

Cardiac Conduction System Arrhythmias and ECGs

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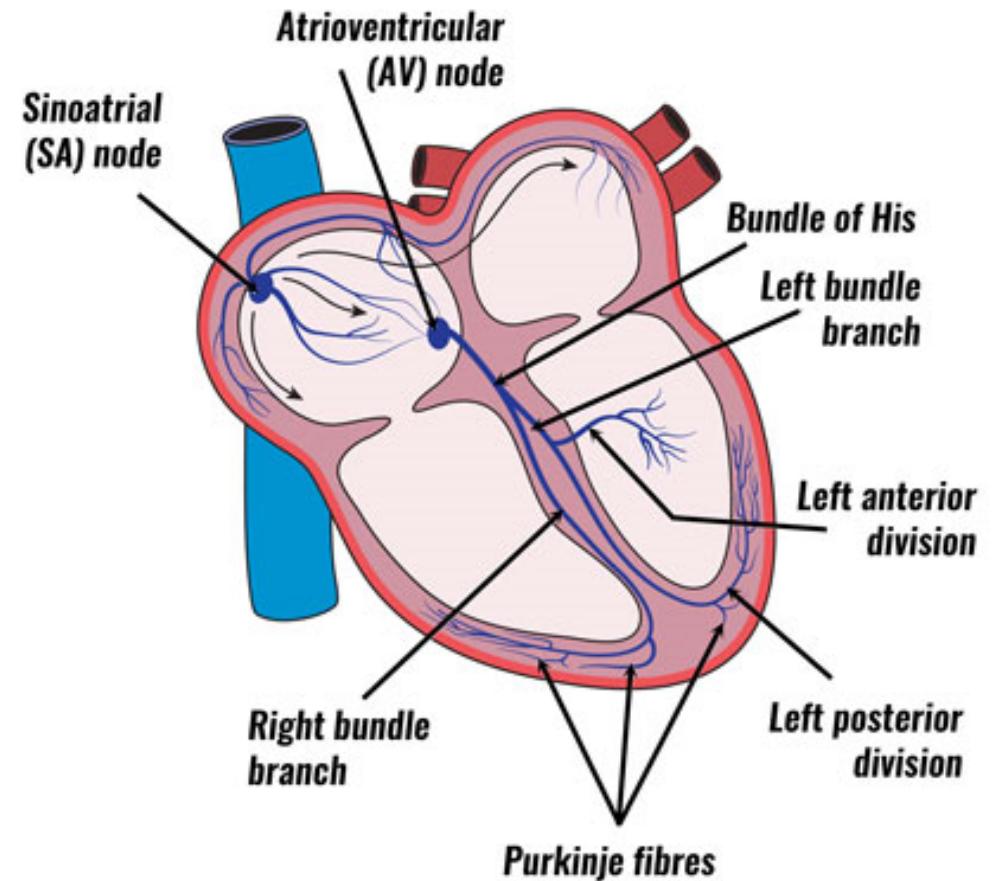
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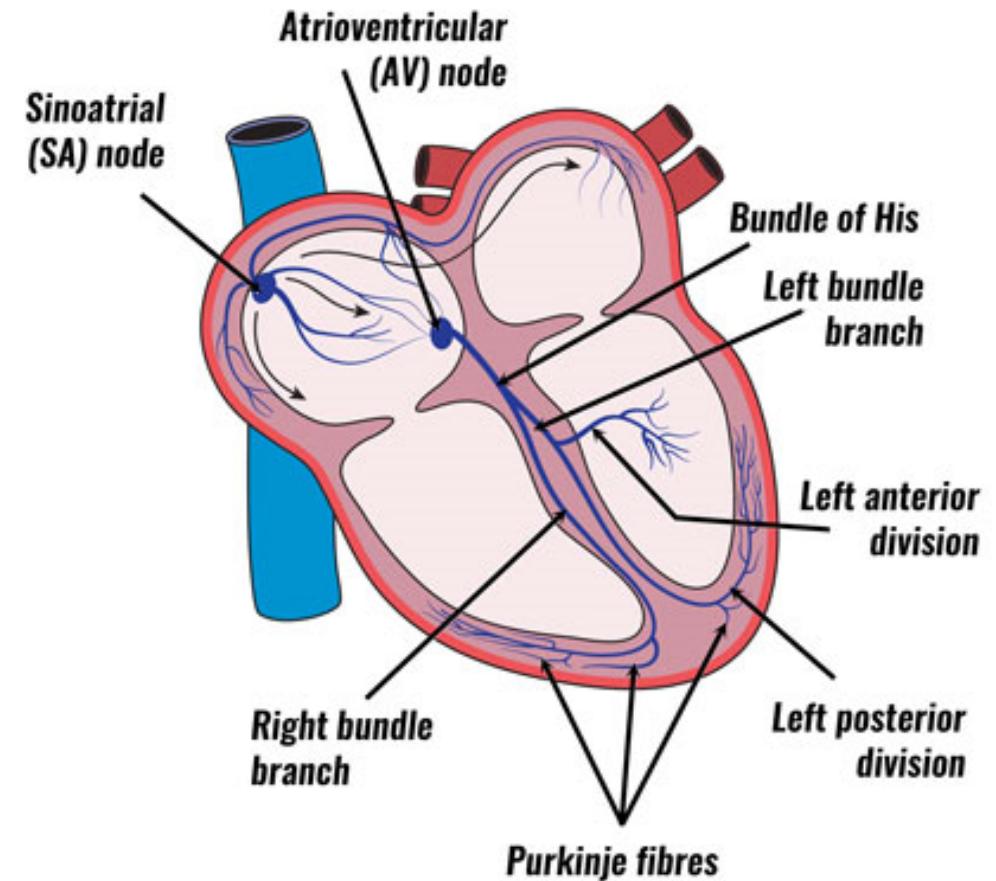
Review

- 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



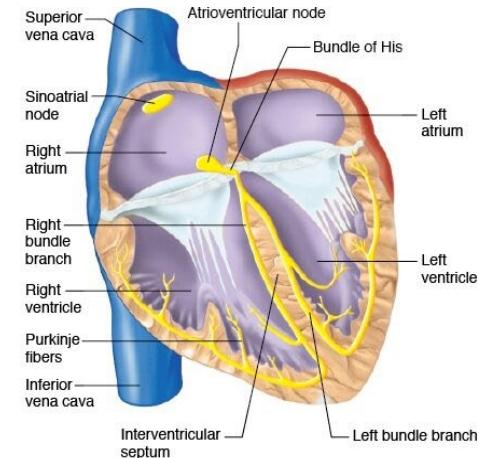
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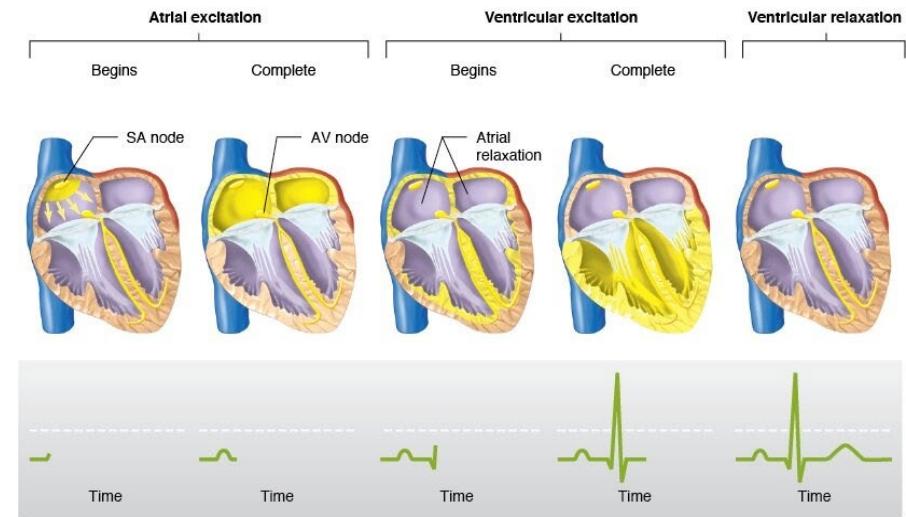


The impulse conduction system consists of four structures

- 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



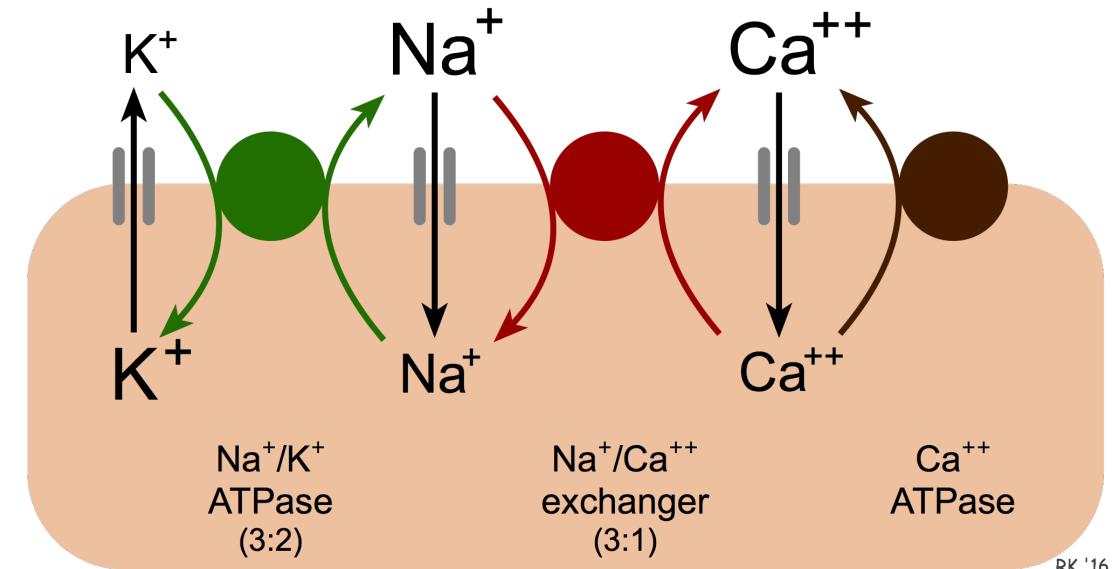
A



B

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.

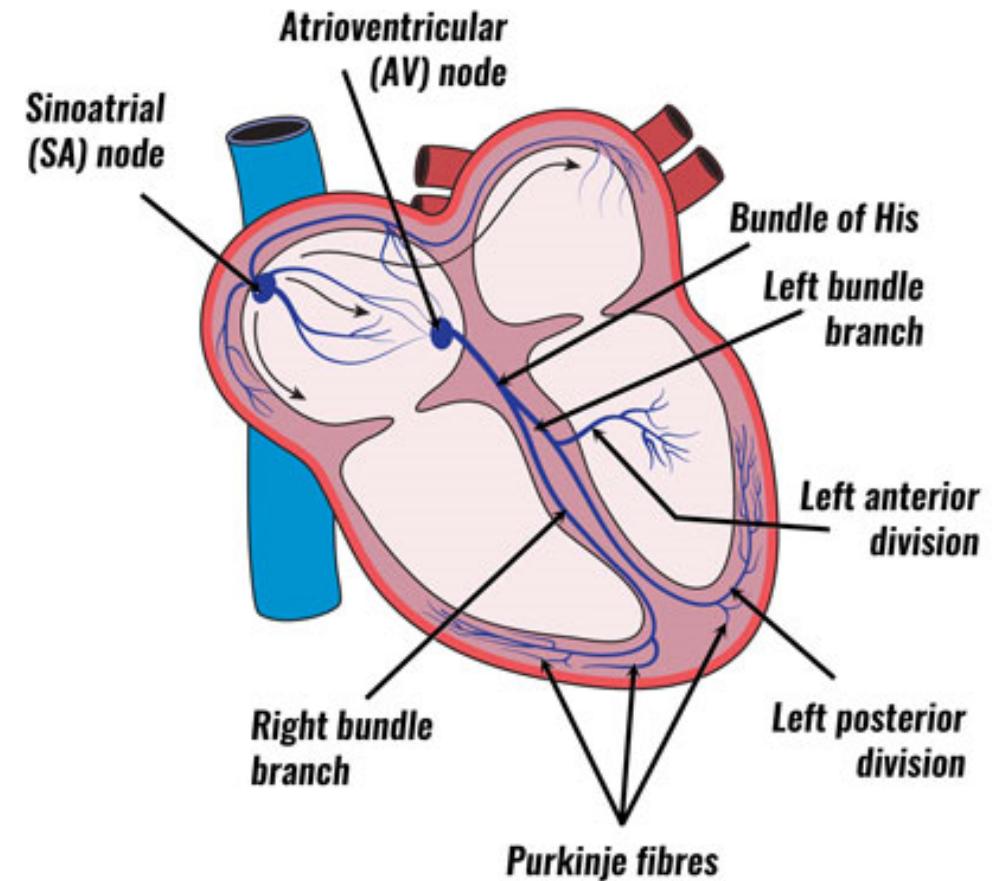


RK '16



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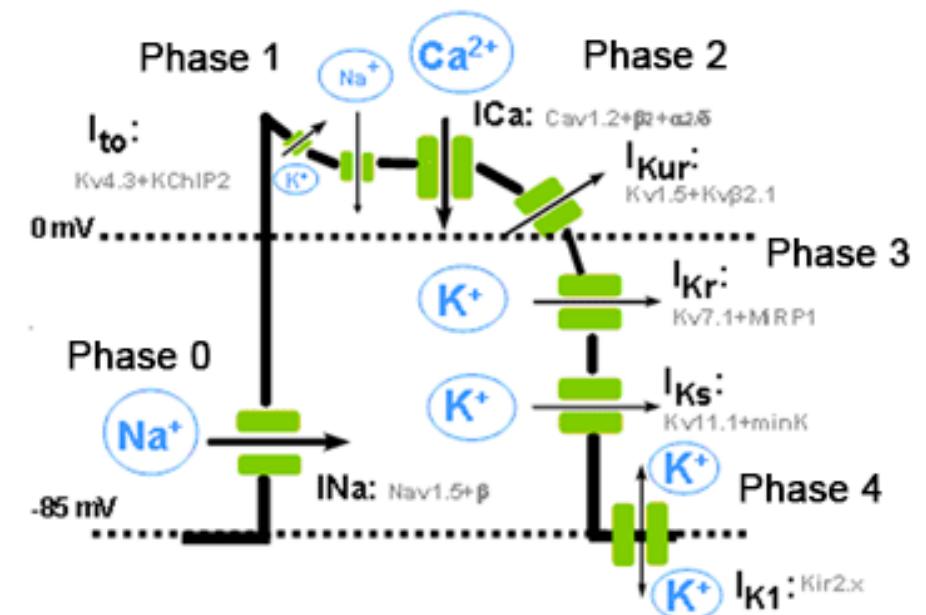


Action potential

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

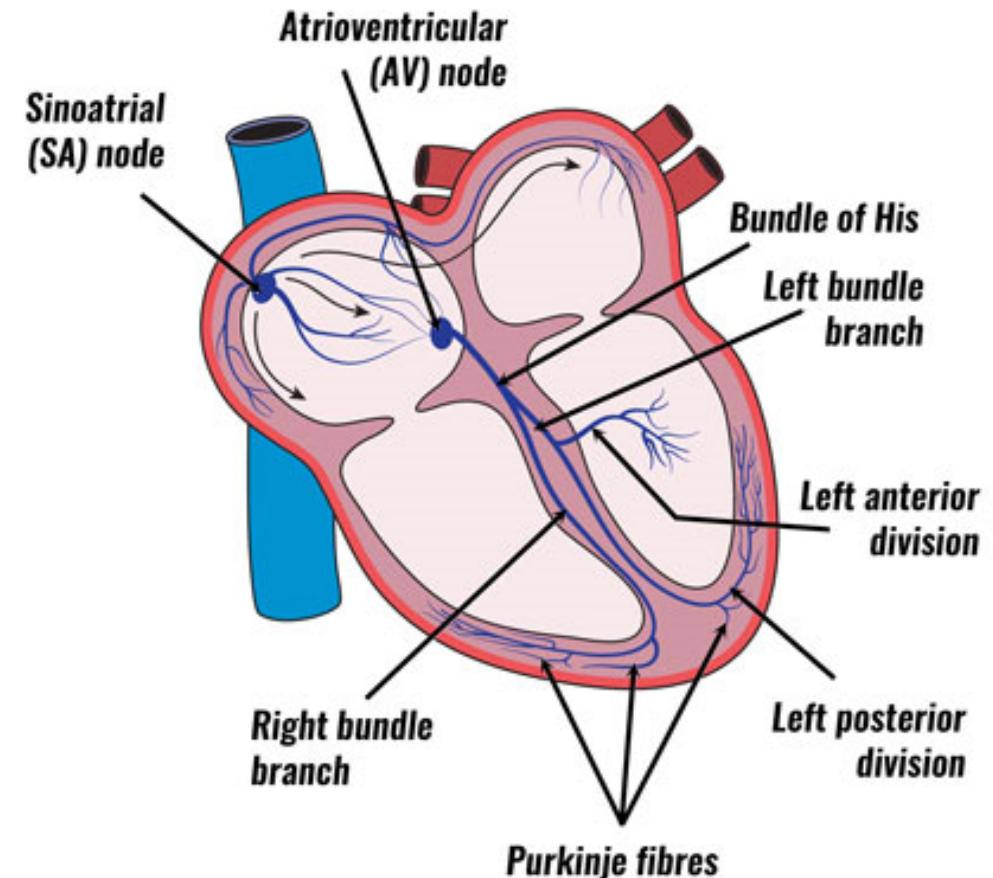
Phases

- 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



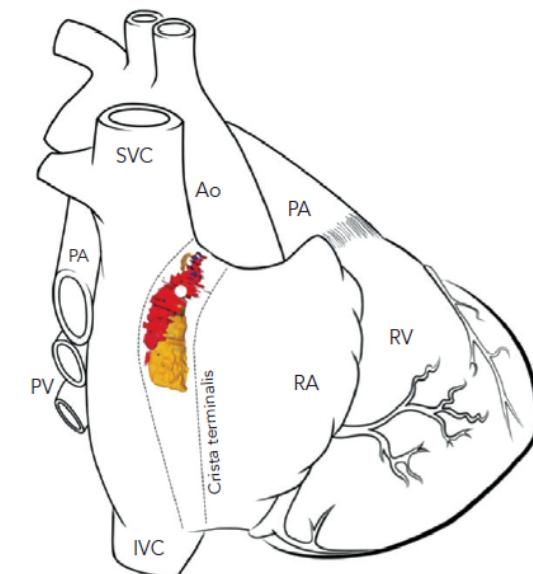
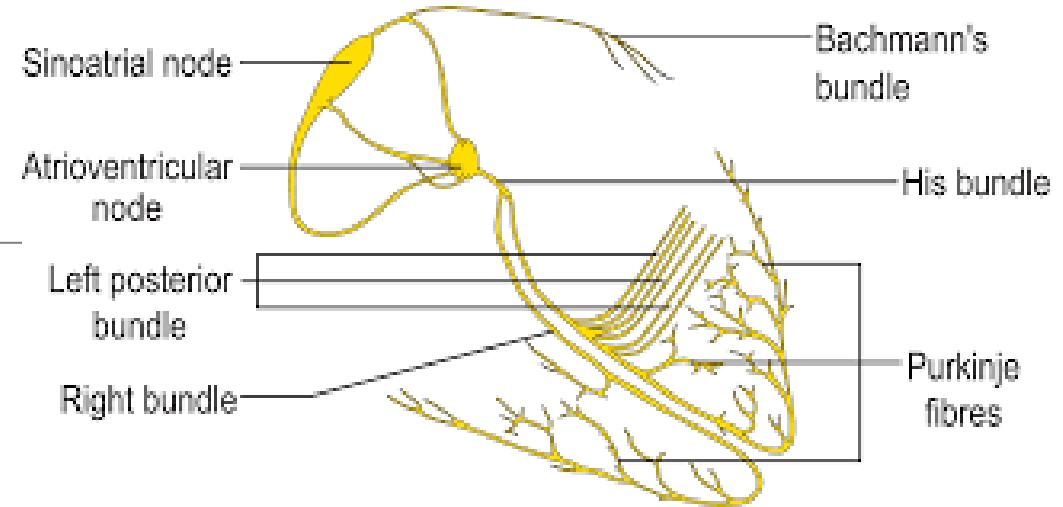
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SA Node

