

## The conduction system

- In today's lecture we will discuss the conducting system of the heart.
- If we placed the heart in a special solution that contains  $\text{Ca}^{+}$  it will keep on contracting, keep in mind that before the mechanical response –contraction- happens, an electrical response (action potential) has to occur, this indicates that there must be an intrinsic source of electrical responses in the heart since we can transport the heart outside of the body in this solution where there are no neural connections.

**Note: the autonomic nerves that supply the heart don't have any influence in terms of initiation of impulses, they only aid in the regulation.**

From that, we conclude that the heart has an intrinsic system responsible for Action Potentials (electrical response) called: *The conduction system of the heart*. It consists of a group specialized cardiac cells, so we are going to discuss their specialization.

### Objectives:

- a) **List the parts of the conduction system.**
  - b) **Explain the mechanism by which these parts can produce AP intrinsically without any extrinsic source; without nerves.**
  - c) **Point out the regulation of the conduction system potential which occurs by an extrinsic source (Autonomic Nerves).**
- The conducting system consists of specialized cardiac muscle cells which are two types:
    - a) Cardiac muscle proper or the contractile muscle fibers.
    - b) Authorhythmic cells; the cells that produce the impulses automatically and regularly, they constitute about 1% of the total cardiac muscle fibers, they are very important, (they form the structures of the conduction system).

### Structures of the conduction system

- **SA node:** located in the posterior wall of the right atrium just below the entrance of the SVC.
- **AV node:** located between the atria and the ventricles, it still in the right ventricle around the fibrous septum that separates the atrium from the ventricle.
- **AV bundle:** Keep in mind that there is no muscular connection between the atria and the ventricles, that's why the atria contract as one unit and the ventricles contract as a totally separate unit but this happens in syncytium. When an impulse spreads through the atria that impulse will only continue to the ventricles if it was conducted from the atria to the ventricles through a wire system, which is called the AV node, which continues as the AV bundle. Sometimes it's called the bundle of His in recognition to the Hungarian scientist who described it.

- **Right and left bundle branches:** The AV bundle divides after running some distance in the interventricular septum into right (to right ventricle) and left (to left ventricle) bundle branches.
  - **Purkinje fibers:** The bundle branches end as Purkinje fibers after supplying 2/3<sup>rd</sup> of the myocardium. Sometimes called cardiac fibers or conducting fibers.
- Some people believe that there are internodal fibers/pathways (anterior/middle/posterior) between the SA node and the AV node are the ones to transmit the impulses between them.
  - Others, including the doctor, believe that the impulse *from the SA node reaches the AV node* through the atrial muscles and not through internodal fibers.
- The difference between these parts is their ability to produce impulses at different rates:
1. **SA node:** is able to produce intrinsic impulses at the rate of **60-80** impulse/min up to 100.
  2. **AV node:** is able to produce intrinsic impulses at the rate of **40-60** impulse/min.
  3. **Purkinje fibers:** (the last structure) are able to produce **15-40** impulse/min; they have the lowest rate of producing the impulse.
- Note that the SA node produces the first impulse, then this impulse is conducted to the AV node through the atrial muscle, then to the bundle of His and from there it goes to Purkinje fibers.
- Since the **SA node** has the highest rate of impulse production, it's the one that sets the rate for the whole heart.
- We can think of it as a train that consists of three carts, each having its own motor and speed, the first cart has the highest speed and the last one is the slowest, if all carts work normally; the speed of the train is the speed of the fastest one, which is the SA node. If the SA node is destroyed or damaged the heart rate will follow the AV node's rate.
- The **SA node** is the normal pacemaker of the heart.
- If the SA node was destroyed or damaged the **AV node** will take over and it's called an **Ectopic pace maker**, because in this case it *discharges at a rate higher than the SA node*. In other words; it's an abnormal site of a pacemaker (the word ectopic means not in its place)
- **AV Block/ Heart Block:** Even if the SA node is intact but the AV node is destroyed; there will be *no conduction of the impulses from the atria to the ventricles* since there won't be a muscular connection between the atria and the ventricle, *In other words: there is NO connection between the SA node and the purkinje fibers, so the ventricles will work at the rate of purkinje fibers.* In this case the rate will be between 15-40 impulse/min.

