

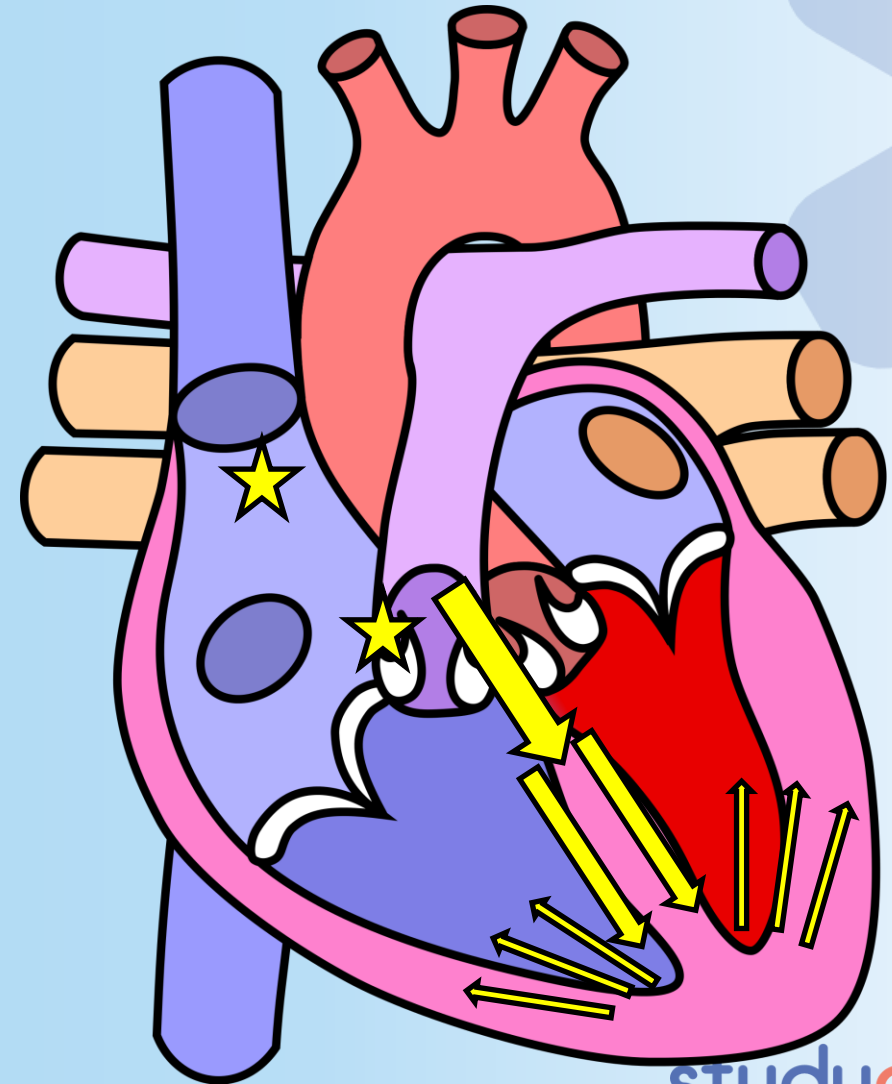
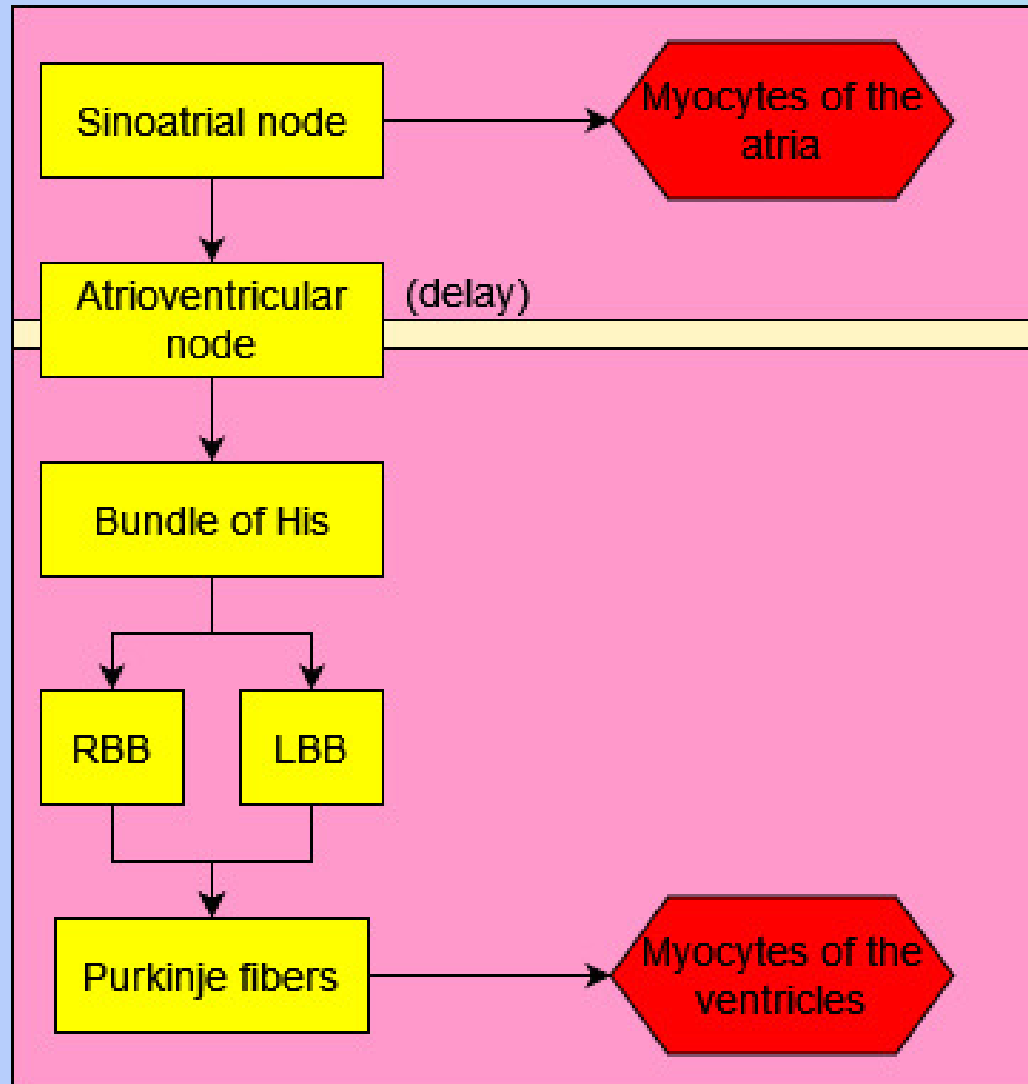
Electrical Activity of the Heart