- 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

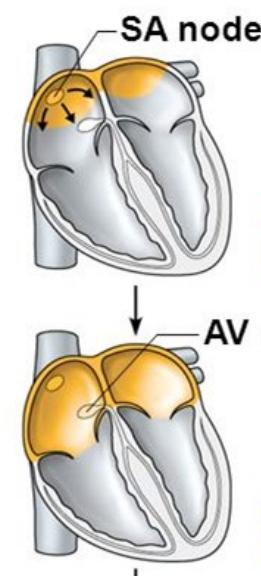


P wave

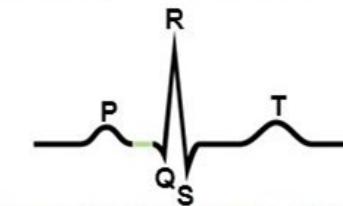
- Depolarization of atrial myocytes result in the “P” wave on the ECG
- The presence of a P wave represent an atrial contraction
- Atria contract forcing blood into the ventricles

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



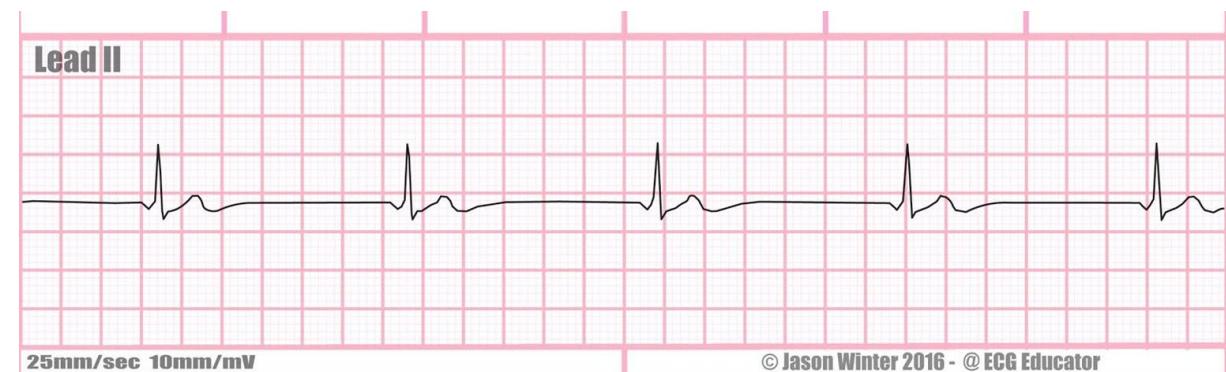
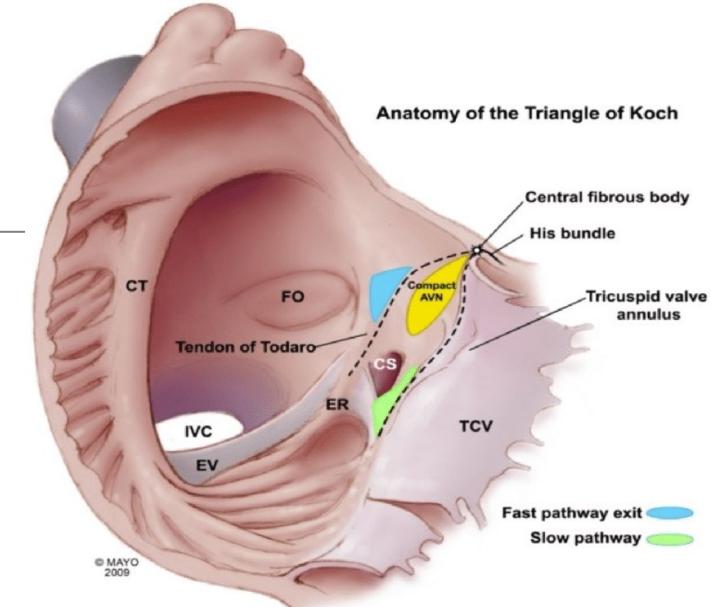
① Atrial depolarization, initiated by the SA node, causes the P wave.



② With atrial depolarization complete, the impulse is delayed at the AV node.

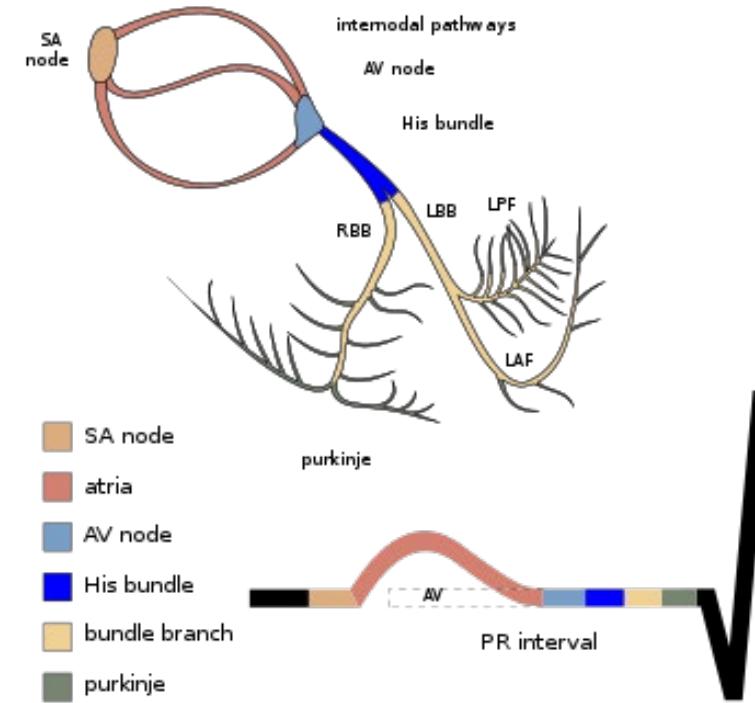
AV node

- 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



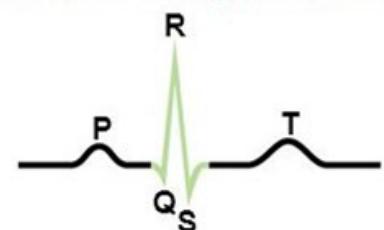
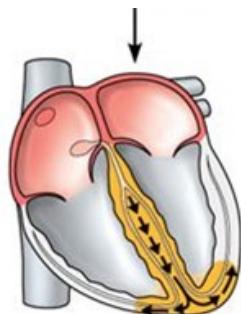
Bundle of HIS

- 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.



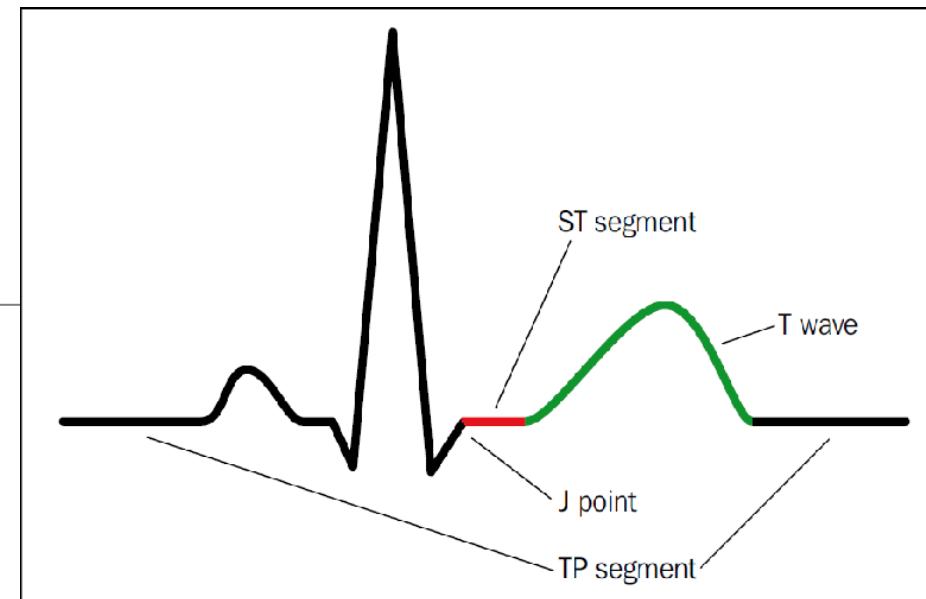
③ AV node depolarizes; Ventricular depolarization begins at apex, causing the QRS complex.
Atrial repolarization occurs.

A simple ECG strip showing a single positive deflection labeled 'R'.	The first (and only) wave is positive and thus an R wave.
An ECG strip showing a large positive deflection labeled 'R' followed immediately by a small negative deflection labeled 's'.	The first wave is large and positive (R), followed by a small negative wave (s).
An ECG strip showing a small positive deflection labeled 'r' followed by a large negative deflection labeled 'S'.	Initially a small positive wave (r), followed by a large negative wave (S).
An ECG strip showing a small negative deflection labeled 'q' followed by a large positive deflection labeled 'R', and finally a small negative deflection labeled 's'.	The first wave is negative and small (q), followed by a large positive wave (R), and finally a small negative wave (s).
An ECG strip showing a large negative deflection labeled 'Q' followed by a large positive deflection labeled 'R'.	Initially a large negative (Q), then a large positive wave (R).
An ECG strip showing a single large negative deflection labeled 'QS'.	A single negative wave is called a QS-complex.
An ECG strip showing a large negative deflection labeled 'Q' followed by a small positive deflection labeled 'r'.	A large negative wave (Q), followed by a small positive wave (r).
An ECG strip showing a large negative deflection labeled 'rs' followed by a large positive deflection labeled 'R'.	The negative wave manages to pass the baseline, and is therefore qualified as an S wave.
An ECG strip showing a small negative deflection labeled 'q' followed by a large positive deflection labeled 'R'.	Initially a small negative wave (q), followed by a large positive wave (R).
An ECG strip showing a single positive deflection labeled 'R'.	Notching on the upstroke of the R wave.
An ECG strip showing a small negative deflection labeled 'r' followed by a large positive deflection labeled 'R'.	The negative deflection does not manage to pass the baseline and can therefore qualify as an s wave.
Two ECG strips showing multiple small, irregular negative and positive deflections that do not form a standard QRS complex.	Examples of fragmented QRS-complexes.



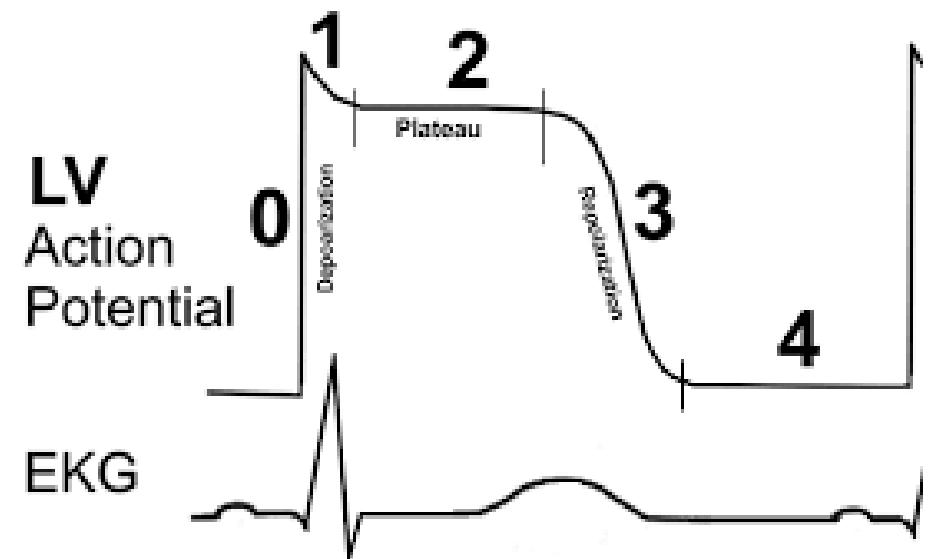
ST Segment

- 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

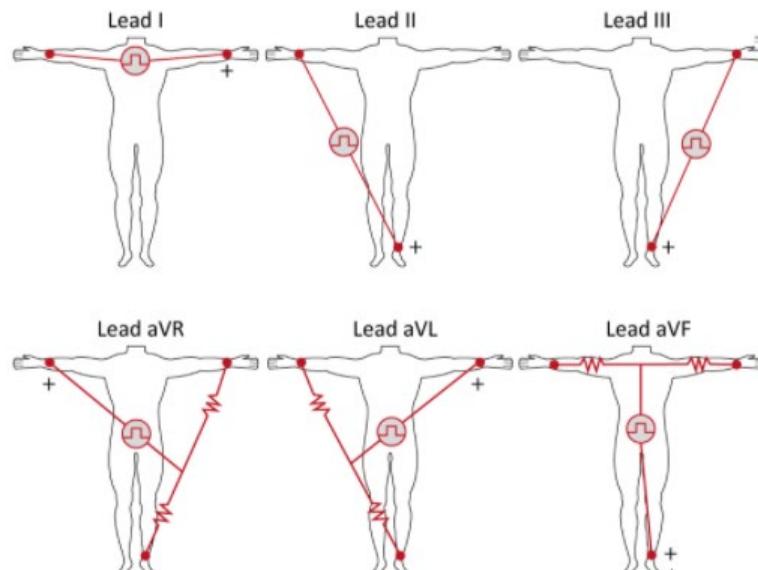


T wave

- 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

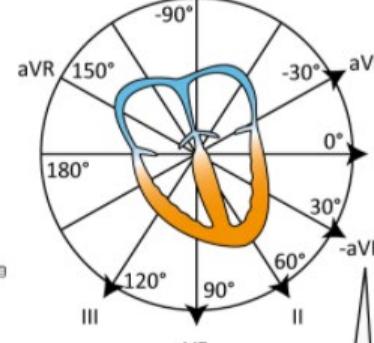


ECG lead placement

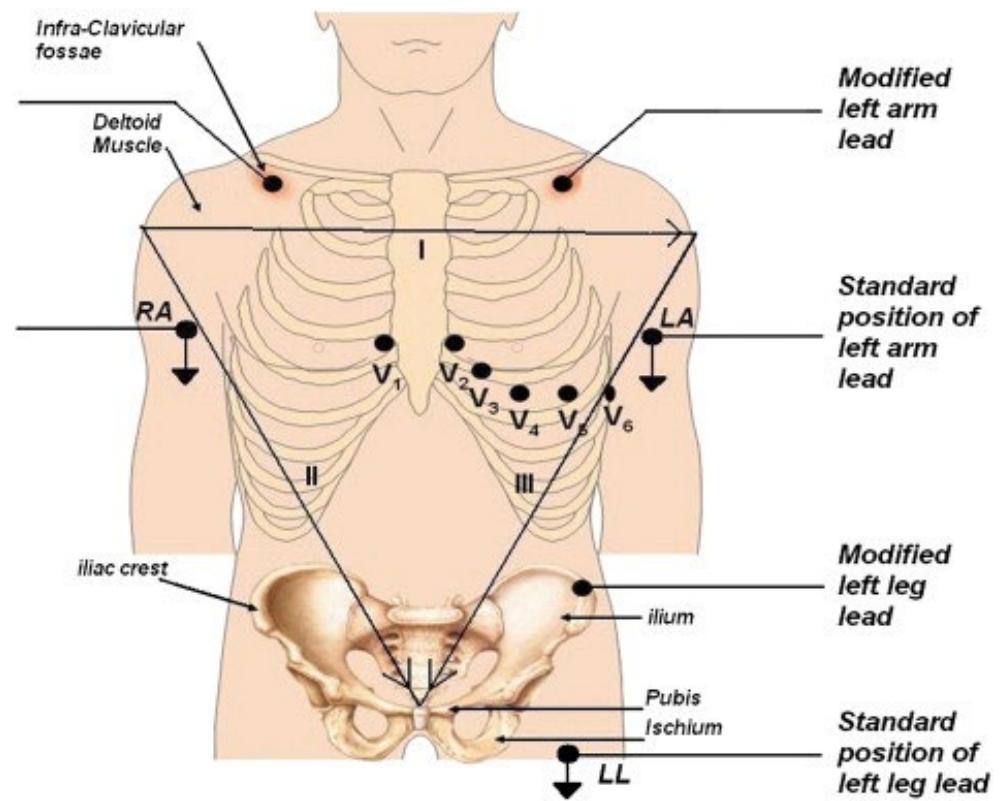


Recall that each lead 'views' the heart from the angle of its positive (exploring) electrode.

