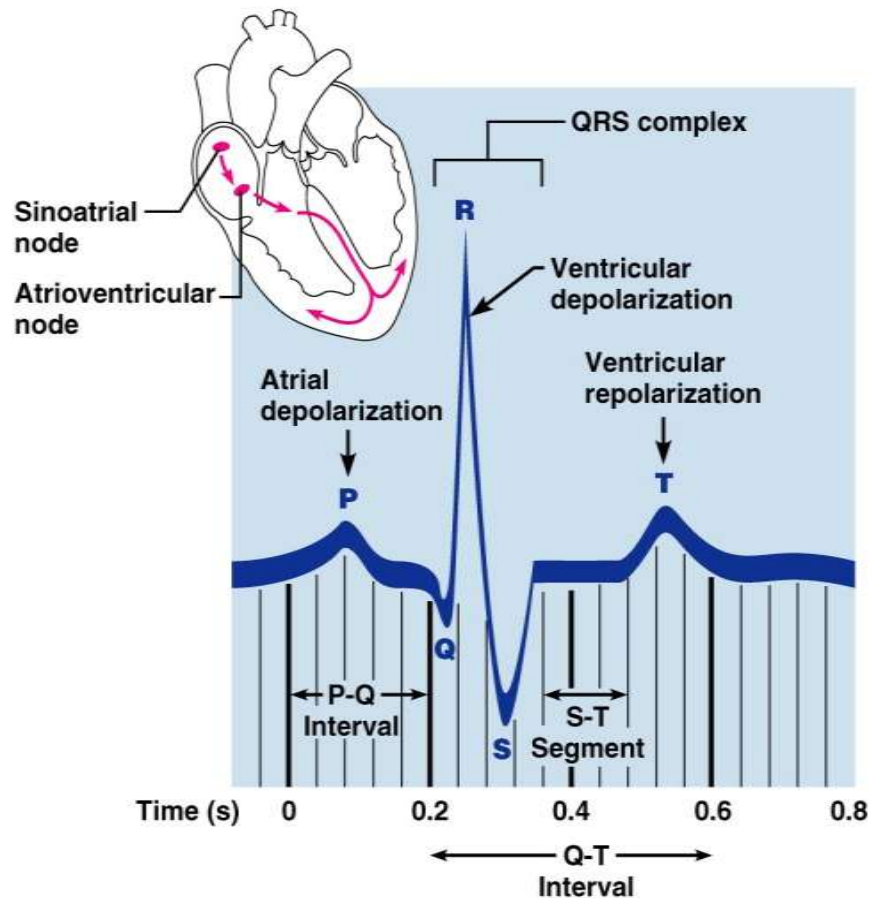
Electrocardiogram (EKG or ECG)

- A graphic record of the electrical activity of the heart.
- **P – wave** : small up ward wave representing atria depolarization – Atrial systole occurs by the end of P wave so, the P wave is atrial excitation .
- **P – Q interval** : Represents the conduction through the atrial musculature , and AV delay (due to passage of action potential through the A – V junction) .



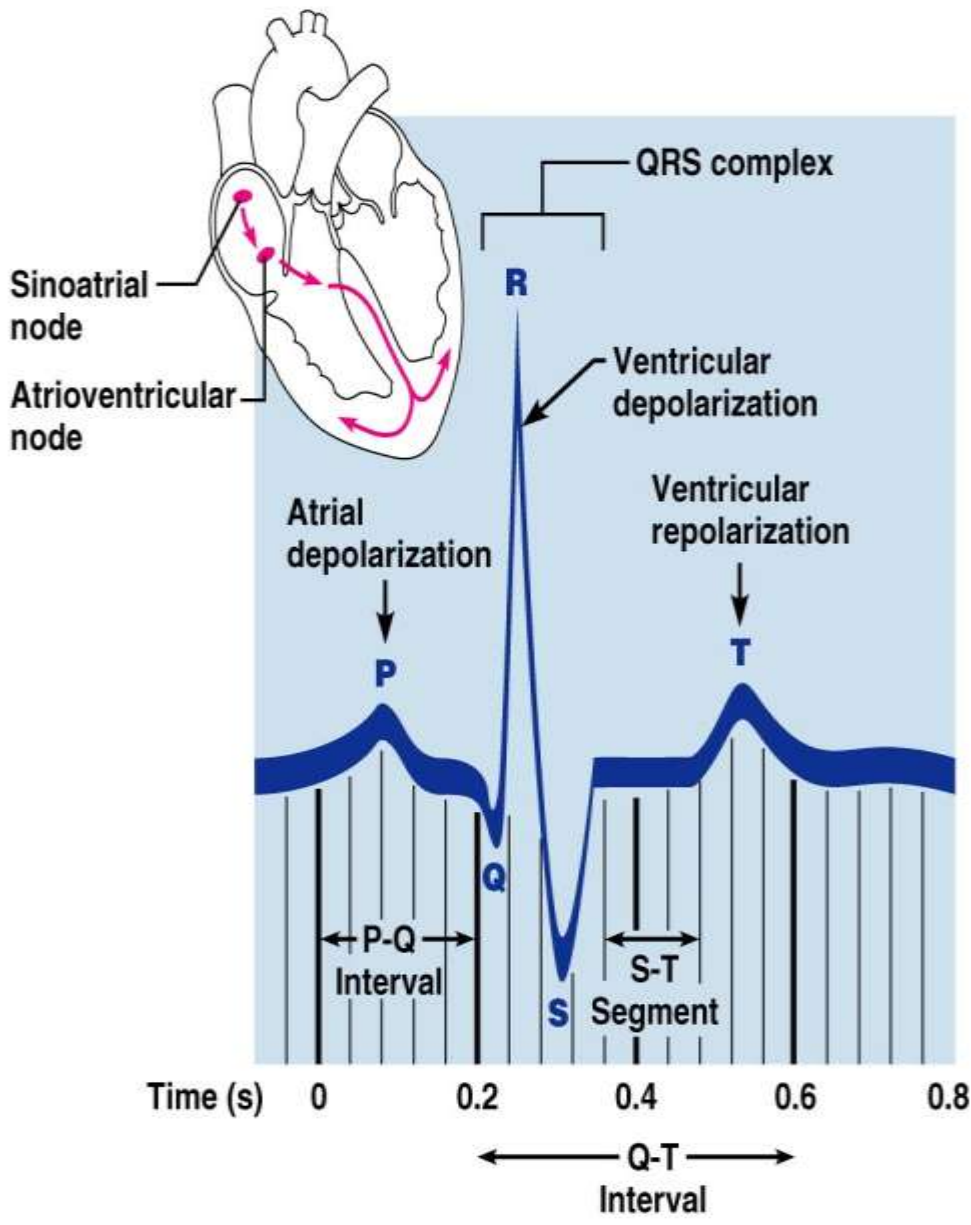
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- **Q wave** : Action potential spreads through the muscle of the septum between the ventricles , mostly from left to right and produces the small , variable , and usually negative wave , the Q wave .
- **R wave** : Action potential spreads through the big mass of ventricular muscle at the apex producing a large positive deflection , called the R wave .



