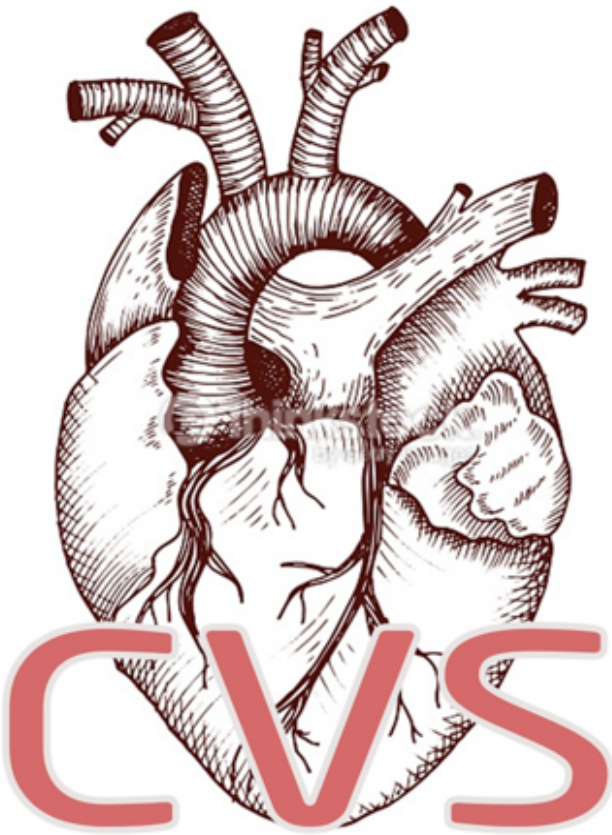

CVS

Physiology

♡ Slide Sheet ❤

Number:	4
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Topics OF THE PREVIOUS LECTURE:

1- The special characteristic of the cardiac cells.
2- We talked about the difference between the action potential in skeletal muscle and cardiac muscle
3- Frank-starling law

Some concepts you must know:

Total tension =active tension +passive tension
We cannot measure the active tension directly so
Active tension =total –passive tension

Passive tension is the tension before the contraction occurs
Active tension →when contraction occurs
Stork volume→ the amount of the blood pumped in each beat
*The law(Frank-starling law) says: within physiologic limits
As the length increases →the tension increases

In the heart the volume of the ventricle is proportional to the length of cardiac muscle fibers
So as the volume increase → the tension increase
Which volume are we talking about here???
-The volume before the contraction of the ventricle (passive/resting tension) we can also call it end diastolic volume EDV
-The volume when contraction (active tension) in the center (ejection from the right ventricle to the pulmonary trunk, or from the left ventricular to the aorta) end systolic volume

We can increase the volume for a limited degree after that we will have a heart failure

I recommend you to watching this video
https://www.youtube.com/watch?v=5SO58NndlPI

Now we will stare the topic of this lecture

This is the conduction system of the heart

Objectives:

1-List the parts that comprise the conduction system
2-Explain the mechanism of slow response action potential (pacemaker potential)
3-Point out the regulation of the conduction system potential by Autonomic Nerves

Resource: Guyton's Textbook of Medical Physiology last edition **(115-120)**

The heart contract without the aid of any external nerve impulses, if you removed the heart from the body in heart transplant, the heart will keep pumping, only if you put it in a solution contains calcium.
So how the contractions happen??
You mast know that for **any muscle to contract** there **must be an action potential**, That means that the source of the action potential comes from ***an intrinsic source*** within the heart itself
(This is called the conduction system of the heart)

The cells of the conduction system are not nerves they are a modified cardiac cells we call them **autorhythmic cells** and they differ from the normal contractile cells (cardiac proper cells).

It’s important not just to know the component of this sys but to understand the mechanism of how these cells produce action potential????