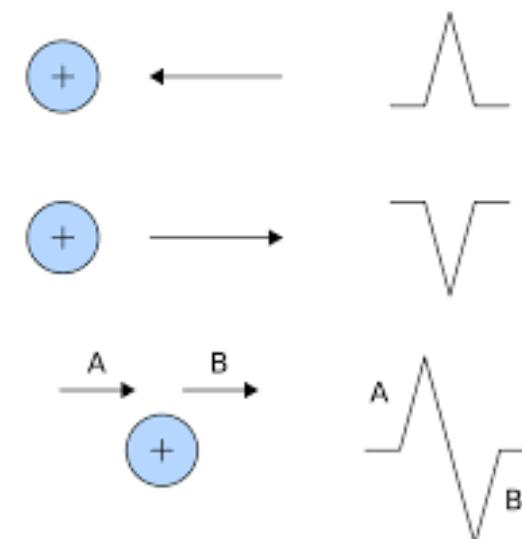
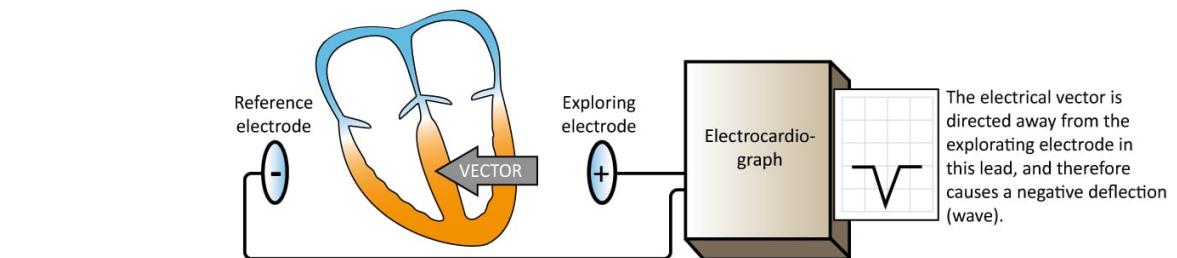
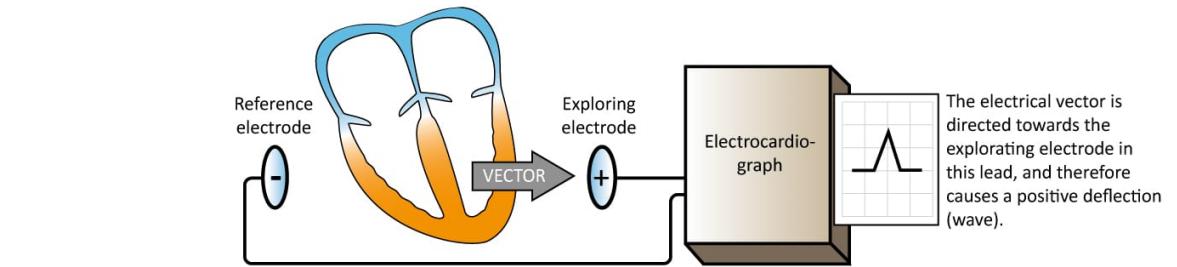
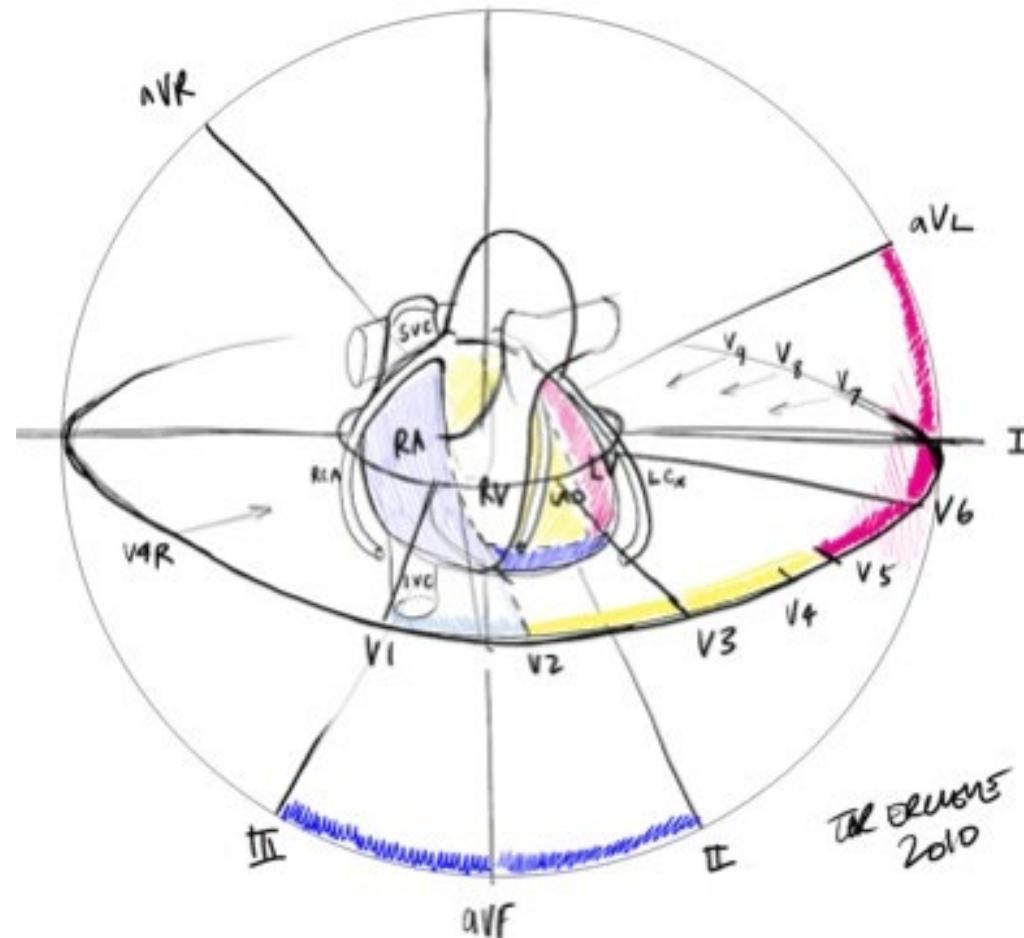
The positive electrode is the exploring electrode. This is defined by the ECG machine.



As noted previously, it is recommended that lead aVR be inverted to lead -aVR, as this fills a gap in the coordinate system and thus facilitates interpretation of the ECG.



ECG AXIS



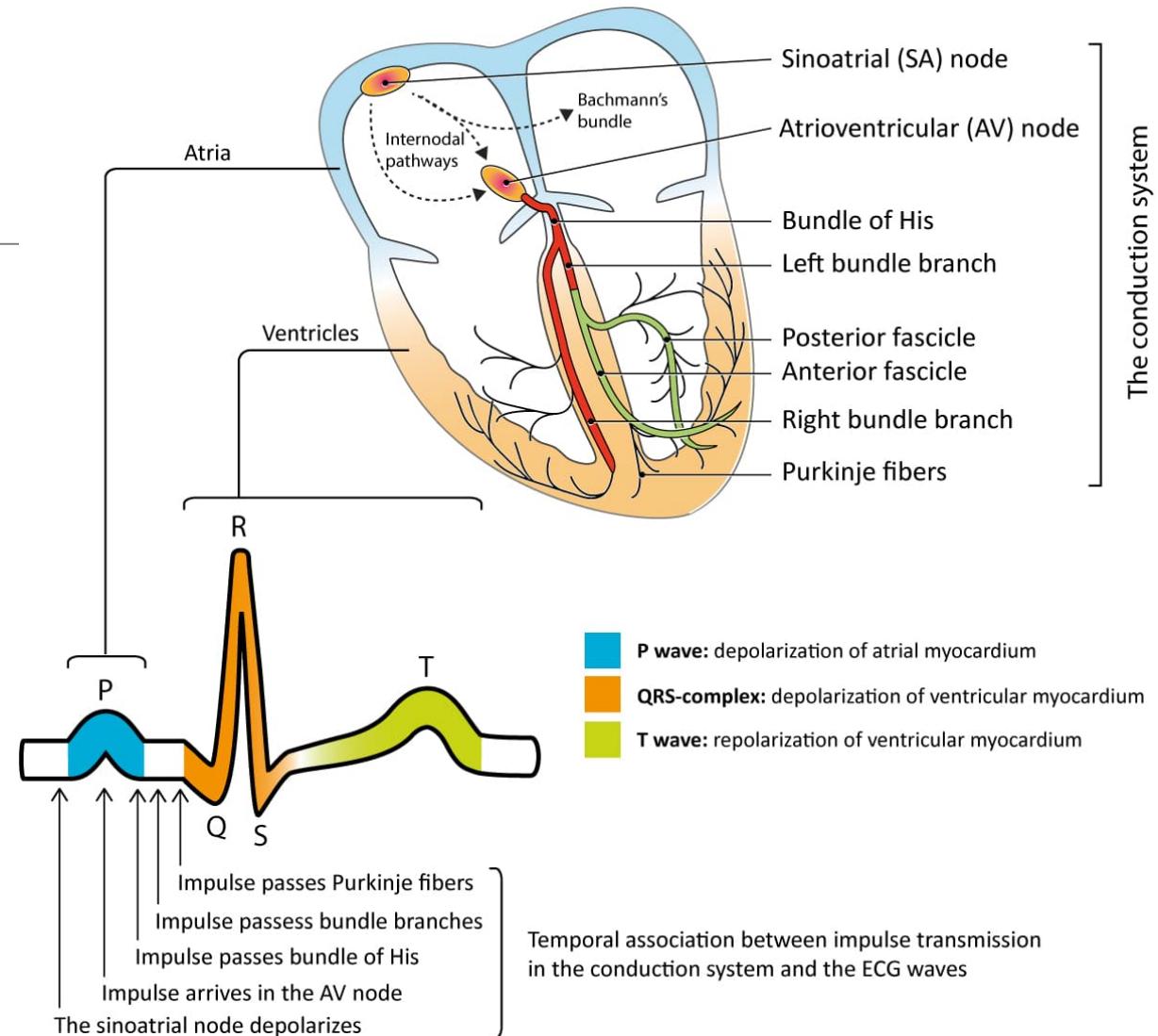
ECG features

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



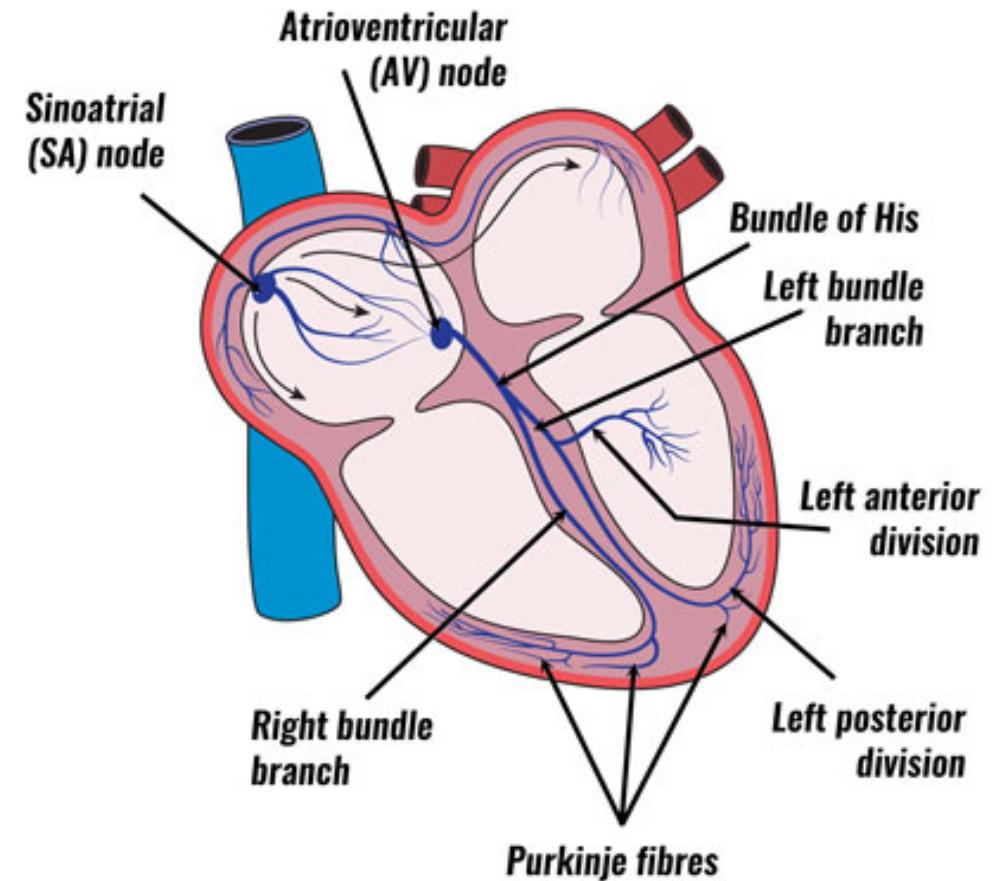
Review

- 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



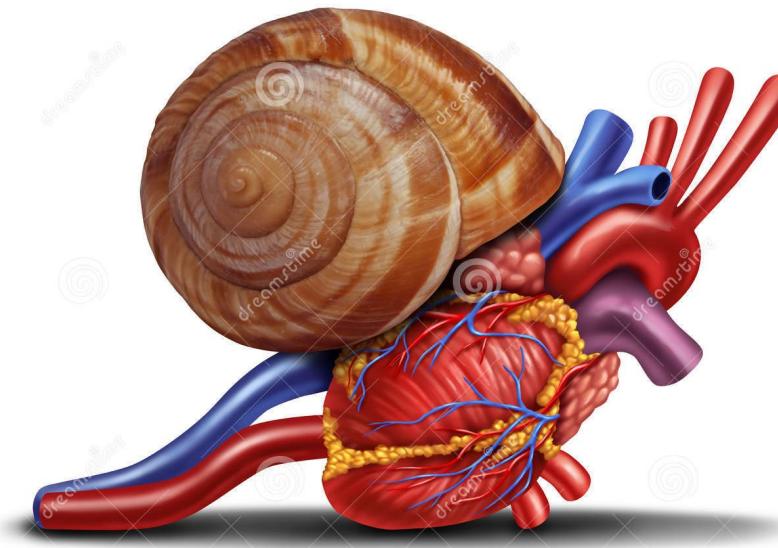
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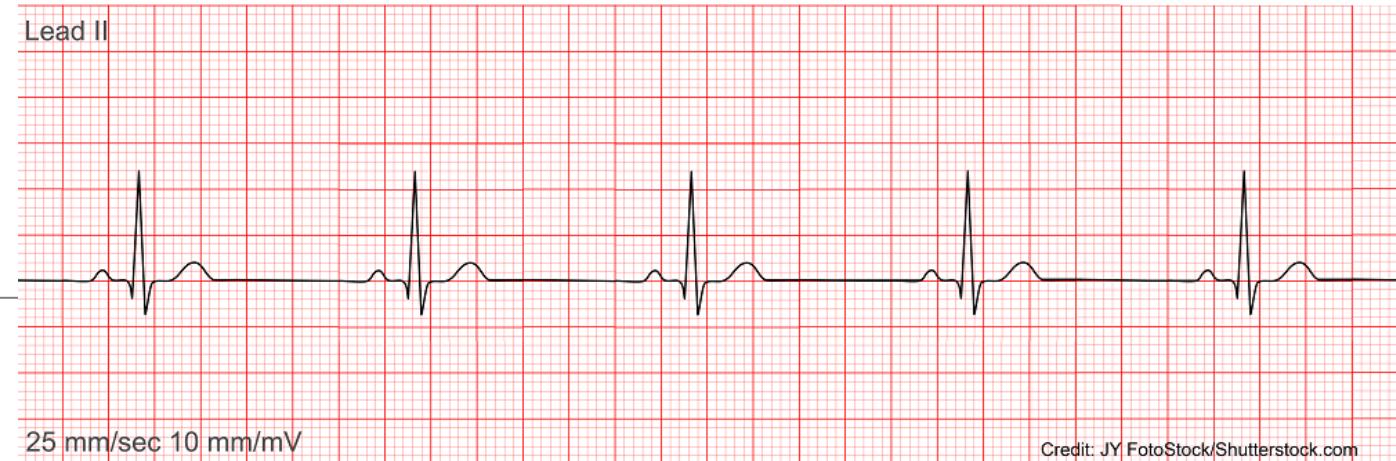
Bradyarrhythmias/ Bradycardia (Slow)

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



Sinus brady

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



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Sinus Pause

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



Sinus arrhythmia

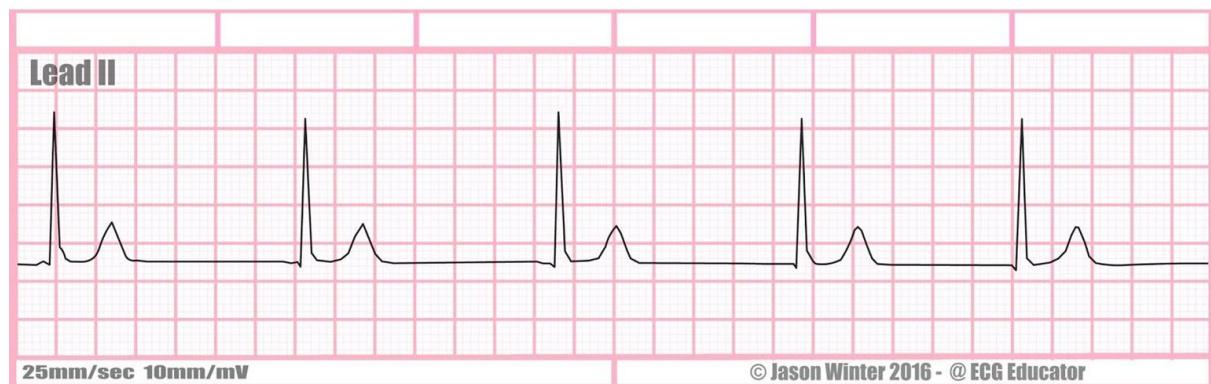
- Beat to beat variation in P-P interval
- All generated by the SA node, irregular