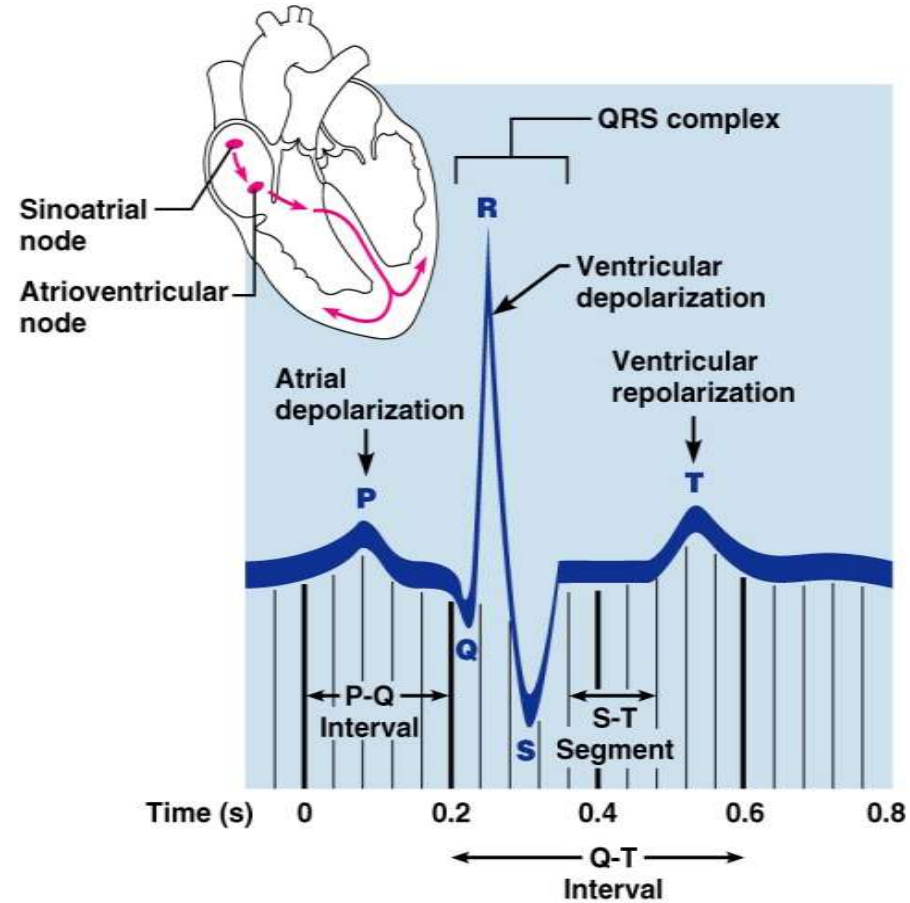
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- **S wave** : Action potential continues to spread through the last part of the bases of the ventricles , producing the usually inverted S wave (front to back Action potential)
- **QRS complex** : indicates ventricular depolarization and coincides with atrial repolarization .
- **S – T segment** : indicate the time between ventricular depolarization and beginning of repolarization .



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- Note: S – T segment is elevated in acute myocardial infarctions . During S-T interval , all of ventricle stays "excited".
- **Q – T interval** : shows events in ventricular activity from beginning of depolarization until end of repolarization .
- **T wave** : Indicates the ventricular repolarization (recovery of the ventricles to the resting state).



