Advanced 12 Lead ECG Interpretation

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Opening Thought

We have a hunger of the mind which asks for knowledge of all around us, and the more we gain, the more is our desire; the more we see, the more we are capable of seeing.

Maria Mitchell

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Advanced Concepts in 12 Lead ECG Interpretation Part 1

Prerequisite Review
Hemiblocks
Chamber Enlargement
Electrolyte and Drug Effects

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Key Concepts You Should Know

- Definitions of Q, R, S waves
- Anatomy of a 12 Lead ECG
  - Which leads look where
  - How leads record
- QRS Axis Calculation
- Right and Left Bundle Branch Block

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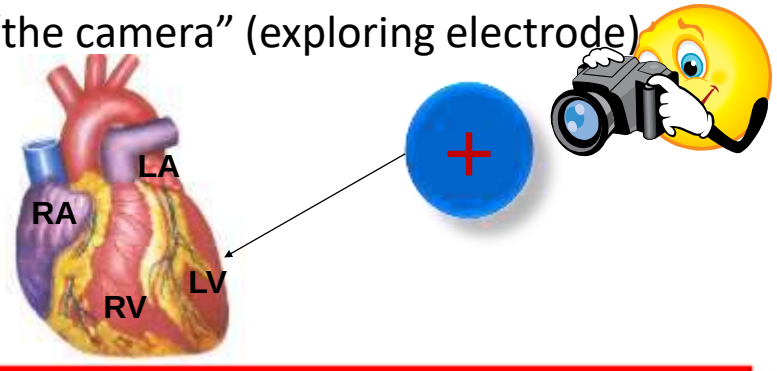
Importance of the Positive Electrode

- Consider the positive pole of each lead as "the camera" (exploring electrode)



Positive pole is where the camera is located. Negative pole tells the camera which way to look.

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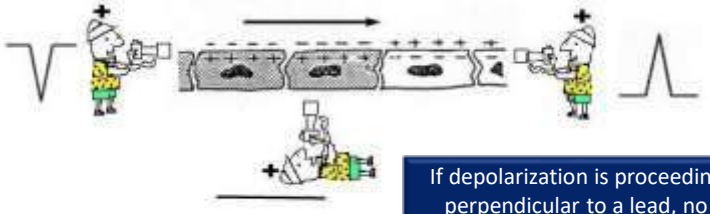
How Leads Record

- Positive electrode is the recording electrode or “camera lens”
- Negative electrode or reference point tells camera which way to shoot
- If positive electrode sees depolarization approaching it, it records an upright complex
- If positive electrode sees depolarization heading away from it, it records a negative complex.



If depolarization is proceeding perpendicular to a lead, no deflection is recorded

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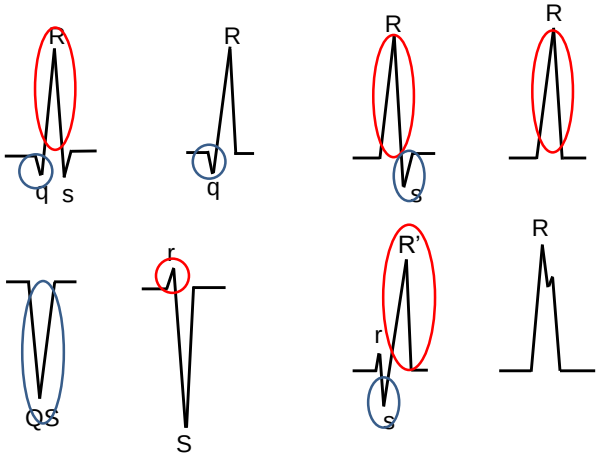
QRS Complexes

- Q wave
  - Initial negative deflection from baseline
- R wave
  - Always an upright deflection
  - If 2 are present, second one is R’
- S wave
  - Negative deflection following an R wave



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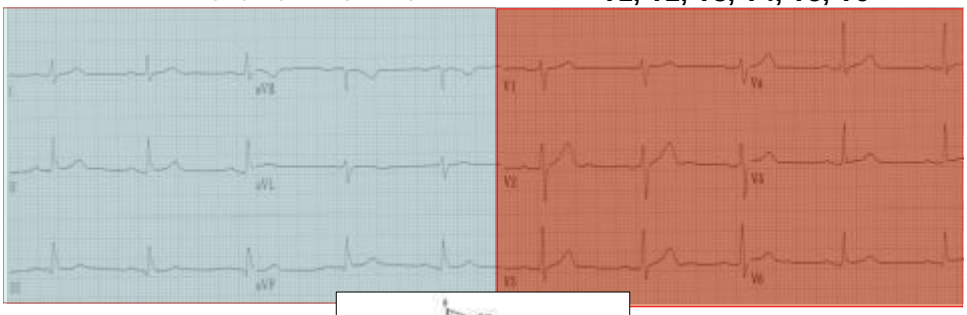
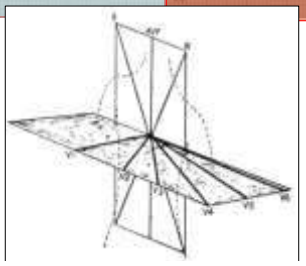
Anatomy of a 12 Lead ECG

Limb Leads: I, II, III, aVR, aVL, aVF

Precordial (Chest) Leads: V1, V2, V3, V4, V5, V6



Frontal plane leads: up/down, right/left



Horizontal plane leads: anterior/posterior, right/left

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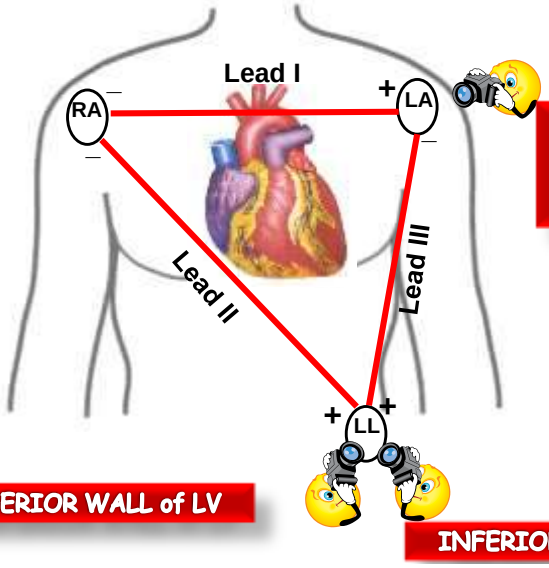
Standard Limb Leads (Dual Electrode Leads)
Leads I, II, III

Lead 1
RA – Left Arm +
High Lateral Wall LV

Lead 2
RA – Left leg +
Inferior Wall LV

Lead 3
LA – Left leg +
Inferior Wall LV



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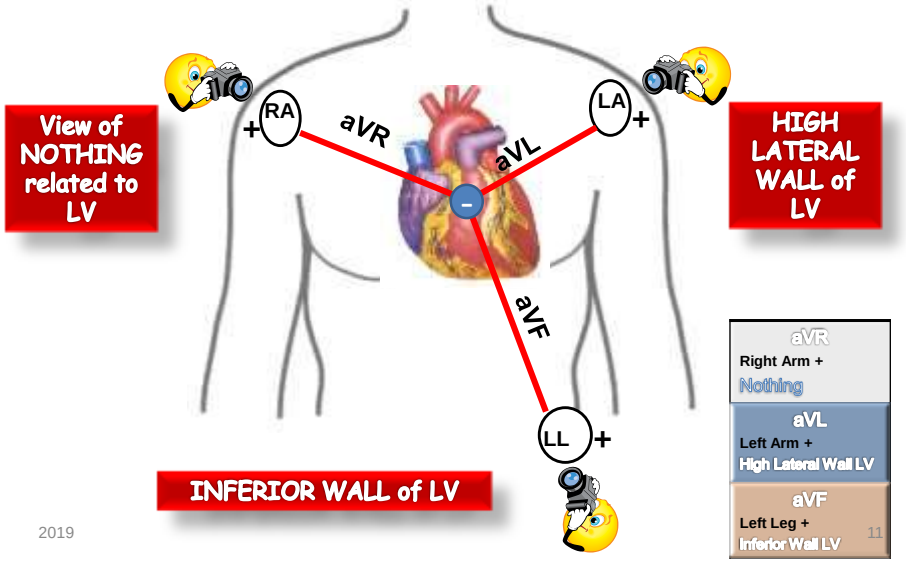
Augmented Limb Leads (Single Electrode Leads)
aVR, aVL, aVF



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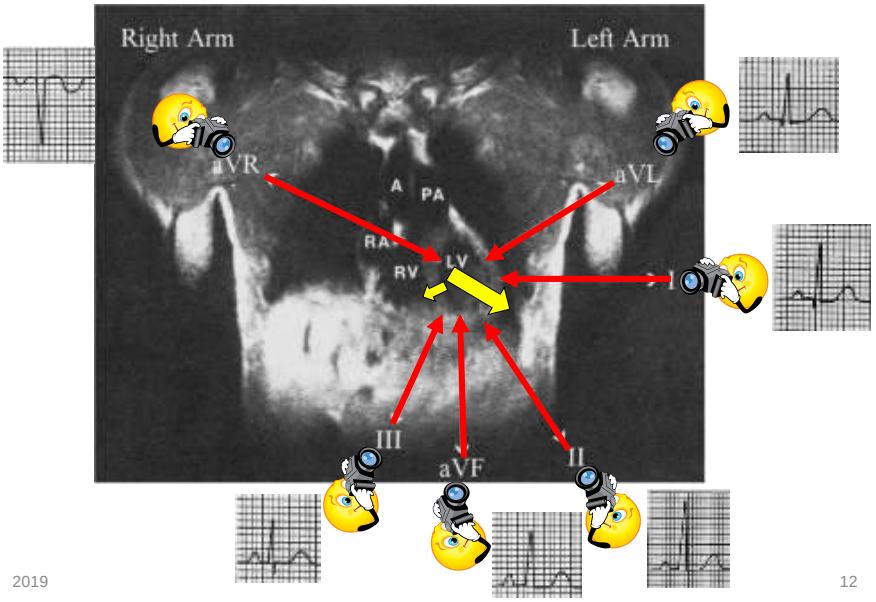
6 Limb Leads



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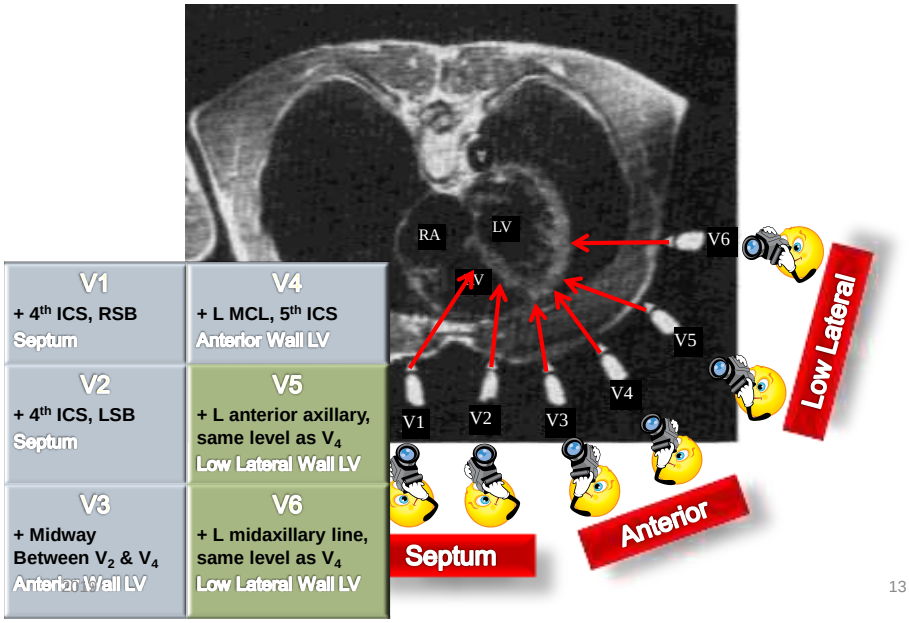
6 Precordial (Chest) Leads



V1 + 4th ICS, RSB Septum	**V4** + L MCL, 5th ICS Anterior Wall LV
V2 + 4th ICS, LSB Septum	**V5** + L anterior axillary, same level as V_4 Low Lateral Wall LV
V3 + Midway Between V_2 & V_4 Anterior Wall LV	**V6** + L midaxillary line, same level as V_4 Low Lateral Wall LV

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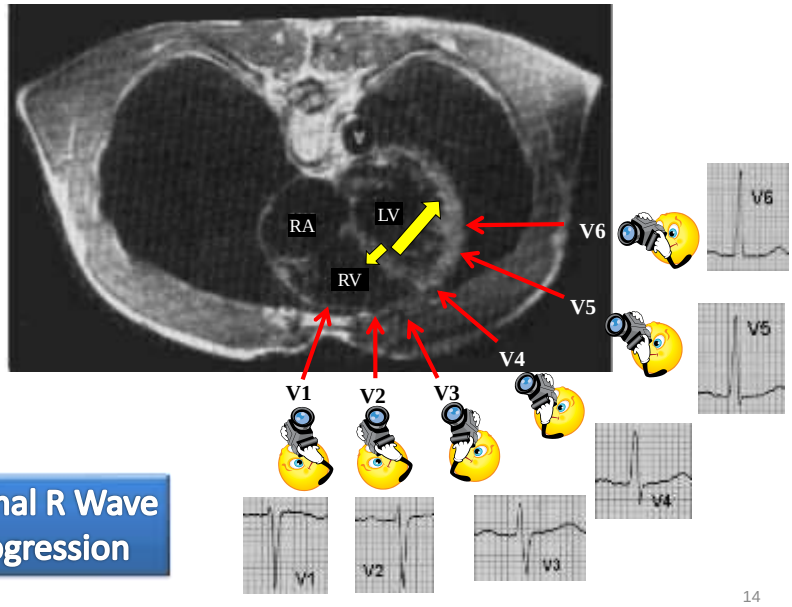
6 Precordial (Chest) Leads



Normal R Wave Progression

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Lead 1	aVR	V1	V4
+ Left Arm High Lateral Wall	+ Right Arm	+ 4th ICS, RSB Septal Wall	+ L MCL, 5th ICS Anterior Wall
Lead 2	**aVL**	**V2**	**V5**
+ Left Leg Inferior Wall	+ Left Arm High Lateral Wall	+ 4th ICS, LSB Septal Wall	+ L anterior axillary, same level as V_4 Low Lateral Wall
Lead 3	**aVF**	**V3**	**V6**
+ Left Leg Inferior Wall	+ Left Leg Inferior Wall	+ Midway Between V_2 & V_4 Anterior Wall	+ L midaxillary line, same level as V_4 Low Lateral Wall

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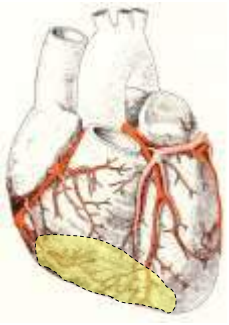
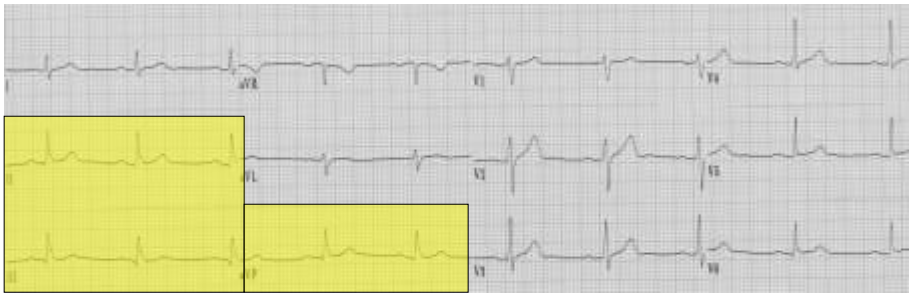
Inferior Leads





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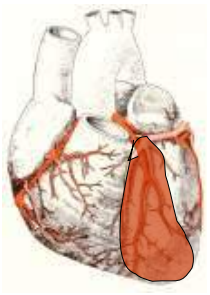
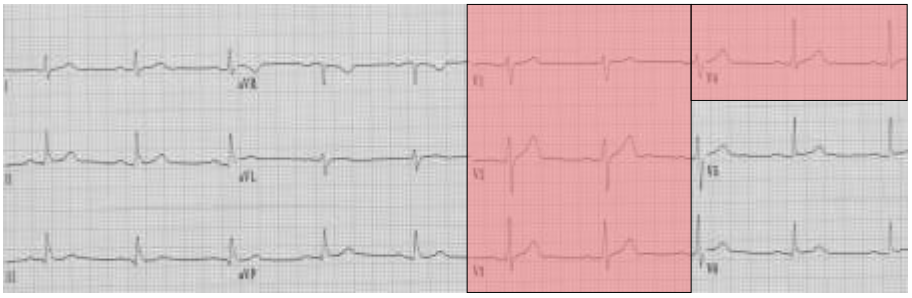
Anterior Leads





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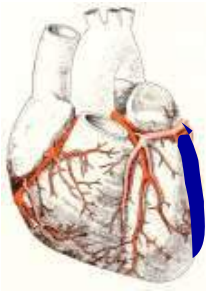
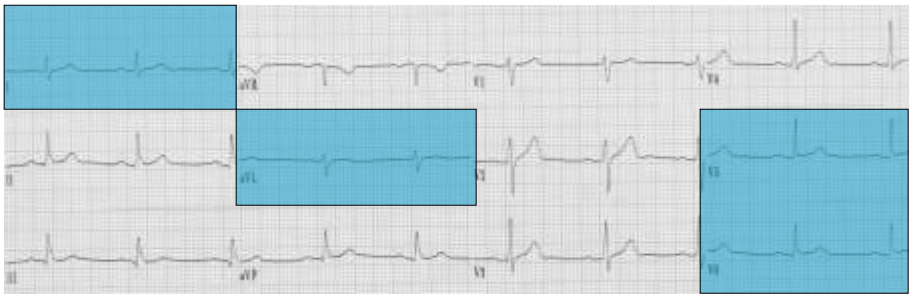
Lateral Leads





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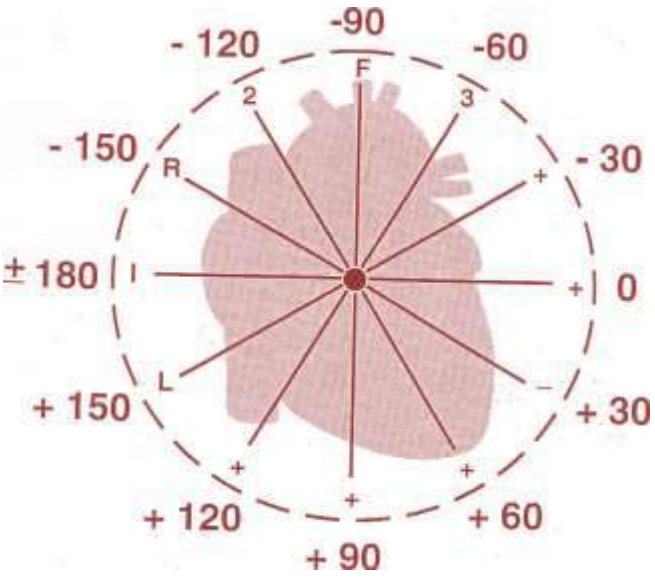
Cardiac Axis



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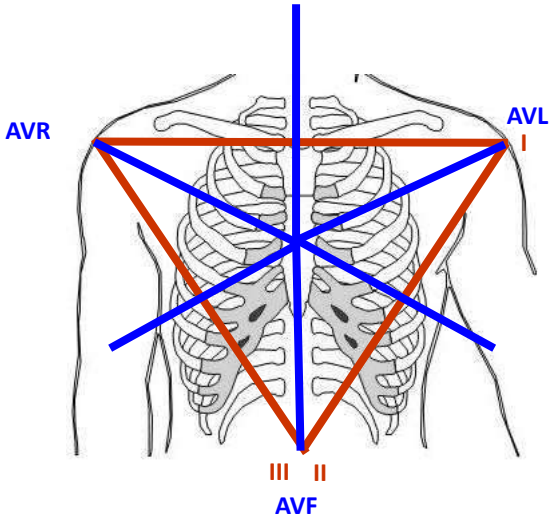

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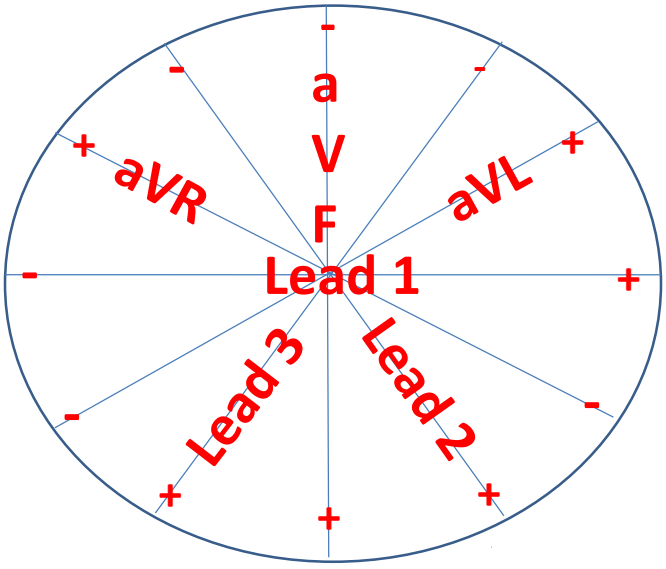
-
a
V
F
-
aVR
+
-
aVL
+
-
Lead 1
+
-
Lead 3
+
Lead 2
-
+
+

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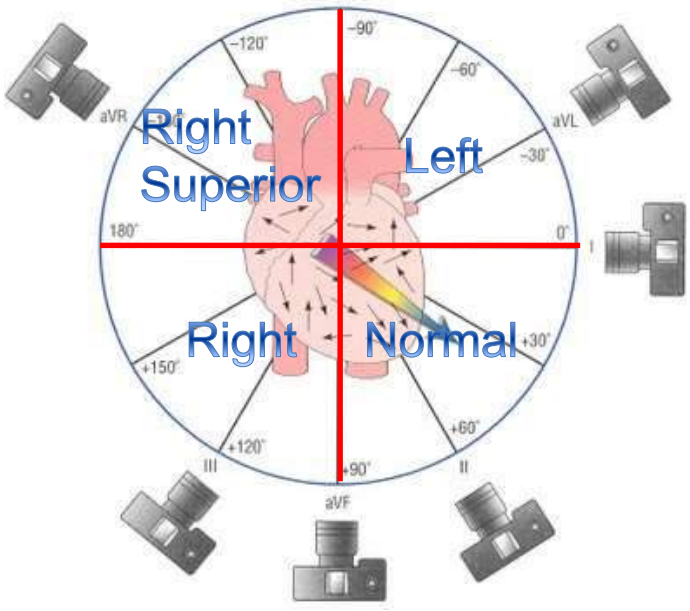
-90°
-120°
-60°
aVR
aVL
Right
Superior
Left
-30°
180°
0°
I
Right
Normal
+30°
+150°
+60°
+120°
III
+90°
II
aVF

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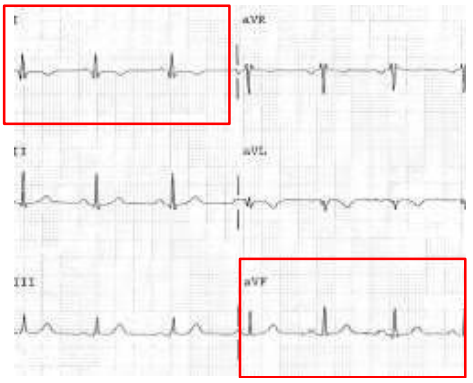
Quick Look Axis Quadrant Determination

- Evaluated QRS in Lead I and aVF
- Left hand QRS in Lead I
- Right hand represents QRS in aVF
- Hand will point in the same direction as the QRS complex



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Right Superior Axis

- Both hands down

Left Hand Lead I | Right Hand Lead aVF

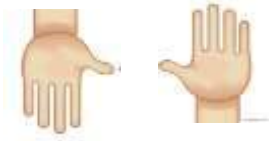
Left Axis

- **Left** hand up

Left Hand Lead I | Right Hand Lead aVF

Right Axis

- **Right** hand up



Left Hand Lead I | Right Hand Lead aVF

Normal Axis

- Both hands up

Left Hand Lead I | Right Hand Lead aVF

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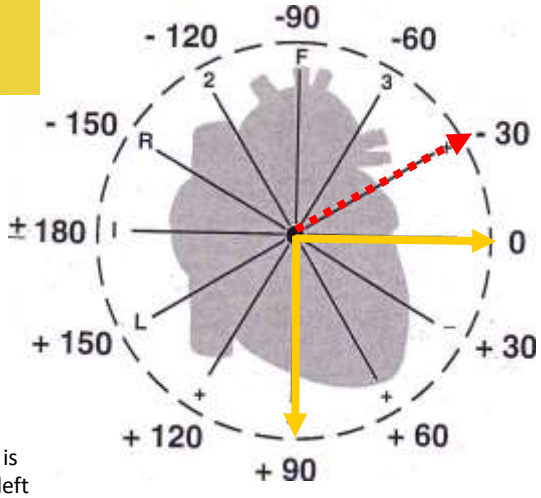
Axis Quadrants:

Adults:
-30◦ to
+90◦

Normal Axis



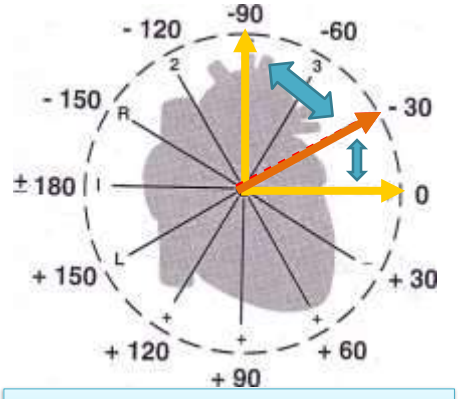
Note: Normal axis is extended into the left quadrant by 30 degrees.

Axis Quadrants:

Leftward Axis and Left Axis Deviation

Adults:
-30◦ to -45◦ Moderate

-45◦ to – 90◦ Marked



Clinical Pearl: The key is to look at lead II. If the QRS is upright in lead II leftward axis is within normal limits. If the QRS in lead II is not upright then left axis deviation is present.

