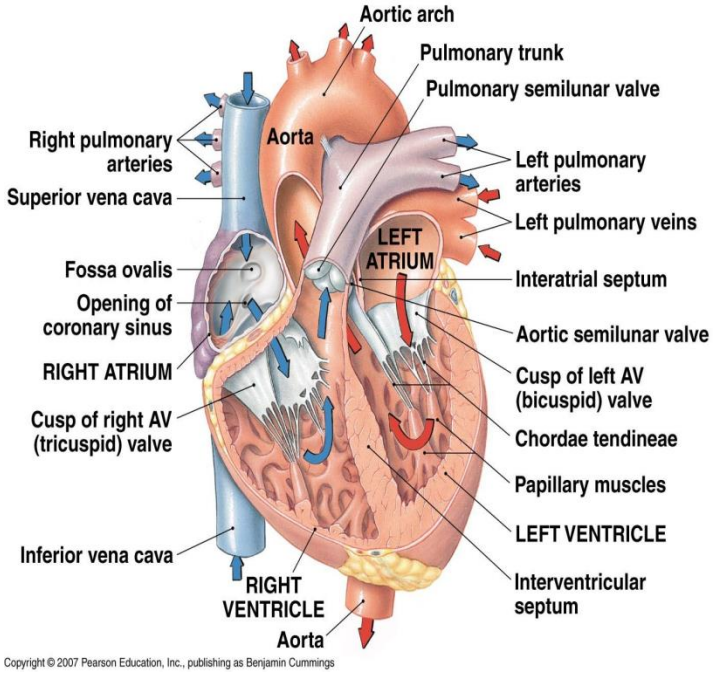
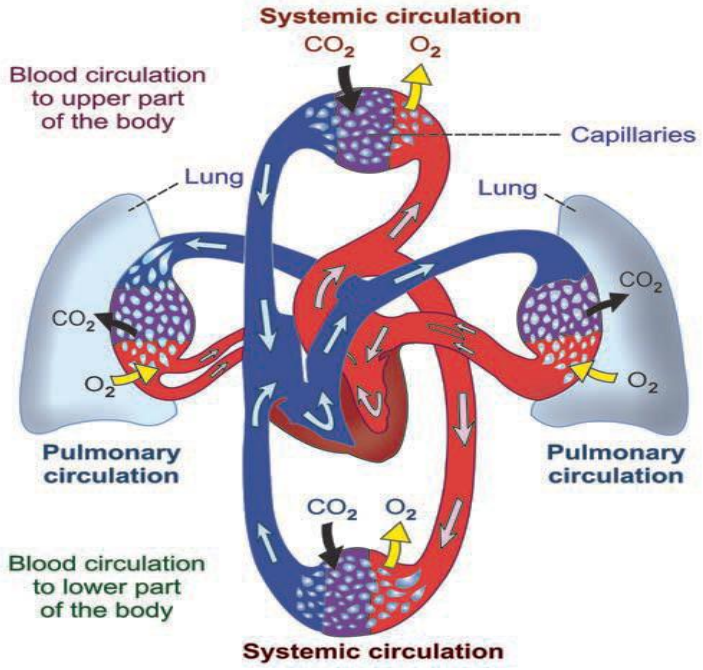
Human Cardiovascular System

Lecture (12) Cardiac Conductivity and Contactility

- The cardiovascular is a **closed transport system** inside which blood circulates **in one direction** by **the action of the valves present in heart and most of veins**.

- The **cardiovascular** system consists of the heart and blood vessels.
- The **heart is muscular organ**. **Each side is formed of two chambers** (**atrium & ventricle**).
- **The atria act mainly as blood reservoirs and ventricles act as a pump**.
- **The musculature of ventricles is thicker than** that of **atria.**





tricuspid valve Separate the right atrium from the right ventricle	**mitral valve (bicuspid).** Separate the left atrium from the left ventricle
pulmonary valve Separate the pulmonary artery from right ventricle	**aortic valve** Separate the aorta from the left ventricle
Right atrium	**Left atrium**
receives and **deoxygenated blood** from the venous system then sends down to the right ventricle	The left atrium receives o**xygenated blood from the lungs**.
Right ventricle	**Left Ventricle**
Receives blood **from the right atrium**; pumps blood into to the lungs through the pulmonary artery. (lesser pulmonary circulation)	pumps the blood from the left atrium out to the body, supplying all organs with oxygen-rich blood. through greater systemic circulation.

