PASSION ACADEMIC TEAM

YU - MEDICINE

Cardiovascular System

Sheet# 1 - PHYSIOLOGY

Lec. Date :

Lec. Title : Electrophysiology Of The Heart

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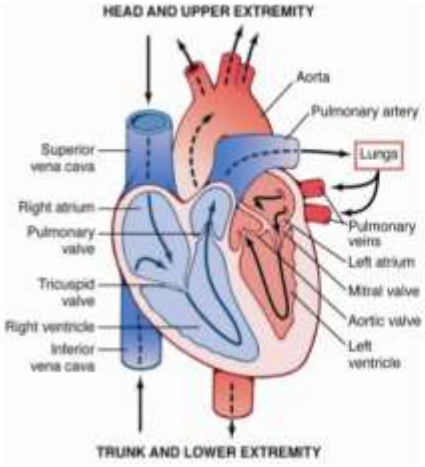
Electrophysiology of the heart

There is no direct contact between the two.

Electrical action potential is conducted by the AV bundle. There is a delay due to the bundles which allows the atria to contract ahead of the ventricles.



- Atrial syncytium
- Ventricular syncytium

- Fibrous insulator exists between atrium and ventricle (why?)

The heart consists of 2 main cell types:

1. Myocardial cells

- form 99% of the heart
- for contraction and relaxation.
- Contain actin, myosin sarcolemma etc. So they are built like underdeveloped skeletal muscles.
- Their sarcoplasmic reticulum (calcium storage units) is not as well developed as the skeletal muscles' sarcoplasmic reticulum is >>>> So calcium is less sufficient in the cardiac muscle >>>> which means there is a lower contraction force.

- Cardiac Muscles:
  - Contractile (myocardial) cells 99 %
  - Autorrythmic & conducting cells 1%.
- In addition to the syncytium property of the cardiac muscle, it also has the properties of
  - Excitability
  - Automaticity and rhythmicity
  - Conductivity
  - Contractility.

2. Autorythmic and conducting cells:

- form the remaining 1%
- for conducting the electrical impulses, these include SAN, AV etc.
- These are self-exciting meaning they generate their own action potential without stimuli in regular repetitive manner.

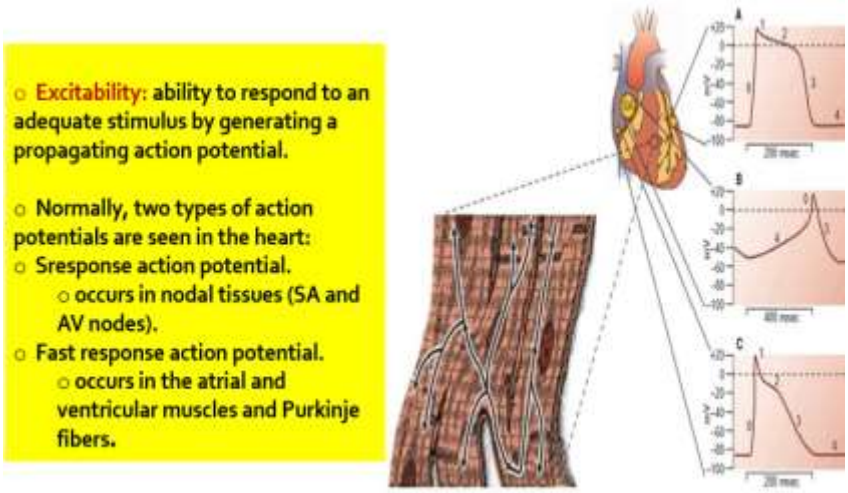
Cardiac cells are connected through intercalated discs >>> These include a combination of **mechanical (desmosomes)** and **electrical (gap junctions) methods.**

Desmosomes allow the cells to contact as a union while gap junctions allow the propagation of electrical impulses.

- **Excitability:** ability to respond to an adequate stimulus by generating a propagating action potential.
- There are 2 types of action potentials:

1)Fast response action potential (figures A and C) that contains 5 phases:

0: initial depolarization
1: initial repolarization
2: plateau
3: final repolarization
4: resting membrane potential



2)**slow response action potential** (figure B) that contains 3 phases:
4, 3 and 0

- Fast response action potential depolarizes rapidly while slow response action potential depolarizes slowly
- Slow response action potential: at the end of final (one and only) repolarization (which is phase 3) the cells begin to depolarize again.
  They have no stability which leads to the curve going upwards due to autorythmicity unlike fast response cells.
  This leads to an absence of resting membrane potential = membrane potential rising after every repolarization.
  Rapid repolarization, unlike in fast response cells.
  Cells auto generate many impulses per minute.

