EKG Refresher

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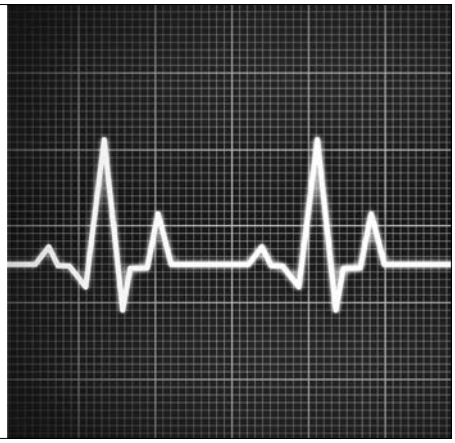
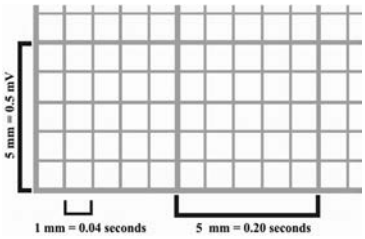


INTRODUCTION

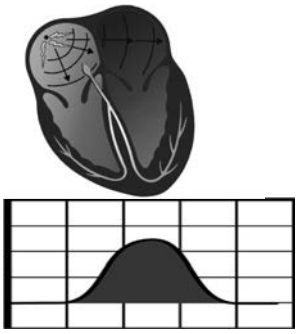
- General Principles
- Enlargement and Hypertrophy
- Arrhythmias
- Conduction blocks
- Preexcitation Syndromes
- Myocardial Infarcts and ischemia
- Electrolyte Disturbances
- Examples

General Principles



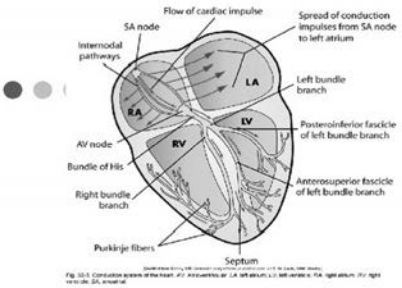
- Smallest box is 0.04 seconds and 1mm
- Bigger boxes 0.2 seconds and 5 mm
  - Composed of 5 small boxes of 0.04 seconds

Atrial Depolarization



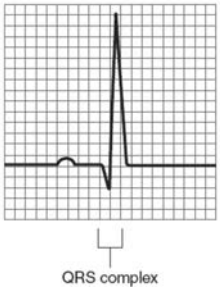
- SA node in the upper Right atrium fires and atria contract first right atrium then left atrium
- Forms a P Wave which consist of the first half right atrium second part left atrium
- Then a pause occurs when the electrical signal reaches the AV node near the intraventricular septum that delay conduction to nearly a pause to allow the atria to complete contraction before contraction of ventricles occurs

Ventricular Depolarization

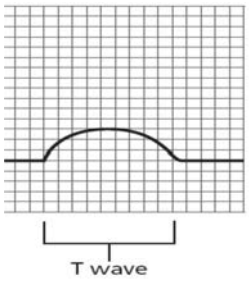


- After the pause from the AV node the current continues down to the path of the ventricles reaching first the
  - bundle of His
  - then Bundle branches
  - and lastly Purkinje fibers
    - Note: LBB splits into
      - Septal fascicle
      - Left anterior fascicle
      - Left posterior fascicle

QRS Complex

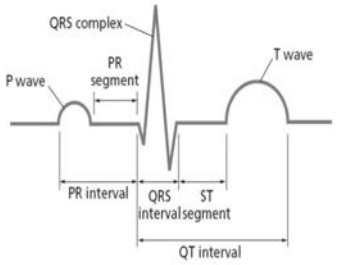


- First part of QRS is from the depolarization of the IV septum via septal fascicle of the LBB
  - Small Q wave
- Both ventricles then depolarize making up the remainder of the complex which structurally depicts of the left ventricle due to size
  - LV is 3 times the size of RV in normal cases

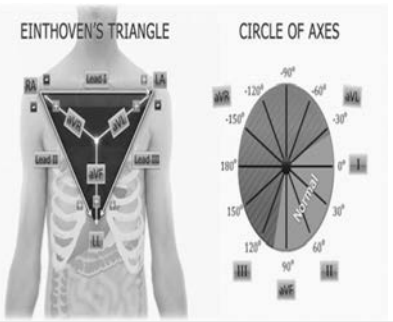


- A much slower electrical current than Ventricular Depolarization that gives us the T wave

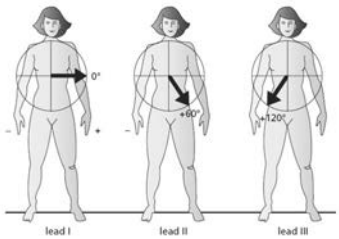
Nomenclature



- PR interval
  - is the start of AD to start of VD
- PR segment
  - end of AD and start of VD
- QRS interval
  - time of VD
- ST segment
  - end of VD to start of VR
- QT interval
  - start of VD to end of VR
- R to R interval
  - 1 cycle

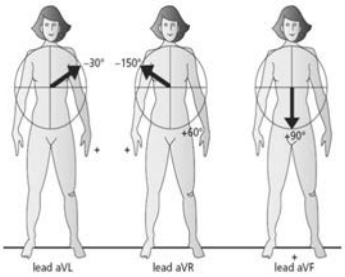


3 Standard limb leads

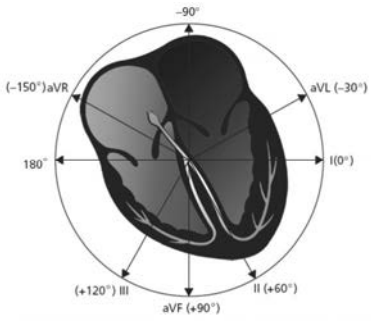


- 3 Standard limb leads
  - Lead I
    - Positive Left arm, negative right arm
    - Vector to 0 degrees
  - Lead II
    - Positive legs, and negative right arm
    - Vector to positive 60 degrees
  - Lead III
    - Positive legs, negative left arm
    - Vector to positive 120 degrees

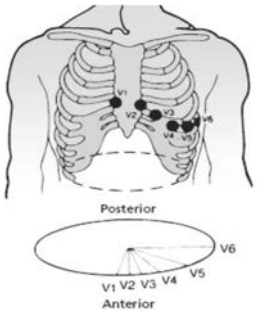
3 Augmented limb leads



- 3 Augmented limb leads
  - aVL
    - Positive left arm, other limbs negative
    - Vector is negative 30 degrees
  - aVR
    - Positive right arm, other limbs negative
    - Vector is negative 150 degrees
  - aVF
    - Positive legs, other limbs negative
    - Vector is positive 90 degrees



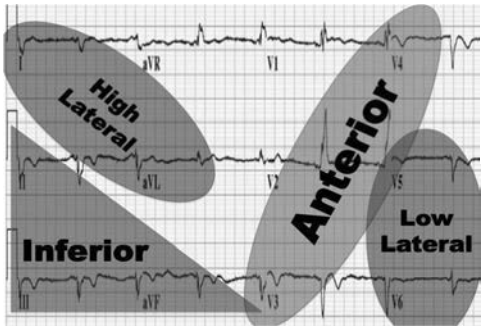
Lead	Angle
Inferior Leads	
Lead II	+60°
Lead III	+120°
Lead aVF	+90°
Left Lateral Leads	
Lead I	+0°
Lead aVL	−30°
Right-sided Lead	
Lead aVR	−150°



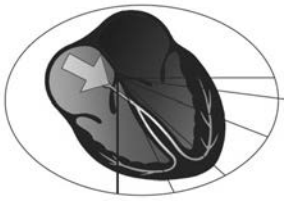
- V1
  - 4th intercostal space of right side of sternum
- V2
  - 4th intercostal space of left side of sternum
- V3
  - Between V2 and V4
- V4
  - 5th intercostal space in the midclavicular line
- V5
  - Between V4 and V6
- V6
  - 5th Intercostal space in the midaxillary line

Categorizing leads

- Leads
  - Anterior leads: V2, V3, V4
  - Left Lateral leads: I, aVL, V5, V6
  - Inferior leads: II, III, aVF
  - Right ventricular: V1, aVR



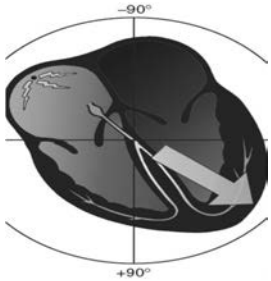
Recap on the P Wave



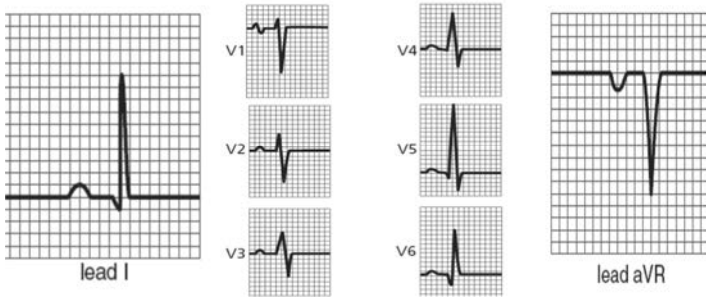
- Current starts in the SA node (upper right atrium) and goes in the direction to AV node on general direction of the left ventricle
- PR interval from start of Atrial depolarization to start of Ventricular depolarization is usually 0.12 to 0.2 seconds (3-5 small boxes)