By Jakub Staniszewski




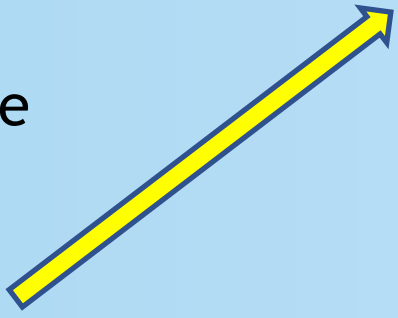
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Anatomy of the Conduction System



Sequence of Conduction & New Questions

1. SA node initiates an action potential.  How is this AP generated?
2. The signal quickly spreads through the atrial muscle.  How can it spread quickly through muscle?
3. The signal arrives at the AV node, and the speed of conduction slows.  How can conduction speed change?
4. The AV node conducts the signal to the bundle of His.
5. The signal continues rapidly down the Purkinje fibers. 
6. The ventricles get the signal and contract.



Unique Properties of Nodal Cells

Automaticity

- SA node cells spontaneously depolarize to threshold, without outside stimulation.
- This creates an AP up to 100 times per minute (intrinsic rhythm).
- AV node cells also spontaneously depolarize, but their intrinsic rhythm is 40-60/minute.

Conduction

- The AV node conducts signals very slowly.
- This is due to narrow fibers, few gap junctions, and a **slow rate of depolarization****.
- Purkinje fibers are wide, have many gap junctions, and depolarize rapidly.

Gap junctions are found in both myocytes and nodal cells. They allow cell-to-cell conduction.