The cardiac muscle consists of:

myocardial contractile cells	myocardial conducting cells
(99%, similar to skeletal muscle but with **intercalated discs**, **gap junctions between its fibers**)	(1%) that is responsible for **initiation (SA node)** and **propagation of action potential to the contractile fibers (AV node, Bundle of His, Right and left bundle branches and Purkinje fibers).**
can't initiate their action potential	contain more sarcoplasm and very little myofibrils.

Physiological properties of the cardiac muscle:

1. **Excitability.**
2. **Rhythmicity.**
3. **Contractility.**
4. **Conductivity.**

1. Excitability

It is the ability of cardiac muscle to respond to an adequate stimulus.

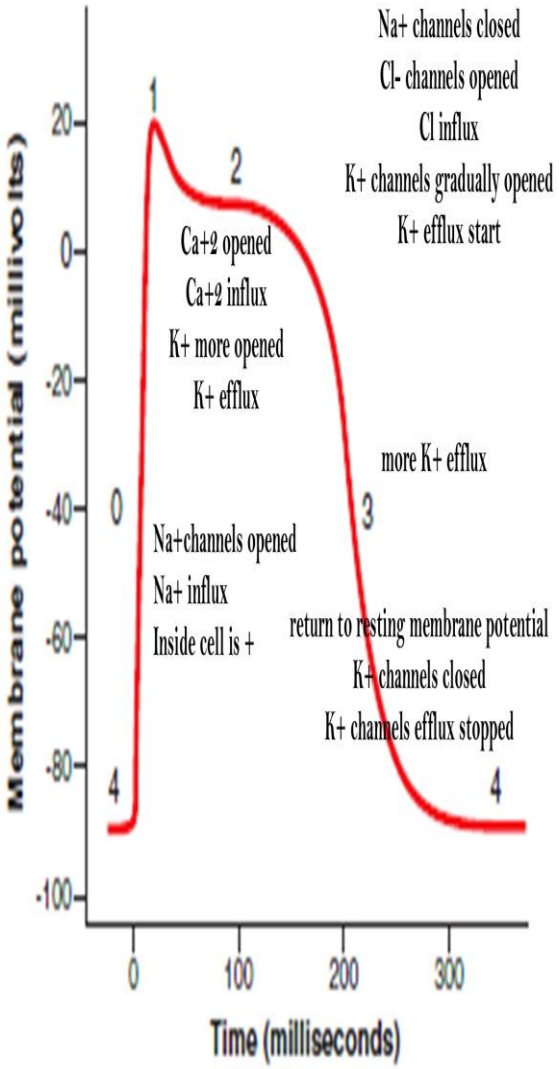
Phase 0 (Rapid depolarization up to +20 mv): This phase is due to the opening of the fast Na+ channels causing a rapid increase in the membrane conductance to Na+.

