Cardiac Conduction System ECG and Arrythmias'

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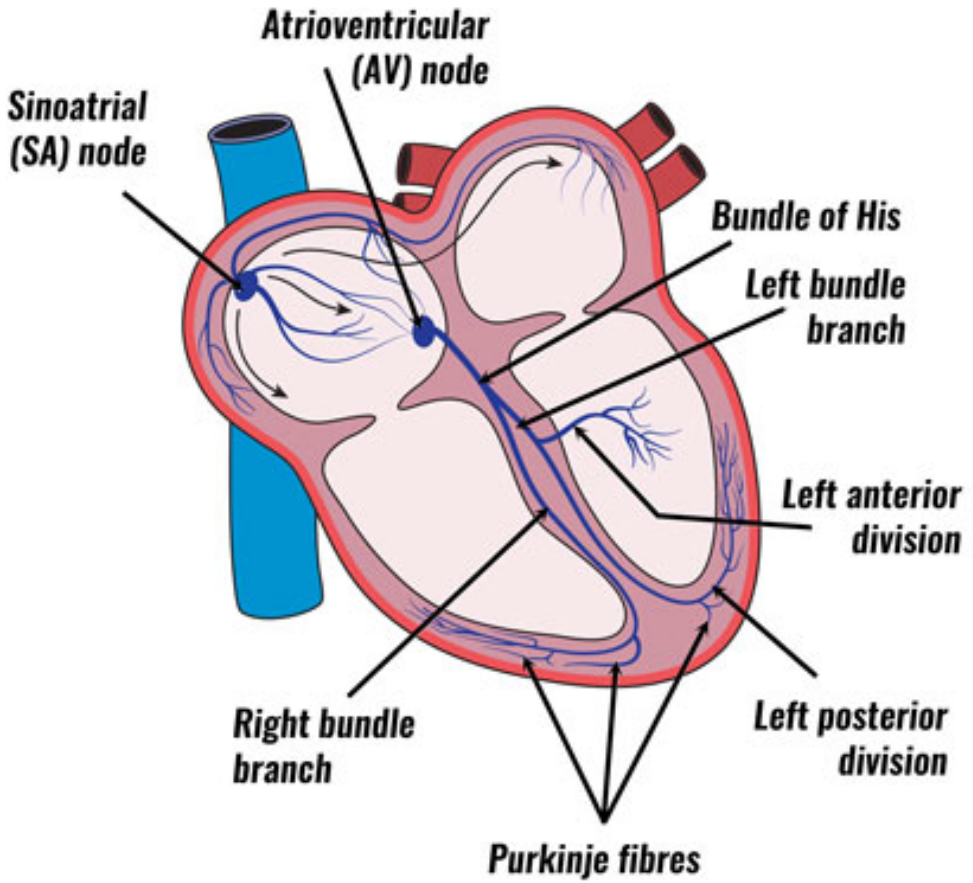
Is an impulse-conducting system that consists of specialized cells that initiate the heart-beat and electrically coordinates contractions of the cardiac chambers

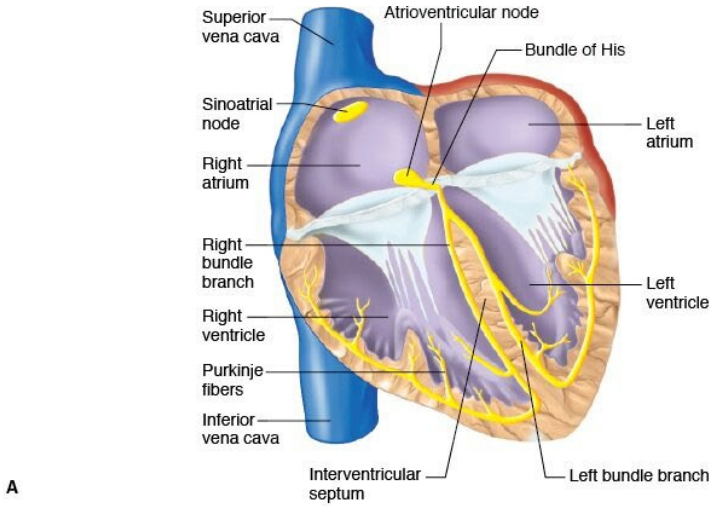
Review

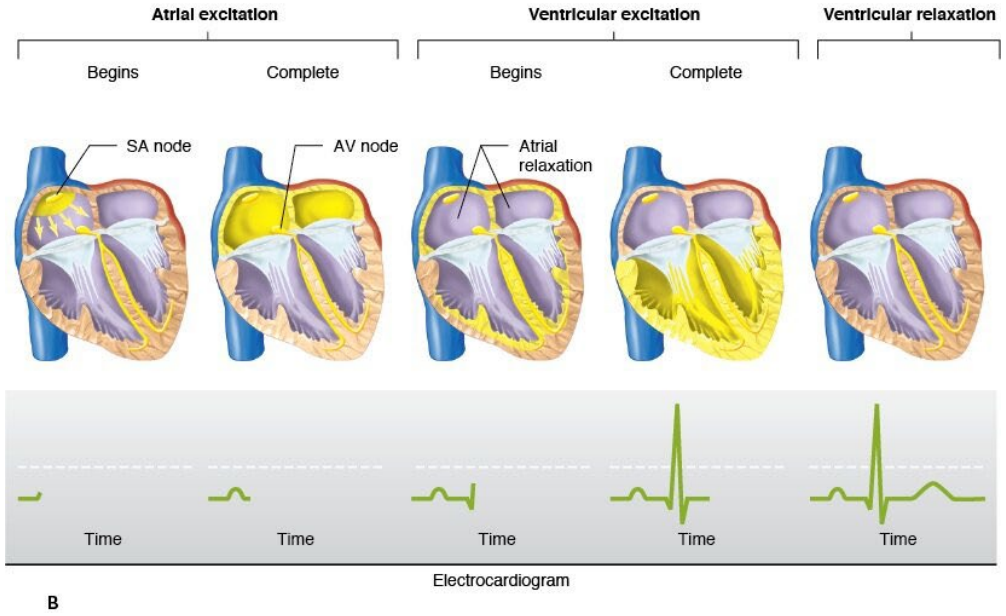
- Basics of electrophysiology
- Action Potential
- The ECG
- Brady arrhythmia’s
- AV Blocks
- Tachy arrhythmia’s
- Ectopics
- STEMI/ NSTEMI



The impulse conduction system consists of four structures

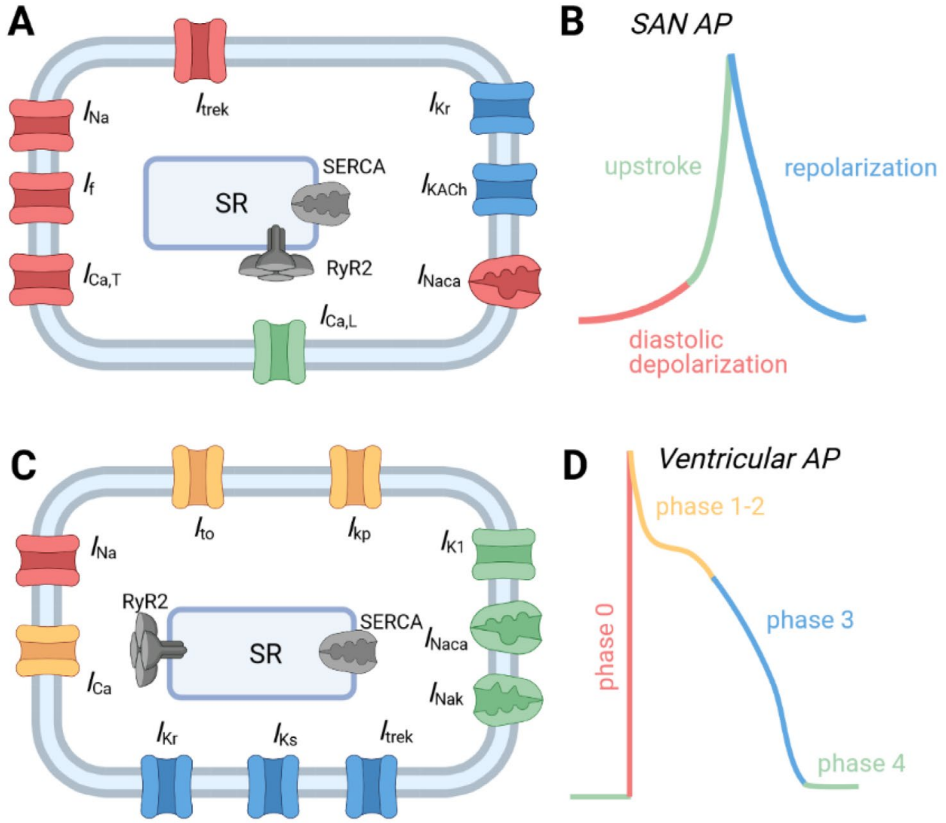
- The sino-atrial node (SA node)
- The atrio-ventricular node (AV node)
- The atrio-ventricular bundle (AV bundle)
- The Purkinje fibers





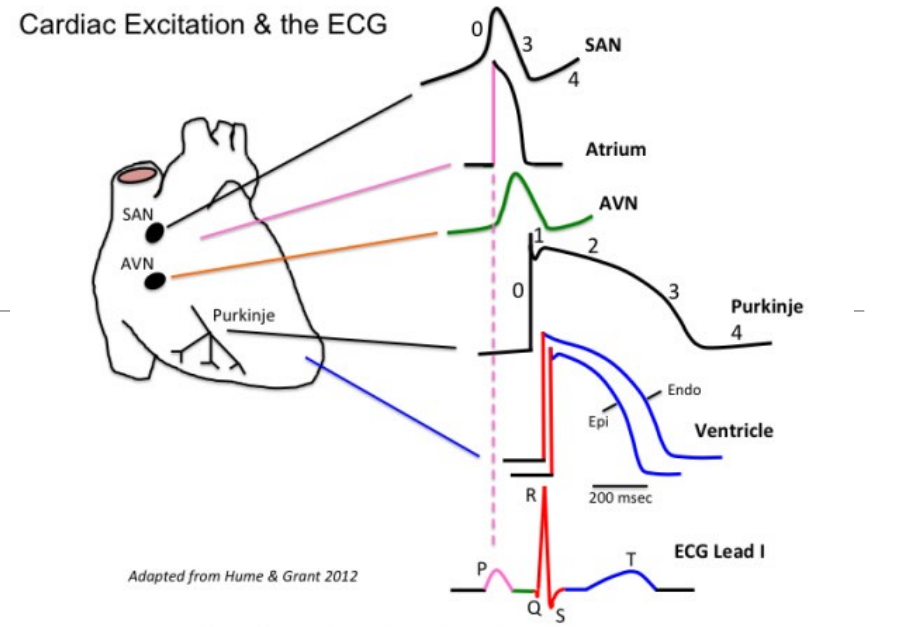
Basics of electrophysiology

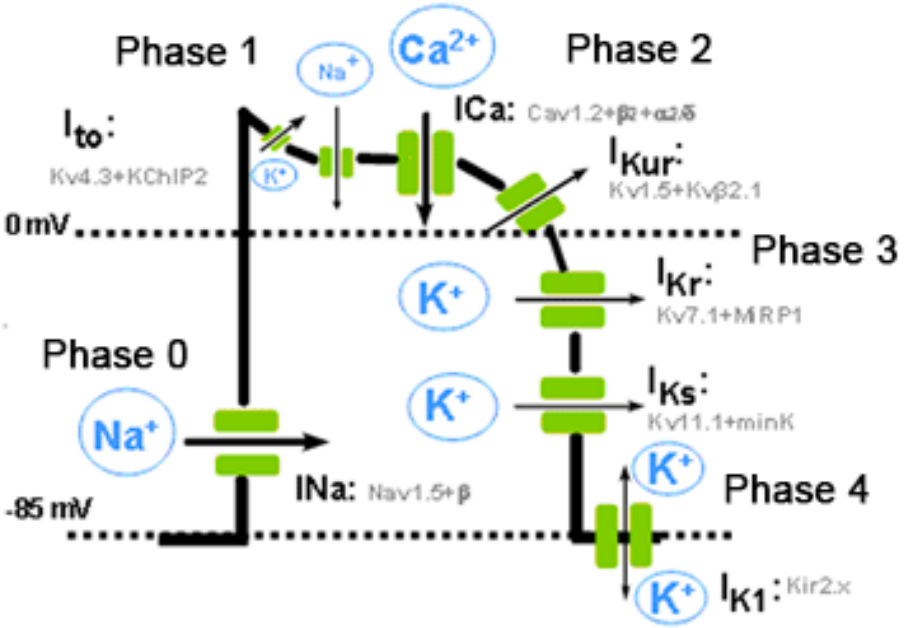
- Ion movement and channels – cardiac cells are separated by a lipid bilayer that is largely impermeable to ions. There are ion channels that maintain the concentration gradients of charge between the inside and outside of cells.
- Resting potential – Is the electrical charge difference between the inside and outside of the cell.