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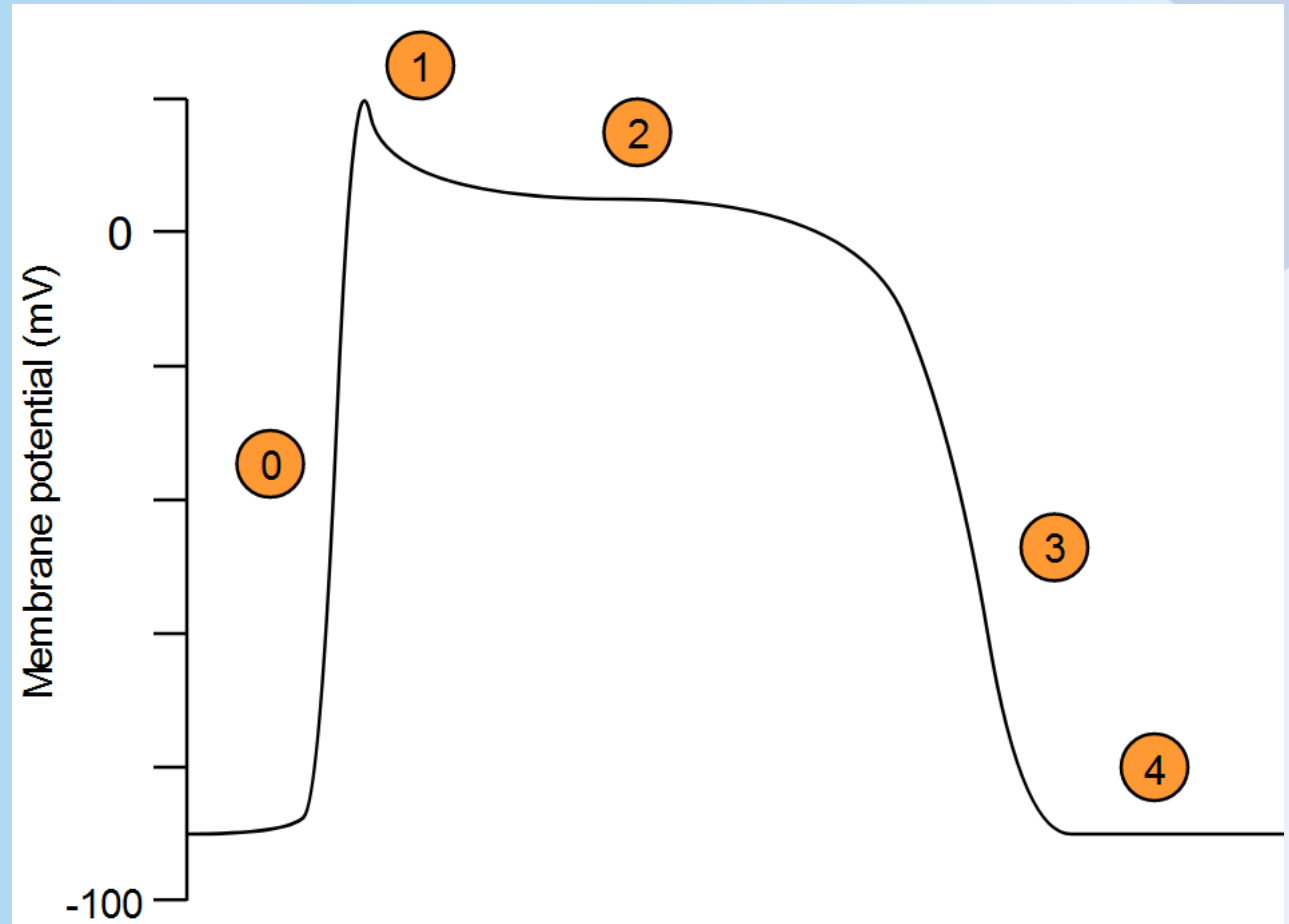
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AP arrives and depolarizes to threshold potential

Non-nodal action potential

Myocytes and Purkinje cells

- 0 Upstroke and depolarization
voltage-gated Na^+ channels
- 1 Early repolarization
transient K^+ channels (ungated K^+ leak)
- 2 Plateau phase
voltage-gated L-type Ca^{2+} channels
- 3 Rapid repolarization
delayed rectifier K^+ channels
- 4 Resting phase
inward rectifier K^+ channels