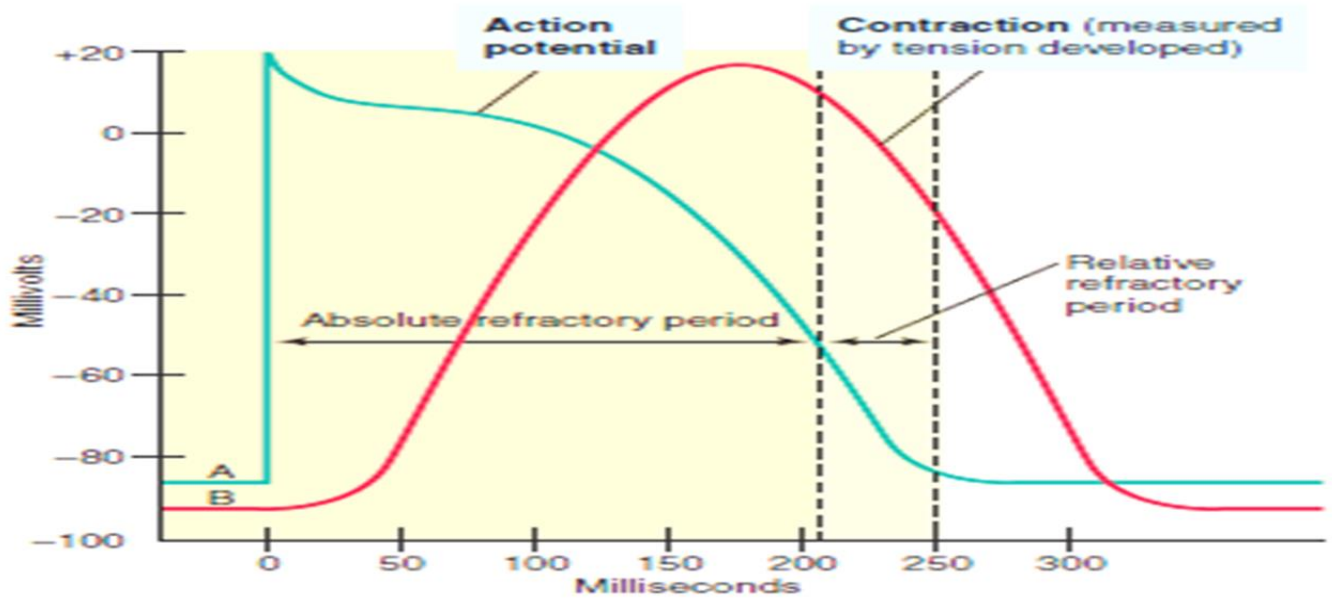
Phase 1 (partial repolarization): Phase 1 of the myocyte action potential occurs with the inactivation of the fast Na+ channels with influx of Cl^- and gradually increasing K^+ efflux.

Phase 2 (plateau phase): This "plateau" phase of the cardiac action potential is due to prolonged increased permeability of cell membrane to Ca^{2+} caused by opening of L-type slow Ca^{2+} channels. It is sustained by a balance between inward movement of Ca^{2+} and outward movement of K^+. The plateau phase increases the action potential time (5-15 times as skeletal muscle) ⟶ long absolute refractory period.

Phase 3 (Rapid repolarization): Due to delayed increased permeability of cell membrane to K^+ ⟶ more K^+ efflux.

Phase 4 (RMP): It is the resting membrane potential (RMP), is about -90 mV.





Absolute refractory period	Relative refractory period	Supernormal phase
✓ The period during which the cardiac muscle cannot respond to any stimulus whatever strong ✓ Occupying the whole systole and early part of diastole (to allow heart filling). **Phase 0 + Phase 1 + Phase 2**	✓ The period during which the cardiac muscle needs stronger stimulus than normal to produce a response called **extrasystole (premature beats).** ✓ Occupies the end of repolarization phase ✓ **Phase 3**	✓ the excitability is more than normal ✓ cardiac muscle can respond to sub-threshold stimulus ✓ begins at the end of relative refractory period ✓ may lead to arrhythmia up to ventricular fibrillation (fatal).
Significance of long absolute refractory period (ARP): 1. Prevention of tetanic contraction of the heart as it is fatal (stop heart pump). 2. Provide rest period for the cardiac muscle so prevent fatigue.	**Extrasystole:** Abnormal systole that are produced by impulses discharged from a hyper-excitable ectopic focus (a focus other than normal pacemaker). Such foci may develop normally as a result of excessive smoking or pathologically due to myocardial ischemia.	

The systole (heart contraction)	diastole (heart relaxation)
reaches its maximum at the end of plateau phase	starts with rapid repolarization which is completed at about the middle of diastole

CONDUCTIVITY

Ability to conduct the excitation wave through the heart. Human heart has a specialized conductive system, through which impulses from SA node are transmitted to all other parts of the heart.