- **Components of the conduction system :**

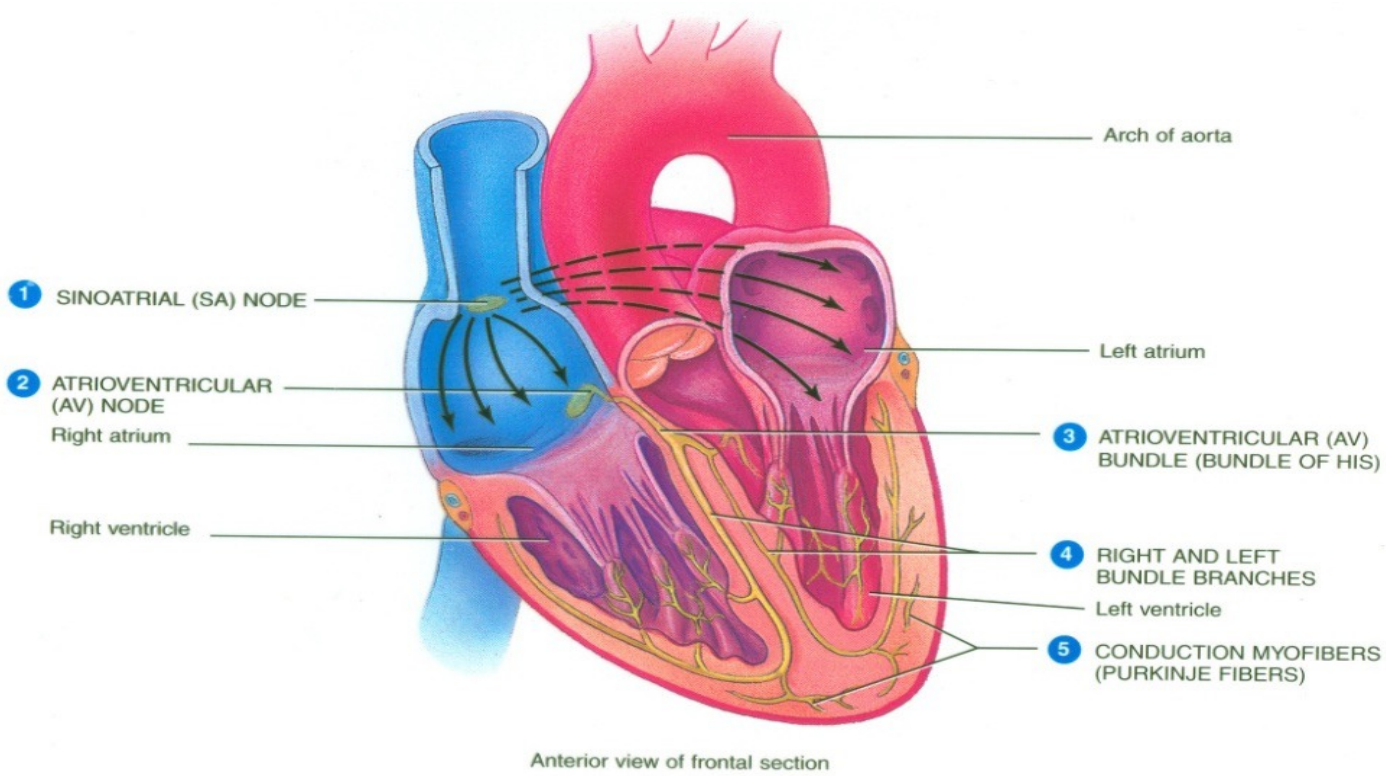
1. Sinoatrial node [SA node]. (In the posterior aspect of the R. atrium just below the entrance of the S.V.C).
2. Atrioventricular node [AV node]. (In the area between the R. ventricle and the R. atrium Connected to the

↓

3. AV bundle (bundle of his).which pierce the atrioventricular septa going to the right and left ventricles as the

↓

4. Bundle branches (right and left). These are subendocardial branches that go to Right and Left ventricles to form >>
5. Purkinje fibers.