Some people can't live with this rate, because they need a higher rate and higher cardiac output, so these patients require an **Artificial pacemaker**. (Placed on the right ventricle to overcome the AV node; produces electrical discharges. Old ones require a battery that lasts for 5-6 years, and they have a fixed rate. While in the new ones the rate can be changed)

- The is the sequence in which the impulse is conducted:

SA → AV → Bundle → Branches → Purkinje fibers

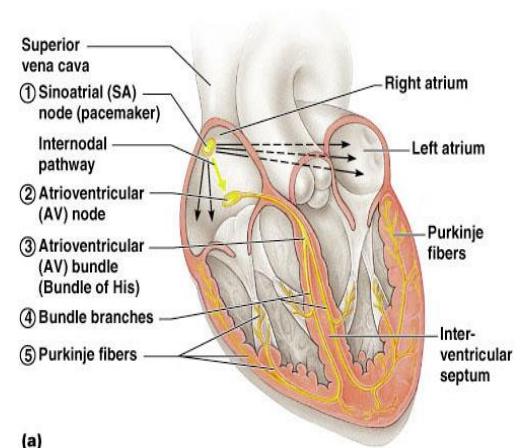
Recall that the SA node has the highest conduction **rate** and Purkinje fibers have the lowest.

- **AV node acts as a delay system:**

- It delays the impulse to ensure that the atria contract and finish their contraction before the ventricles start to contract (we don't want the atria and the ventricles to contract together).
- Normally the atria contract and then the ventricles contract, they don't contract together, that happens only in abnormal conditions.
- The **AV node** has the **slowest speed of conduction** → 0.02 m/sec; this is to delay the conduction of the impulse.
- The **fastest speed of conduction** is through the **Purkinje fibers** → 4-5 m/sec; this is *to make sure that the impulses are conducted through to all parts of the ventricles at the same time*, and this ensures that all the cells of the ventricles will contract as one unit and makes them an effective pump. This is essential for the normal function of the heart.
- If not all of the cells of the ventricles contracted at the same time, and some fibers were contracted while others are relaxed, this is called: **Ventricular Fibrillation**, which is lethal.
- We can survive **atrial fibrillation**, however, because the contraction of atria is not essential for the normal function of the heart, remember that the contraction of the atria aids only in the flow of 20% of the blood into the ventricles and the other 80% flows passively
- The difference in conduction speeds is due to the difference in size between them: The SA and AV nodes are rounded with less gap junction. The diameter of the Purkinje fibers is larger.
- The conduction rate of atrial and ventricular muscle cells is moderate → 0.5-1 m/sec
- In the case of **Bundle branch block**: the ventricular cells that are supplied by that blocked branch will receive impulses through the ventricular muscles, at a speed of 0.5m/sec which is lower than the normal conduction rate. Therefore the ventricles go through a period of prolonged depolarization (the doctor will discuss it again in ECG).

- Usually the conduction through the nodes is unidirectional, but it might be reversed.

- The **SA node** conducts the impulses to the **AV node** through the *muscles of the atria* (as we mentioned, this is what the doctor believes)
- The AV node delays the impulses to allow the atria to contract, and then the **bundle branches** spread the impulses in the walls of the ventricles, and then by the action of the **purkinje fibers** the impulses are spread put to all the muscles of the ventricle.



*Let's sum up:*

- SA node: Specialized cardiac muscle connected to atrial muscle. Acts as a pacemaker.
- Internodal fibers: Transmit cardiac impulses throughout atria. There are anterior, middle, and posterior internodal pathways.
- AV node: delays the impulse conduction to allow the atria to contract before the ventricle contract. (the amount of seconds of delay is not required from us)
- AV bundle: the right and left branches send the impulses to the right and left ventricular walls correspondingly. One way conduction.
- Purkinje fibers: have the fastest speed of conduction, although they have the lowest rate of production of intrinsic impulses; because their size is larger (they have a larger diameter than the others). They contain many gap junctions at intercalated disks.