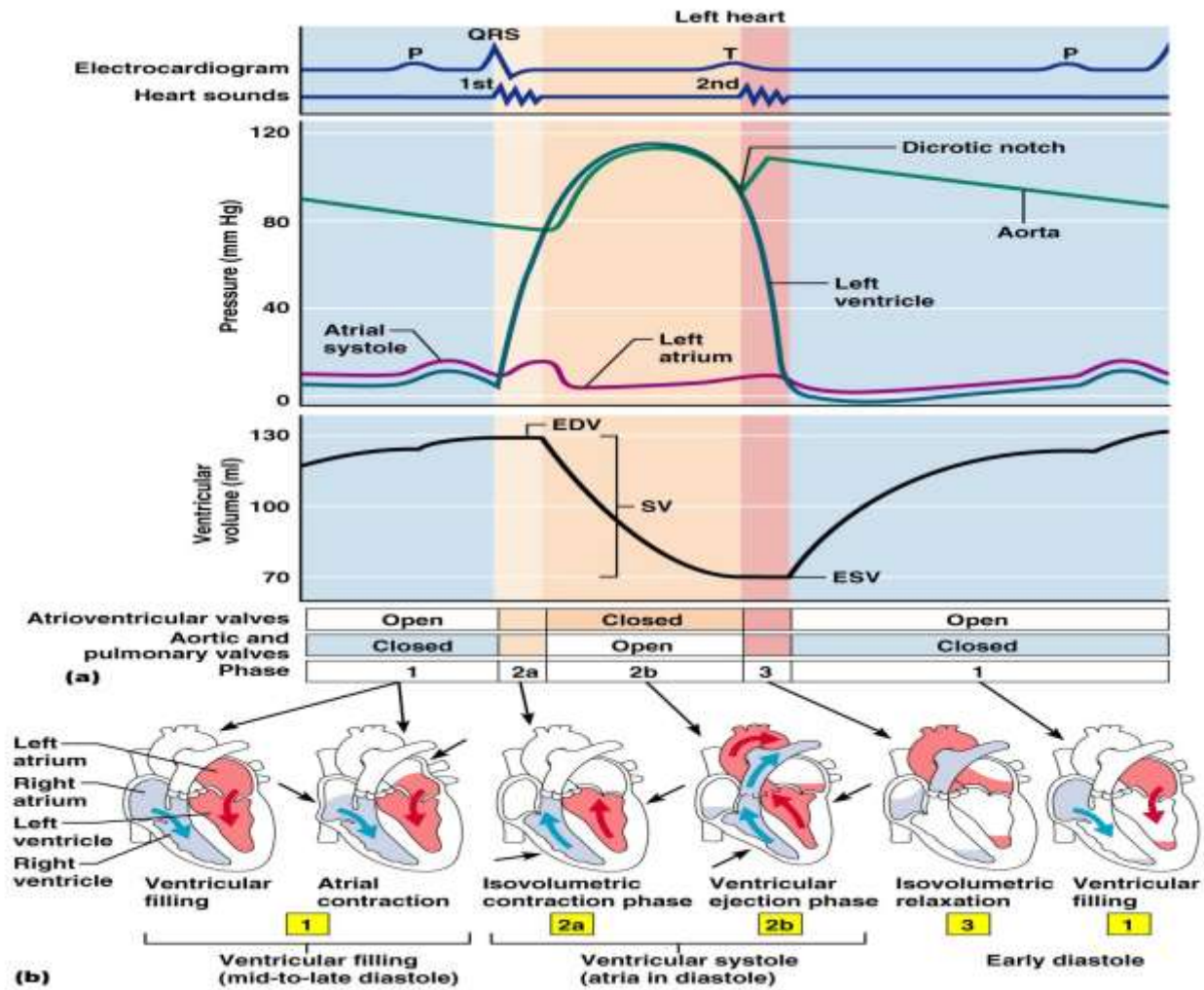
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Cardiodynamics

- 1. A **cardiac cycle** (or a heartbeat) consists of **3** phases of events.
- **Relaxation period** – when all 4 heart chambers are relaxed, all 4 heart valves are closed, and blood is coming into the 2 atria through the veins.
- **Ventricular filling**- when 70% of the blood in atria flow passively into ventricles due to gravity, followed by 30% of the blood being pumped by **atrial systole** (contraction of atria) ; during this phase, **AV** valves are open while **SL** valves are still closed.
- **Ventricular systole**- after all the blood in atria gets into ventricles, **AV** valves close, atria go into relaxation (**atrial diastoles**), and ventricles contract to pump the blood into exiting arteries (**LV** into aorta and **RV** into pulmonary trunk) which opens the **SL** valves.

-
- **SL** valves will close after almost all ventricular blood is ejected (there is always 60 ml blood remained in each ventricle, a volume called **end-systolic volume** or **ESV**). Then the ventricles relax (**ventricular diastole**), and a new cardiac cycle will follow.
 - 2. Each cardiac cycle takes about **0.8** second to complete.
 - 3. Toward the end of ventricular filling, when pressure builds up in the ventricles **AV** valves begin to close. Now papillary muscles contract, pulling the chordae and cusps, to prevent eversion (over- bulging) of the cusps into the atria.
 - 4. In each cardiac cycle, the two atria contract and relax simultaneously- a phenomenon called **atrial syncytium**. The same is true for the ventricles, which is known as **ventricular syncytium**. [when cardiac muscles in heart chambers lose this syncytium ability, fibrillation occurs which can be fatally out of control].