Anterior view of frontal section

Some believe that there are internodal fibers between the AV node and the SA node through which, the pulses move and reach AV node
(The doctor doesn't believe that). Anterior, posterior, and middle internodal bundles
The doctor believe that the cells of the atrium are what conducting the pulses to the A.V node

All of these parts are able to produce intrinsic pulses by themselves with different rates; these cells are called **autorhythmic cells**: they can depolarize and repolarize

Independent from external innervations they form 1% of the heart
The rest 99% → contractile

There are differences between them in:

1. Their Intrinsic rate:

The number of (impulses, action potential, firing rate, discharge rate) all are generated per minute.
SA node: 70-80/min.
AV node: 40-60/min.
Purkinje fibers: 15-40/min.

1. The ***SA node*** generate the first impulse due to its intrinsic activity (it is the normal **pacemaker** of the heart) pace means speed , anything else is considered ectopic.
2. The impulse is conducted through the muscles of the atria.
3. The ***AV node*** receives impulse from the SA node through muscle fibers within both atria.
4. The impulse is then conducted through the bundles (bundle of his and bundle branches) till reaching the ***Purkinje fibers.***

Are these cells supplied by nerves??

- **Actually yes they are but there is difference in their supply**

  -The sympathetic [comes from cardiac plexus] supply all parts of the heart.

  -<u>The parasympathetic [comes from vagus nerve] only supplies the AV node and SA node and the right and left atria</u>. There are no fibers to the rest of the heart (to ventricles).

  This supply is only for regulation of the conduction sys.

  We will talk more about this later in this sheet.

The pacemaker of the heart is the S.A node because it has the highest rate.

So what if the S.A node not functioning???

Think of them as carts of a train, the higher cart lead ;)

- Normally the ventricle contract within a rate that is 70-80 (pace maker) ,this means that the Purkinje is also working within a rate that is 70-80, the intrinsic rate of Purkinje here is said to be suppressed, this is called **over drive suppression,** the same is for the AV node.

  In other words, normally SA node will produce impulses at 70-80 , AV node will conduct these impulses at the same rate 70-80 (here we say that its own rate has been suppressed) and Purkinje will conduct the impulses at the same rate too. At the end, the ventricles will contract at this rate.

So, the heart beats by a rate equals the highest intrinsic rate, normally SA node has that the highest rate. Once SA node is nonfunctional, the train will move but with the speed of the AV node, and this is not the normal pacemaker → ((ectopic pacemaker)

- If the AV node is destroyed (even if the SA node is intact)

  - the <u>ventricle</u> will contract at the rate of Purkinje fibers (15-40). Why?

The ventricle will not receive signal, since there is no connection between the atria and the ventricle).

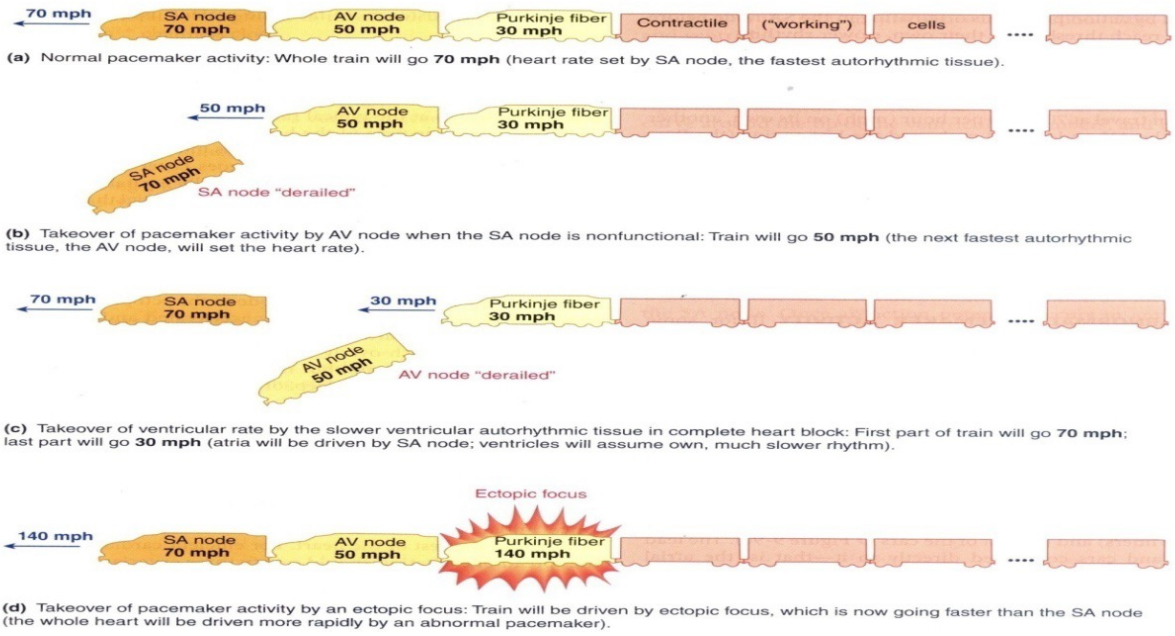
We have septa (fibrous tissue), separate the atria from the ventricles and doesn't conduct impulses.