- aVR
- Lead I
- V6
- Lead III
- V1

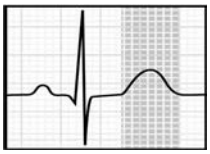
QRS Complex

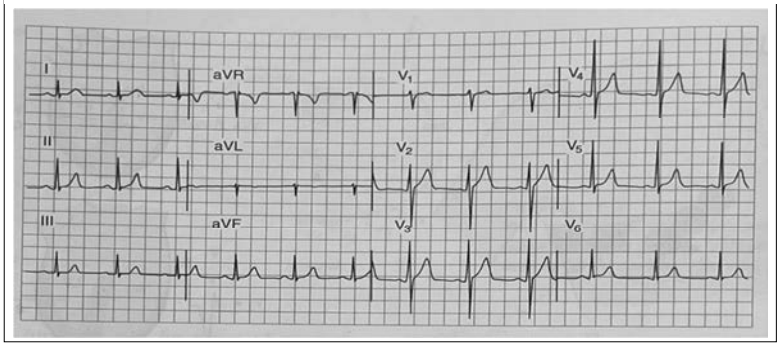


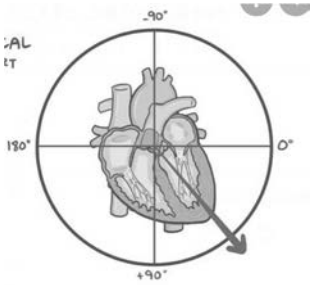
- Starts off after ther AV node current runs down bundle of HIS then down small LBB to the small septal fascicle creating a small septal Q wave, then followed by massive positive deflection creating the R wave followed by a deep negative deflection
- QRS Interval duration of QRS usually last 0.06-0.1 seconds



- Most T waves positive in leads with tall R waves

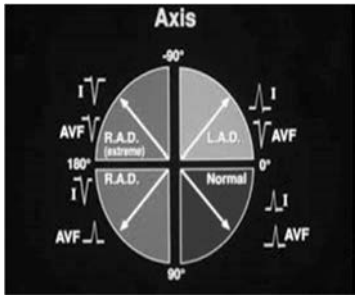






- Our normal electrical axis goes in the vector direction to the left lower quadrant from 0 degree to +90 degrees.
- To help determine axis on EKG use lead I (0 degrees) and aVF (+90)
  - Positive QRS in Lead I lets us know vector is between -90 and +90 degrees
  - Positive QRS in aVR lets u know vector is between 0 to +180.
  - To find specifically find the biphasic then plot perpendicular to it

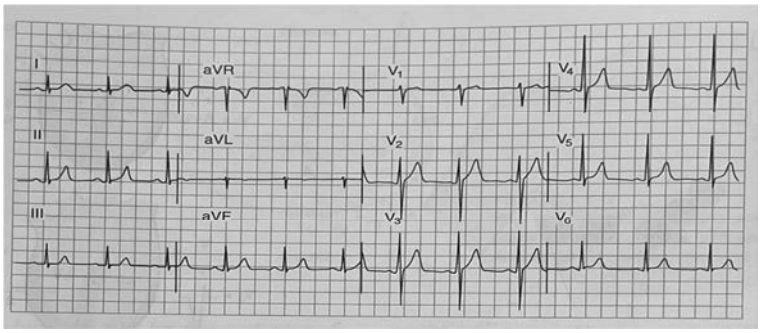
4 different axis

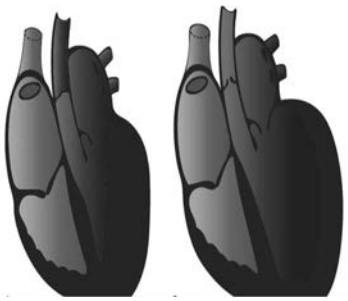


- Left axis deviation:
  - Lead I is positive and aVF is negative
  - From 0 to -90 degrees
- Right axis deviation
  - Lead I is negative and aVF is positive
  - From +90 to +180
- Extreme axis deviation
  - Lead I and aVF negative
  - From +180 to -90

QRS Deflection		Axis
Lead I	Lead aVF	
+	+	Normal
+	-	LAD
-	+	RAD
-	-	Extreme Axis

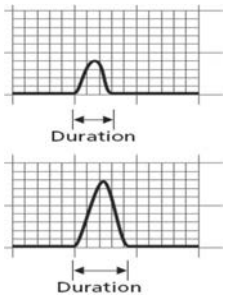
- Remember the medical student thumb technique
- Let's try on the next slide





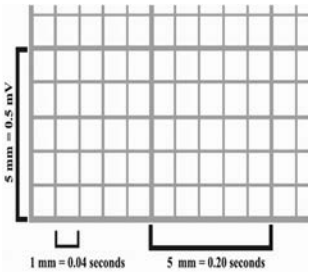
- Enlargement gives reference to dilatation of any of the 4 chambers of the heart
  - Usually referred more to Atrial enlargement but can also be ventricle
  - Valvular disease can be major causes such as mitral and aortic regurgitation
- Hypertrophy in EKGs gives reference to the increase in mass of a ventricle
  - Having to work out harder increasing in size due to causes such as HTN and Aortic stenosis

EKG changes hypertrophy or enlargement



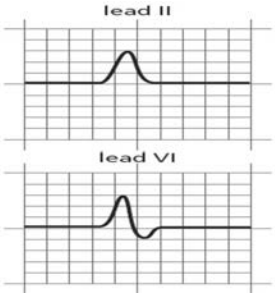
- Longer depolarization period leads to an increases in duration (width)
- Increase in voltage from increase mass leads to an increase in amplitude (height)
- Increase in size can cause a shift in the electrical axis vectors

Atrial enlargement



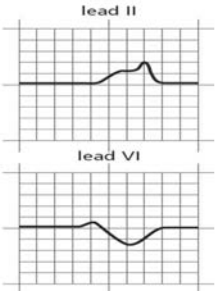
- P wave should be less than 0.12 seconds (3 small boxes) in duration and voltage should be less than 2.5 mm (2.5 small boxes up)

Right atrial enlargement



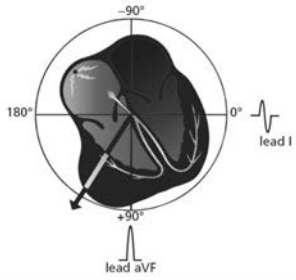
- With Right Atrial Enlargement duration does not increase but height does, the duration does not change because the second part of the p wave is left atrium in source
- Tallest P wave now in lead aVF or lead III no longer II
- Best view is in lead II and V1
- Criteria needs to be P waves with height >2.5 mm in a inferior lead (II, III, aVF)

Left Atrial Enlargement



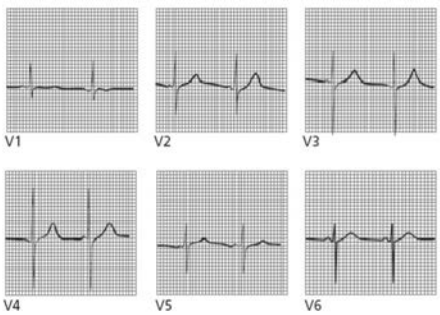
- Left Atrial Enlargement involves the 2nd part of the P wave causing an increase in height and width
- Criteria is
  - in V1 the second part of the P wave must have a drop >1 mm below the baseline
  - also the left atrial portion of the P wave must have a width >0.04 seconds

Right Ventricular Hypertrophy



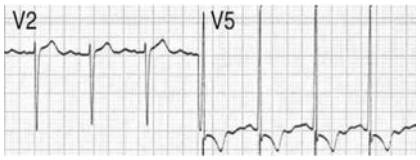
- Usually seen in Right axis deviation
  - Lead I negative aVF positive
- Common causes of RVH Pulmonary disease or Congenital heart disease

RVH



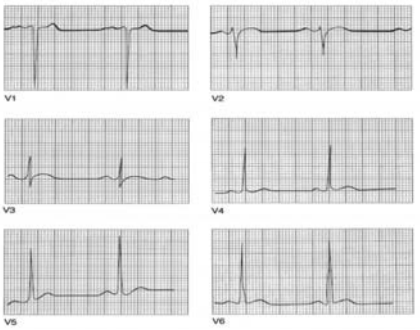
- Precordial leads V1-V5 mildly reversed
- Lead V1 now R waver is taller than the S wave
- Lead V6 now S wave is taller than the R wave

Left Ventricular Hypertrophy



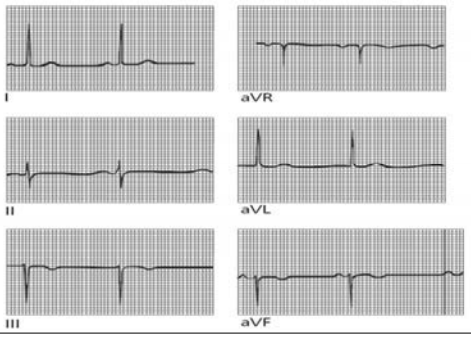
- Common causes HTN, aortic stenosis
- Usually Left axis deviation noted but not diagnostic needs to meet criteria for true diagnosis, remember Lead I positive and aVF negative; -90 to 0 degrees
- Key features are:
  - enlarged R wave height in leads over the left ventricle
  - S wave height enlarged in leads over the right ventricle

Left Ventricular Hypertrophy Criteria



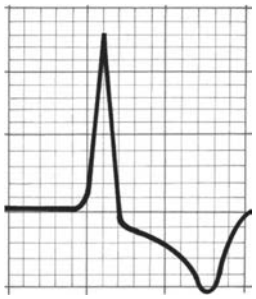
- Precordial Leads Criteria
  - R wave height in lead V5 or V6 plus the S wave height in V1 or V2 >35 mm
  - R wave height in lead V5 >26 mm
  - R wave height in lead V6 >20 mm
  - R wave height in V6 > R wave height in V5