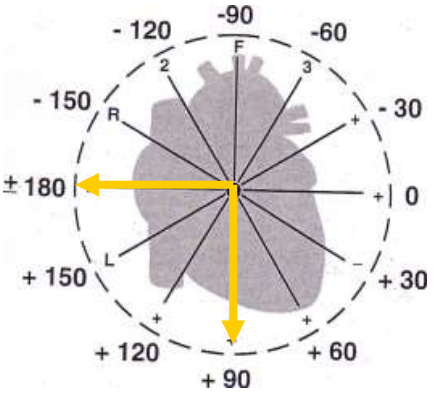
Causes:

- Aging (changes in left anterior fascicular conduction)
- Left ventricular hypertrophy
- Acute MI (peri infarction block)
- Congenital heart disease
- Hyperkalemia
- Marked: Associated with left anterior fasicular block

Axis Quadrants:

Right Axis Deviation

+90 to +120: Moderate
+120 to +180: Marked



Causes:

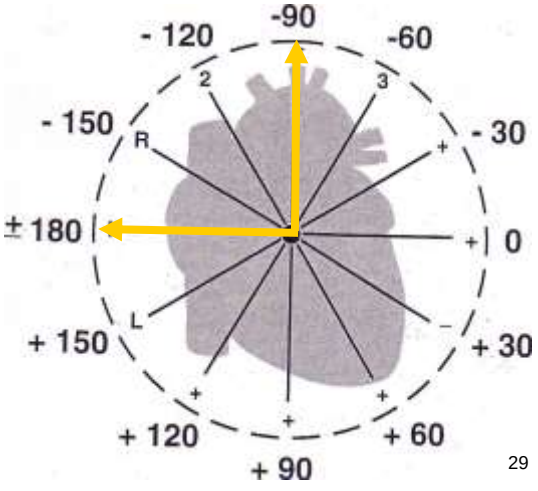
- Infants / Children
- RV Hypertrophy
- Chronic Lung Disease
- Pulmonary Hypertension
- **Pulmonary Embolus**
- Marked: Often associated with left posterior fascicular block

Axis Quadrants:

Extreme (Right Superior) Axis Deviation

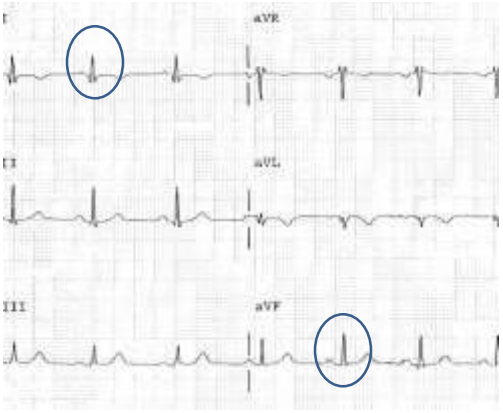
Causes:

- Ventricular Tachycardia
- Other significant conduction abnormalities

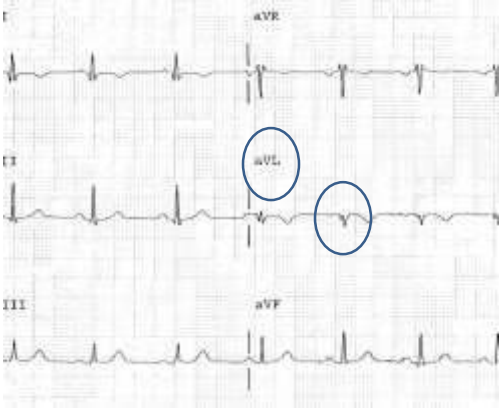


Calculating Degree of Axis

- Before you consider the degree of axis – always know the direction.
- Which quadrant do you expect?



Calculating Degree of Axis "*E*SPN" Method – Step 1

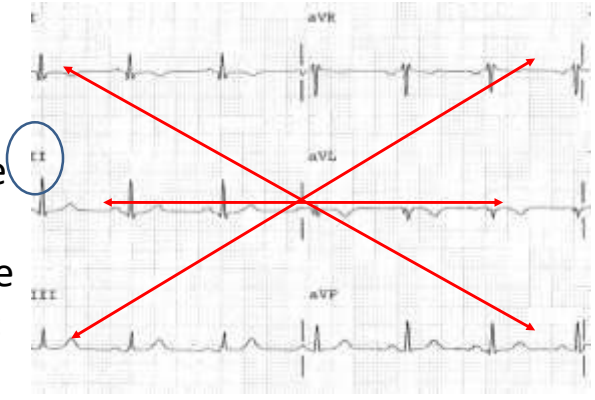


- **E=Equiphasic**
  - Find the QRS complex in the limb leads which is the most equiphasic

    OR
  - Find the smallest QRS complex (height of R wave minus depth of S wave)
- We are looking for the lead recording closest to baseline

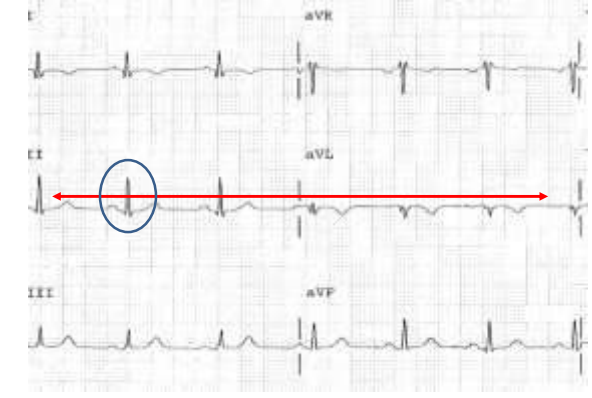
“ESPN” Method – Step 2

- **S=Sister Lead**
  - Utilizing the “Criss-Cross” method Find the “Sister” lead to the lead with the most equiphasic QRS complex



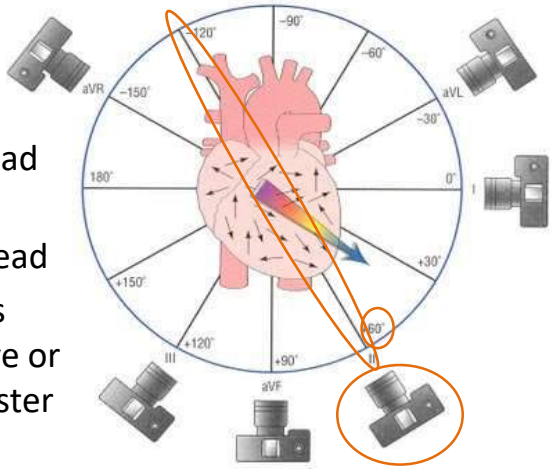
“ESPN” Method – Step 3

- **P/N = Positive or Negative**
  - Is the “Sister” lead positive or negative?



"ES*PN*" Method – Step 4

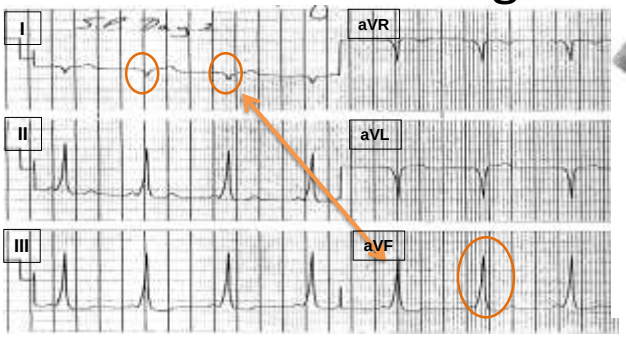
- Go to the AXIS Wheel
- If positive: locate the positive pole of that lead
- If negative: locate the negative pole of that lead
- What degree of AXIS is assigned to the positive or negative pole of the sister lead

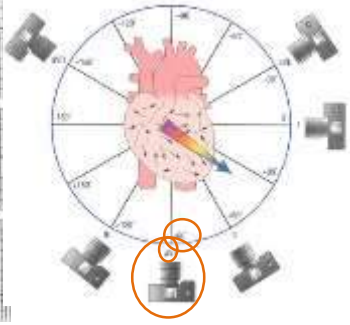


Axis Practice

Calculating Degrees

Calculate Degree of Axis





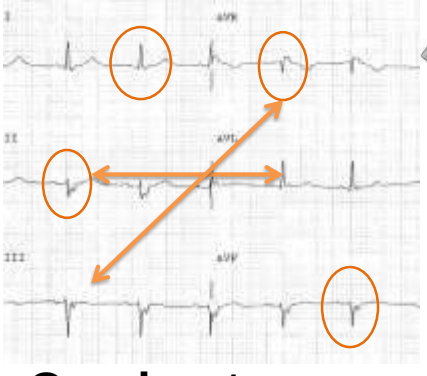
Quadrant: Right Hand Up – Right Axis

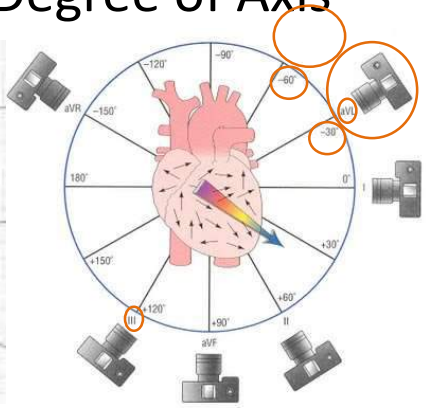
Equaphasic**:** Lead I

Sister**:** Lead aVF

P/N: Positive

Calculate Degree of Axis





Quadrant: Left Hand Up – Left Axis

E: Lead II

S: Lead aVL

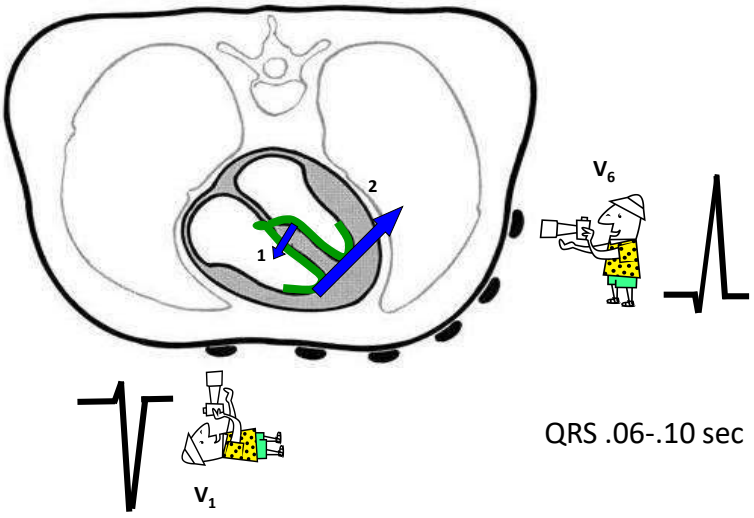
P/N: Positive

E: Lead aVR

S: Lead III

P/N: Negative

Normal Depolarization



QRS .06-.10 sec

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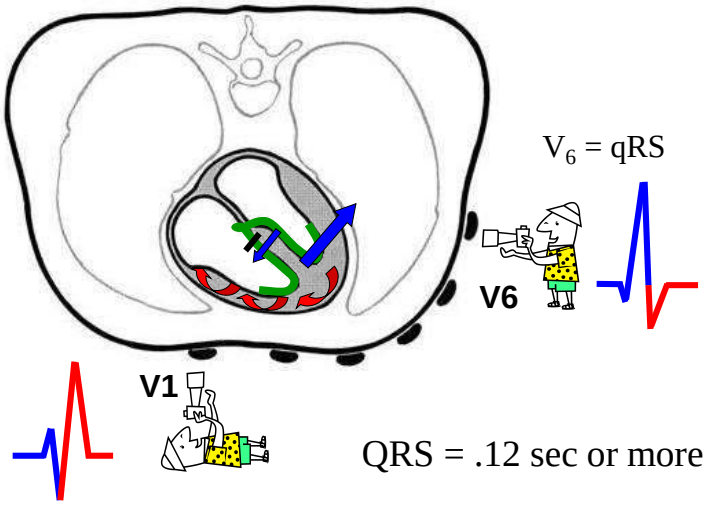
Right Bundle Branch Block



V_6 = qRS

V_1 = rsR'

QRS = .12 sec or more

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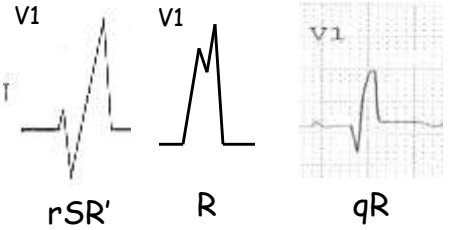
Right Bundle Branch Block

- V1
  - Triphasic complex rsR' (rsr', rSR') pattern
  - Or an M shaped R wave with right peak taller
  - Or a qR pattern (In patients with septal infarct)
- V6
  - Triphasic complex
  - Large R is maintained
  - qRs with wide S waves

  Note: Also a wide S wave in lead 1



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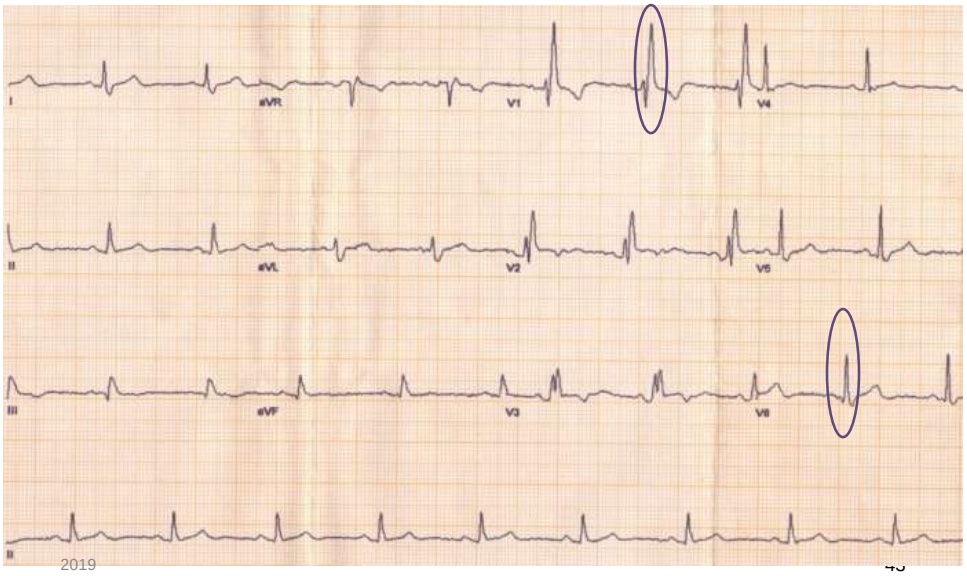

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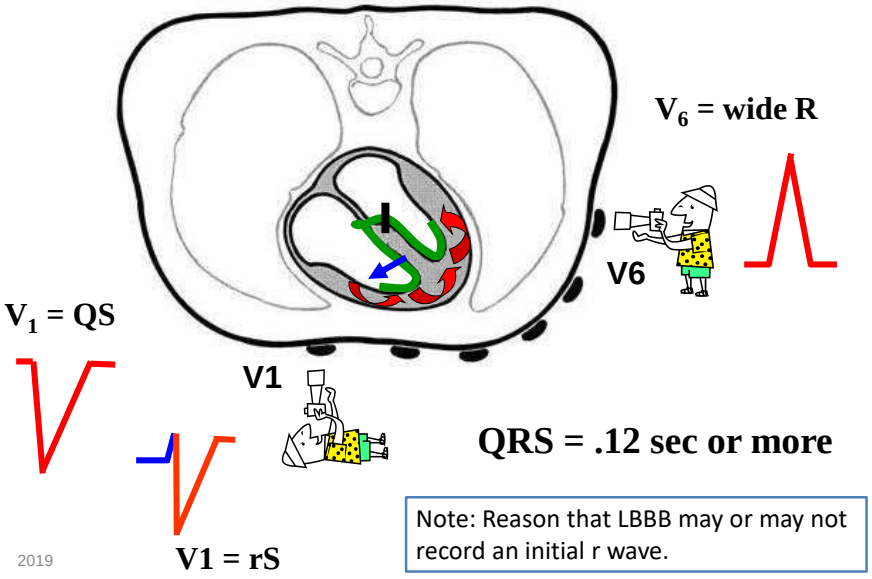
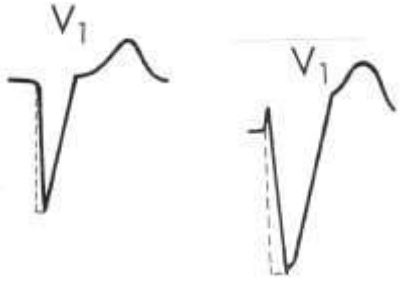
Left Bundle Branch Block

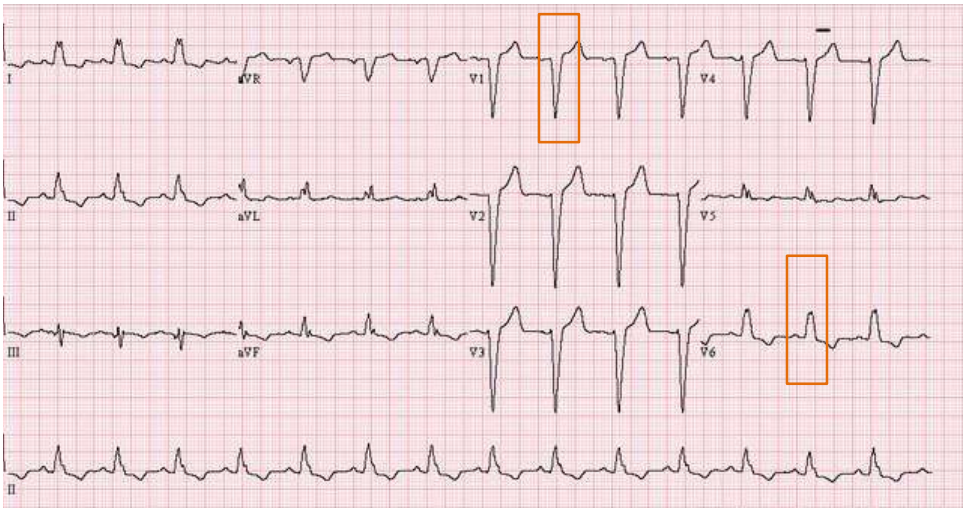


Note: Reason that LBBB may or may not record an initial r wave.

Left Bundle Branch Block

- V1
  - Wide QS or rS complex - negative
  - Slick downstroke
  - Time to nadir <0.06 sec
- V6
  - Large R wave is maintained
  - Wide R wave with no initial septal q wave





Additional diagnostic criteria include:
Broad slurred or notched R waves in leads 1, aVL, V5, and V6.

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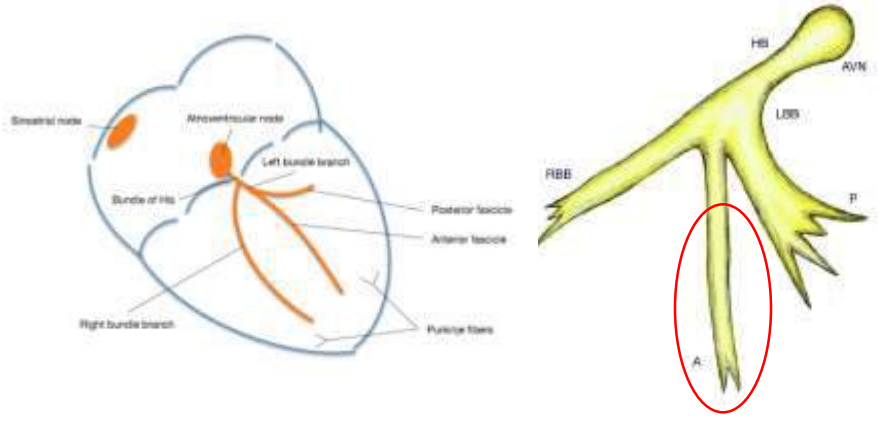
Left Anterior Hemiblock (LAHB)

Hemiblock and Fascicular Block are synonymous



Anterior fascicle is vulnerable: All blood supply from septal perforator of left anterior descending artery.

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Left Anterior Hemiblock

Causes

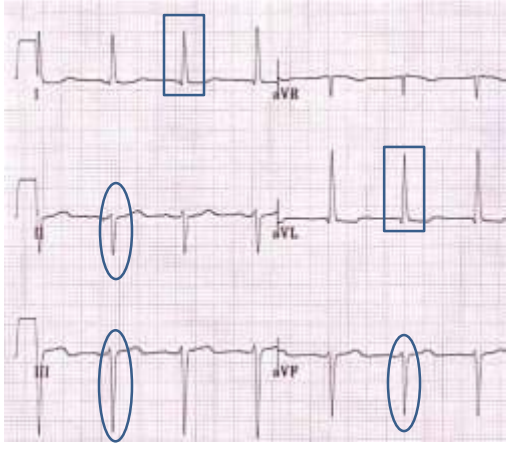
- Disease of left ventricular outflow tract
- Ischemia / injury of anterior septum or anterior lateral wall
- HTN
- Coronary artery disease
- Aortic valve disease
- Cardiomyopathy
- Closure of septal defect
- Surgical complication
- Lev and Lenègre diseases

Implications

- May be incidental finding in a healthy person
- Not benign in a hospitalized patient
  - Associated with increased mortality and morbidity in patients with coronary artery disease or myocardial infarction
  - In association with RBBB: very high risk
- May conceal the ECG signs of myocardial infarction and left ventricular hypertrophy

Left Anterior Hemiblock Recognition

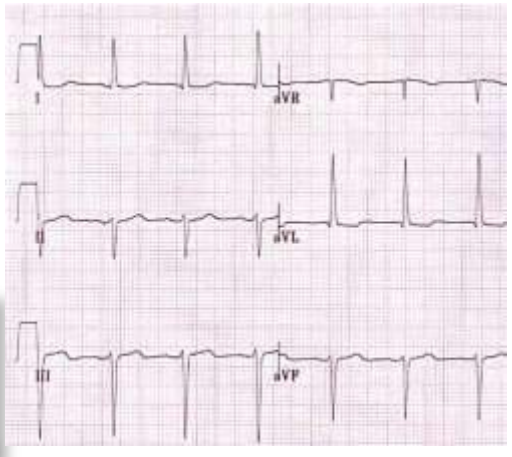
- **Lead 2, Lead 3 and aVF**
  - **rS pattern**
  - **Small r waves**
  - **Slightly wide / deep S waves**
  - **Increased limb lead voltage**
- Lead 1 and aVL
  - qR pattern



Note: Diagnostic criteria is qR in aVL with a R peak time of ≥ 45 msec

Left Anterior Hemiblock: Additional Diagnostic Criteria

- Normal QRS duration
- Left axis deviation
  - - 45 ° to -90 °
  - Common at – 60 °



Clinical Pearl:

Key for recognizing -60° Axis

aVR is most equiphasic limb lead in presence of leftward axis

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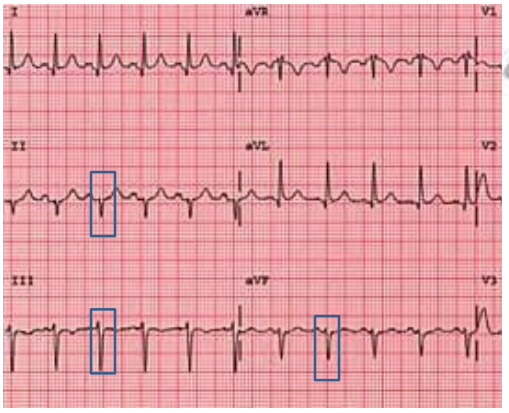

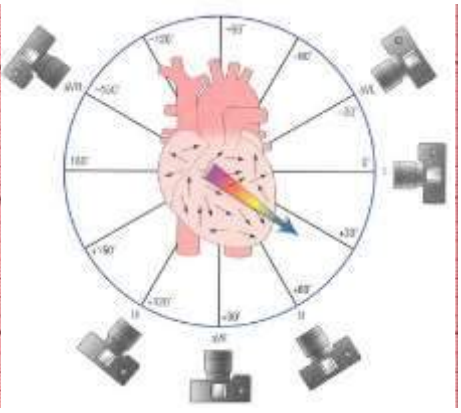

Handy method for axis tells direction of depolarization

ESPN method: II and aVR are most equiphasic

Left

Degree of deviation about -45

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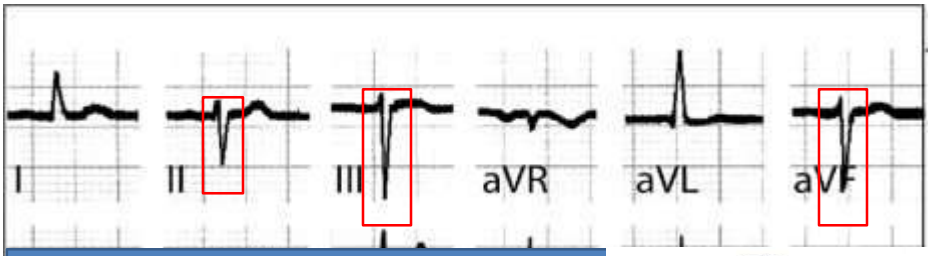
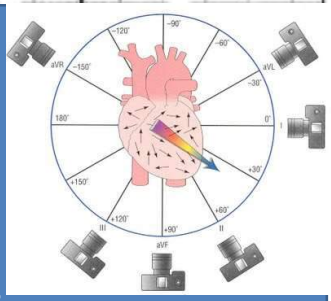


Determine Quadrant of axis: Left
ESPN Method

E: aVR
S: Lead III
P/N Negative

Negative pole of Lead III: -60 degrees



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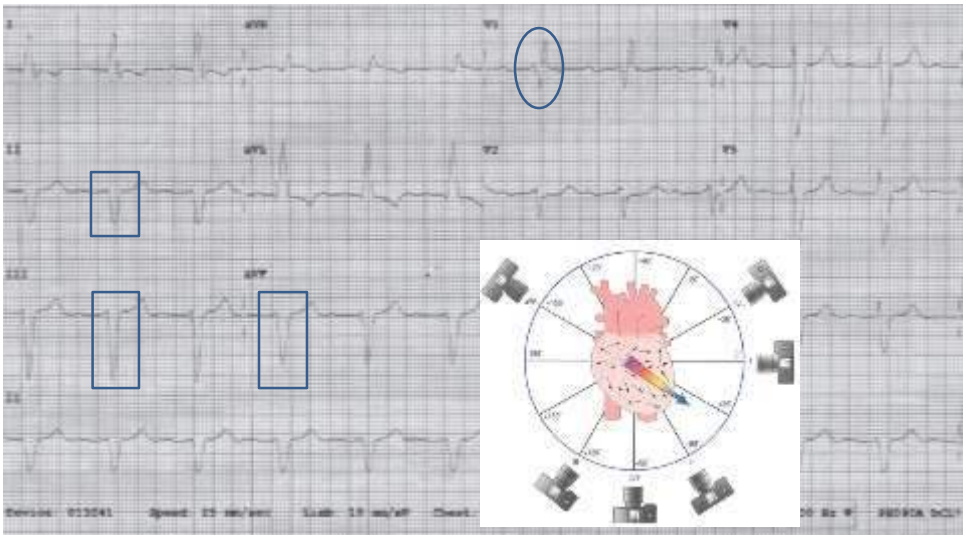

Note lead aVR is most equiphasic in presence of leftward axis.