Steep phase 0 = fast rate of conduction

Non-nodal action potential

Myocytes and Purkinje cells

0 Upstroke and depolarization
voltage-gated Na⁺ channels

1 Early repolarization
transient K⁺ channels

2 Plateau phase
voltage-gated L-type Ca²⁺ channels

3 Rapid repolarization
delayed rectifier K⁺ channels

4 Resting phase (-90 mV)
inward rectifier K⁺ channels

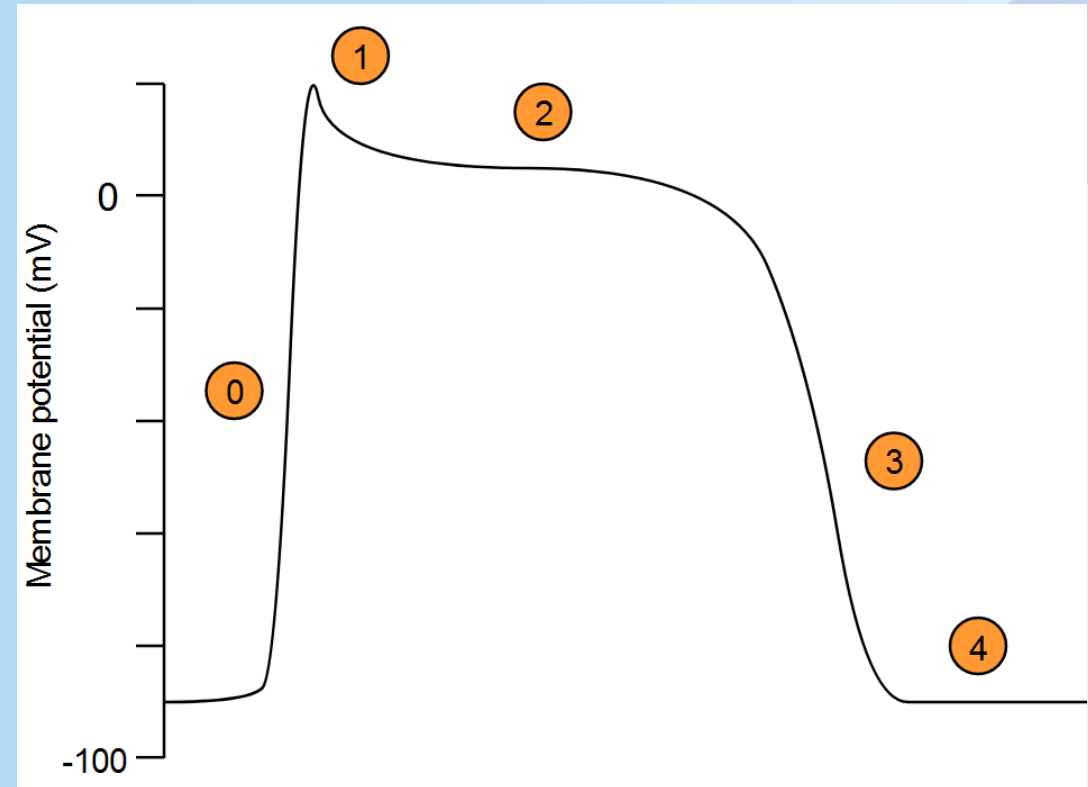
	↑	↑	↓
	Sodium (Na)	Calcium (Ca)	Potassium (K)
0	At -65mV, massive Na ⁺ influx.	-	Ungated K ⁺ leak
1	Na ⁺ channels inactivated.	-	K ⁺ efflux
2	-	Ca ²⁺ influx	K ⁺ efflux
3	-	Ca ²⁺ channels inactivated	K ⁺ efflux
4	-	-	K ⁺ at equilibrium potential (-90mV)

Effect on hemodynamics

A long plateau phase:

- Allows enough time for the ventricles to empty during systole.
- Prevents re-excitation of the muscle before the next beat (refractory period)

The plateau phase is longer than systole. The heart muscle can completely relax before the next contraction occurs.



Other important channels in myocytes

Ryanodine receptor → opens in response to Ca^{2+} binding, and releases more Ca^{2+} into the cytoplasm from the SR (Ca^{2+} induced Ca^{2+} release).

- This channel is responsible for triggering muscle contraction.

SERCA → pumps Ca^{2+} back into the sarcoplasmic reticulum during the plateau phase.

- This pump ensures that the heart muscle is ready for the next contraction. Allows the heart muscle to relax!

$\text{Na}^+/\text{Ca}^{2+}$ exchanger → pumps Ca^{2+} into the extracellular space.

- This pump ensures that the myocyte/Purkinje cell is ready for the next AP.