➤ The **ANS** with its branches; sympathetic and parasympathetic, supply all parts of the heart:

1. **Sympathetic**: Atria, SA node and AV node, Ventricles (all heart).  
These fibers come from the cardiac plexus.
2. **Parasympathetic**: Atria, SA node and AV node. Very little or no supply to the ventricles and has very minimal effect on them, here we are taking about neural stimulation, but as for chemical stimulation due to the release of Ach by Parasympathetic fibers, it has an effect on the ventricles since they have muscarinic receptors.  
These fibers come from the two vagus nerves.

The innervation to the ventricles is mainly by the sympathetic and the parasympathetic has a minimal or no role.

- ✓ Keep in mind that the **Vagus Nerve** *supplies* the SA and the AV node but it *doesn't supply* the *purkinje fibers* because they are *found in the ventricle*.

## How does the conductive system intrinsically produce impulses (intrinsically active)? What is the mechanism?

- This diagram represents the AP of the **contractile muscle proper**.
- We have already mentioned that the conducting system cells are **specialized cardiac cells**; there specialization in terms of:

### Anatomy:

- They are rounded, except the Purkinje fibers.
- They lack/ very little contractile muscle fibers → unable to contract.

### Function:

- They are leaky to  $\text{Na}^+$ . Not through voltage gated channels, instead, the membrane contains leaky  $\text{Na}^+$  Channels.

During phase 4 for **contractile muscle proper**: membrane potential returns to -90.

- **BUT in autorhythmic cardiac cells; since these cells are Leaky to  $\text{Na}^+$ :**

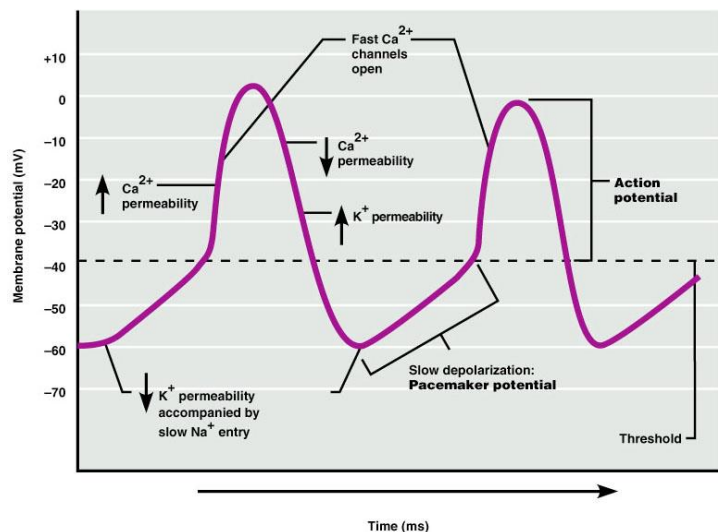
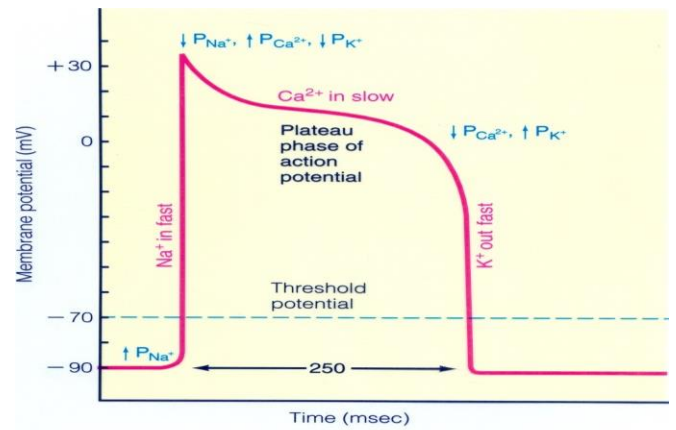
- There is  $\text{Na}^+$  influx during **Phase 4**, and the resting membrane potential will never reach -90 → it will be less negative: -60 or -65. During Phase 4, the membrane potential is depolarizing; therefore it is called the **slow depolarization phase**. As  $\text{Na}^+$  enters until the threshold is reached slowly.
- This gives enough time for the *slow inactivation gates to close before the fast activation gates open*. Therefore, Sodium will not enter.
- Recall: Fast Sodium channel has two gates: the activation/M gate (fast) and the inactivation/H gate (slow), the *difference is in the timing and not in the voltage requirement*, their threshold is the same but their response is different.
- The threshold for the  $\text{Na}^+$  channels is the same as that of the  $\text{Ca}^{2+}$  channels, therefore, when the threshold is reached, the slow  $\text{Ca}^{2+}$  channels open →  $\text{Ca}^{2+}$  influx → Rapid depolarization. This is **Phase 0**. (There is influx of  $\text{Ca}^{2+}$  but there is no  $\text{Na}^+$ ; because the  $\text{Na}^+$  channels are closed.)
- There is no phase 1 and no plateau.
- Phase 3 is due to the efflux of  $\text{K}^+$