LVH Criteria

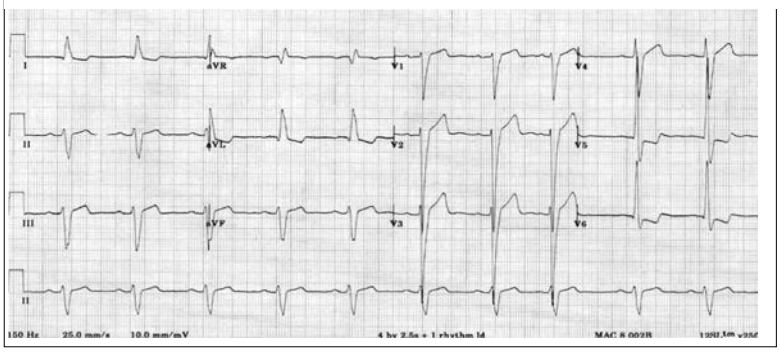


- Limb Leads Criteria
  - R wave height in lead aVL >11 mm
  - R wave height in lead aVF >20 mm
  - R wave height in lead I > 13 mm
  - R wave height in lead I plus the S wave height in lead III > 25 mm

Ventricular secondary effects

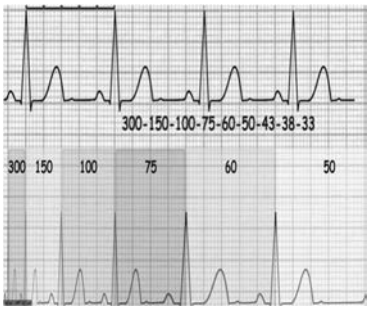


- Know as secondary repolarization abnormalities
  - 1) Downslopping ST segment depression
  - 2) T wave inversion
  - Mechanisms of action theories to be due to strain
- If seen most common in
  - RVH in V1 and V2
  - LVH in I, aVL, V5, and V6



- A disturbance in rate, regularity, site of origin, or conduction
- Common causes
  - Hypoxia: lung disease, PE
  - Ischemia\irritability: myocarditis
  - Sympathetic stimulation: hyperthyroid, CHF, CNS, exercise
  - Drugs
  - Electrolyte disturbance: K, Ca, Mg
  - Bradycardia: bradytachycardia: SSS
  - Stretch: enlargement and hypertrophy, CHF, Valve disease

Calculate the Rate

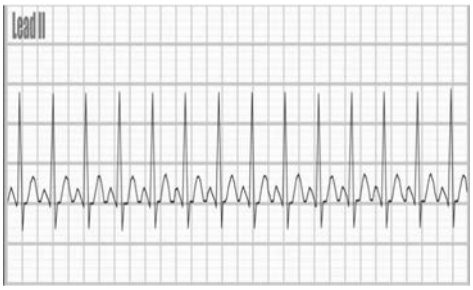


- Quick 3 step method to determine heart rate
  - Locate a R wave close to a darker line
  - Count the large squares until the next R wave
  - Quick ways is to divide 300 by the number of big boxes
    - So if 1 box is the R to R = 300 bpm
    - …. If 2 boxes the R to R = 150 bpm
    - …. If 3 boxes the R to R = 100 bpm
    - …. If 4 boxes the R to R = 75 bpm
    - …. If 5 boxes the R to R = 60 bpm
    - …. If 6 boxes the R to R = 50 bpm

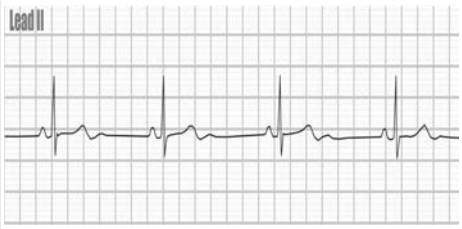
Types of Arrhythmias

1. Sinus arrhythmias of sinus origin: start with depolarization of the SA node but either to slow or fast or irregular
2. Ectopic rhythm: electrical current arising from other foci than the SA node
3. Reentrant arrhythmias: Trapped electrical current within a circuit, it's shape and track is made by an anatomic or electrical anomaly
4. Conduction blocks: starts at the SA node and has "road blocks" along the normal pathway
5. Preexcitation syndromes: Electrical current uses a accessory pathway that bypasses the normal electrical direction, a "short cut"

Arrythmias of Sinus Origin, Sinus Tachycardia

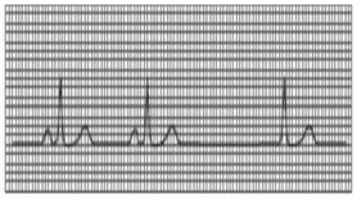


- Sinus tachycardia
  - Exercise, stimulants
  - CHF
  - Lung disease
  - Hyperthyroid
  - HR > 100 bpm for Cardiologist >110 bpm
- Inhalation = increases HR



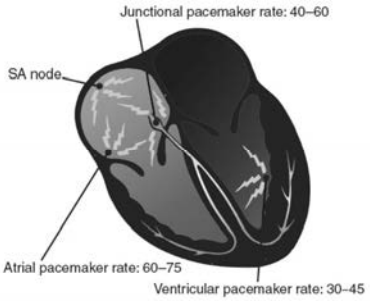
- Sinus Bradycardia
  - Medications: beta blockers, Calcium channel blockers, opioids
  - Athletes
  - HR <60 BPM
- Expiration = slows HR

Sinus arrest, Asystole then escape beats



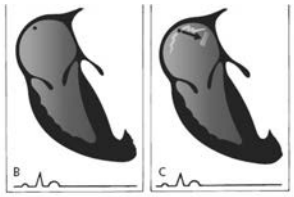
- When the SA node stops sinus arrest occurs, prolonged sinus arrest with no other electrical activity occurs it is called Asystole
- During sinus arrest other myocardial cells can fire and act similar to pacemaker cells and create escape beats which rescue the pathway
- In example notice no P wave in the junctional escape beat

Nonsinus Pacemakers



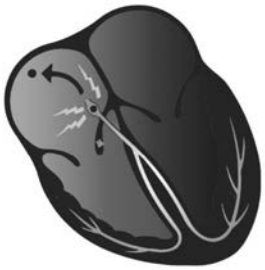
- Atrial pacemaker cells have a rate of 60-75 bpm
- Pacer cells around the AV node are called junctional pacemakers which fire at 40-60 bpm
- Ventricular pacer cells fire at 30-45 bpm
- Any of these can assist when the SA node is not firing adequately, the most common helper are the junctional pacers
  - These escape beats (will have no P wave) but retrograde P waves may follow

Sinus arrest vs. Sinus block



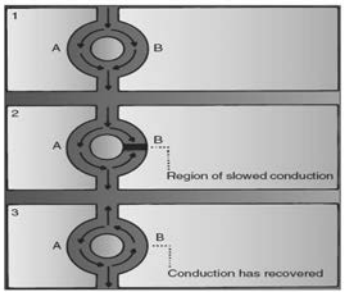
- Sinus arrest
  - there is a malfunction of the sinus mechanism to fire its current
  - No electrical activity
- Sinus exit block
  - Failure of current to leave the SA node and into the atria
  - Electrical activity

Ectopic Rhythms



- Rhythms that originate from other areas of the atria non SA node
- They can be single or sustained beats
- Formed by intrinsic pacemaker electroactivity at a single foci or roaming one
  - Can be enhanced by stimulants, digitalis toxicity, beta agonist, caffeine, alcohol, illicit drugs

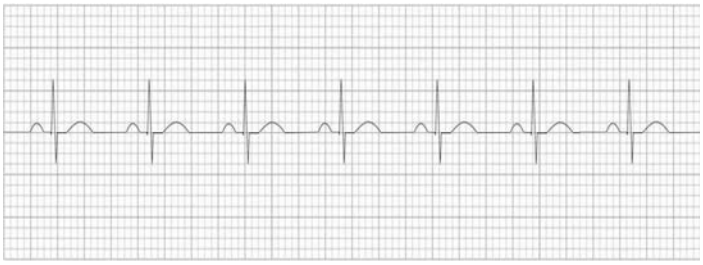
Reentrant Rhythms



- Once again a current formation is originated from non SA node and takes over the conduction pathway; a problem of impulse transmission
- 1) is normal in our diagram
- 2) there is slowed conduction by ischemia or fibrosis that causes a delay which throws off the cycle causing to pathway A to now circle back around
- 3) a new reentrant conduction is now formed and overrides SA node flow

The big 4 questions when analyzing a EKG

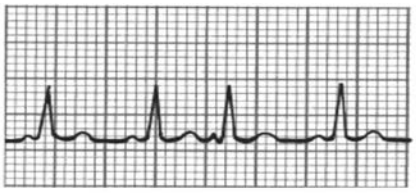
1. Are P waves present?
   - Check Lead II and aVR for positive p waves
   - If yes then origin from the atria
   - If no P waves; then it arises below the atria in the AV node or ventricles
   - If abnormal p wave location
     - Think retrograde P waves
2. Are the QRS complexes narrow or Wide
   - <0.12 seconds or > 0.12 seconds
   - Narrow QRS means normal current flow a wide QRS means origin is in the ventricles moving slower and causing a longer duration
3. What is the relationship between the P waves and the QRS Complexes?
   - Does P wave always precede? Are they 1:1, sinus or atrial origin?
4. Is the Rhythm Regular or Irregular?
   - We will get to this in the next slides



- Premature Atrial Contractions (PACs)
- AV Nodal Reentrant Tachycardia (Paroxysmal SVT)
- Atrial Fibrillation
- Atrial Flutter
- Multifocal Atrial Tachycardia
- Paroxysmal Atrial Tachycardia (Ectopic atrial tachycardia)
- AV reciprocating tachycardia

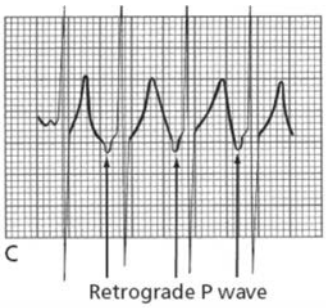
- Originate in the atria or AV node
- Can be 1 beat or sustained, can last second to a lifetime
- Look for P waves in Lead II or V1