Cardiac cycle



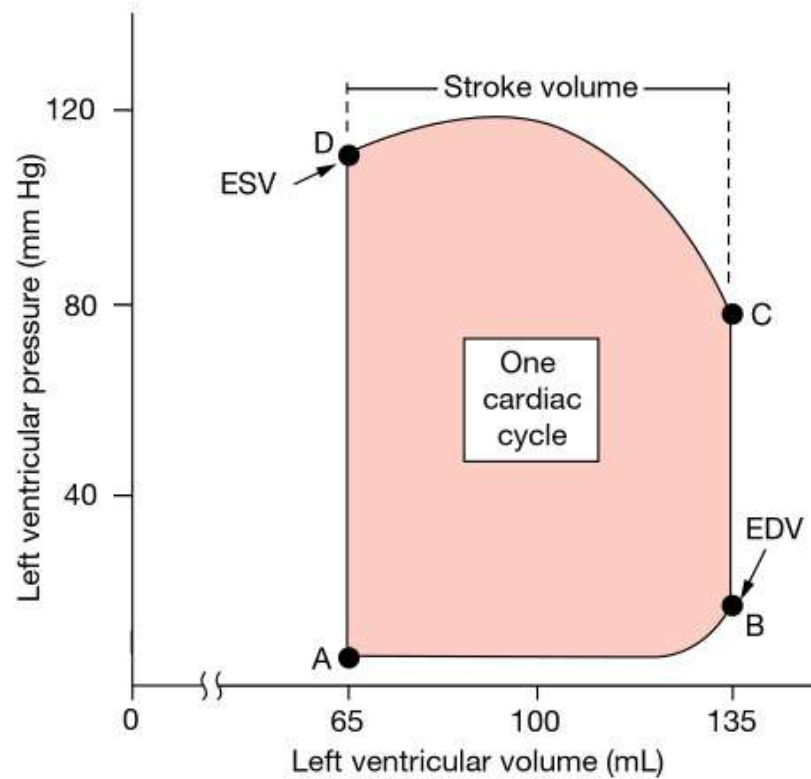
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- **Cardiac output** : volume of blood pumped by the heart each minute = stroke volume (volume of each beat) \times Heart rate (C.H.R) [# beats / min]
 - Stroke volume : determined by the volume of blood in the Heart at the beginning of systole (**end diastolic volume**) minus the amount of blood remaining in the ventricles when the valves close at the end of systole (**end systolic volume**) .
 - $SV \text{ (ml / beat)} = EDV \text{ (120 ml)} - ESV \text{ (50 ml)} = 70 \text{ ml / beat}$
 - Note : End diastolic volume is the amount of blood that collects in a ventricle during diastole.
 - End systolic volume is the amount of blood remaining in the ventricle after it has contracted.
 - End systolic volume is the amount of blood remaining in the ventricle after it has contracted.

Left ventricular pressure-volume change

KEY

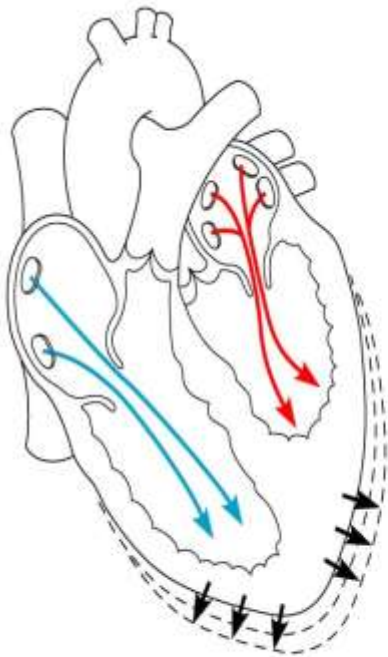
EDV = End-diastolic volume
ESV = End-systolic volume



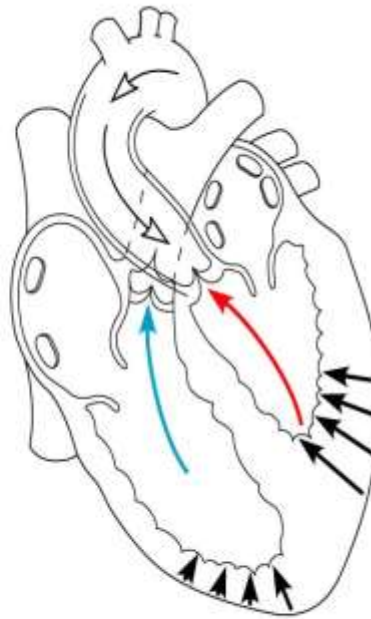
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Degree of stretch of Heart muscle:

Frank – Starling law: stretching muscle fibers, increase their length and produces increased contractile force of the heart. The contractile force is the preload (degree of stretch of cardiac muscle cells just before they contract).

