
Acknowledgement of Country
We acknowledge the Aboriginal people as the Traditional Owners of this land and we pay our respects to Elders past and present.
www.kinyalerrk.com.au

Cardiac Conduction System Arrhythmias and ECGs

BIANCA COELHO

LEAD CARDIAC PHYSIOLOGIST

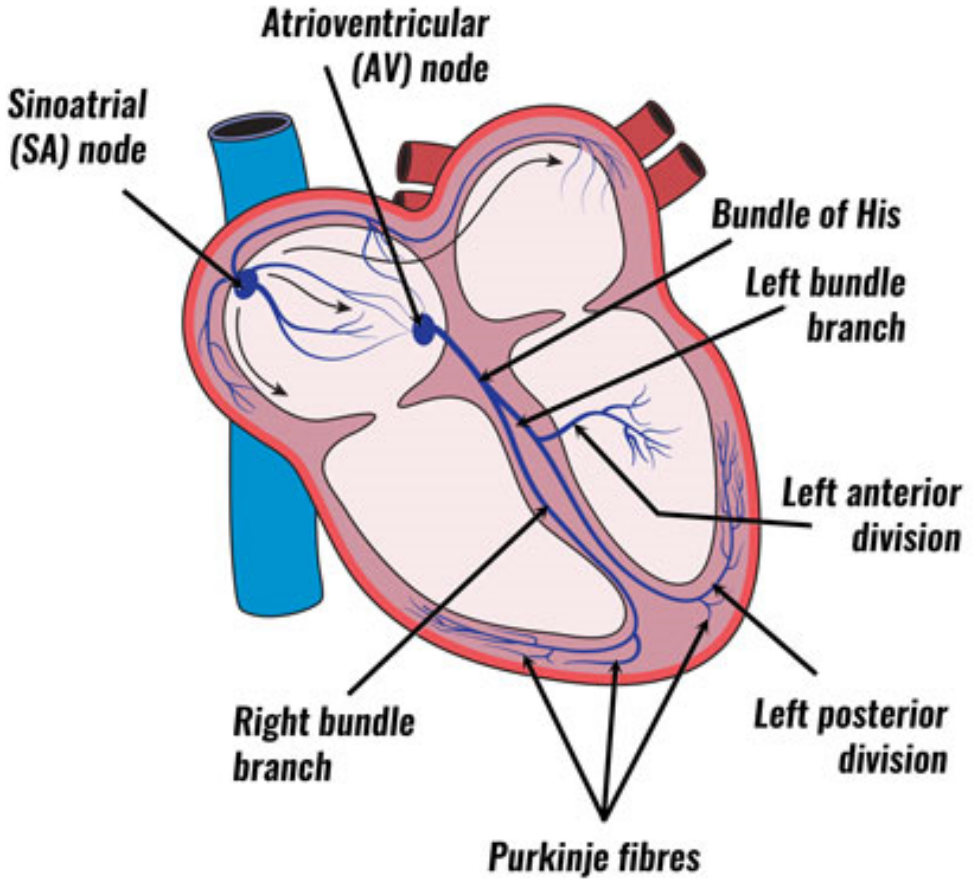
MQHEALTH CARDIOLOGY

DMU (CARDIAC) ACP (CARDIAC)

BIANCA.COELHO@MQHEALTH.ORG.AU

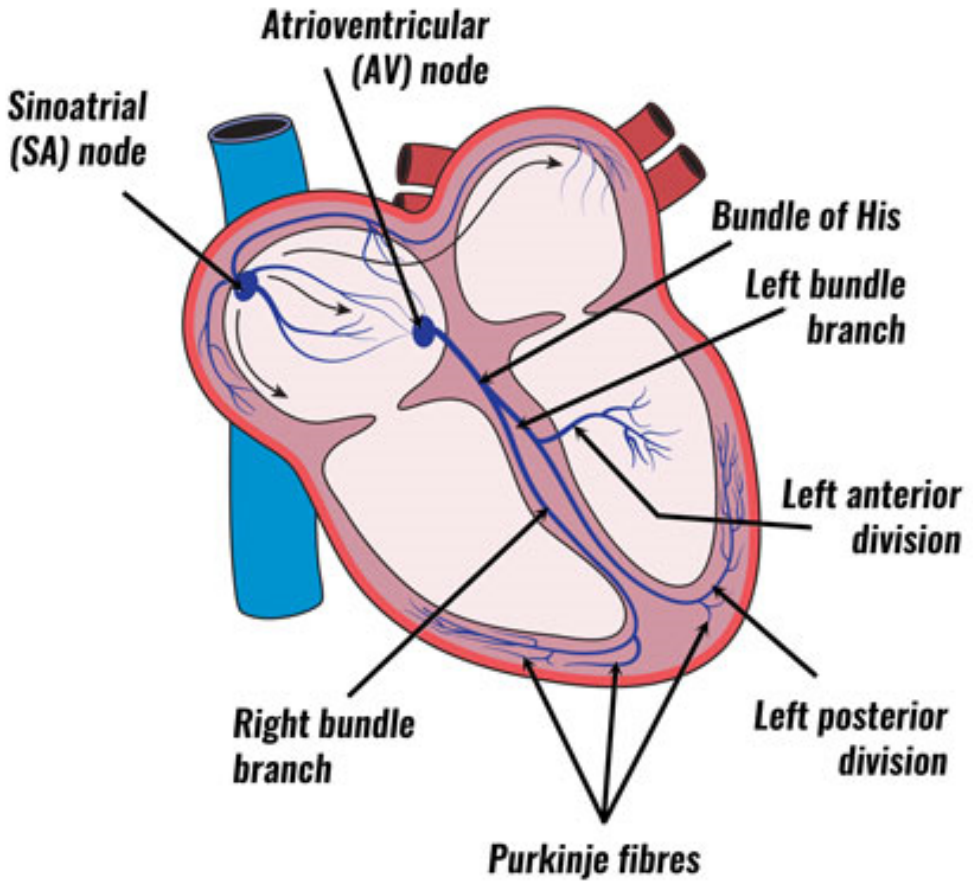
Review

- Basics of electrophysiology
- Action Potential
- The ECG
- Brady arrhythmia’s
- AV Blocks
- Bundle Branch Blocks
- ST elevation myocardial infarction/ Non ST elevation myocardial infarction
- Ectopics and Tachy arrhythmia’s
- Atrial fibrillation / flutter



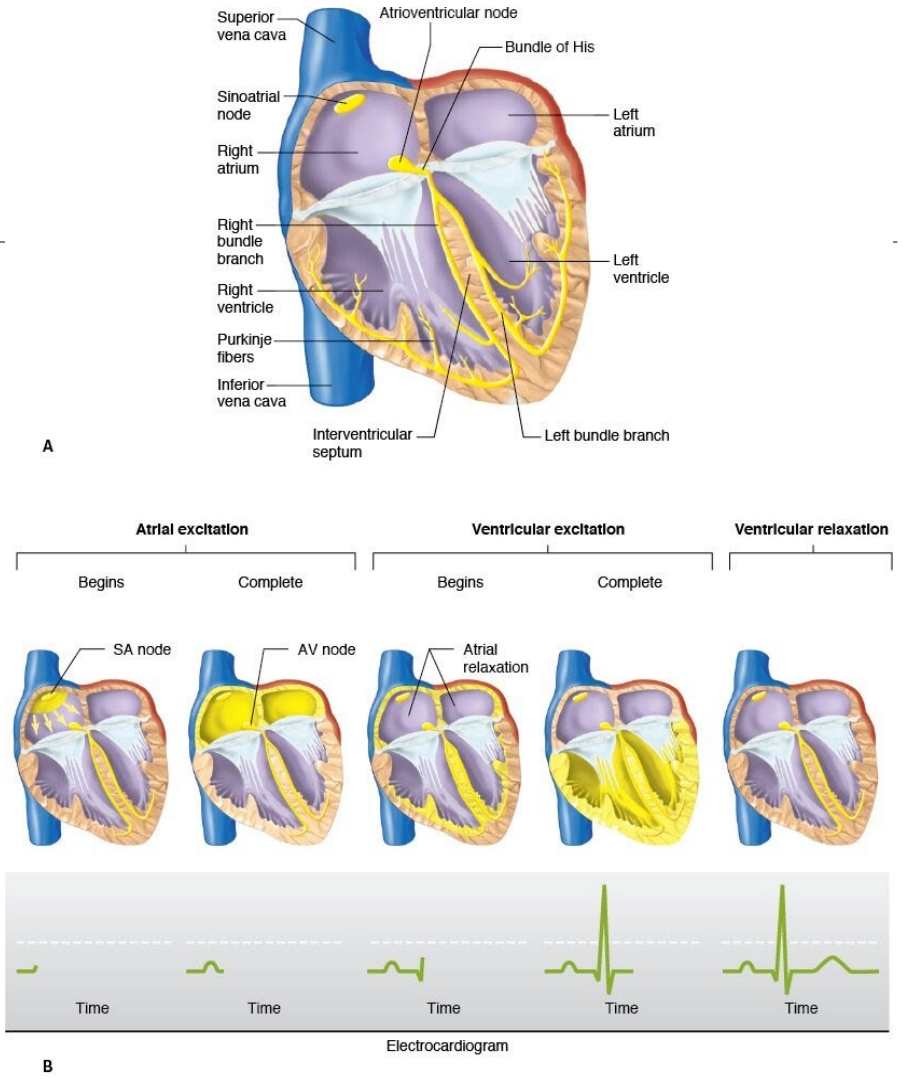
Review

- Basics of electrophysiology
- Action Potential
- The ECG
- Brady arrhythmia's
- AV Blocks
- Bundle Branch Blocks
- ST elevation myocardial infarction/ Non ST elevation myocardial infarction
- Ectopics and Tachy arrhythmia's
- Atrial fibrillation / flutter



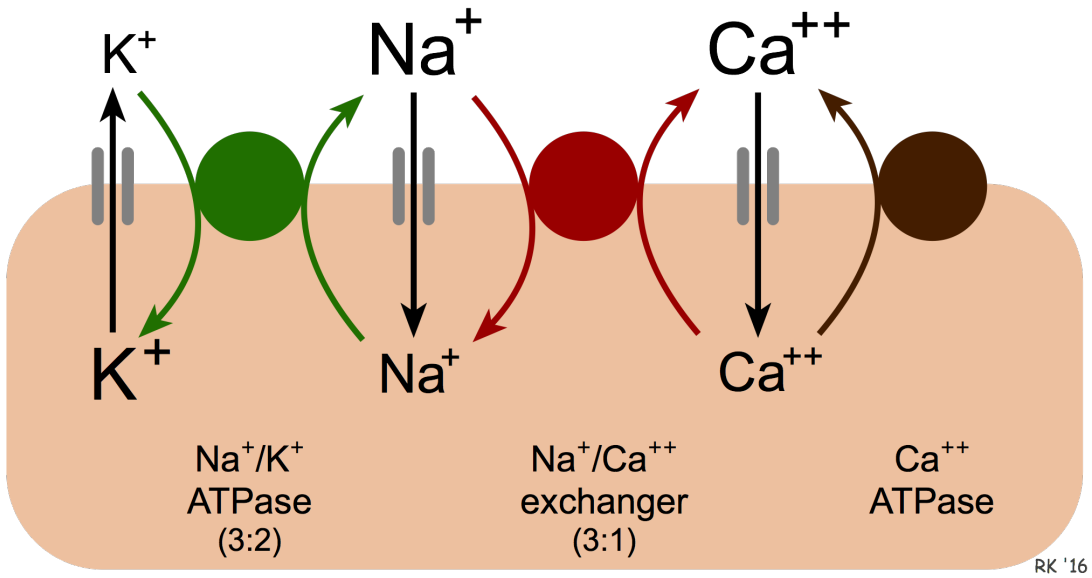
The impulse conduction system consists of five structures

- The sino-atrial node (SA node)
- The atrio-ventricular node (AV node)
- Bundle of HIS
- The Purkinje fibers
- Fascicles and the left and right bundles



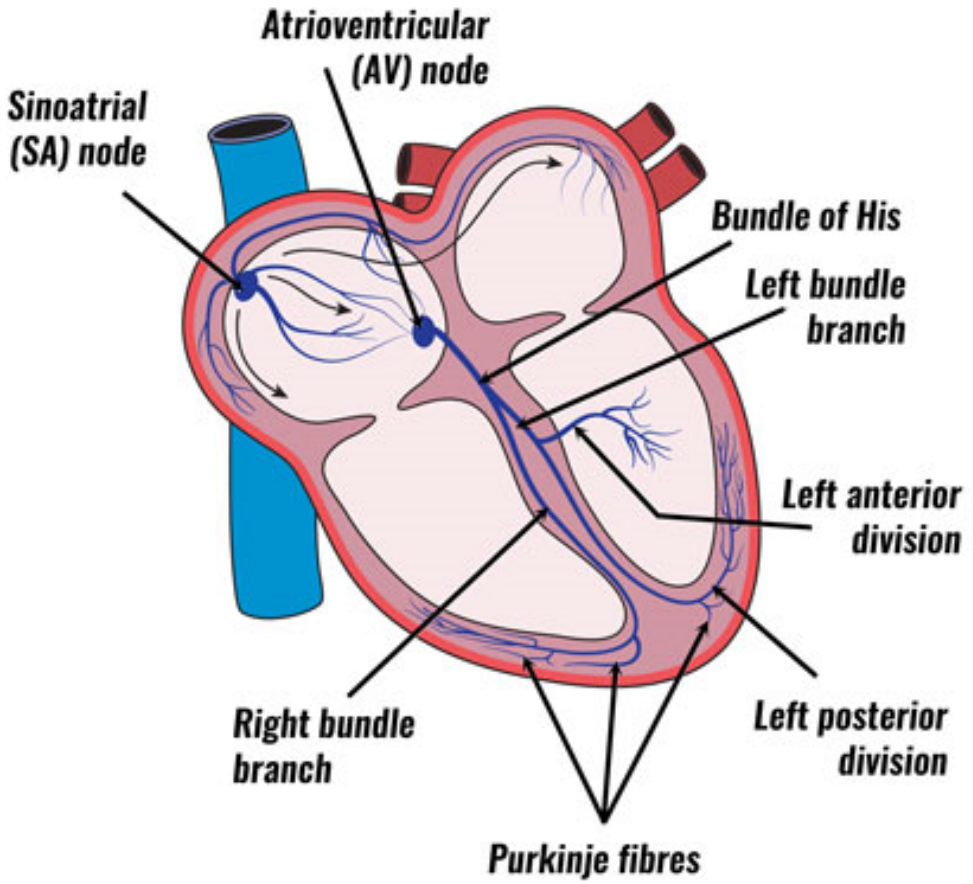
Basics of electrophysiology

- 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.



Review

- Basics of electrophysiology
- Action Potential
- The ECG
- Brady arrhythmia’s
- AV Blocks
- Bundle Branch Blocks
- ST elevation myocardial infarction/ Non ST elevation myocardial infarction
- Ectopics and Tachy arrhythmia’s
- Atrial fibrillation / flutter

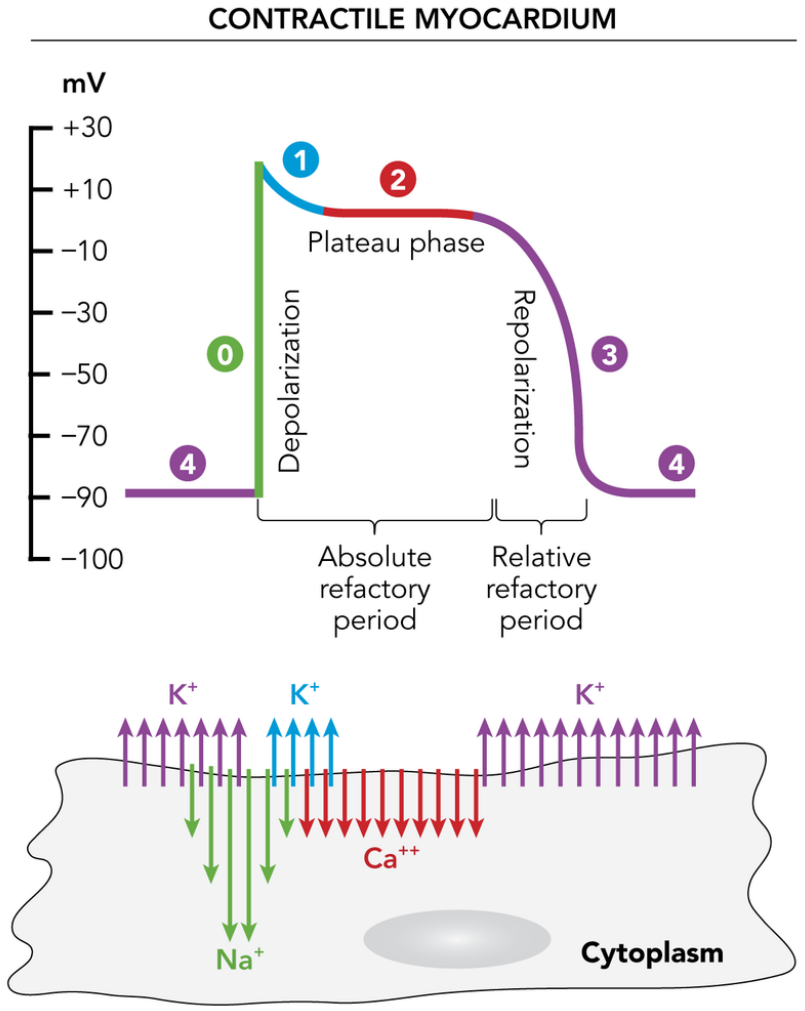


Action potential

- 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

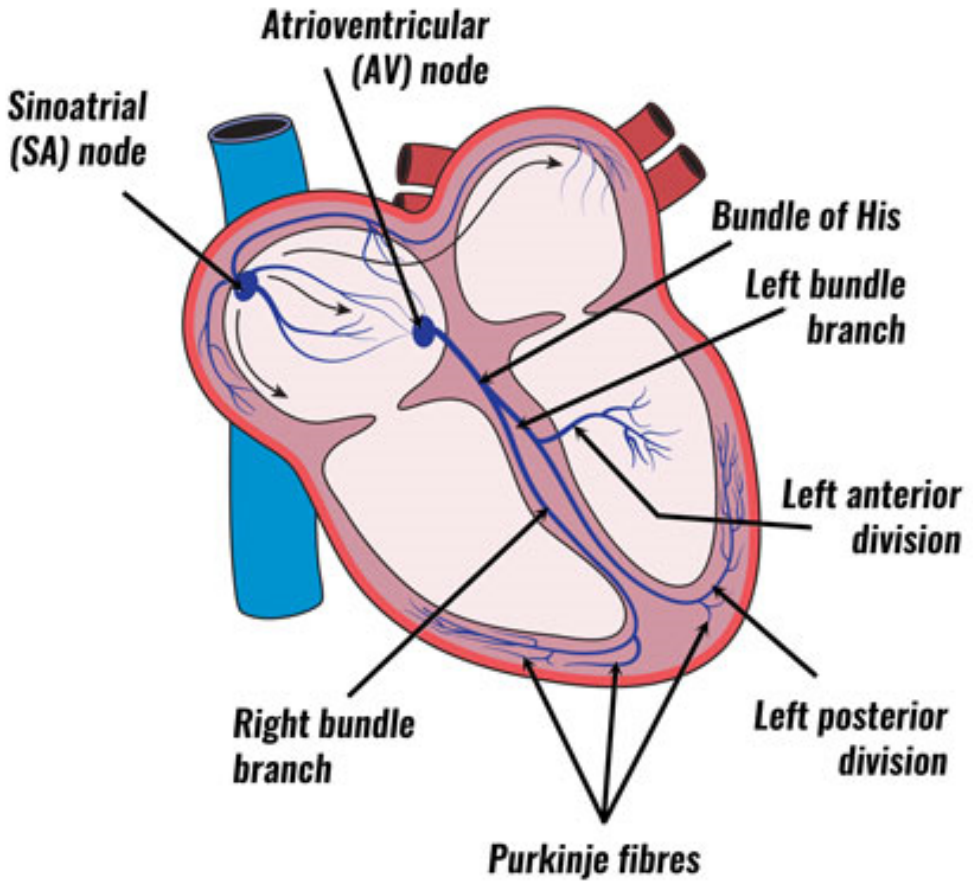
Phases

- Phase 0 – Depolarization – influx of Na + , rapid upstroke of action potential
- Phase 1 – Transient repolarization – Inactivation of Na+
- Phase 2 – Plateau Phase – Na+ closed, voltage maintained by Ca + moving in
- Phase 3 – Rapid repolarization – Ca + close , K + remains open
- Phase 4 – Diastole/ resting state



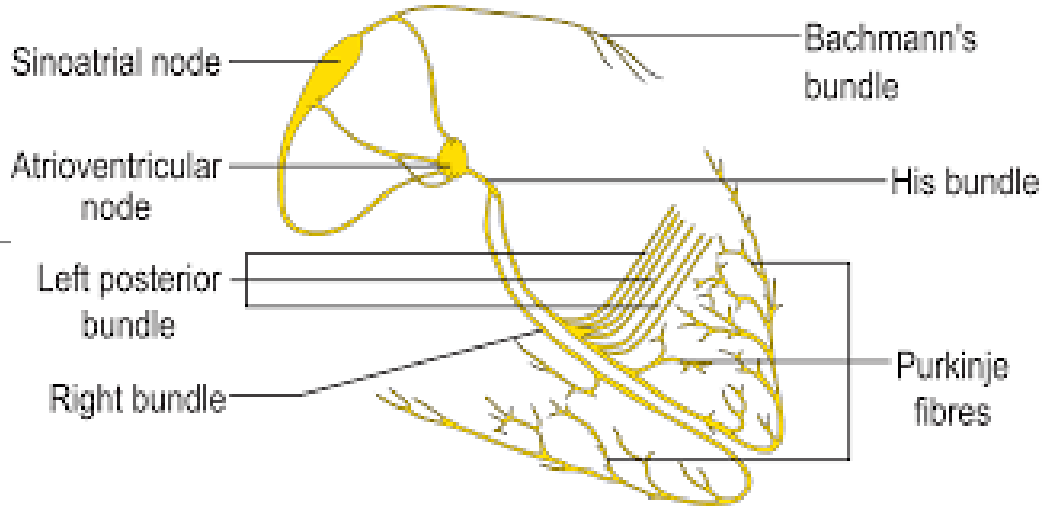
Review

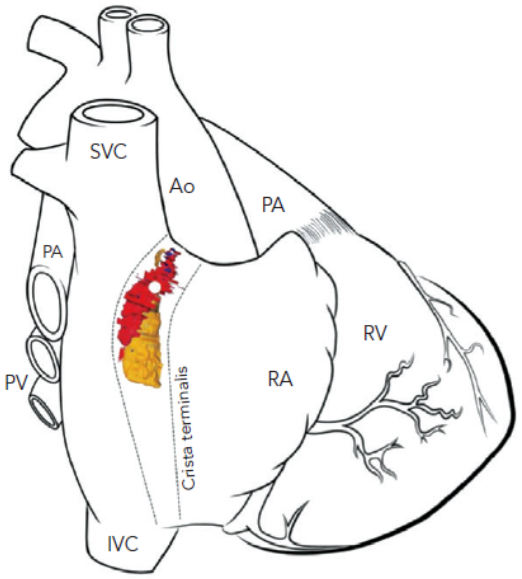
- Basics of electrophysiology
- Action Potential
- The ECG
- Brady arrhythmia's
- AV Blocks
- Bundle Branch Blocks
- ST elevation myocardial infarction/ Non ST elevation myocardial infarction
- Ectopics and Tachy arrhythmia's
- Atrial fibrillation / flutter