PACs



- PACs
  - Origin is the atria
  - Faster P wave
  - Different shaped from prior P waves
- They are conducted normally to the ventricles and have narrow QRS complexes

AV Nodal Reentrant Tachycardia (Paroxysmal SVT)



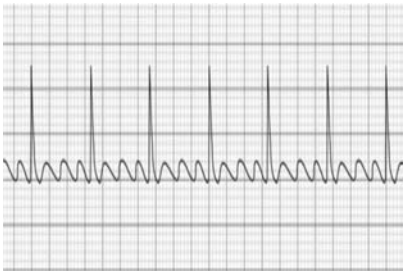
- Common
- Rapid onset, can be set off by a PAC or Junctional premature beat (similar to escape beat but happens earlier)
- Seen in healthy hearts
- Regular rhythm, rate 150-250 bpm
- As the name implies has a reentrant pathway track within the AV node
- Lead II or III has retrograde P wave
  - Pseudo R' in lead V1 that reflects a superimposed retrograde P wave in QRS
- Narrow QRS

Carotid massage effects

- Right carotid stimulates the SA node thru vagal input
- Left carotid stimulates the AV node
- Can stop or slow down reentrant currents

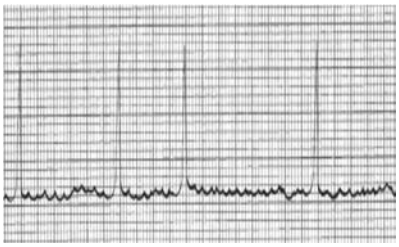
- However caution if you do choose to do it

Atrial Flutter



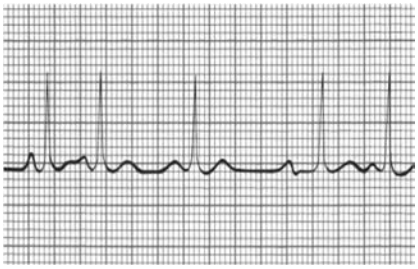
- Atrial origin, famous sawtooth appearance
- Regular rhythm with rate of 250-350 bpm
- Created by a reentrant pathway that cycles around the annulus of the tricuspid valve
- Depolarization in atrium is so fast that P waves demarcated by the baseline are not visualized, instead a continues up and down flutter wave is seen, usually best seen in Lead II and III
- The AV node cannot process the rapid flow from the atrium and unable to keep up with QRS leading to what is a AV block
- A. Flutter usually has a 2:1 AV block (2 flutter waves per 1 QRS)
- Carotid massage makes it worst increase to up to 5:1

Atrial Fibrillation



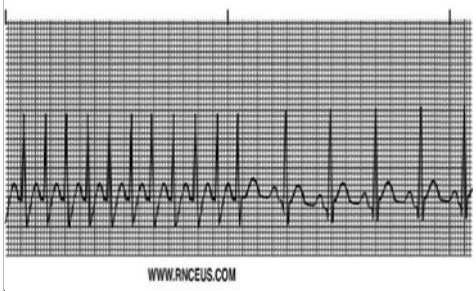
- Erratic atrial electrical activity, can fire up to 350-500 currents to the AV node which cannot process all and with an average rate of 120-180 bpm
- Caused by several small reentrant pathways swirling around
- No real P waves
- Baseline will appear nearly flat with mild fibrillation "undulating" waves
- Irregularly irregular appearance of QRS without P waves is key

Multifocal Atrial Tachycardia and Wandering Atrial Pacemaker



- Irregular, rate of 100-200 bpm, when rate is under 100 its called WAP
- Unsystematic discharges of multiple different ectopic atrial foci
- Not affect by Carotid massage
- Has clear P waves before QRS unlike A fib
- Criteria must have 3 different P wave morphologies

Paroxysmal Atrial Tachycardia



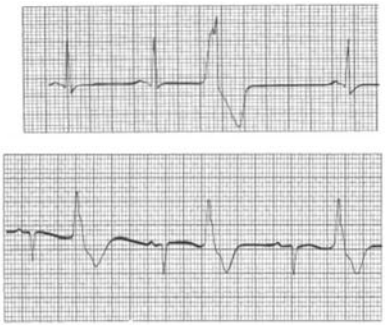
- Regular rhythm with rate of 100-200 bpm
- From a reentrant pathway within the atria or from increased automaticity of ectopic atrial focus
- Has a "warm up" and "cool down" state that helps distinguish between PSVT
- No affected by Carotid massage

Ventricular Arrythmias

- Premature Ventricular Contractions
- V Tachycardia
- V Fibrillation
- Accelerated Idioventricular Rhythm
- Torsade de Pointes

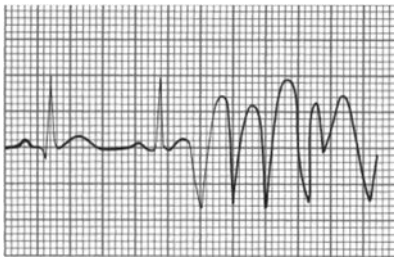
- Arise the below the AV node
- Mild to life threatening

PVCs



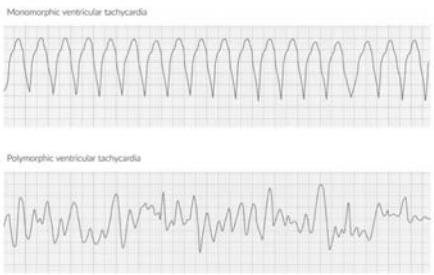
- Most common of the Ventricular arrythmias
- Wide and abnormal QRS due to VD does not follow the traditional Ventricular circuit
- QRS >0.12 seconds (3 smalls boxes) in most leads
- May have retrograde P waves or no P waves
- usually has a pause before the next beat, if no pause called a "interpolated PVC"
- Commonly seen on their own
- Caution if seen after a MI which can trigger V tach or V Fib
- Bigeminy when 1:1 one normal sinus run and one PVC
  - Trigeminy is when 2:1 two normal sinus beats and one PVC

PVCs rule of malignancy



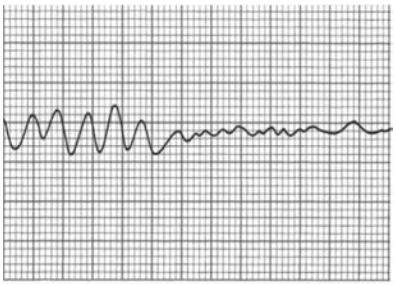
1. Recurrent PVCs
2. Consecutive run of PVCs of 3 or more
3. Different PVC morphologies
4. PVCs on T waves of prior cycle named ""R on T" phenomenon, very vulnerable time during VR and can cause V Tach to start
5. A PVC during a new MI

Ventricular Tachycardia



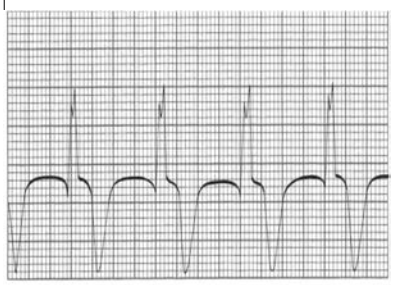
- A run of ≥3 consecutive PVCs
- Rate of 120-200 bpm
- Sustained if > 30 seconds
- Can be uniform or polymorphic
  - Uniform seen more in healed infarcts or scarred myocardial tissue
  - Polymorphic seen in acute coronary ischemia, new infarct, electrolyte changes, prolonged QT interval

Ventricular Fibrillation



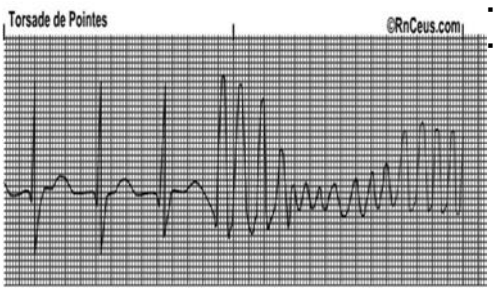
- Cardiac emergency can lead to sudden death
- Can be either (fine) smooth undulating or (coarse) spasmodic
- No clear cut QRS complexes
- No Cardiac output
- Causes
  - MI, Heart failure, hypoxemia, hypercapnia, shock, hypotension, electrolyte disturbance, stimulant drug overdose

Accelerated Idioventricular Rhythm



- Benign seen after new MI or after PCI
- Regular rhythm and rate of 50-100
- Likely ventricular escape focus
- Rarely sustained, when drops under 50 just idioventricular rhythm
- No P wave with wide QRS

Torsade de Pointe

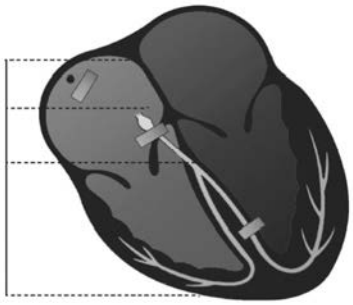


- "Twisting of the points"
- A derivative of V Tach with prolonged QT interval
  - Can be from congenital
  - Electrolyte disturbance Ca, Mg, K
  - Acute MI
  - Meds: SSRI, Tricyclics, antipsychotics, fluoroquinolones, Zofran, Azithromycin, …
  - PVC falling on T wave
  - Undulating around the baseline with change in height