- The intrinsic frequency of SAN is around 80 impulses per minute making it the normal pacemaker of the heart because of its highest intrinsic frequency.
- Fast response cells have higher propagation but lower impulse generation count when compared to slow response cells.

Phase 0: initial depolarization:

- is caused by an increase in sodium influx into the cell.
- This influx is produced when the membrane is suddenly depolarized and the resting membrane potential increases from -90 millivolt to -65 millivolt.

- This changed in the membrane's potential is due to stimulation >>>> This leads to a transient increase in sodium conduction >>>> Sodium channels open briefly then close.
- This is due to them being self-limiting channels.
- The same stimulation that opens them closes them too.

Five distinct phases:
Phase 0: Initial rapid depolarization (upstroke):
- fast voltage-activated sodium Na+ channels

Phase 1: Initial repolarization:
- opening and then closing of transient outward (TO) K+ channels and are designated as I_{tofast} and I_{toslow}

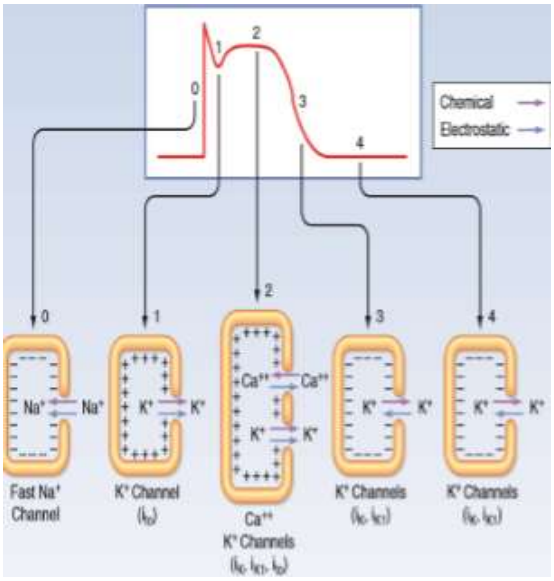
Phase 2: Plateau:
- the influx of positive current from calcium approximately matches the remaining K+ efflux carried through a few open K+ channels in the membrane.

Phase 3: Final repolarization:
- Inactivation of inward Ca+2 current
- Open of outwardly rectifying potassium channels I_K: I_{Ks} and I_{Kr}
- activation of inwardly rectifying K+ channels: I_{K1}

Phase 4: Resting potential:
- Mainly due to K+ outflow (along its conc. gradient) through inward rectifying K+ channels (i_{K1}).
- sodium-potassium pump
- 3Na+-Ca++ antiporter 1

الملخص:.....

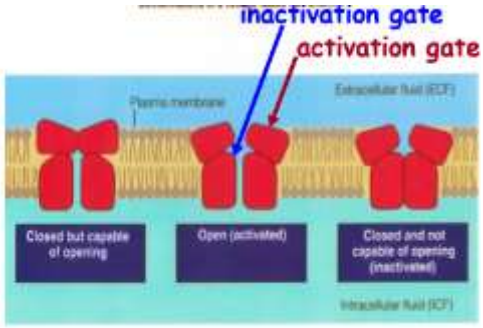
1)stimulation of the membrane
2)resting membrane goes from -90 mv to -65 mv
3)transient sodium influx into the cell due to the sodium channels being self-limiting
4)phase 0! initial depolarization



- There are 2 types of sodium channels.

1) activation gates
2) inactivation gates

Channel is closed due to activation gate being closed and inactivation gate being open.
=activation gate(closed) + inactivation gate(open) = closed sodium channel that does not open unless there is stimuli
Stimuli causes depolarization from -90 to -65 (threshold value)>>>>>> conformational change in channel proteins >>>>>The gates switch due to this change and open the sodium channel. Transient sodium influx leads to phase 0.
The gate is self-limiting so, inactivation gate opens leading to inactivation state = termination of phase 0.



Long retracting period of action potential:
Caused by sodium activated voltage channels.
When the sodium channels are inactivated they cannot be reopened>>>> This stops another action potential from generating.
The cells are in effective/absolute refractory period. This is to prevent tetanic contraction.
There is no contraction merging and no retardation of ventricular contraction.
The heart pumps normally due to normal blood filling the heart.

phase1: (initial repolarization)

- is happening when Na+ voltage channels are closed and transient outward K+ channels (Ito) are opened.
- both chemical and electrostatic forces drive K+ efflux and generating repolarization.
- Repolarization is a stage of an action potential in which the cell experiences a decrease of voltage due to the efflux of potassium (K+) ions along its electrochemical gradient. This phase occurs after the cell reaches its highest voltage from depolarization

Phase2: (plateau)

- Long lasting type L calcium channels once open inactivate slowly >>>> This leads to a long lasting calcium current/influx >>>> long plateau phase (Phase 2).
- Potassium out flux continues but it is equal to calcium = flat phase. Positive ions (calcium) in equals the Negative ions (potassium) out.
- Long lasting type L calcium channels are activated during depolarization when the membrane potential is -20 mv and they are fully activated at 0 mv , Transient type T calcium channels: activated at more negative (-70 mv). So, establish the resting membrane potential.
- Calcium that enters during phase 2 is essential in contraction as we already said that the sarcoplasmic reticulum is not well developed here so the amount of calcium is not sufficient making the cells require extracellular fluid calcium.