SA Node

- Depolarizes at 60-100 bpm
- Controlled by the autonomic nervous system, vagus nerve
- Parasympathetic input slows down the rate of action potential production
- 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.
- This ensures simultaneous and coordinate, right and left atrium depolarization



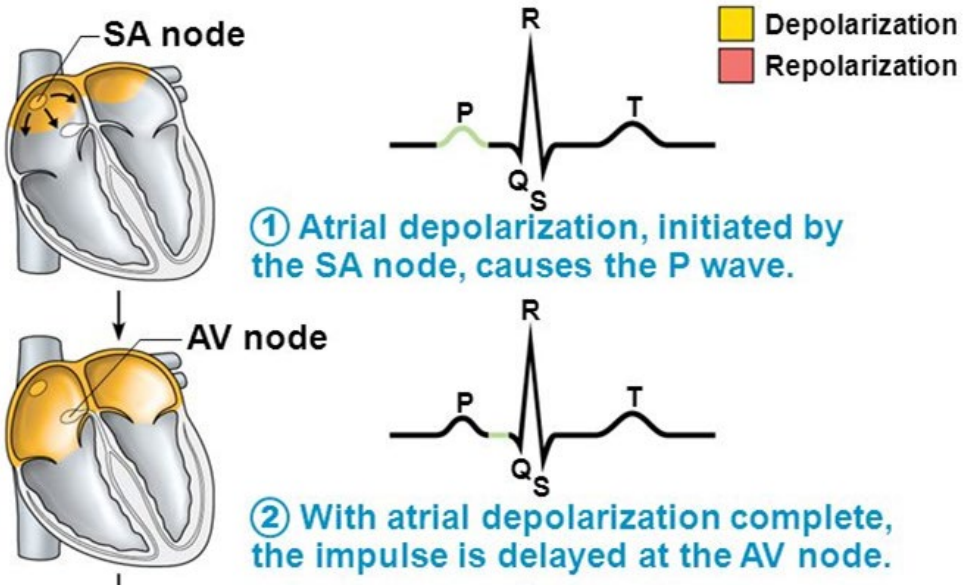


P wave

- 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

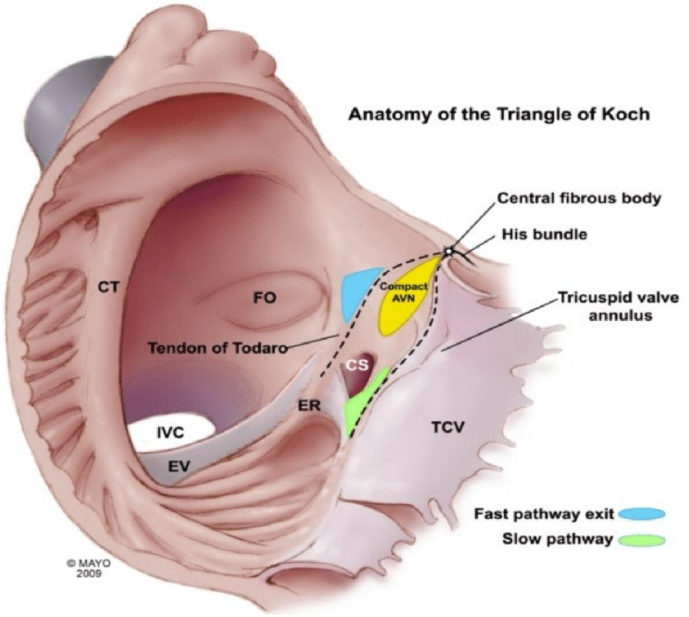
<u>Normative values</u>

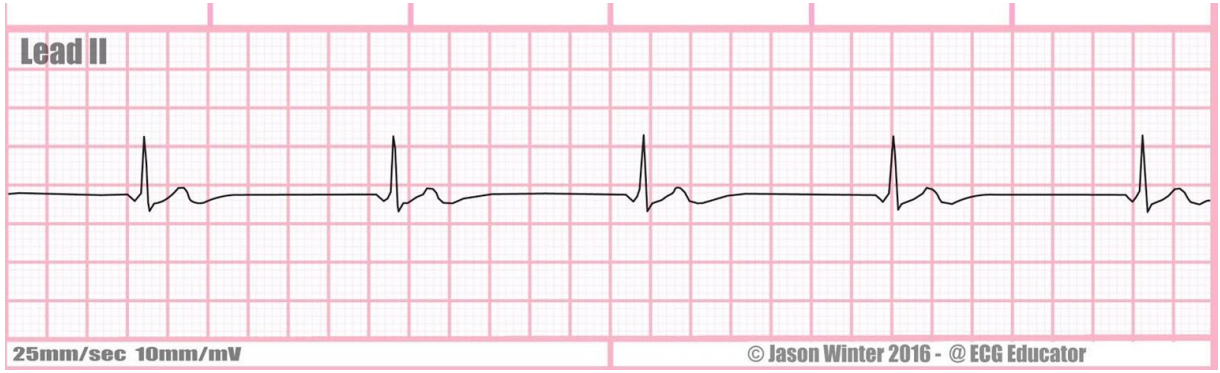
- P wave – upright in lead I, II
- <120 ms width
- <2.5 mm tall
- Inverted in avR
- V1 – biphasic
- PR interval – is the time between when the impulse travels from the atria to the AV node, allowing the ventricles to fill with blood
- > 120 ms – 200 ms



AV node

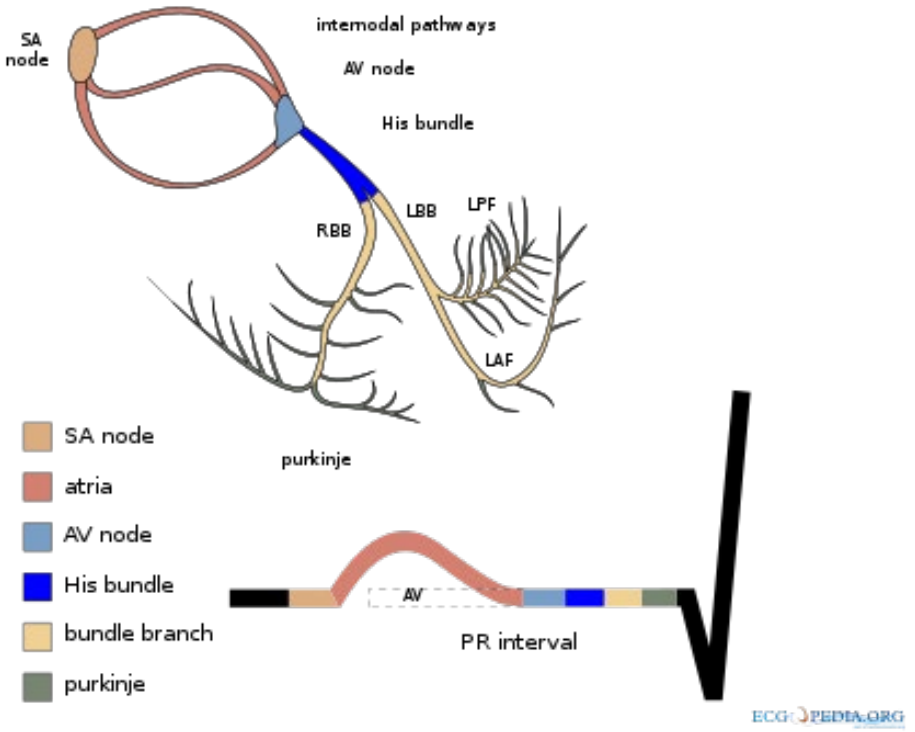
- Compact node , located at the apex of the Triangle of Koch.
- Made up of a cluster of cells, that can collect impulses and funnel it towards the His Purkinje system
- AV node has the ability to slow impulses
- AV node can also function as a secondary pacemaker
- AV node generates impulses at the rate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





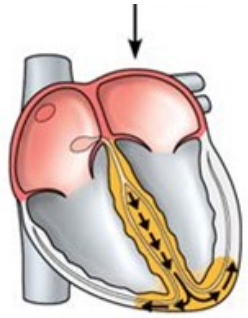
Bundle of HIS

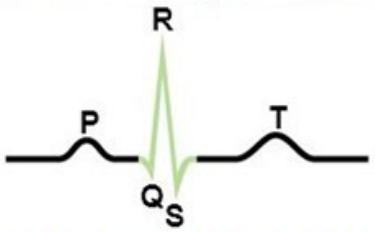
- Located between the atria and the ventricles
- Bifurcates into the left and right bundle
- Can conduct impulses at faster rates than myocytes
- PR interval is representative of HIS BUNDLE conduction
- Right and Left Bundles
- Runs along the septum
- RBB – thin , long and branches into Purkinje fibers
- LBB – thick and branches into left anterior fascicle and left posterior fascicle.
- This allows coordinated contraction of the RV and LV.



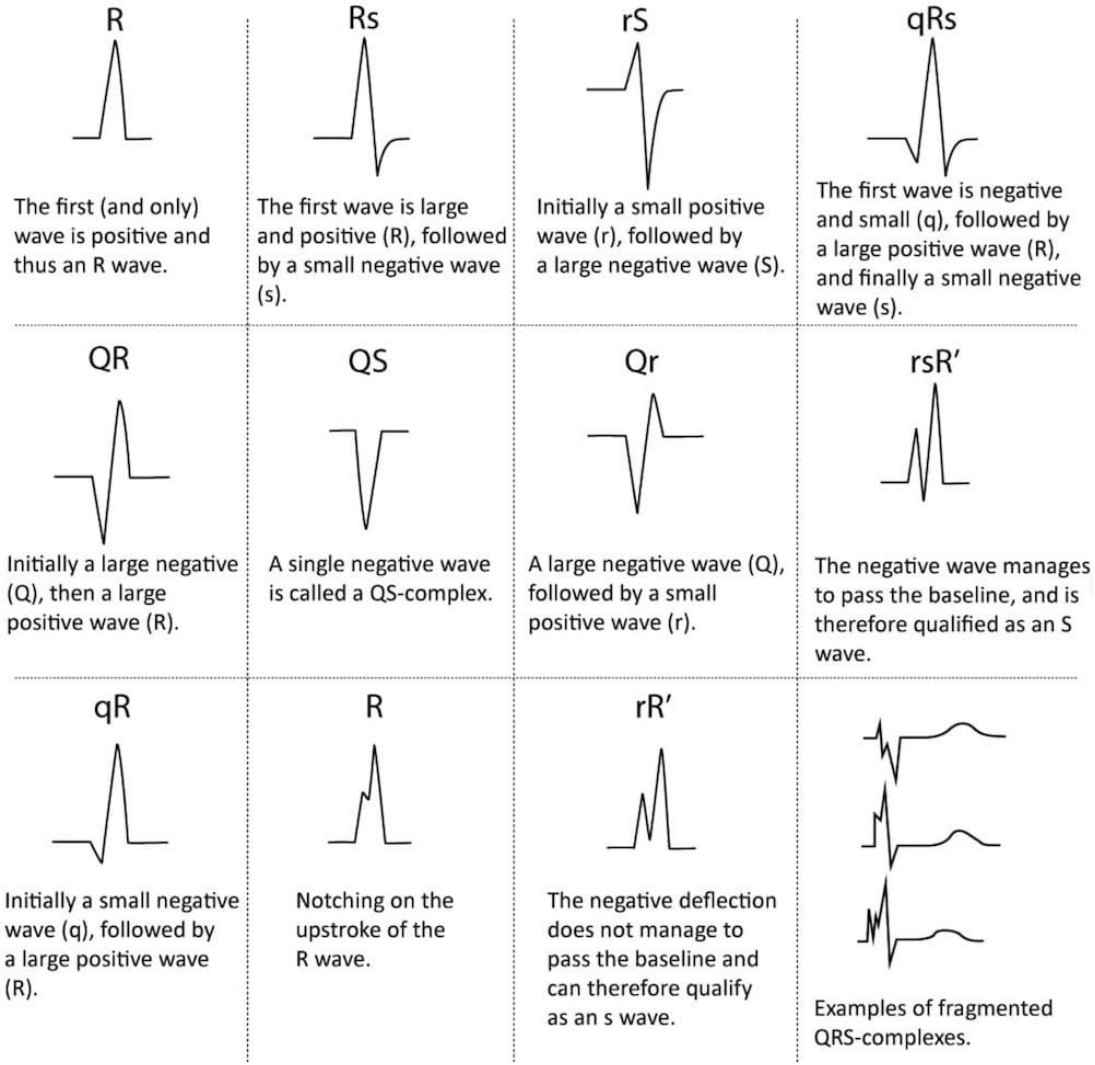
QRS Complex

- 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.



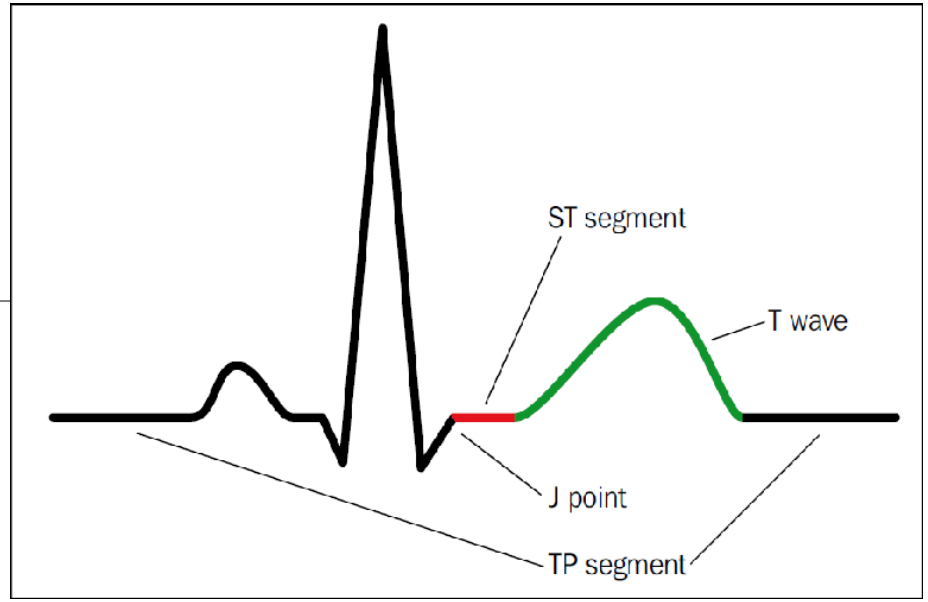


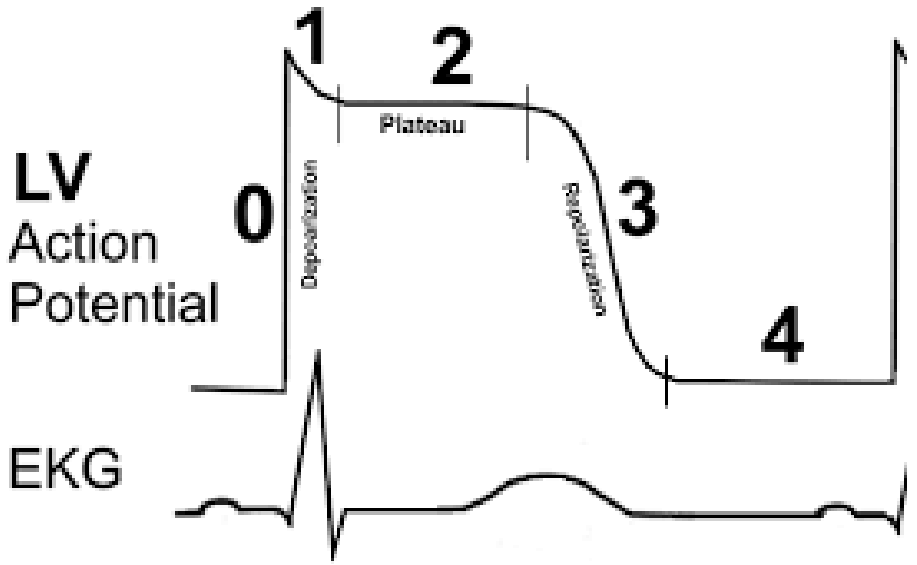
③ AV node depolarizes; Ventricular depolarization begins at apex, causing the QRS complex.
Atrial repolarization occurs.



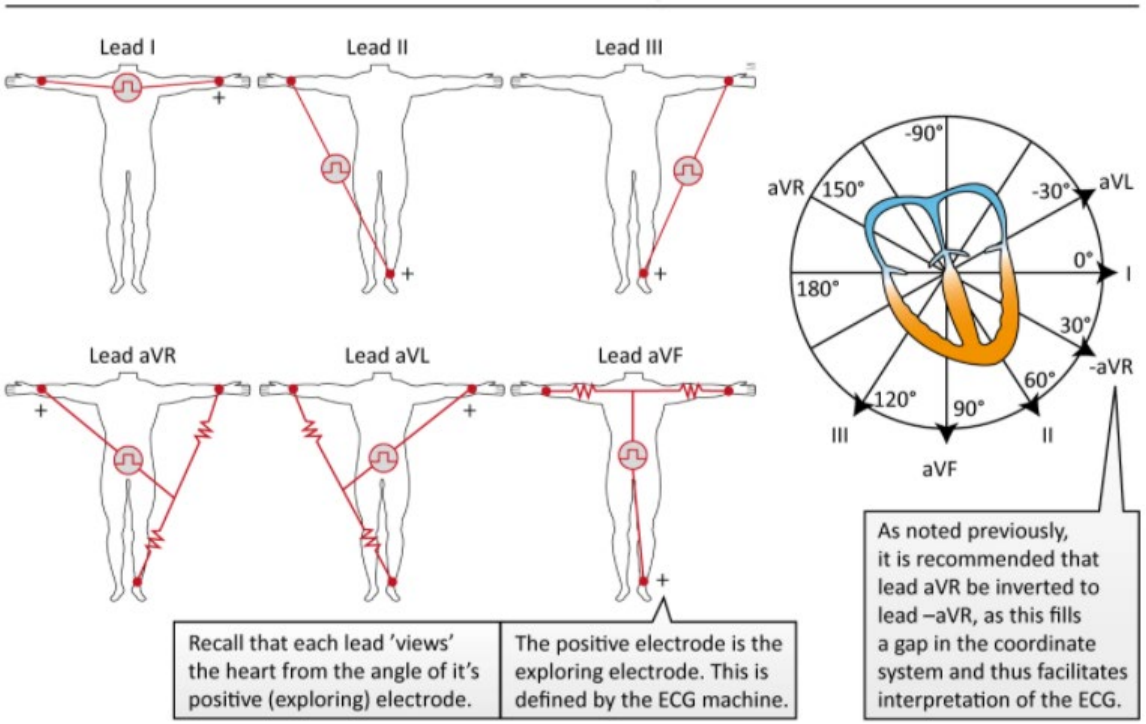
ST Segment

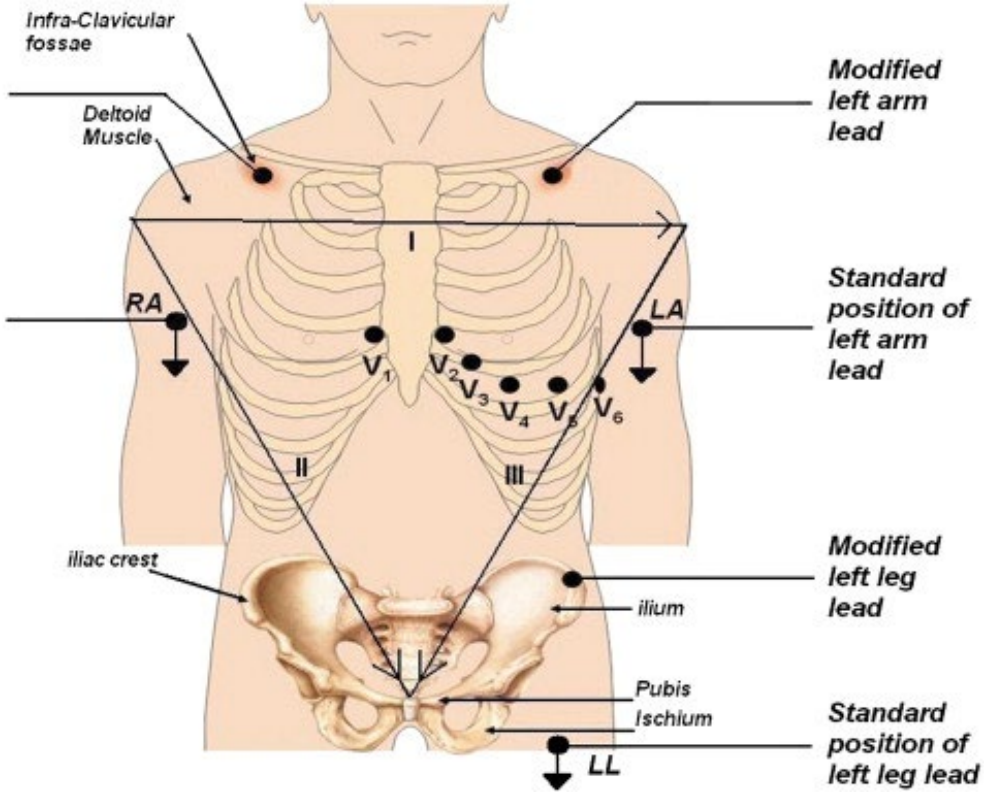
- Measured from the offset of the QRS complex to the onset of the T wave
- Time between ventricular depolarization and repolarization
- Isoelectric
- **J point**
- Where the QRS complex meets the ST segment
- **T wave**
- Ventricular repolarization
- Phase 3 / 4 action potential
- Important for assessing Ischemia and electrolyte abnormalities
- Positive in most leads except , avR, v1 , III
- Amplitude < 5 mm in limb leads and < 10 mm in precordial leads