Recap Supraventricular and Ventricular Arrhythmias

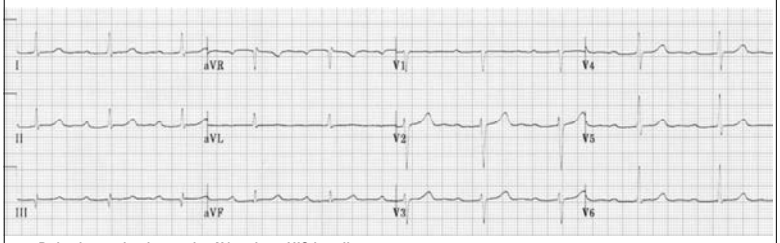
- Supraventricular
  - Narrow QRS <0.12 sec
  - Carotid massage may improve
- Ventricular
  - Wide QRS >0.12 sec
  - Not affected by Carotid massage

Conduction Blocks



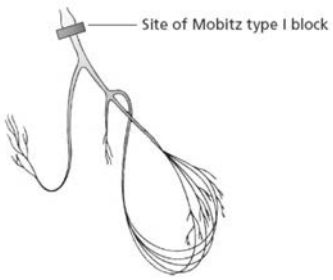
1. Sinus node block
   - Sinus exit block
     - SA node fires routinely but is blocked and not relayed by atrial tissue
     - Already discussed this earlier
2. AV block
   - A block between AV node and HIS bundle most common
3. Bundle branch block
   - Block at one ventricular branch or both or partial in left bundle

AV Blocks: First degree AV Block



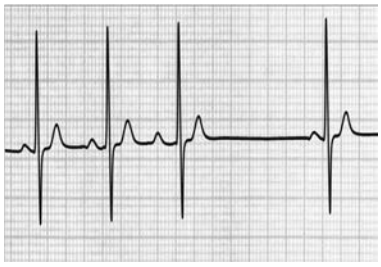
- Delay in conduction at the AV node or HIS bundle
  - More of a delay than a true block
- PR interval > 0.2 seconds (1 big box)
- Common and usually asymptomatic

AV Block: 2nd degree



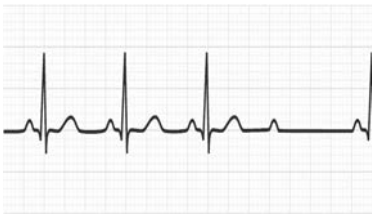
- AV node is not processing all atrial currents and will have a >1:1 P wave to QRS
- 2 type of 2nd degree block
  - Mobitz type 1 (Wenckebach)
  - Mobitz type 2

Mobitz Type I Wenckebach



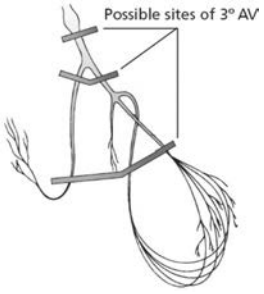
- Block within the AV node
- The block is variable and increasing with each proceeding impulse
- The new impulses encounter a longer delay in the AV node until a impulse fails to make it through the AV node creating 'dropped' QRS
- In other words PR interval that prolongs until a QRS is dropped
- Repeats this pattern

Mobitz Type 2

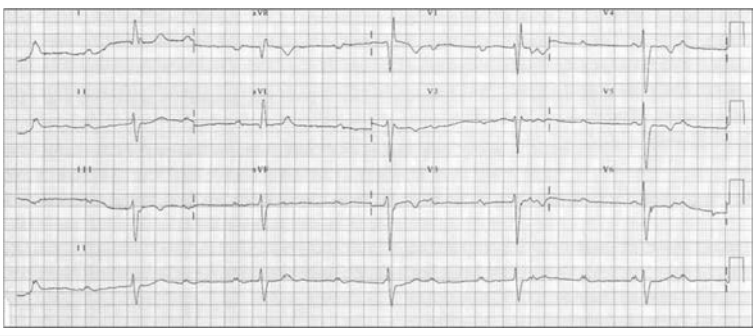


- Block is below the AV node in the HIS bundle or near it
- A "all or nothing" phenomenon
  - 2 or more normal cycles proceeded by a P wave with no QRS behind it
  - Inconsistent, has ratios of 2:1, 3:2 etc
- To differentiate from type 1 with Type 2 there is no PR interval lengthening
- More concerning than type 1

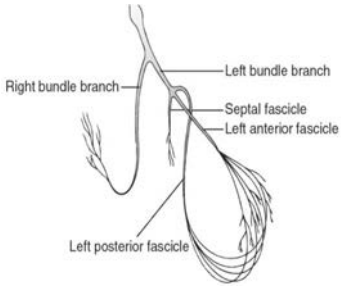
3 Degree AV Block



- No atrial current makes it to the ventricles
  - Ventricles react with escape beats with at rate of 30-45
- Complete heart block
  - Can be at the AV node or lower
- Now the atria is still beating at its 60-100 rate
- To diagnosis needs
  - AV dissociation = Atria and Ventricles beating independently with ventricles much slower rate than atria

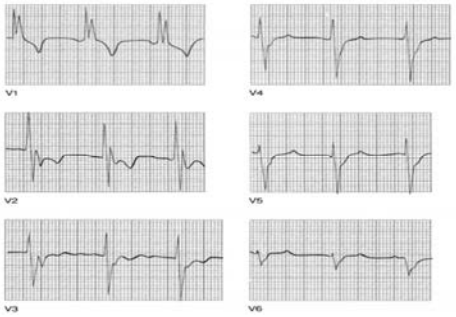


Bundle Branch Blocks



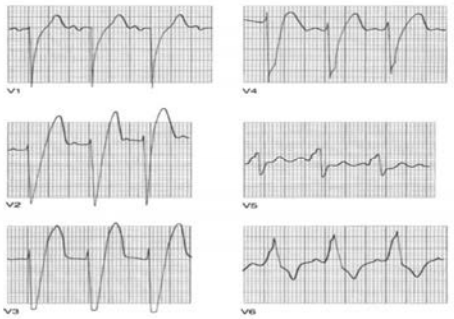
- Conduction delay or block thru the Right or Left bundle branches
- To help make diagnosis the QRS will have several changes
- Incomplete BBB
  - When LBBB or RBBB appear but QRS is 0.10 to 0.12 seconds

Right Bundle Branch Block



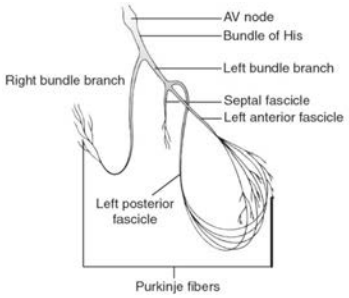
- Right ventricle depolarization is delayed and does not start until LV is nearly depolarized
- This delay in RV depolarization prolongs the total time for total VD creating >0.12 second QRS
- The wide QRS has a unique shape over the RV V1 and V2 unopposed after the LV has finished its cause a second R wave R prime making a rabbit ears look
- In the lateral leads it causes a reverse change of deep S waves

Left Bundle Branch Block



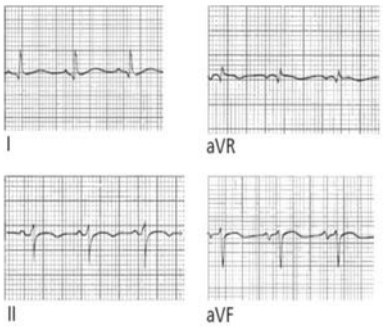
- LV depolarization is delayed
- QRS >0.12 seconds
- QRS over Lateral leads will have change in morphology with a lag in the rise of tall R waves that have notches on top and or broad
- QRS on RV leads will have broad S deep waves
- Ventricular hypertrophy cannot be diagnosed if BBB are present

Hemiblocks



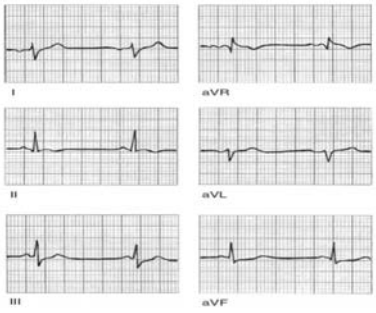
- Found on one of the Left bundle branch
- Left anterior or Left posterior
- Can affect the axis deviation
- Left ant. Fascicle lies superior and lateral to Left posterior fascicle
- QRS is not widen unlike BBB

Left anterior hemiblock



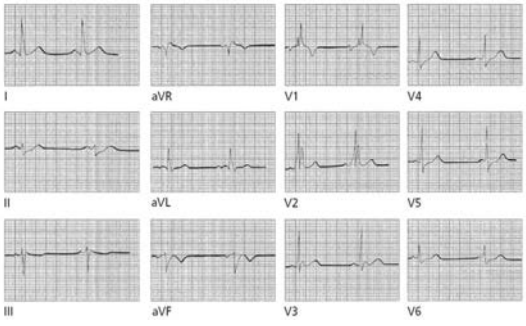
- The pathway down LAF is blocked and the current from LPF wraps around to assist
- Causing a Left axis deviation positive in lead I and negative in aVF
- Tall positive R waves are seen in left lateral leads from this hemiblock

Left Posterior Hemiblock



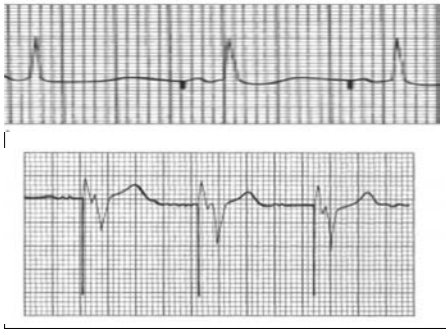
- Reverse from LAF
- The pathway down LPF is blocked and the current from LAF wraps around to assist
- Causing a Right axis deviation positive in aVF and negative in lead I
- Tall positive R waves are seen in inferior leads and deep S wave in Lateral leads