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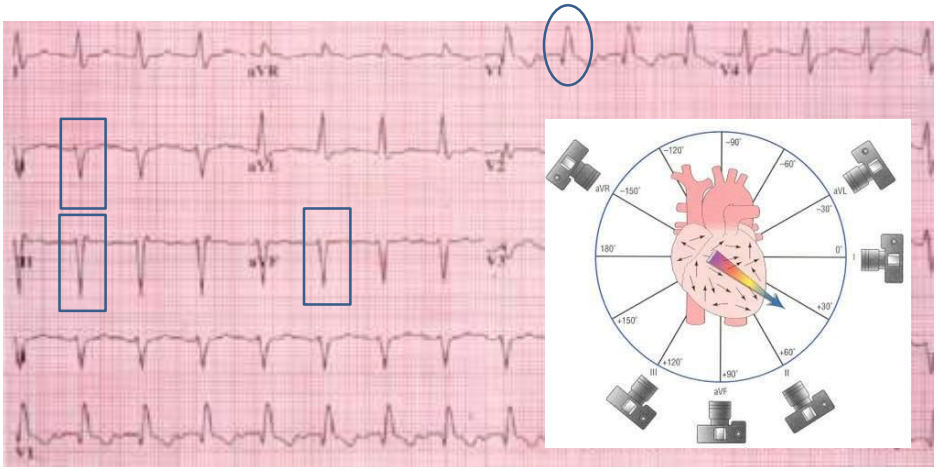
Also note the 1st degree AVB.
This used to be called trifascicular block.



Note lead aVR is the most equiphasic in presence of leftward axis.

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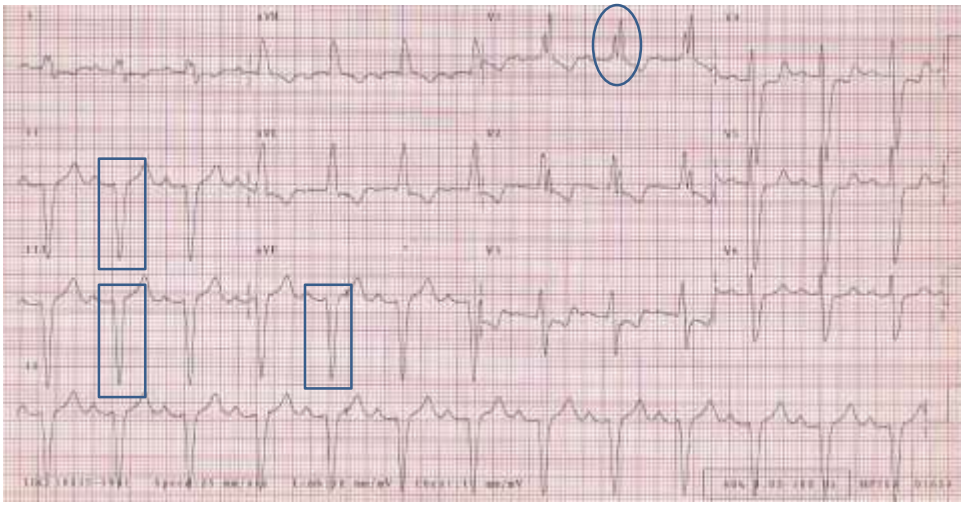

INTERESTING: Lead 1 is most equiphasic in presence of leftward axis.

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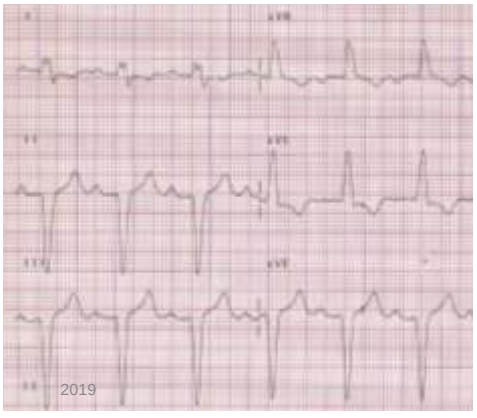
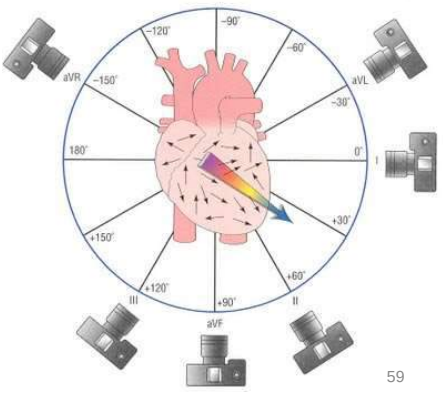
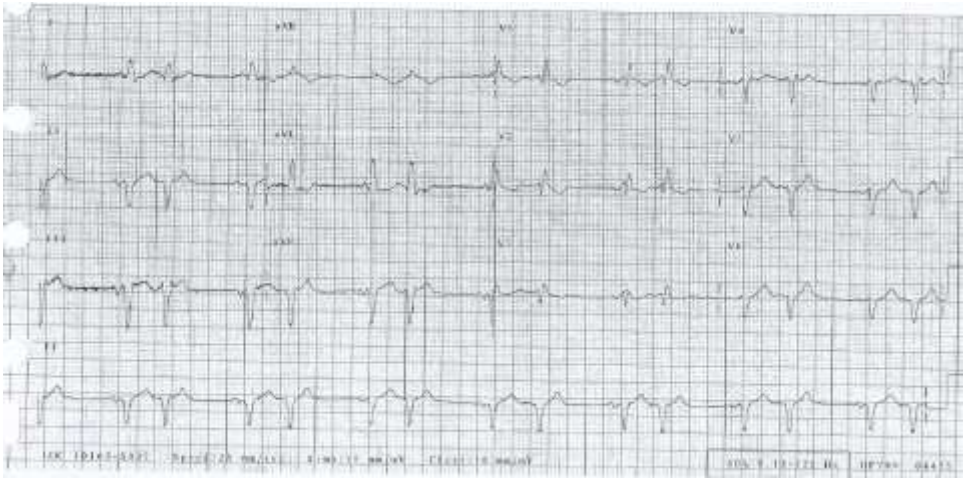
Let's Do the ESPN Method

- Lead 1 is most equiphasic
- Sister lead is lead aVF
- aVF is negative

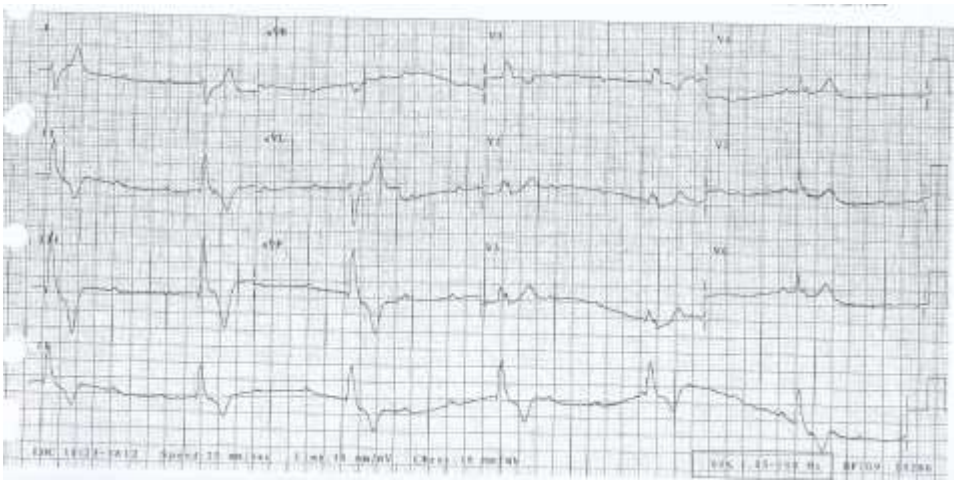




Case Study ECG #1 Admission



Case Study ECG #2



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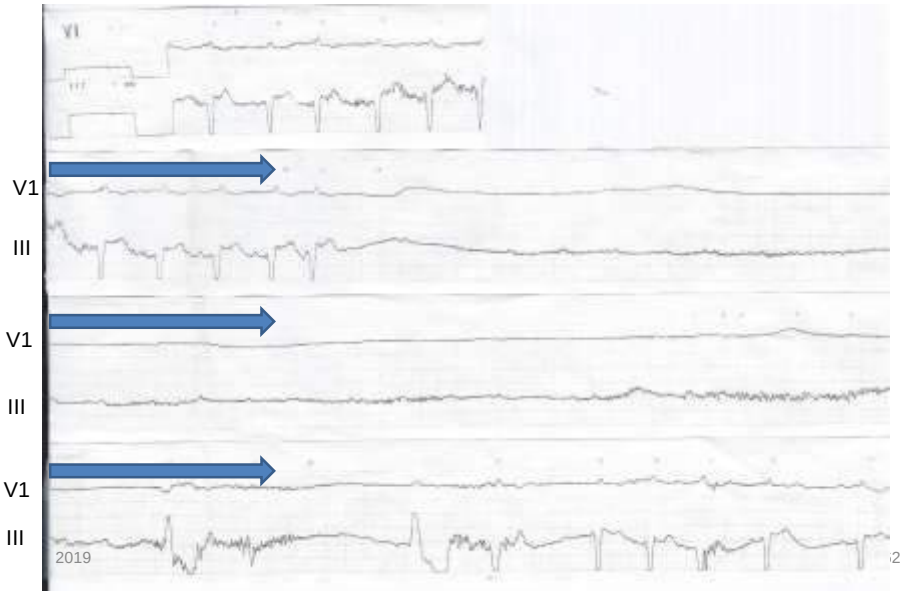
Case Study ECG #3



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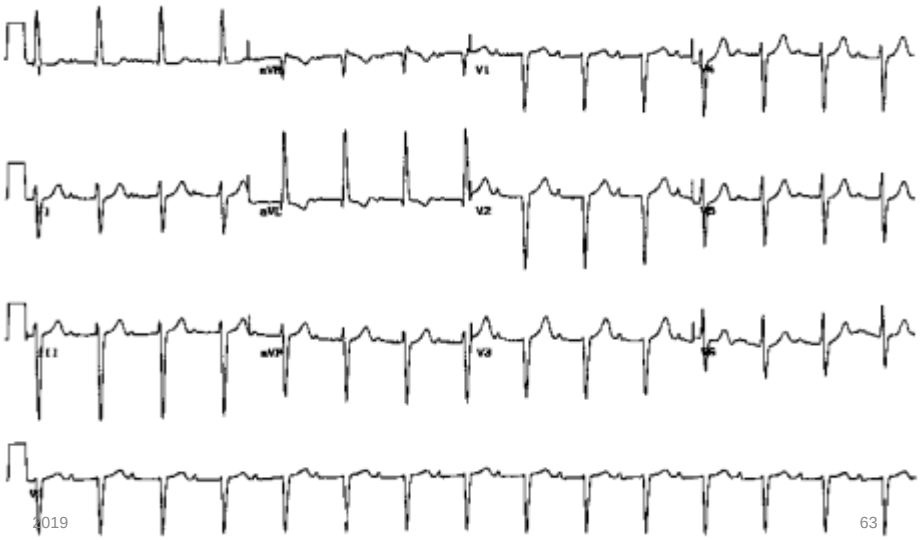
ECG #1



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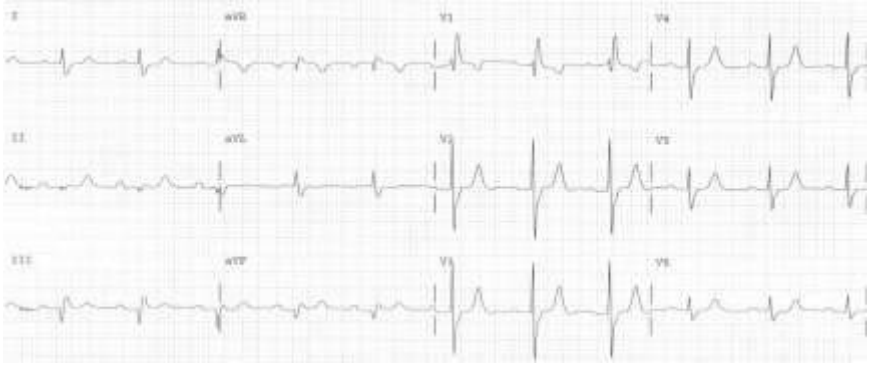
ECG #2



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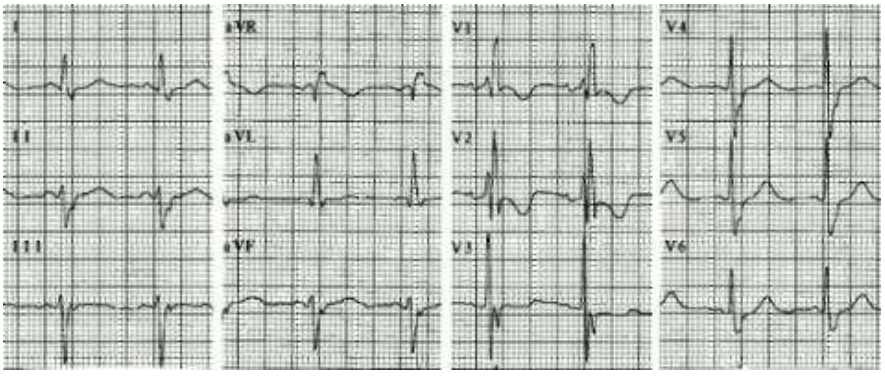
ECG #3



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Left anterior HemiBlock during acute MI

LAHB estimated to occur in approximately 7% to 15% of anterior / anteroseptal myocardial infarctions.

When combined with RBBB the stakes are higher.

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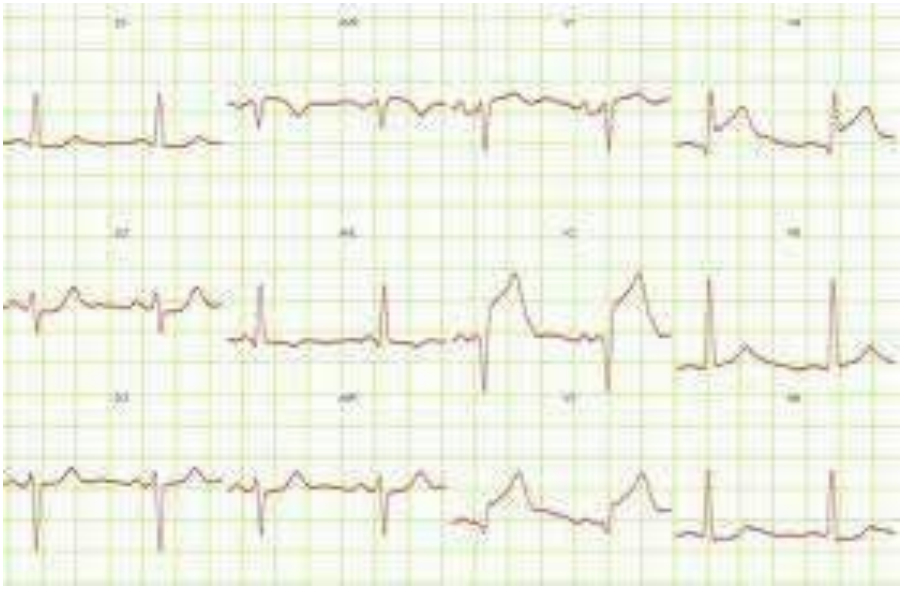

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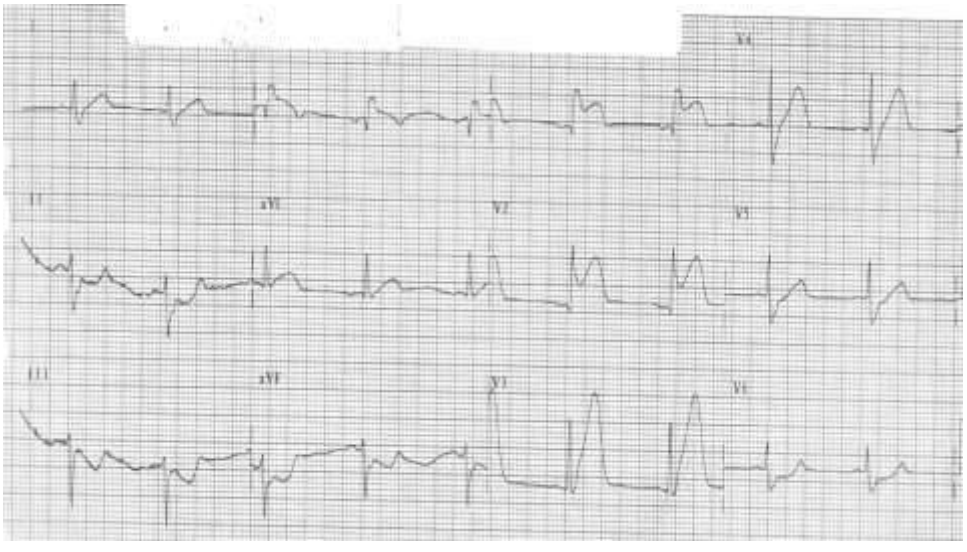

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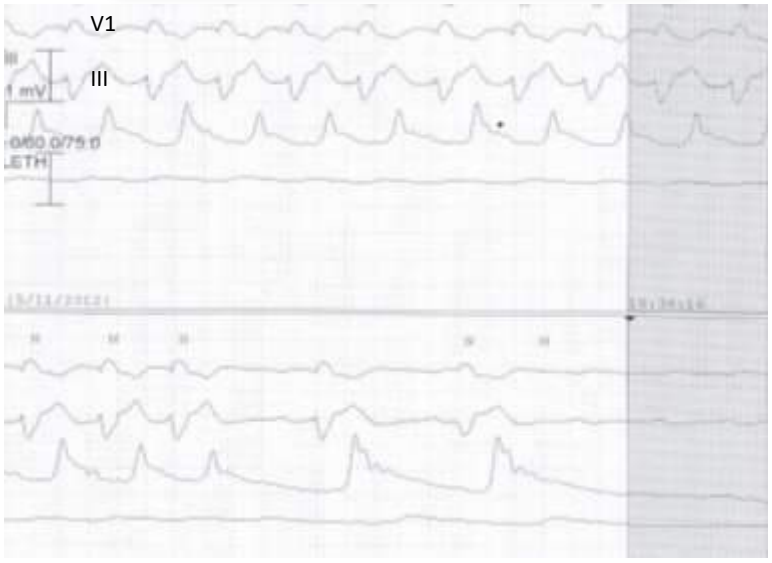
Using the Bedside Monitor: V1 and Lead III



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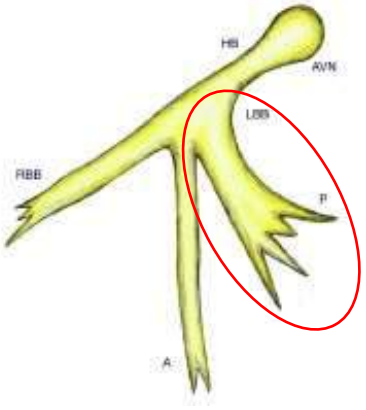
Left Posterior Hemiblock



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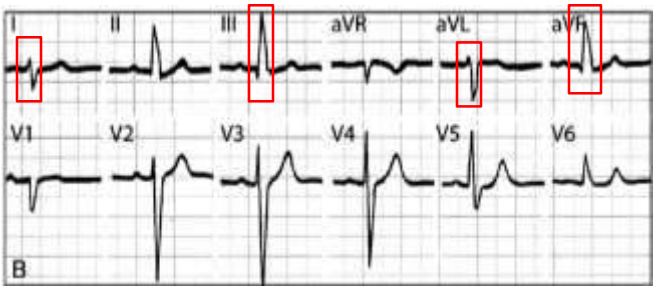
Recognition of Left Posterior Hemiblock

- rS in I and aVL
- qR in III and aVF



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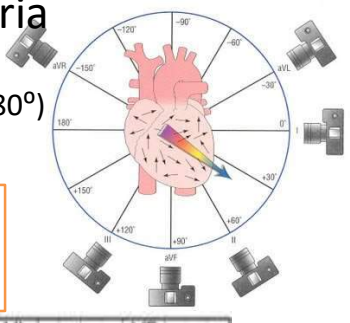
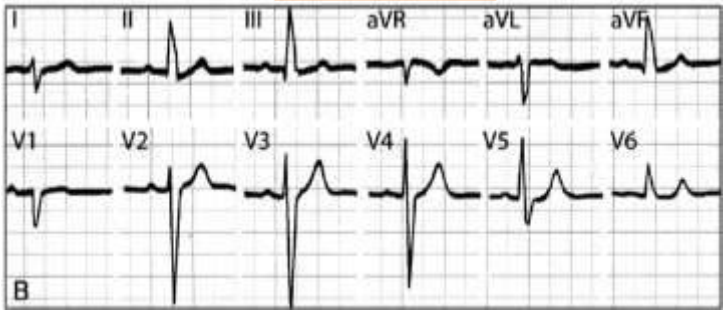
Left Posterior Hemiblock: Additional Diagnostic Criteria



- Right Axis Deviation (+90º to +180º)
  - *Rule out other causes of RAD*
- Normal QRS

Let's use your hands to calculate axis



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Causes of Left Posterior Hemiblock

- Does not occur in healthy people without cardiac disease
- Associated with many myocardial diseases.
- When it occurs in acute MI it is usually associated with RBBB and carries a poor prognosis
  - Highest incidence in inferior or posterior MI
  - Incidence is < 0.8%

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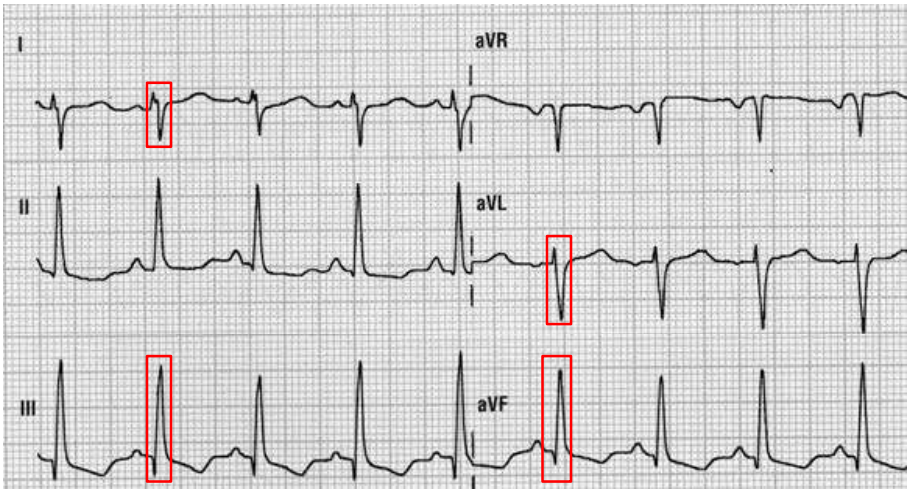

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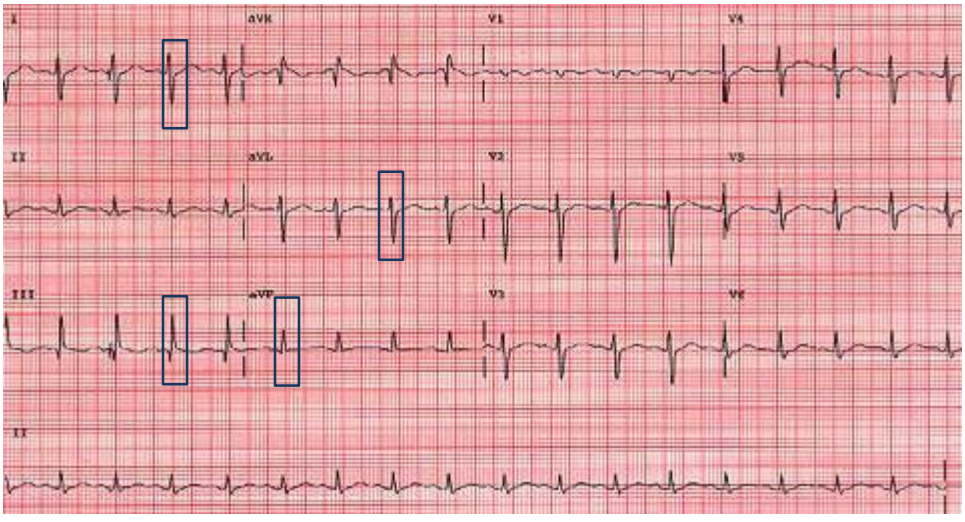

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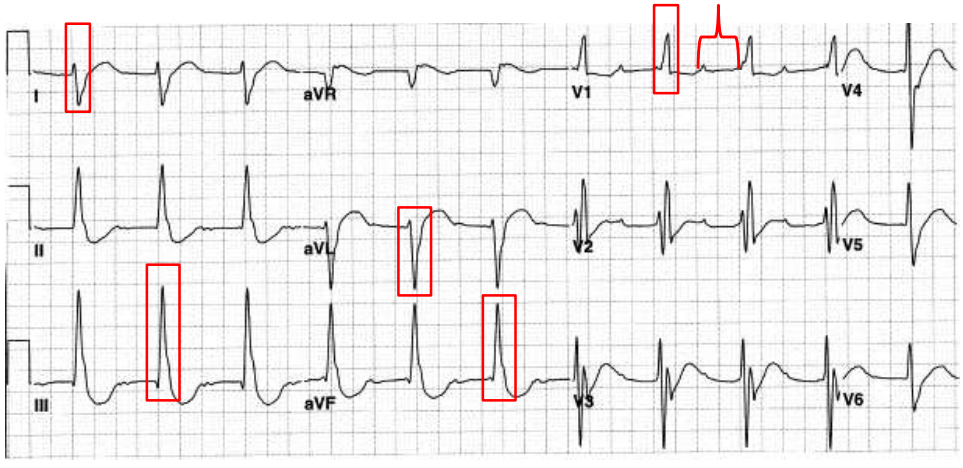