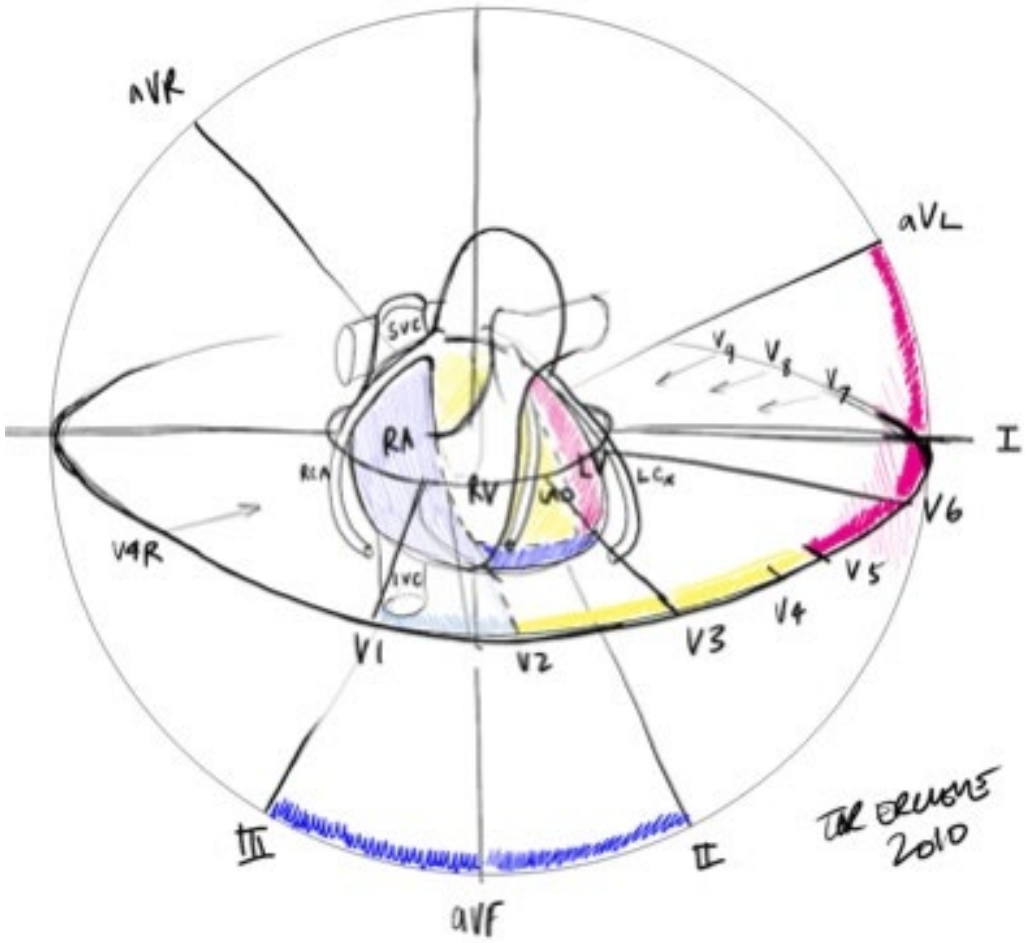


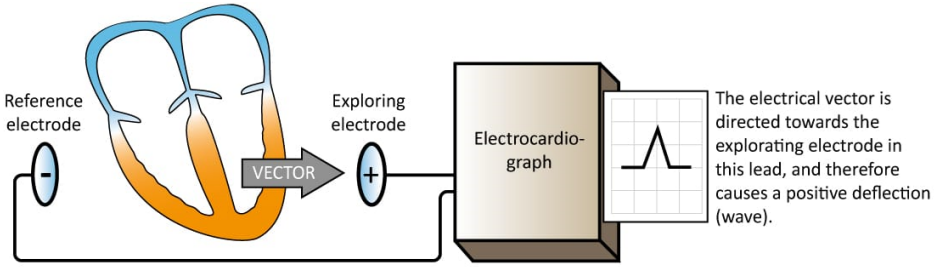
ECG lead placement

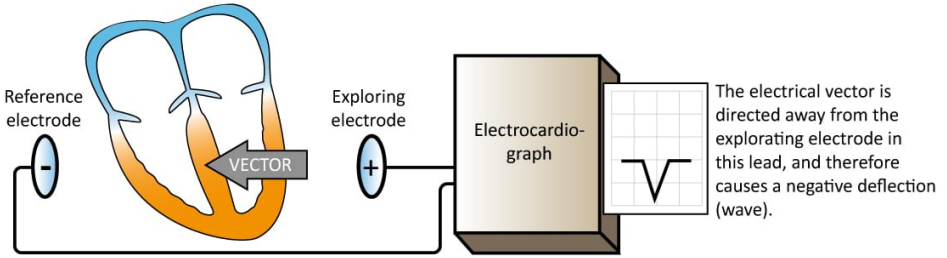


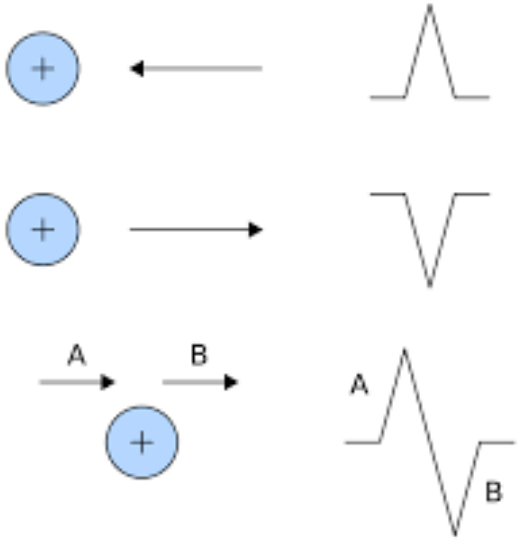


ECG AXIS





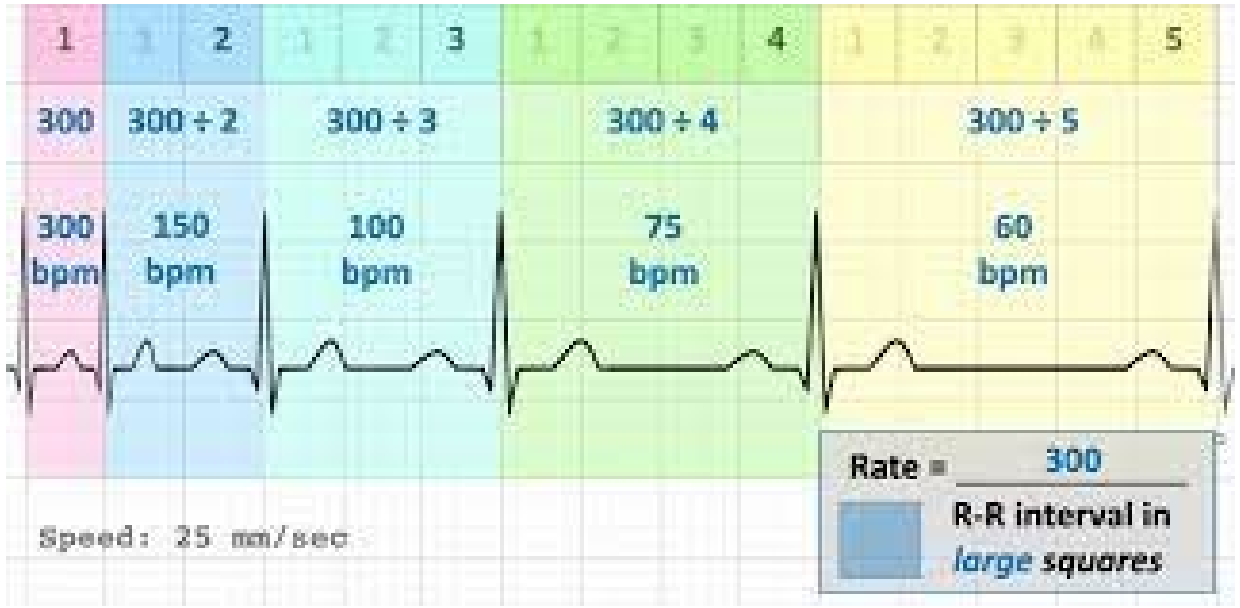




MQ Health
MACQUARIE UNIVERSITY
HEALTH SCIENCES CENTRE

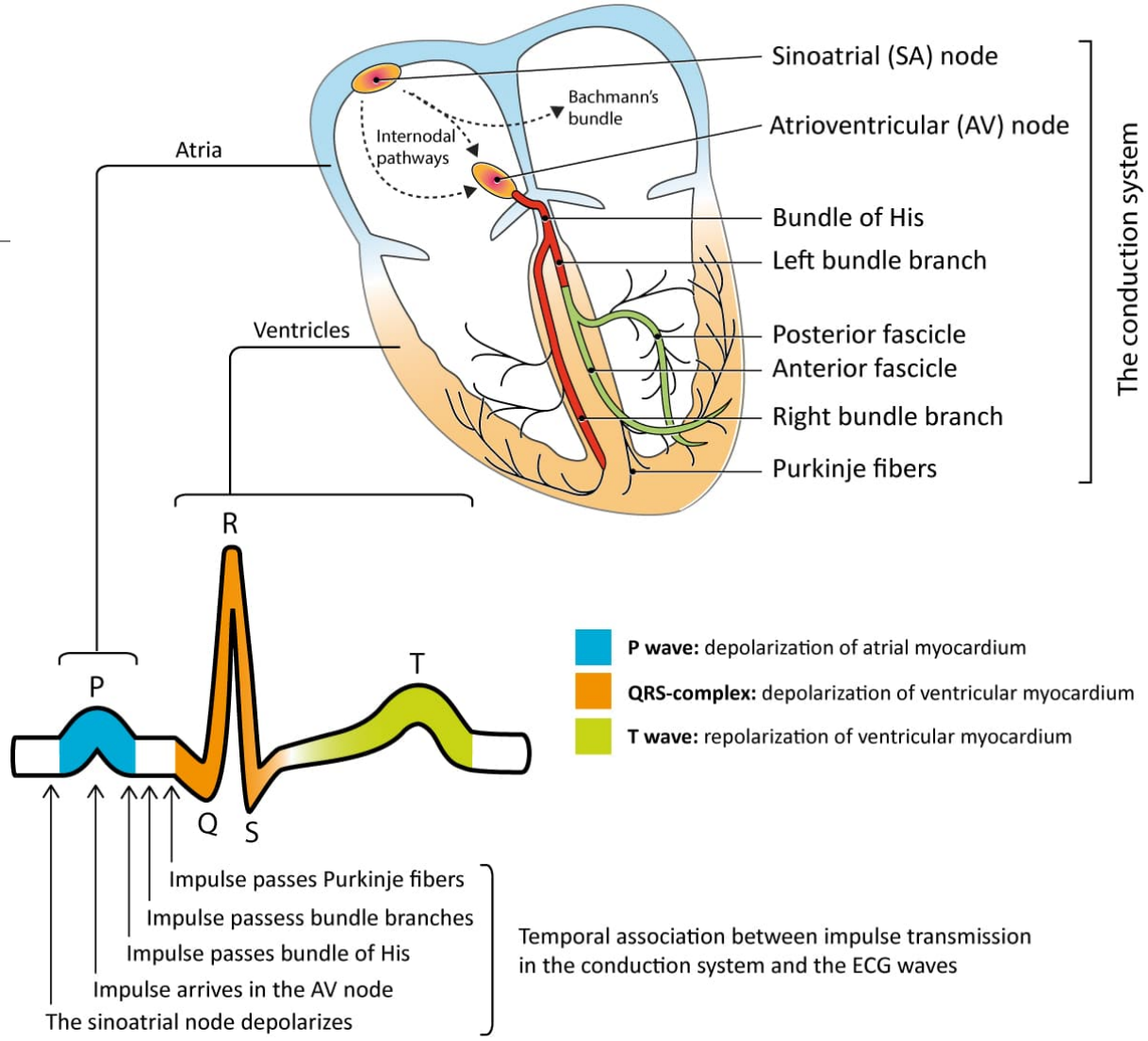
ECG features

- Heart rate = 300/ no. of large squares
- Is there a P wave - atrial contraction confirmed
- Is there a QRS complex - ventricular contraction confirmed
- Is there a T wave - ventricular repolarization confirmed



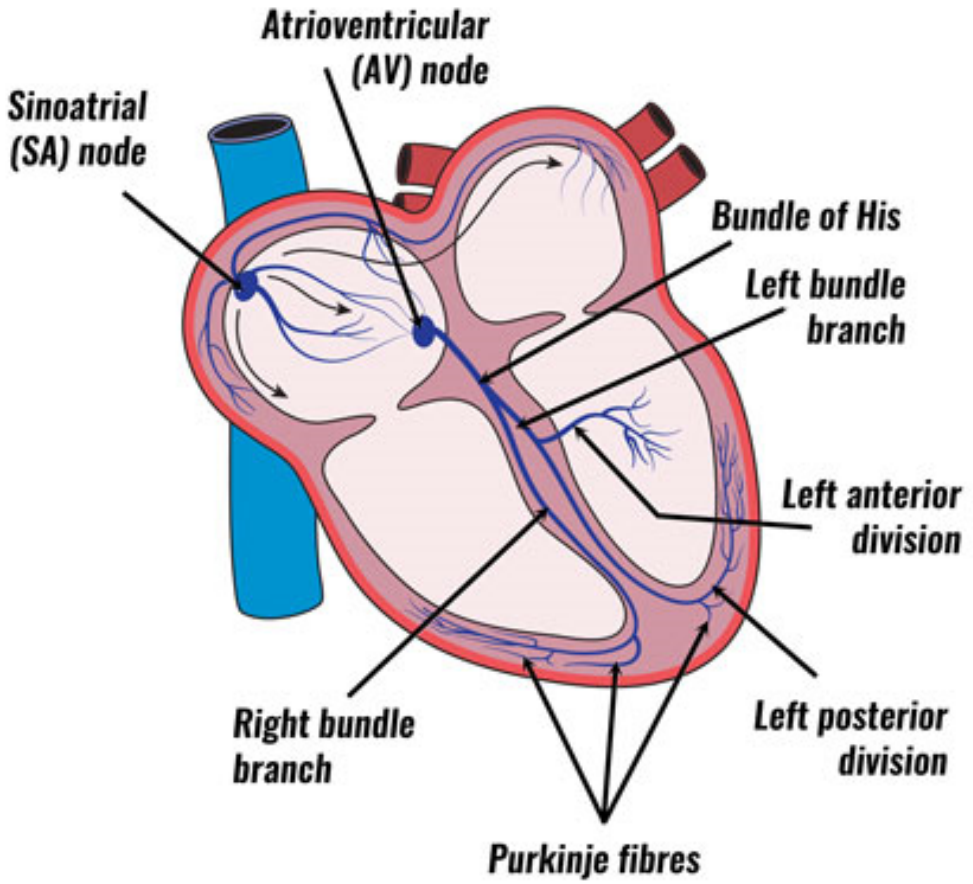
Review

- Sinus node - atrium - P wave - primary pacemaker - atrial contraction
- AV node - inferior atrium - HIS bundle - PR interval – isoelectric line
- Purkinje fibres - left and right bundle - QRS - depolarisation - ventricular contraction
- T wave - repolarization



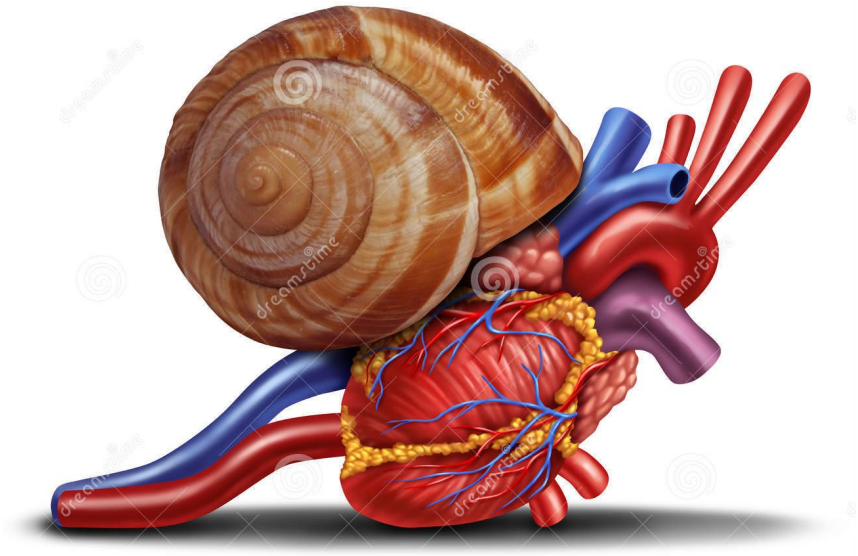
Review

- Basics of electrophysiology
- Action Potential
- The ECG
- Brady arrhythmia’s
- AV Blocks
- Bundle Branch Block
- ST elevation myocardial infarction/ Non ST elevation myocardial infarction
- Ectopics and Tachy arrhythmia’s
- Atrial fibrillation / flutter



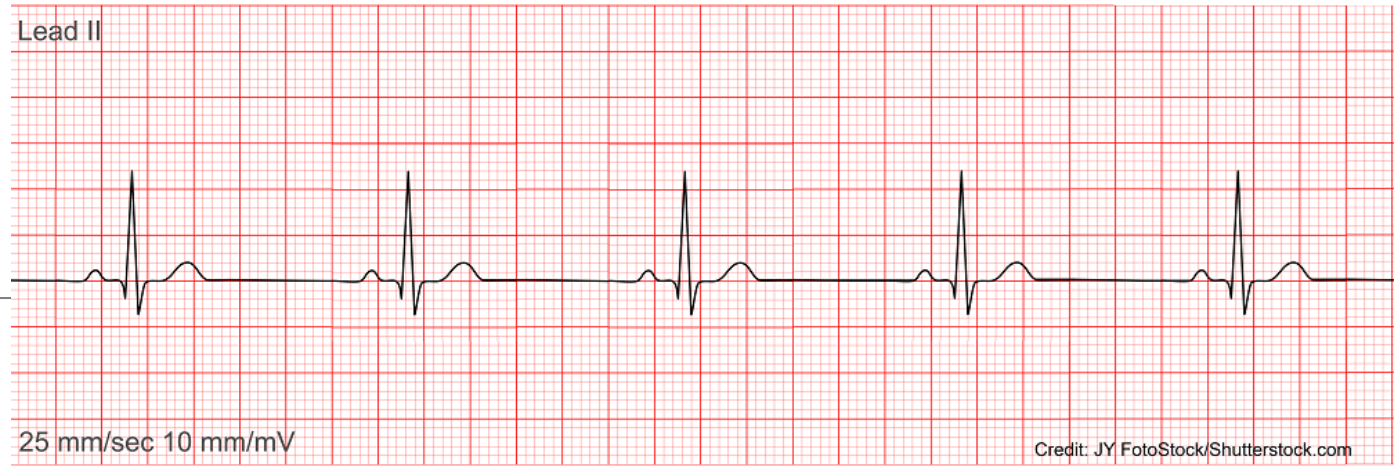
Bradyarrhythmias (Slow)

- SA dysfunction/ Sick sinus syndrome
- Bradycardia
- Sinus pauses
- Sinus arrythmia