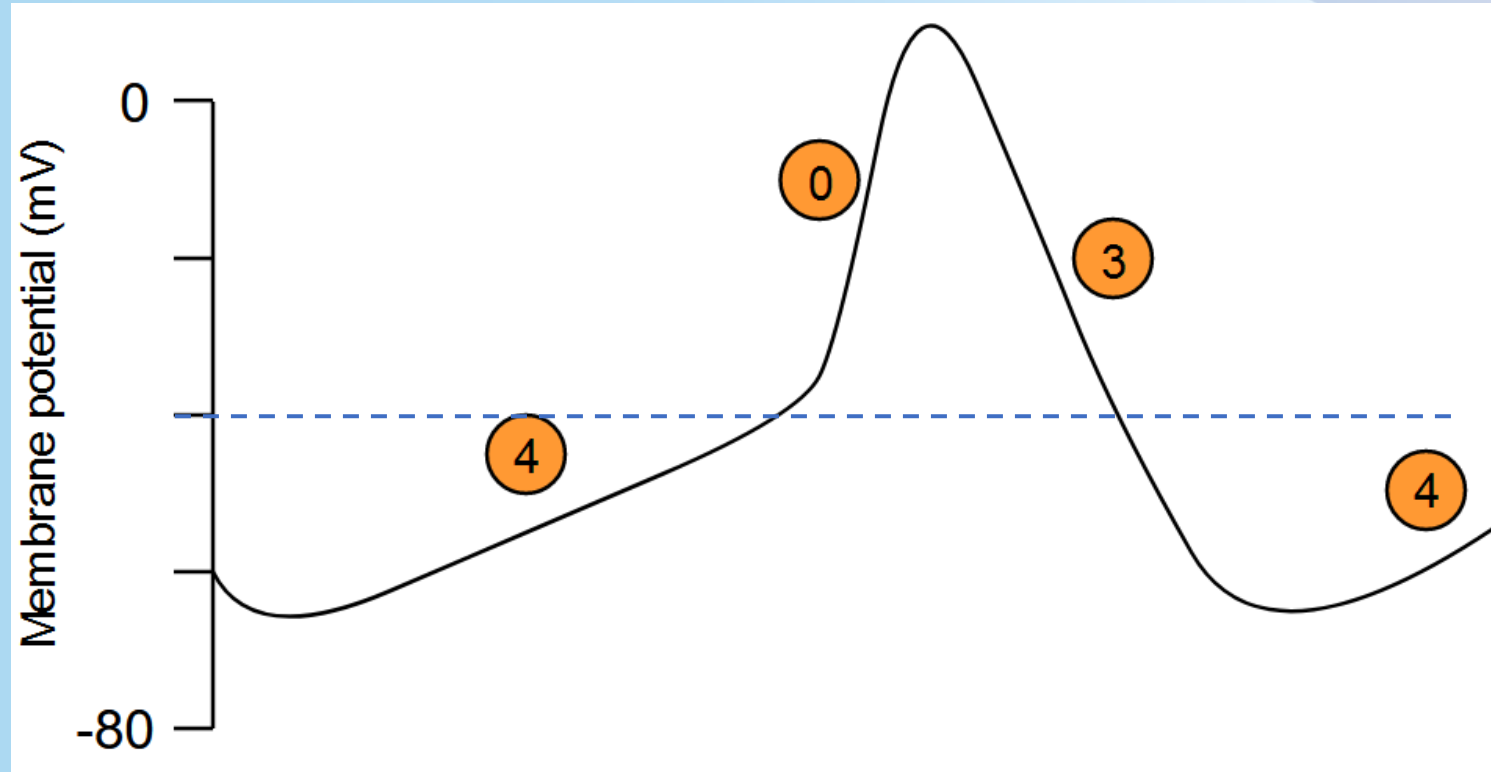
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Nodal action potential

SA and AV nodes

- 4 Slow spontaneous depolarization
funny Na^+ channels / T-type Ca^{2+} channels
- 0 True depolarization
voltage-gated L-type Ca^{2+} channels.
- 3 Repolarization
delayed rectifier K^+ channels
- 4 Slow spontaneous depolarization
funny Na^+ channels



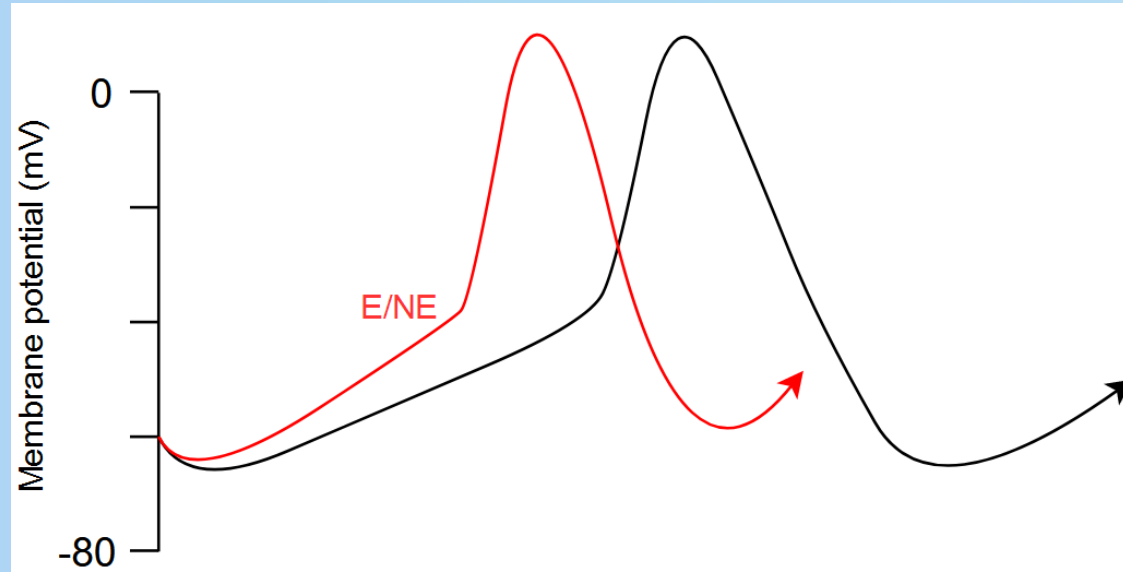
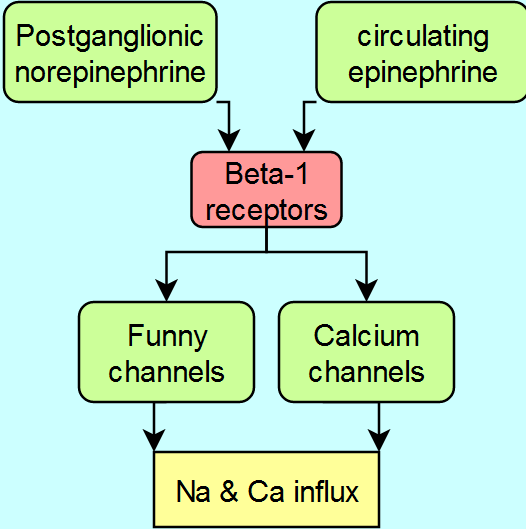
Note that phase 0 is not as steep as in the myocyte!