Action potential

- When cardiac cells are stimulated appropriately ion channels open and close in s sequential manner
- Each channel has a characteristic pattern of activation and inactivation that determines the progression of the electrical signal





Action potential phases

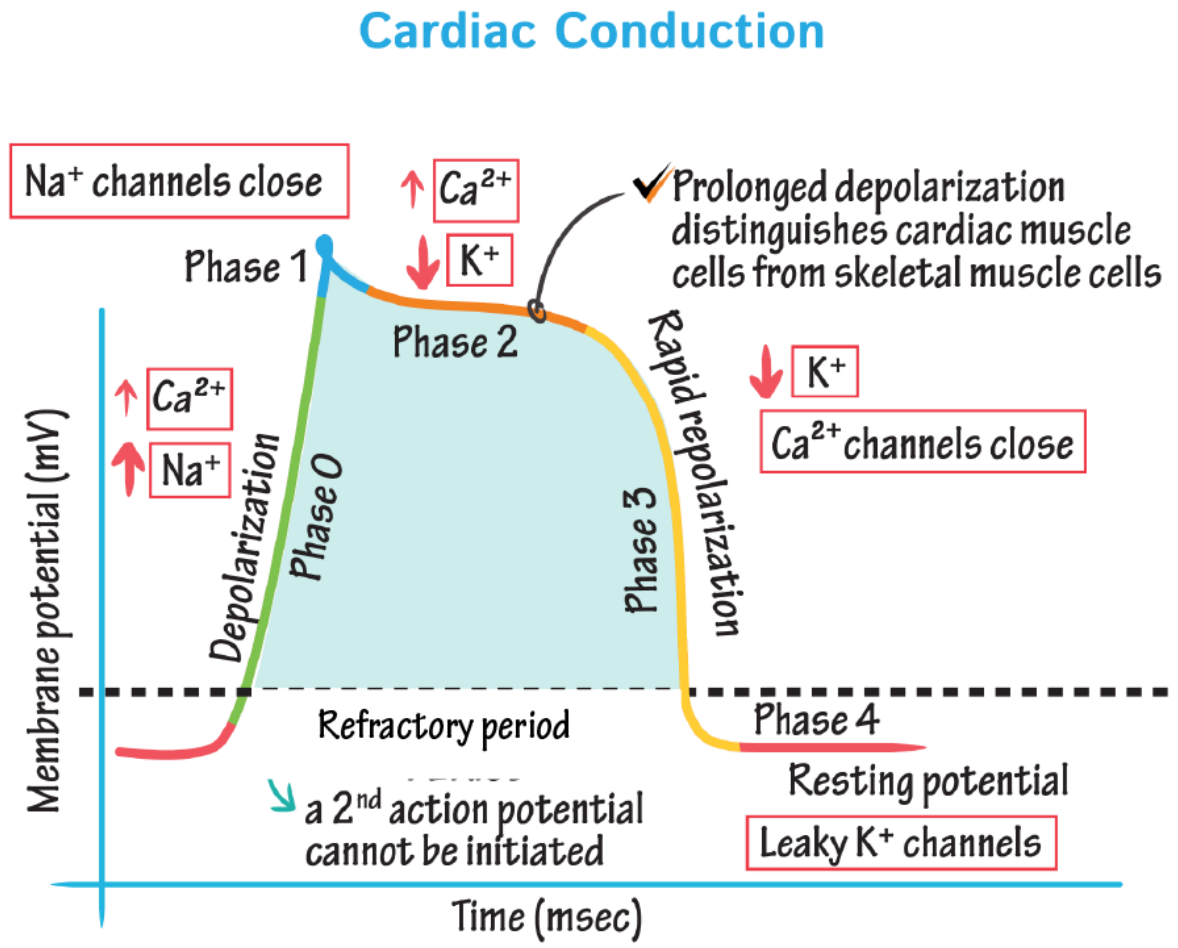
Phase 0 – Depolarization

Phase 1 – Transient repolarization

Phase 2 – Plateau Phase

Phase 3 – Rapid repolarization

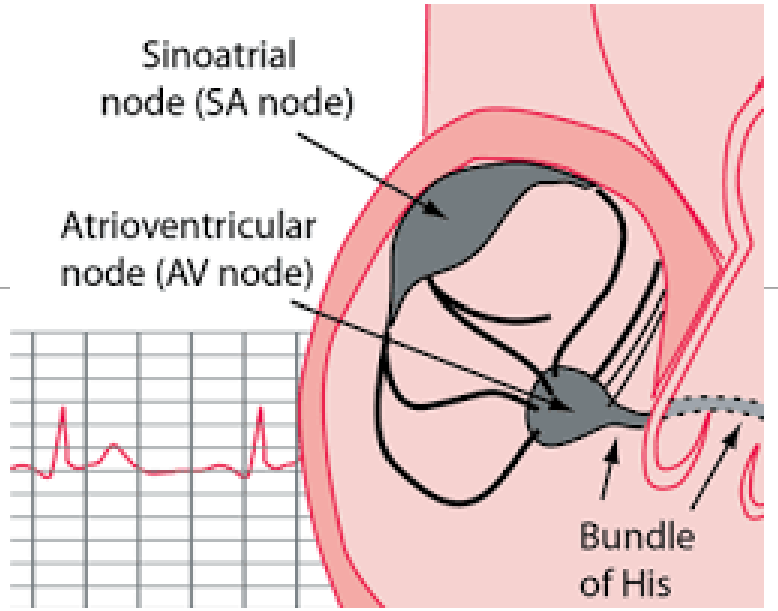
Phase 4 – Diastole/ resting state

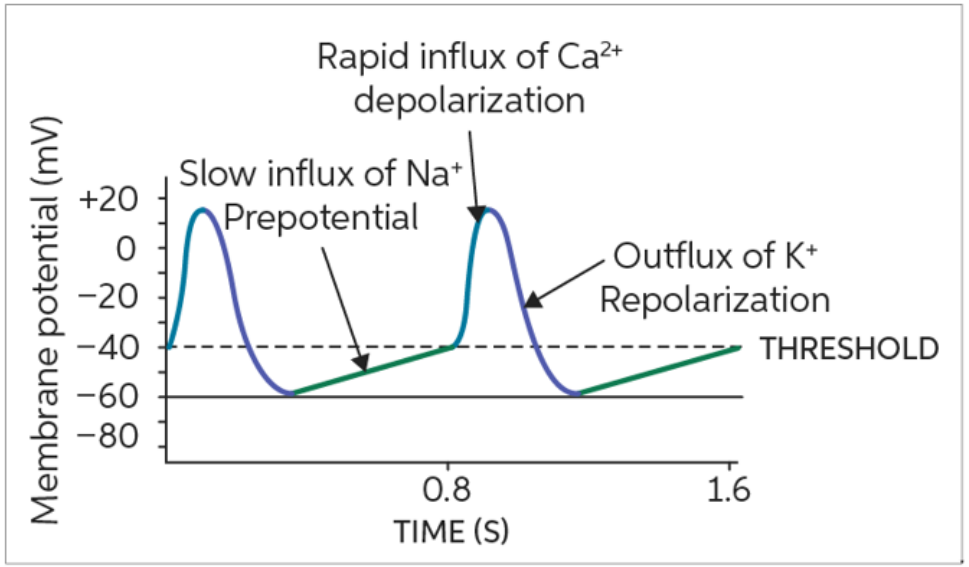


Electrocardiograph

SA Node

- It is located at the junction of the crista terminalis in the upper wall of the right atrium and the opening of the superior vena cava.
- At rest, the SA nodal myocytes depolarize at an intrinsic rate between 60 and 100 beats per minute
- The autonomic nervous system tightly controls input into the sinus node.
- Parasympathetic input slows down the rate of action potential production
- Parasympathetic input is carried via the Vagus nerve
- sympathetic input increases the rate of action potential production, via nor adrenaline.
- Impulses are distributed across to the left atrium via inter nodal tracts, known as Bachmann's bundle.



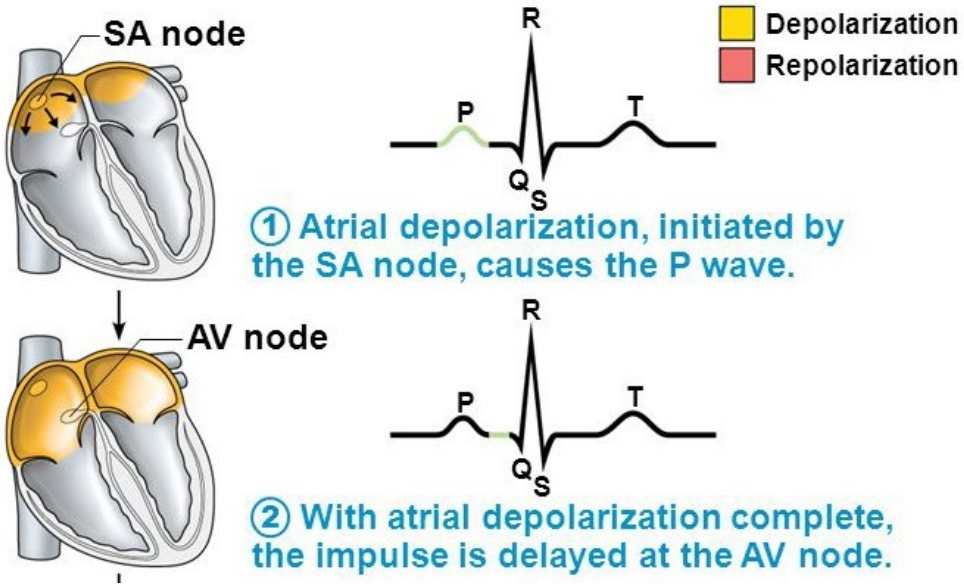


Action potential of SA node

P wave

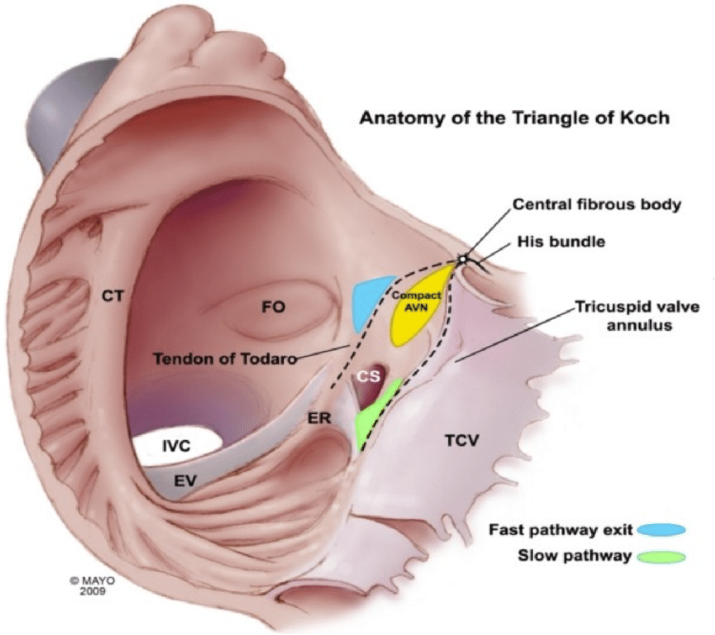
- Depolarization of atrial myocytes result in the “P” wave on the ECG
- The presence of a P wave represent an atrial contraction
- Atria contract forcing blood into the ventricles