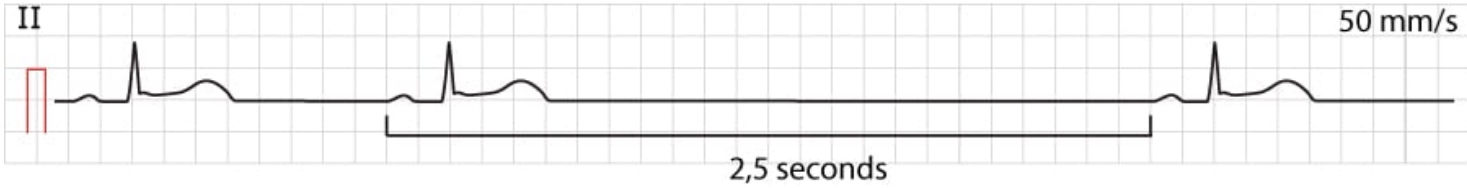
Sinus brady

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



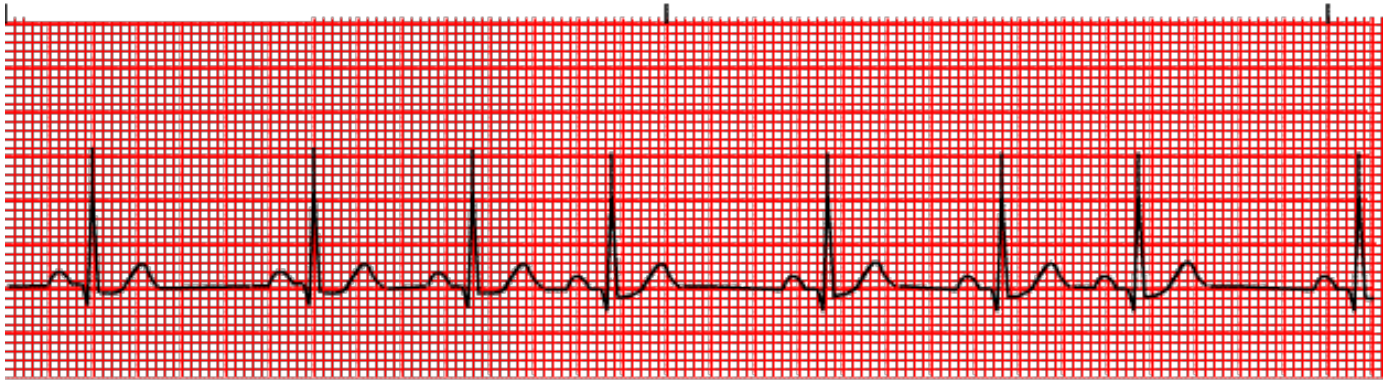
Sinus Pause

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



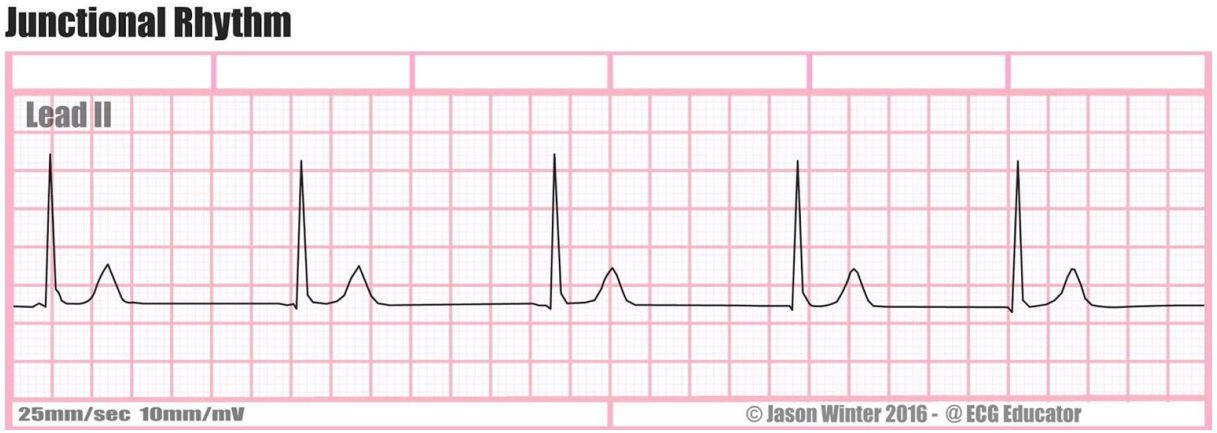
Sinus arrythmia

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



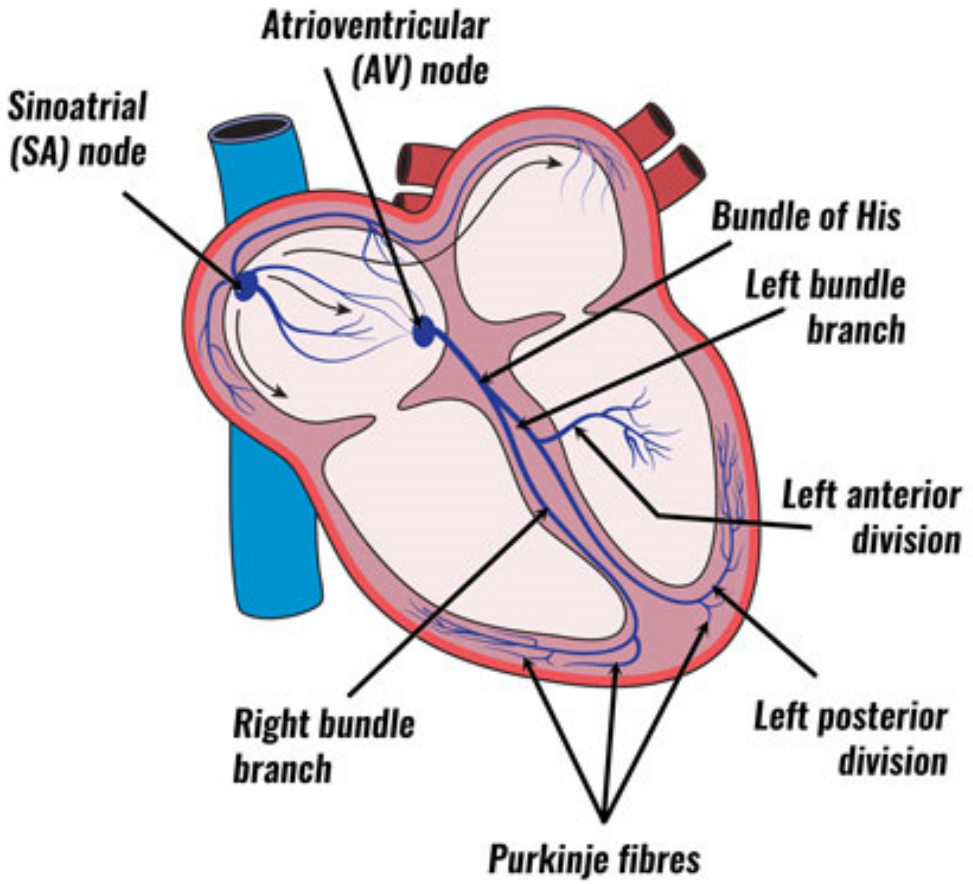
Junctional Rhythm

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



Review

- Basics of electrophysiology
- Action Potential
- The ECG
- Brady arrhythmia’s
- AV Blocks
- Bundle Branch Blocks
- ST elevation myocardial infarction/ Non ST elevation myocardial infarction
- Ectopics and Tachy arrhythmia’s
- Atrial fibrillation / flutter



AV BLOCKS

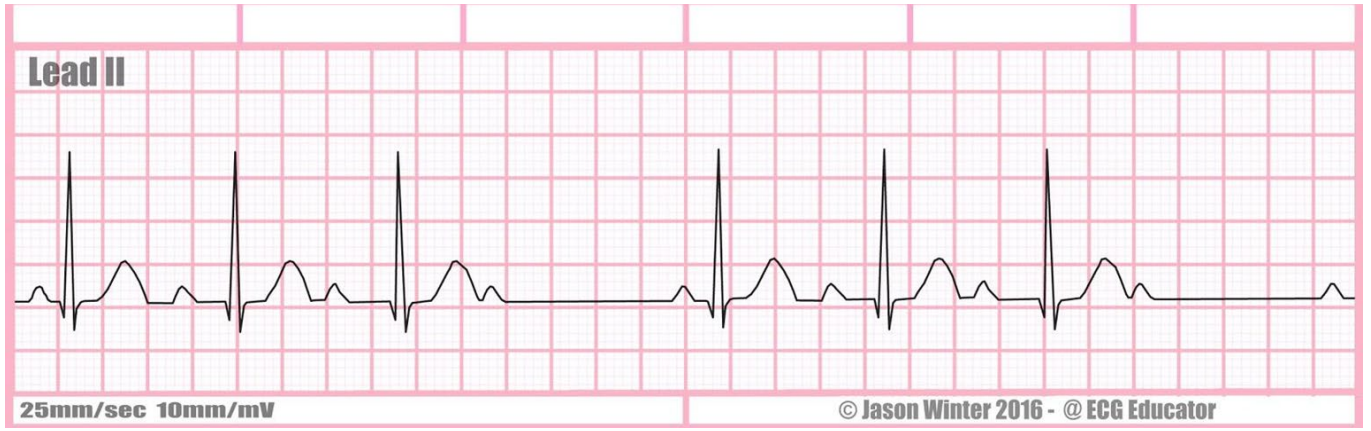
- 1 deg AV Block
- 2 deg AV Block
- 3 deg/ complete AV Block

1ST degree AV block

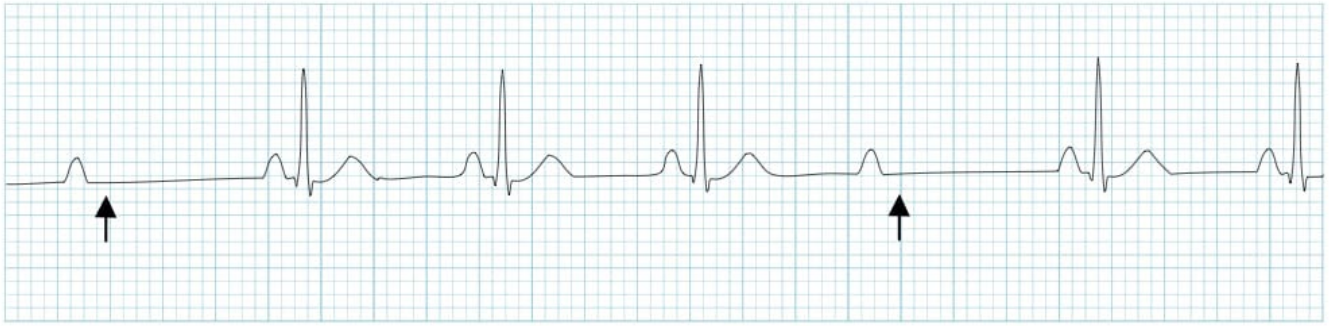
- PR interval >200 ms
- Rarely symptomatic
- Usually improve on exercise

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

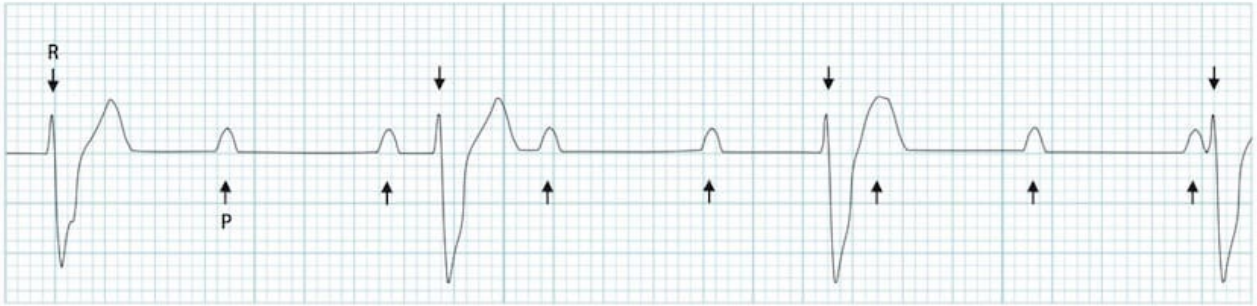


2nd degree AV block, Type II Mobitz



- 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

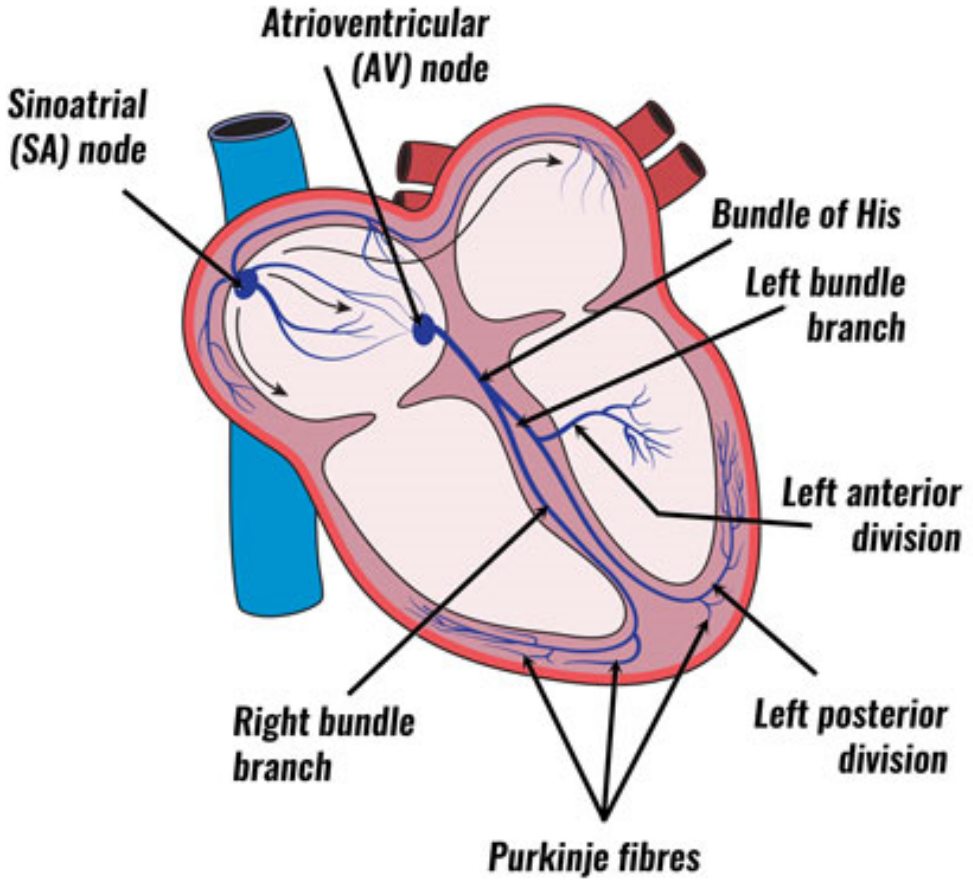
Complete or 3rd degree AV Block



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

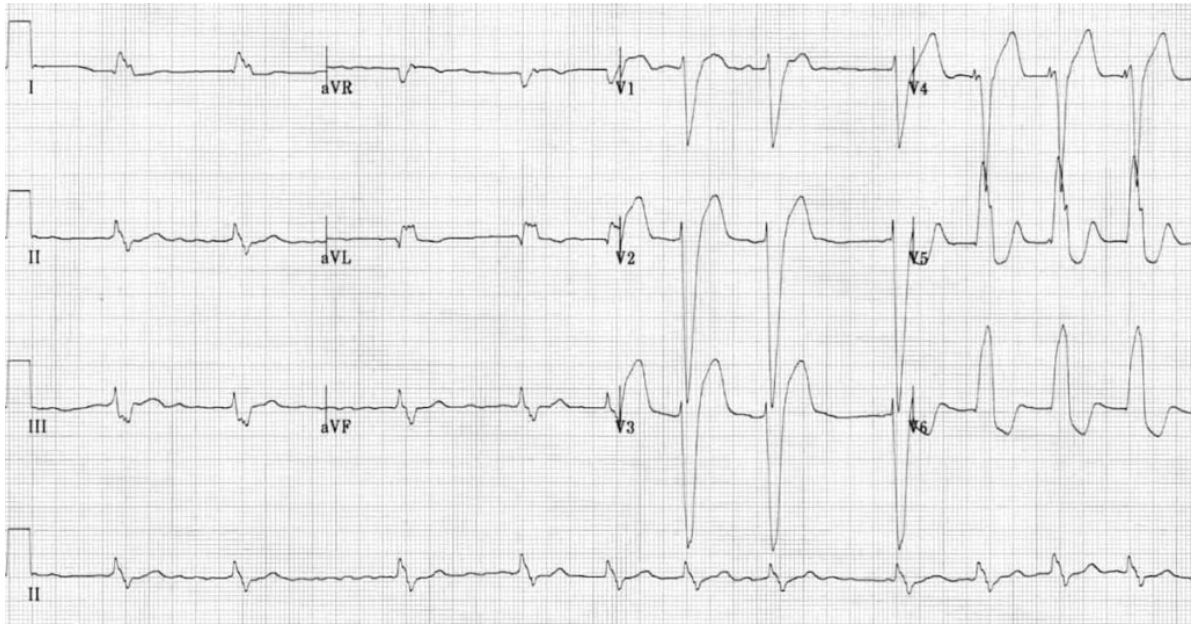
Review

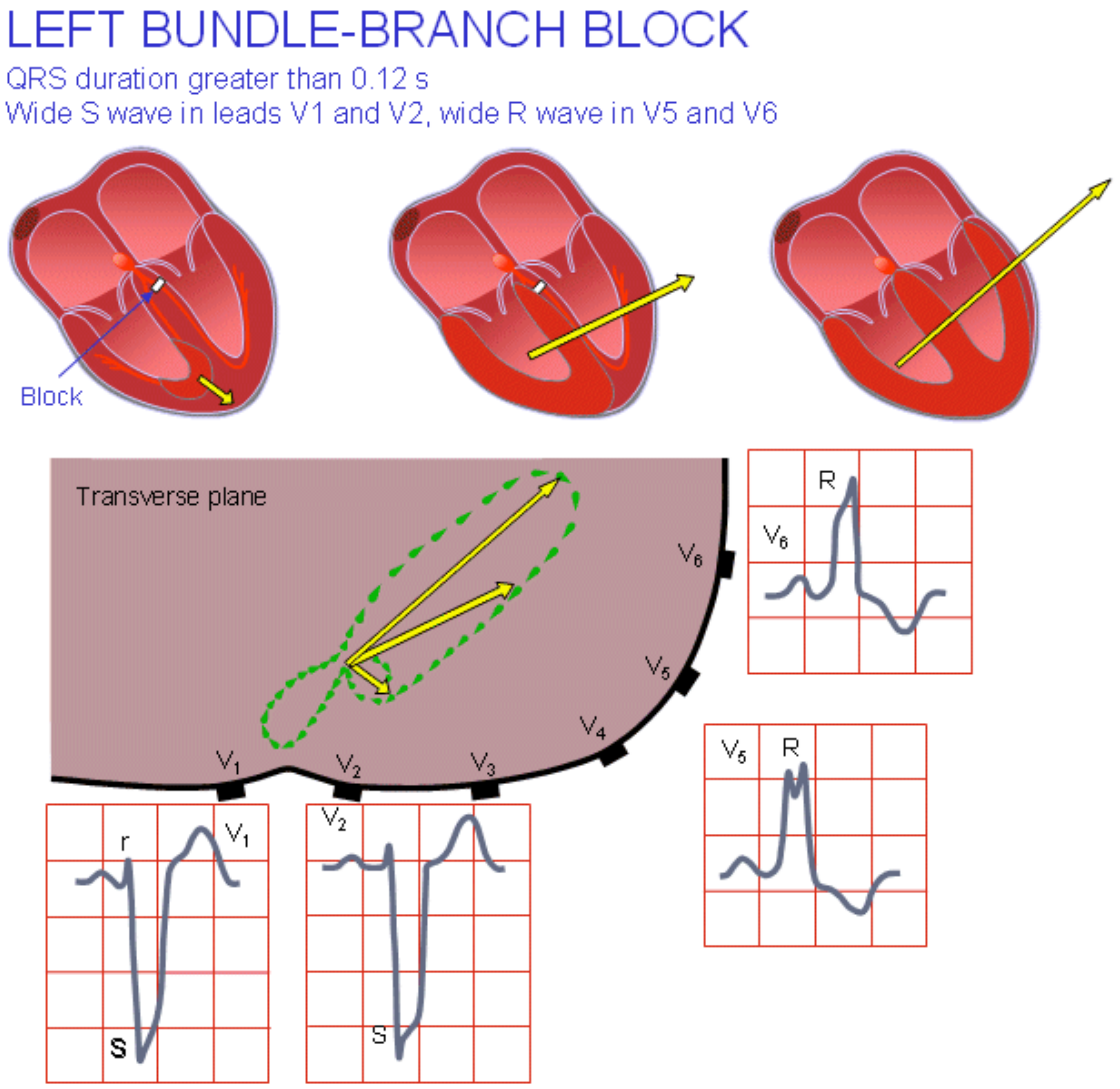
- Basics of electrophysiology
- Action Potential
- The ECG
- Brady arrhythmia's
- AV Blocks
- Bundle Branch Blocks
- ST elevation myocardial infarction/ Non ST elevation myocardial infarction
- Ectopics and Tachy arrhythmia's
- Atrial fibrillation / flutter