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Other Conduction Delays

- QRS duration > 110 msec (0.10 sec) in adults is considered abnormal
- Incomplete RBBB: QRS duration 110 to 120 msec with rsr' in V1
- Incomplete LBBB: QRS duration 110 to 119 msec with presence of LV hypertrophy pattern

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- QRS > 110 msec may also be a non specific conduction disturbance
- QRS width can change in response to injury or ischemia
  - Peri infarction block with a pathological Q wave
  - Peri ischemic block – transient with acute injury

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Chamber Enlargement (Hypertrophy)

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More on Chamber Enlargement

- Chambers can be enlarged for one of two reasons:
  - Increased pressure resulting in hypertrophy
    - Example: Aortic valve stenosis results in left ventricular hypertrophy
  - Increased volume resulting in dilatation
    - Example: Mitral valve regurgitation results in left atrial volume overload and subsequent enalrgement
- The ECG is not the ideal tool for differentiating the cause of enlargement

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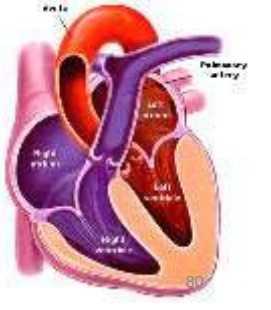
Right Atrial Enlargement

- Right atrial enlargement is caused by conditions that increase volume or pressure in the right atrium
  - Tricuspid stenosis or regurgitation
  - High right ventricular pressure
  - Right ventricular hypertrophy
  - Pulmonary artery hypertension
  - Chronic lung disease
  - Pulmonic valve stenosis or regurgitation
  - Congenital heart disease



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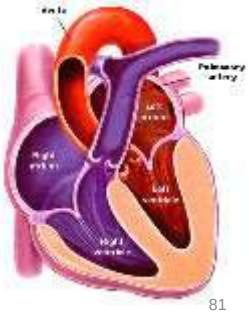
Left Atrial Enlargement

- Left atrial enlargement is caused by conditions that increase volume or pressure in the left atrium
  - Mitral stenosis or regurgitation
  - Systemic hypertension
  - Left ventricular failure
  - Left ventricular hypertrophy



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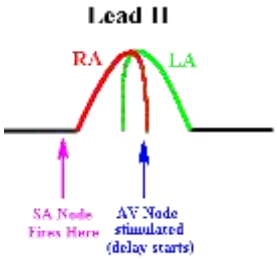
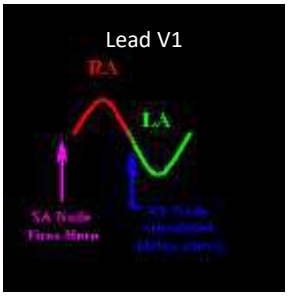
P Waves



- P waves represent atrial depolarization and spread of electrical impulse through the atria
  - Upslope of P represents depolarization of right atrium
  - Downslope of P represents depolarization of left atrium



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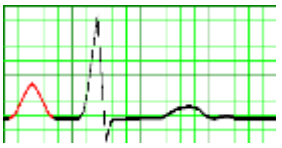
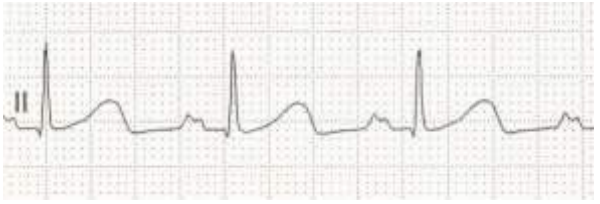
P Waves: Lead II

- Tall P waves could indicate RA hypertrophy



- A fat P wave could indicate LA hypertrophy



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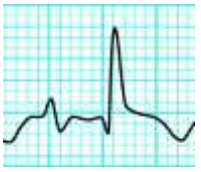
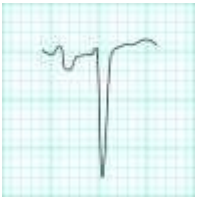
P Waves: V1

- A predominate 1st half of biphasic P wave could indicate RA hypertrophy



- A predominate 2st half of a biphasic P wave could indicate LA hypertrophy



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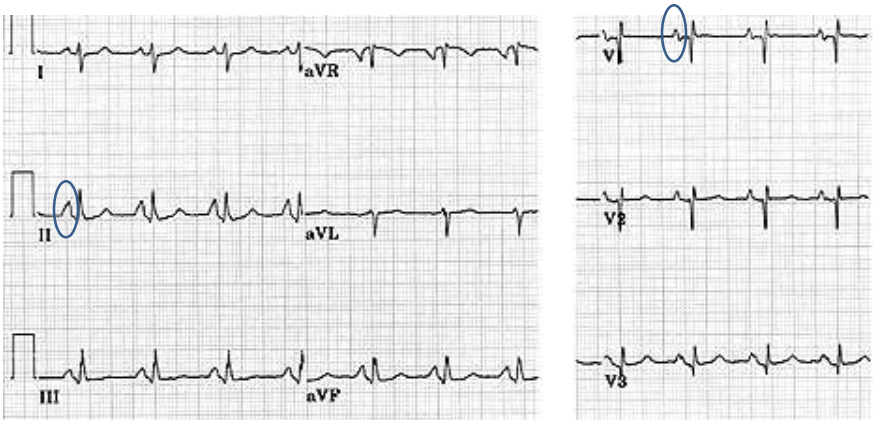
RA Hypertrophy



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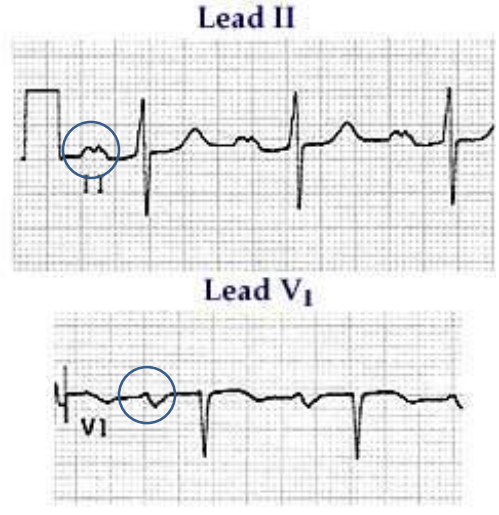
LA Hypertrophy



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ECG in Ventricular Hypertrophy

- ECG criteria used to identify ventricular hypertrophy are not very reliable
  - Good specificity
    - When ECG changes of ventricular hypertrophy are seen there is usually hypertrophy present
  - Very poor sensitivity
    - ECG changes are not always seen even when there is hypertrophy present
- Patients who meet one set of criteria may not meet another
- QRS voltage is influenced by age, gender, race and body build

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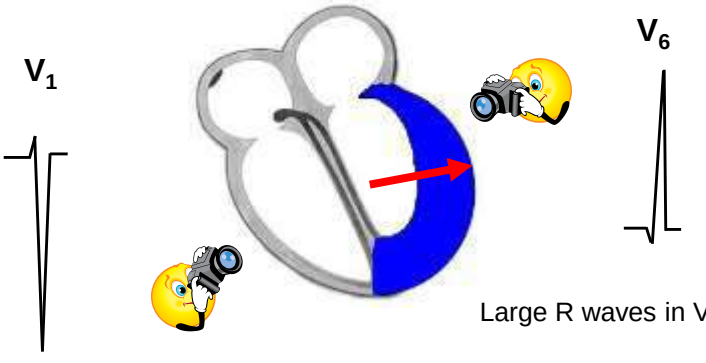
Left Ventricular Hypertrophy



Large S waves in V_1, V_2

Large R waves in V_5, V_6

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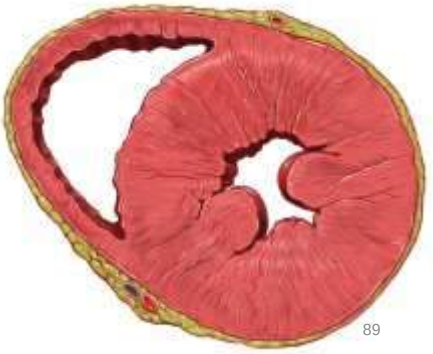
Causes of LVH

- Chronic hypertension (most common)
- Aortic stenosis or insufficiency
- Hypertrophic cardiomyopathy
- Coarctation of aorta



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LVH

- Common reason for false positive ST elevation
- Anatomic LVH may be present in absence of ECG criteria
  - To ascribe ST elevation to LVH the ECG must meet the voltage criteria

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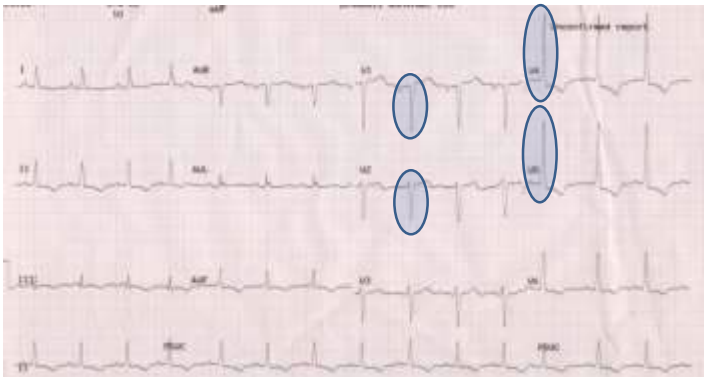
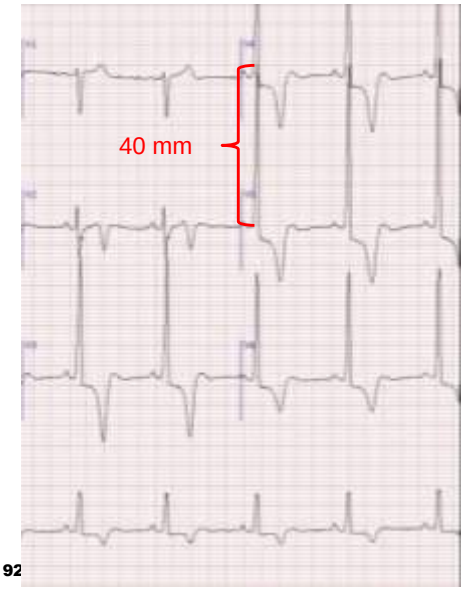
Left Ventricular Hypertrophy

- **V1 and V2 (V3)**
  - Deeper than normal S waves
  - Small r waves
- **V5 and V6 (V4)**
  - Taller than normal R waves
  - Small S waves



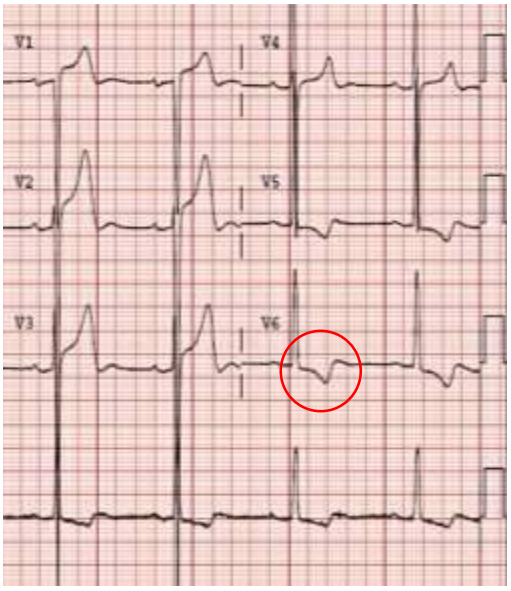
LVH Voltage Criteria

- One or more voltage criteria
- Only applicable if QRS is < 120 ms
- **Precordial lead voltage criteria**
  - R-wave in V5 or V6 > 26 mm
  - R-wave in V5 or V6 + S-wave in V1 > 35 mm
  - Largest R-wave + largest S-wave in precordial leads > 45 mm



ST – T Wave Changes Secondary to LVH

- ST elevation is generally discordant
  - ST elevation in V2 -V3 (V1)
  - ST elevation in lead III
  - ST depression in V4-V6
    - Previously called strain pattern
    - Down sloping – not horizontal
- Not due to LVH
  - ST elevation in lateral leads
  - ST depression in V2-V3



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Estes Scoring System for LVH

R or S in limb lead: 20 mm or more OR S in V1, V2, or V3: 25 mm or more OR R in V4, V5, or V6: 25 mm or more	3
Any ST shift (without digitalis) Typical "strain" ST-T (with digitalis)	3 1
LAD: -15 degrees or more	2
QRS width = 0.09 sec or more	1
Intrinsicoid deflection V5 or V6 = 0.04 or more	1
P-terminal force in V1 > .04	3
Total Possible Points	13
5 = LVH; 4 = probable LVH	

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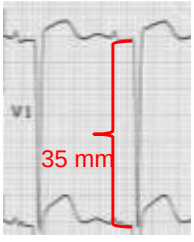
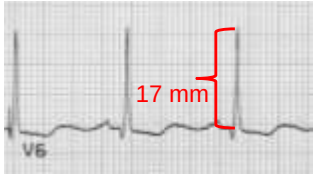
Sokolow and Lyon Criteria for LVH

- S wave in V_1 + R wave in V_5 or V_6 (whichever is larger)
  - If greater than 35 mm = LVH





Total = 52 mm

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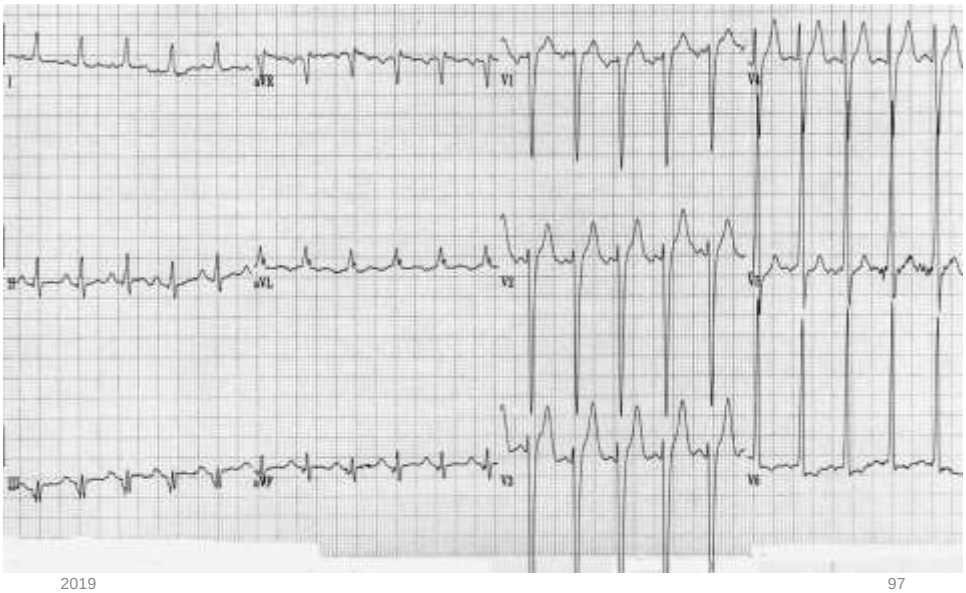
LVH



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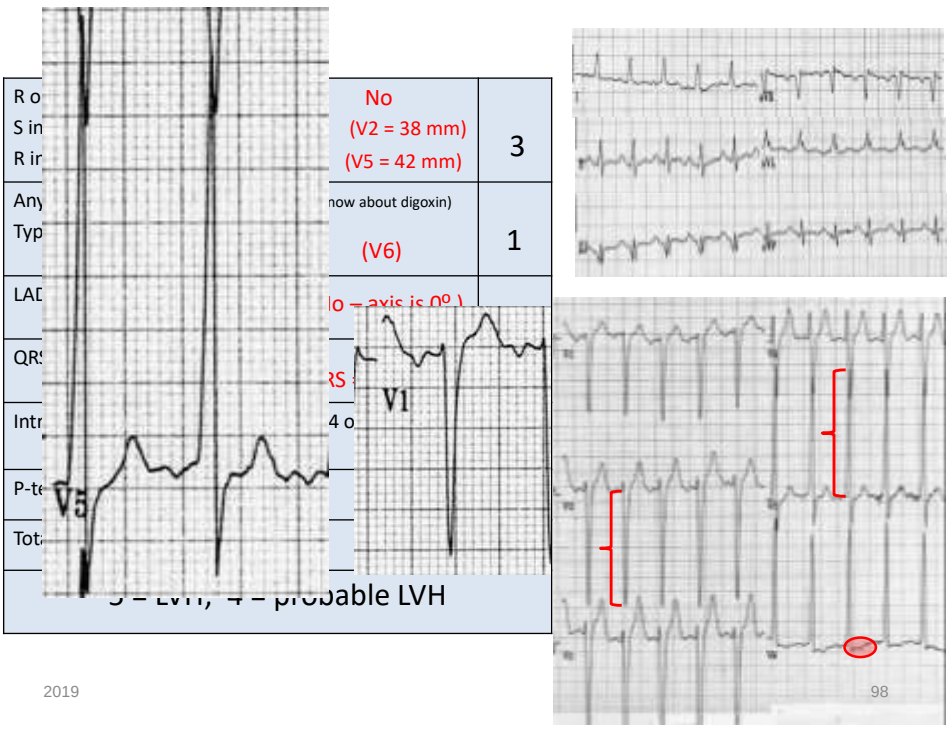

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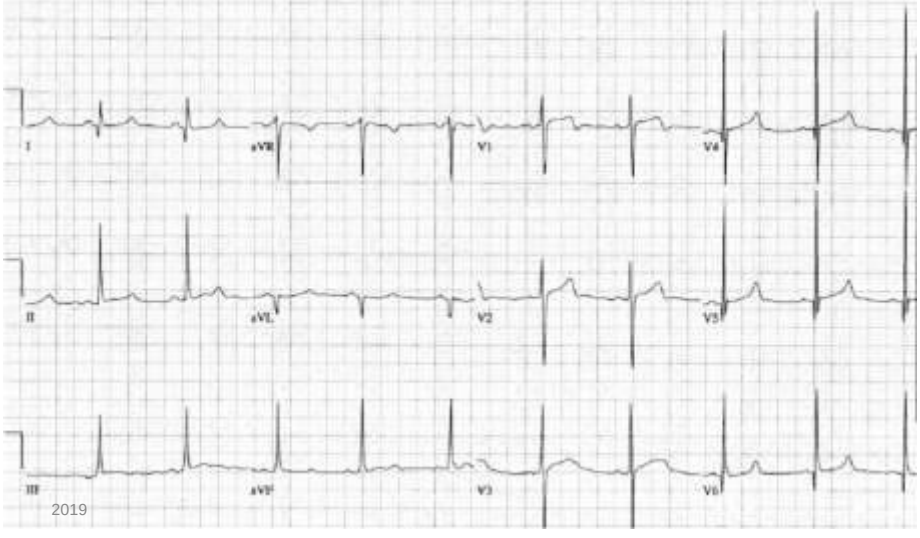
ECG #4

R-wave in V5 or V6 > 26 mm
R-wave in V5 or V6 + S-wave in V1 > 35 mm
Largest R-wave + largest S-wave in precordial leads > 45 mm

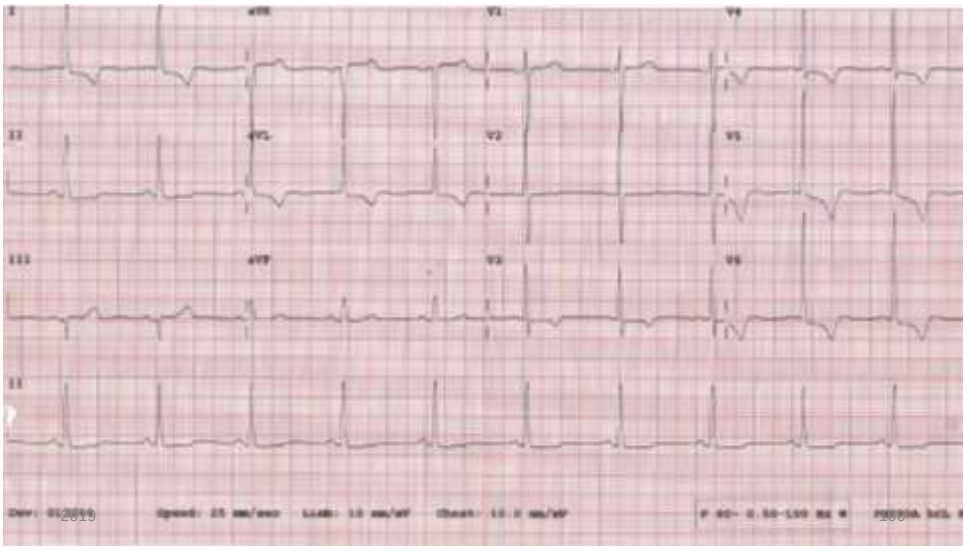


2019

99

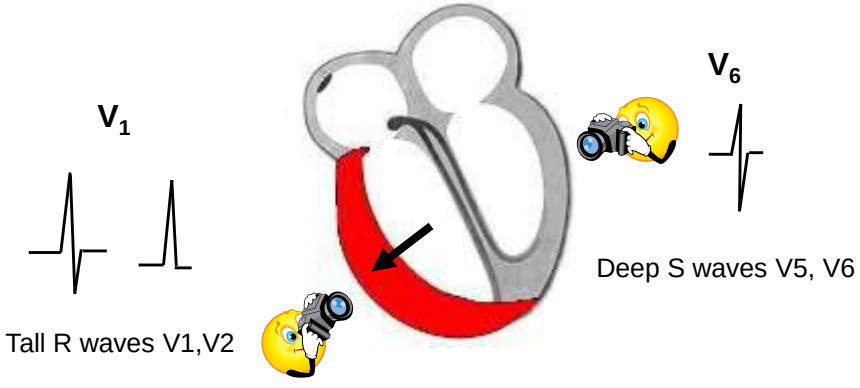
ECG #5

R-wave in V5 or V6 > 26 mm
R-wave in V5 or V6 + S-wave in V1 > 35 mm
Largest R-wave + largest S-wave in precordial leads > 45 mm



100

Right Ventricular Hypertrophy



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Causes of RVH

- Mitral valve disease
- COPD
- Pulmonary hypertension
- Pulmonic stenosis
- Tricuspid insufficiency

102

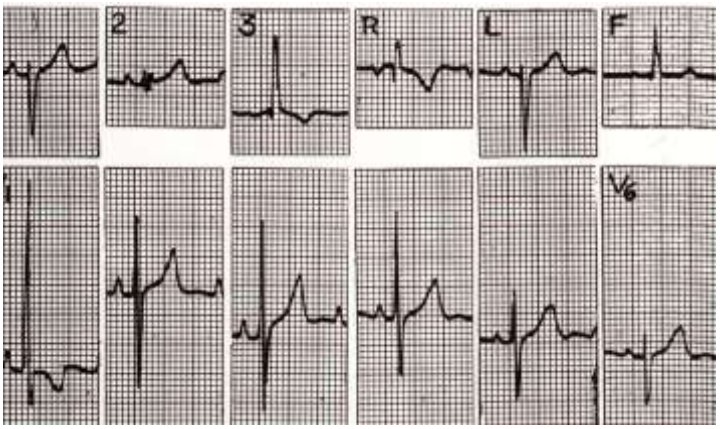
RVH

- Large R waves in RV leads V_1 and V_2
- Deep S waves in the LV leads (V_5, V_6)
- Axis is often deviated to the right (> +90°)
- ST-T strain pattern in RV leads (V_1-V_2) and in leads II, III, and AVF
- Intrinsicoid deflection is often delayed to 0.05 sec in V_1-V_2

103

Right Ventricular Hypertrophy

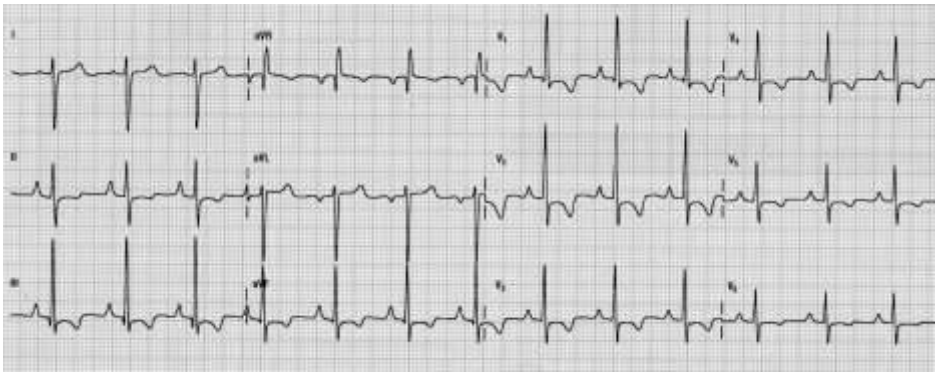
- Right Axis deviation is one of earliest signs
- Reverse R wave progression
- Dominant R wave in V1 and V2 (QRS width is normal)
- Deep S wave in V5 and V6



ECG Clues to RVH

- RAD > + 90 degrees
- R in $V_1 \geq 7$ mm
- R in V_1 + S in V_5 or $V_6 \geq 10$ mm
- R/S ratio in V_1 = 1.0 or more ($R \geq S$)
- S/R ratio in V_6 = 1.0 or more ($S \geq R$)
- V_1 ID = .04 or more
- Incomplete RBBB
- Right atrial enlargement

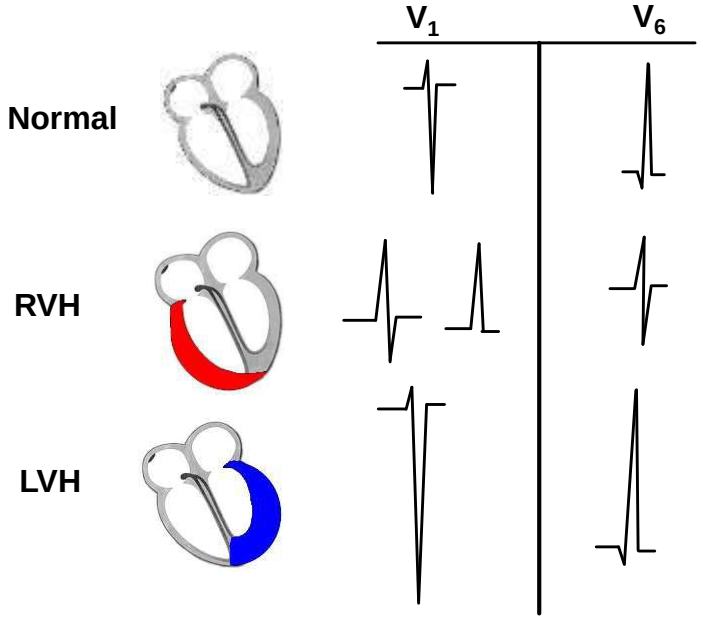
RVH



Right axis deviation? + 150º
R in V1 ≥ 7mm? Yes: 17mm
R in V1 + S in V5 or V6 ≥ 10 mm? Yes: 22mm
R/S ratio in V1 ≥ 1 (R = or > S)? Yes
S/R ratio in V6 ≥ 1 (S = or > R)? No