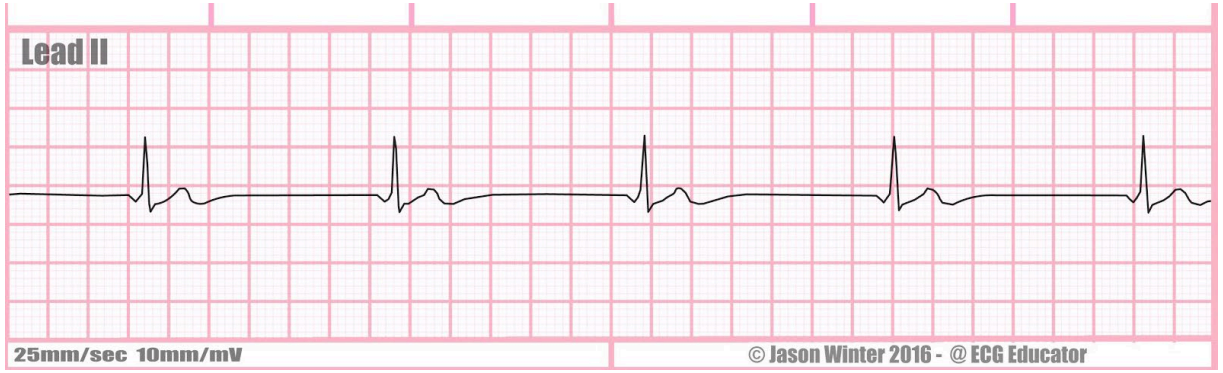
- Normative values
- P wave – upright in lead I, II
- <120 ms width
- <2.5 mm tall
- Inverted in avR
- V1 – biphasic
- PR interval – is the time between when the impulse travels from the atria to the AV node, allowing the ventricles to fill with blood
- > 120 ms – 200 ms



AV node

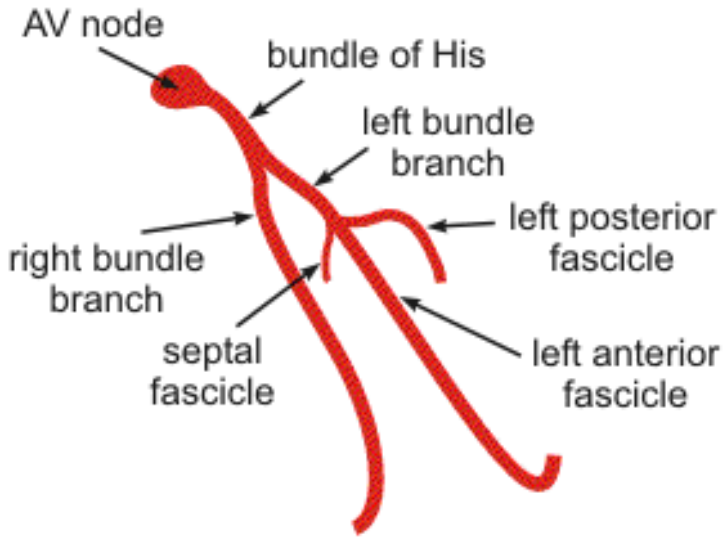
- Compact node , located at the apex of the Triangle of Koch.
- Made up of a cluster of cells, that can collect impulses and funnel it towards the His Purkinje system
- AV node has the ability to slow impulses
- AV node can also function as a secondary pacemaker
- AV node generates impulses at the rate of 40-60 bpm
- Affected by autonomic tone

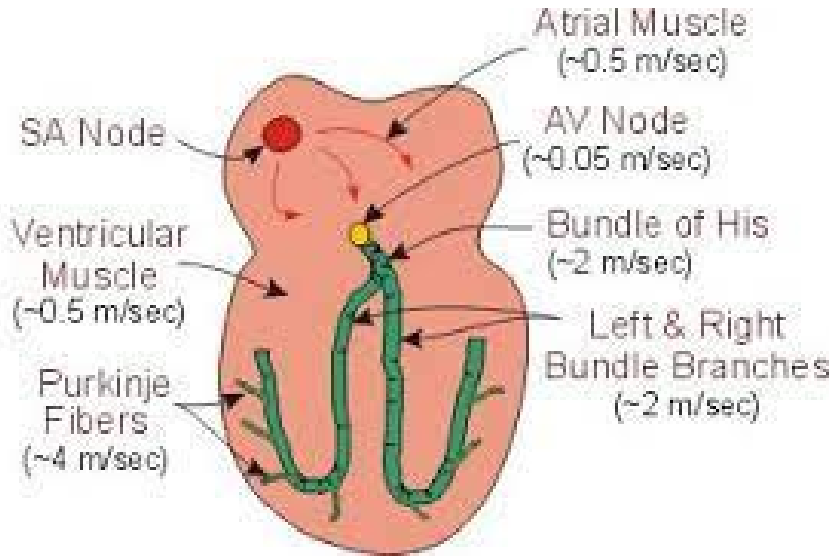




Bundle of HIS

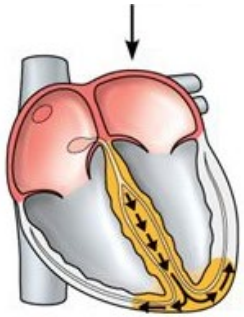
- Located between the atria and the ventricles
- Fibrous sheath located between the atria and ventricles
- The bundle of His consists of wide, conducting muscle fibers that carry the cardiac impulse through the insulating annulus fibrosus into the fibrous upper part of the ventricular septum.
- It then bifurcates into the left and right bundle
- Can conduct impulses at faster rates than myocytes
- PR interval is representative of HIS BUNDLE conduction
- Right and Left Bundles
- Runs along the septum
- RBB – thin , long and branches into Purkinje fibers
- LBB – thick and branches into left anterior fascicle and left posterior fascicle.
- This allows coordinated contraction of the RV and LV.

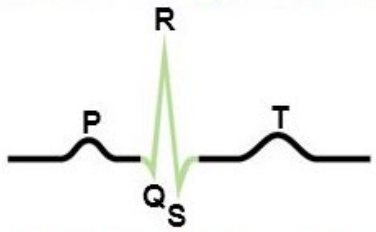




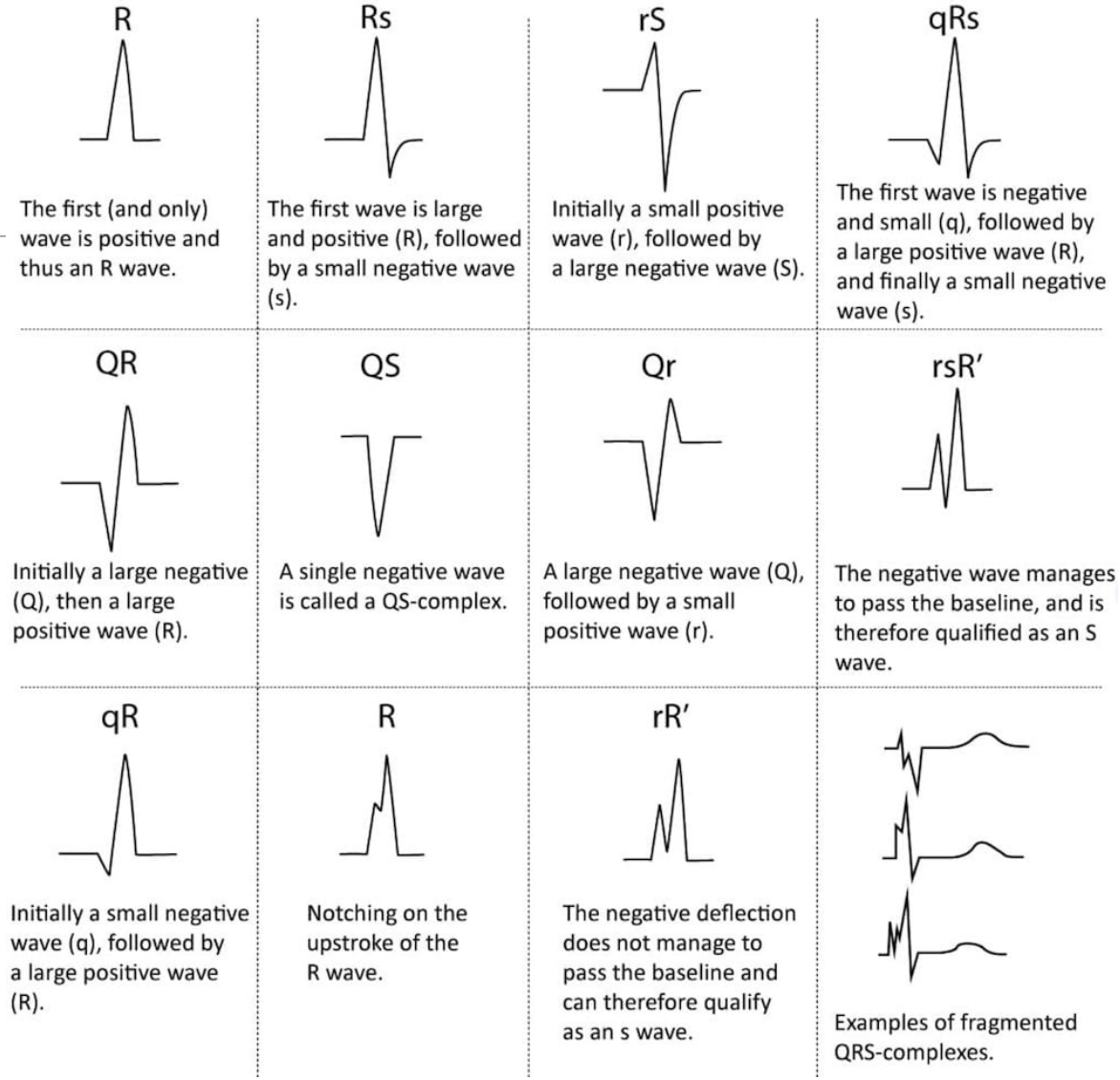
QRS Complex

- After the impulse reaches the AV node, it travels to the Purkinje system via the HIS bundle
- This results in ventricular contraction
- This is much larger wave form and it depolarizes a larger amount of muscle tissue in comparison to the atria and so higher voltages are involved.
- 100 ms , abnormal if > 120 ms.





③ AV node depolarizes; Ventricular depolarization begins at apex, causing the QRS complex.
Atrial repolarization occurs.