Left Bundle Branch Block

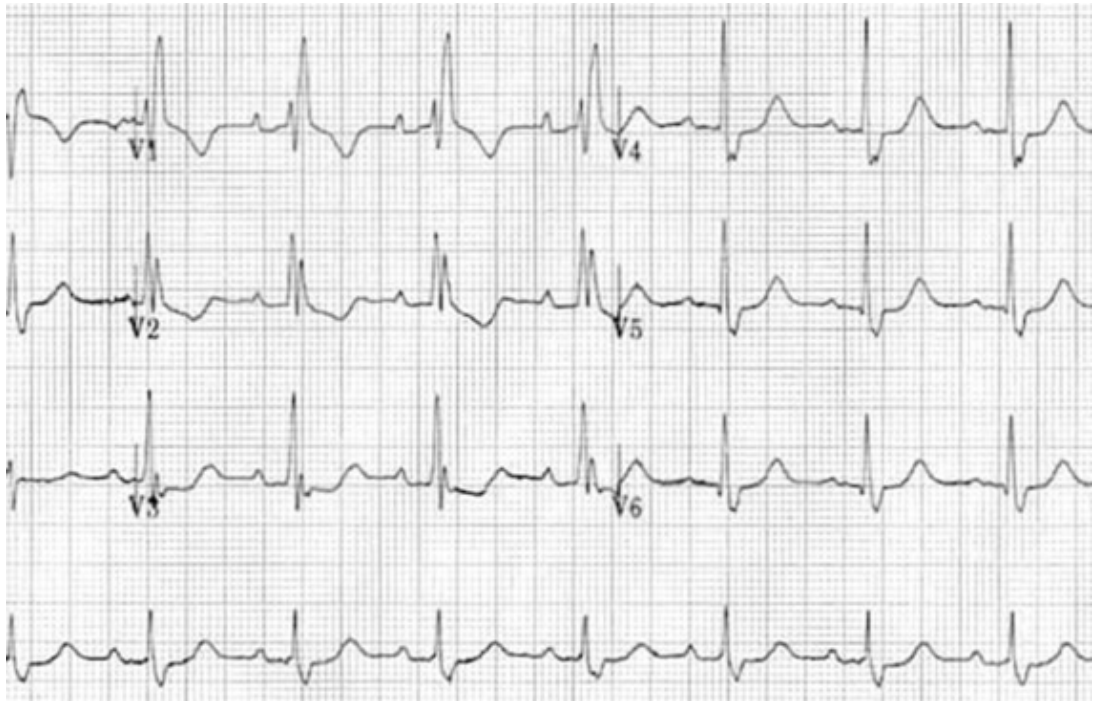
- Wide QRS
- Negative in V1

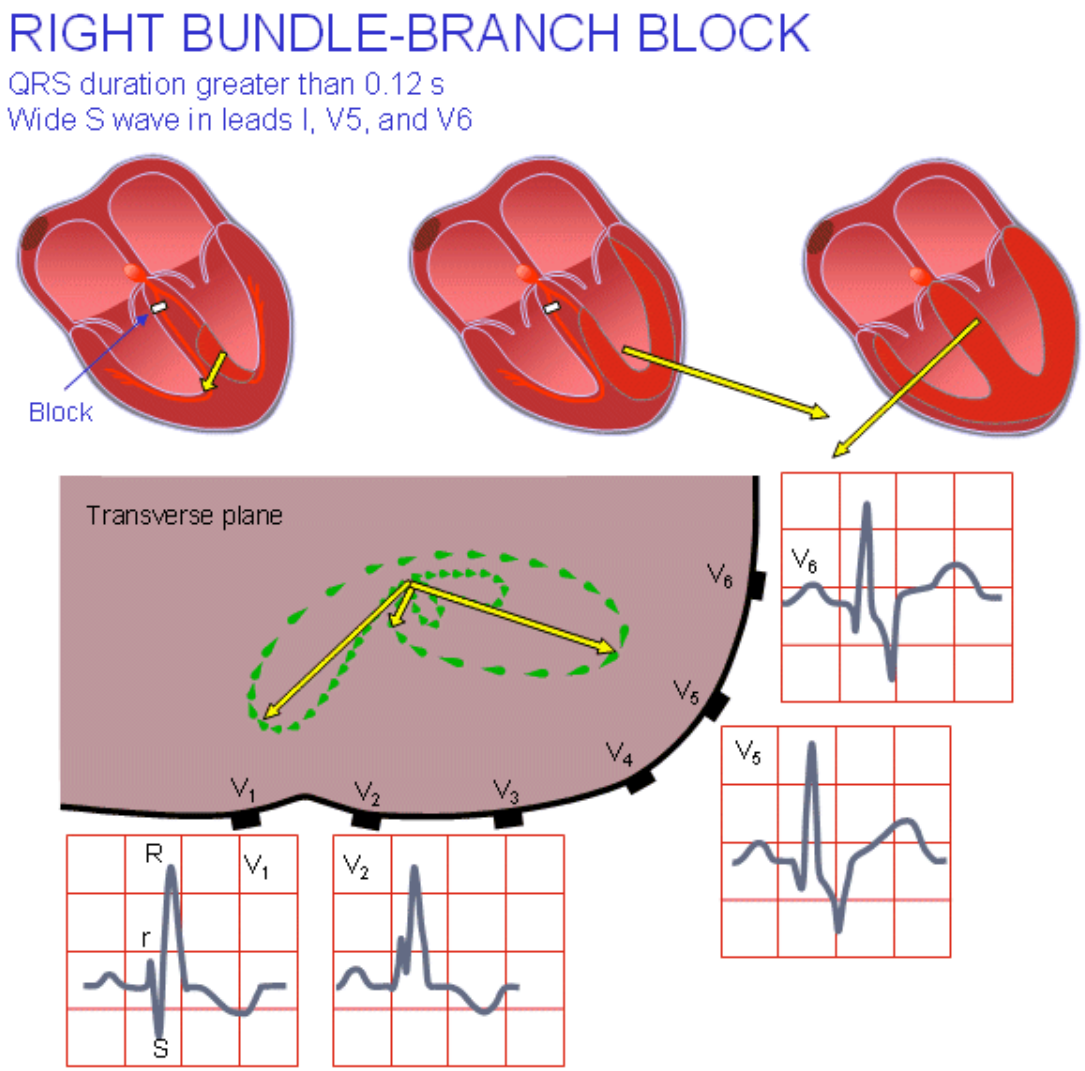




Right Bundle Branch Block

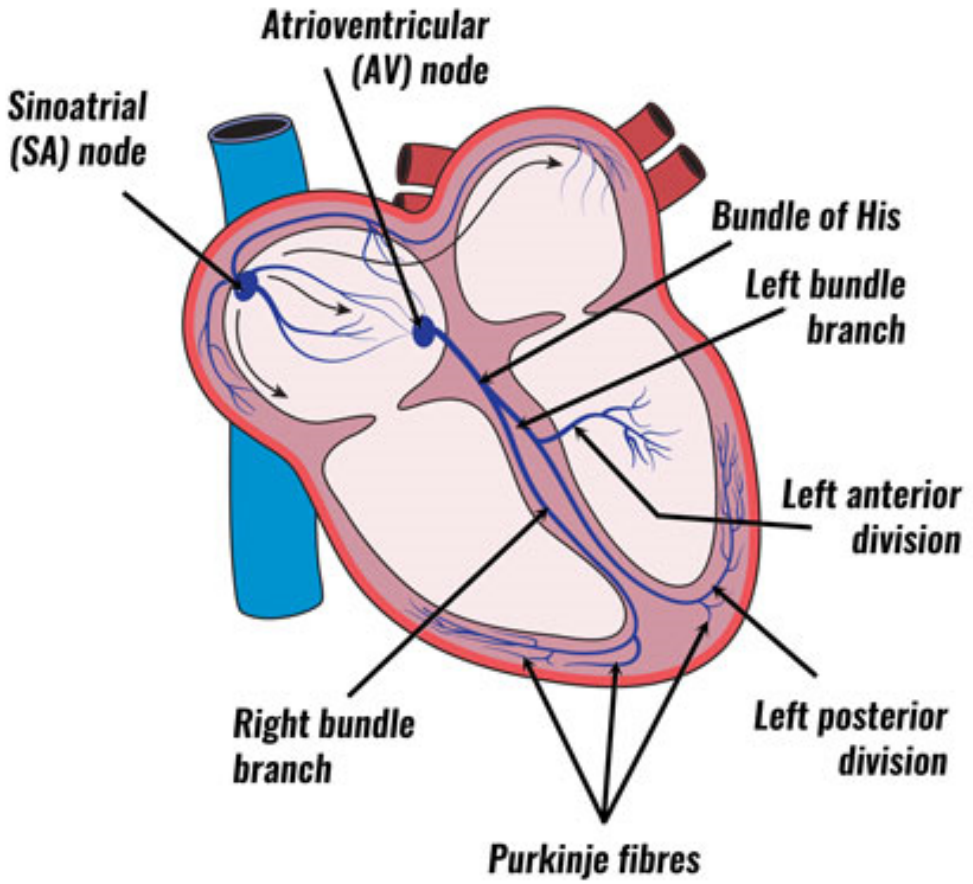
- Dominant R wave in V 1
- RsR pattern
- S wave in lateral leads, V5, V6





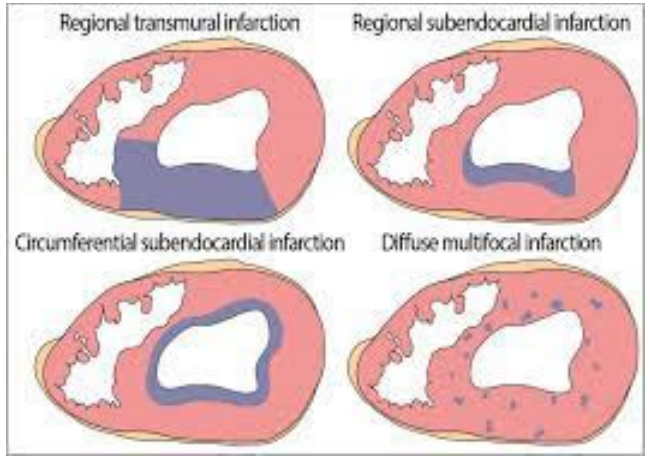
Review

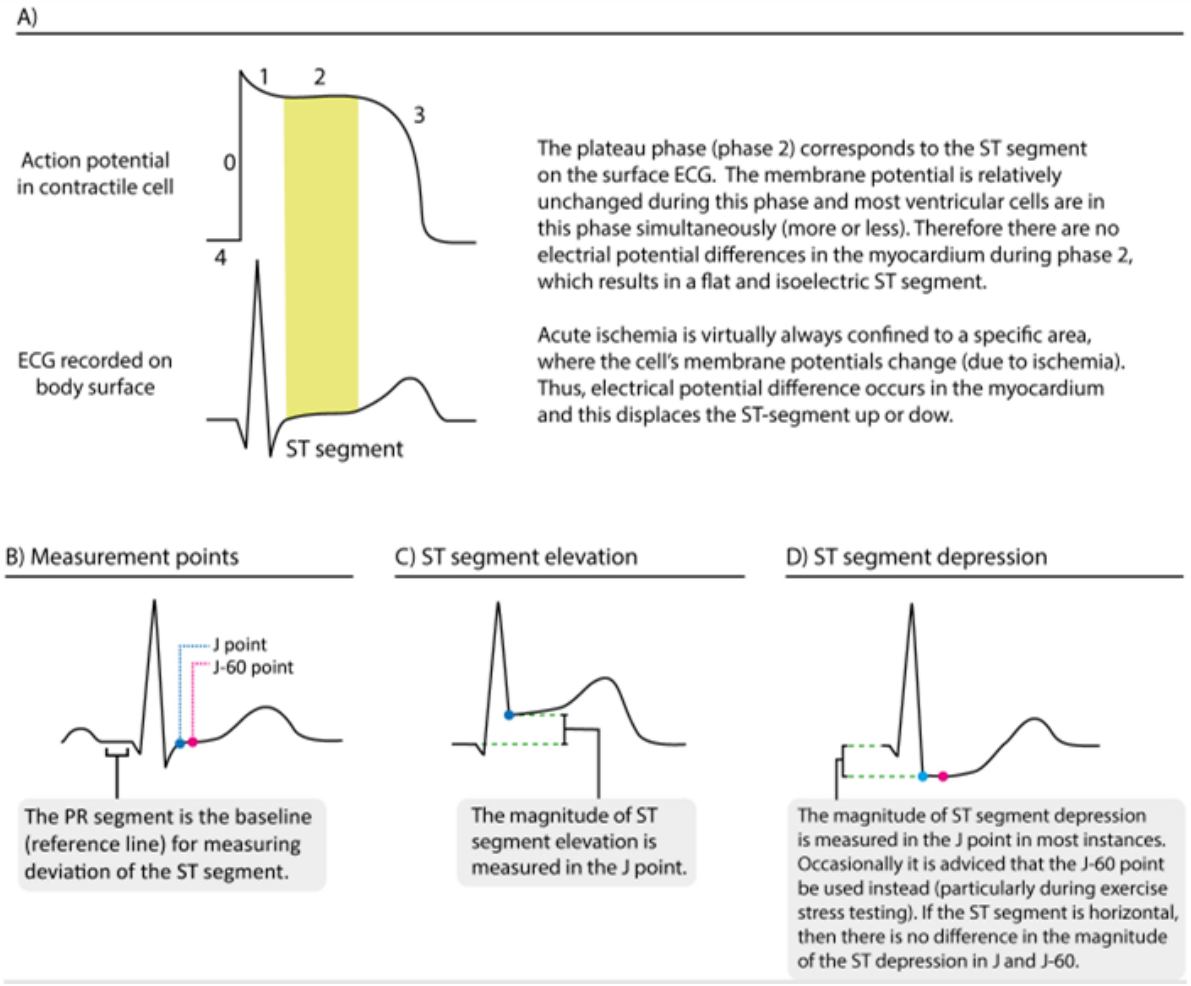
- Basics of electrophysiology
- Action Potential
- The ECG
- Brady arrhythmia's
- AV Blocks
- Bundle Branch Blocks
- ST elevation myocardial infarction/ Non ST elevation myocardial infarction
- Ectopics and Tachy arrhythmia's
- Atrial fibrillation / flutter

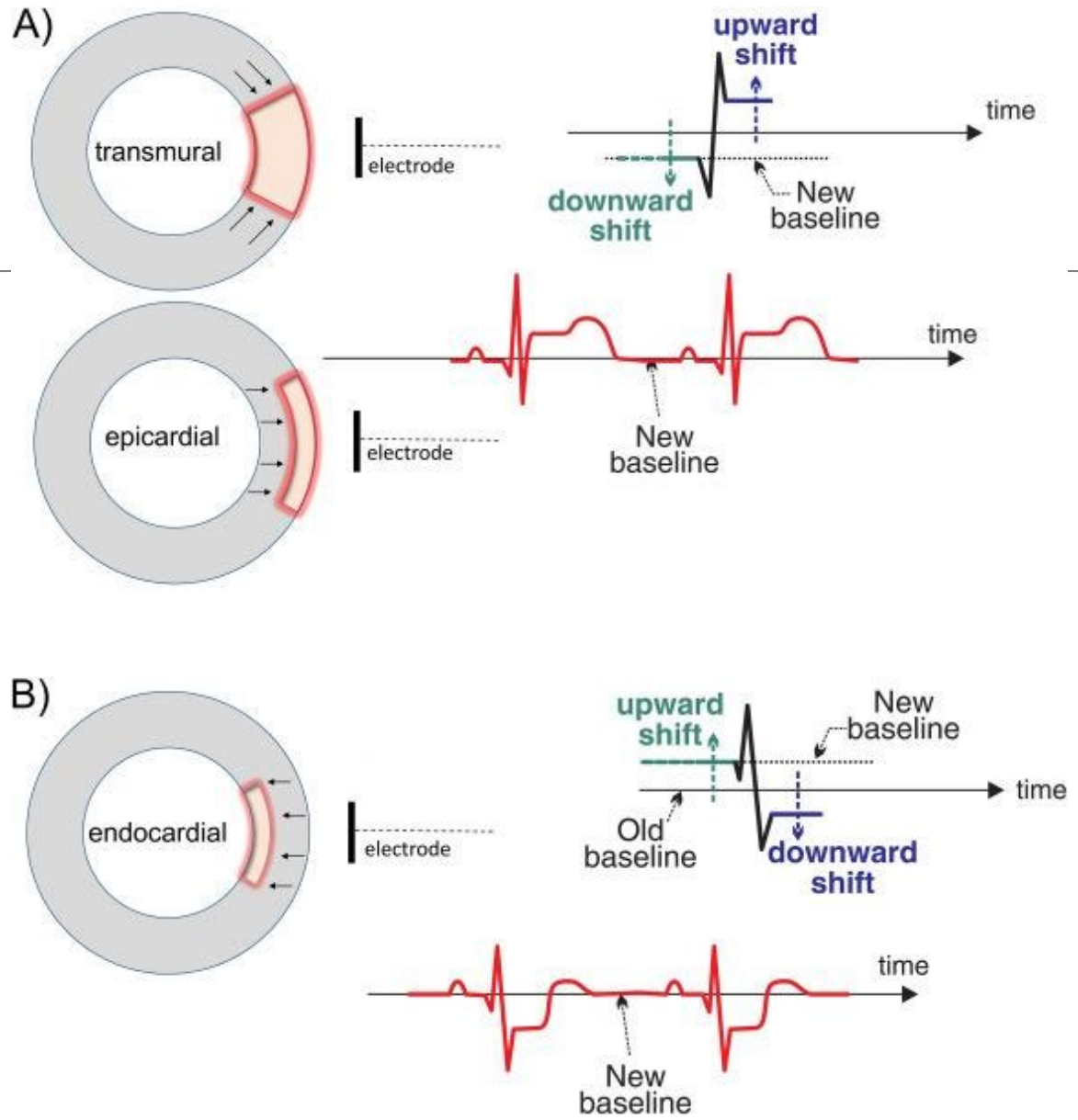


Ischemia

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

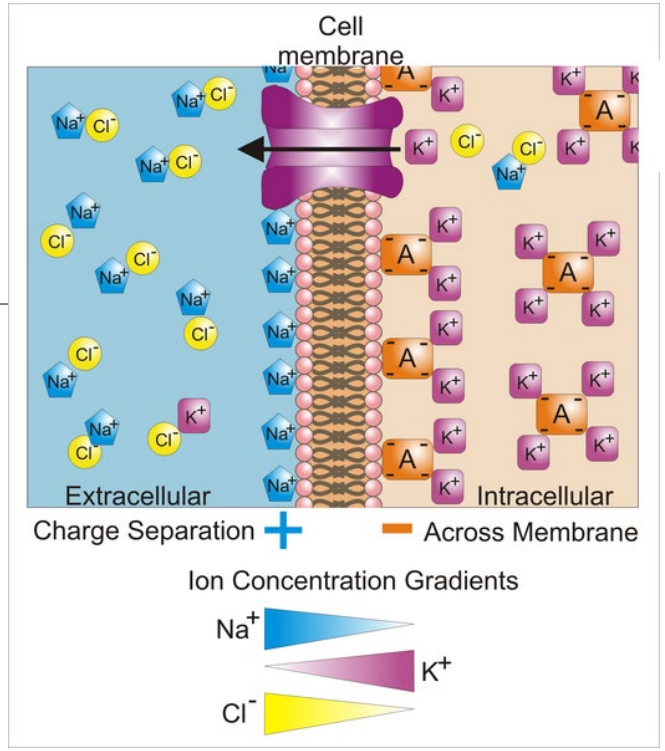


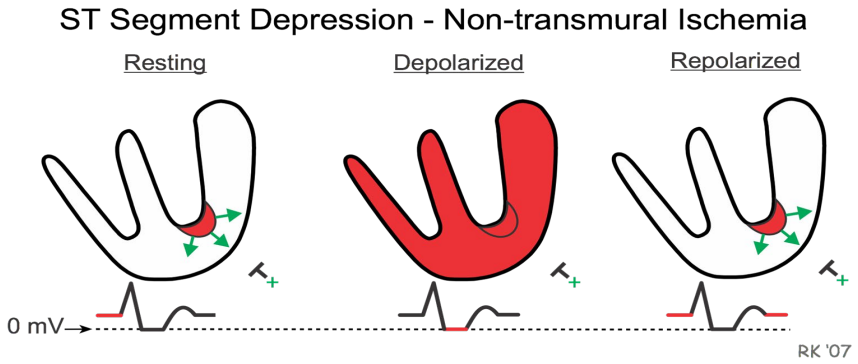


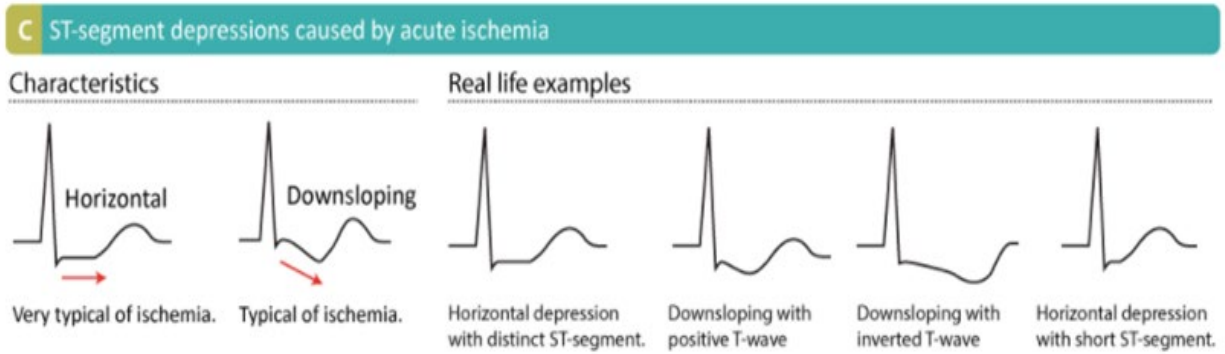

A)
transmural
electrode
upward shift
time
downward shift
New baseline
epicardial
electrode
time
New baseline
B)
endocardial
electrode
upward shift
New baseline
time
Old baseline
downward shift
time
New baseline

Non ST elevation myocardial infarction (NSTEMI)

- Ischemia causes cells to have a short action potential
- Earlier than normal depolarization
- ECG wave front moves towards the normal myocardium
- Characterized by horizontal or down slopping depression

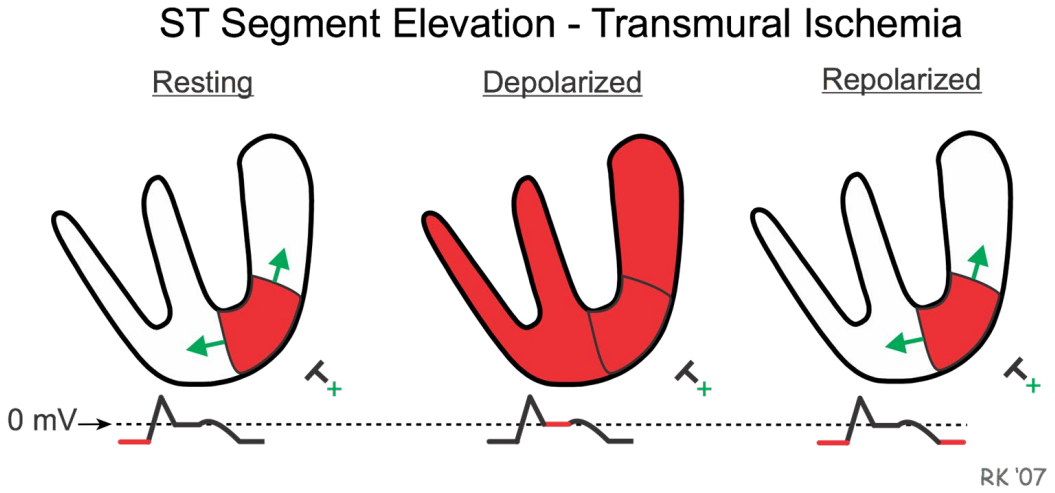


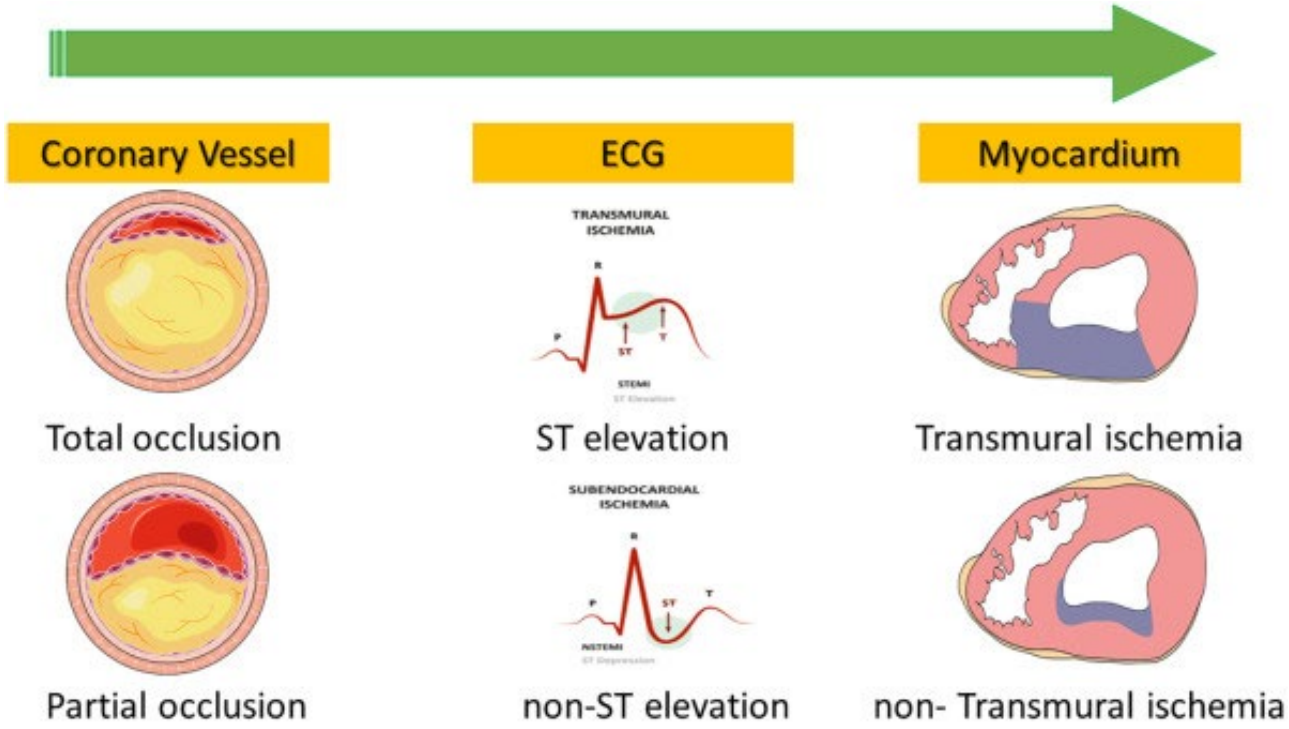




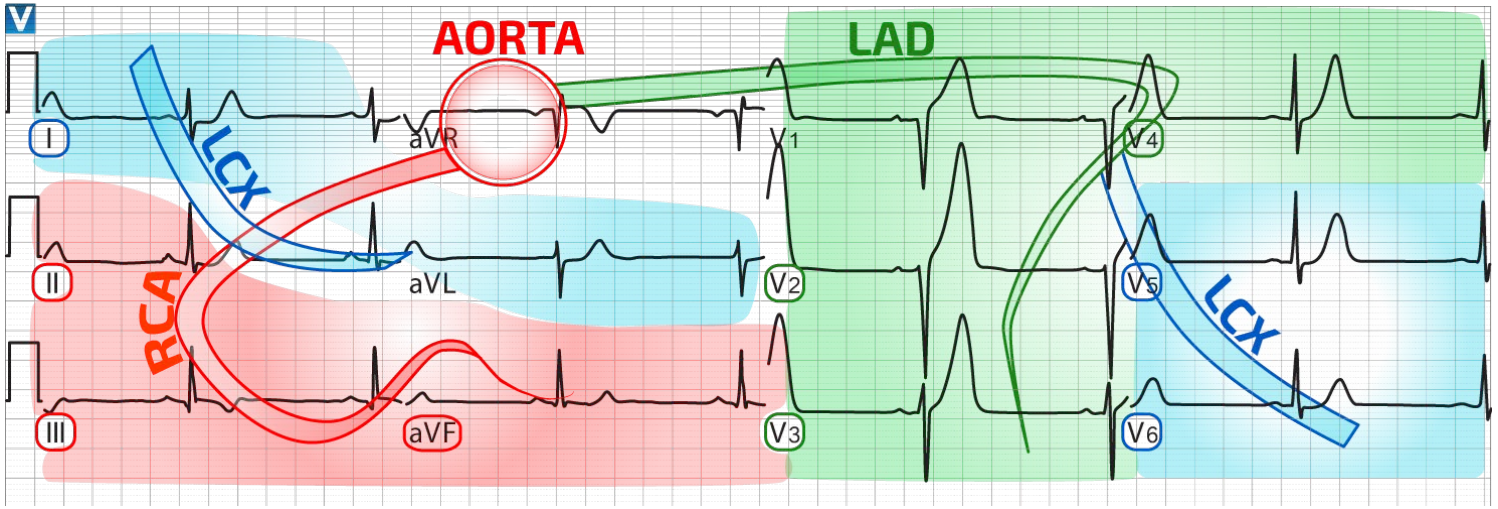
ST elevation myocardial infarction (STEMI)