ID in V1 ≥ .04 sec? Yes: .04
Incomplete RBBB pattern? No
ST-T strain in II, III, aVF? Yes
Right atrial enlargement? Yes

106



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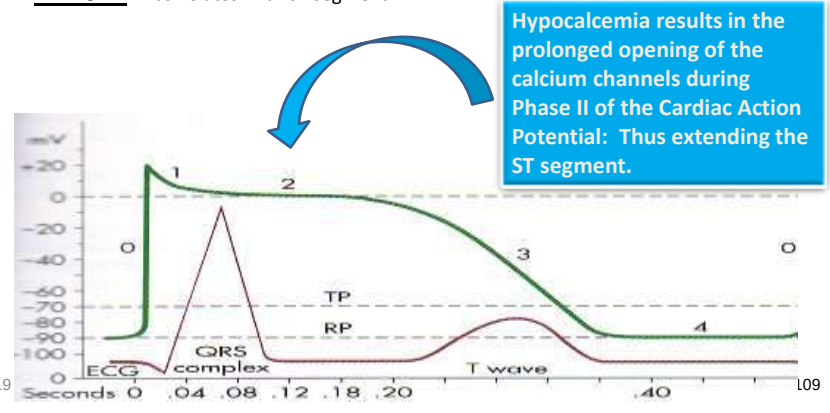
Electrolyte Effects

- Potassium and calcium are the two electrolytes with the most influence on the ECG
- Changes are non specific
  - ECG cannot be considered diagnostic of an electrolyte abnormality
  - Electrolyte abnormalities can occur in the absence of ECG changes
- Magnesium abnormalities aren't revealed by changes on the ECG
  - Can result in cardiac arrhythmias
  - Magnesium is treatment of choice in Torsades de Pointes

108

Action Potential of Cardiac Cells

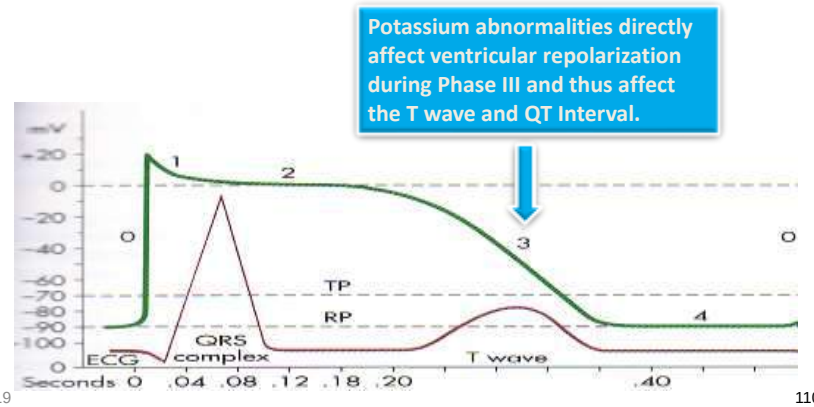
- Phase 0: Rapid depolarization – <u>Sodium Influx</u> (beginning of QRS complex)
- Phase 1: Brief, rapid initiation of repolarization
- Phase 2: Slowing of the repolarization – <u>Calcium Influx</u> – correlates with ST segment



109

The Electronics

- Phase 3: Sudden acceleration in the rate of repolarization - Potassium Efflux – Correlates with T wave
- Phase 4: Resting membrane potential



Hyperkalemia ECG Changes

Tall narrow peaked T waves

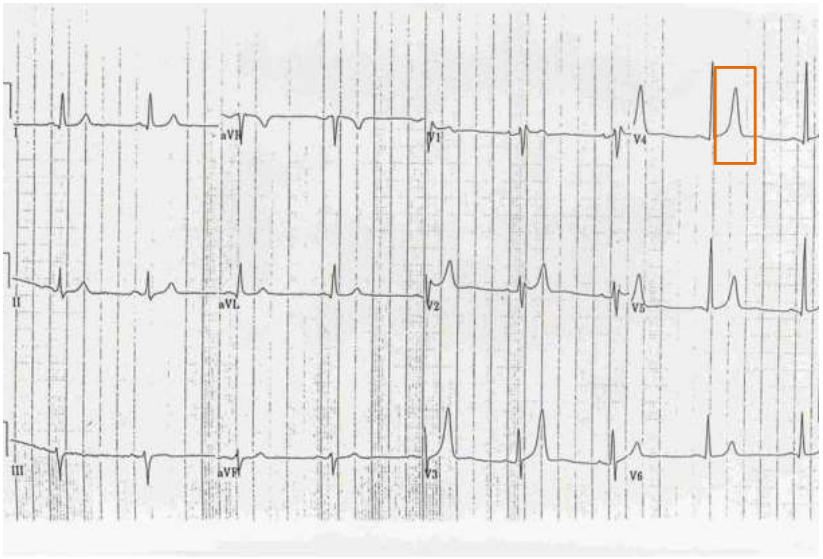
Wide QRS

Prolonged PR and flattened to absent P wave

Dysrhythmias

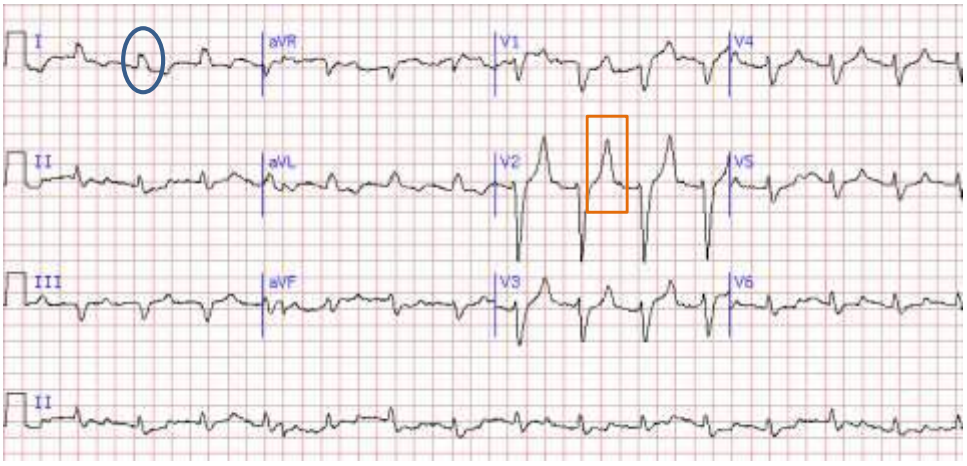
- √ Bradycardia / heart block
- √ Sine wave pattern
- √ Asystole

Hyperkalemia



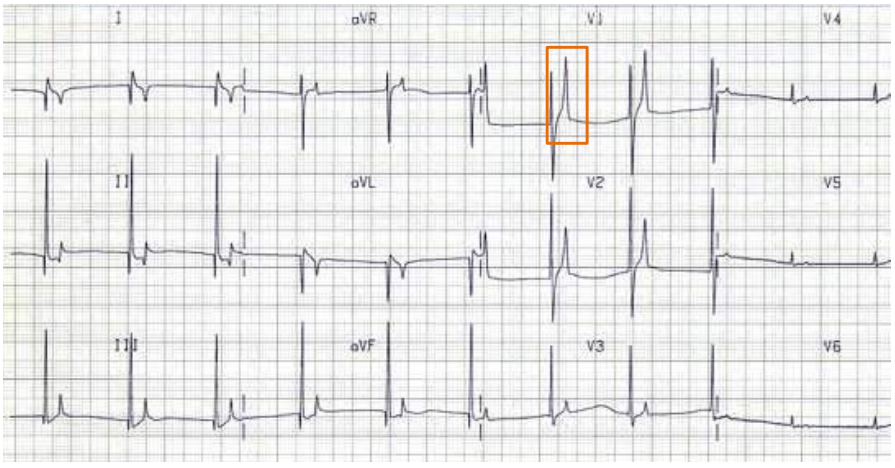
112

Hyperkalemia



113

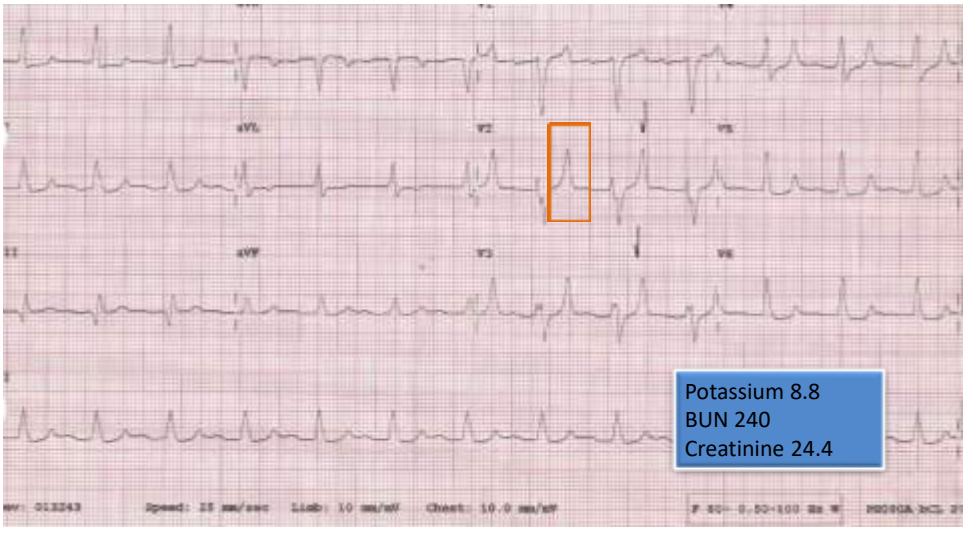
Hyperkalemia



Note: This is not a normal sinus rhythm.

114

Hyperkalemia



Note: Prolonged PR interval and flattening of the P wave.

115

Hypokalemia: ECG Changes

- Mild hypokalemia: delays ventricular repolarization
  - ST depression, flattening of T wave, inverted T wave
  - Heightened U waves, prolonged QT interval
- Increases risk for Torsades de Pointes
- Lowered threshold for ventricular fibrillation and reentrant tachycardias
- Severe hypokalemia
  - Increased PR interval
  - Increased QRS interval

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Clinical Pearls

Digoxin

- Hypokalemia increases risk of digoxin toxicity.

Class III Antiarrhythmics

- Hypokalemia increases the risk of Torsades de Pointes with potassium channel blocking medications

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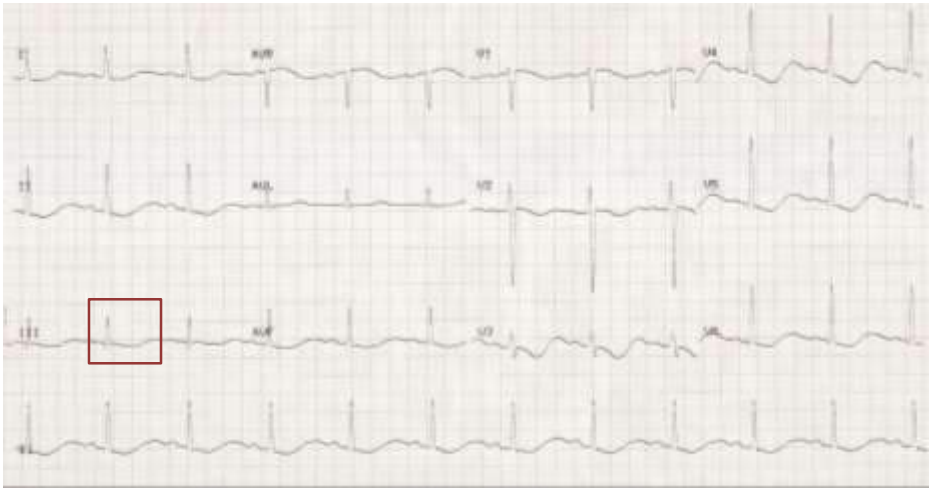
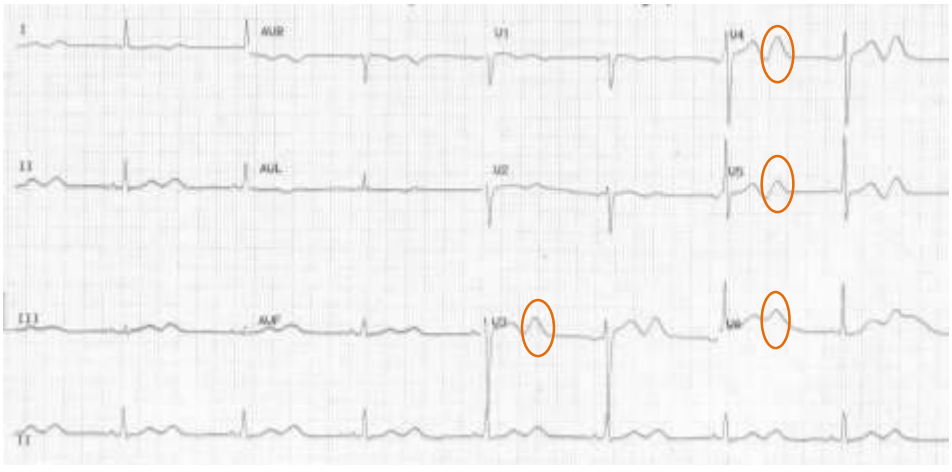
Hypokalemia



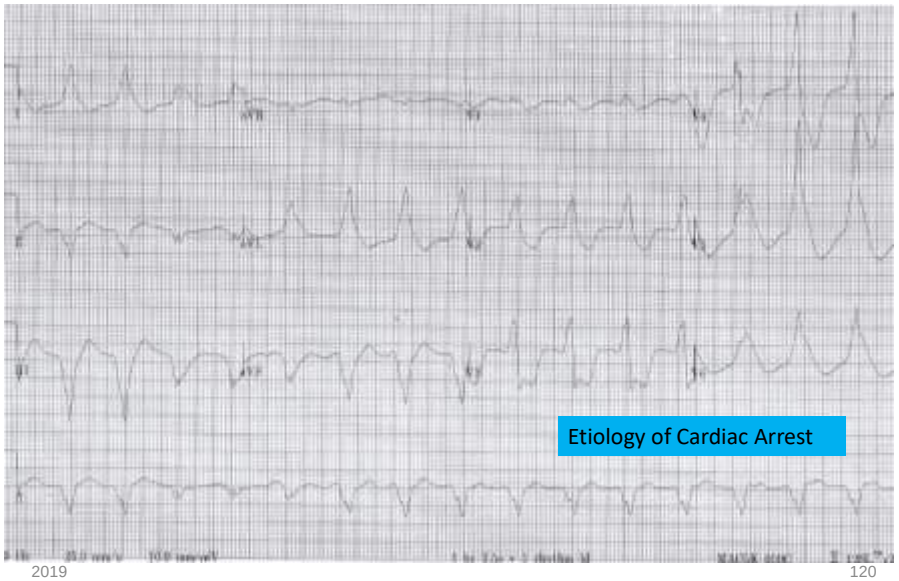
118

Hypokalemia



119

Hypokalemia: Severe



120

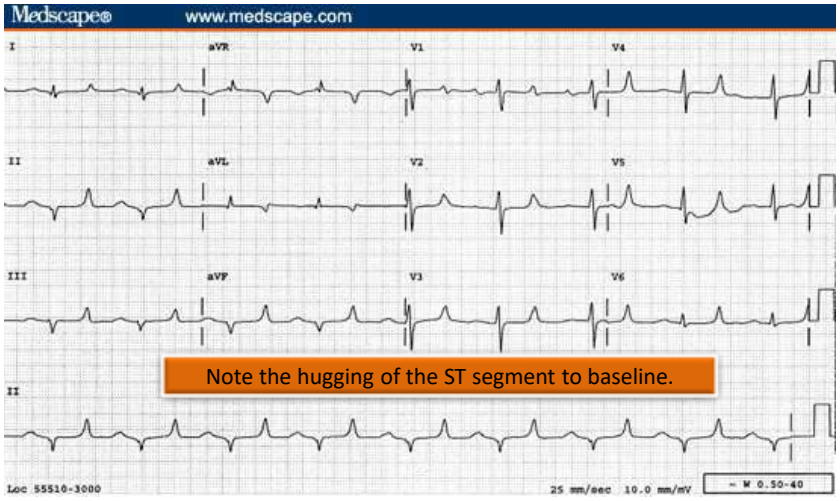
Hypocalcemia: Cardiac Effects

Hypocalcemia results in the prolonged opening of the calcium channels during Phase II of the Cardiac Action Potential: Thus extending the ST segment.

- Cardiovascular effects:
  - Decreased contractility
  - Hypotension
  - Prolonged QT
    - ST segment hugging baseline for extended period (Normal up to 0.15ms)
    - QT prolongation is not due to delay in ventricular repolarization
  - Torsades de pointes
  - Bradycardia / heart block
  - ***Digitalis insensitivity***
  - Heart failure
  - Cardiac arrest

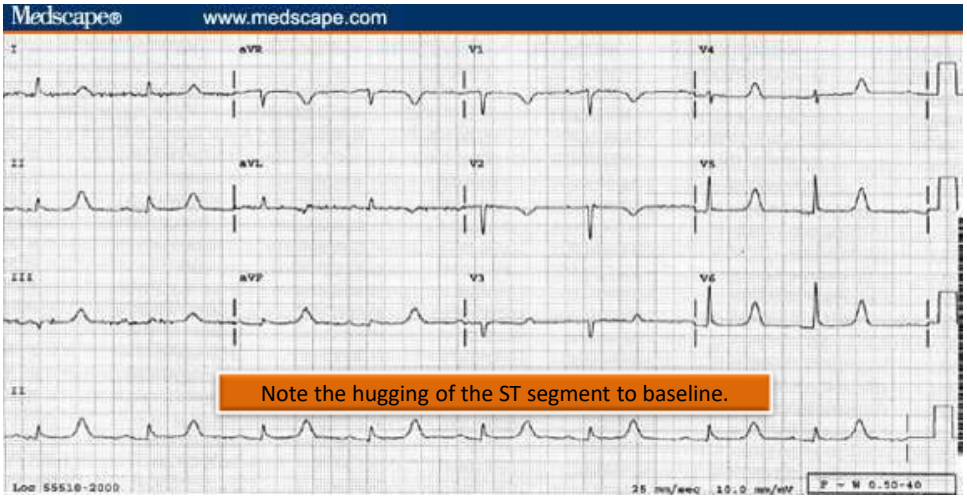
121

Hypocalcemia



122

Hypocalcemia



124

Hypercalcemia

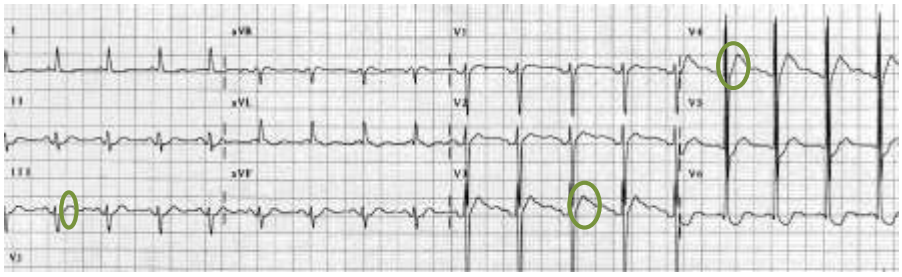
- Cardiac symptoms:
  - Hypertension (may be offset by co-existing dehydration)
  - Cardiac ischemia
  - Arrhythmias (conduction abnormalities)
  - Digitalis toxicity.

- **ECG signs**
  - **Shortened QT segments (secondary to shortened ST segments)**
  - **Short ST segments can cause ST to merge with T wave (similar to what occurs with hyperacute T wave in a STEMI)**

- Life threatening signs and symptoms are rare unless calcium levels reach > 14 mg/dL.

125

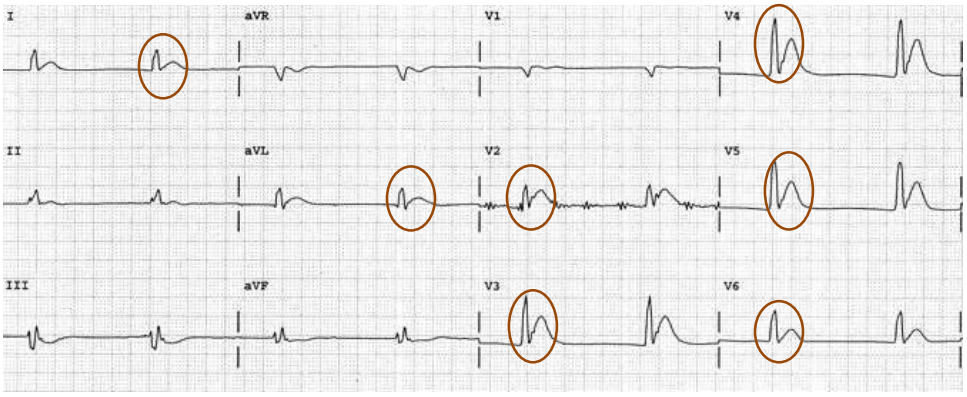
Hypercalcemia



Note lack of horizontal component of ST segment and abrupt take off of T wave after QRS.

126

Hypercalcemia



Note lack of horizontal component of ST segment and abrupt take off of T wave after QRS.

127

Clinical Pearl

Calcium imbalances can alter appearance of ECG but are less likely to cause cardiac arrhythmias than potassium imbalances.

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Drug Effects

Digoxin

- Increases vagal activity
- Digoxin decreases conduction velocity through the AV node
  - HOWEVER: Sympathetic stimulation easily overrides the inhibitory effects of digoxin on AV node conduction
- The conduction velocity increases in the atria, but decreases in the AV node.
- **Automaticity is also increased, in the atria,** AV node, Purkinje fibers and ventricles.

Digoxin Toxicity

- EKG Changes with Toxicity
  - Increased automaticity with impaired conduction is common (example: PAT with AV Block)
- Other Signs and Symptoms of Toxicity
  - N & V (most frequent 1st sign), HA, Confusion
  - Visual disturbances: halos, change in color perception

Digitalis Effect on ECG

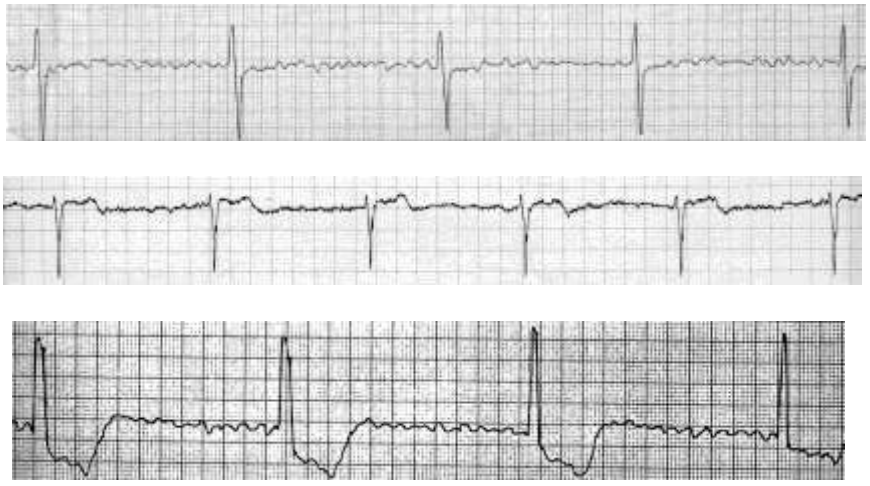
- **Sagging depression of ST segment in leads with positive QRS**

- Reduced T wave amplitude
- Possible T wave inversion
- Increased U wave amplitude

PR interval may also be prolonged.

- Difficult to evaluate if hypertrophy or BBB

Atrial Fibrillation with Complete Heart Block



133

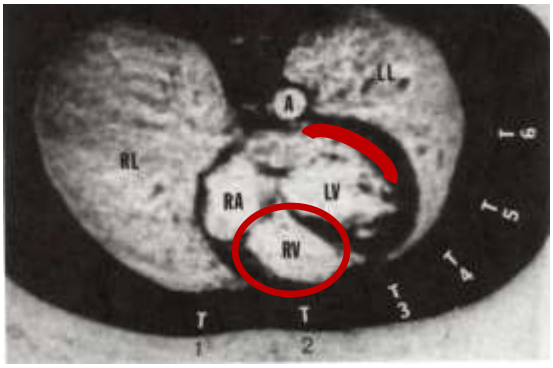
Advanced Concepts in 12 Lead ECG Interpretation

ST Segments & T Waves
Patterns of Injury & Ischemia
Myocardial Infarction Mimics

134

Note!

- Posterior wall of the left ventricle and the right ventricle are not captured on the standard 12 lead ECG.



135

THE ST SEGMENT and T WAVE

ABNORMALITIES RESPESENT ABNORMALITIES IN VENTRICULAR REPOLARIZATION

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MUST DIFFERENTIATE BETWEEN PRIMARY OR SECONDARY ST AND T WAVE CHANGES

Primary versus Secondary Repolarization Abnormalities

- **Primary Changes**
  - Changes in the **REPOLARIZATION** phases of the action potential
  - Occur in the *absence* of changes in **DEPOLARIZATION**
  - Ischemia, myocarditis, drugs, toxins, electrolyte abnormalities (calcium, potassium), abrupt HR changes, hyperventilation, body position changes, catecholamines, sympathetic stimulation, temperature changes
  - Normal QRS axis with an abnormal direction of the T wave usually indicates a primary change

Primary versus Secondary Repolarization Abnormalities

- **Secondary Changes**
  - ST and T wave changes result from *changes* in the sequence and /or duration of ventricular **DEPOLARIZATION**
  - Usually associated with changes in the QRS shape and duration (but not always)
  - ***Changes*** the ***sequence of depolarization altering*** the ***repolarization*** sequence
  - BBBs, ventricular pre excitation, ectopic and paced ventricular beats

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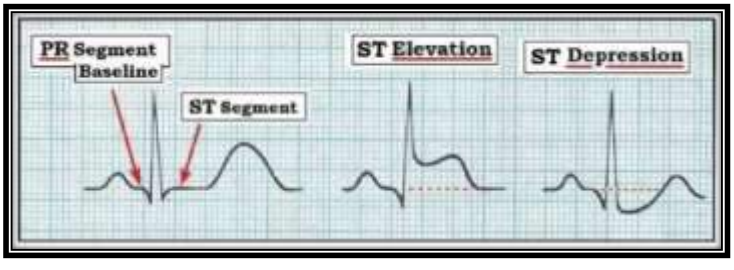
ST Segment

- In limb leads the ST segment is normally isoelectric but may be slightly elevated or depressed by less than 1mm
- In precordial leads ST segment elevation is normally not more than 1 to 2 mm (small elevation normal in many people)



Clinical Application:
1) Do not accept any elevation in limb leads
2) Do not accept any depression in chest leads

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Thresholds for ST Elevation

Gender	Age	Leads	Threshold
Men	≥ 40	V2, V3	2 mm
Men	≥ 40	All except V2, V3	1 mm
Men	< 40	V2, V3	2.5 mm
Women	All ages	V2,V3	1.5 mm
Women	All ages	All except V2, V3	1 mm
Men	≥ 30	V3R, V4	0.5 mm
Men	< 30	V3R, V4R	1 mm
Women	All ages	V3R, V4R	0.5 mm
Men	All ages	V7 thru V9	0.5 mm
Women	All ages	V7 thru V9	0.5 mm

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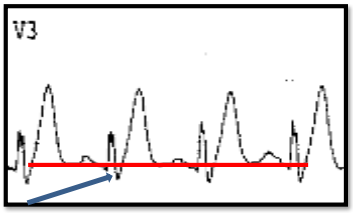
Thresholds for ST Depression

Gender	Age	Leads	Threshold
Men	All ages	V2, V3	0.5 mm
Women	All ages	V2, V3	0.5 mm
Men	All ages	All except V2, V3	1 mm
Women	All ages	All except V2, V3	1 mm

142

The "J" Point

- Point where the QRS complex and the ST segment meet.