- **Phase 4:** slow depolarization phase

- **Phase 0:** due to  $\text{Ca}^{2+}$  influx

We don't have Phase 1 or Phase 2

- **Phase 3:**  $\text{K}^+$  efflux



- **The phases are therefore: 4/Slow depolarization → 0 → 3 →** this kind of response is called **the slow response action potential** (The one for cardiac proper muscle is called fast response). The impulse is automatic, as long as Na keeps leaking inside there will be an AP, and it is rhythmic.

- This action potential is autorythmic, produced by the SA node → through atrial muscles → the AV node → AV bundle → purkinje fibers → Ventricles
- They differ in their rates of producing impulses, and this is due to the differences in their leakiness to Sodium: The SA node is *more leaky to  $Na^+$*  than the AV node, and both of them are *more leaky to  $Na^+$*  than the purkinje.

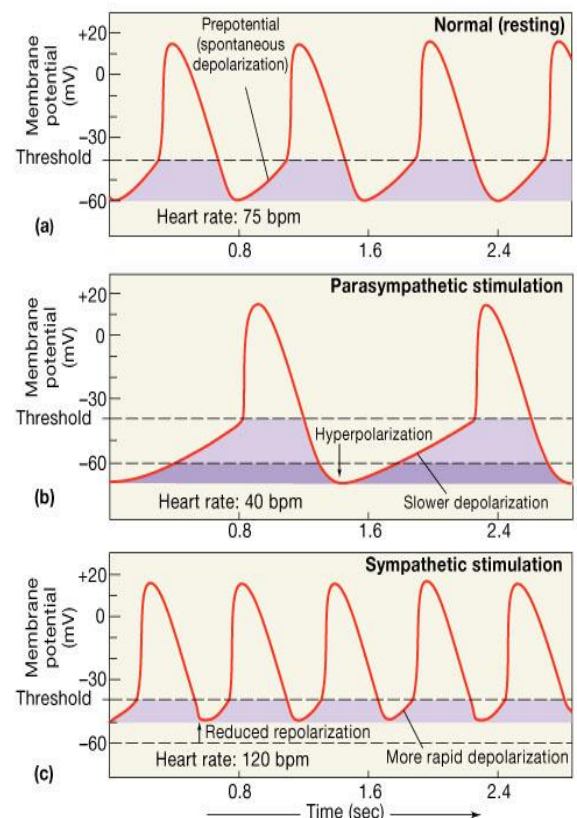
- At Phase 4/ slow depolarization: the angle of this depolarization is the **slope**. The steeper the slope → the higher the heart rate. The less steep the slope → the lower the heart rate.
- The slope for the SA node is higher than the AV node which is *higher than the slope for the Purkinje fiber*.
- Speeds of conduction
  - SA node: Slow
  - Ventricular and Atrial muscle: Moderate
  - AV node: Slowest
  - Purkinje fibers: Fastest