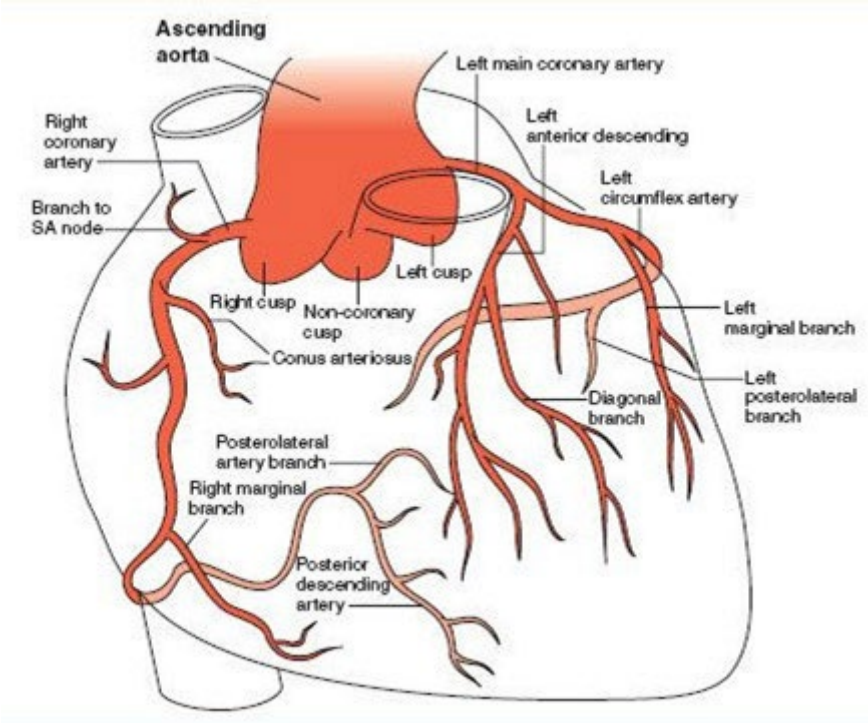
- Ischemic event that causes damage to the entire tissue, transmural injury
- Due to abrupt blockage of a coronary artery
- Earlier than normal depolarisation
- ECG wave front moves away from the injured myocardium towards normal tissue




Coronary Vessel
ECG
Myocardium
TRANSMURAL ISCHEMIA
R
P
ST
T
STEMI
Total occlusion
ST elevation
Transmural ischemia
SUBENDOCARDIAL ISCHEMIA
R
P
ST
T
NSTEMI
Partial occlusion
non-ST elevation
non- Transmural ischemia

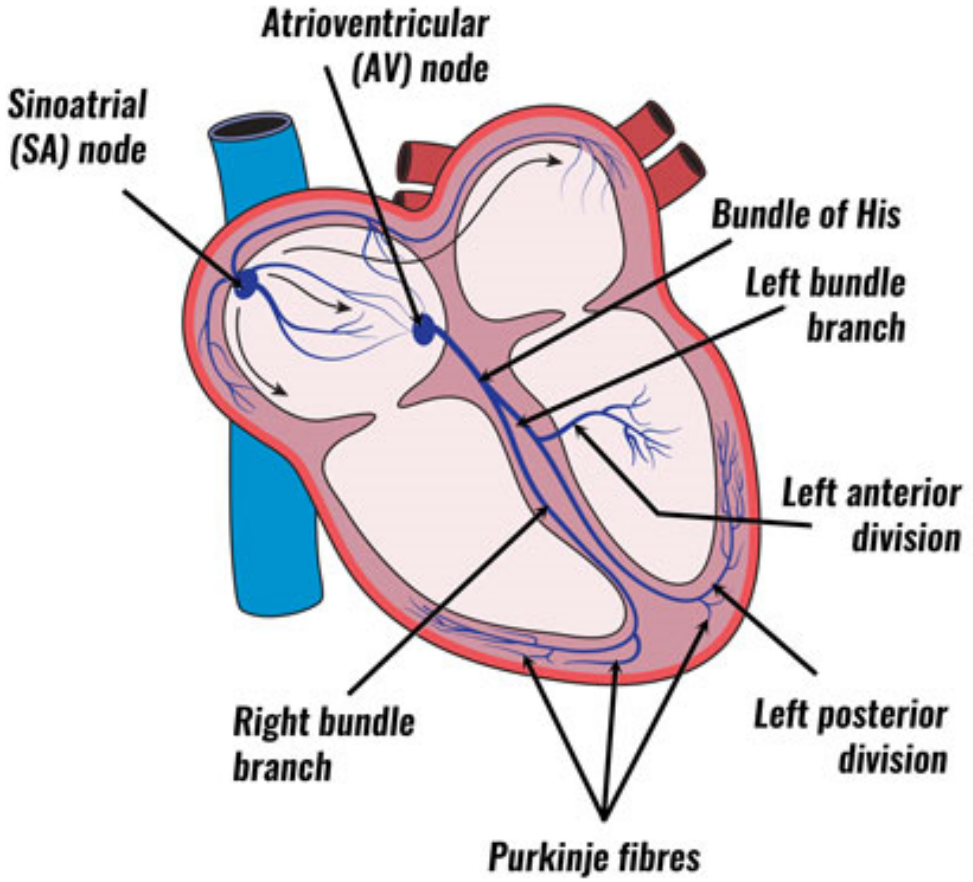
ECG leads and coronary arteries





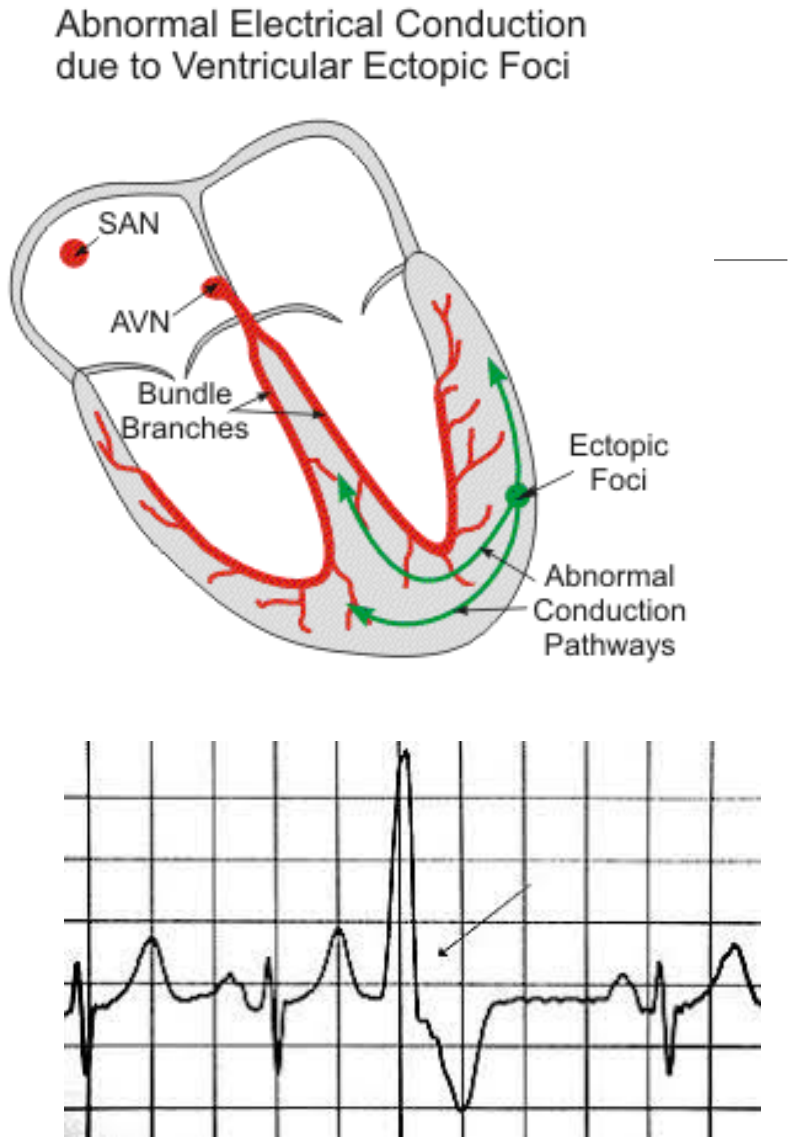
Review

- Basics of electrophysiology
- Action Potential
- The ECG
- Brady arrhythmia's
- AV Blocks
- Bundle Branch Blocks
- ST elevation myocardial infarction/ Non ST elevation myocardial infarction
- Ectopics and Tachy arrhythmia's
- Atrial fibrillation / flutter



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



Tachyarrythmias (FAST)

Ventricular arrhythmias

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

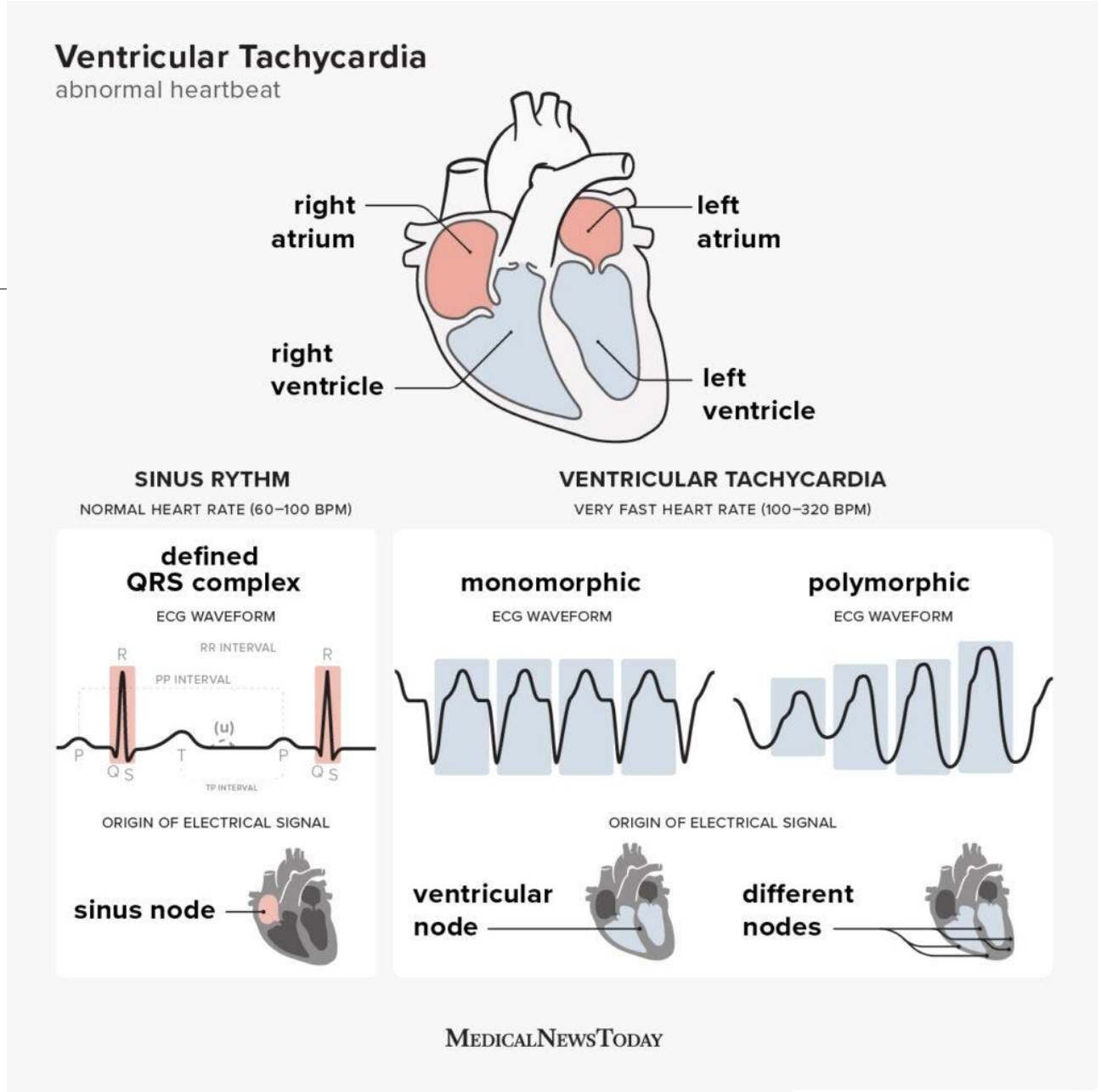
Monomorphic

- Uniform stable QRS morphology

Polymorphic

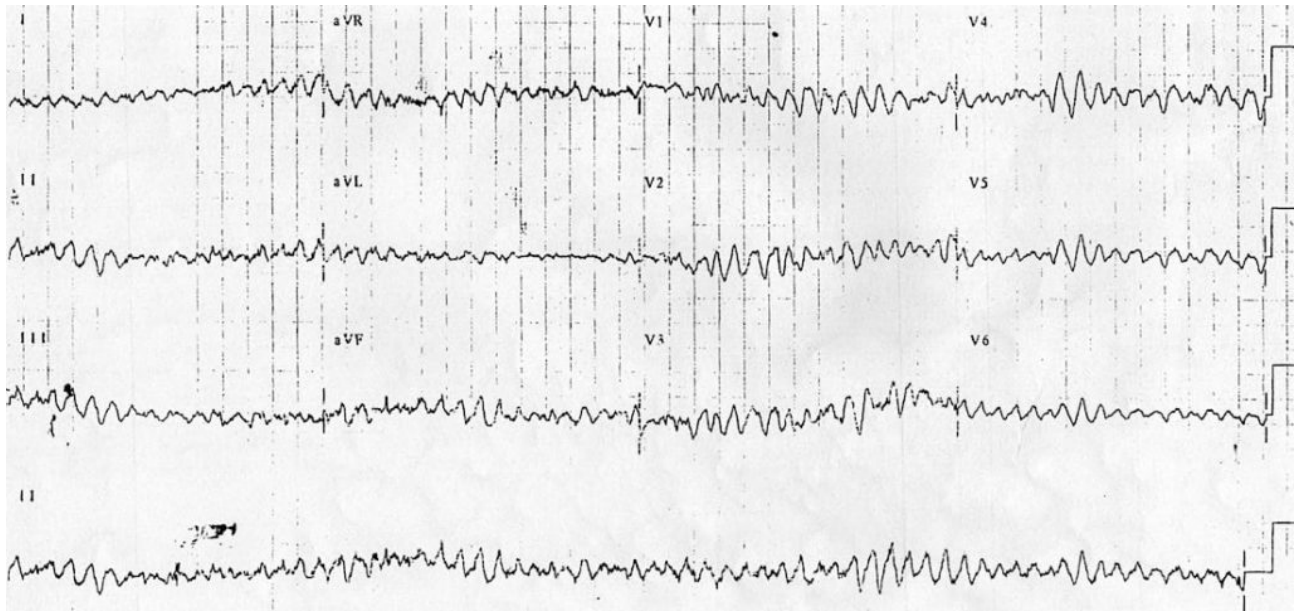
- 200 bpm
- Hemodynamically unstable
- Can degenerate into VF

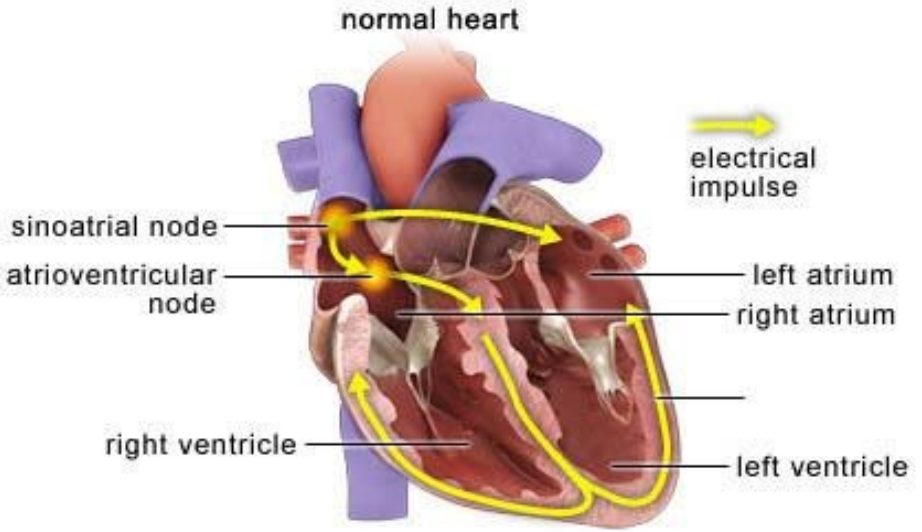
Causes :
Idiopathic
Ischemia, scar
Myopathy – dilated , HCM
Infiltration – Sarcoidosis , Amyloid
Long QT / Brugada syndrome

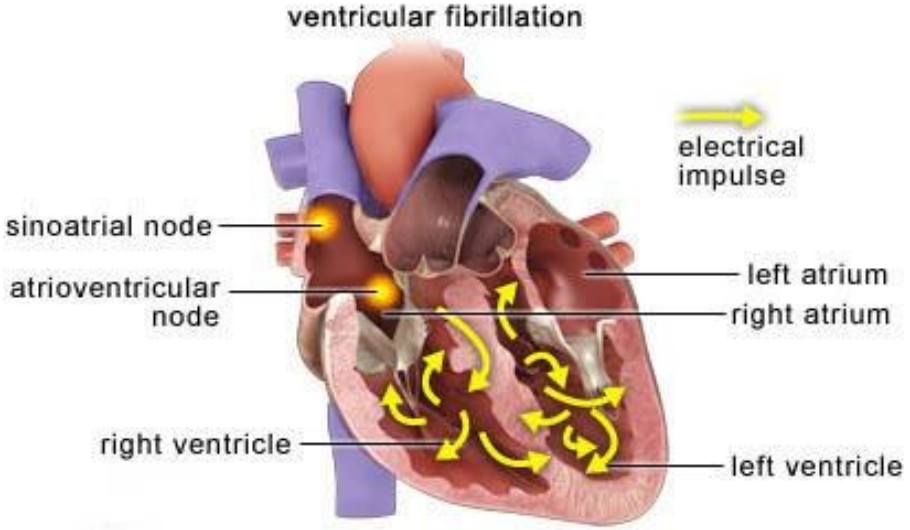


Ventricular fibrillation

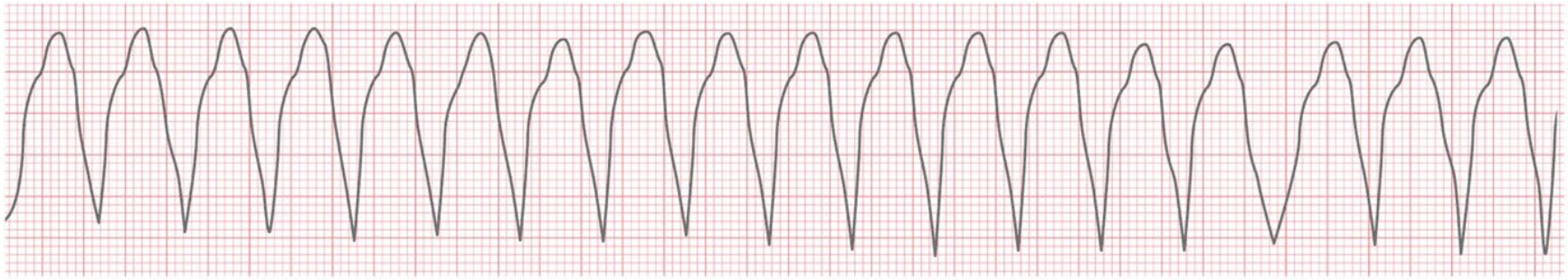
- No uniform ventricular depolarization
- 300 bpm, irregular
- Most common cause of sudden cardiac death
- Can cause syncope
- Treatment – defibrillator / anti-arrhythmic therapy