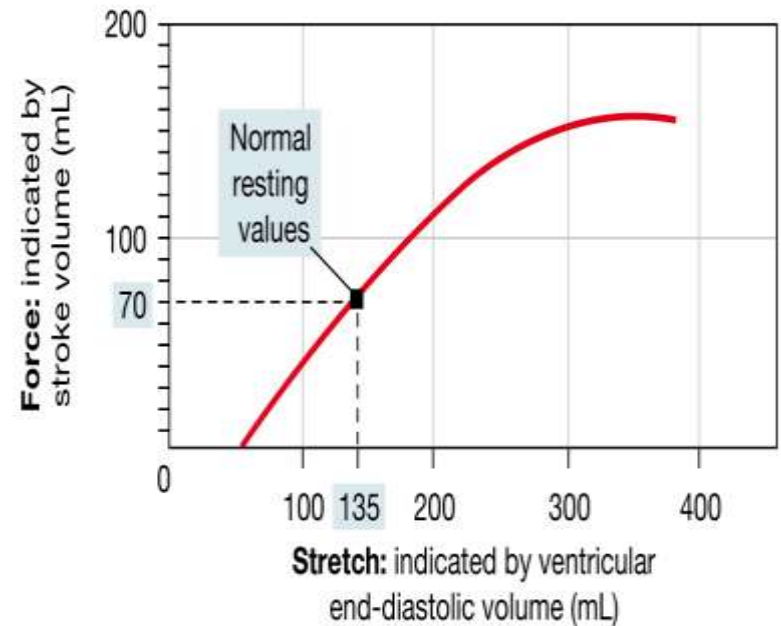


(a) Preload



(b) Afterload

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Auscultation

- The process of studying heart sounds using a stethoscope.
- Each cardiac cycle generates 4 heart sounds, usually only the first two sounds are audible.
- **S1 (or "lubb) sound** is the **loudest**, caused by the **closure of AV valves** at the beginning of ventricular systole; it matches the R wave in ECG.
- **S2 (or " dupp") sound** is **second loudest**, caused by the **closure of SL valves** at beginning of ventricular diastole; it matches the T wave in ECG.
- **S3 sound** is **noises** generated by **ventricular filling** in the first 1/3 a trial blood flow into ventricles.
- **S4 sound** is **noises** generated by **atrial systole** in the remaining 30% of atrial blood being pumped into ventricles

Sinus rhythm

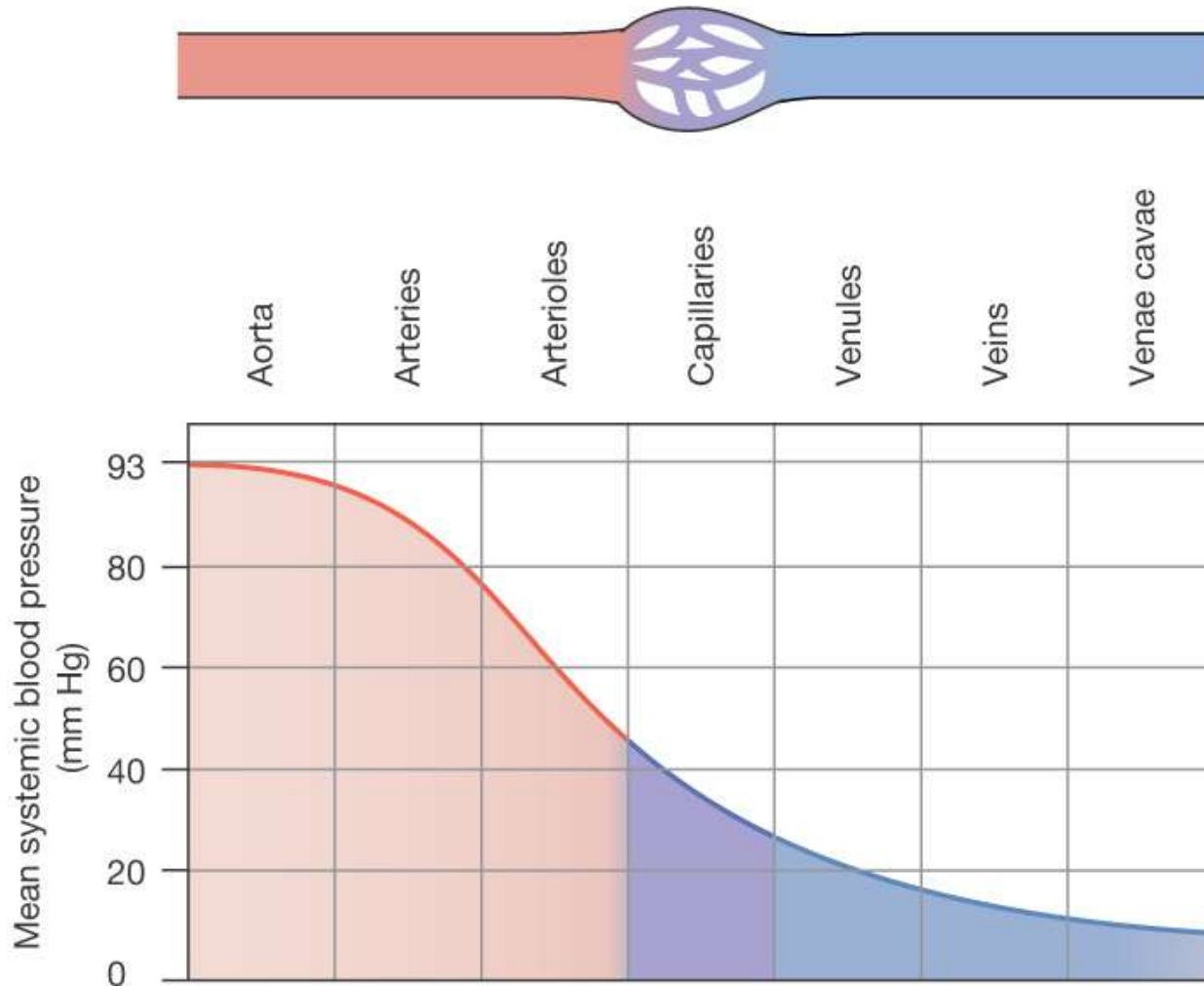
- Normal cardiac action or heart rate for female is about 72-80 beats/min, for male, about 64-72 beats/ min., and for fetus, about 140 - 160 beats/min.
- **Arrhythmias** are any abnormal hear beat.
- **Bradycardia** is slow heart rate (<60 beats/min) which is normal during sleep, but can be induced by low body temperature, parasympathetic stimulation, and certain drugs. It is indicated by a Short PQ interval and a flat T wave on the ECG.
- **Tachycardia** is fast heart rate (> 100 beats/min) which is normal during exercising or excitement, but can be induced by high body temperature, sympathetic stimulation, drugs, heart diseases, anemia , or shock. It is indicated by a lack of P,Q, S, or T wave, with only high-frequency of upward and downward deflections on the ECG.