Junctional Rhythm

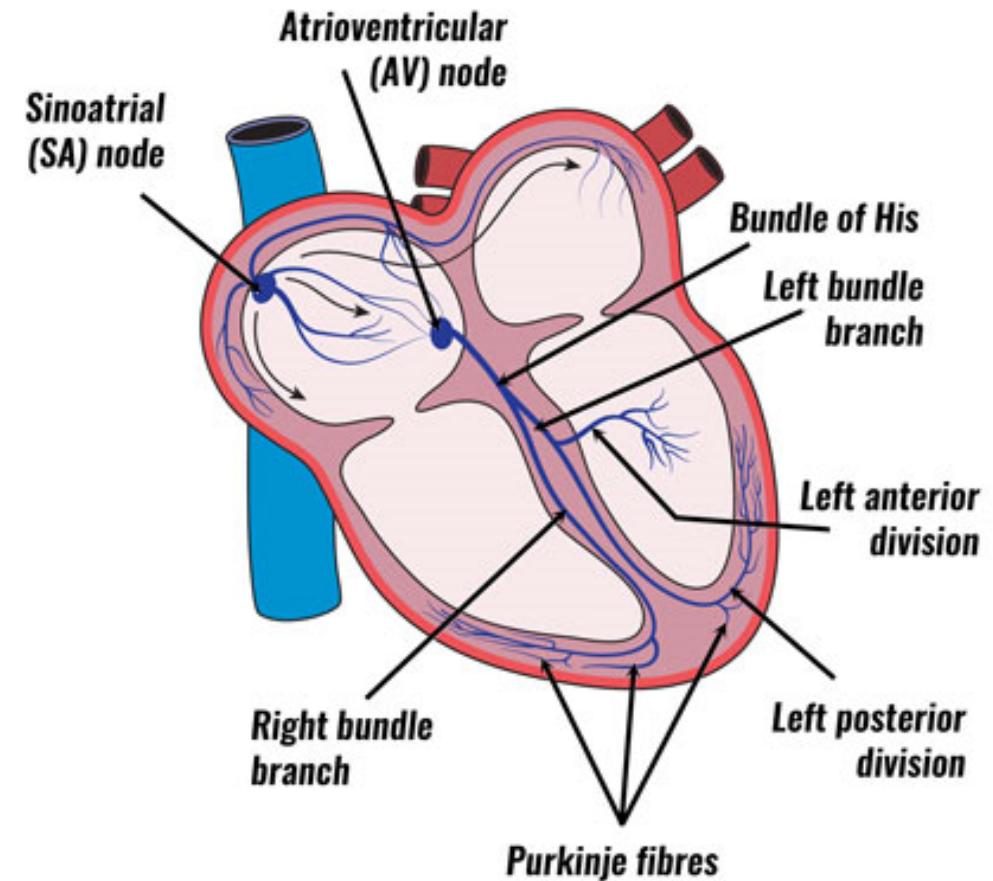
- Lack of P waves
- Regular R-R intervals , narrow complex QRS
- AV node is the pacemaker
- 40-60 bpm (rate of AV node)

Junctional Rhythm



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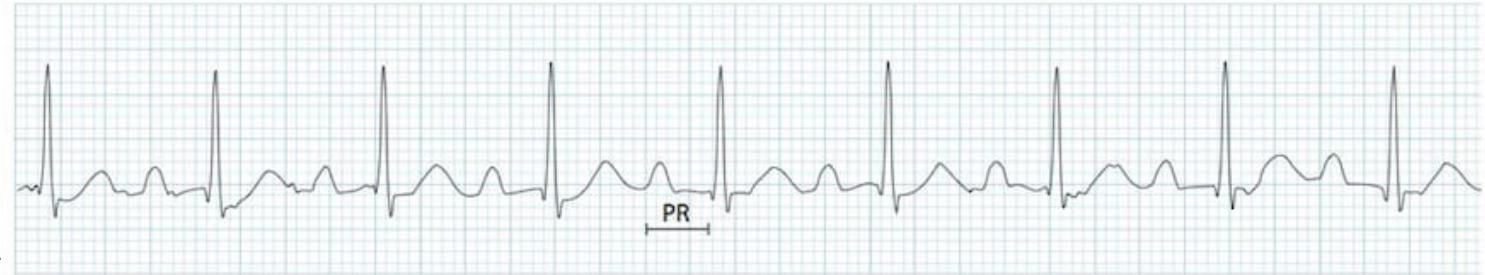
AV BLOCKS

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



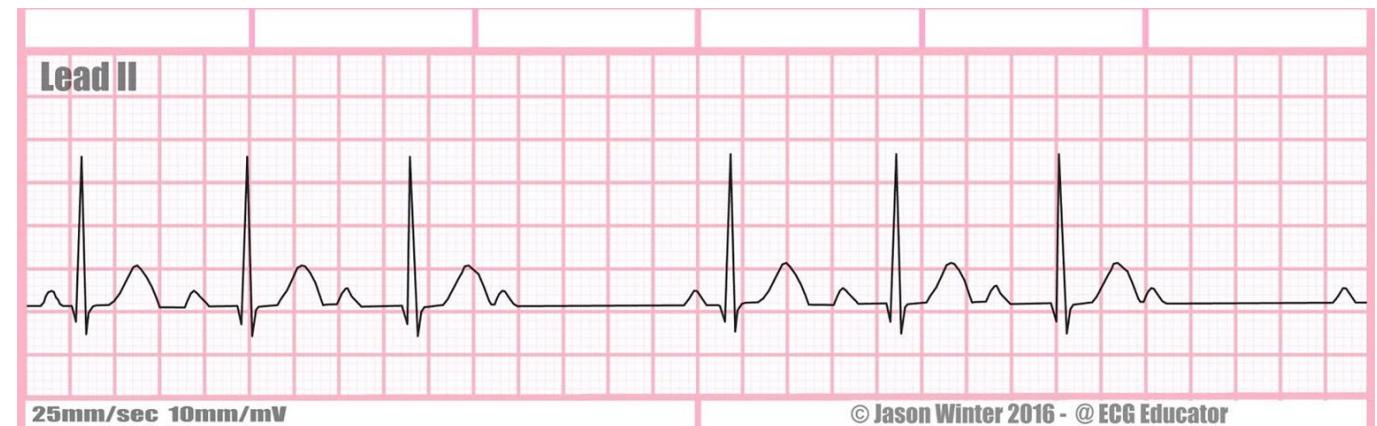
1st degree AV block

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



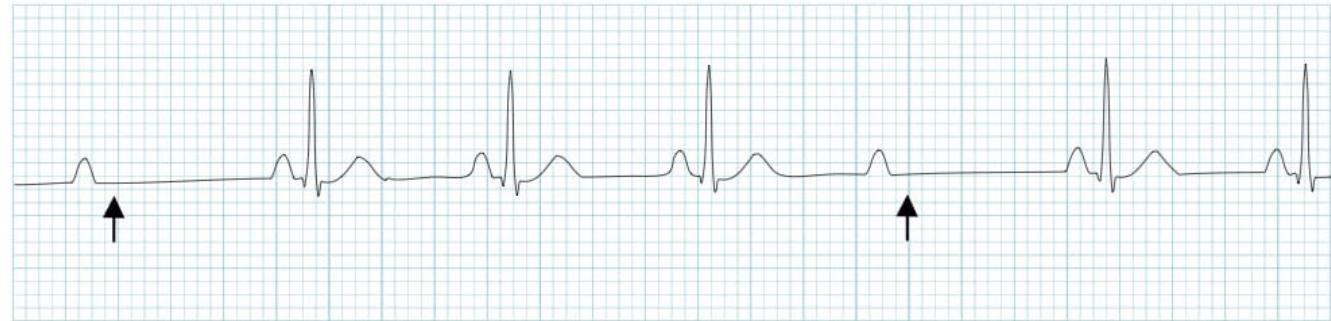
2nd degree type 1, Mobitz type 1

- 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



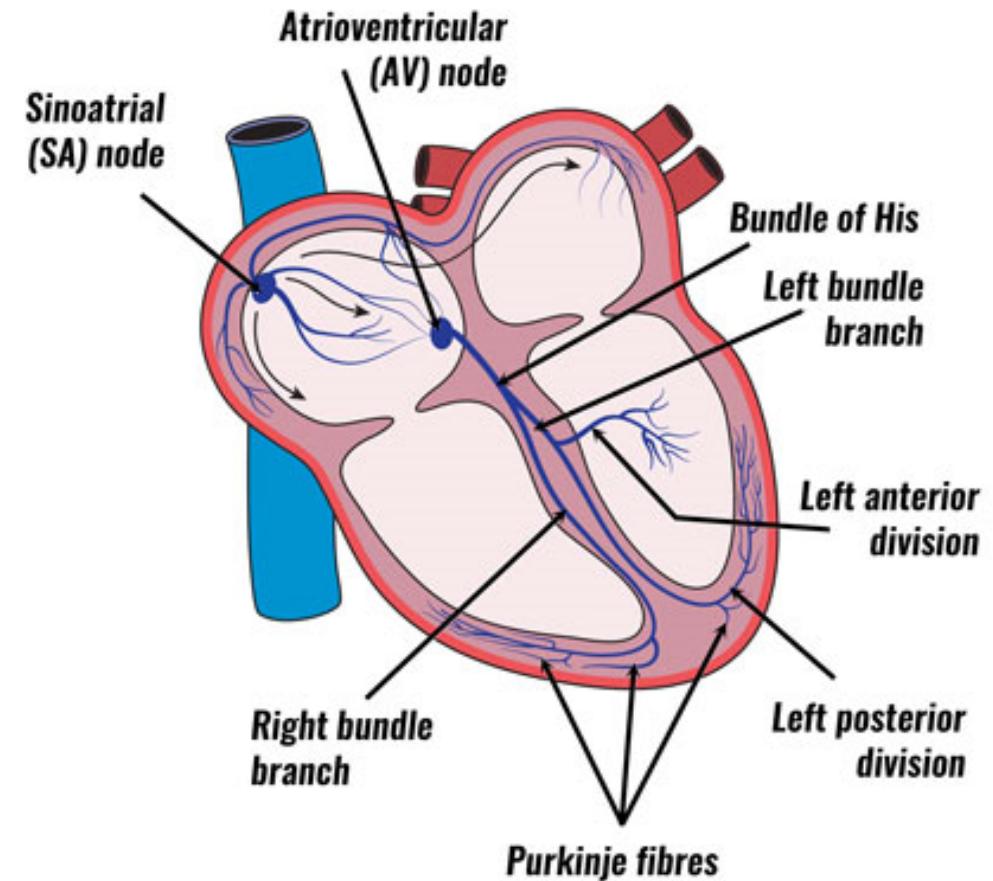
Complete or 3rd degree AV Block

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



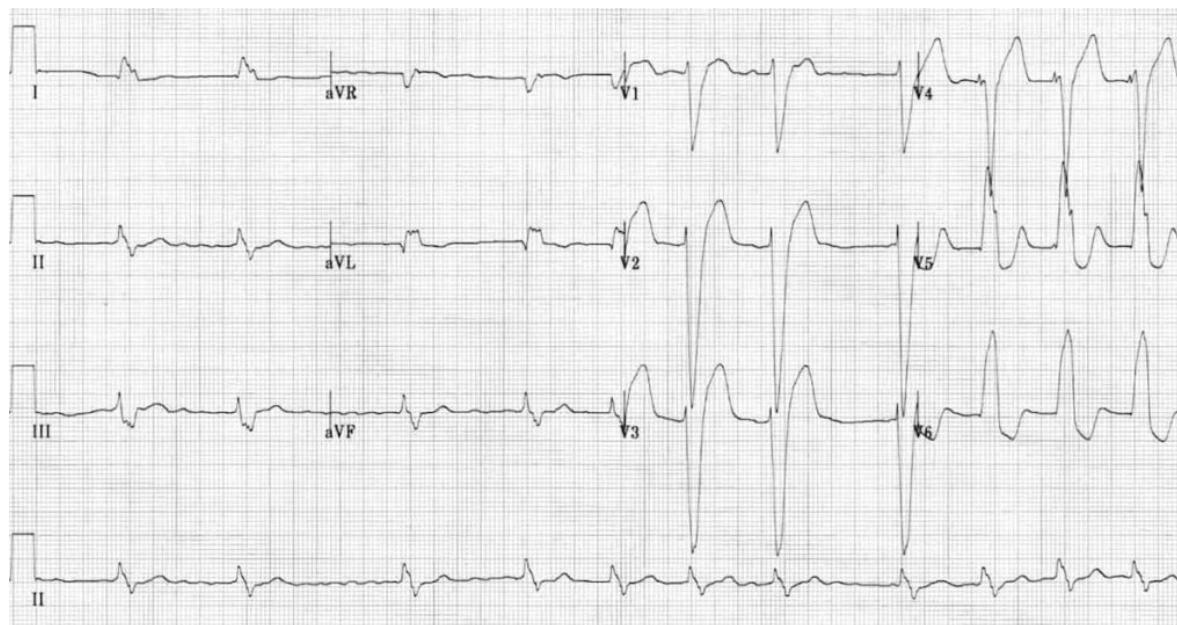
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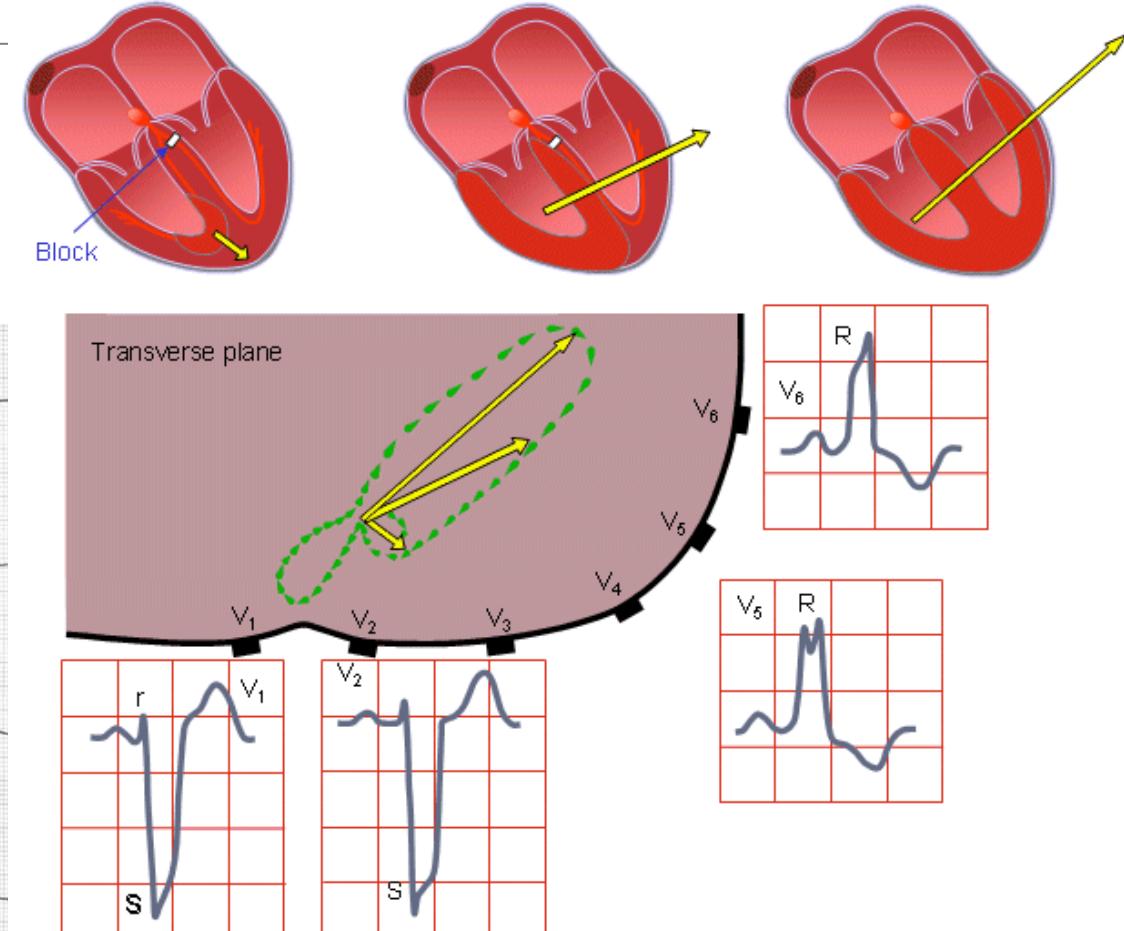
Left Bundle Branch Block

- Wide QRS , dyssynchrony
- Negative in V1
- Causes – infiltration, aortic valve procedures



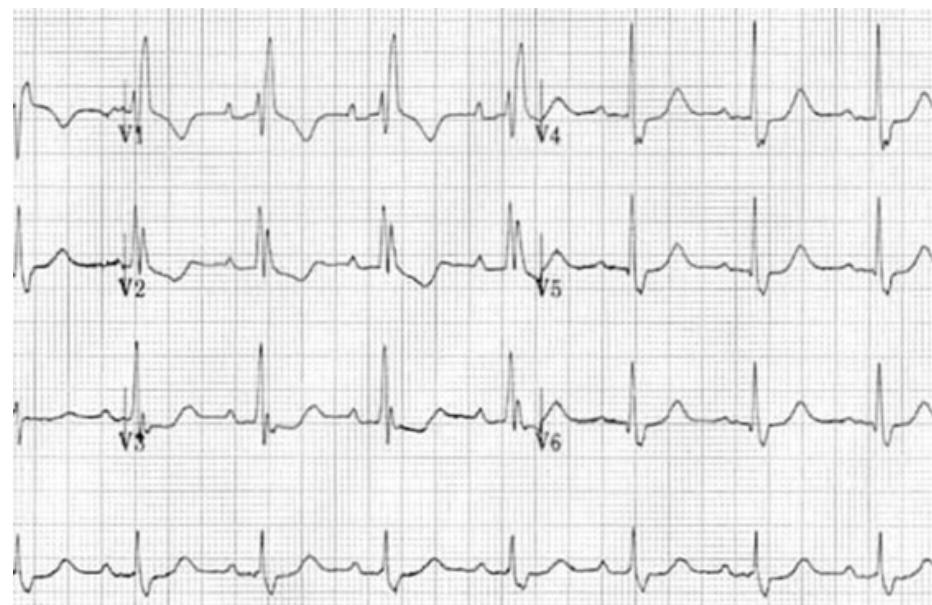
LEFT BUNDLE-BRANCH BLOCK

QRS duration greater than 0.12 s
Wide S wave in leads V1 and V2, wide R wave in V5 and V6