ST Segment

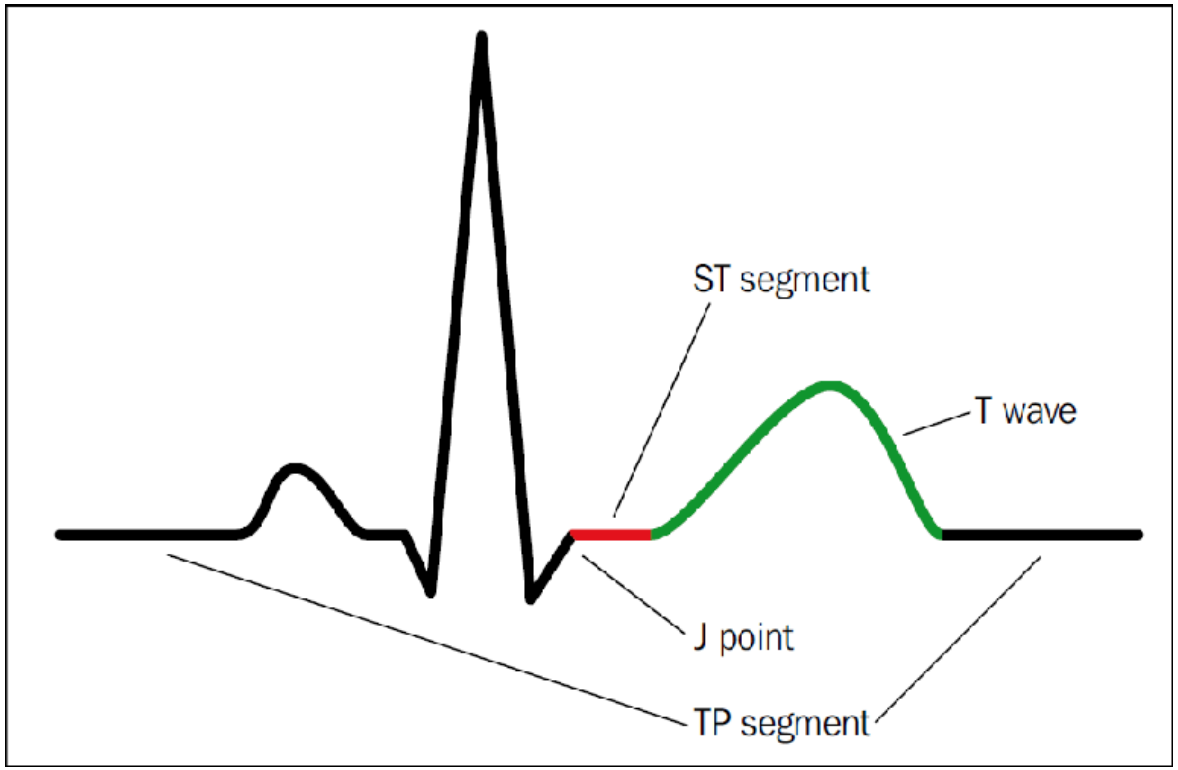
- Measured from the offset of the QRS complex to the onset of the T wave
- Time between ventricular depolarization and repolarization
- Isoelectric

J point

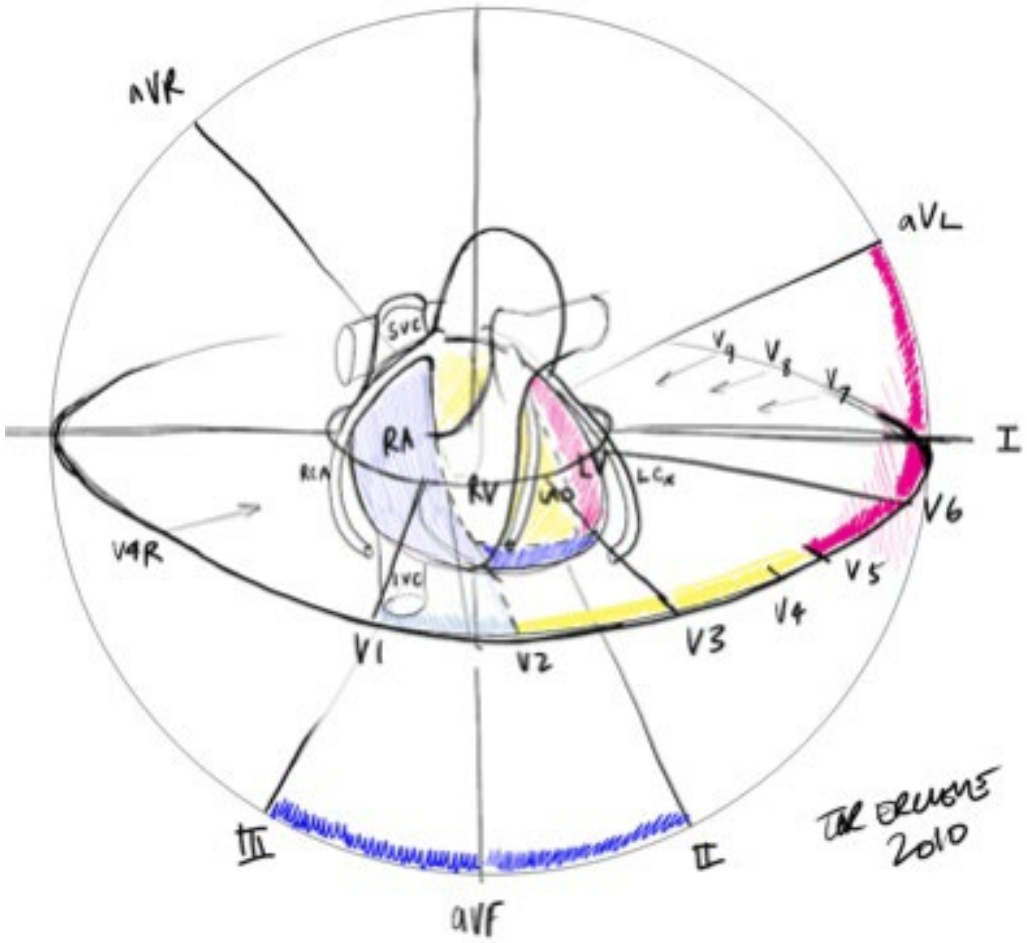
- Where the QRS complex meets the ST segment

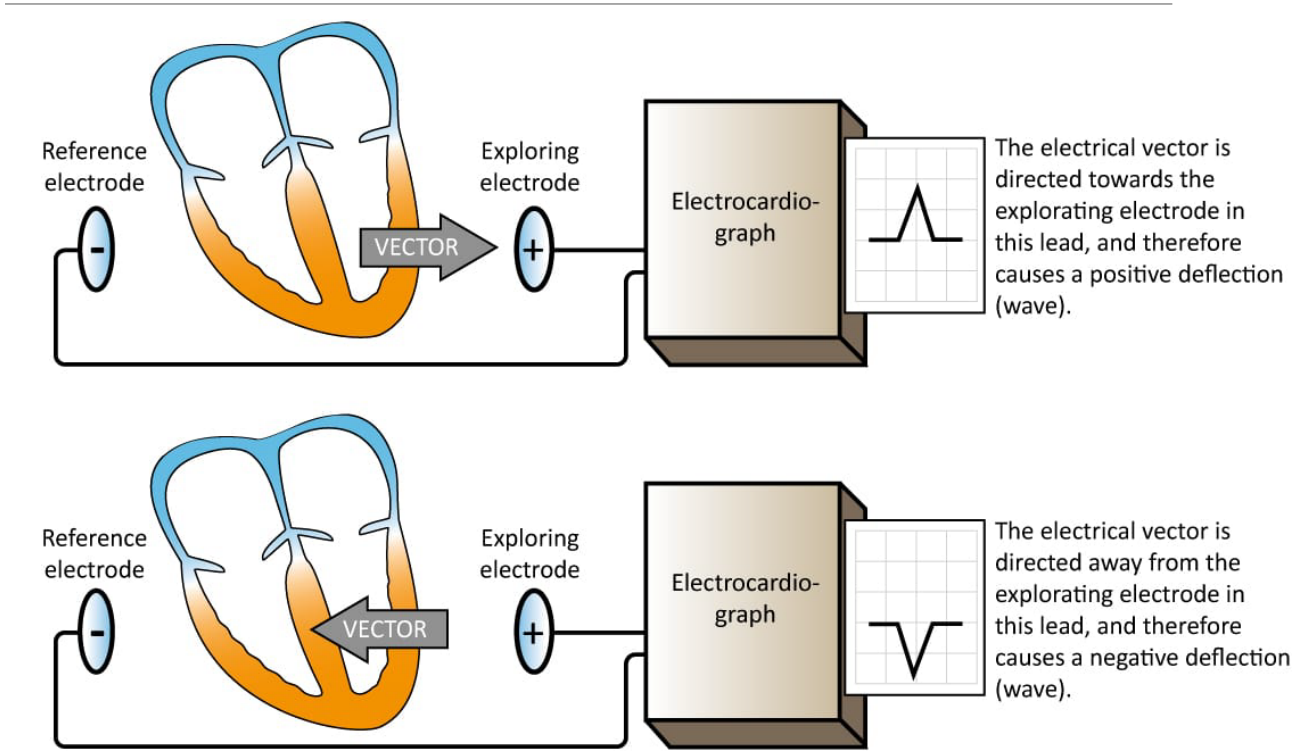
T wave

- Ventricular repolarization
- Phase 3 / 4 action potential
- Important for assessing Ischemia and electrolyte abnormalities
- Positive in most leads except , avR, v1 , III
- Amplitude < 5 mm in limb leads and < 10 mm in precordial leads



ECG AXIS


aVR
aVL
I
V6
V5
V4
V3
V2
V1
V4R
II
III
aVF
SVC
IVC
RA
RV
LV
2010


Reference electrode
Exploring electrode
VECTOR
Electrocardio-graph
The electrical vector is directed towards the exploring electrode in this lead, and therefore causes a positive deflection (wave).
Reference electrode
Exploring electrode
VECTOR
Electrocardio-graph
The electrical vector is directed away from the exploring electrode in this lead, and therefore causes a negative deflection (wave).

Bradyarrythmia’s

SA dysfunction/ Sick sinus syndrome

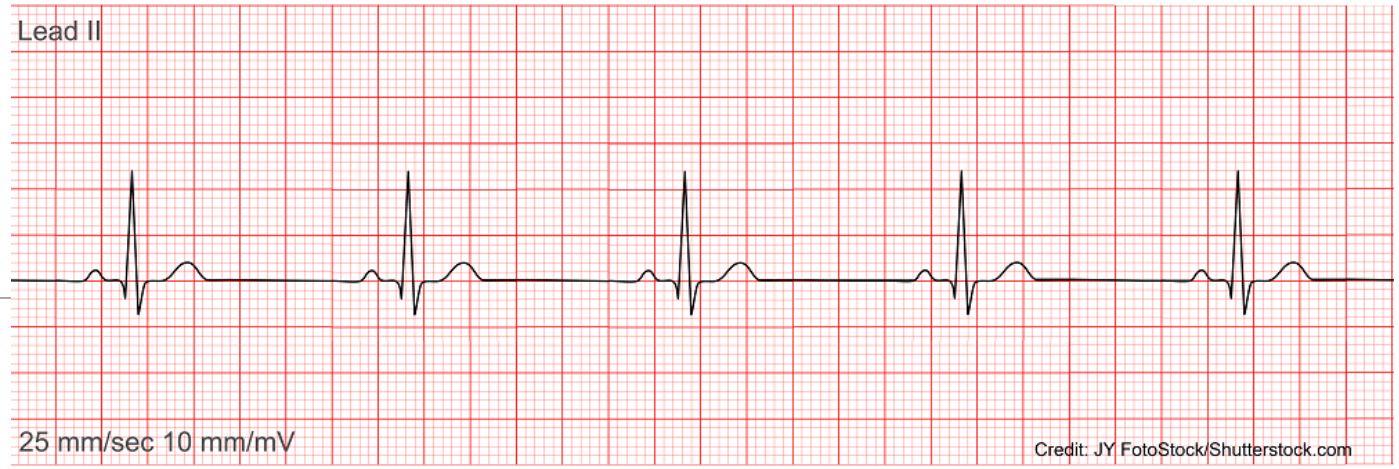
- Bradycardia
- Sinus pauses
- Sinus arrythmia