-
- **Flutter** is very high heart rate (>250 beats/min) which is usually pathological (e.g. bacterial infection or inflammation of myocardium). It is indicated by many small, unrecognized waves, then a big upward/downward wave on the ECG.
 - **Fibrillation** is high but uncoordinated heart rate caused by regions of myocardium contracting and relaxing independently (lack of syncytium). **Atrial fibrillation** is not very serious if ventricles are functioning normally. **Ventricular fibrillation** is usually fatal (the most common cause of sudden death) where blood cannot be pumped properly into the lungs and body tissues. It is indicated by extremely irregular waves on the ECG , and usually Q and S waves are absent.

Blood pressure (120/80 mmHg)

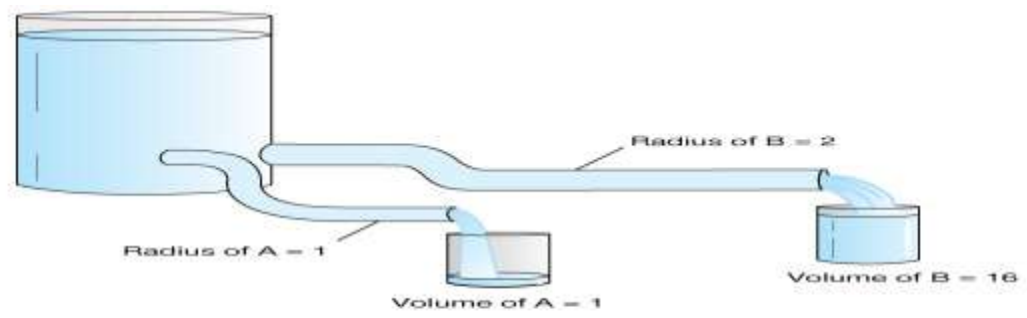
- Systolic pressure is the highest pressure attained in the aorta and peripheral arteries when blood is ejected by the ventricles into the aorta .
- Diastolic pressure is the lowest pressure attained during relaxation or diastolic phase of the heart .
- Mean arterial blood pressure is the highest in the aorta and large arteries (100 Hg) .
- It sharply decreases in small arteries and even more in arterioles (30 mmHg)
- After passage through the capillaries and in the small veins the pressure is about 15 mmHg .
- It falls to about 5mmHg upon entering the R. atrium

Pressure gradient in the blood vessels



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- Blood pressure is sensed by baroreceptors in the carotid sinuses and arch of the aorta . They send impulses to the cardiovascular centers in the brain stem which then send signals back to the peripheral vasculature and heart .
- The effector mechanism that maintains constancy of BP is dual [BP is increased by either increased CO or increased PR (peripheral resistance)] .
- Change in the diameter of arterioles , controlled y the vasomotor center of the medulla oblongata , regulate peripheral resistance . Resistance to flow is increased by decreasing blood vessel radius .