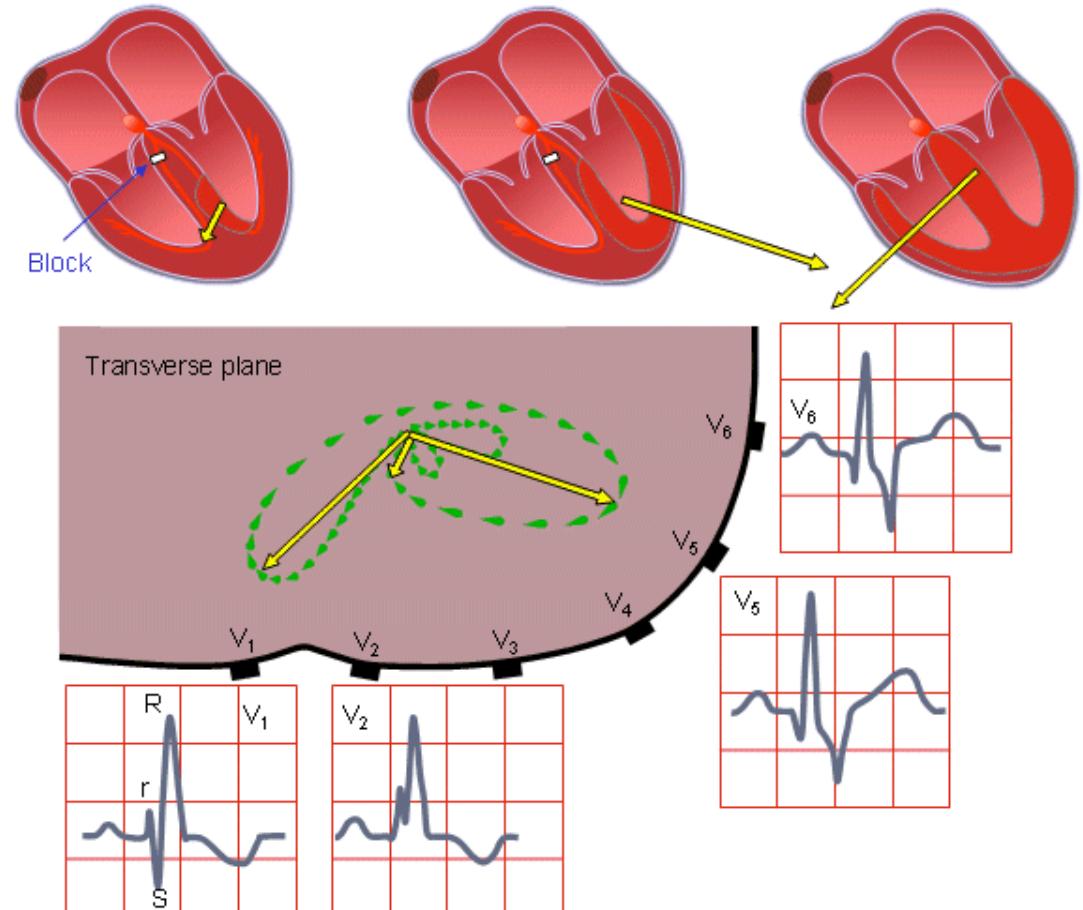
Right Bundle Branch Block

- Dominant R wave in V₁, dyssynchrony not as consequential
- S wave in lateral leads, V₅, V₆
- Causes – trauma, dilatation



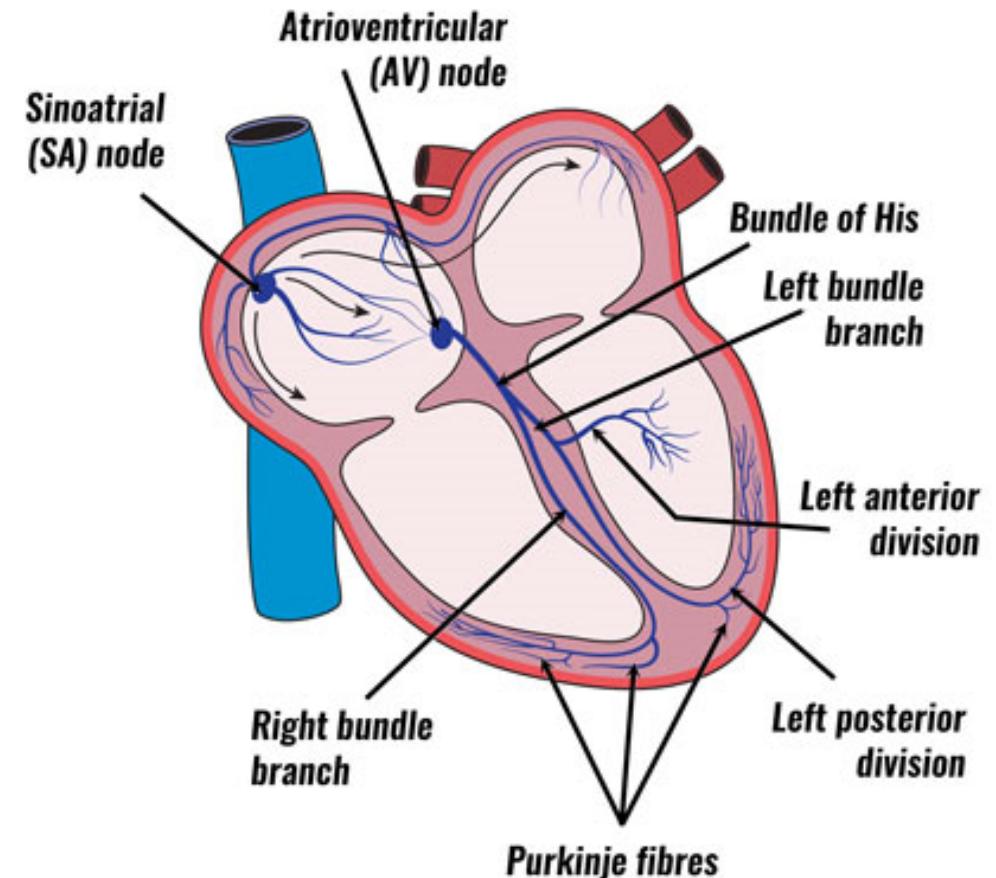
RIGHT BUNDLE-BRANCH BLOCK

QRS duration greater than 0.12 s
Wide S wave in leads I, V₅, and V₆



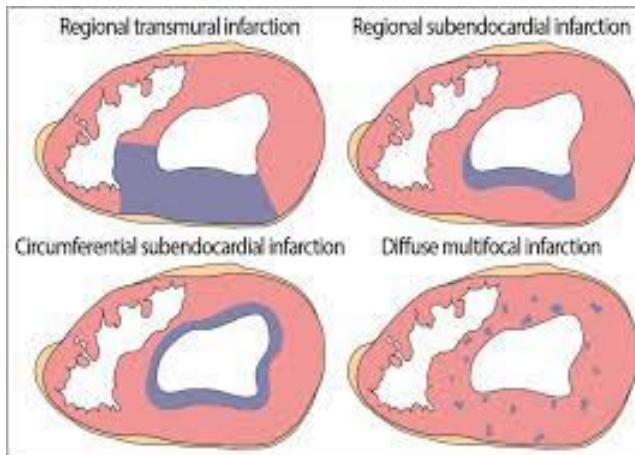
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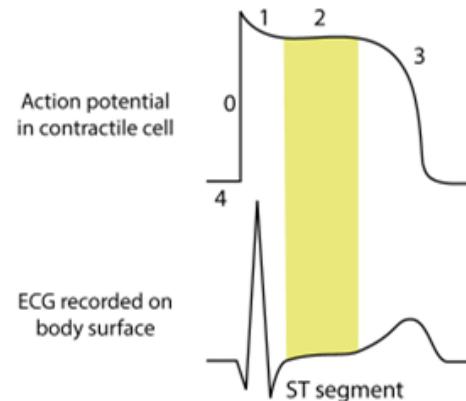


Ischemia

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



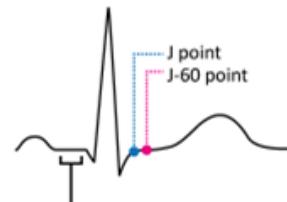
A)



The plateau phase (phase 2) corresponds to the ST segment on the surface ECG. The membrane potential is relatively unchanged during this phase and most ventricular cells are in this phase simultaneously (more or less). Therefore there are no electrical potential differences in the myocardium during phase 2, which results in a flat and isoelectric ST segment.

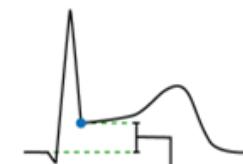
Acute ischemia is virtually always confined to a specific area, where the cell's membrane potentials change (due to ischemia). Thus, electrical potential difference occurs in the myocardium and this displaces the ST-segment up or down.

B) Measurement points



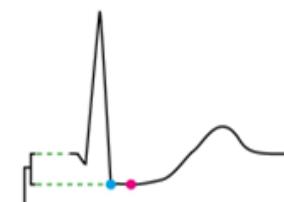
The PR segment is the baseline (reference line) for measuring deviation of the ST segment.

C) ST segment elevation



The magnitude of ST segment elevation is measured in the J point.

D) ST segment depression

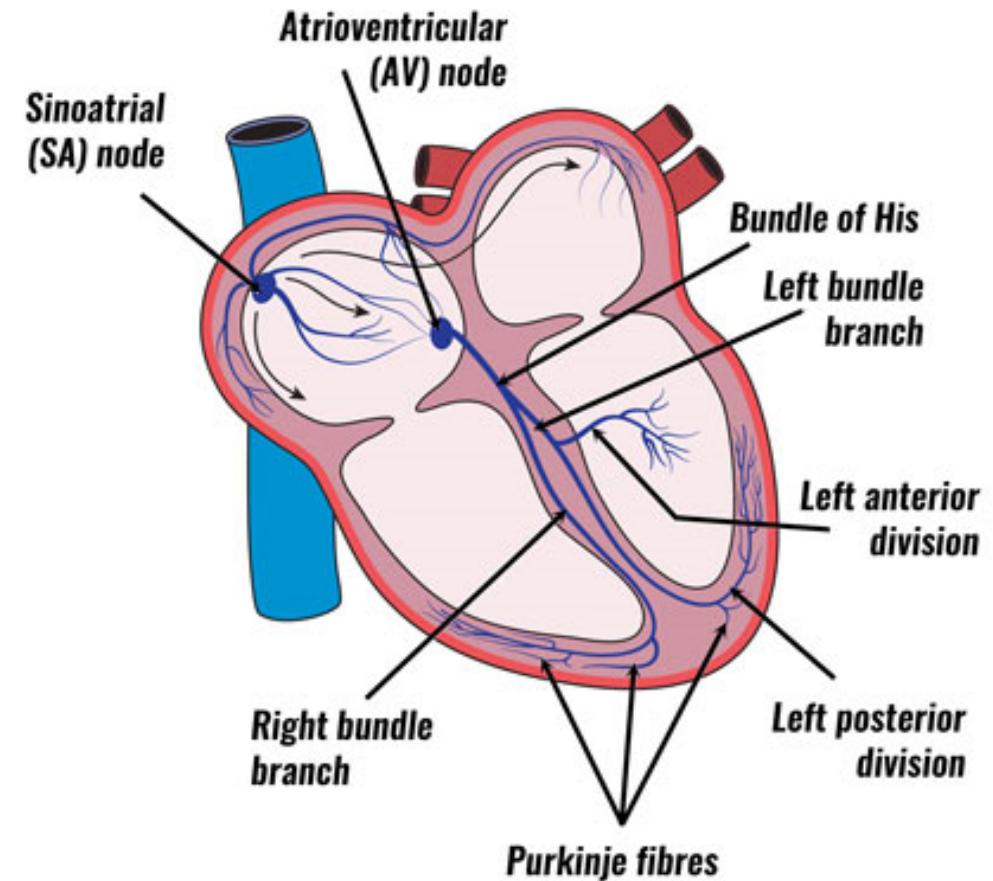


The magnitude of ST segment depression is measured in the J point in most instances. Occasionally it is advised that the J-60 point be used instead (particularly during exercise stress testing). If the ST segment is horizontal, then there is no difference in the magnitude of the ST depression in J and J-60.



Review

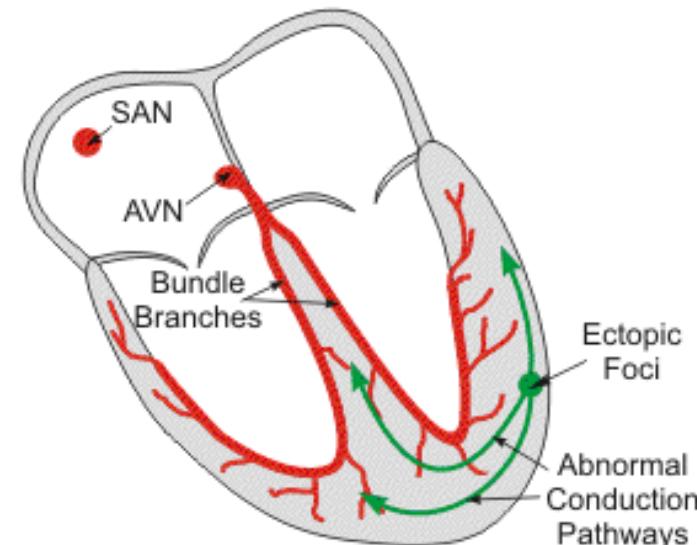
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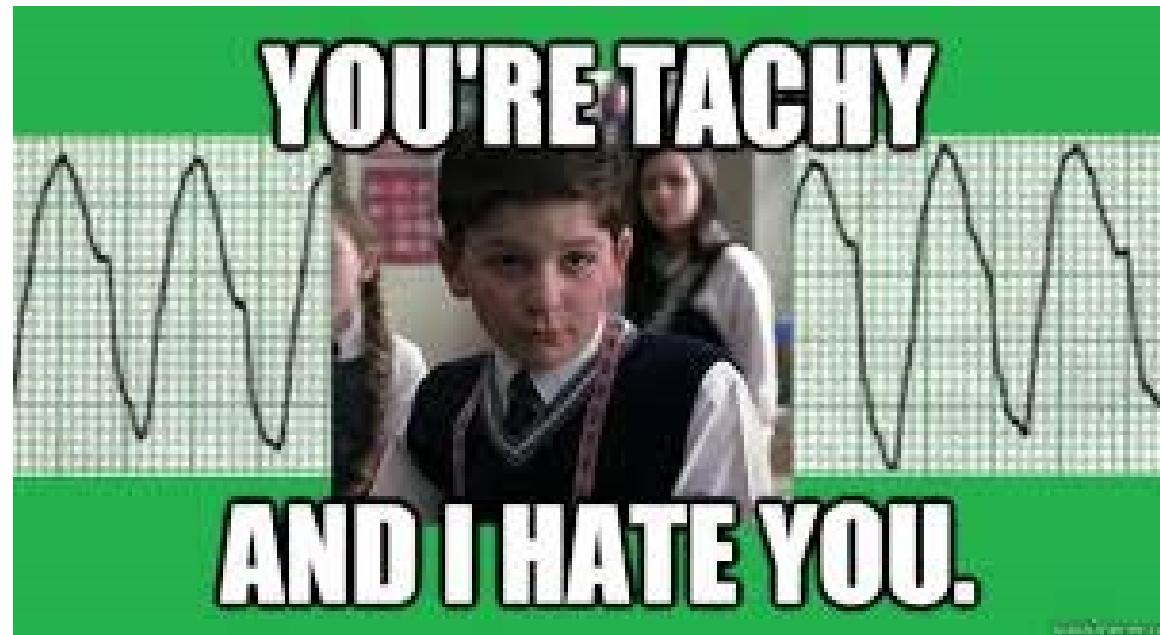
Ectopics

- Pacemaker sites outside the normal pathway
- 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

Abnormal Electrical Conduction due to Ventricular Ectopic Foci



Tachyarrhythmias (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

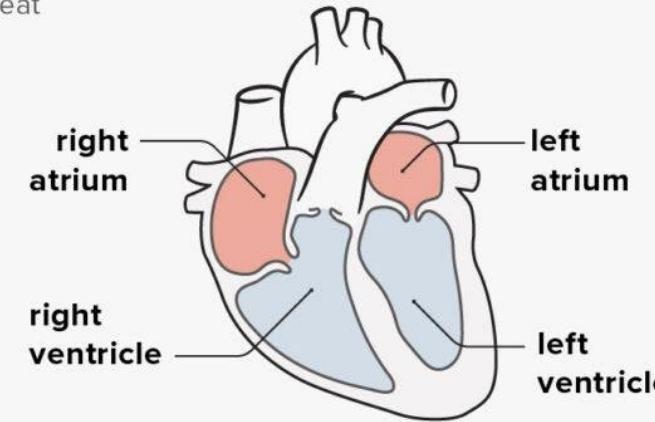
Myopathy – dilated , HCM

Infiltration – Sarcoidosis , Amyloid

Long QT / Brugada syndrome

Ventricular Tachycardia

abnormal heartbeat

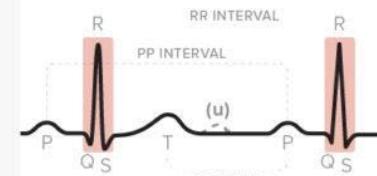


SINUS RHYTHM

NORMAL HEART RATE (60–100 BPM)

defined QRS complex

ECG WAVEFORM



ORIGIN OF ELECTRICAL SIGNAL

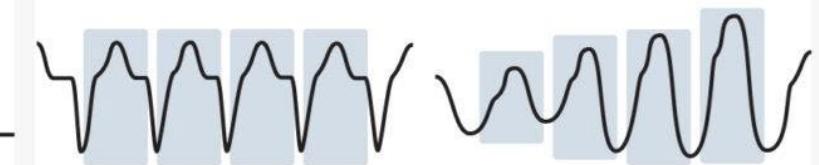


VENTRICULAR TACHYCARDIA

VERY FAST HEART RATE (100–320 BPM)