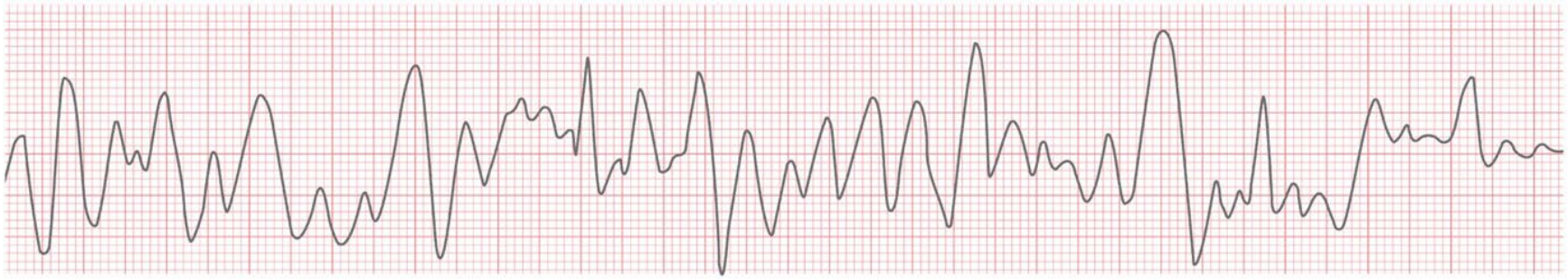




Monomorphic ventricular tachycardia

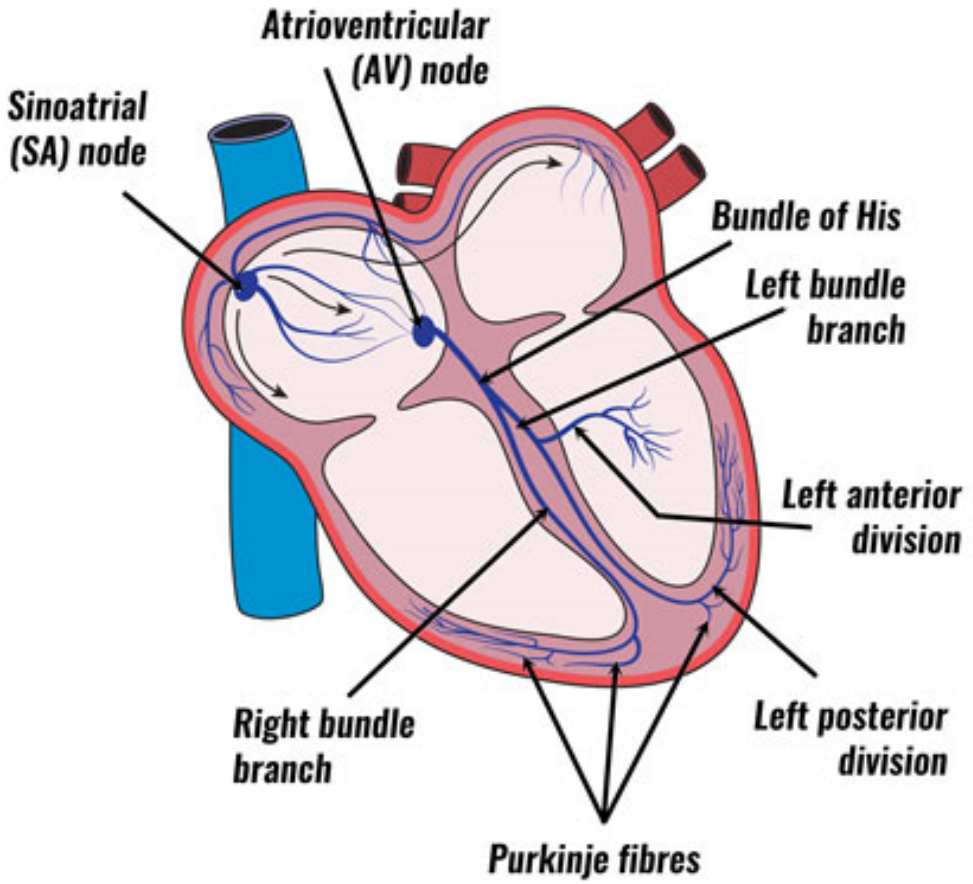


Polymorphic ventricular tachycardia

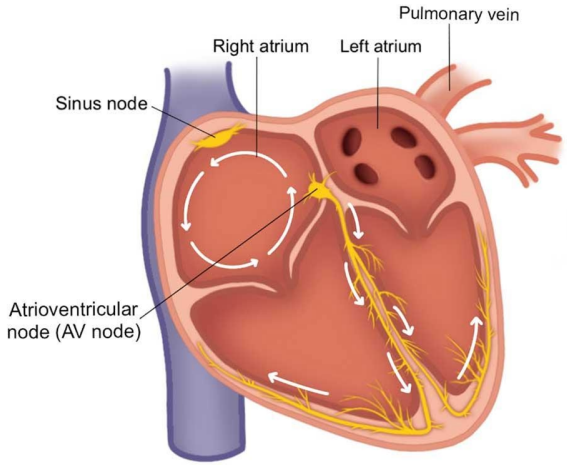


Review

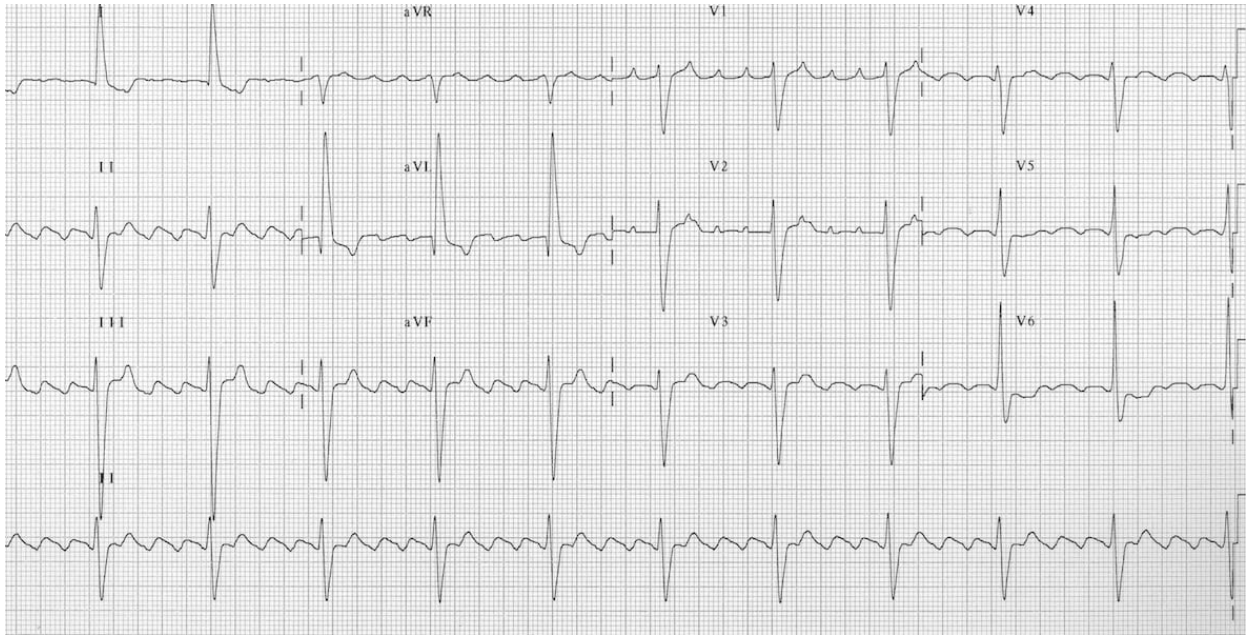
- Basics of electrophysiology
- Action Potential
- The ECG
- Brady arrhythmia's
- AV Blocks
- Bundle Branch Blocks
- ST elevation myocardial infarction/ Non ST elevation myocardial infarction
- Ectopics and Tachy arrhythmia's
- Atrial fibrillation / flutter



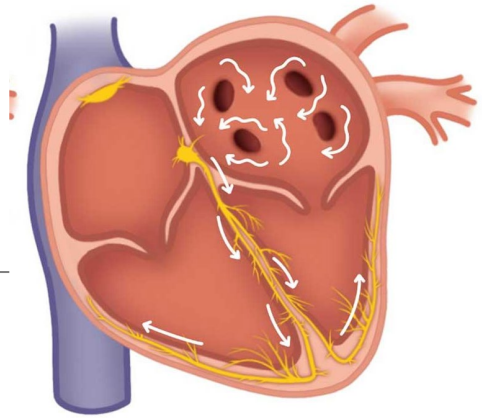
Atrial Flutter



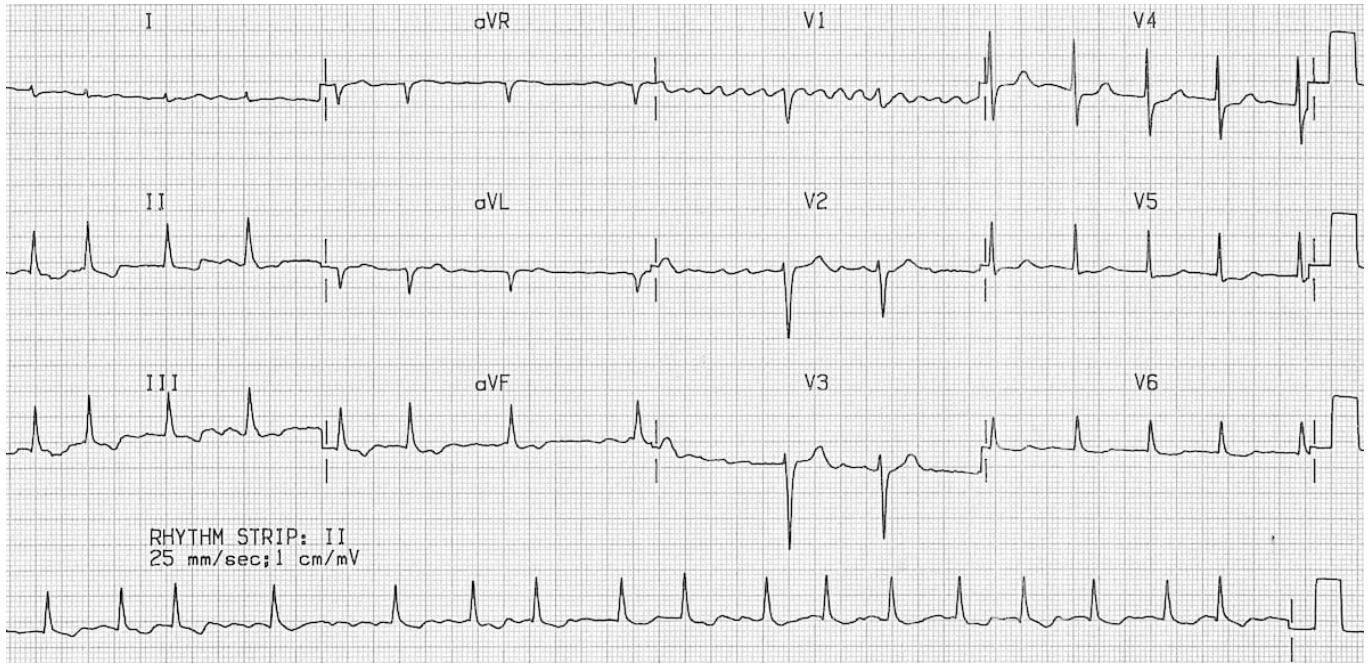
- Narrow complex tachycardia
- Regular atrial activity at ~300 bpm
- Loss of the isoelectric baseline
- “Saw-tooth” pattern of inverted flutter waves in leads II, III, aVF
- Upright flutter waves in V1 that may resemble P waves
- Ventricular rate depends on AV conduction ratio



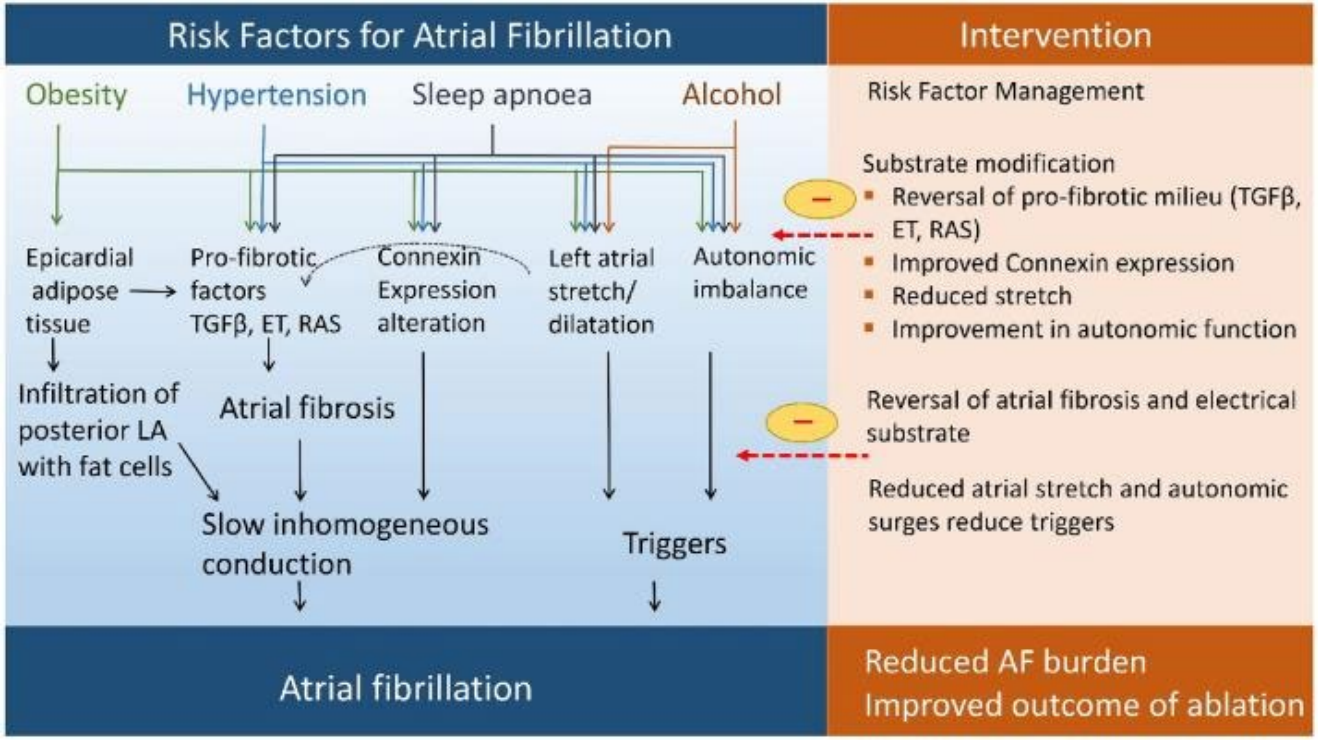
Atrial fibrillation

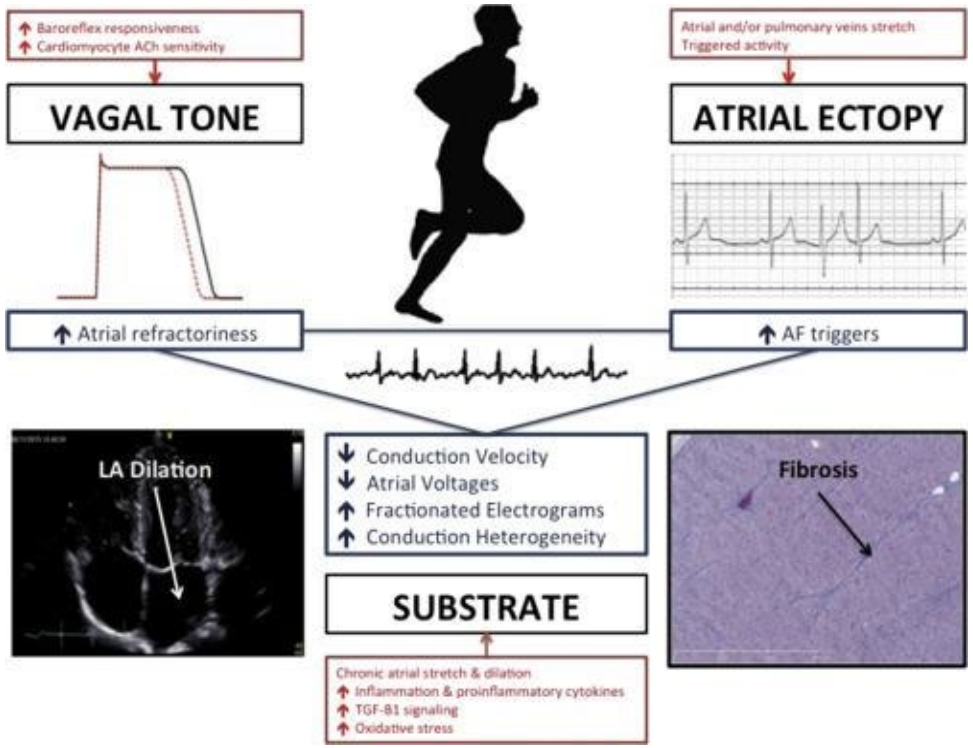


- Irregularly irregular rhythm
- No P waves
- Absence of an isoelectric baseline
- Irregular R-R interval/ventricular rate
- Fibrillatory waves may be present and can be either fine (amplitude < 0.5mm) or coarse (amplitude > 0.5mm)
- Paroxysmal AF – episodes that last less than a week
- Persistent – AF lasting more than a week at a time
- Long standing – continuous AF episodes lasting more than a year
- Chronic AF – where the physicians have accepted that the AF is there and no further attempt to maintain sinus rhythm is made



Risk Factors



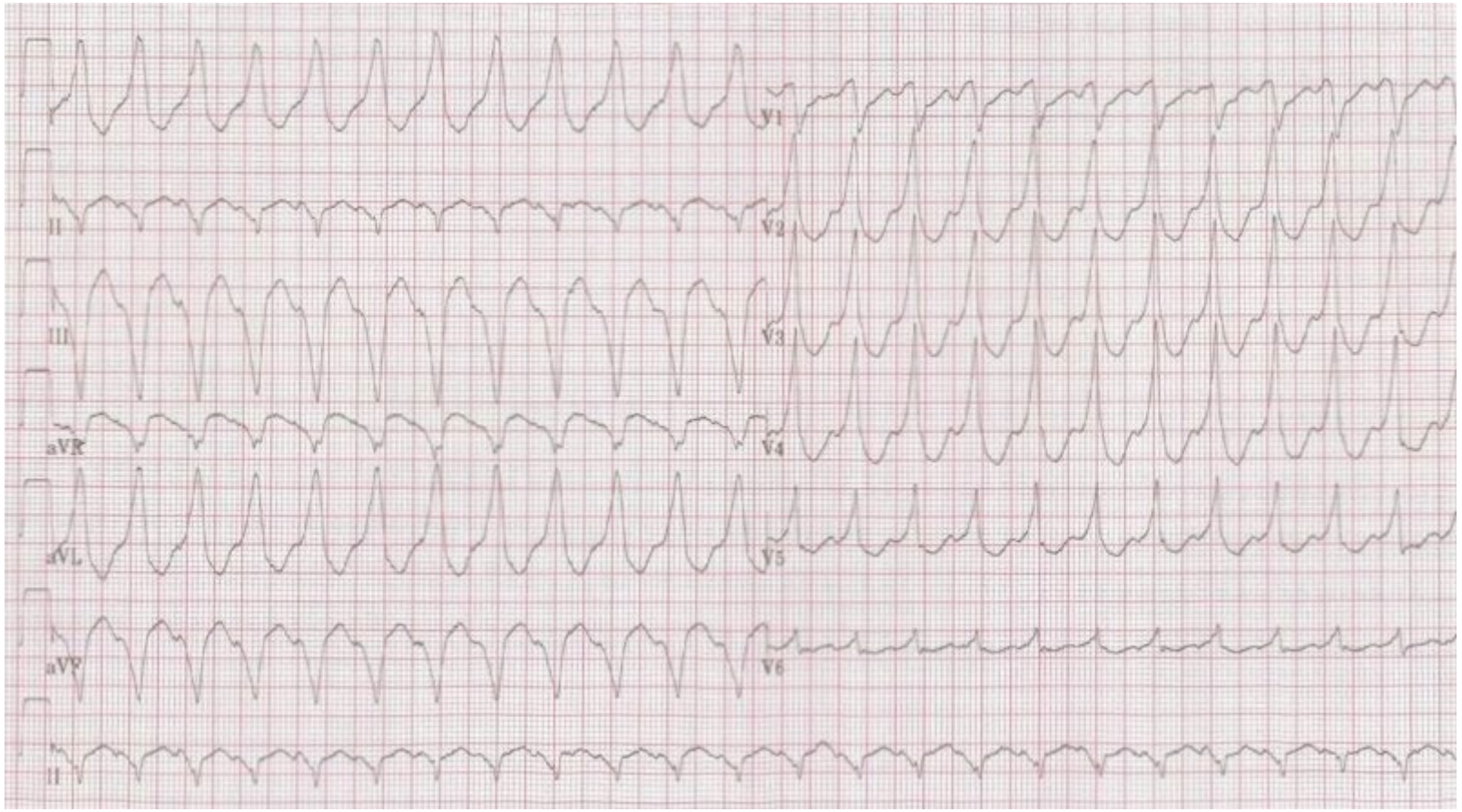


Review

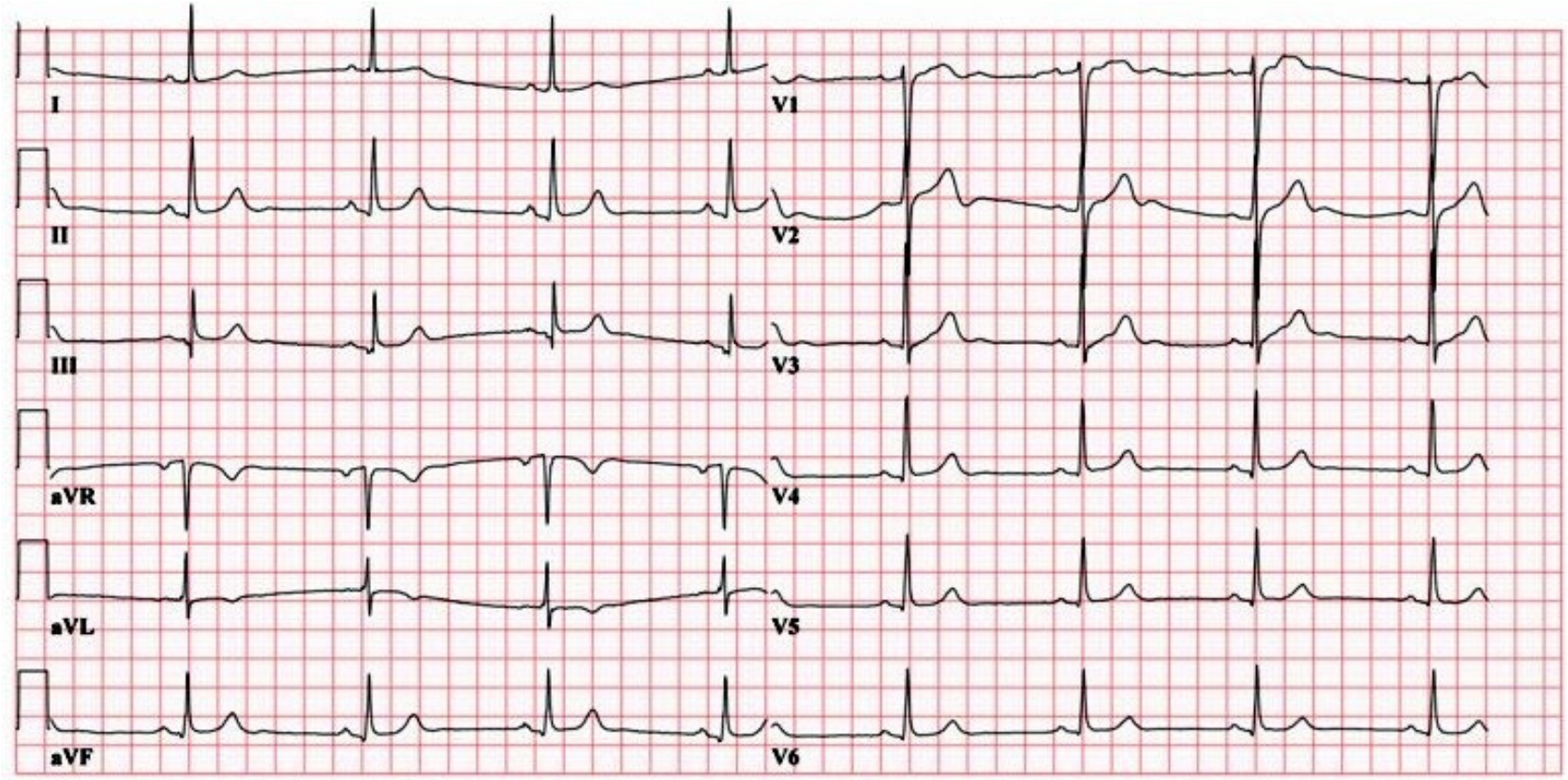
- Brady - sinus node - slow
- AV Block - AV node - slow
- ST depression - Ischemia
- ST elevation - infarction
- Tachy – atria -ventricles- fast
- Ventricular tachy / fibrillation - ventricles fast
- Atrial flutter / fibrillation - atria fast