Bifascicular blocks



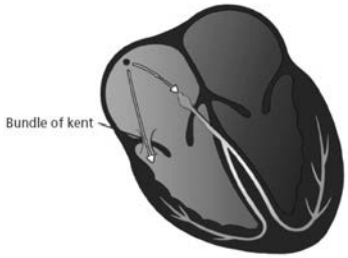
- RBBB and a hemiblock either LAF or LPF
- With a RBBB there will be a QRS >0.12 seconds, RSR' in V1, V2
  - If LAF will have Left axis deviation
  - If LPF will have Right axis deviation

Pacemakers on EKGs



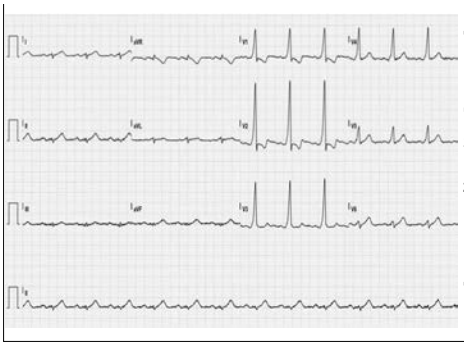
1. Atrial Pacemaker
   - Pacer spike followed by a P wave then normal PR interval and QRS
2. Ventricular Pacemaker
   - Bizarre and wide QRS similar to a PVC
   - May see P wave
3. Dual Chamber
   - 2 spikes one before a P wave and one before a wide bizarre QRS

Preexcitation Syndromes



- There is usually a 0.1 second pause at the AV node
- in Preexcitation there is a accessory pathway that bypasses the AV node to reach the ventricles with no delay
  - In other words a "short cut"

Wolff-Parkinson-White

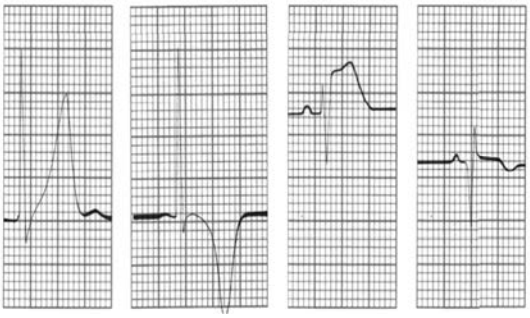


- This short cut pathway is a discreet conduction pathway that connect both atria and ventricles it can be Left atrium to left ventricle or right sided
- Criteria:

1. PR interval being shortened <0.12 seconds
2. QRS complex to widen >0.10 seconds including a "delta wave"
   - Wide because premature activation adds a upstroke wave to the normal QRS increasing its width overall

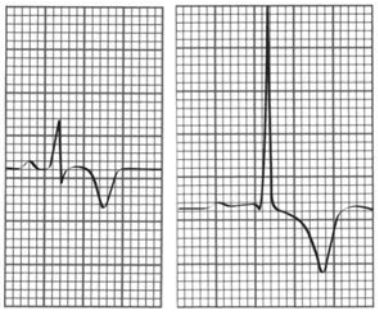
- Can occur with A. Fib and SVT

Myocardia Ischemia and Infarction



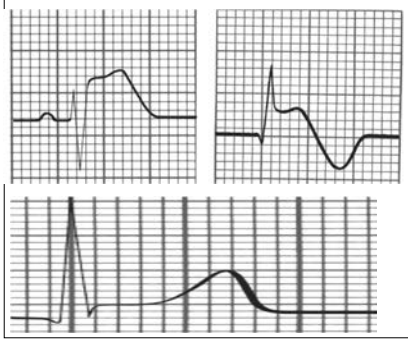
- 3 general EKG changes noted on a ST elevated Myocardial Infarction (STEMI)
  1. T waves peak then invert
  2. ST segment elevation
  3. Q waves appearing

Hyperacute T waves or Inverted T waves



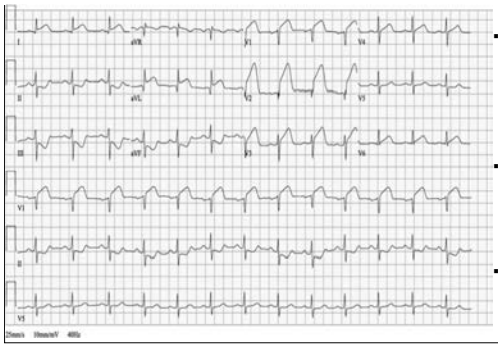
- Reflection of ischemia = lack of blood flow, initially peak then several hours later invert
- They can switch back to normal in cases of ischemia, in cases of infarct they stay inverted for month to years
- TWI can been seen with BBB and hyperventilation
- In cases of ischemia are usually symmetrical
- In cases with prior known TWI they can revert back to normal in ischemia or new infarct
- Normal in some athletes isolated to V1, V2, V3 or lead III

ST segment



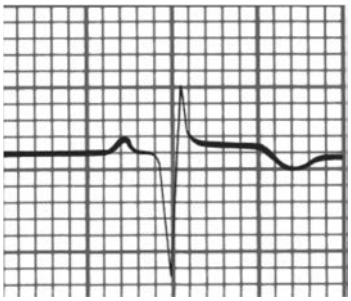
- 2nd change that happens acutely in a STEMI
- Significant amount of injury if this is present
- Use the TP segment to reference the ST segment
- Return to baseline in several hours, persistent can be from a ventricular aneurysm
- Junction point elevation common in healthy young folks in V1, V2, V3 has a small notch or slur downsloping of the R wave
- True ischemia ST is bowed upward

ST Elevation Criteria



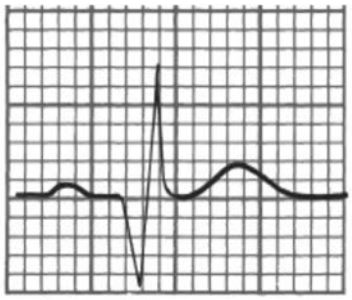
- ST elevation In V2 V3
  - Men <40 yo; >2.5 mm increase
  - Men >40 yo; >2.0 mm increase
  - Women >1.5 mm increase
- ST elevation in other leads
  - Men <40 yo; >1 mm increase
  - Men >40 yo; >1 mm increase
  - Women >1 mm increase
- ST elevation must be in at least 2 leads

Q Waves



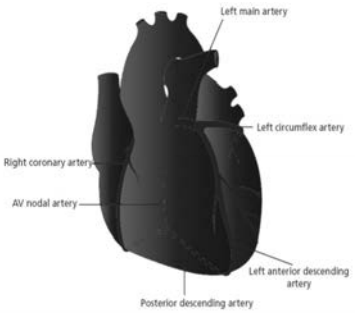
- New Q waves indicate irreversible damage
- Diagnostic for an MI
- Can be seen several hours after a STEMI, usually after ST elevation has gone down
- Can have them for life
- When a area of myocardium is permanently damaged that area will have a negative deflection creating the Q wave
- Leads distant from the infarcted tissue can have ST segment depression
- Ex. Lead III should be positive

Q wave criteria



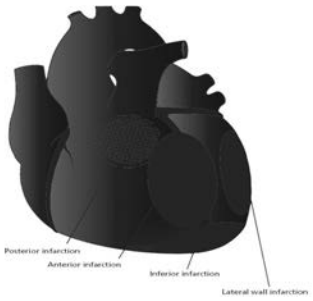
- Ischemia Q Waves are wide and deep
  1. Must have >0.04 seconds
  2. Depth must be 25% of the height of the R wave of the same QRS
- Normal Q waves
  1. Small in lateral I, aVL, V5, V6
  2. Q wave in only V3 no other leads

Locating the infarct



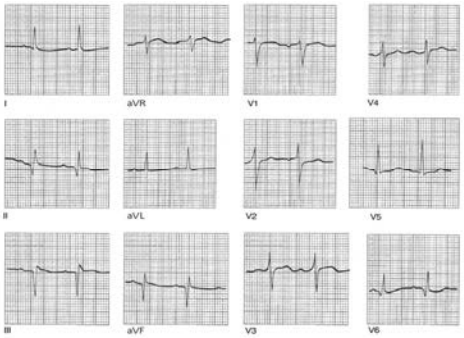
- Right coronary artery passes between the Right atrium and Right ventricle then moves to the posterior surface of the heart
  - Descending branch feeds the AV node
- Left main artery splits into the Left anterior descending artery and left circumflex artery
  - LAD goes between the 2 ventricles and feeds the anterior wall of the heart and most of the Interventricular septum
- Circumflex artery goes between the Left atrium and Left ventricle and lateral wall of the left ventricle

Locations of Infarcts



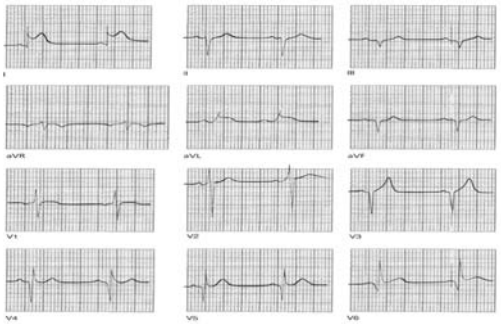
1. Inferior infarct: diaphragm surface of the heart caused by occlusion of RCA or descending branch
   - Inferior leads: II, III, aVF
   - Reciprocal in Anterior lateral leads
2. Lateral infarct: left lateral wall of heart, occlusion of LCA
   - Left lateral leads: I, aVL, V5, V6
   - Reciprocal in inferior leads
3. Anterior infarct: anterior surface of the Left Ventricle, occlusion of the LAD
   - Any precordial lead can change V1-V6
   - If occlusion is of Left main artery can cause a anterolateral infarct with precordial and lead I and aVL
   - Reciprocal is inferior leads
4. Posterior infarct: posterior surface of the heart, occlusion of RCA
   - usually occurs with inferior or lateral infarcts
   - to diagnosis needs to see in reciprocal changes in anterior leads with tall R wave and ST depression in leads V1-V3
   - Mirrors image of a anterior infarct