- The <u>atria</u> will contract at the rate of SA node.

* The damage in the AV node is called AV block (heart block), the impulse will not reach Purkinje fibers.

- what if the Purkinje have an increased abnormal beats 120per min it will lead the heart (the highest rate) this is also not normal →ectopic
  but the atria will contract at the SA node

So when we say ectopic it may be high or low rate (not the normal pacemaker)



(a) Normal pacemaker activity: Whole train will go **70 mph** (heart rate set by SA node, the fastest autorhythmic tissue).

(b) Takeover of pacemaker activity by AV node when the SA node is nonfunctional: Train will go **50 mph** (the next fastest autorhythmic tissue, the AV node, will set the heart rate).

(c) Takeover of ventricular rate by the slower ventricular autorhythmic tissue in complete heart block: First part of train will go **70 mph**; last part will go **30 mph** (atria will be driven by SA node; ventricles will assume own, much slower rhythm).

(d) Takeover of pacemaker activity by an ectopic focus: Train will be driven by ectopic focus, which is now going faster than the SA node (the whole heart will be driven more rapidly by an abnormal pacemaker).

Student question if the AV node is damaged we will have two rates (atrial and ventricular rate)
When we are measure the heart rate which one we are measuring??
Answer:

Acutely when we take the heart beats it belong to the ventricle rate not to the atrium
We can measurer the atrial rate but with a different way we will talk about later

Pathway of the Heartbeat

- Begins in the sinoatrial (S-A) node
- Internodal pathway to atrioventricular (A-V) node (if they actually present)
- Impulse delayed in A-V node (allows atria to contract and finish their contraction before ventricles contract) because they should not contact at the same time it will be useless
- A-V bundle takes impulse into ventricles
- Left and right bundles of Purkinje fibers take impulses to all parts of ventricles

Conduction speed:

Conduction Speed (conduction rate):

- **SA node**: slow speed of conduction.
- Ventricular and Atrial muscle : Moderate speed
- **AV node**: slowest speed of conduction.
- **Purkinje fibers**: Fastest speed of conduction

-The conduction rate is **fastest** in the Purkinje, 4m /sec, due to higher number of gap junctions -look like the normal contractile muscles with large diameter and low resistance- to make sure that ventricles will receive the impulse at the same time and contract at the same time, otherwise, each ventricular fiber contracts dependent from the others, this is called ***ventricular fibrillation,*** it's lethal and the physician should interfere to relief the condition. But it has the lower intrinsic action in the system

-The conduction rate is **slowest** in AV node, why? To assure that the atria and ventricles will not contract at the same time; atria contracts and finish its contraction then ventricles are able to contract and this is mediated through AV node which delays the impulse.

-The conduction is by definition unidirectional, this is due to refractory period (anything behind the impulse will not be polarized again for a while so the wave can't go back).