AV conduction abnormality

- 1 deg AV Block
- 2 deg AV Block
- 3 deg/ complete AV Block

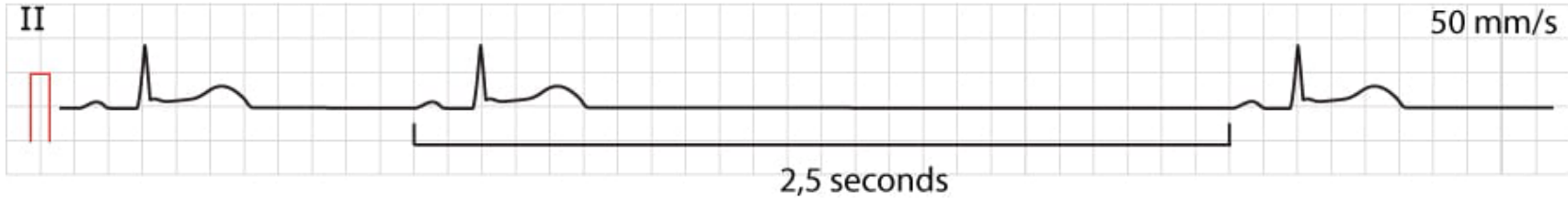
Sinus brady

- HR < 60 bpm
- ECG feature, not a diagnosis



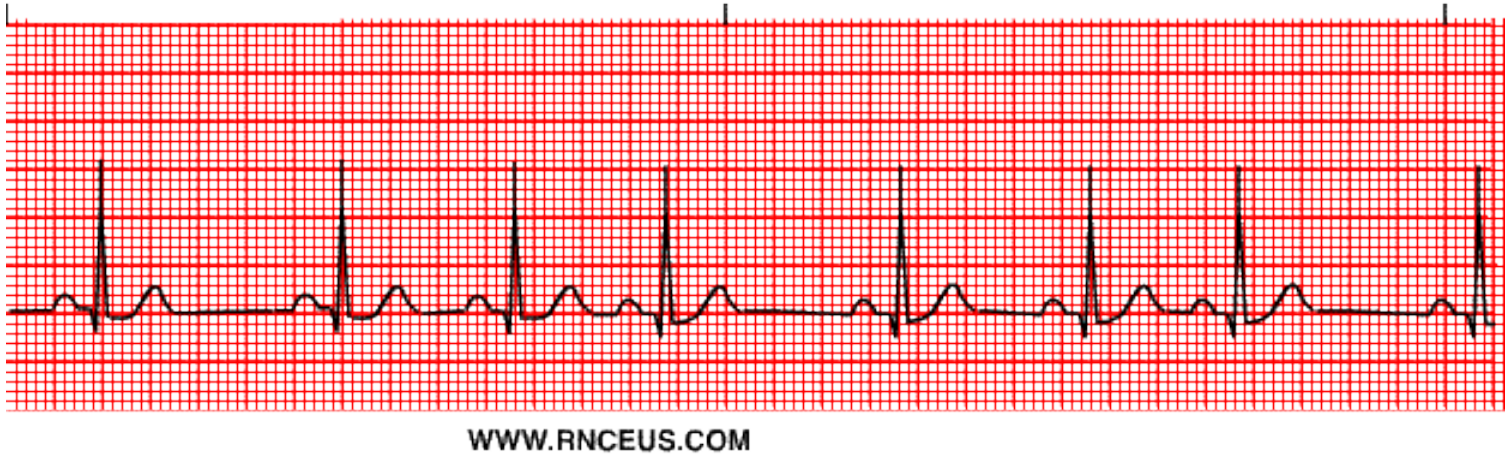
Sinus Pause

- Usually > 2 secs
- Failure of SA node to generate action potential



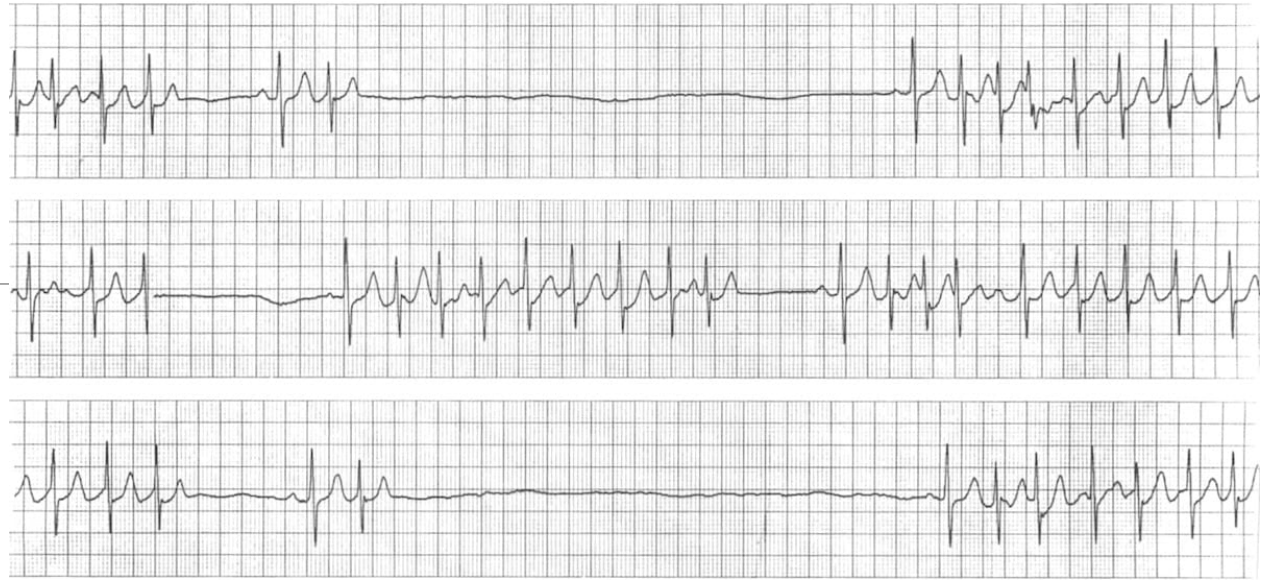
Sinus arrythmia

- Beat to beat variation in P-P interval
- All generated by the SA node , irregular



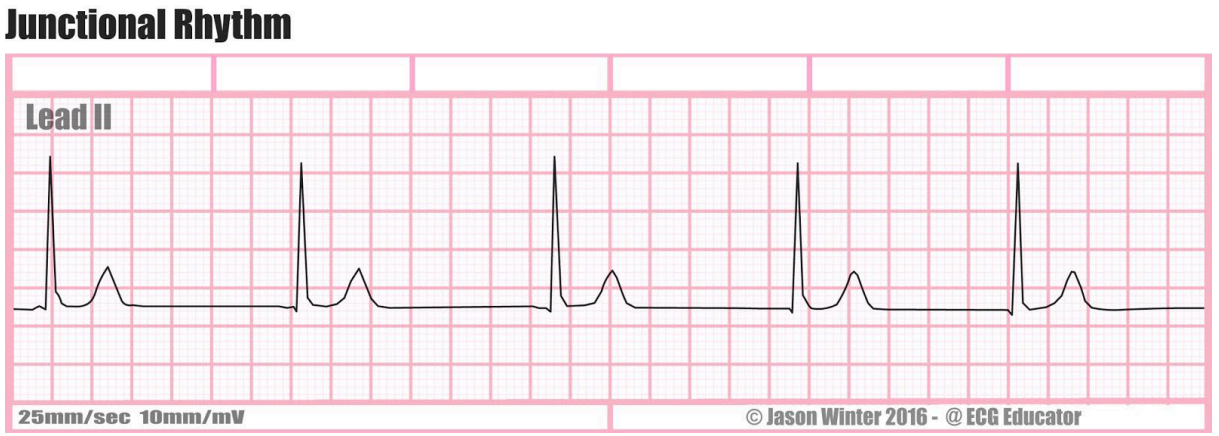
Tachy/ Brady syndrome

- bradycardia alternating with paroxysmal supraventricular arrhythmias, most frequently atrial fibrillation (AF)
- Patients are symptomatic to the change in heart rate



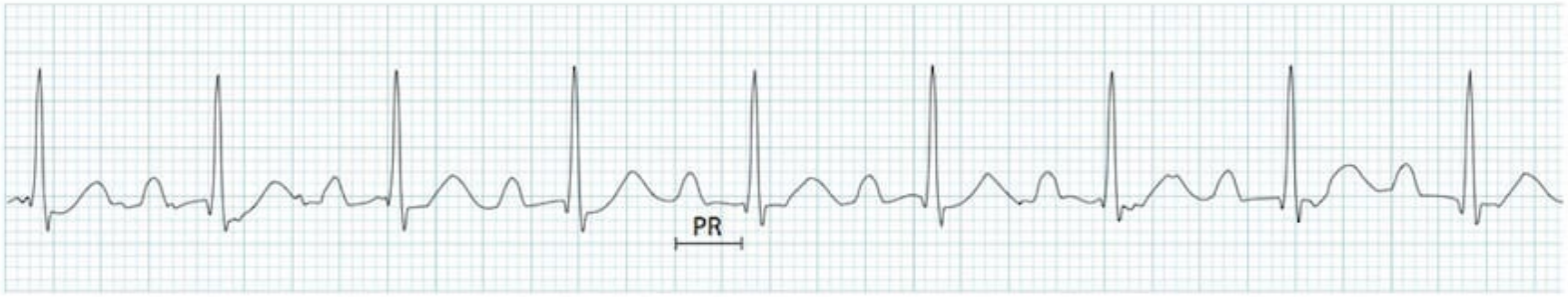
Junctional Rhythm

- Lack of P waves
- Regular R-R intervals , narrow complex QRS
- AV node is the pacemaker
- 40-60 bpm (rate of AV node)