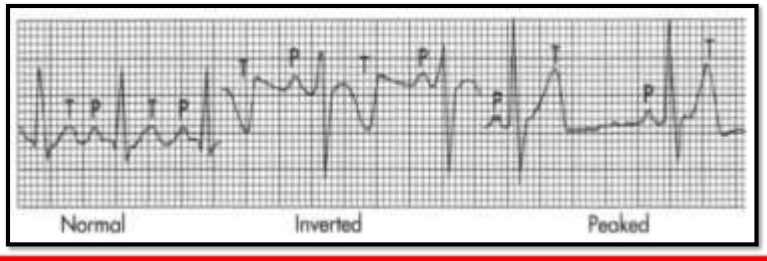


Clinical Application:
There can be ST segment elevation with no J point elevation.

143

T Waves

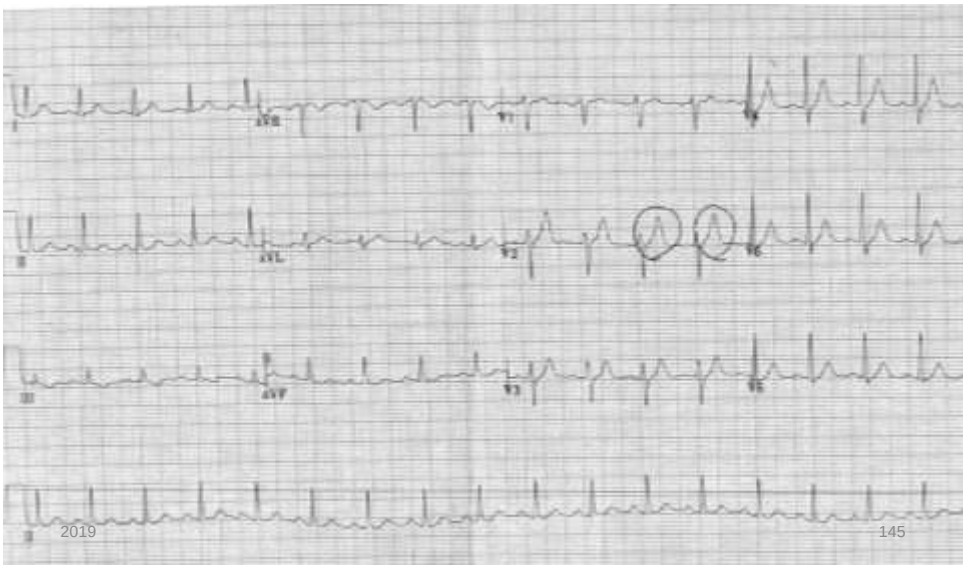
- Represents ventricular repolarization
- Slightly asymmetrical
- Usually upright
- Most likely abnormal If inverted in two contiguous leads
- Not normally > than 5mm (limb leads) to 10 mm (precordial) high



Clinical Application:
3) Never tolerate a T wave that is too big in any lead.

144

ECG A



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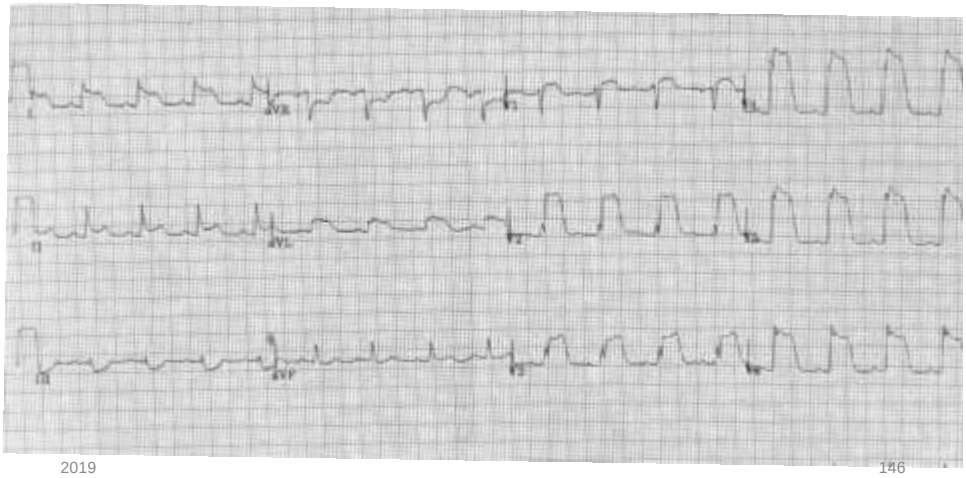
ECG B (2 hours later)



146

ECG Assessment Priorities
When Assessing for Injury or Ischemia

1) Assess for ST segment elevation first
   - ST elevation and need for reperfusion

2) Assess for T wave inversion next
   - Non STEMI or
   - Unstable angina
     - ischemia

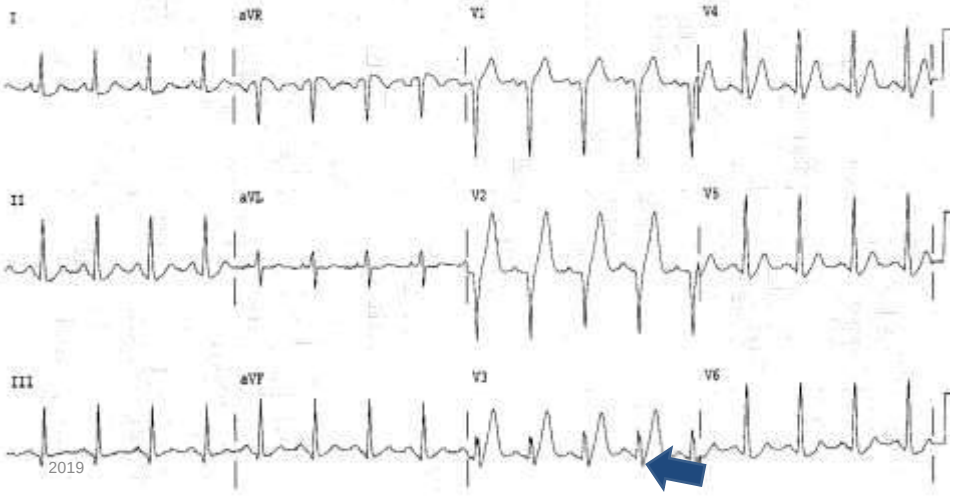
NonStemi: Troponin Abnormally Elevated
Unstable Angina: Troponin Normal

3) Assess for ST segment depression thirdly
   - Supply and demand ischemia (often in V5 regardless of vessel occlusion)
   - OR – reciprocal changes to ST elevation
   - **Clinical application: Supply and demand ischemia is typically not the primary problem in patients at rest**

Hyperacute T Wave

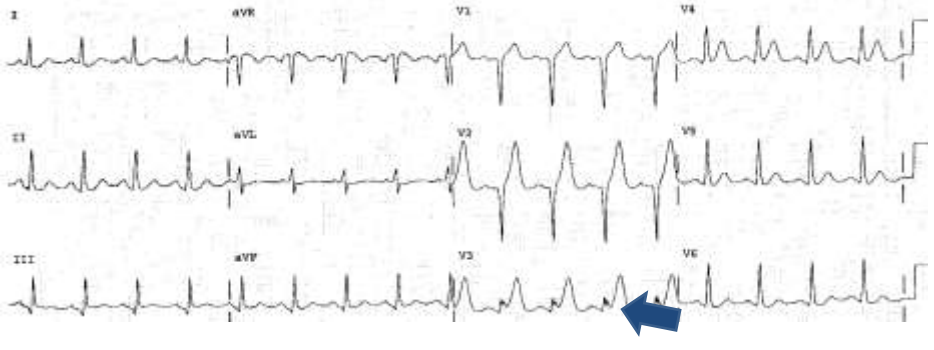
Note: Hyperacute T waves can occur with 2 minutes of a coronary occlusion

Hyperacute T Waves - STEMI



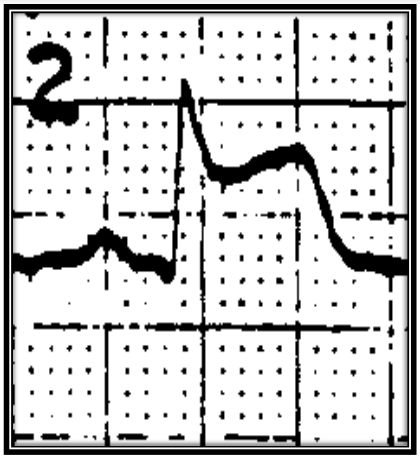
149

Post Hyperacute T Waves - STEMI



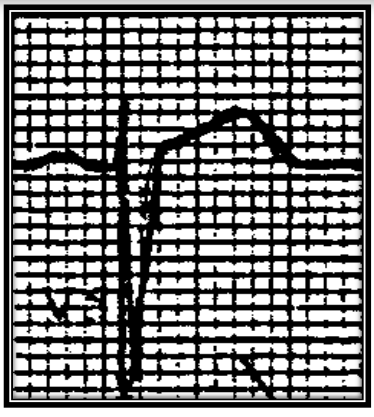
150

J Point Elevation



151

Subtle ST Elevation Forming Larger T Wave



Note: ST elevation is often subtle in the limb leads in an acute MI.

152

T Wave Inversion: Key Points

- T wave should be positive in lead I and II
- **Normal inversion is rare in V2 – V6**
- Inversion in lead III, aVL and aVF may be normal
- Inversion in V1 is common - always compare to previous ECG

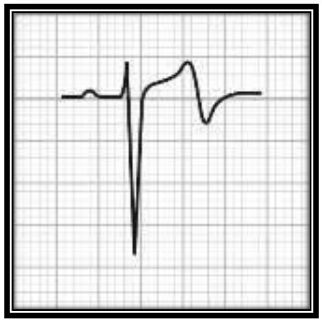
T Wave Inversion Associated With Ischemia /Infarction

- Deep T wave Inversion
- Disproportionate T wave Inversion (in relation to QRS voltage)
- New or changing T wave Inversion
- QTc usually increased

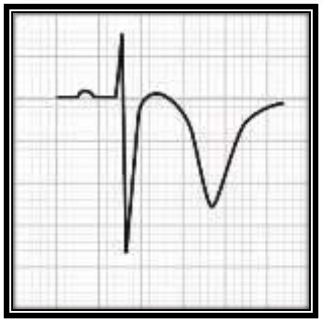
153

2 Types of T Wave Inversion

Terminal T wave inversion



Symmetrical T wave inversion



154

More on T Wave Inversion

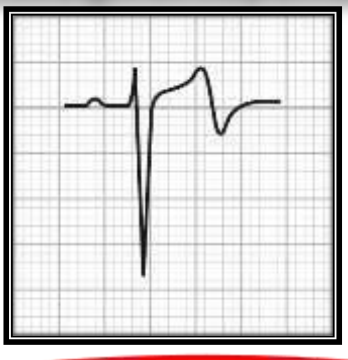
- T wave inversion is a "warning" for ACS (either unstable angina or NonSTEMI) unless……

  – T wave inversion occurs after a STEMI

  - After a STEMI T wave inversion is expected
  - Terminal T wave inversion is a sign of reperfusion after a STEMI
  - Symmetrical T wave inversion will develop after terminal T inversion

Terminal T Wave Inversion: A Sign of Reperfusion

- "



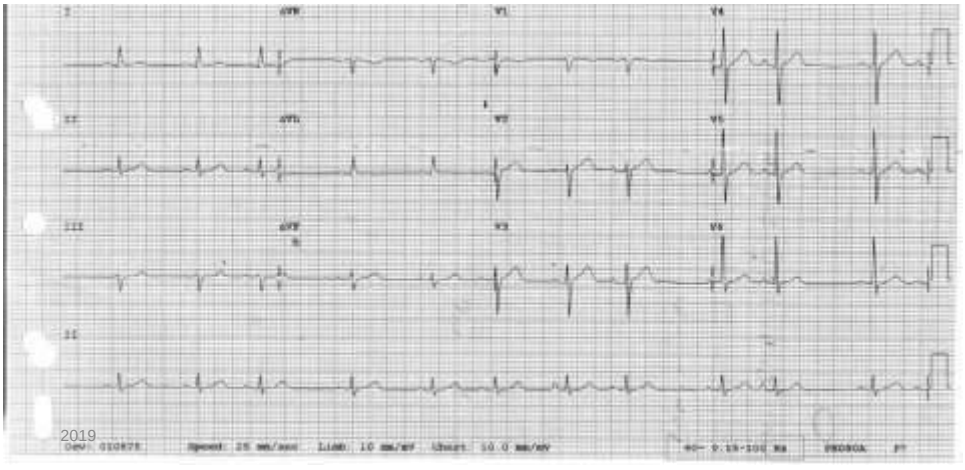
This pattern frequently occurs after successful reperfusion in STEMIs. It is an expected finding after reperfusion.

Wellen's Syndrome

- **T wave inversion occurring in leads V2 and V3**
  - With or without other ST/T wave changes in the other precordial leads
  - In patients with a history of intermittent symptoms consistent with unstable angina
  - Often during pain free period
  - 76% symmetrical T wave inversion
  - 24% terminal T wave inversion
- Has also been referred to as LAD coronary T-wave syndrome
- Associated with recent injury or ischemia caused by a LAD lesion (proximal to septal perforator), and placing the patient at risk for anterior wall myocardial infarction in the absence of coronary revascularization

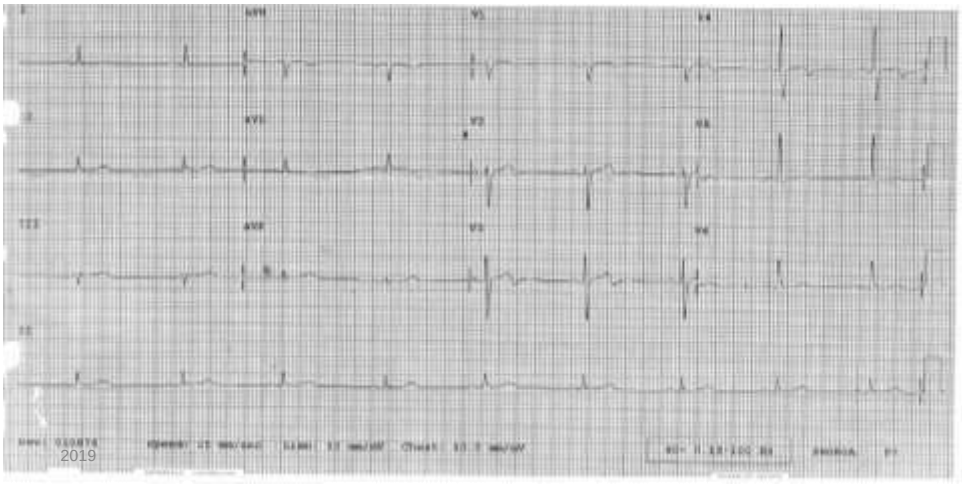
157

Practice ECG 1 of 3



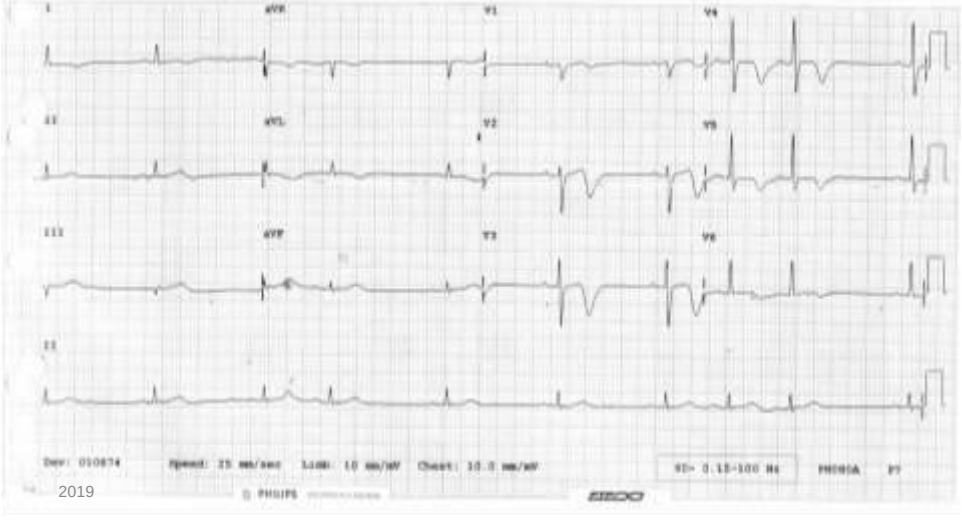
158

Practice ECG 2 of 3



159

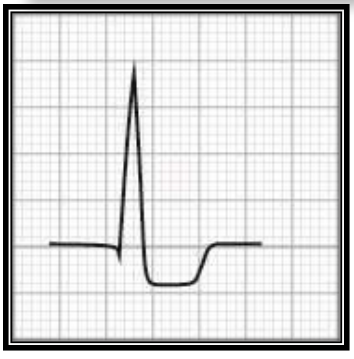
Practice ECG 3 of 3



160

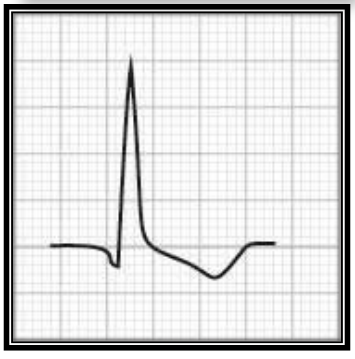
ST Depression

Horizontal ST segment depression



Can be seen in exercise stress testing with supply and demand ischemia.

Down sloping of ST segment



Often seen with left ventricular hypertrophy.

Clinical Pearls

ECG

- Be suspicious of horizontal ST segment depression in patient at rest.
- Suspect left main disease (or significant 3 vessel disease) when diffuse depression and ST elevation in lead aVR (and V1 to lesser extent)

Presentation

- Assess for reasons for supply and demand ischemia at rest (i.e. low hemoglobin).
- Rule out medical reason for falls, motor vehicle crashes, and other trauma (i.e. syncope or near syncope due to cardiac cause).

Live Content Slide

When playing as a slideshow, this slide will display live content

Poll: When evaluating the ECG in a patient having chest pain you note ST elevation in aVR and lead V1 with diffuse ST segment depression in the remainder of the limb leads an precordial leads.

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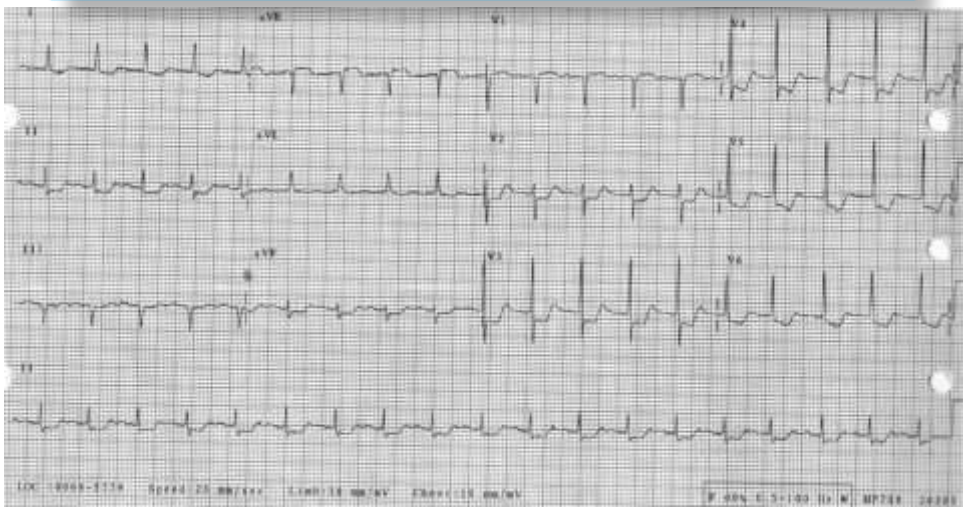
Practice ECG



2019 **Admitted with fall. Fracture femur admitted to surgical floor.** 164

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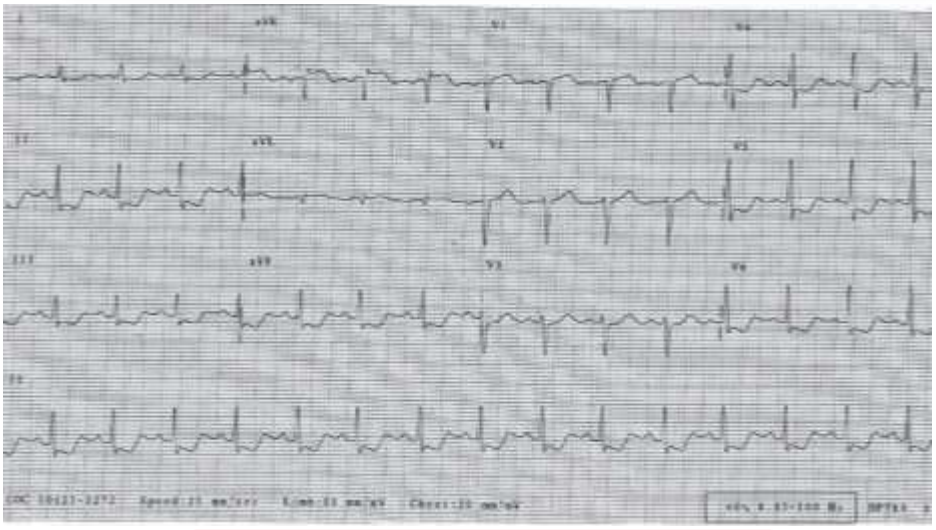
Practice ECG



Developed chest pain in PACU.

Non Specific ST – T Wave Changes That May Indicate Ischemia

- Inverted U waves
- Prolonged ST Segment
- Flat T waves
- Non specific changes in particular need correlated with the patient's clinical condition

Interpreting the ECG

EVOLUTION OF MI

ECG Evolution of a STEMI

- Evolutionary changes occur due to prolongation of the cardiac action potential in regions of stunned myocardium adjacent to the necrotic area.
- Prolongation in repolarization affects the voltage and polarity of the T wave.
- Ventricular remodeling can produce differences in electrical fields that may be responsible for the evolutionary changes.

ECG Changes After STEMI

Non Reperfused

- T wave enlargement
- ST elevation
- Q wave formation or loss of R wave amplitude
- ST stabilization
- **T wave inversion** (within 48 - 72 hours) before ST resolution
- ST resolution
- **T waves stays inverted** for period of time (takes weeks to months)
- Possible disappearance of Q waves

2019

Reperfused

- T wave enlargement
- ST elevation
- Earlier ST normalization and stabilization
- T wave inversion may accelerate
  - Terminal T wave inversion initially
  - T waves deepen symmetrically over time
  - **T waves stays inverted** for period of time (takes weeks to months)
- Q wave development is less pronounced or even absent

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ECG Evolution of a STEMI

T wave inversion within first 3 days predictive of large amount of stunned myocardium.

- **Early terminal T wave inversion can occur with successful reperfusion.**
- **T waves typically stay inverted for weeks to months**
- **Persistent negative T waves or an increase in number of negative T waves during the 6 month period post discharge is associated with less regional wall motion recovery, more ventricular enlargement, and decreased left ventricular function over time.**

2019

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ECG Evolution of a STEMI

- ST segment back to baseline occurs as result of myocardial cell death and reduction in injury current.
  - One of final evolutionary changes.
  - Abrupt resolution of ST elevation indicates reperfusion.
  - Persistent ST elevation is predicator of adverse outcomes.