## Extrinsic Regulation of the Heart Beat

We mentioned that the nerve supply of the heart is through sympathetic and parasympathetic fibers. Now we will discuss how they induce their effects:

- **Sympathetic:** release both Epinephrine and Norepinephrine → increase the permeability of cardiac cells to  $Na^+$  and  $Ca^{2+}$  and decreases the permeability to  $K^+$ .
- **Parasympathetic:** release Ach → increases the permeability of cells to  $k^+$  and decreases it to  $Na^+$  and  $Ca^{2+}$ .

Changing the heart rate is called the **Chronotropic Effect** (if it is +ve, there is an increase in the heart rate and if -ve there is a decrease).



### Sympathetic:

- By increasing the permeability to  $\text{Na}^+$ , the slow depolarization phase will be faster and the resting membrane potential will be less negative (Instead of -60 it will be -55) → therefore the rate increases → **+ve Chronotropic Effect**.
- By increasing Ca permeability, it increases the intracellular  $\text{Ca}^{2+}$  → increase the contractility/ the force of contraction and this is called a **+ve inotropic effect**.
- The slope of the slow depolarization phase is **more** steep.
- Supply all parts of the heart.
- Increases the speed of conduction of impulse: **+ve Dromotropic effect**.

(Refer to the figure above for better understanding)

### Parasympathetic:

- Increases the permeability of cardiac cells to  $\text{K}^+$  (more K efflux) → the resting membrane potential is more negative (Instead of -60 it will be -65) → cells require more time to reach the threshold → It has a **-ve chronotropic effect**: it decreases the heart rate.
- The slope of the slow depolarization phase is **less** steep.
- The ventricles aren't supplied by the vagus nerves so there is *minimal/no effect on their contractility*.
- It supplies the atria and the SA, AV nodes
- **-ve Dromotropic effect**

*Always keep in mind that the peak never changes, because the action potential is all or none, but the rate and the slope of depolarization can change.*

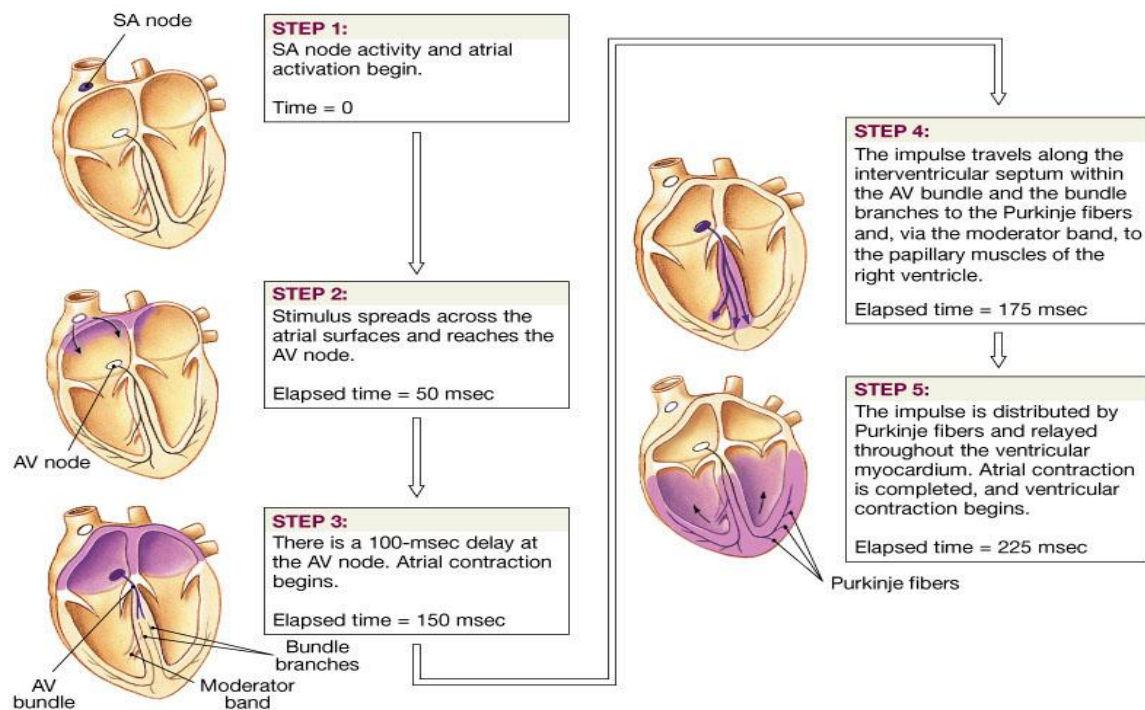
Check out this summary from slide 26:

Effect of autonomic nerve activity on the heart		
Region affected	Sympathetic Nerve	Parasympathetic Nerve
SA node	Increased rate of diastole depolarization ; increased cardiac rate	Decreased rate of diastole depolarization ; Decreased cardiac rate
AV node	Increase conduction rate	Decreased conduction rate
Atrial muscle	Increase strength of contraction	Decreased strength of contraction
Ventricular muscle	Increased strength of contraction	No significant effect



*To sum up:*

- **Sympathetic:** from the cardiac plexus. Supplies all parts of the heart (atria, ventricle and all parts of the conduction system). Increase the permeability of the cardiac cells to  $\text{Na}^+$  and  $\text{Ca}^{++}$  i.e Positive Chronotropic and positive Inotropic action
- **Parasympathetic:** from Vagus nerves. Supply mainly the atria, SA and AV nodes, very little supply to ventricles. Increase the permeability of the cardiac cells to  $\text{K}^+$  and decrease its permeability to  $\text{Na}^+$  and  $\text{Ca}^{++}$ . Negative Chronotropic effect and no significant Inotropic effect.



The steps 1, 2,3,4,5 explain how the impulse spreads through the heart

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## Overdrive Suppression and Ventricular Escape

- If there is massive stimulation of the parasympathetic (abnormal) → the heart rate might stop: the ventricles will stop contracting, no cardiac output and the brain won't be supplied with blood. → The patient will undergo Syncope (coma) and will fall down.
- After 15-30 seconds, the patient will wake up but their *heart rate will be that of the Purkinje fibers*: 15-40. Why does this happen?
  - The rates of the Purkinje fibers and the AV node are normally suppressed by the SA node's higher rate. This is called **overdrive suppression** (their own rate is suppressed and they receive a higher rate by the SA node.)
  - It takes some time for the Purkinje fibers to take over (15-13 seconds). Then the person wakes up due to **ventricular escape** → the ventricles escape the effect of the parasympathetic innervations since they are not supplied by it.
- This happens in **Stokes-Adams syndrome**: a vascular syndrome whereby vessels from the carotid press on the vagi and stimulate them. If this stimulation is extensive it may cause syncope. The person faints for a little while because there is no heart beat then wakes up because of the ventricular escape.

*To sum up:*

- The heart stops beating for (15-30) seconds due to over drive suppression and it then returns to its function due to ventricular escape.
- This happens due to:
  - AV Block
  - SA node failure to initiate a beat
  - A failure of the conductivity from the SA node to the AV node
  - Stokes-Adams syndrome
- The Purkinje fibers take over due to ventricular escape; because they aren't supplied by the parasympathetic fibers.
- The doctor read slide 34:
  - Parasympathetic (vagal) nerves, which release Ach at their endings, innervate S-A node and A-V junctional fibers proximal to A-V node. This causes hyperpolarization because of increased K<sup>+</sup> permeability. This causes decreased transmission of impulses maybe temporarily stopping heart rate. Ventricular escape occurs.

The end  
Smile