Nodal action potential

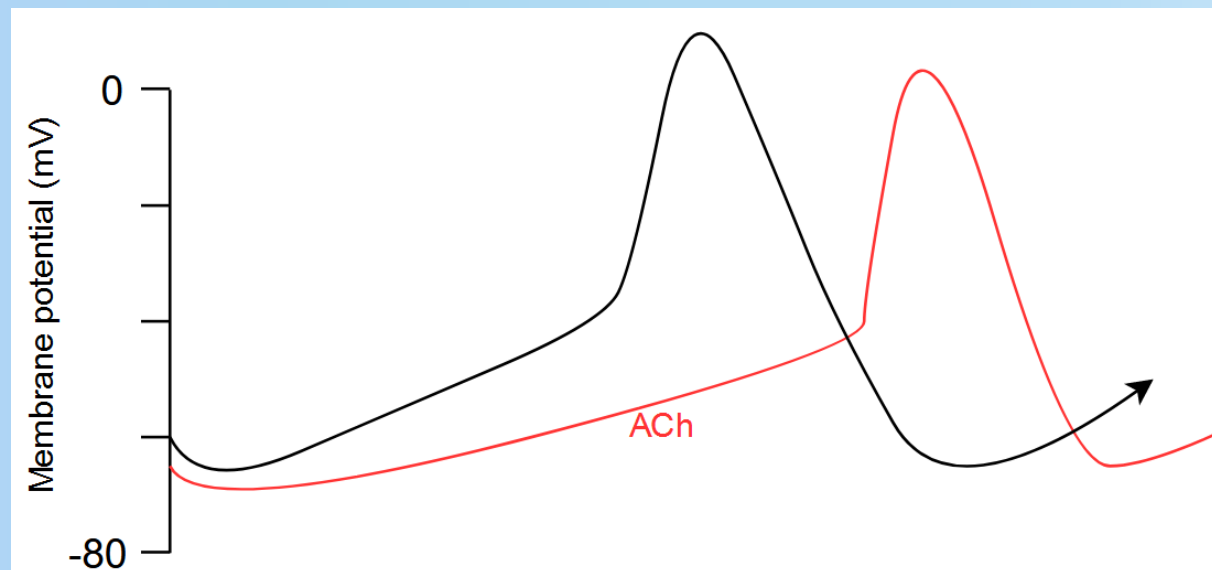
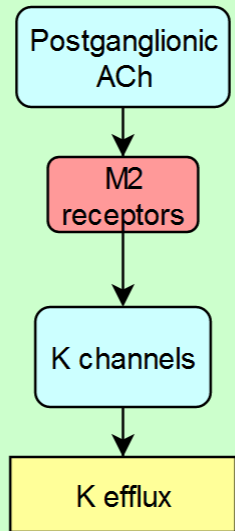
SA and AV nodes

	Sodium (Na)	Calcium (Ca)	Potassium (K)
4 Slow spontaneous depolarization funny Na ⁺ channels / T-type Ca ²⁺ channels	<u>Na⁺ funny channels are open</u>	T-type Ca ²⁺ channels are open	Slight K ⁺ efflux
0 True depolarization voltage-gated L-type Ca ²⁺ channels.	Na ⁺ funny channels closed	<u>Ca²⁺ influx</u>	-
3 Repolarization delayed rectifier K ⁺ channels	-	Ca ²⁺ channels closed	K ⁺ efflux

Autonomic control of nodal tissue



- Greater influx of Na^+ and Ca^{2+}
- Threshold reached sooner
- Shorter phase 4
- **Increased heart rate**



- Greater efflux of K^+
- Threshold reached later
- Longer phase 4
- **Decreased heart rate**

To prove to your family that you are, indeed, learning medicine.