Phase 3: final repolarization:

- calcium channels have closed but efflux of potassium continues =repolarization
- The positive currents flowing inward and outward become almost equal during this stage.

Phase 4: Resting potential:

- Mainly due to K+ outflow (along its conc. gradient) through inward rectifying K+ channels (iK1).
- sodium-potassium pump
- 3Na+-Ca++ antiporter 1

Refractory periods:

- period during which normal cardiac impulse cannot excite an already excited area. Sodium channels cannot reopen.
- Happens during relaxation. Sufficient stimulus >>> normal stimulus = extra systole/premature beat. This contraction happens earlier than the normally expected one arising from AV node. = abnormal stimulation.

❖ Extra systole occurs earlier. Normal impulse from systole does not invoke an impulse. =no response =no contraction. So we can see a long pause after extra systole (compensating pause). The magnitude of contraction after compensation pause is very high. Why?

- There are another area of pacemaker cells of heart becoming hyper excitable known as >>>ectopic focus
- Hyper excitable and they are able to generate current and make irregularity of heart beats.

EXTRASYSTOLES : are premature beat (contractions) that are produced by impulses discharged from a hyperexcitable ectopic focus (= a focus other than the S-A node) during diastole i.e. during the RRP.

- **ventricular extrasystoles**
- **supraventricular extrasystole**

- If ectopic focus located in atrial region it called as supraventricular extra systole

❑ **Automaticity** is self-excitation (i.e. ability of spontaneous generation of action potentials independent or extrinsic stimuli).
❑ **Rhythmicity** is the regular generation of these action potentials.
❑ **Autorhythmic** cells present in:
(A) The nodal system:
(1) The sinoatrial node (SA), which is located in the wall of the right atrium near the opening of the superior vena cava.
(2) The atrioventricular node (AV), which is located at the base of the right atrium near the interventricular septum.
(B) The His-Purkinje systems:
(1) The bundle of His.
(2) The Purkinje fibers

❑ Normally the SA-node has the fastest inherent rate of discharge (100-110 /minute) and is thus the **normal pacemaker** of the heart. Other cells are known as **latent pacemakers.**

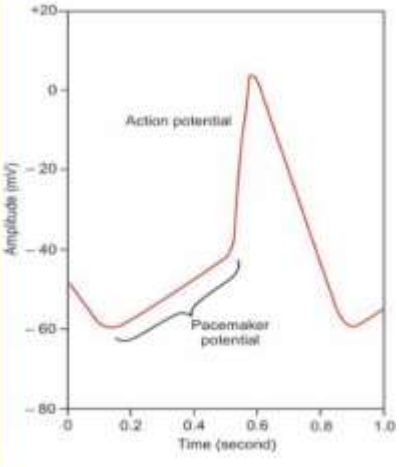
Location	Intrinsic Firing Rate (Impulses/min)
Sinoatrial node	70–80
Atrioventricular node	40–60
Bundle of His	40
Purkinje fibers	15–20

Laten pacemaker
- If the SA node firing rate decreases, or stops completely.
- If the intrinsic rate of firing of one of the latent pacemakers should become faster than that of the SA node.
- If the conduction of action potentials from the SA node to the rest of the heart is blocked.

Overdrive suppression
Chronotropism:
+ ve chronotropic
-ve chronotropic

- SA node and laten pacemakers have the capacity for spontaneous depolarization.
- The pacemaker cell with the fastest rate for phase of depolarization sets the heart rate. This is called overdrive suppression.

- The pacemaker cells in the nodal tissue have unstable resting membrane potential of -55 to - 60 mV.
- Gradual depolarization occurs spontaneously till a threshold is reached (- 40 to- 45 mV) at which an action potential is initiated
- This gradual depolarization is called **pacemaker potential , prepotential or diastolic depolarization.**



- The AV node has the fastest rate for phase of depolarization so it sets the heartrate.
- These cells are called the heart's pacemaker.
- SA node overdrives the other laten pacemakers so it sets the heart rate.
- They also have the short test action potential duration.
- So, they recover faster and are ready to fire another action potential before the other cell types are ready.
- Laten pacemakers have the chance to drive the heartrate only if the SA node is damaged/the laten pacemakers become faster than the SA node.

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