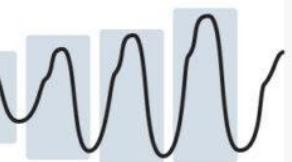
monomorphic

ECG WAVEFORM



polymorphic

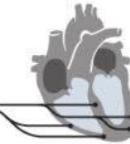
ECG WAVEFORM



ORIGIN OF ELECTRICAL SIGNAL



ventricular node
different nodes



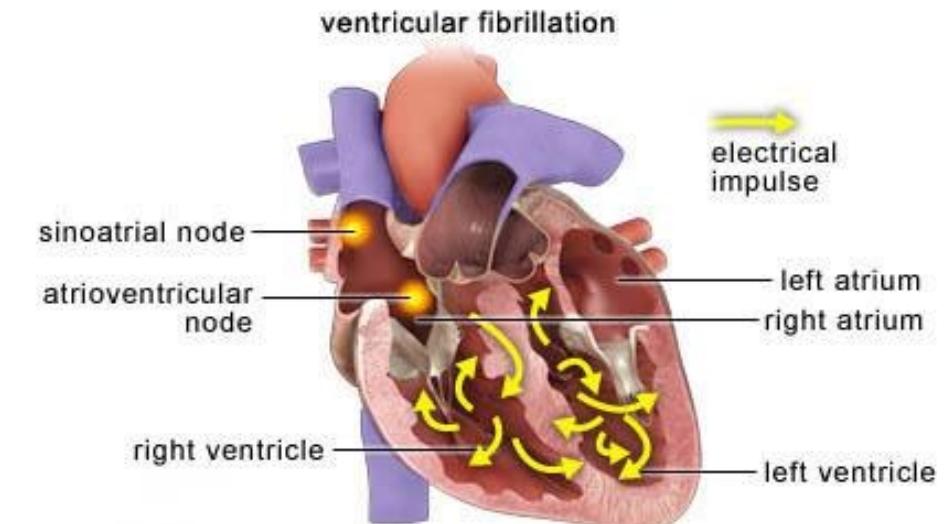
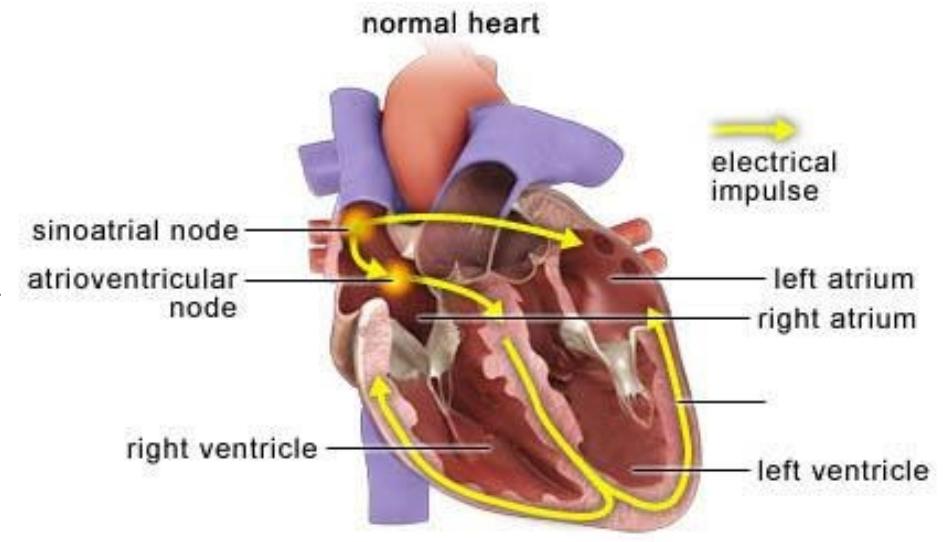
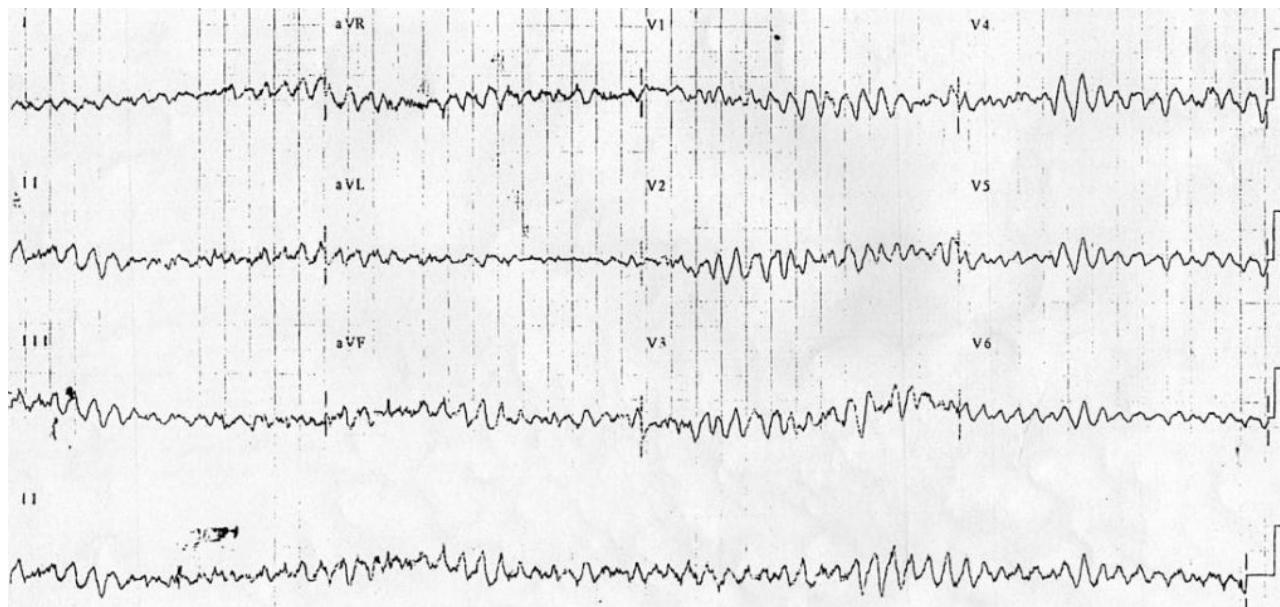
MEDICALNEWS TODAY



MQ Health
MACQUARIE UNIVERSITY
HEALTH SCIENCES CENTRE

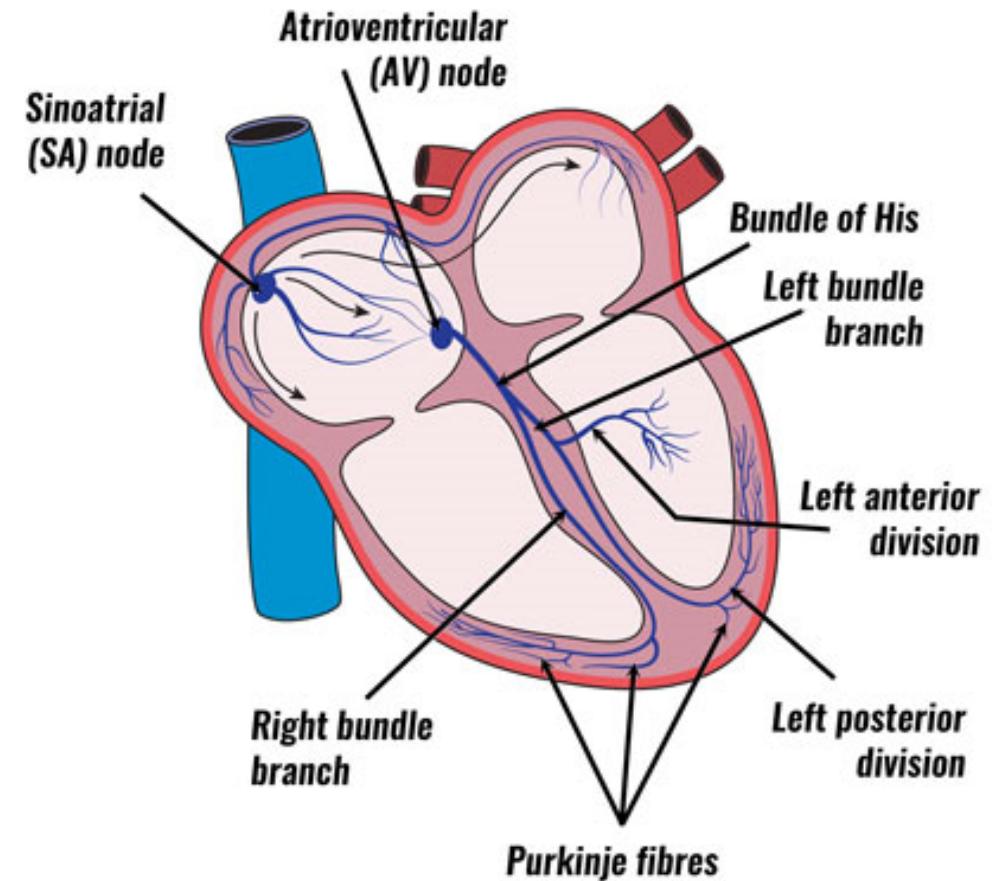
Ventricular fibrillation

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



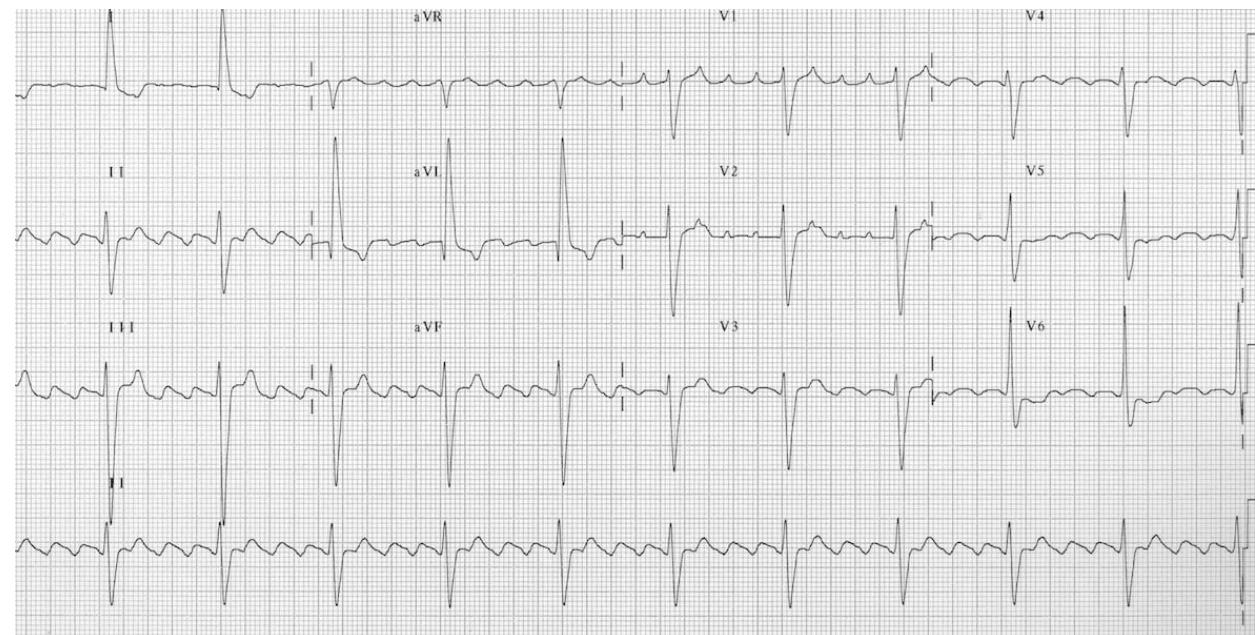
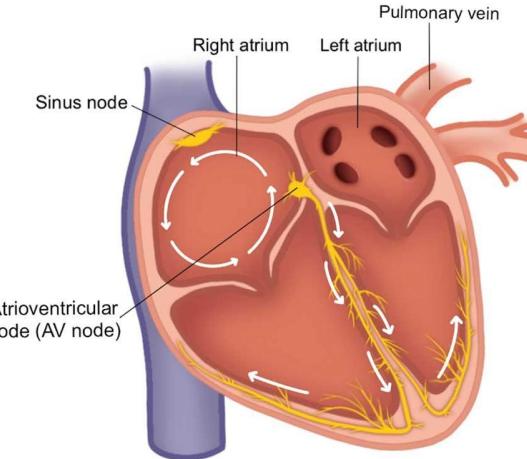
Review

- 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



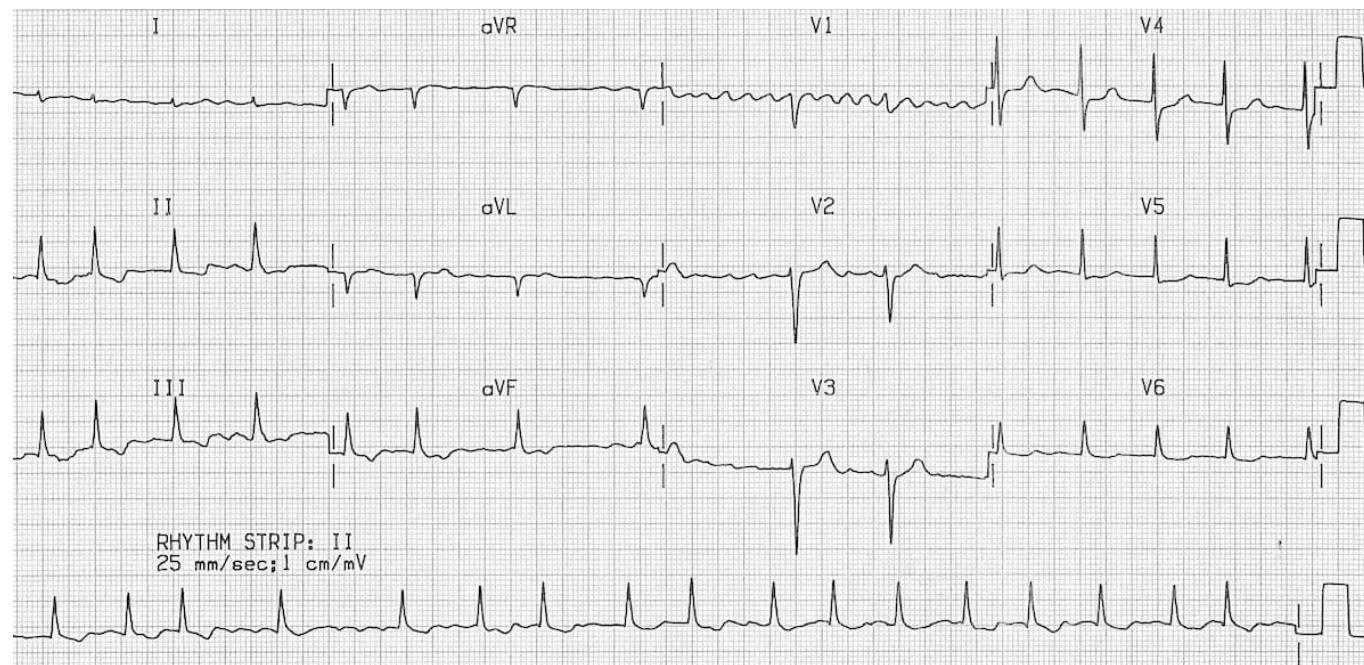
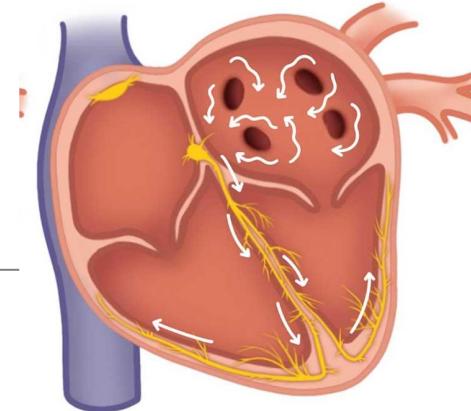
Atrial Flutter