Aka Clinical correlate

What effect would a K^+ channel blocker have on the non-nodal AP?



K^+ efflux is responsible for repolarization



Repolarization is delayed
Plateau phase is prolonged

K^+ channel blockers prolong the refractory period, making them effective anti-arrhythmic drugs.

What effect would a T-type Ca^{2+} channel blocker have on the nodal AP?



Ca^{2+} influx is partly responsible for spontaneous depolarization



Takes longer to reach threshold
SA node rate is slowed

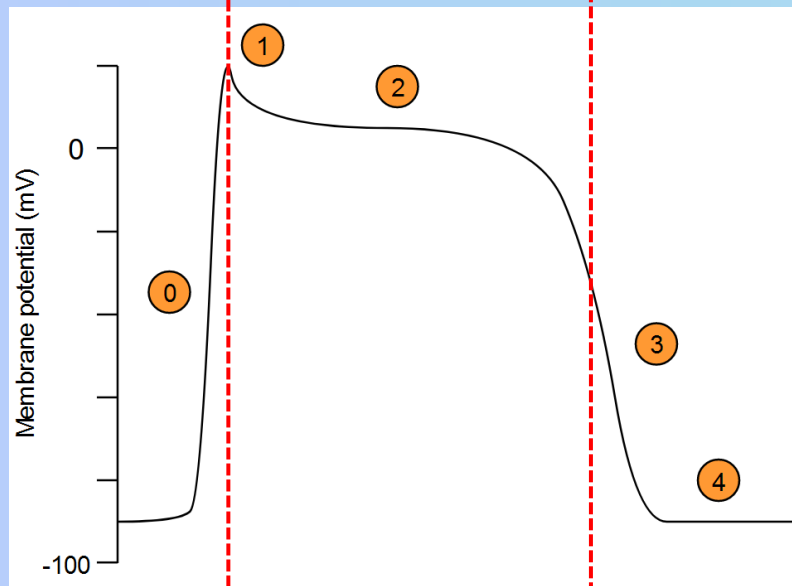
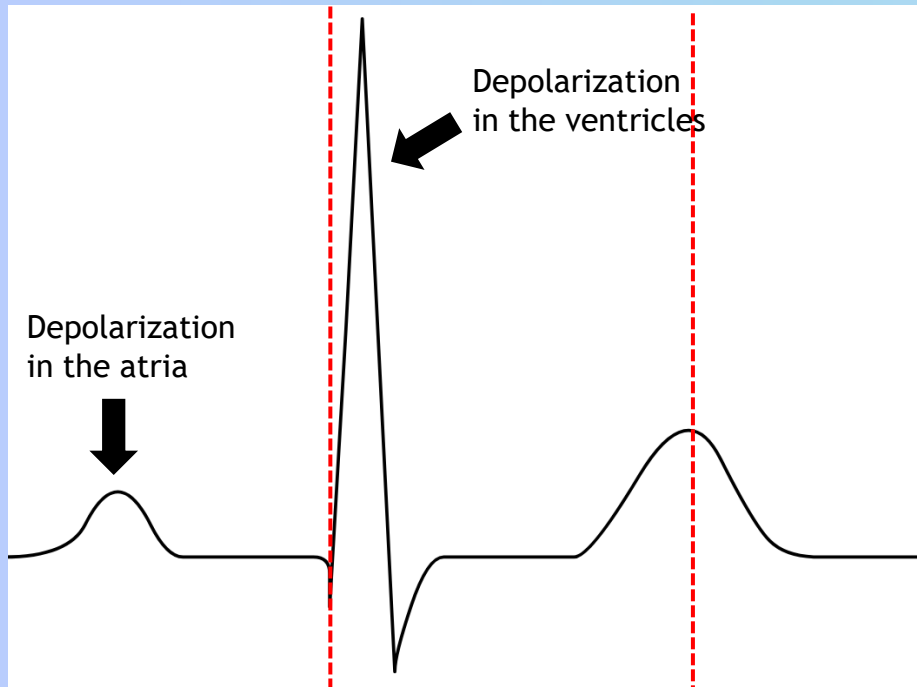
Ca^{2+} channels are everywhere! Slowed HR is only one of many effects that CCBs exert!