Note:

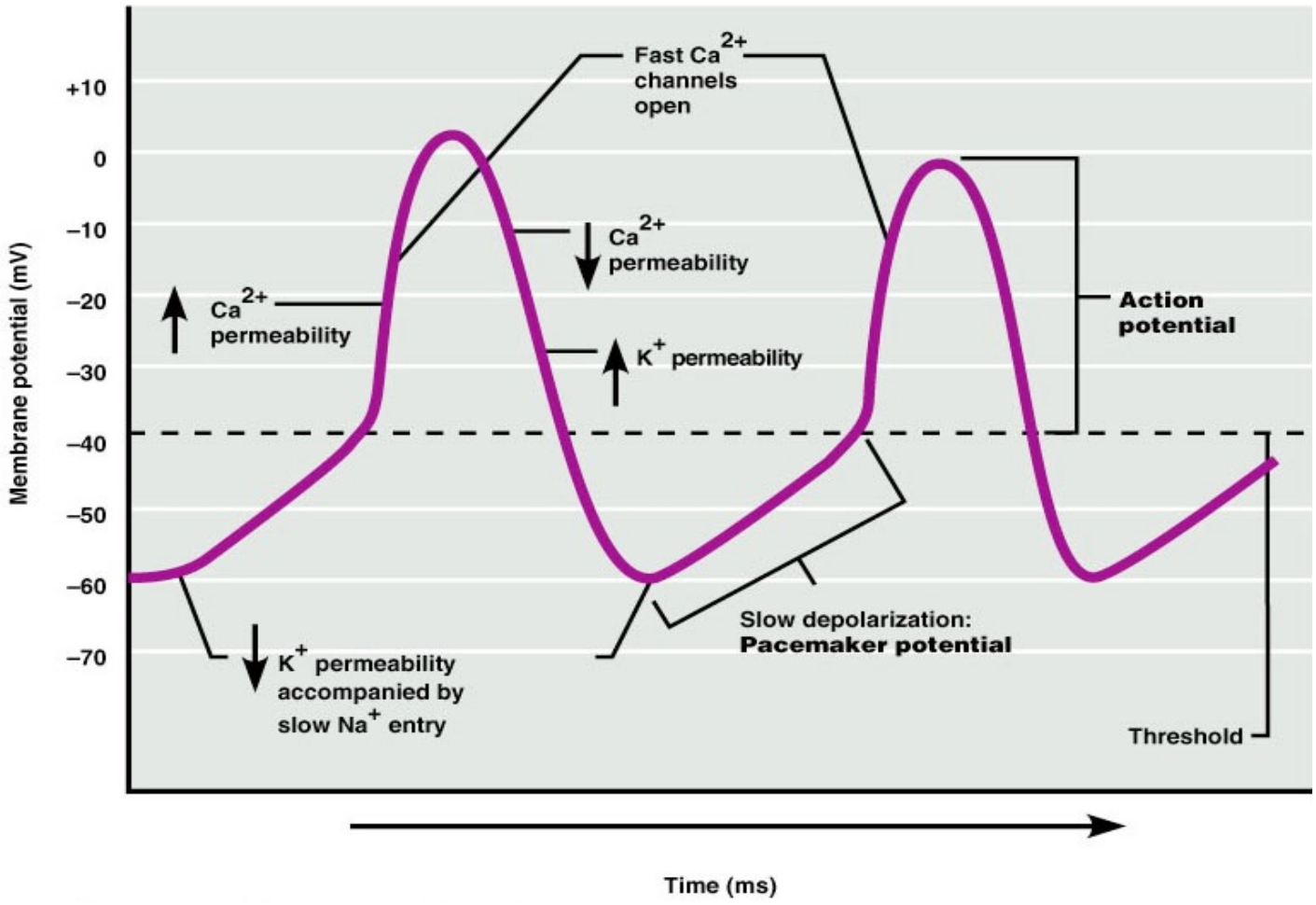
If the branches (**Bundle of His)** are damaged the rate will be conducted by the Ventricle cells but they will be slower

Now we will talk about the machine
How these cells generate intrinsic action potential????