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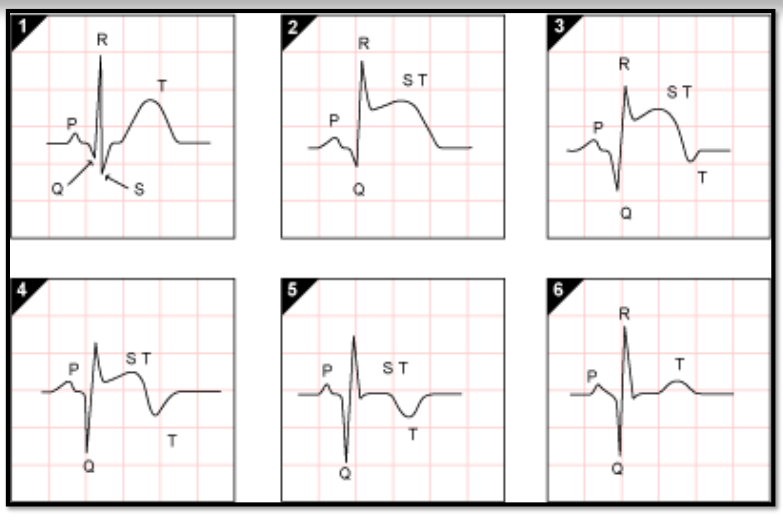
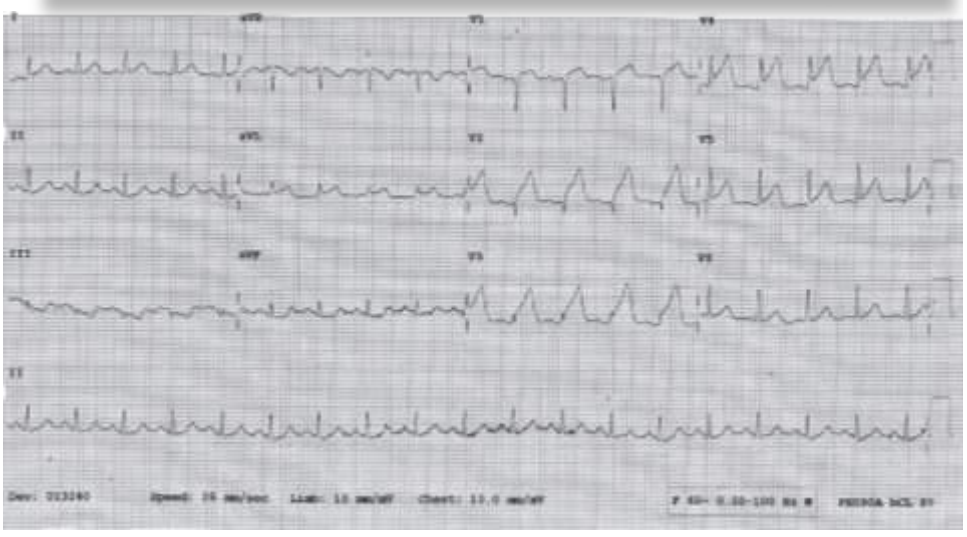
ECG Evolution of a STEMI



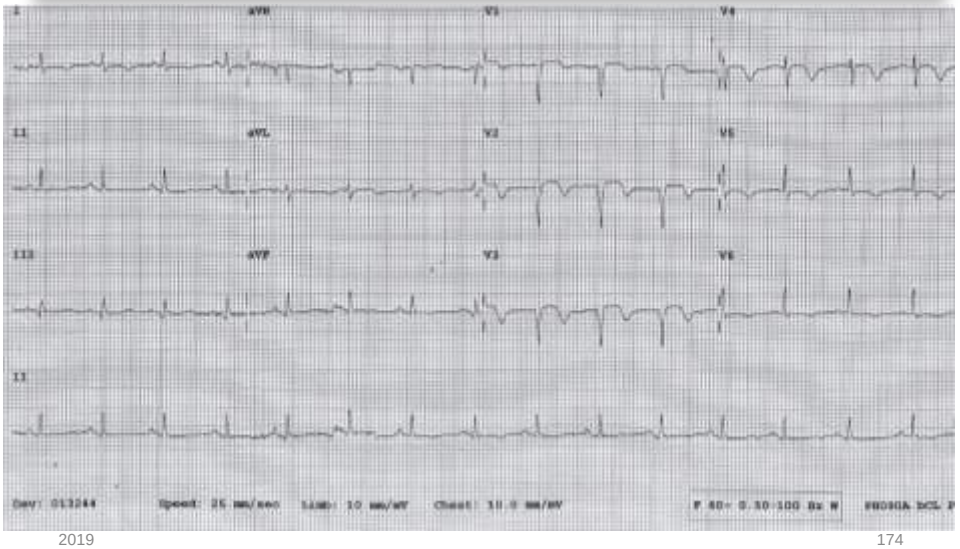
172

Practice ECG 1 of 2



173

Practice ECG 2 of 2



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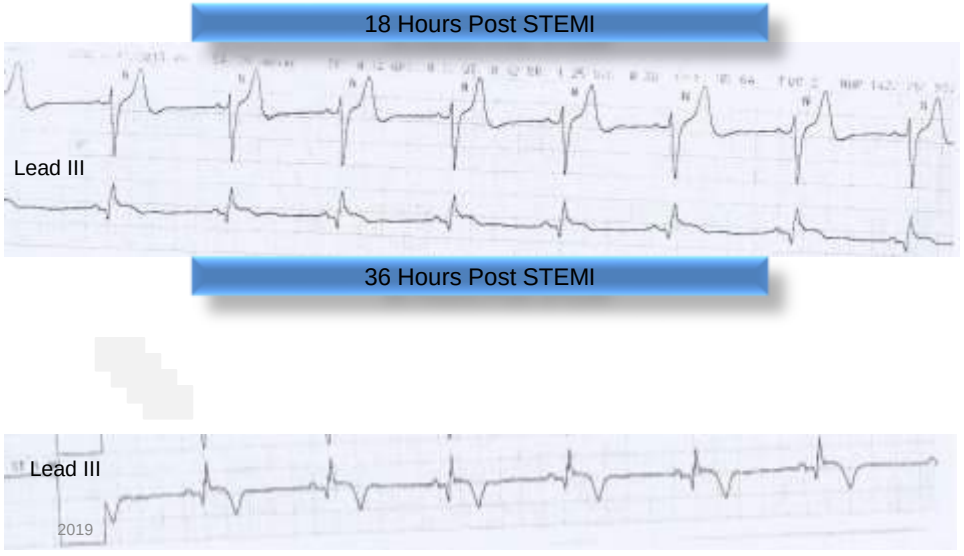
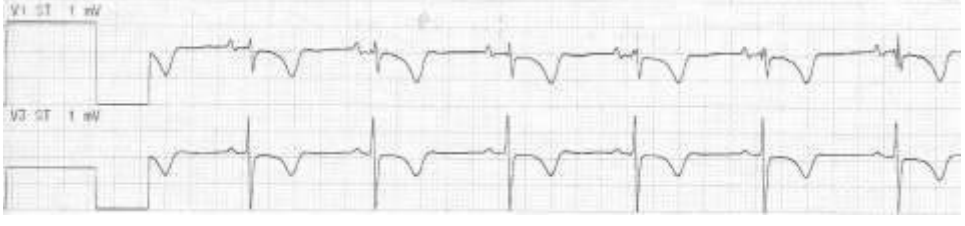
Inferior Posterior STEMI



175

36 hours Post Anteroseptal STEMI



2019 176

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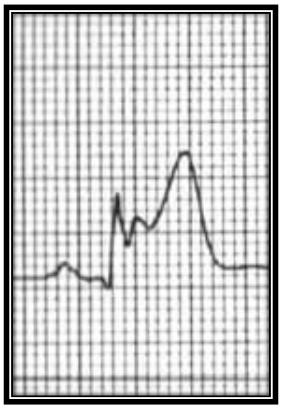
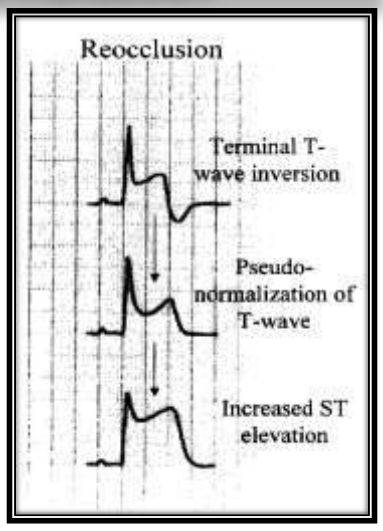
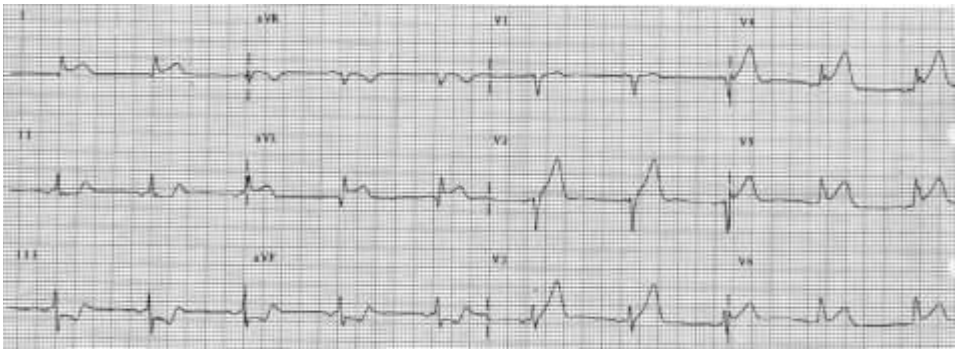
ST Evolution: Pseudo Normalization





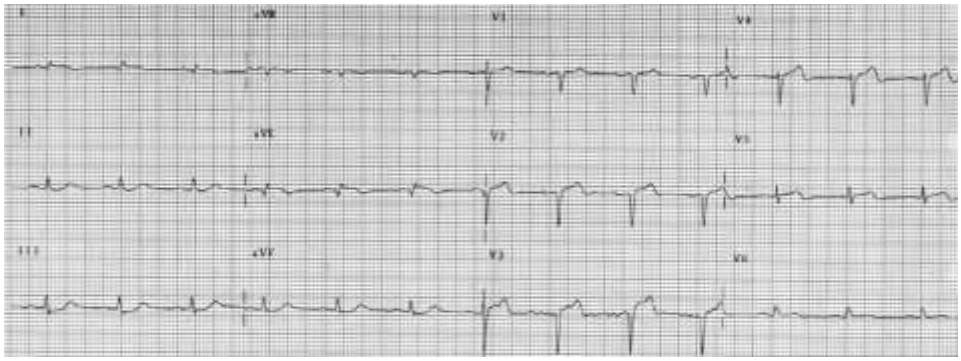
177

Practice ECG 1 of 2



178

Practice ECG 2 of 2



Note: Importance of ST segment monitoring or serial ECGs.

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Interpreting ECG

RECIPROCAL CHANGES

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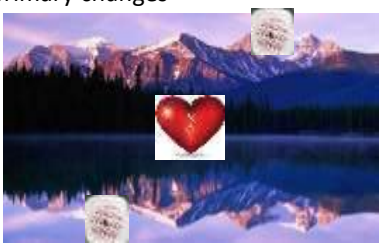
Reciprocal Changes

- Primary Change is most important – look for:
  - ST Elevation: ACS (STEMI)
  - T Wave Inversion: ACS (Non STEMI or UA)
  - ST Depression (ischemia)
- Reciprocal Changes
  - ST segment depression in leads reciprocal (opposite) those with ST elevation
  - Reciprocal changes can help confirm primary changes

Clinical Application:

Before calling ST segment depression ischemia – double check the reciprocal leads for missed ST segment elevation.



181

Lead 1 Left Arm High Lateral Wall	**aVR** Right Arm Posterior Wall	**V1** 4th ICS, RSB Septal Wall	**V4** L MCL, 5th ICS Anterior Wall
Lead 2 Left Leg Inferior Wall	**aVL** Left Arm High Lateral Wall	**V2** 4th ICS, LSB Septal Wall	**V5** L anterior axillary, same level as V_4 Low Lateral Wall
Lead 3 Left Leg Inferior Wall	**aVF** Left Leg Inferior Wall	**V3** Midway Between V_2 & V_4 Anterior Wall	**V6** L midaxillary line, same level as V_4 Low Lateral Wall

2019

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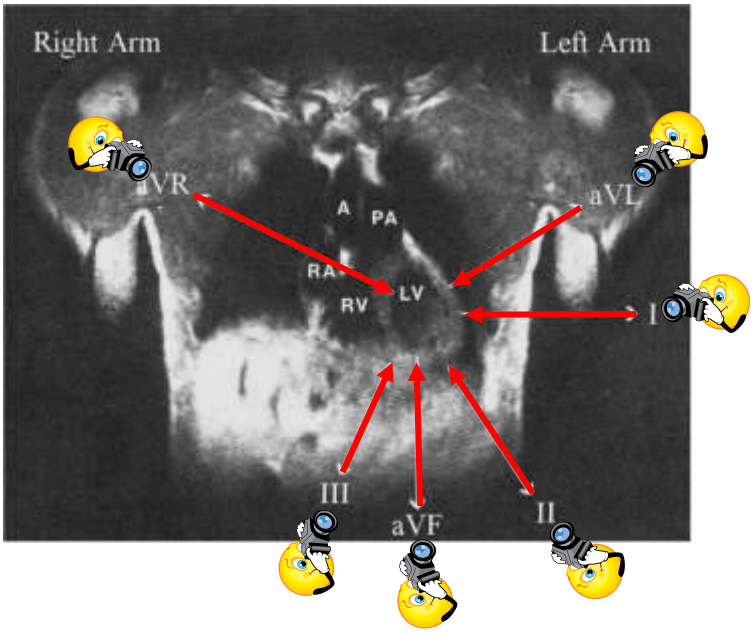
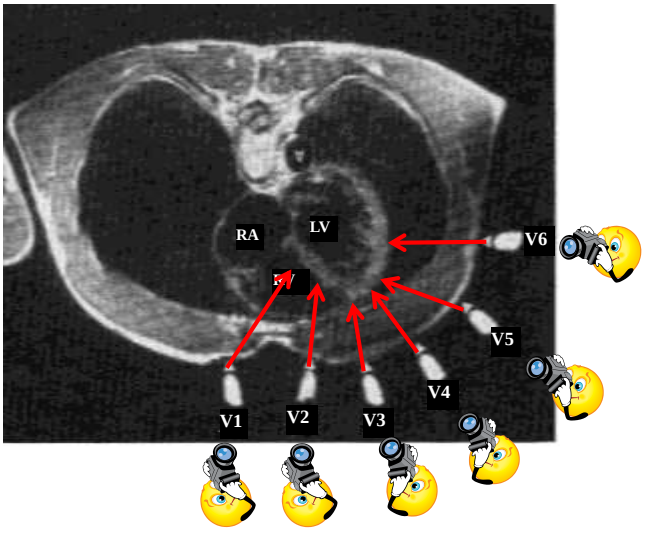


183

6 Precordial (Chest) Leads



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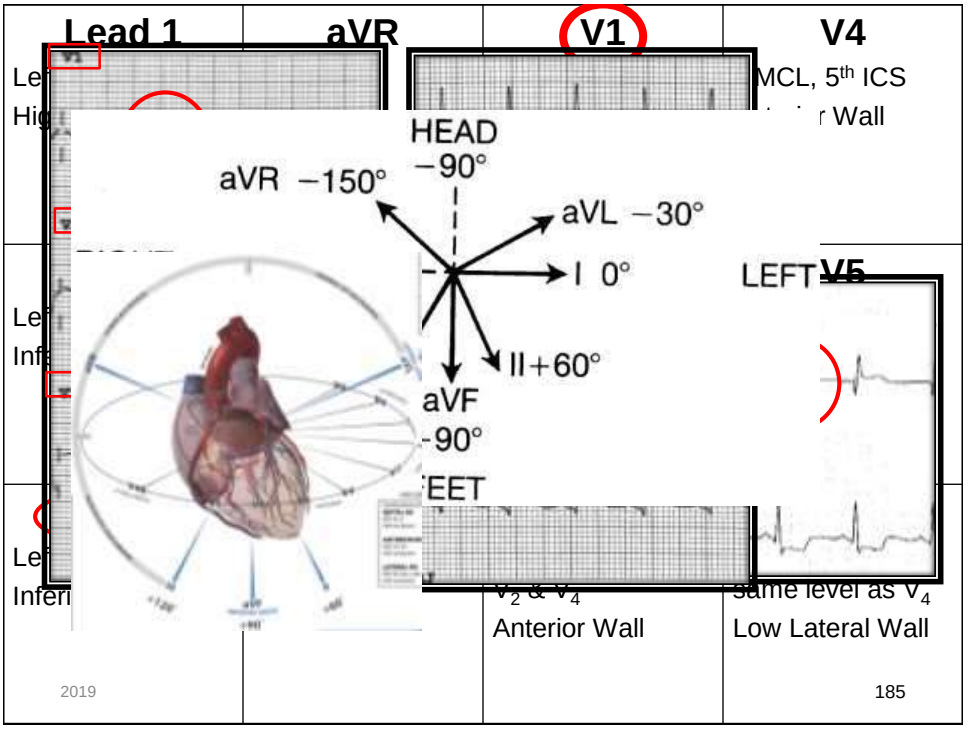
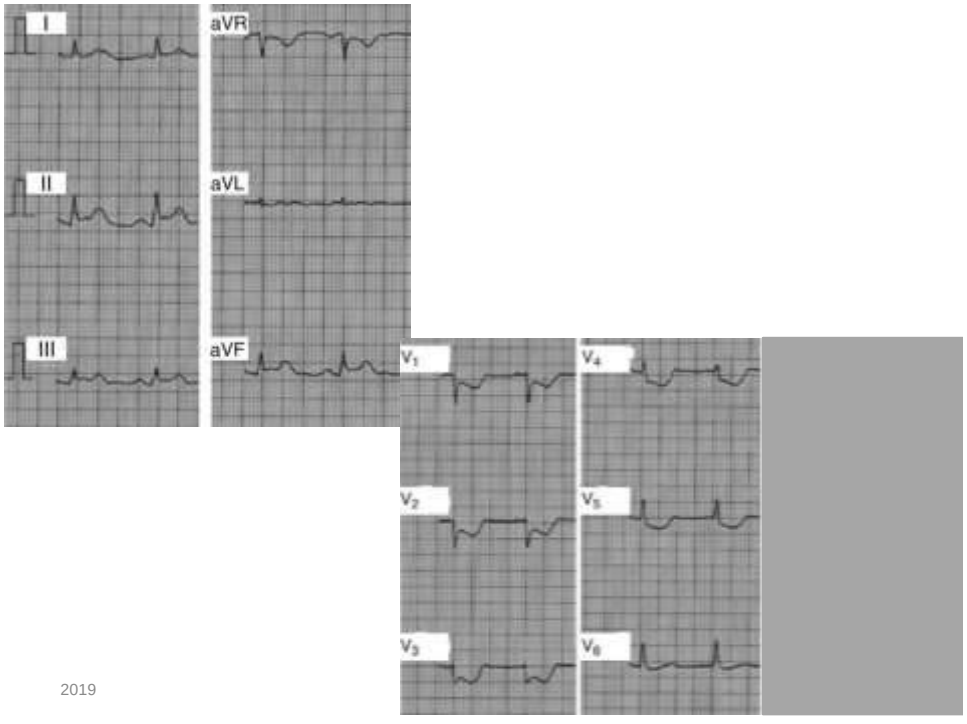
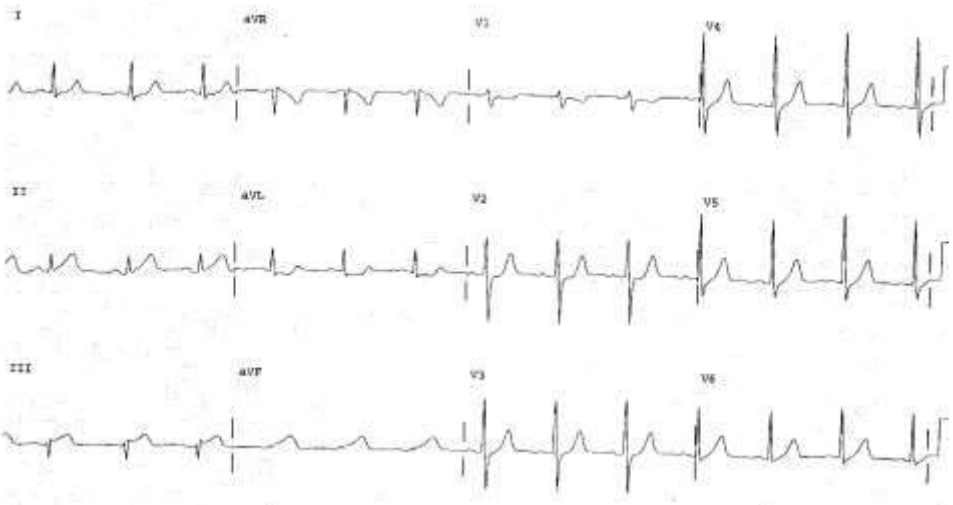

Lead 1
aVR
V1
V4
MCL, 5th ICS
Wall
HEAD
−90°
aVR −150°
aVL −30°
I 0°
II+60°
aVF
-90°
EET
LEFT
V5
Anterior Wall
same level as V4
Low Lateral Wall
2019
185

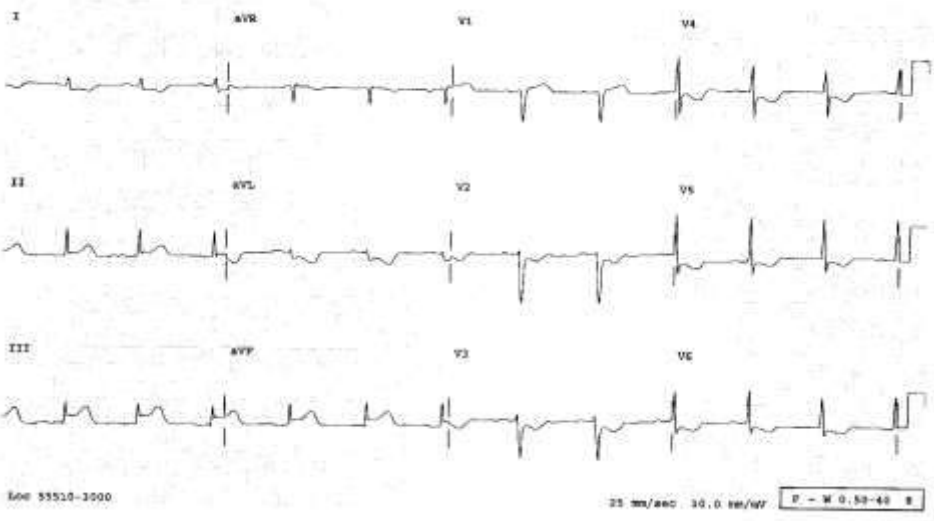
185

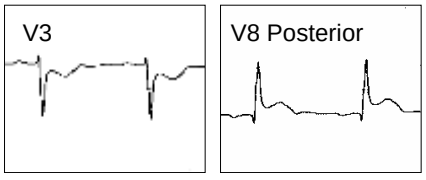

I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
2019

189


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

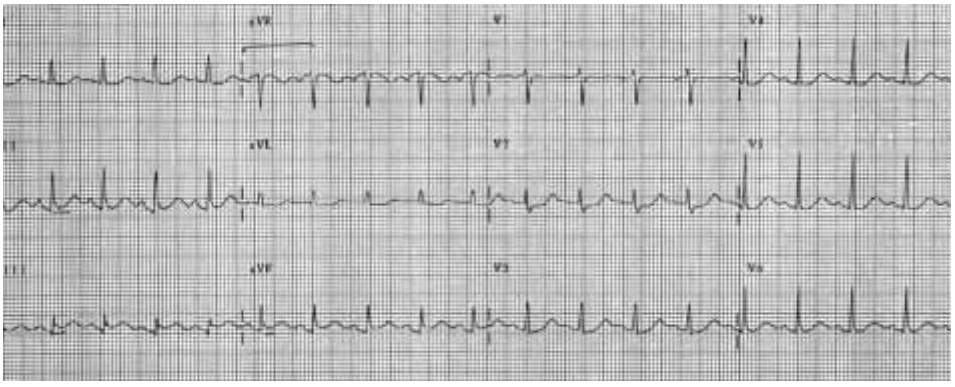
191


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


V3
V8 Posterior

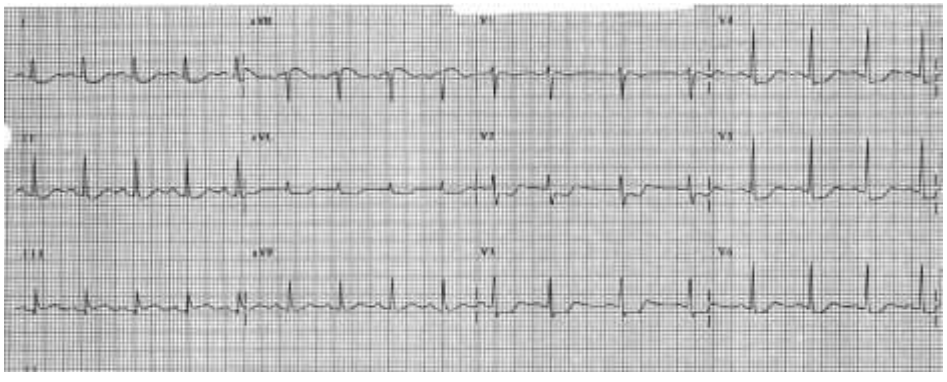
192

ECG 1 of 3



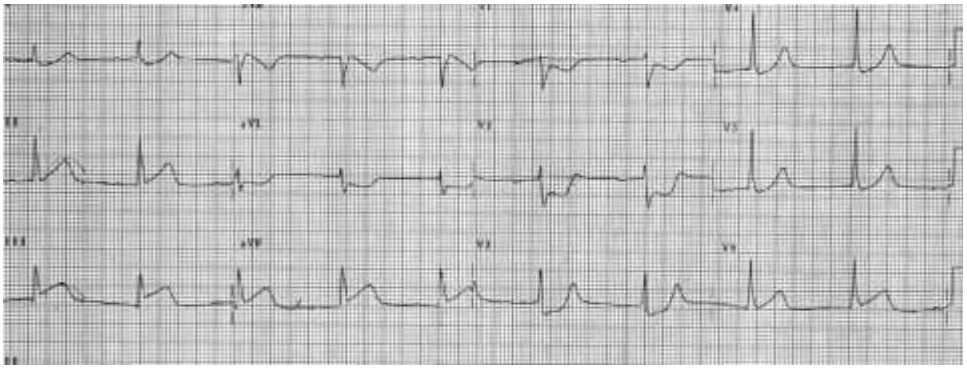
193

ECG 2 of 3



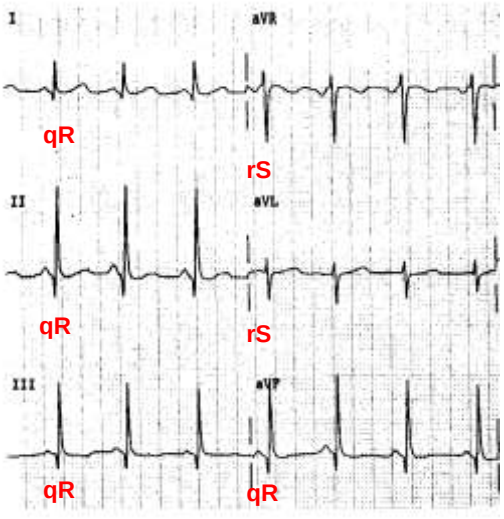
194

ECG 3 of 3



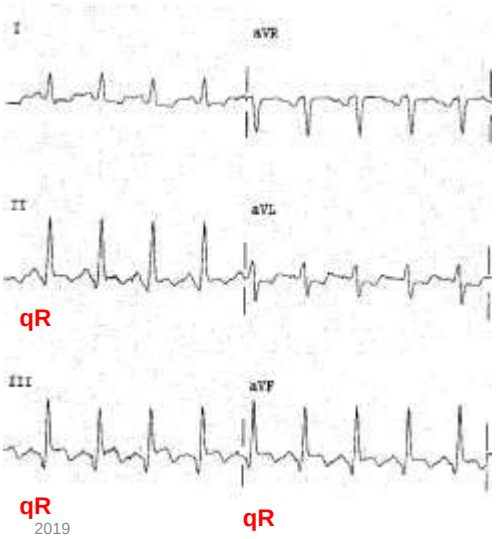
195

Normal Septal Q Waves



196

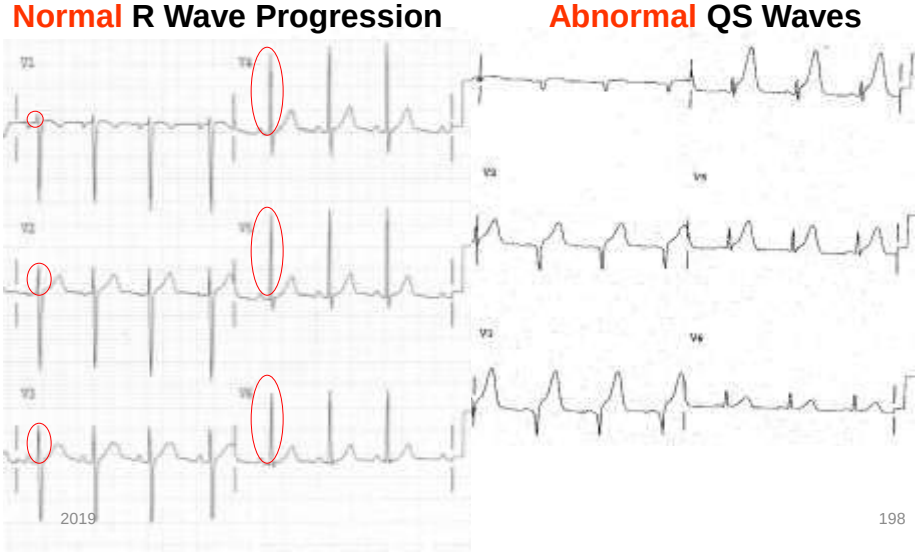
Abnormal QR Waves



- Abnormal Q Waves
  - ≥0.04 sec (1 small box)
  - > 25% of R Wave

197

Abnormal QS Waves



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Q Waves & Diagnosis of AMI

- Q waves in leads with ST elevation
  - Supports diagnosis of STEMI
  - QR Pattern
    - More likely acute
    - Smaller area of infarct
  - QS Pattern
    - More likely old MI or late in course
    - Larger area of infarct
    - May represent ventricular aneurysm
- Q waves in leads without the ST elevation
  - Second MI
  - Higher risk for LV dysfunction

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Q Waves

- Q waves may be present very early in STEMI and should not eliminate reperfusion
  - Q wave or loss of R wave may occur within 1 hour in anterior wall MI
- Q waves may disappear
- STEMI more likely to produce Q waves than NSTEMI
- Q Wave does not necessarily indicate transmural involvement

200

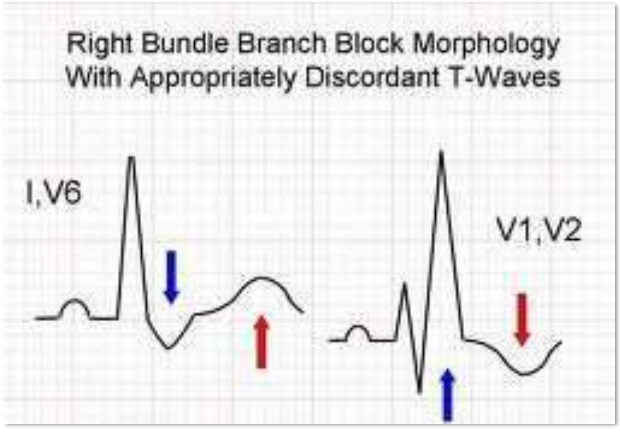
Welcome Back! Let's have a great afternoon.