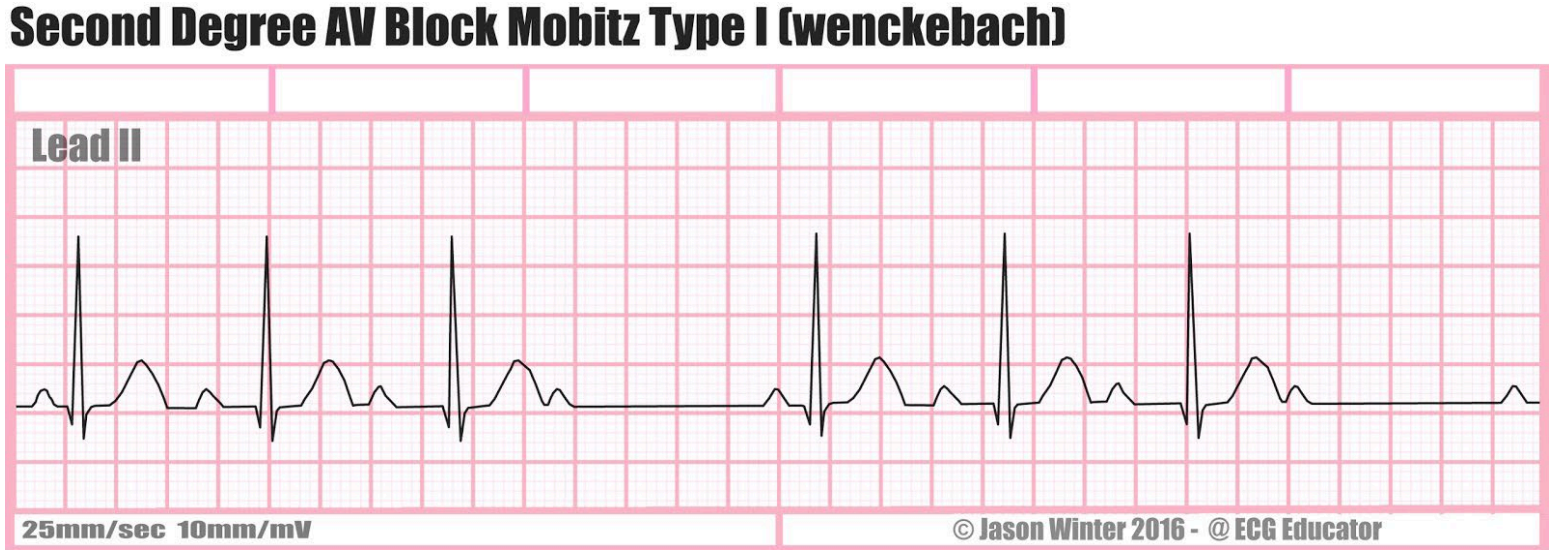
1ST degree AV block

- PR interval >200 ms
- Rarely symptomatic
- Usually improve on exercise



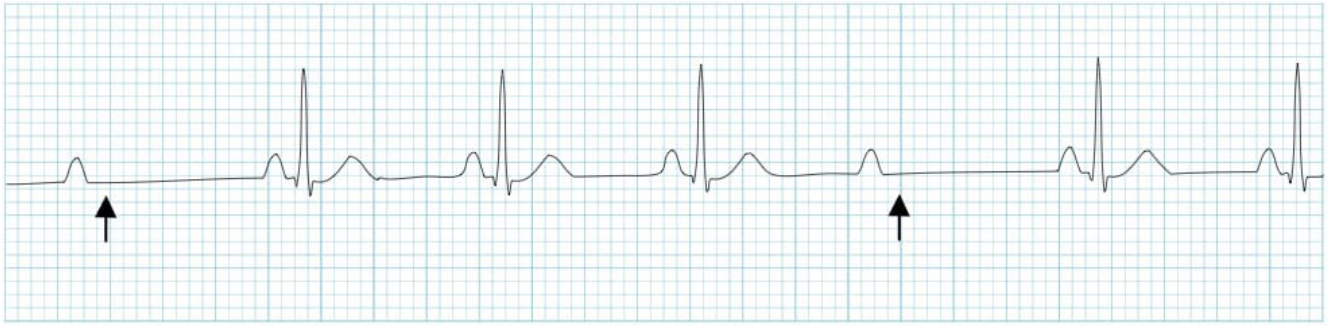
2nd degree type 1, Mobitz type 1

- Intermittent failure of the AV node to conduct impulses
- Block occurs within the AV node
- Progressively prolonged PR interval , followed by a block
- Can occur due to high resting vagal tone



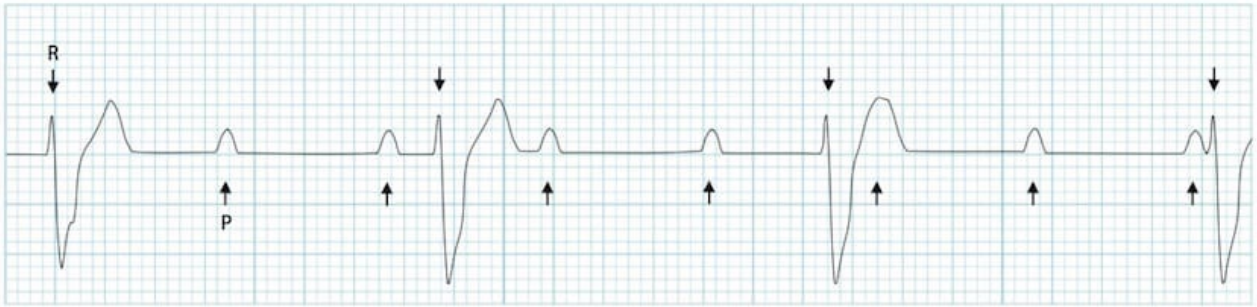
2nd degree AV block, Type II Mobitz

- Fixed 3:1 or 2:1 AV conduction
- PR intervals are fixed
- Irreversible and degenerative condition, patients are at high risk of CHB
- Short term fix – temporary pacing wires
- Long term – permanent pacemaker



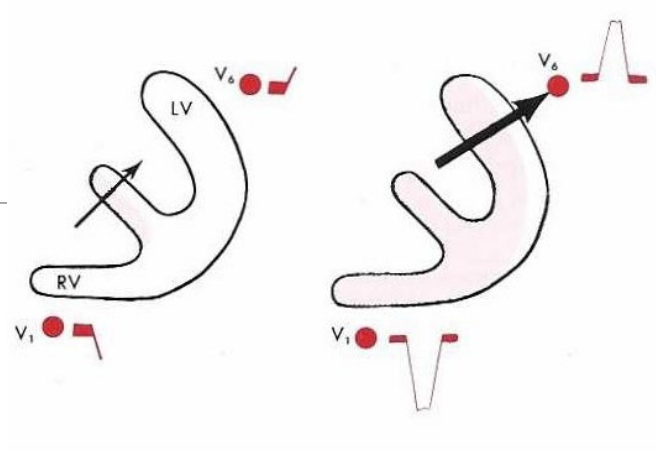
Complete or 3rd degree AV Block

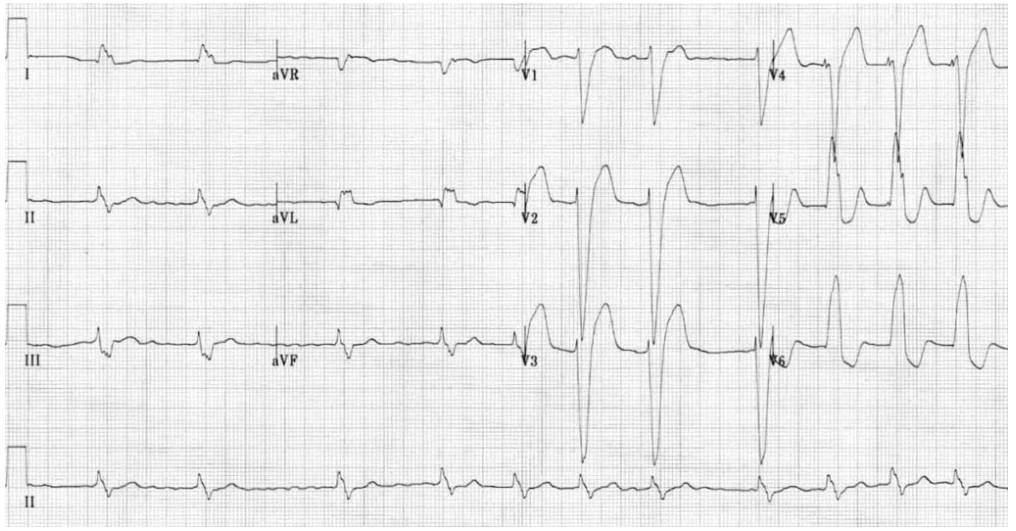
- Total absence of AV conduction
- Junctional rate will be present
- SA node will depolarize the atrium at a different rate to the junction
- High risk of hemodynamic collapse