CONDUCTIVE SYSTEM IN HUMAN HEART:

Conductive system of the heart is formed by the modified cardiac muscle fibers. These fibers are the specialized cells, which conduct the impulses rapidly from SA node to the ventricles.

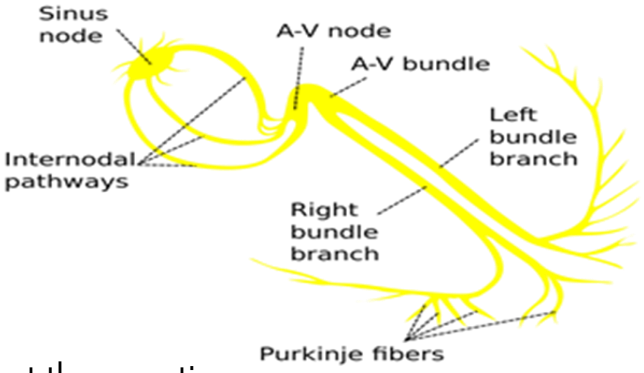
Components of Conductive System in Human Heart

1. AV node.

2. Bundle of His.

3. Right and left bundle branches.

4. Purkinje fibers to each ventricular fiber to contract all at the same time.



❑ Velocity of conduction:

- In A-V node = 0.2 meter/second (delayed conduction).
- In bundle branches and Purkinje fibers = 4 meter/second (rapid conduction).
- In AV bundle, ventricular and atrial muscles = one meter/second.

Thus, the velocity of impulses is maximum in Purkinje fibers (more gap junctions) and minimum at AV node (AV nodal delay, few gap junctions).

✓ Significance of AV nodal delay:

1- Give enough time for blood to pass from atria to ventricles. If conduction at AV node is rapid, atria and ventricles contract at the same time.

2- Protects the ventricles against the pathologically high atrial rhythms as atrial flutter and atrial fibrillation.

Rhythmicity

It is the ability of the cardiac muscle to beat (contract and relax) regularly.

Rhythmicity is myogenic (started from the cardiac muscle).

Rhythmicity of different parts of the heart:

The sino-atrial (S-A) node rhythm (Sinus rhythm)	The atrio-ventricular (AV) node rhythm	Purkinje fibers rhythm
100-110 discharges per minute	60 discharges per minute	25-40 discharge/minute
and it acts as the **pacemaker of the heart**	secondary pacemaker and acts only if the S-A node is damaged	tertiary pacemaker
The highest rhythmicity as it has rapid recovery from action potential than other cardiac muscles.	It is the only electrical pathway from atrium to ventricle (main function of AV node).	acts only if the impulse conduction via A-V node is completely blocked and damage AV bundle

❑ Factors affecting rhythmicity:

Nervous factors	Physical factors	Chemical factors
♘ Sympathetic⟶↑ rhythmicity of the whole heart ♘ ParaSympathetic⟶↓ rhythmicity of the whole heart	♘ Moderate warming ⟶↑ rhythmicity ♘ Moderate cooling ⟶↓ rhythmicity ♘ Excessive cooling ⟶Stop rhythmicity	♘ Adrenaline, thyroxine⟶↑ rhythmicity ♘ Acetylcholine ⟶↓ rhythmicity ♘ Moderate acidity ⟶↓ rhythmicity ♘ Moderate alkalinity ⟶↑ rhythmicity
	secondary to changes in the metabolic activity of the pacemaker cells in the SAN	♘ Excessive Acidity or Alkalinity ⟶ Stop rhythmicity

➢ Arrhythmias:

These are abnormalities in the number or regularity of heart beating due to:

1. Disturbances in impulse formation: Due to either: -

- Alteration of SA node activity (sinus tachycardia or bradycardia).