- 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

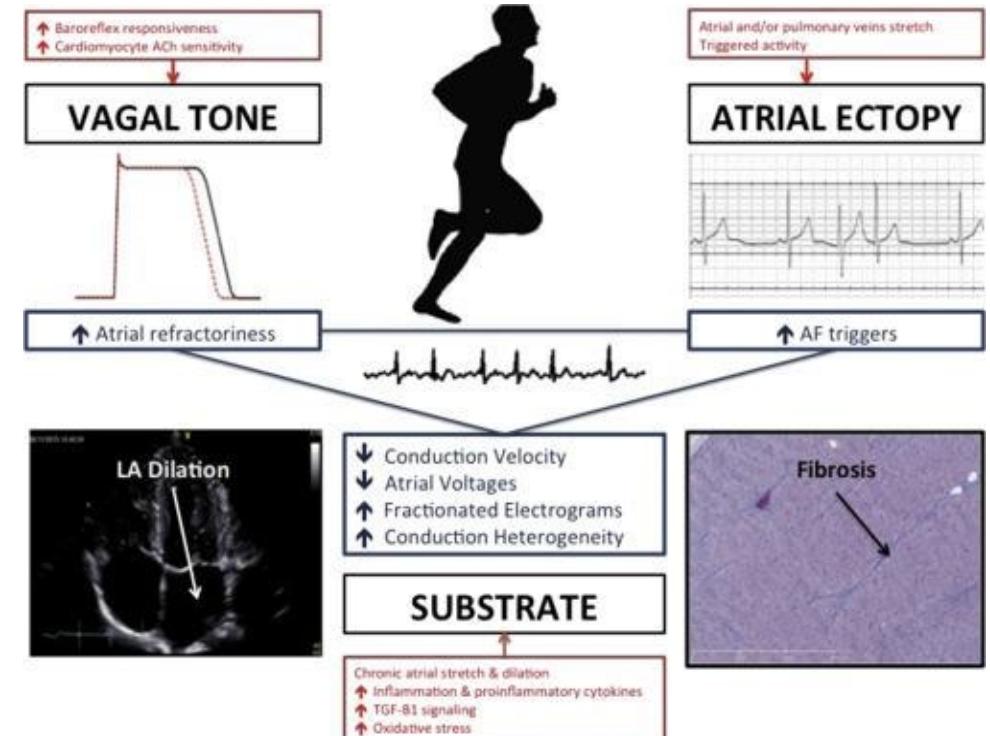
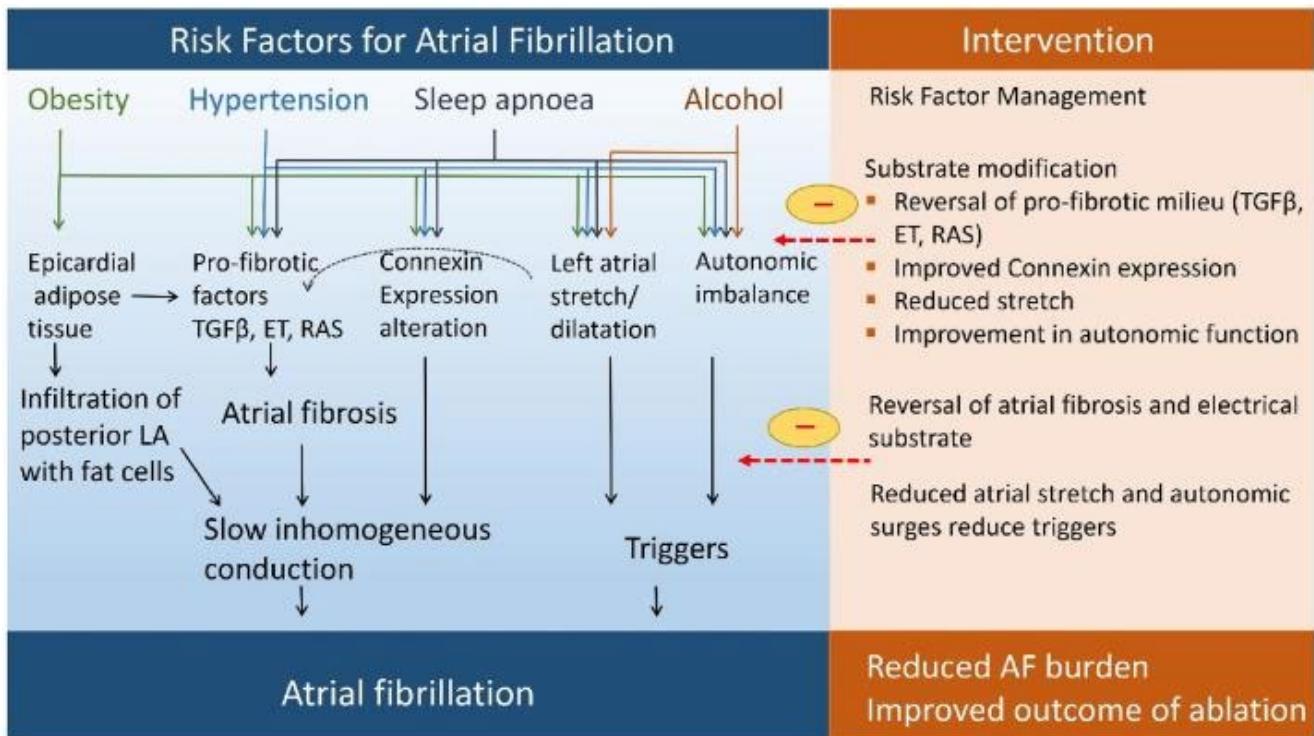


Atrial fibrillation

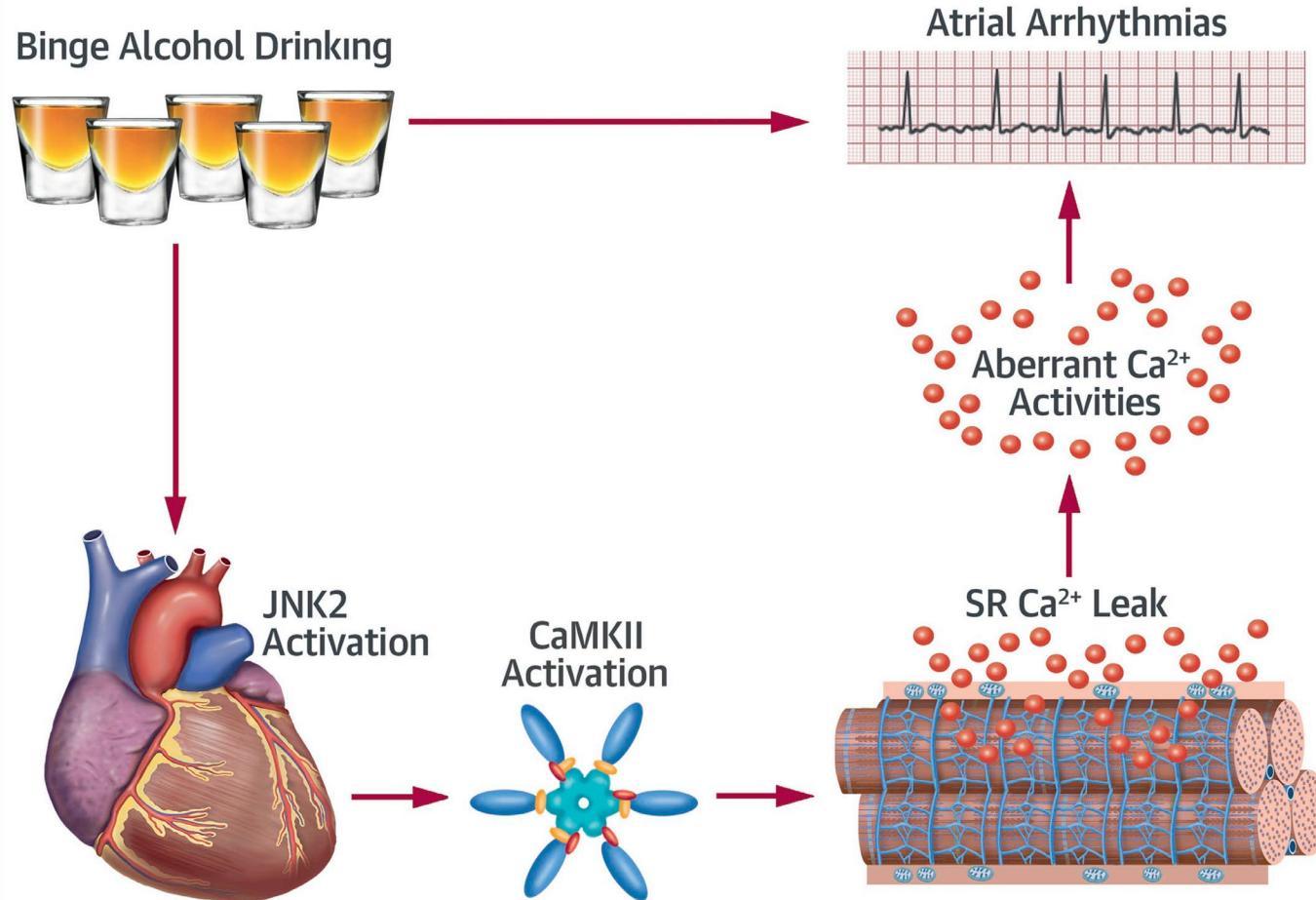
- Irregularly irregular rhythm
- No P waves
- Irregular R-R interval/ventricular rate
- Fibrillatory waves may be present
- Paroxysmal AF – episodes that last less than a week
- Chronic AF – where the physicians have accepted that the AF is there and no further attempt to maintain sinus rhythm is made
- High risk of stroke



Risk Factors



CENTRAL ILLUSTRATION: Alcohol-Evoked JNK Activates CaMKII to Promote Atrial Fibrillation



Yan, J. et al. J Am Coll Cardiol. 2018;71(13):1459-70.

Review

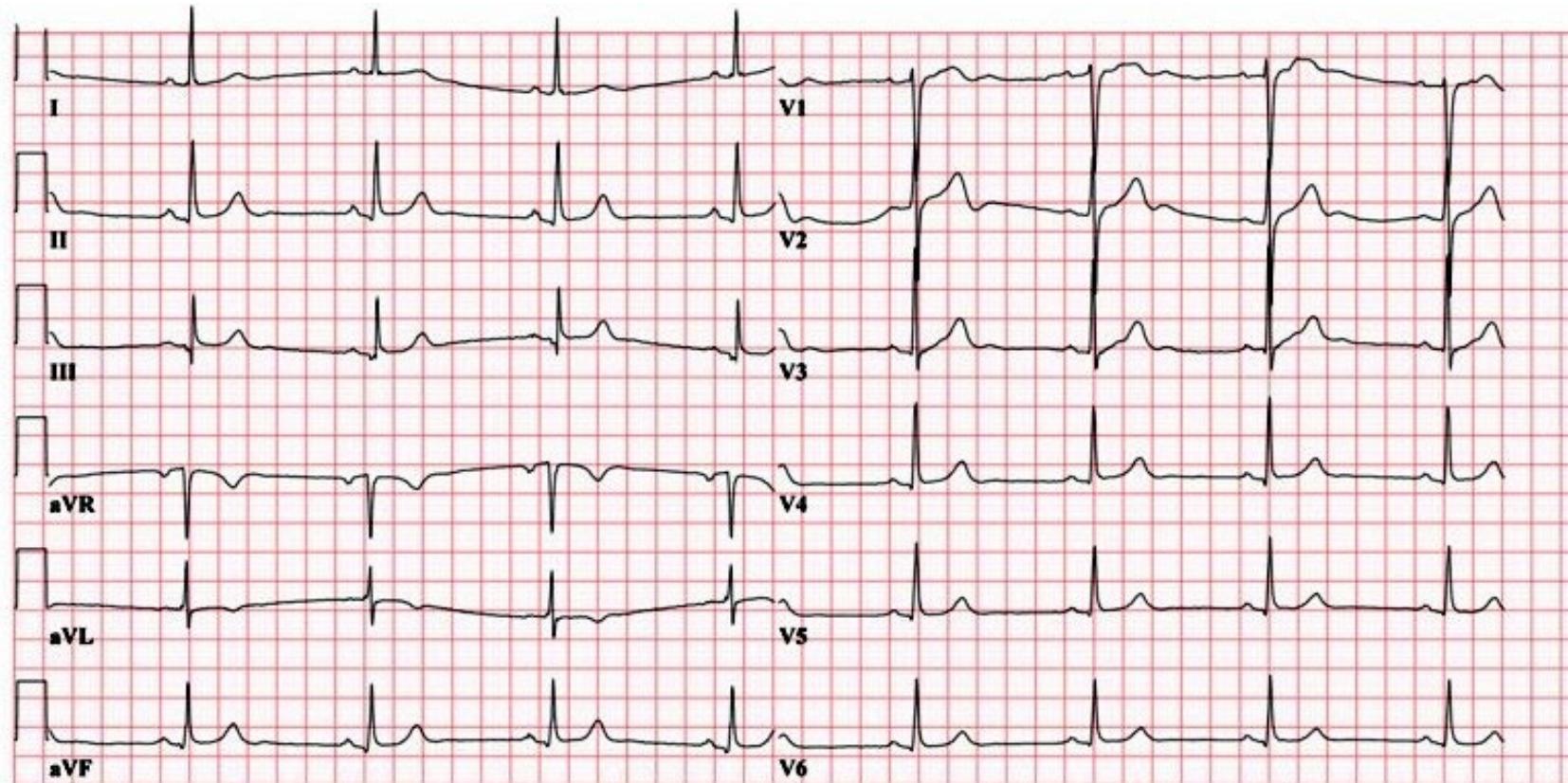
- Brady - sinus node - slow
- AV Block - AV node - slow
- ST depression - Ischemia
- ST elevation - infarction
- Tachy – atria -ventricles- fast
- Ventricular tachy / fibrillation - ventricles fast
- Atrial flutter / fibrillation - atria fast

CASES

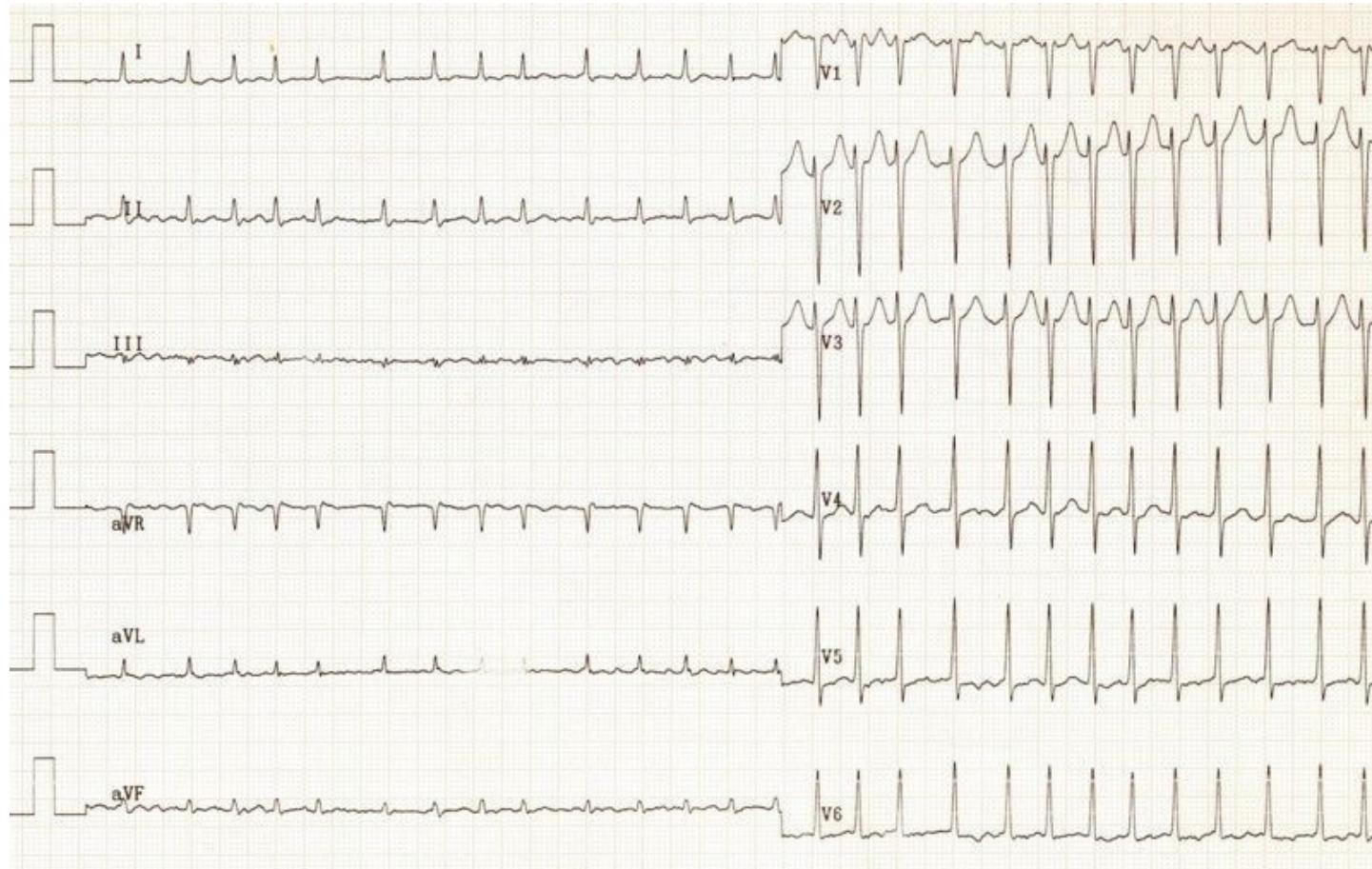
A patient has a 24 hour ECG monitor put on. You find this rhythm occurring at 2 am, what is going on ? Patient does not report any symptoms



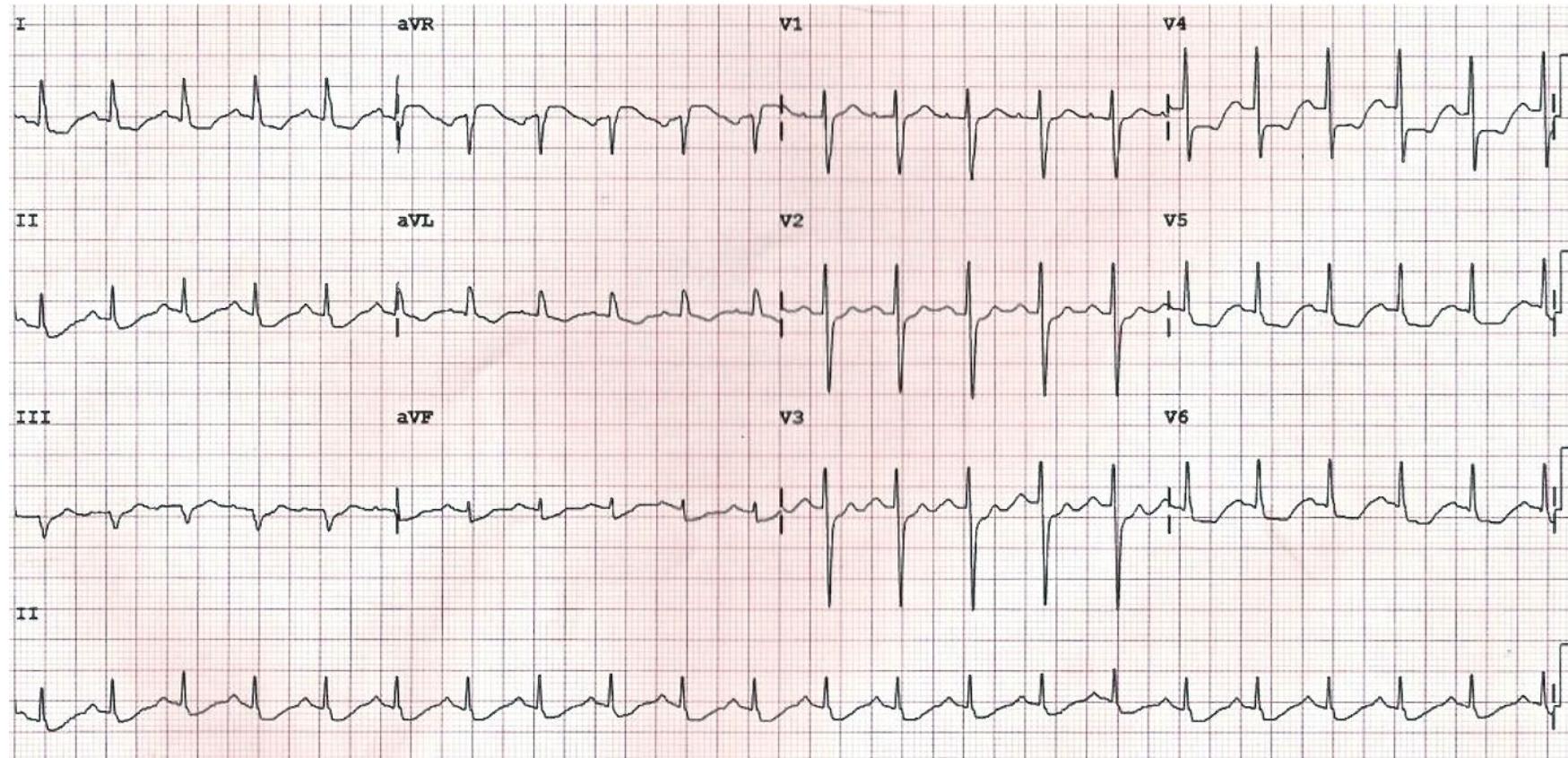
A 72 year old lady presents with collapse. This is her ECG. Present your findings. How would you proceed?