Left Bundle Branch Block

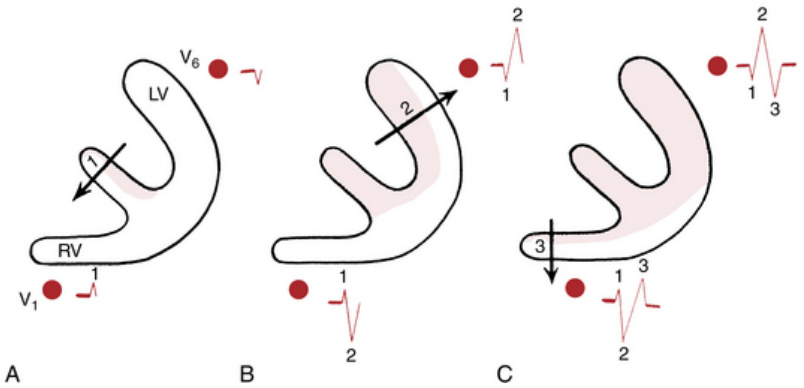
- Wide QRS
- Negative in V1

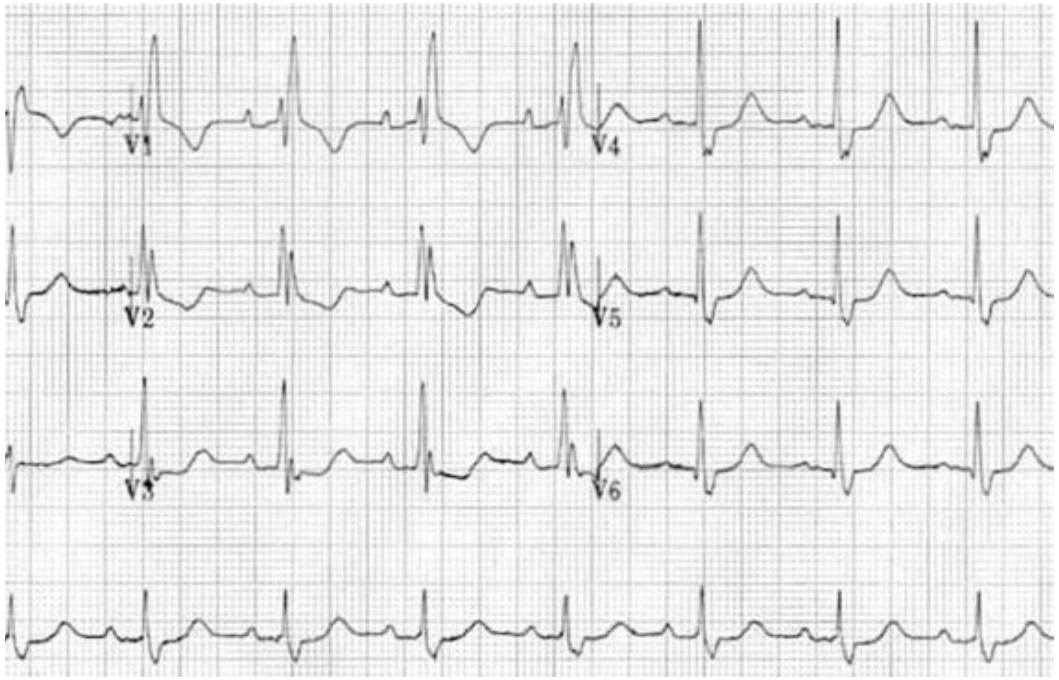




Right Bundle Branch Block

- Dominant R wave in V 1
- RsR pattern
- S wave in lateral leads, V5, V6





Ventricular arrythmia's

- Sustained > 30 secs
- Wide QRS > 120 ms , > 100 bpm

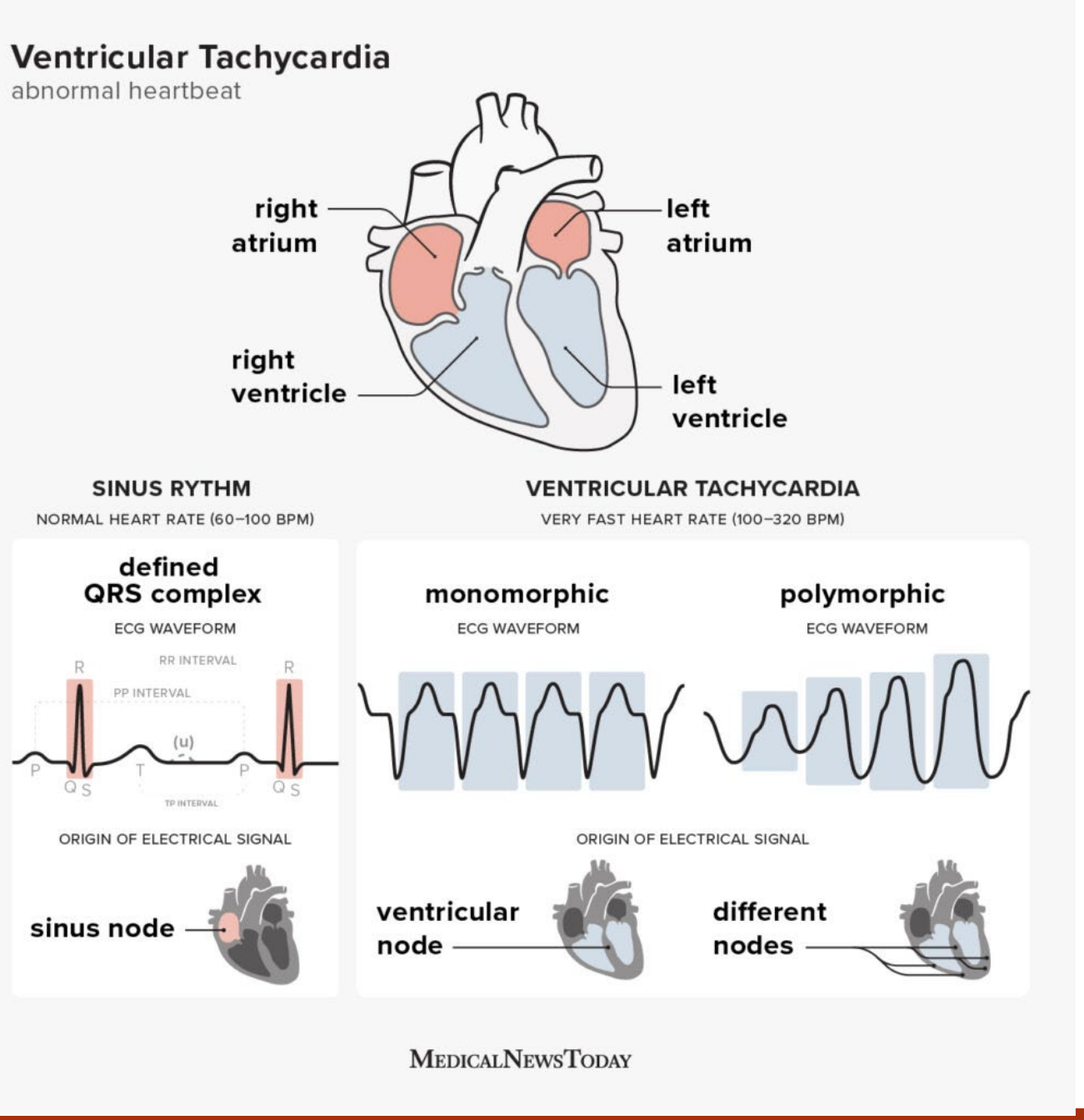
Monomorphic

- Uniform stable QRS morphology

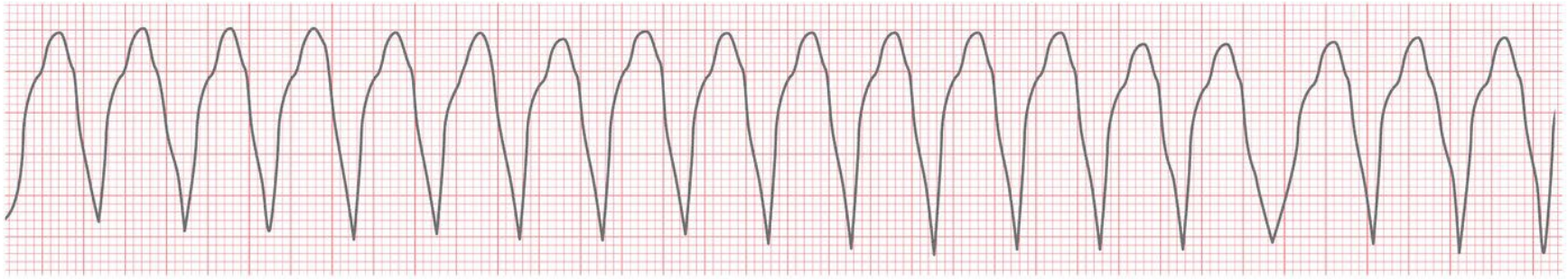
Polymorphic

- 200 bpm
- Hemodynamically unstable
- Can degenerate into VF

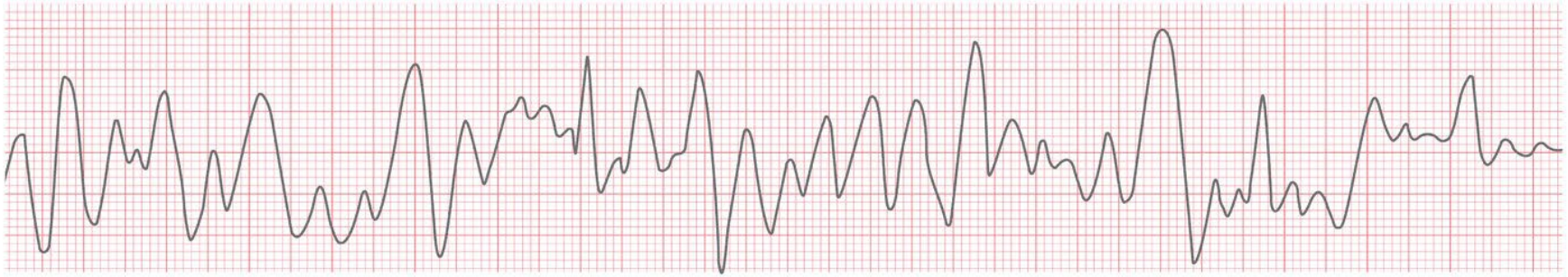
Causes :
Idiopathetic
Ischemia, scar
Myopathy – dilated , HCM
Infiltration – Sarcoidosis , Amyloid
Long QT / Brugada syndrome



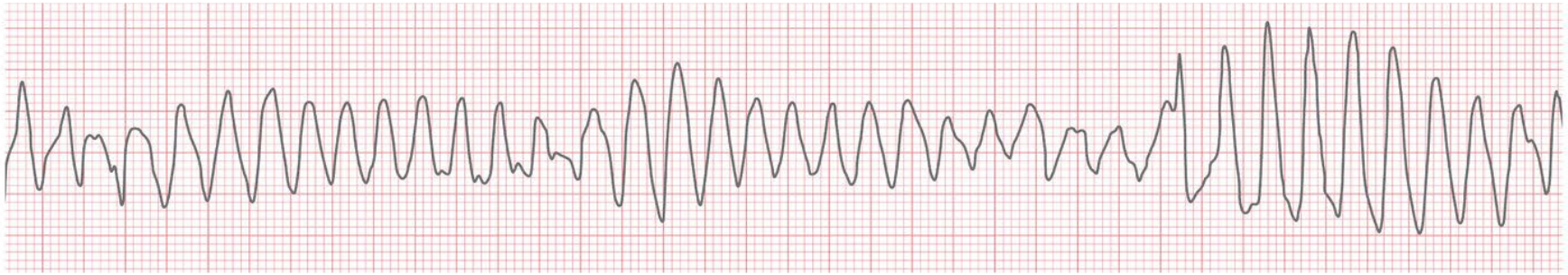
Monomorphic ventricular tachycardia



Polymorphic ventricular tachycardia

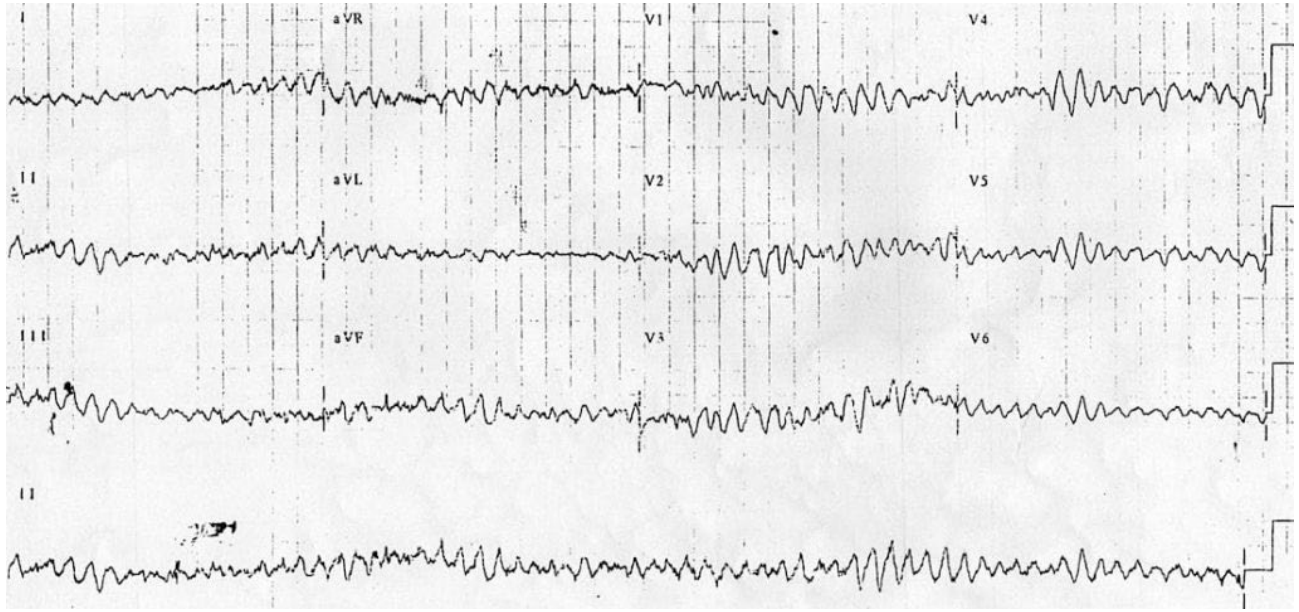


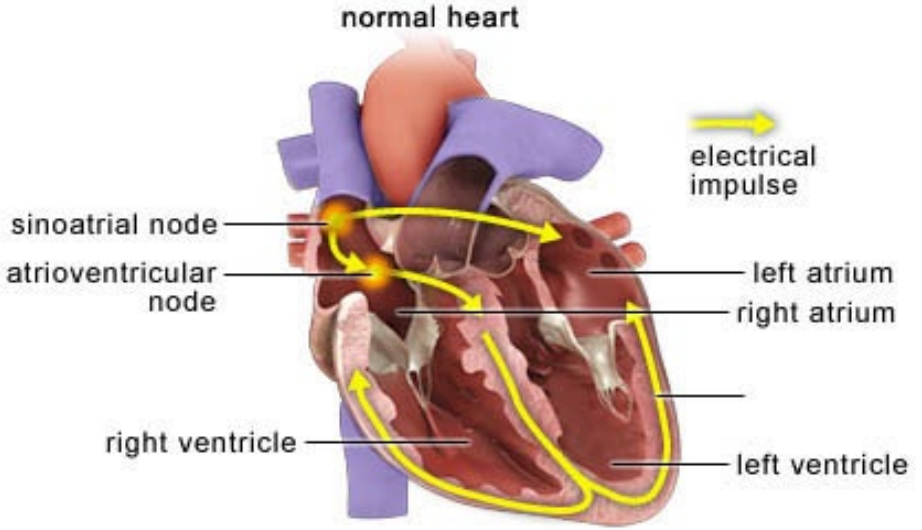
Torsades de pointes

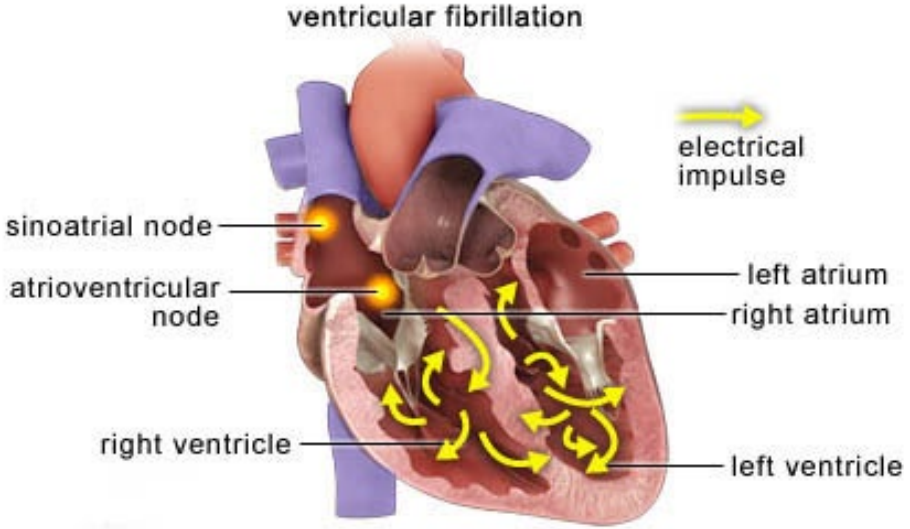


Ventricular fibrillation

- No uniform ventricular depolarization
- 300 bpm, irregular
- Most common cause of sudden cardiac death
- Can cause syncope
- Treatment – defibrillator / anti-arrhythmic therapy