First;
Remember the fast response action potential (action potential proper):

1-startes at resting membrane potential of -90
2-fast depolarization due to opening of fast voltage gated Na+ channels
4- Phase 1 → partial repolarization
5- Phase 2 plateau (due to ca+ influx)
6- Phase 3 repolarization due to voltage gated K+ channels

Tissue	Conduction rate (meter/sec)
Atrial muscle	**0.3**
Atrial pathways	1
AV node	0.05
Bundle of His	1
Purkinje system	4
Ventricular muscle	0.3-0.5



- On the other hand these cells (SA node, AV node, Purkinje fibers) in terms of physiology the cells are modified: they are leaky to sodium (leaky channels not voltage gated channels), in phase 4, sodium will follow its electrochemical gradient, move toward intracellular compartment (inside cells) and cause slow depolarization to reach the threshold.
- The resting potential will be less negative reaching -60 never -90.

By looking to previous diagram above, you can notice that:
• In **phase 4** (from -60 to threshold) we have **slow** diastolic **depolarization,** we reach the threshold slowly.
• Thus the inactivation gate of sodium channel (H gate) the "slow gate" will have enough time to close before the M gate the "fast gate" opens.
So, **Na ions will not enter through fast channels.** طيب أنا كده استفدت ايه ؟

• **Phase zero** (fast depolarization) -when we reach the threshold slowly- is due to **slow voltage calcium channels (Ca++ influx only),** and this depolarization is slower than the depolarization of action potential for contractile cardiac fibers which is fast depolarization.

• There is no phase1 (partial repolarization) nor phase 2 (plateau)
• There is **phase 3** which represents the repolarization by increasing the permeability for K+ ions
This is called **slow response action potential**.
Note: these cells are called automatic cells or autorhythmic cells because normally without any stimulation (intrinsically), whenever the cell reach -60, a slow depolarization will occur until we reach the threshold and then we will have an action potential.

- **Depolarization or phase 0 → due to ca2+ influx**
- **No plateau**
- **No phase 1**
- **A.V node is less leaky to Na+ → it will reach the threshold later (the slow depolarization phase is slower) → rate is slower**
- **Purkinje fibers → less leaky than A.V node → the rate is slower**
- **That's why there is difference in their intrinsic rate.**

Autonomic innervations of the heart:

The heart intrinsically can pump blood and generate action potentials.
• But if the heart can intrinsically pump, then why we have the autonomic nerves then?

We have both sympathetic and parasympathetic innervations:
1. ***Sympathetic*** from ***cardiac plexus*** supplies all the heart (ventricles and atria).
2. ***Parasympathetic*** from **vague nerve,** supply only the atria (SA node and AV node), don't affect the ventricles

The sympathetic stimulation (giving epinephrine/ norepinephrine):

1. Increase permeability to sodium and calcium and decrease for K+.
2. The resting membrane potential become less negative, the slow depolarization occurs faster (increasing the slope of phase 4), the rate increases

Strength of contraction also **increases due to higher calcium entering. (**The effect on other cells of the heart not the SA node because SA node does not have contractile tissue)

3. The increase in the rate is referred to **as positive chronotropic effect.**
4. The increase in the strength of contraction (contractility) is referred to **as positive inotropic Effect**
5. The sympathetic also have **positive dromotropic effect** "higher rate of conduction".

Parasympathetic stimulation (giving acetylcholine):

1. Affect only the atria including SA node and AV node.
2. Increase permeability to potassium. (k+ will efflux)
3. Decrease permeability to Na+ and Ca++.
4. The resting membrane potential becomes more negative.
5. Slow depolarization occurs slower (decreasing the slope of phase4), so the Heart rate decreases thus **negative chronotropic effect.**
6. No effect on the contractility of ventricles.
7. Causes **negative inotropic and dromotropic effect** on the atria only.

In both types of stimulation (the slow and the fast) the peak **doesn't change;** because action potential follows "the all or none principle".
Take a look to the slides

(الأحلام الكبيرة لا تسمح لأصحابها بهناء النوم, ولا لذة الركون أو رغد المُستقر)