Resistance $\propto \frac{1}{\text{radius}^4}$	
Tube A	Tube B
$R \propto \frac{1}{1^4}$	$R \propto \frac{1}{2^4}$
$R \propto 1$	$R \propto \frac{1}{16}$

Flow $\propto \frac{1}{\text{resistance}}$	
Tube A	Tube B
Flow $\propto \frac{1}{1}$	Flow $\propto \frac{1}{\frac{1}{16}}$
Flow $\propto 1$	Flow $\propto 16$

Cardiac Output

Increased blood volume entering heart



Increased stretch of myocardial fibers



Greater force of myocardial contraction

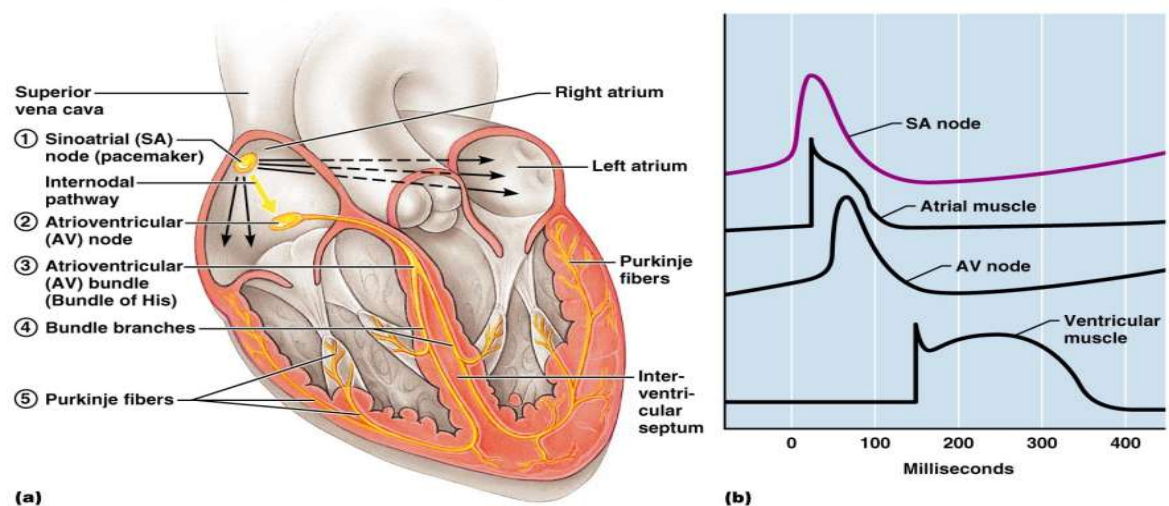
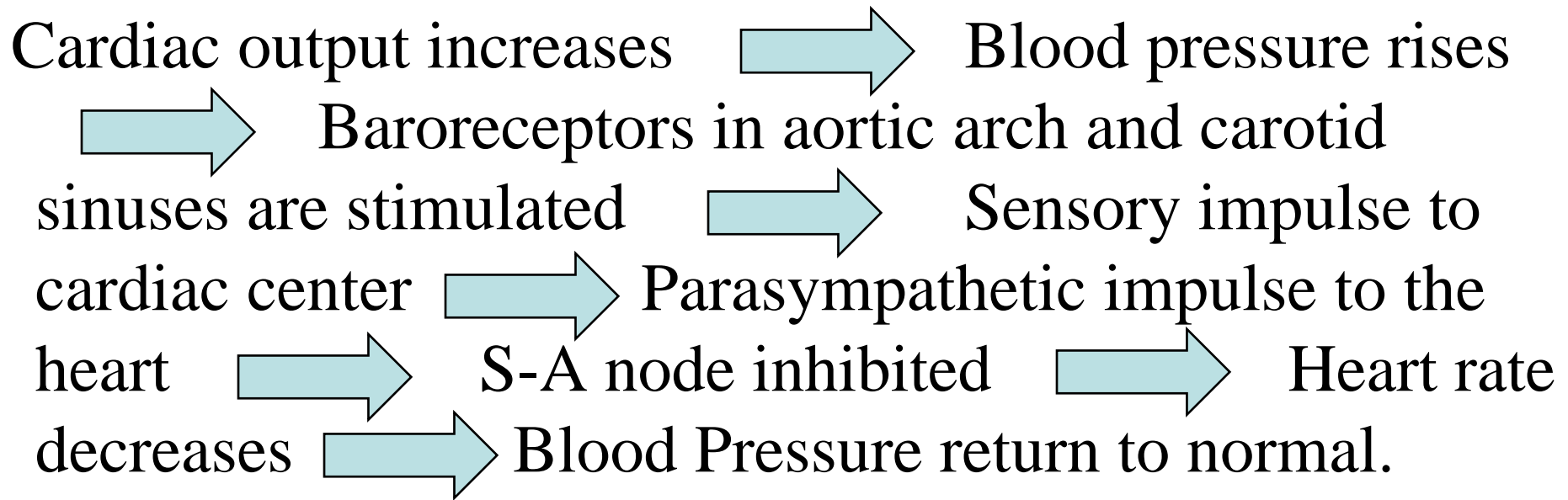