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The ECG and the AP



- The ECG is a recording that is taken by placing electrodes on the skin and recording electrical activity.
- The electrical activity is the product of myocardial depolarization and repolarization.
- Phase 0 → ventricular depolarization
- Phase 3 → ventricular repolarization

The AP represents activity at a single point. It cannot be measured clinically.

The ECG represents activity in the entire heart.

ECG Components

P Atrial depolarization, beginning in the SA node.

Atria contract - heart in diastole.

QRS Ventricular depolarization

Ventricles contract - heart in systole.

T Ventricular repolarization

Ventricles are relaxed.

- PR interval corresponds to AV conduction velocity
- QT interval corresponds to length of ventricular AP.

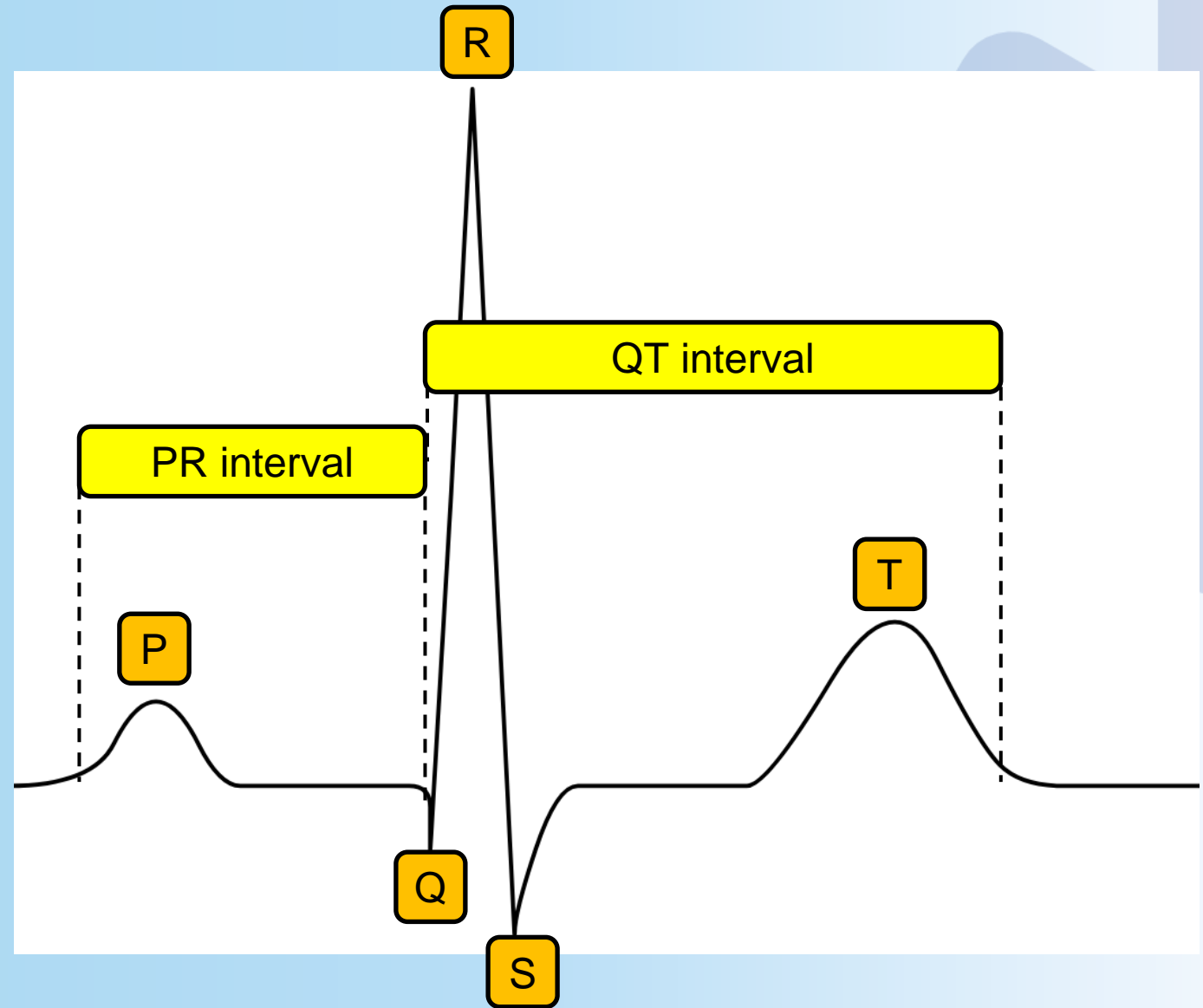


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**Thanks for listening,
it's time for a quiz!**

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