Inferior MI



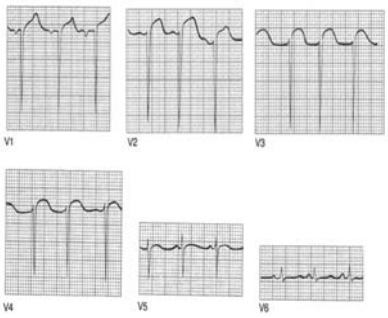
- Inferior infarct: diaphragm surface of the heart caused by occlusion of RCA or descending branch
  - Inferior leads: II, III, aVF
  - Reciprocal in Anterior lateral leads

Lateral MI



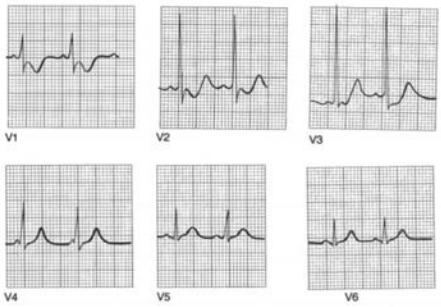
- Lateral infarct: left lateral wall of heart, occlusion of LCA
  - Left lateral leads: I, aVL, V5, V6
  - Reciprocal in inferior leads

Anterior MI



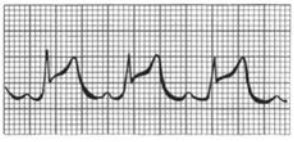
- Anterior infarct: anterior surface of the Left Ventricle, occlusion of the LAD
  - Any precordial lead can change V1-V6
  - If occlusion is of Left main artery can cause a anterolateral infarct with precordial and lead I and aVL
  - Poor R wave progression
  - Reciprocal is inferior leads

Posterior MI



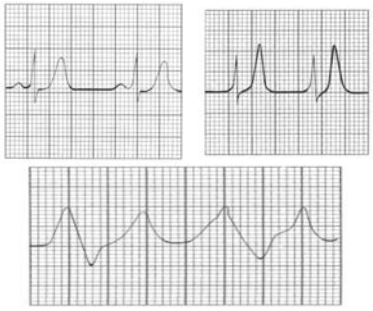
- Posterior infarct: posterior surface of the heart, occlusion of RCA
  - usually occurs with inferior or lateral infarcts
  - to diagnosis needs to see in reciprocal changes in anterior leads with tall R wave and ST depression in leads V1-V3
  - Mirrors image of a anterior infarct

Misc. MI related info



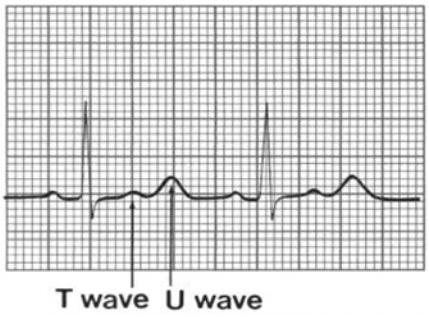
- Right Ventricle MI
  - Inferior MI with V1 changes
  - Preload sensitive caution with nitrate can cause severe hypotension
- New LBBB
  - Treat as a new MI, remember ischemia thought to play a role in LBBB
- Prinzmetal Angina
  - Coronary vasospasms
  - ST elevation quickly reversible with nitroglycerin

Electrolyte Disturbances

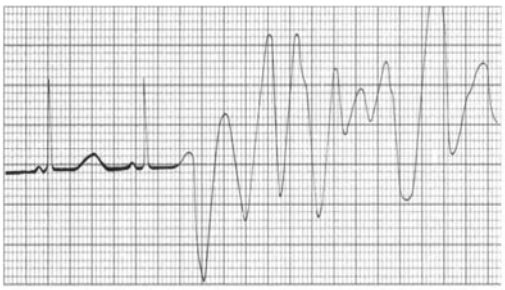


- Hyperkalemia
  - Increase in Potassium initially causes a increase in T wave height nearly all leads distinguishing from Mis
  - If potassium continues to increase it will prolong the PR interval and flatten the P waves
  - The QRS will widen and merge with the T waves if potassium worsens creating a sine wave pattern
    - Hyperkalemia distinguished from other wide QRS with right axis deviation
  - Risk of Ventricular fibrillation

Hypokalemia

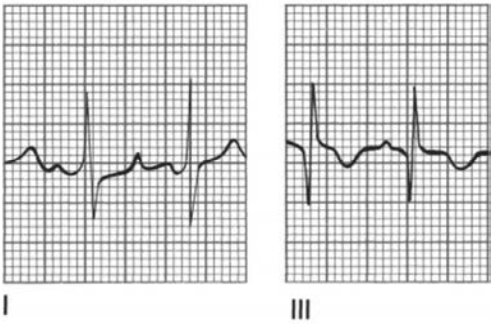


- Can cause
  - ST depression
  - Flatten the T waves and prolonged QT interval
  - Appearance of U waves, seen after the T wave
  - Severe hypokalemia can lead to ST elevation, SVT, and V Tach



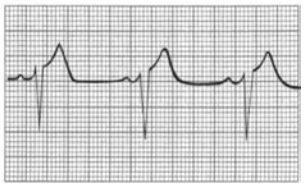
- Hypocalcemia
  - Prolongs the QT interval
- Hypercalcemia
  - Shortens the QT interval
- Torsade de Pointes
  - Prolonged QT interval

Acute PE

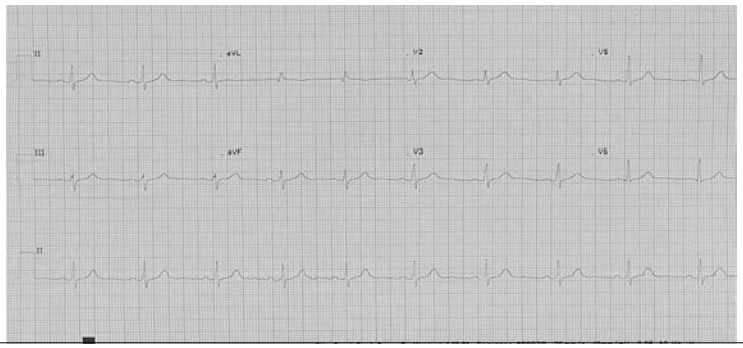


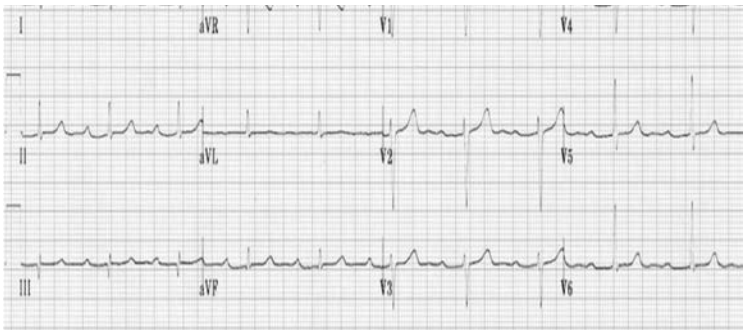
- Massive PE can cause
  - RVH due to a dilated ventricle
  - RBBB
  - Large S waves in lead I and deep Q waves in lead III called the S1Q3 pattern
  - TWI in V1, V2
  - Seen with A. Fib and Sinus tachycardia

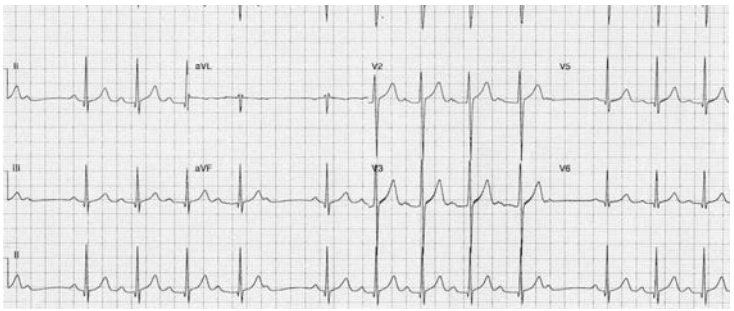
Acute Pericarditis

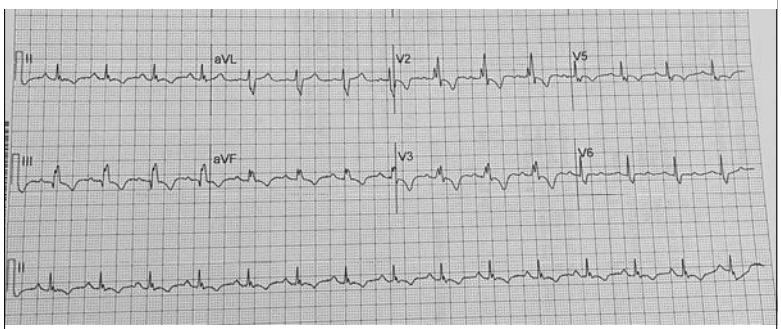


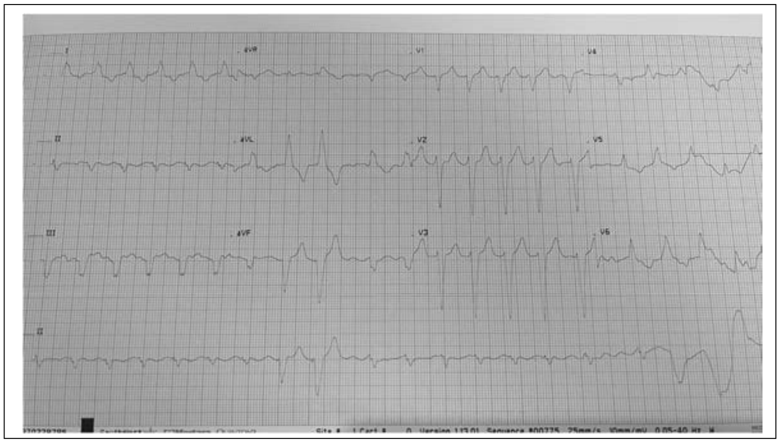
- ST elevation and flat T waves or TWI seen throughout all leads
- STs are upward concave (saddle shaped)
- No Q waves seen
- If effusion will cause
  - Decreased voltage on EKG
  - Electrical alternans
    - Change in electrical axis