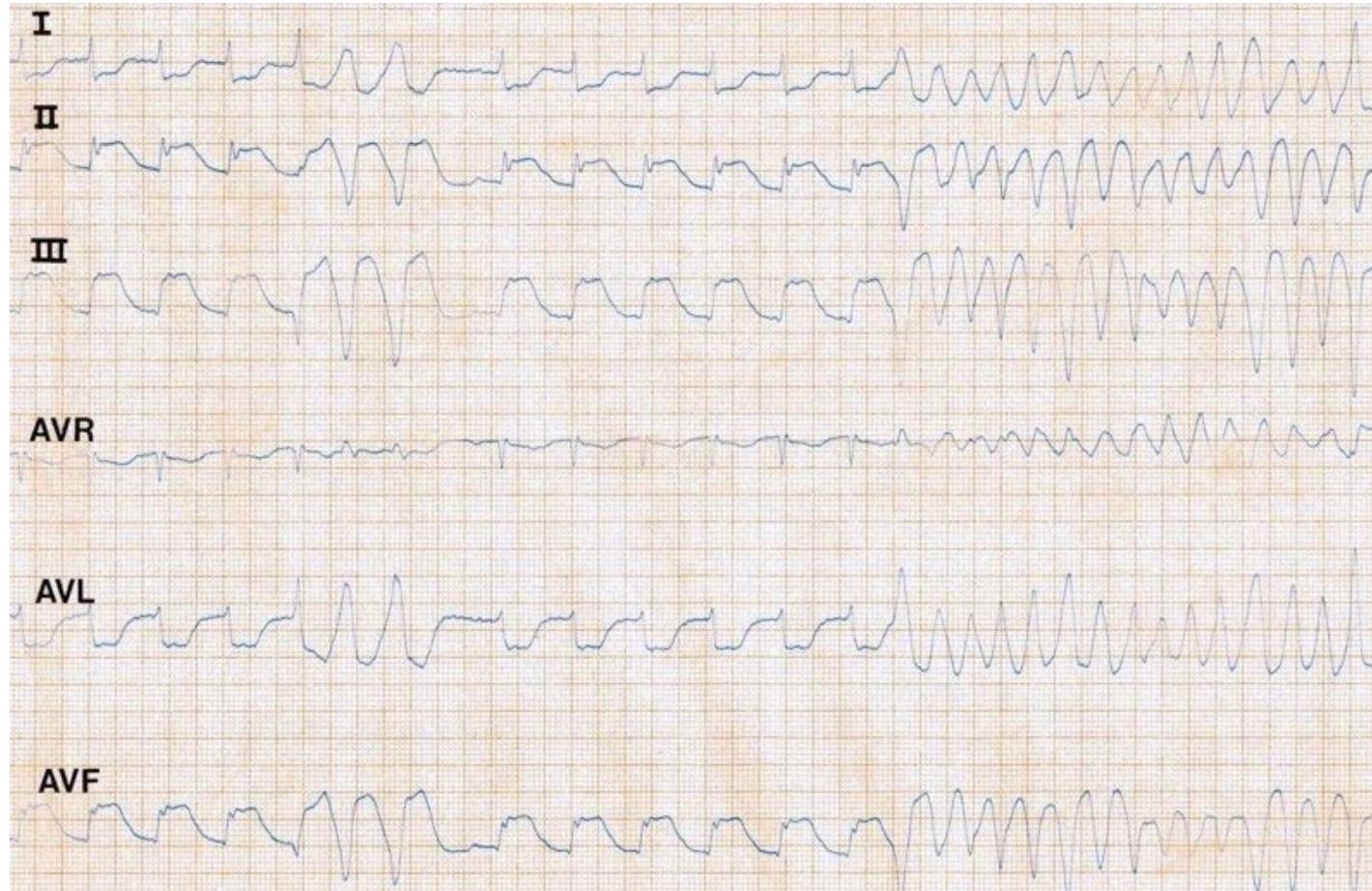
35 yr old male presents to ED, after a night of binge drinking over the weekend



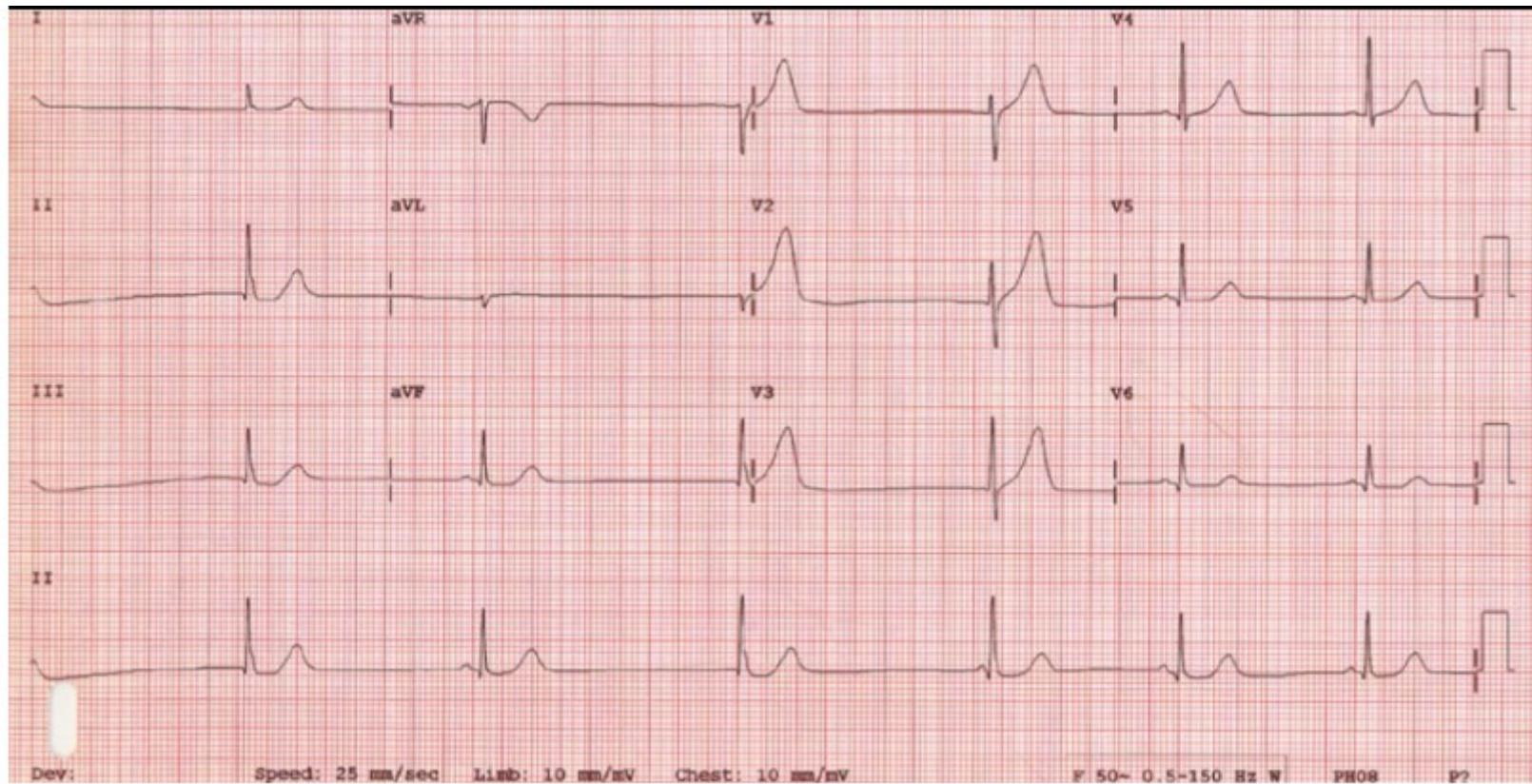
A 65 year old woman presents with chest pain radiating to her jaw and down her left arm. It feels like her 'normal' angina but on this occasion it has not eased with GTN spray.



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?

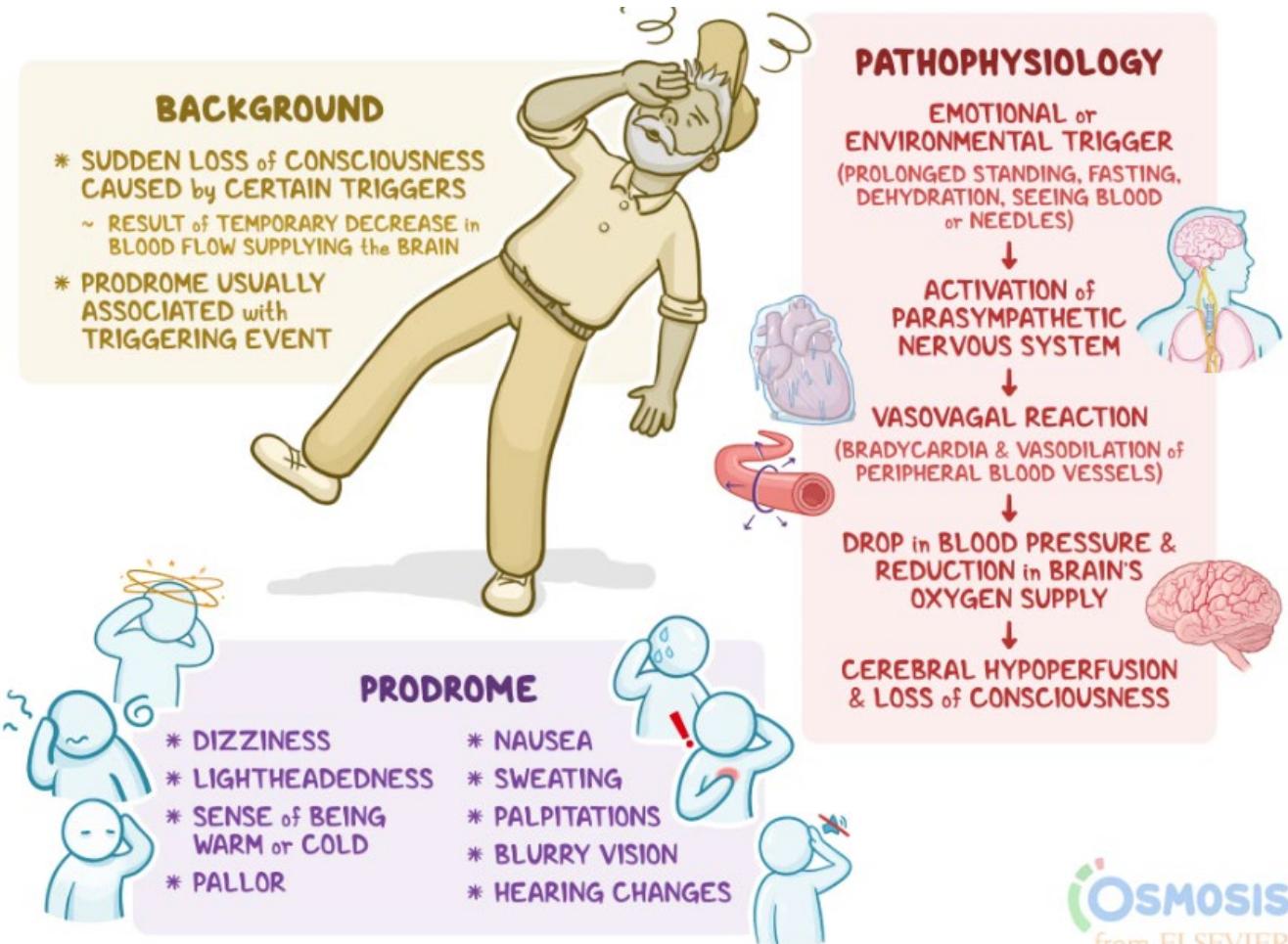


50 year old male, passes out while witnessing a car accident. EMS staff on scene get this ECG

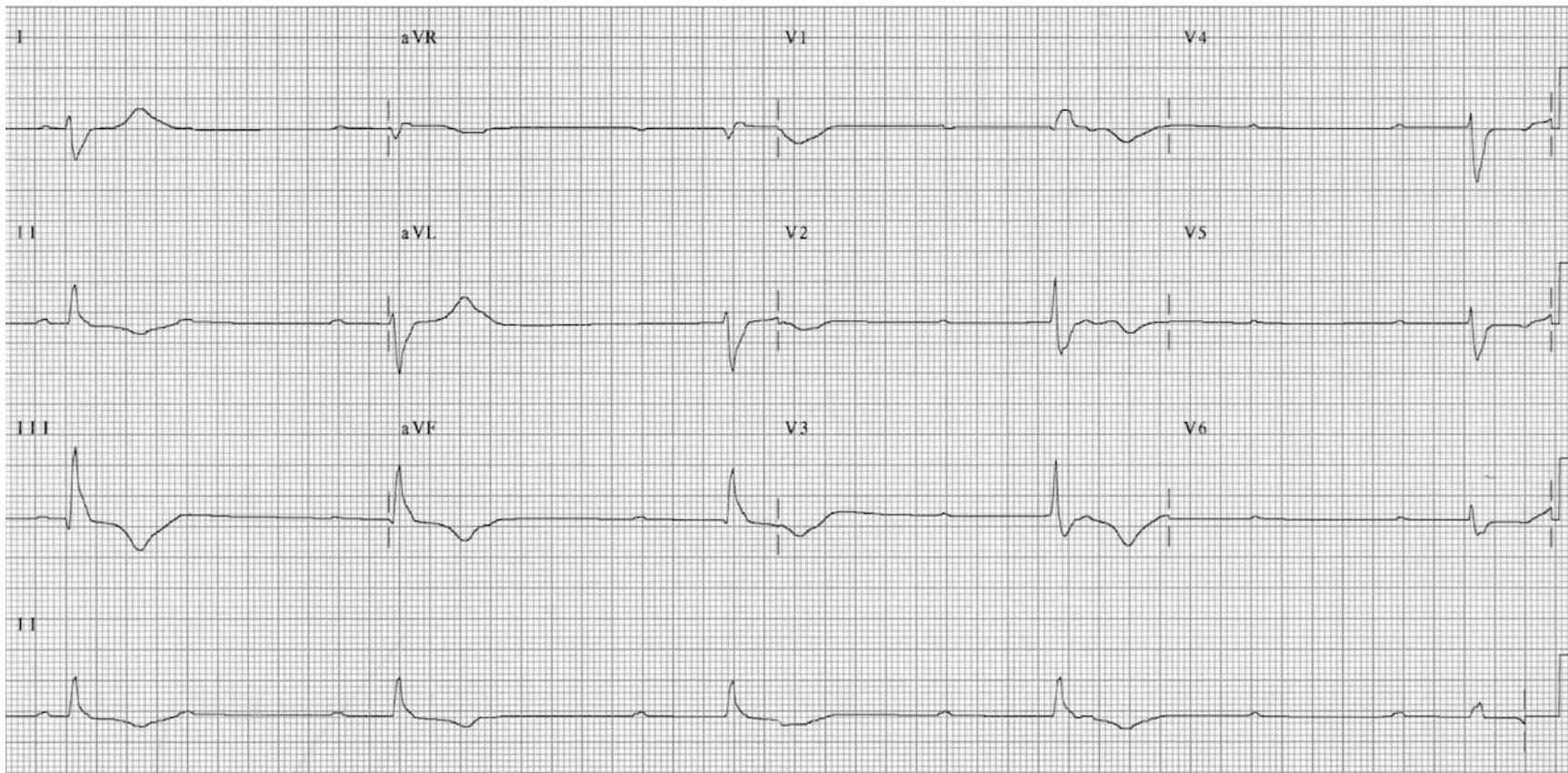


Vasovagal Syncope

- Trigger
- Increased vagal firing
- Decrease in HR – can be several seconds long
- Decrease in blood flow return to the heart
- Drop in mean blood pressure -> loss of consciousness



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



55 yr old , feels palpitations after 5 cups of coffee



Patient reports for an annual medical check up. What do you think is going on ?



THANK YOU FOR LISTENING

Further reading or courses

Cardiac Physiology in Practice – www.cardiacphysinpractice.com

Life in the fast lane – www.litfl.com

Professionals in Cardiac Sciences Australia – www.picsa.org.au