- Presence of ectopic foci:

Atrial extrasystole	Ventricular extrasystole	Paroxysmal atrial tachycardia (Attack)	Paroxysmal ventricular tachycardia (Attack)
Place: Atrium **Cause:** Ectopic focus **Beats:** only one more **SA node:** work **Pcaemaker:** SA node	**Place:** Ventricle **Cause:** Ectopic focus **Beats:** only one more **SA node:** work **Pcaemaker** SA node	**Place:** Atrium **Cause:** Ectopic focus **Beats:** 150-220/min **SA node:** Suppressed **Pcaemaker** Ectopic focus	**Place:** Ventricle **Cause:** Ectopic focus **Beats:** 150-220/min **SA node:** work **Pcaemaker:** SA in atrium. focus in ventricle
Atrial flutter	**Atrial fibrillation**	**Ventricular fibrillation**	
Place: Atrium **Cause:** Ectopic focus **Beats:** 250-350/min **SA node:** work **Pcaemaker:** SA node **AV node:** conduct 180-230 beat/min	**Place:** Atrium **Cause:** Ectopic focus **Beats:** 350-500/min **SA node:** work **Pcaemaker** SA node **AV node:** conduct 180-230 beat/min	**Place:** ventricle **Cause:** multiple ectopic foci **Beats:** irregular **SA node:** work No pump Death	

2. <u>Disturbances in impulse conduction:</u> heart block.

Heart block: It means failure of conduction of impulses from S-A node down to the ventricles. It

CONTRACTILITY

Ability of cardiac muscle to contract and push blood into the circulation.

➢ **Myocardial excitation contraction coupling:**

- As in skeletal muscle, the depolarization wave causes release of Ca^{+2} ions into the muscle sarcoplasm from the sarcoplasmic reticulum. This is called activator Ca^{+2}.

- Relaxation of cardiac muscle occurs as a result of release of actin-myosin combination by decreasing the intracellular Ca^{+2} to its pre-contraction level by: active reuptake of Ca^{+2} to sarcoplasmic reticulum and active pumping of excess Ca^{+2} outside by anti-port Na^{+} -Ca^{+2} exchanger carrier.

Myocardial contraction	Myocardial relaxation
depolarization wave⟶ Ca^{+2} ions release from the sarcoplasmic reticulum⟶↑intracellular Ca^{+2} ions⟶Contraction	Ca^{+2} ions reuptake to sarcoplasmic reticulum / active pumping of excess Ca^{+2} outside by anti-port Na^{+} -Ca^{+2} exchanger carrier ⟶↓intracellular Ca^{+2} ions⟶Relaxation

➢ Properties of cardiac muscle contraction:

All or none rule	Staircase phenomenon	Frank Starling's law
♞cardiac muscle either contracts maximally or does not contract at all ♞cardiac contraction occurs remain constant ♞No contraction at all occurs on using a sub-threshold stimulus	♞rapidly repeated stimulations → Gradual ↑ increase in muscle contraction	♞↑The initial length of cardiac muscle fibers during diastole→↑ Contraction of cardiac muscle (Directly proportional within limit) ♞Overstretching of the muscle fibers causes marked decrease in contractility ♞ The heart is dilated but the contraction is weak

➢ Factors affecting contractility

Nervous factors	Physical factors	Chemical factors
♞Sympathetic→↑intracellular Ca^{2+}→↑ contractility of the whole heart ♞ ParaSympathetic→↓intracellular Ca^{2+}→↓ contractility of the whole heart (Atrial muscle only)	♞Mild warming →↑intracellular Ca^{2}+& ATP formation→↑ contractility ♞Mild cooling →↓ intracellular Ca^{2}+& ATP formation→↓ contractility ♞Excessive cooling →↓ ATP formation→Stop contractility ♞Excessive heat →↓ ATP formation→ Stop contractility	♞Adrenaline, thyroxine, digitalis→↑ contractility ♞Acetylcholine →↓ contractility ♞Moderate acidity →↓ contractility ♞Moderate alkalinity →↑ contractility ♞Excessive Acidity or Alkalinity → Stop rhythmicity ♞↑K^{+}→ ↓lowering cell-resting action potential → preventing repolarization→ ↓contractility. ♞↑Ca^{+2}→ prolonging systole on the expense of the diastole→ preventing repolarization→ ↑contractility.