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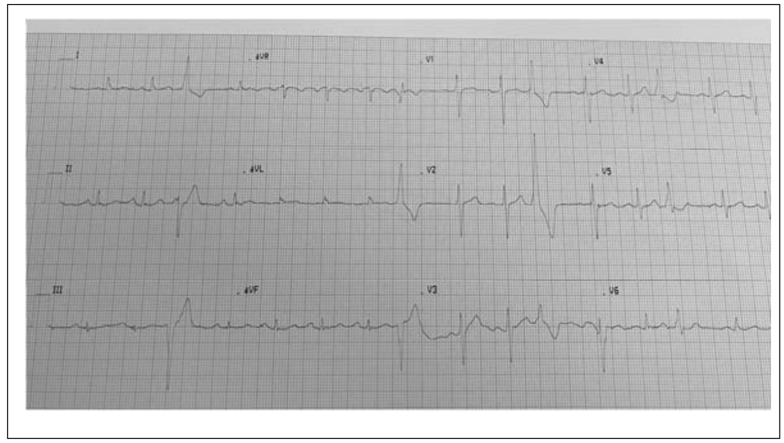
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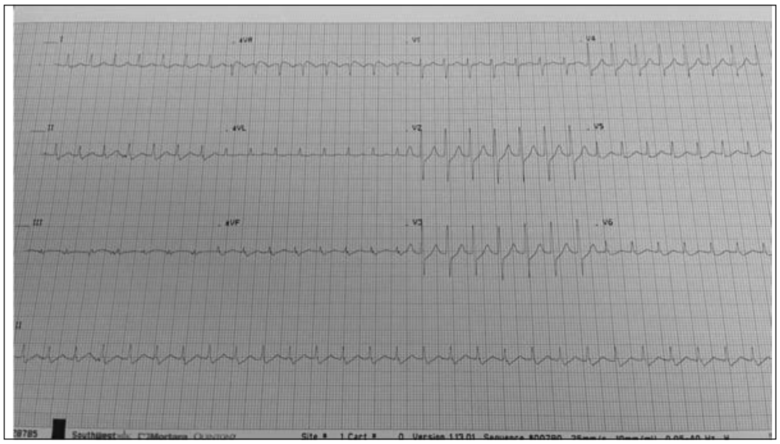
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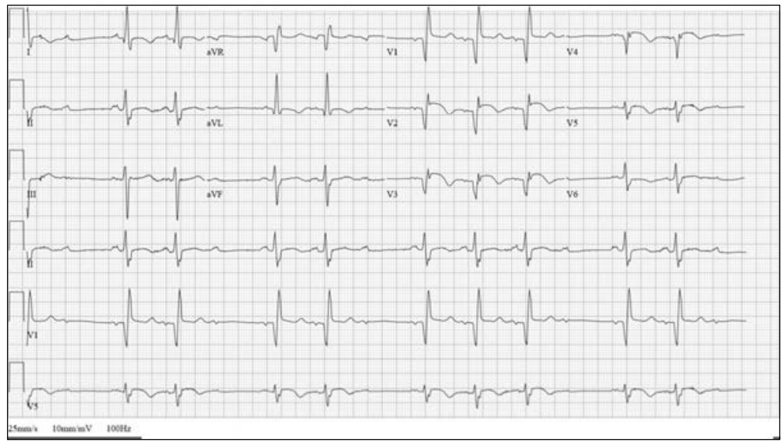
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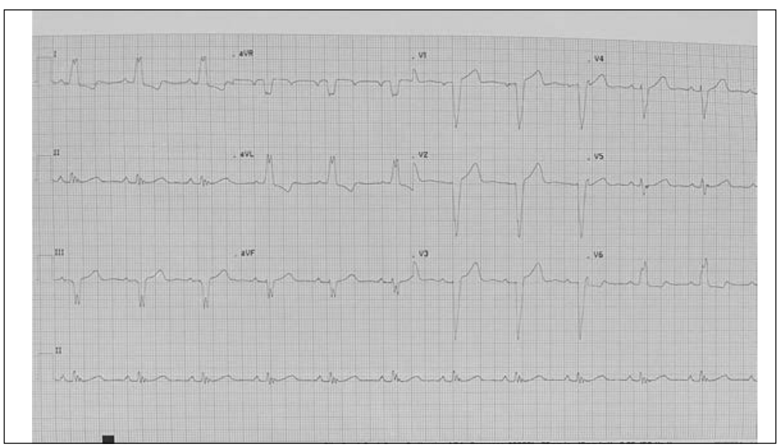
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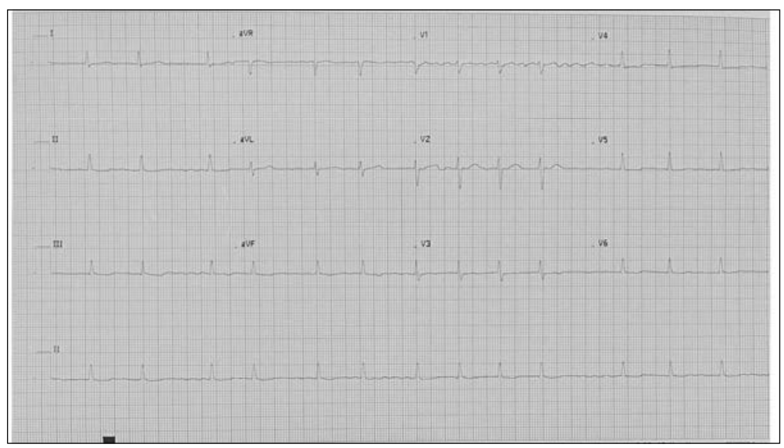
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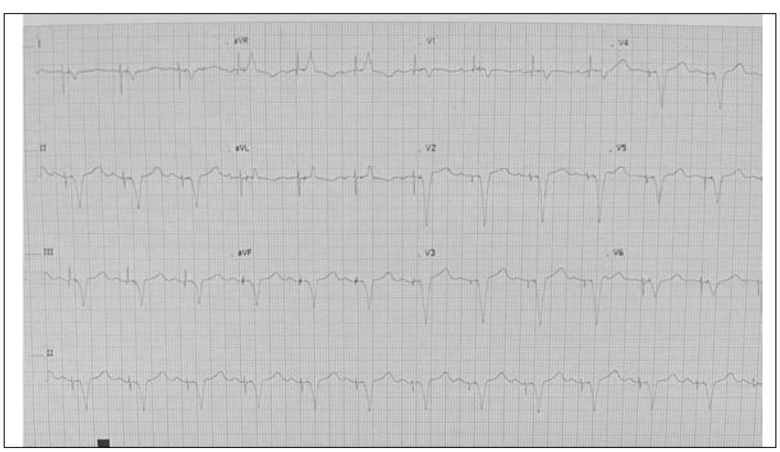
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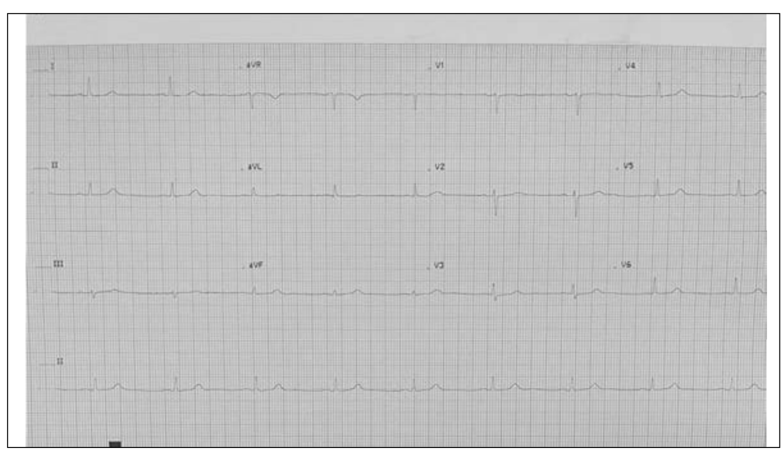
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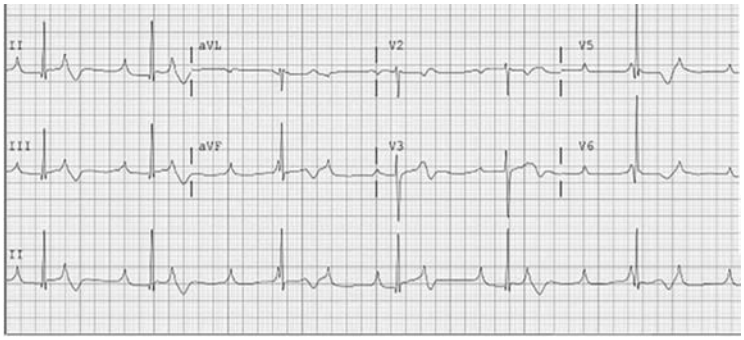

Citations

- Thaler, Malcolm. The Only EKG Book You'll Ever Need. Wolters Kluwer Health, 2017. [Wolters Kluwer].
- Loscalzo, Joseph. Harrison's Cardiovascular Medicine. McGrawHill Medical, 2010.

- Contact info: John.Villasenor@optum.com