Conduction System of the Heart.. Follow ...

Slow Response Action Potential (pacemaker potential).. slide 21

Remember:
- SA node + AV node + Purkinje >> 1% of the cardiac muscle
- They are modified cardiac muscles in term of structure and so in term of function.
- Structure > they are: 1- rounded  2- No contractile fibers (myosin & actin)
3- leaky to Na+ (Resting can’t reach -90 potential, it just reaches ~ -60)
why ?? Before reaching -90 there will be an electrochemical gradient for Na+ → Na+ will leaks to inside (influx) preventing potential from reaching -90.

↓

Then Na+ will continue the influx until reaching the threshold ..
** This influx is SLOW to the extent that the inactivation gate will close (Subsequently, Na+ can’t be leak even if the activation gate is opened)
so >>> there is No phase 0! and there is No plateau coz slow Ca++ channels are already opened to compromise phase 0.

↓

Repolarization (K+ channels open,[Na+,Ca++] influx ceases) also can’t reach -90 because of the same reason .. >> Na+ starts to leak again and make another slow depolarization until reaching threshold and so on..

That what is called Autorythmic (intrinsic) action potential.

i.e. the inherent leakiness of the sinus node to Na+ and Ca++ causes their self-excitation.

AV node: The same thing except that it is less leaky to Na+ so Resting potential will be more –ve (The slope of the slow depolarization phase will be lower) >> we need more time to reach the threshold >> slower action potential >> so Heart rate will be lower.
Purkinje: even less..

Slide 22

Speed of conduction:
* AV node: Slowest conduction >> AV Delay > the impulse is delayed in the AV node > so the atria contract and finish their contraction before the impulse reaches ventricles (i.e. giving the atria time to empty their blood into the ventricles before ventricular contraction begins).

* Purkinje: Fastest, why? to conduct the impulse to the whole muscle (ventricles) at the same time.
[Both ventricles depolarize at the same time and so > contract at the same time >> Efficient Pump.]

AV Block

When AV node is blocked > Ventricles will follow the rate of Purkinje fibers (15-40) >> patients will feel tired from a little exercise.

**Management: Using an Artificial Pacemaker with a Battery.
(putting the battery under the cubital skin .. then connecting it to the heart via (anticubital vein > axillary > subclavian > brachiocephalic > superior vena cava > atrium > Right Ventricle).
*New one > Real time pacemaker that make the beats (rate) according to the status of the patient.
*This Battery is changeable (every certain period we remove it and put another one).
Extrinsic innervation of the heart

By Autonomic nervous system >> Not important for initiation but for Regulation.

<table>
<thead>
<tr>
<th></th>
<th>Sympathetic</th>
<th>Parasympathetic</th>
</tr>
</thead>
<tbody>
<tr>
<td>From</td>
<td>Cardiac plexus that comes from sympathetic chain</td>
<td>Both Vagi</td>
</tr>
<tr>
<td>Supply</td>
<td>SA, AV, atria, ventricles</td>
<td>SA, AV, atria, little if not to ventricles</td>
</tr>
<tr>
<td>neurotransmitter</td>
<td>Epi., Norepi. (adrenaline, noradrenaline)</td>
<td>Acetylcholine</td>
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</table>

Effects on the Heart >>

**Sympathetic**: increase permeability of cardiac cells to Na+ and Ca++. 

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**+ve Chronotropic**

1- Depolarization is going to be faster (higher slope) 
2- Resting potential will become less -ve 
   we reach the threshold faster 
   heart rate increases.

Note:
Chrono = timing
( effect is on the heart rate)

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**+ve Inotropic**

More Ca++ inside the cell 
↓
Increase Contractility
(force of contraction)

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Also there is a **+ve Dromotropic** effect (increase rate of conduction of impulse)
**Parasympathetic:**

- Increase permeability of cardiac cells to K+ and decrease permeability to Na+ and Ca++. 

- More negative resting potential.

- Slower depolarization (lower slope) → heart rate decreases.

So >> **+ve Chronotropic** effect.

Is there any effect on contractility (inotropic)?

No ... Because parasympathetic doesn’t supply ventricles.

What part of the graph doesn’t be affected by ANS?? the Maximum.

**Ventricular escape**

If the vagus is stimulated extensively Heart will stop > Cardiac Arrhythmia.

But as the purkinje don’t receive impulses from the vagus > after 15-30min. they start impulses and contraction (purkinje renew the rate) > so the ventricles escape..

**Overdrive suppression**

Normally purkinje are suppressed by the overdrive of SA node!

**Slide 28** the last part of the heart to get depolarization is the posterior aspect of the left ventricle.

Ectopic pacemaker >> slides
Moving to the next topic ...

**ECG**

In German language > EKG

Remember:
- electrical changes of the heart are Depolarization and Repolarization.
- heart consists of two syncytia (atrial and ventricular)

So, we are going to record:
Atrial depolarization and repolarization waves + Ventricular depolarization and repolarization waves.

Note: in Ventricular fibrillation we are talking about single muscle fibers in the ventricle (i.e. every muscle fiber works by itself) > no pumping activity then death.
  MI lead to arrhythmia that can cause fibrillation.

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Monophasic potential: Cardiac muscle action potential
we put one electrode inside the cardiac cell and the other outside (we have phase 0,1,2,3.. all phases in one side > above the line)

Biphasic potential: ECG
Both electrodes on the surface (in one side)
waves could be above or below the line which is > isoelectric line.
Slide 3:

recording the Depolarization wave A,B and Repolarization wave C,D

- When the membrane is depolarized, there is no potential difference (because both electrodes on the same electrical level).
- A: There is a depolarization wave (-ve outside and +ve inside) so, there is a potential difference that progress until reaching midway >> maximum potential difference.
  - then we proceed beyond the midway so, potential difference will decrease (pointer goes back)
- Until reaching complete depolarization (no potential difference) >> B
  This is the plateau..
- C: Repolarization starts (pointer moves in the opposite direction) and progress until reaching midway >> maximum potential difference but opposite ...
- then reaching complete repolarization >> D

International Standards:
** Depolarization wave > Above isoelectric line.
** Repolarization wave > Below isoelectric line.
Normal ECG:

**P wave**: atrial depolarization

**QRS complex**: ventricular depolarization

**T wave**: ventricular repolarization

Notes:
1. Ventricular depolarization occurs at the same time of atrial repolarization, and because the ventricle is too much muscular >> too much electricity, its depolarization sparks over atrial repolarization .. and don’t be shown on the graph.

**In AV block ..**

It might be shown up ... ( P and QRS are far away from each other )

2. T wave > even it is a repolarization wave, but it is an upward reflection .. WHY? The repolarization starts from the other end of the muscle.

Explanation .. Reverse area of repolarization:

In the heart depolarization goes from endocardium to epicardium and from base to apex .. while repolarization goes from epicardium to endocardium and from apex to base .. WHY?

*remember that depolarization usually followed by contraction and repolarization followed by relaxation. So when depolarization occurs in the ventricle and followed by systole >> the intracardiac pressure will increase particularly on the endocardium and this delays the repolarization in it (by changing electrical environment) .. this delay happens to the extent epicardial repolarization occurs first .. ( i.e. endocardial action potential is longer than the epicardial one ).

Don’t forget to refer to the slides.

Best of luck .. Hadeel Abudari 😊