Greater stroke volume



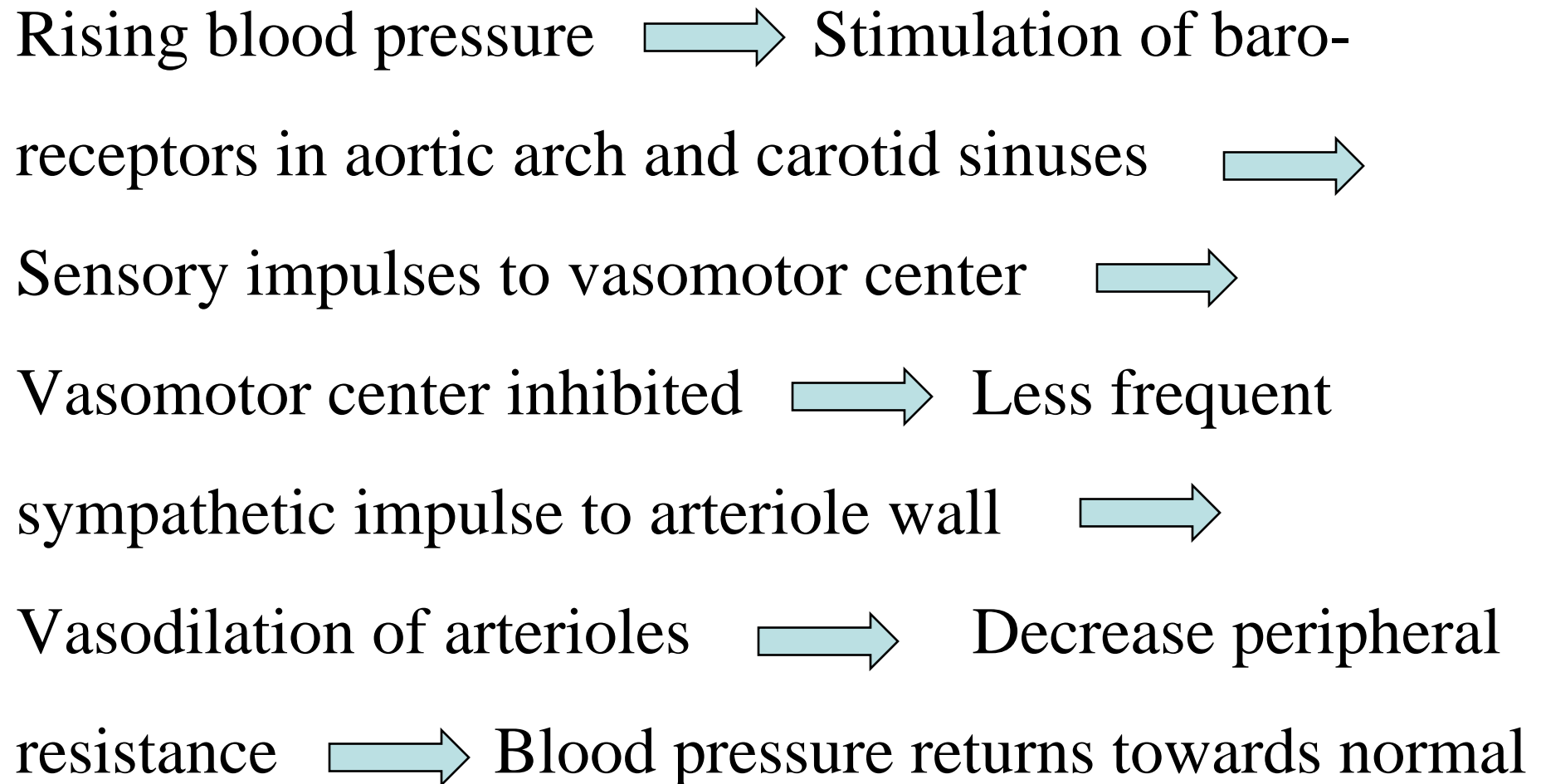
Increased cardiac output .

Mechanism regulating B.P. by inhibiting S-A node



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Mechanism regulating B.P. by dilating arterioles



Renal Control of B.P.

Reduced blood flow to kidneys \longrightarrow Kidneys
releases renin \longrightarrow Renin leads to the production
of angiotensin II \longrightarrow Angiotensin II causes vaso-
constriction \longrightarrow Blood pressure elevated \longrightarrow
Increase blood flow to the kidneys.

Risk factors for stroke

- 1. Alcohol consumption
- 2. Diabetes
- 3. Elevated serum cholesterol
- 4. Family history of cardiovascular disease
- 5. Hypertension
- 6. Smoking
- 7. Transient ischemic attacks

Drugs to treat hypertension

- Angiotensin – converting enzyme inhibitors :
Block formation of Angiotensin II , preventing vasoconstriction .
- Beta blockers : lower heart rate .
- Ca⁺⁺ channel blockers : Dilate blood vessels by keeping ca ions out of muscle cells in vessel walls .
- Diuretics : increase urine output , lowering blood volume .

Atheroscleroses

- Atherosclerosis of arteries can **occlude blood flow** to the heart and brain and is a causative factor in up to 50% of all deaths in U.S.A., Europe, and Japan .
- A . Atherosclerosis begins with injury to the endothelium , the movement of monocytes and lymphocytes into the tunica interna and the conversion of monocytes into macrophages that engulf lipids . Smooth muscle cells then proliferate and secrete extra cellular matrix .
- B . Atherosclerosis is promoted by such risk factors as smoking , hypertension , and high plasma cholesterol concentration .
- LDLs which carry cholesterol into the artery wall , are oxidized by the endothelium and are a major contributor to atherosclerosis .

- occlusion of blood flow in the coronary arteries by atherosclerosis may produce ischemia of the heart muscle and angina pectoris , which may lead to myocardial infarction .
- The EKG can be used to detect abnormal cardiac rates , abnormal conduction between the atria and ventricles , and other abnormal patterns of electrical conduction in the heart .