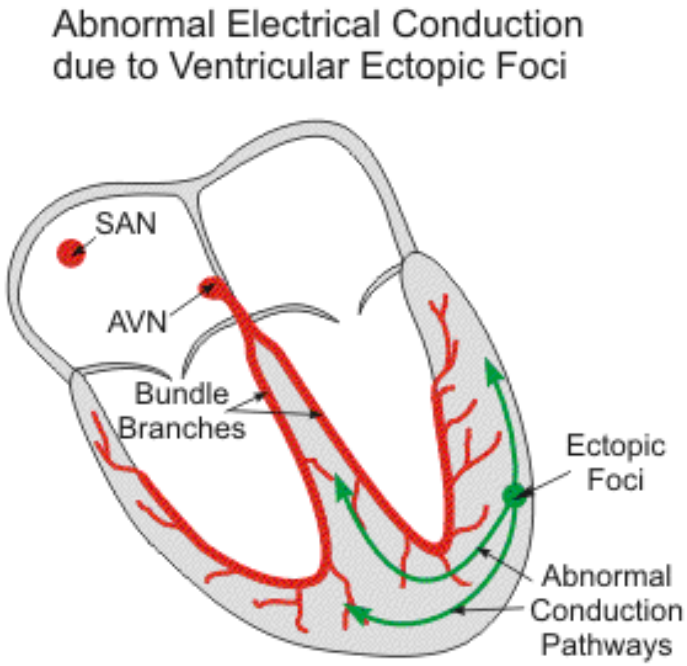


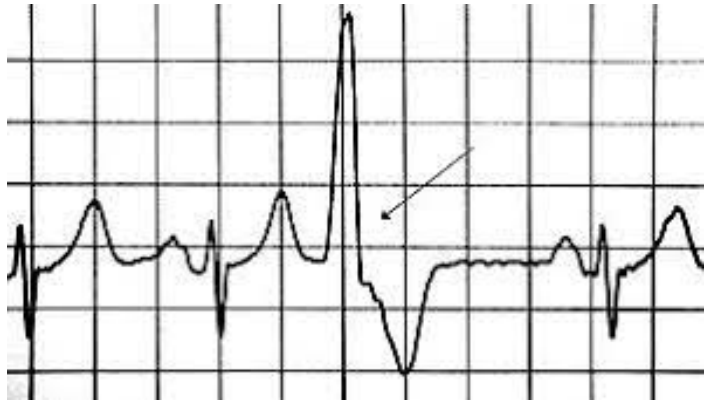




Ectopics

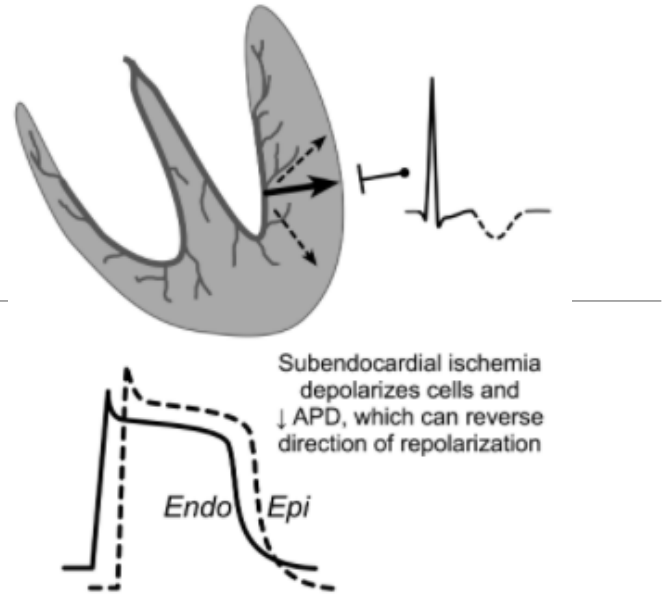
- Pacemaker sites outside the heart
- Can occur in both atria and ventricles
- Occur in addition or can take over the normal pacemaker of the heart
- Does not use the His-Purkinje system to depolarize the heart
- Longer contraction





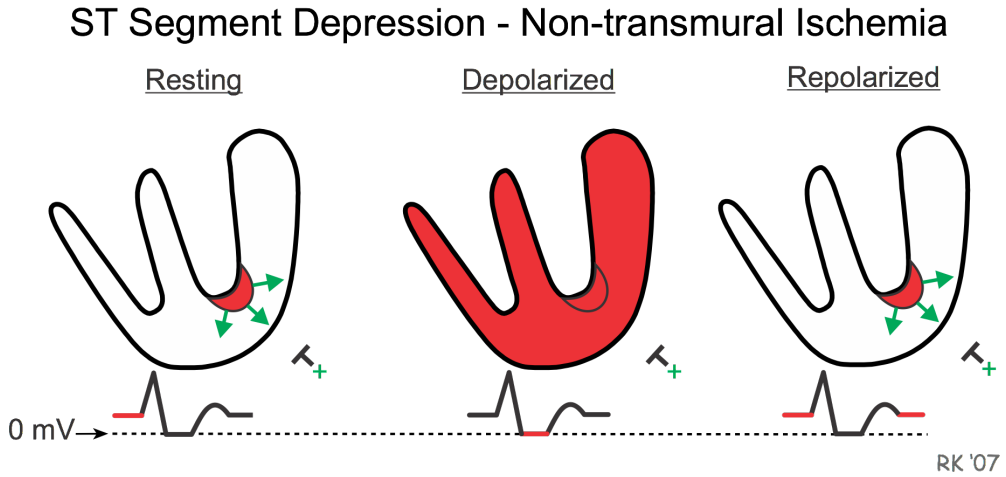
Ischemia

- Inadequate coronary blood flow causing reduced blood flow, hypoxia and cell death
- Subendocardial – ST depression
- Transmural – ST elevation



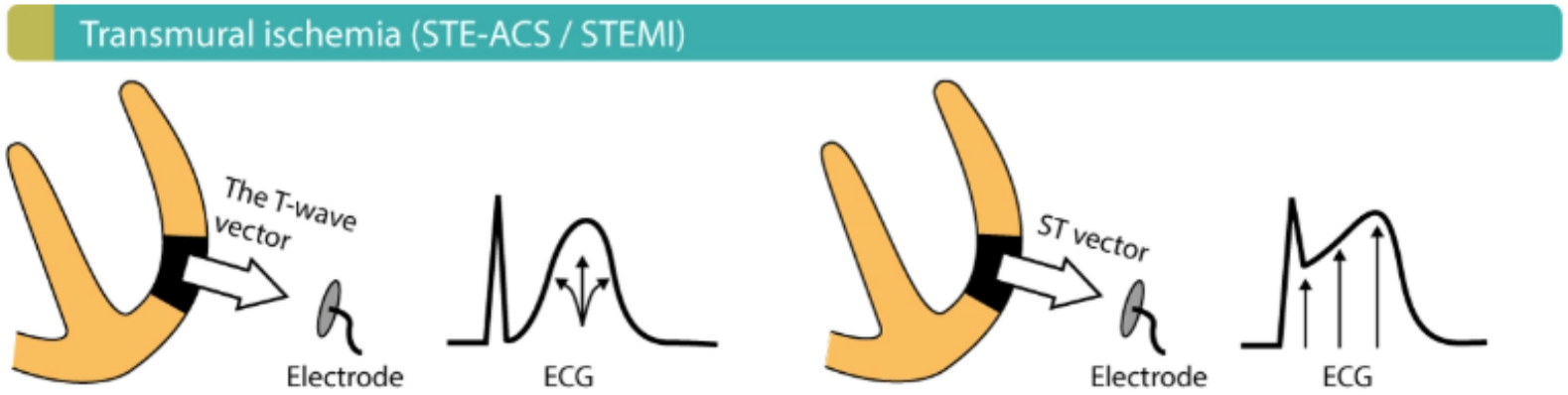
Non ST elevation myocardial infarction (NSTEMI)
ST depression

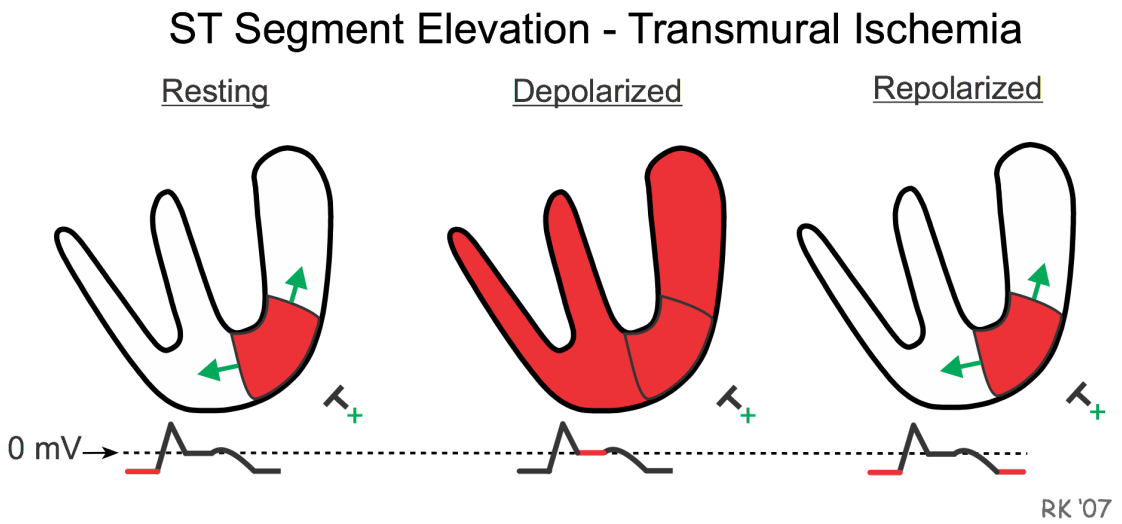
- Ischemia causes cells to have a short action potential
- Earlier than normal depolarization



ST elevation myocardial infarction

- Ischemic event that causes damage to the entire tissue, transmural injury





ECG leads and coronary arteries