CASES

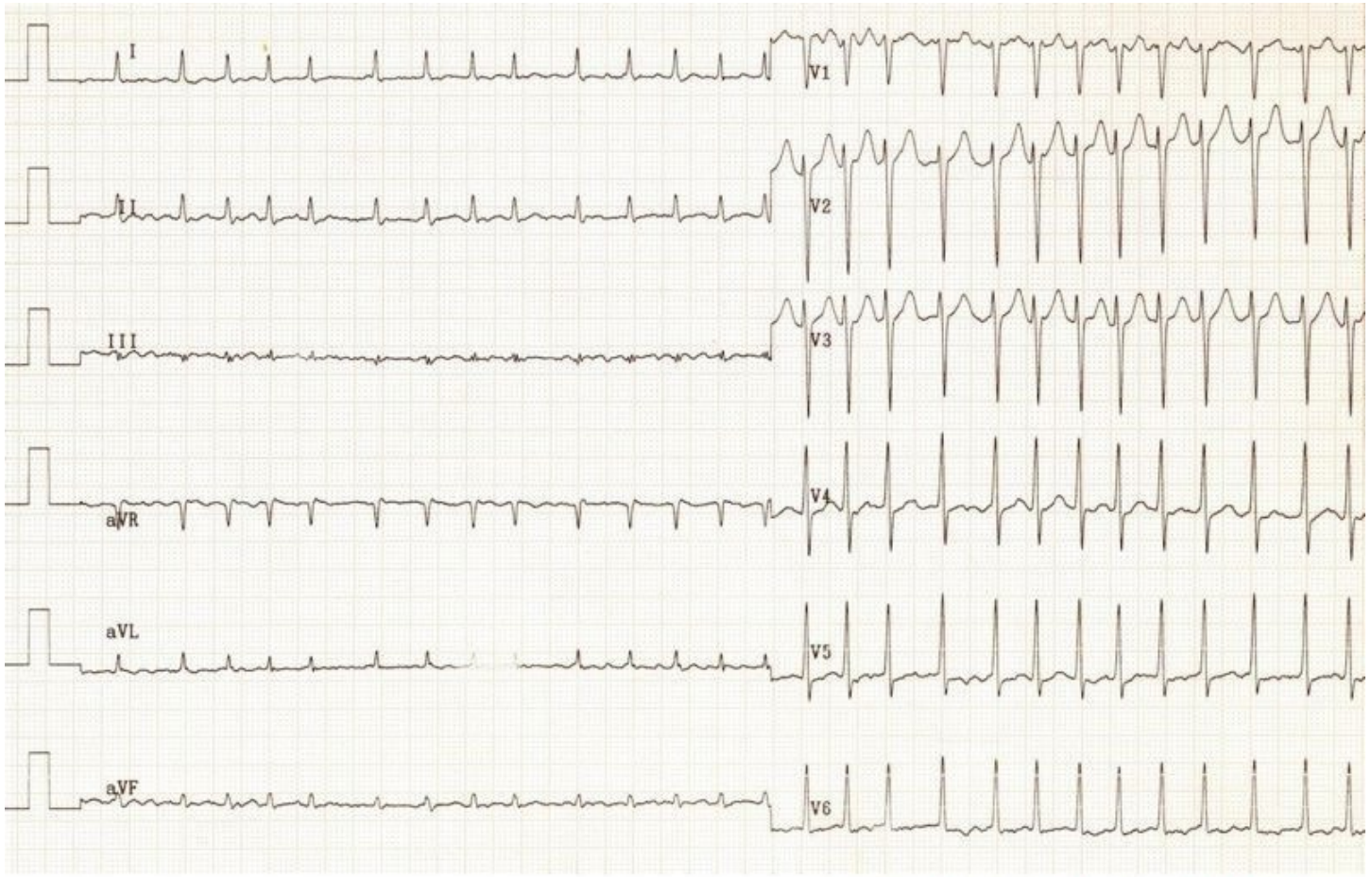
A 65 year old man with a history of ischaemic heart disease is found unresponsive. He has no central pulse and is making no respiratory effort.



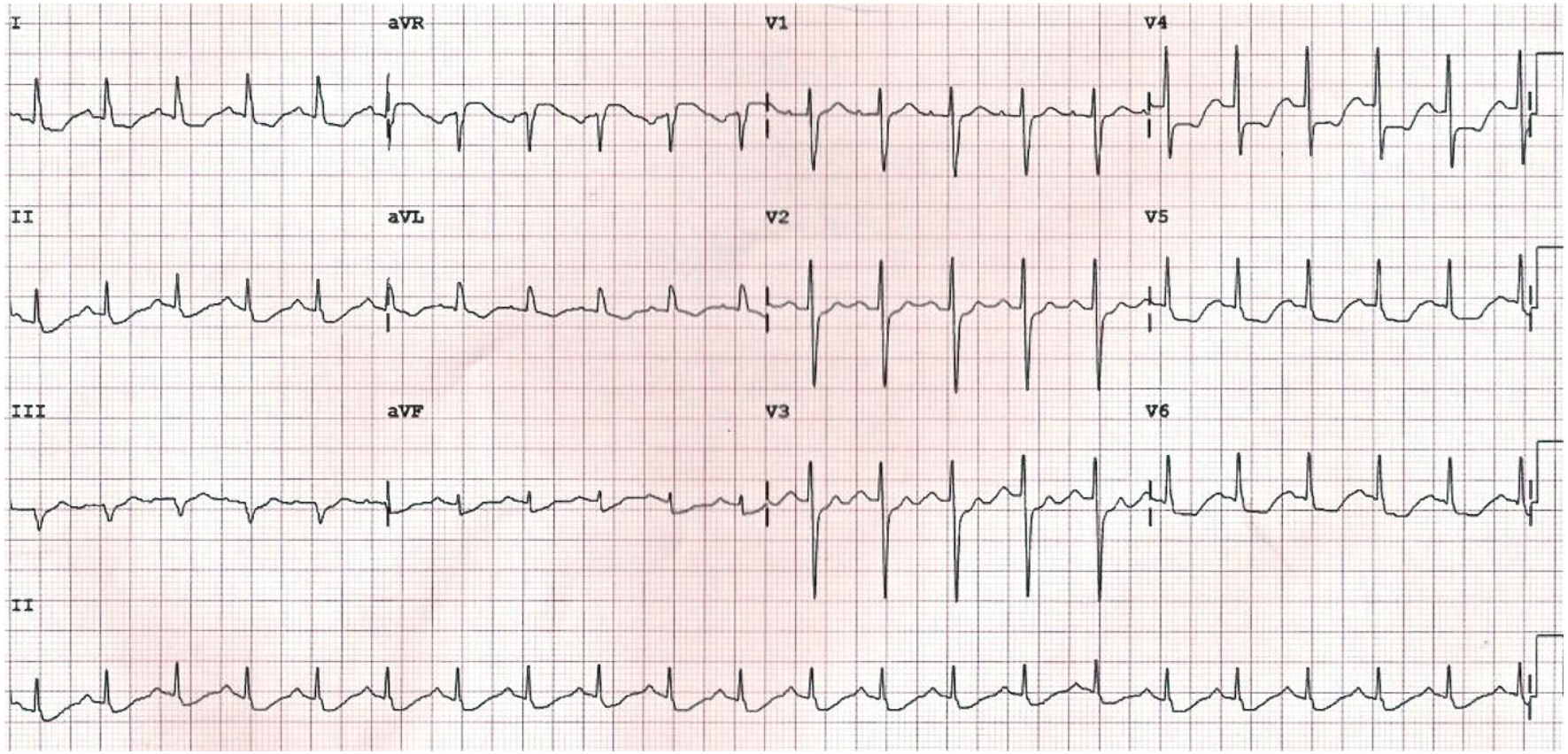
A 72 year old lady presents with collapse. This is her ECG. Present your findings. How would you proceed?



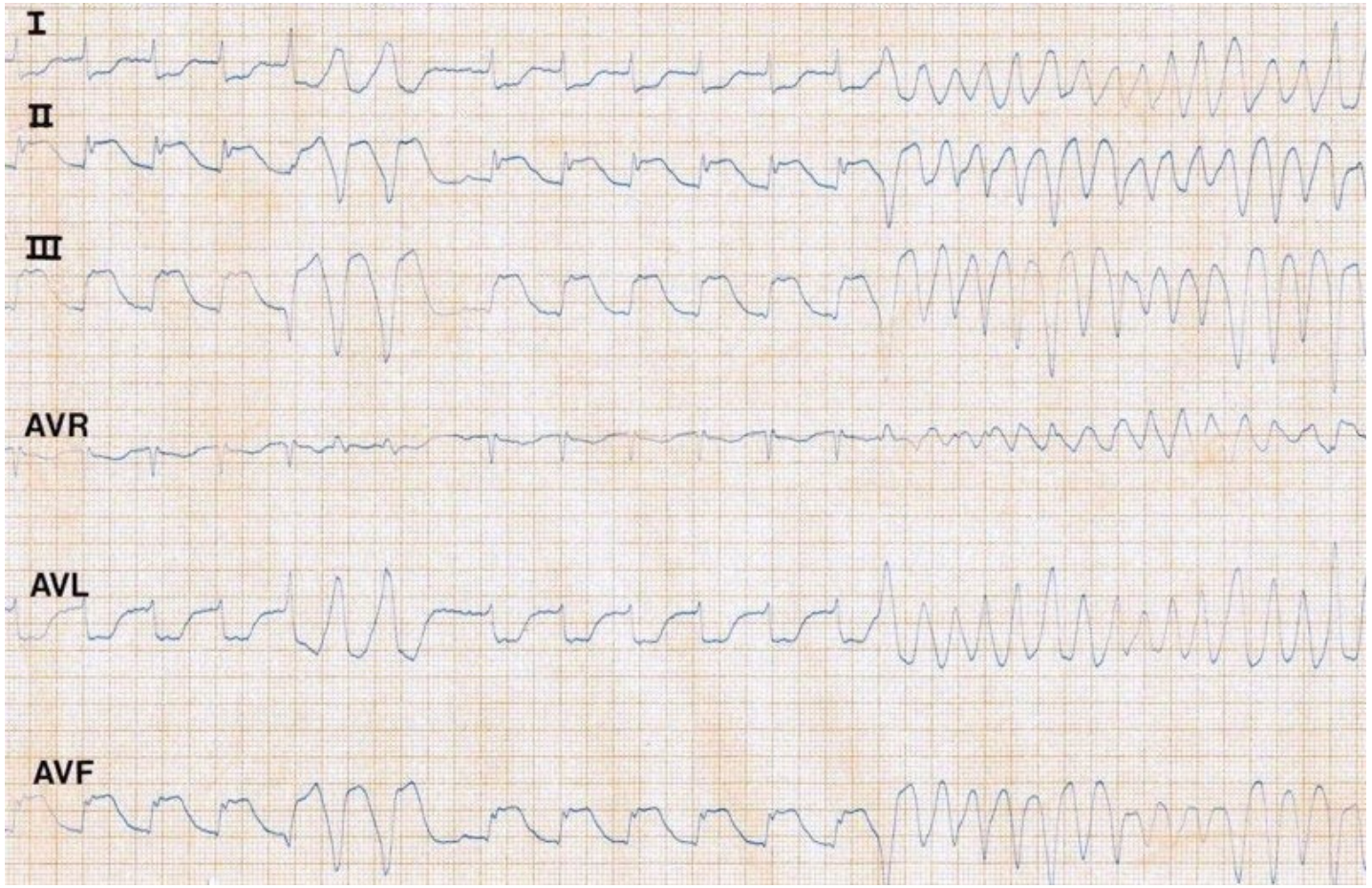
35 yr old male presents to ED, after a night of binge drinking over the weekend

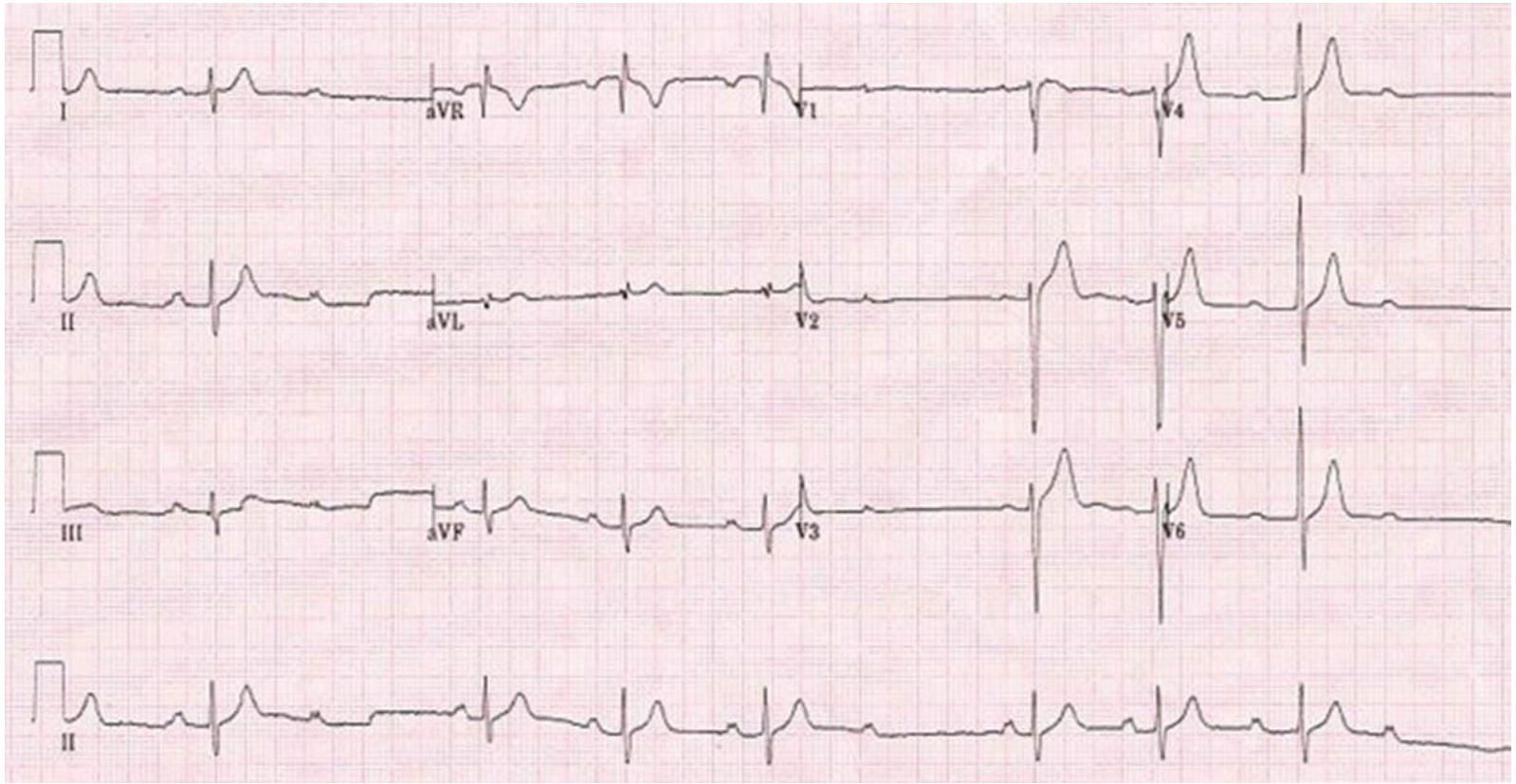


A 65 year old woman presents with chest pain radiating to her jaw and down her left arm. It feels like her ‘normal’ angina but on this occasion it has not eased with GTN spray.

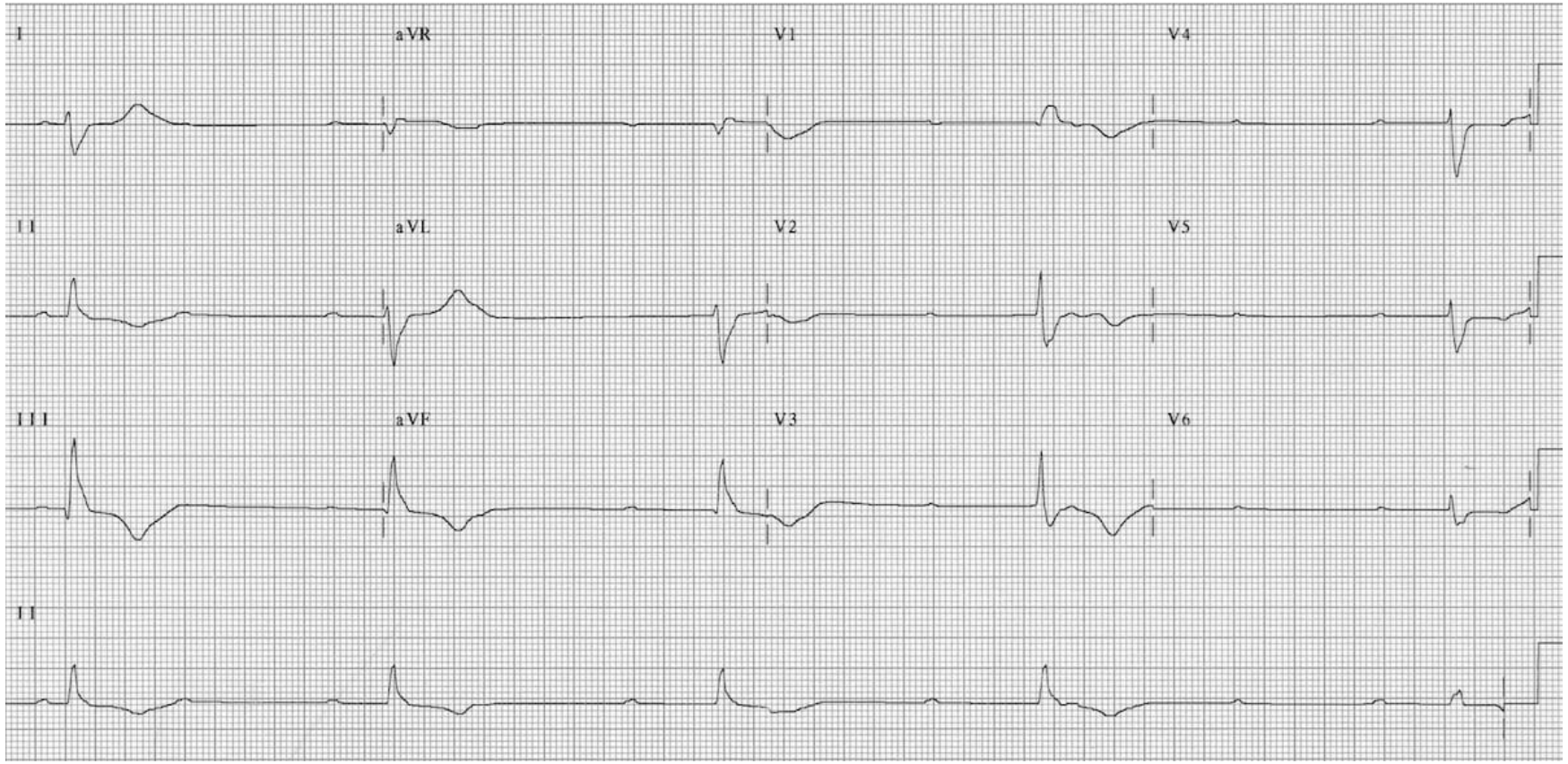


A 58 year old man who attends the emergency department with chest pain then loses consciousness whilst he is having his initial ECG. He has no central pulse and is taking occasional deep breaths. What is going on?




I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

Patient presented with a black out and a HR of 27 bpm. What do you think is happening ?



THANK YOU FOR LISTENING

Acknowledgments

Cardiac Physiology team @ MQHealth Cardiology
Dr Hari Raju, MQHealth Cardiology
Miriam Norman, Royal Hobart Hospital

Further reading or courses

Cardiac Physiology in Practice – ECG course , unaccredited
Life in the fast lane – Australian website, largest national ECG library