205

INJURY AND ISCHEMIA WITH BBB

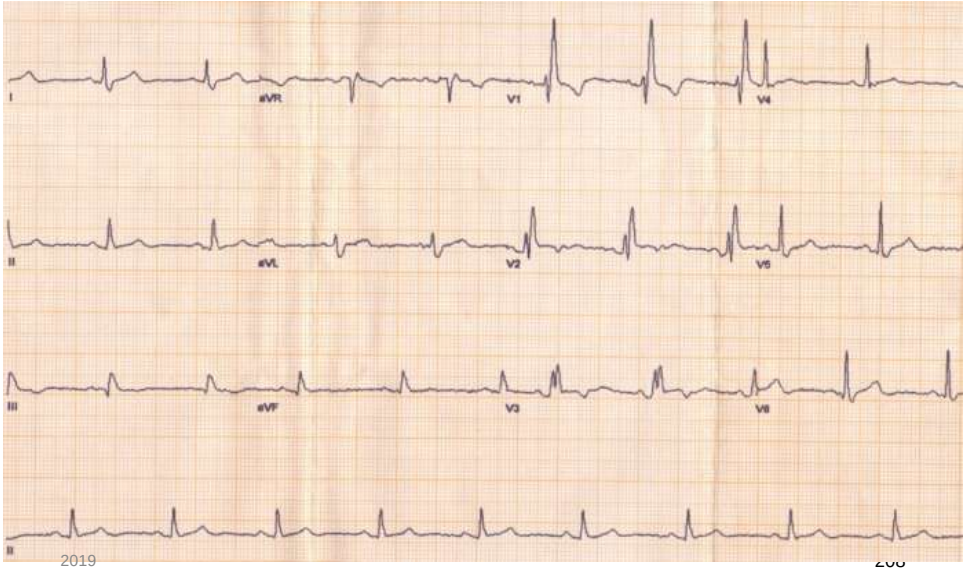
206

NORMAL ST-Twave Changes RBBB

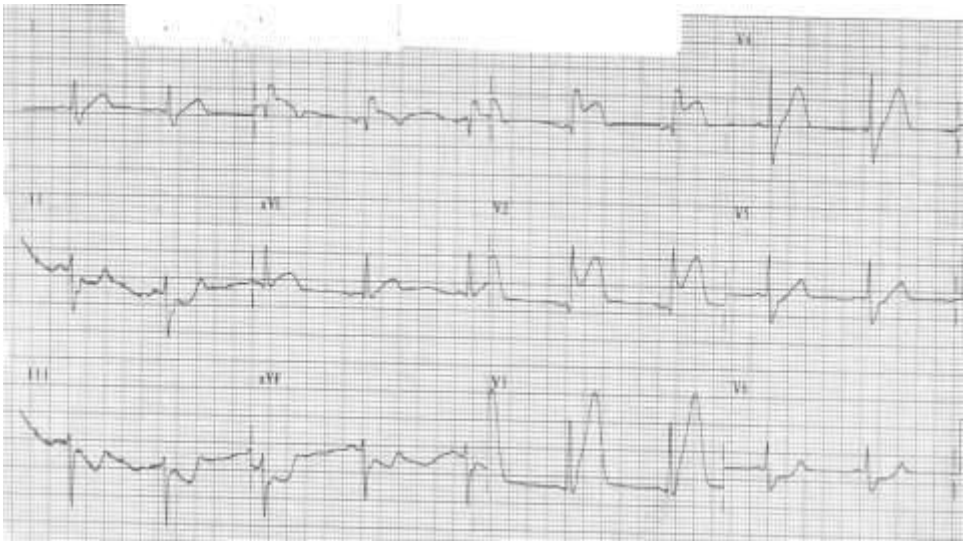


ST should be at baseline.
Presence of any ST elevation or depression in RBBB is abnormal.
Presence of concordance is abnormal.

207



208



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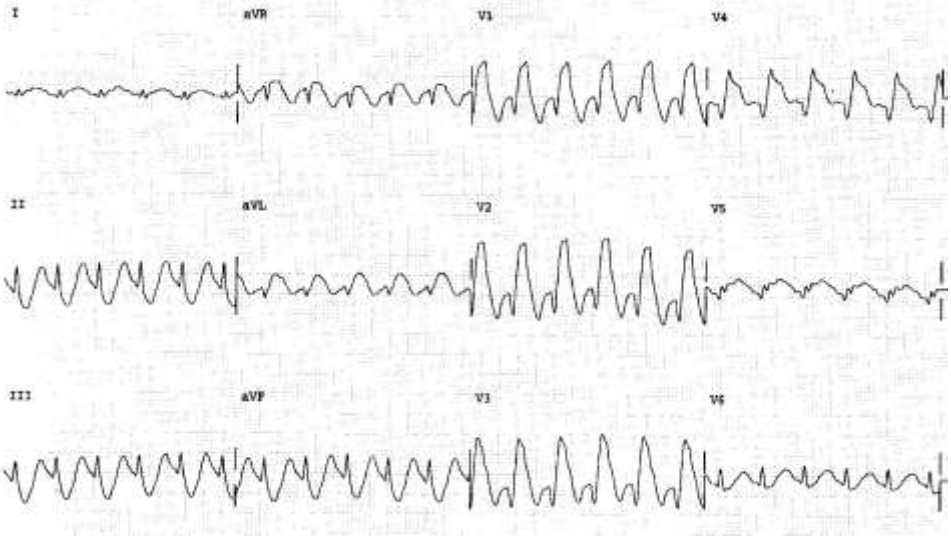
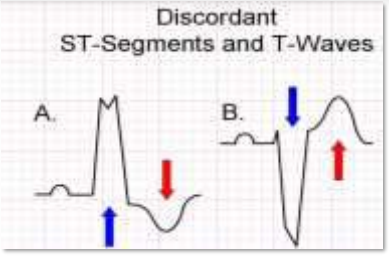


210

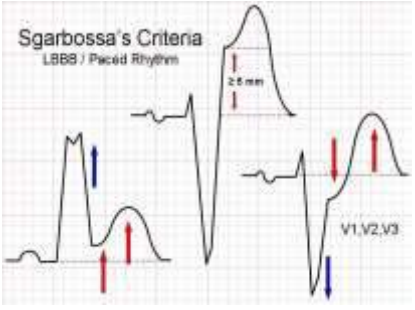
NORAL ST-T wave Changes LBBB and Paced Rhythm

ST usually not at baseline normally in LBBB.

Normal to have DISCORDANCE.



Identifying Infarction in LBBB

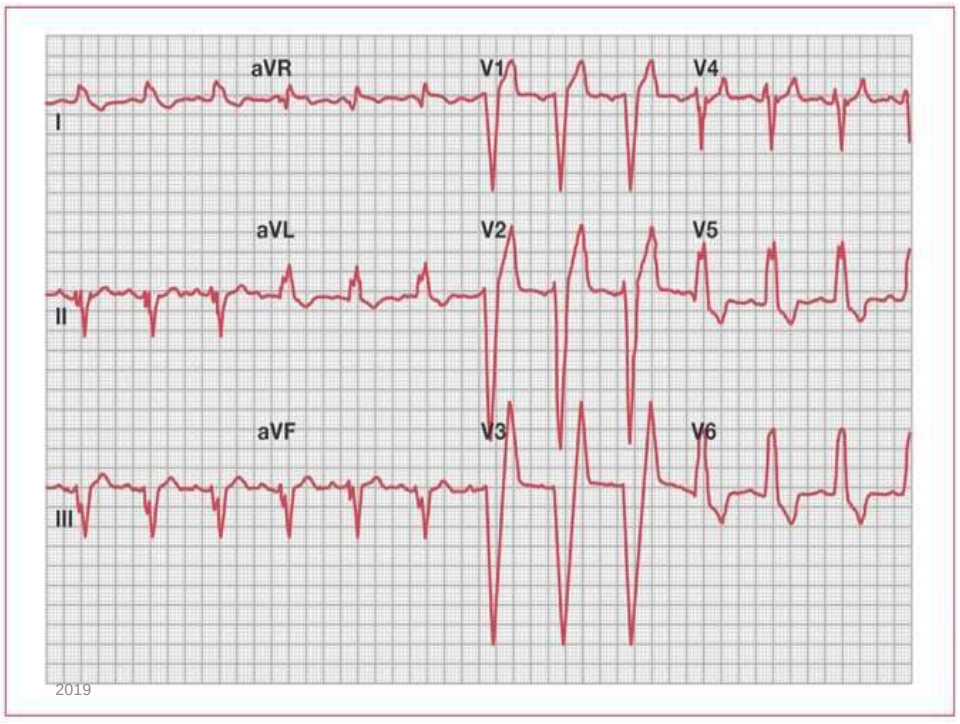


211

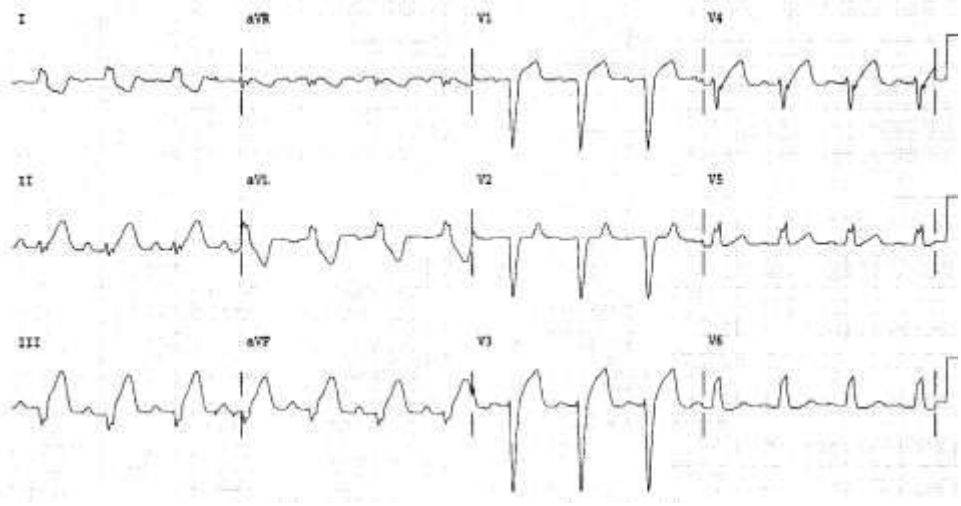
More on New or Presumed New LBBB

- Left BBB is common reason for delayed or withheld reperfusion
- New or presumed to be new LBBB and clinical signs of AMI may be indication for reperfusion therapy
  - Acute LBBB from MI is uncommon
  - LBBB sign of underlying structural heart disease
    - Increased risk of CV mortality
- Old LBBB with increased ST elevation or specific indictors should also receive reperfusion

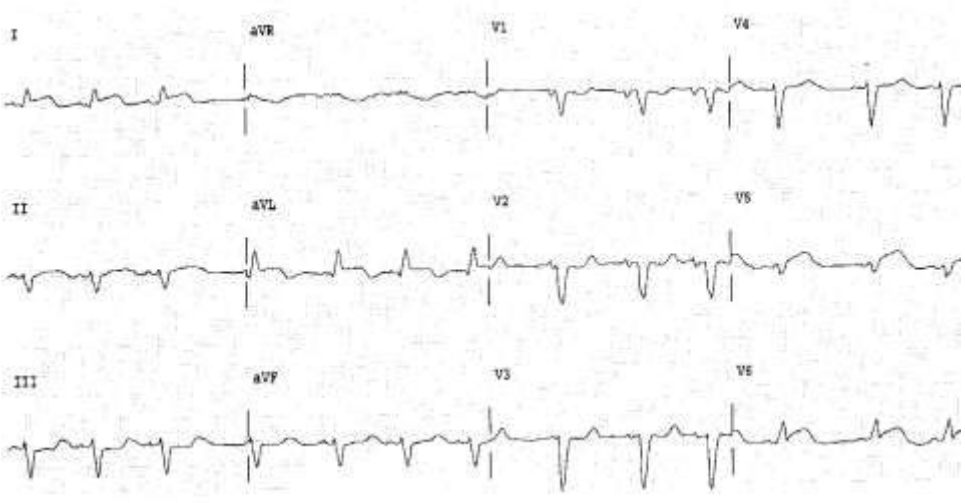
212


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

213


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

214



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USING VECTORS TO DETERMINE CORONARY OCCLUSIONS

216

ST Segment Deviation Vector in Anterior Wall MI

- **Occlusion proximal to first septal and first diagonal branch**
  - Primary area of injury is basal part of LV
  - **ST segment deviation vector points to base (to lead aVR and aVL) and away from apex (inferior leads)**

Septal branch perfuses sub AV nodal conduction system

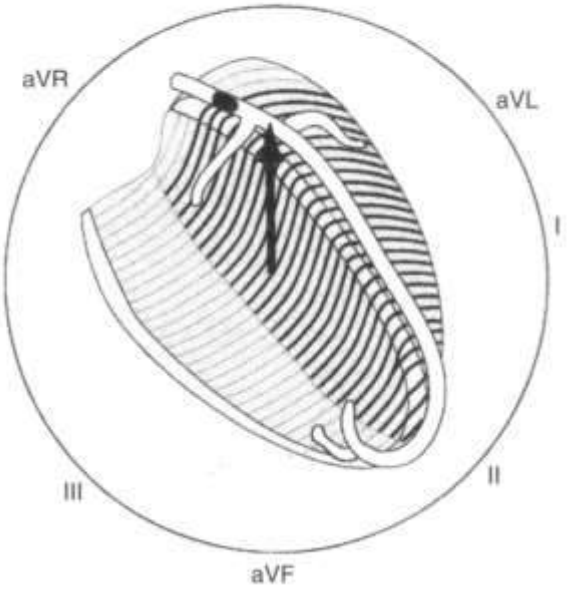
- **Occlusion between first septal and first diagonal branch**
  - Dominant area of injury in high anterolateral area of LV
  - **ST segment deviation vector points toward aVL and away from lead II**

The ECG in Anterior Wall MI

LAD OCCLUSION PROXIMAL TO FIRST SEPTAL AND FIRST DIAGONAL BRANCH

Apart from ST elevation in the precordial leads V_1 to V_4, the frontal ST vector points toward the base of the heart.

- ST elevation in aVR, aVL, and V_1
- ST depression in II, III, aVF, V_5, and V_6
- Acquired right bundle branch block (qR in lead V_1) with or without hemiblock indicates a proximal LAD occlusion.

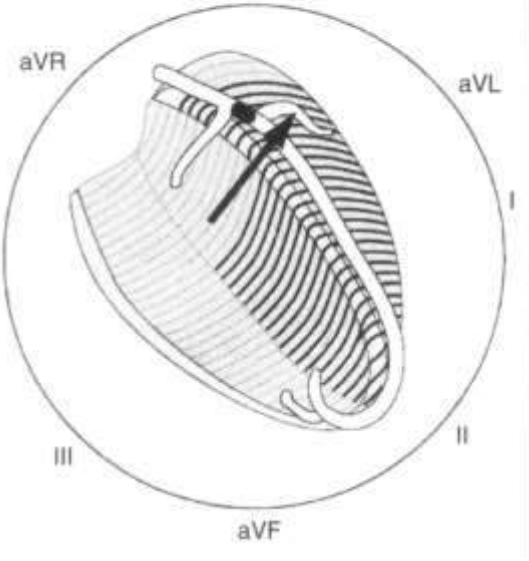


The ECG in Anterior Wall MI

LAD OCCLUSION BETWEEN THE FIRST SEPTAL AND FIRST DIAGONAL BRANCH

Apart from ST elevation in precordial leads V_2 to V_5, the frontal ST vector points toward aVL and away from III.

- ST elevation in I and aVL
- ST depression in III
- ST isoelectric in II



ST Segment Deviation Vector in Anterior Wall MI

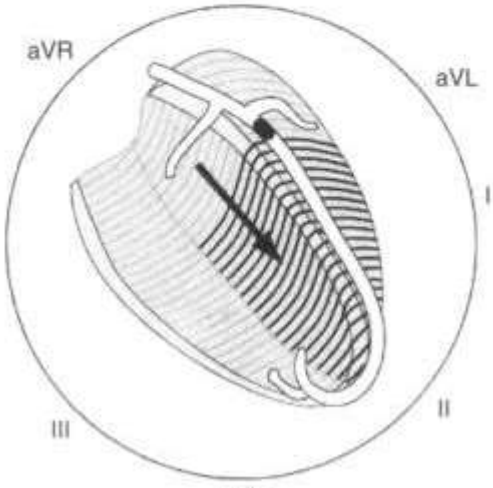
- **Occlusion distal to the diagonal branch**
  - **ST segment deviation vector points to apex**
    - **ST elevation in inferior leads (II > III)**
- **Left Main Coronary Artery Occlusion**
  - **ST segment deviation vector points to lead aVR**
    - **ST elevation in aVR**
    - **Less ST elevation in V1**
    - **ST depression in leads V2 – V6 (posterior wall injury) – circumflex occlusion**

The ECG in Anterior Wall MI

DISTAL LAD OCCLUSION

Apart from ST elevation in V_2 to V_6, a frontal ST deviation vector points in an apical direction.

- ST elevation in the inferior leads with lead II greater than lead III



2019

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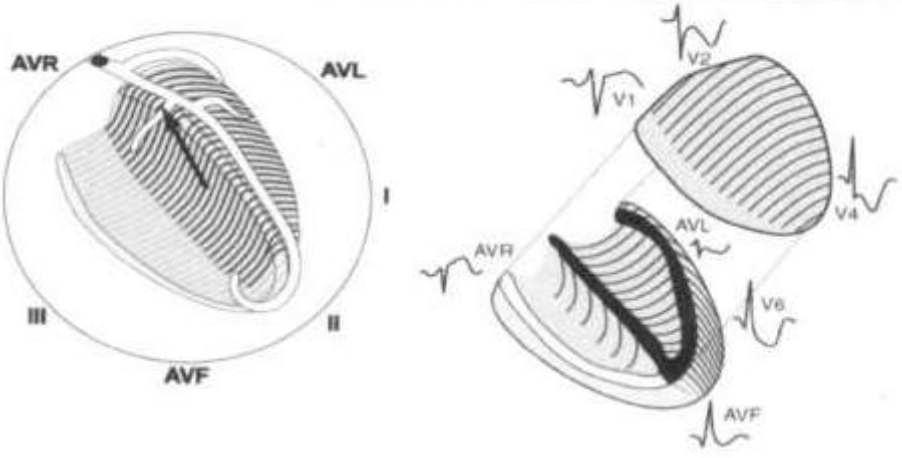
The ECG in Left Main Occlusion



The combination of acute ischemia in the LAD and CX region results in a frontal ST deviation vector pointing toward aVR and a horizontal ST deviation vector directed away from the posterior wall.

2019

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Limitations of ST Segment Deviation Vector Analysis

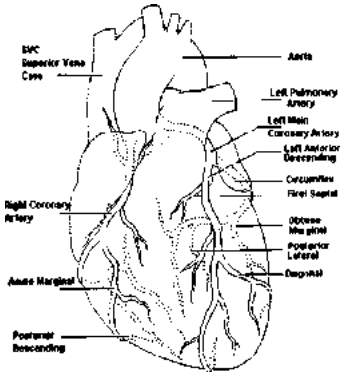
- Previous MI / preexisting ST / T wave abnormalities
- Multi vessel disease
- Altered ventricular activation
- Dominant or small coronary arteries
- Coronary artery anomalies

Specific Types of MIs

Nuances

Anterior MI

- Proximal LAD
  - Proximal to first diagonal
    - Anterolateral
  - Proximal to first septal perforator
    - Anteroseptal
- Mid LAD
  - Anterior MI



LAD: Anterior Wall, High Lateral Wall, Septum

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Complications of Anterior MI

- Myocardium at risk
- Mortality and morbidity
- Post Infarction ejection fraction

Urgency for Reperfusion!

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Complications of Anterior MI

- Tachycardia
  - Sinus tachycardia
  - Atrial tachycardia
  - Ventricular tachycardia
- Right BBB and left anterior hemiblock
- Complete heart block
- Ventricular septal defect
  - New loud systolic murmur
- Cardiogenic shock
- Long term ventricular modeling and heart failure

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ECG Changes: Anterior MI

- **ST Elevation**
  - **V_3, V_4 Anterior Wall**
  - V_1, V_2 Septum

Various locations in the LAD will affect ST deviation vector with varying results on ST segment changes in the limb leads.

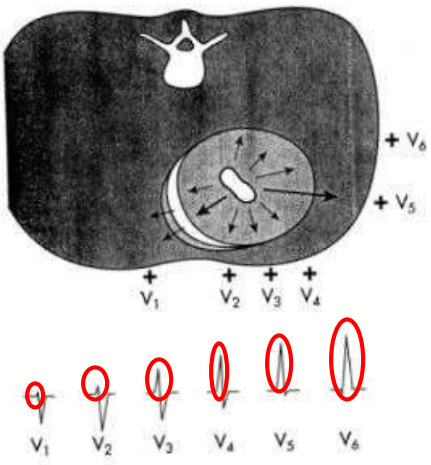
For Example: Occlusion proximal to diagonal branch results in ST elevation in Leads I and aVL.

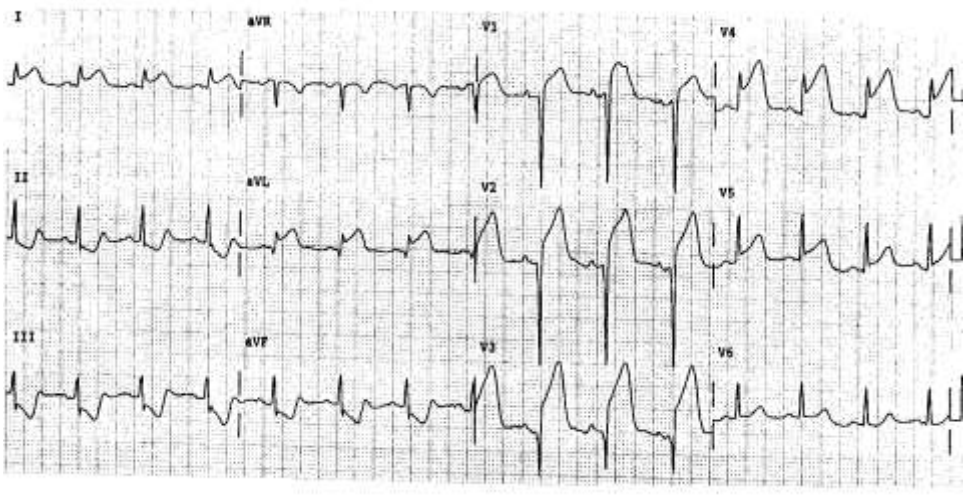
For Example: Occlusion proximal to septal perforator shifts vector to right and results in additional ST elevation in Lead aVR.

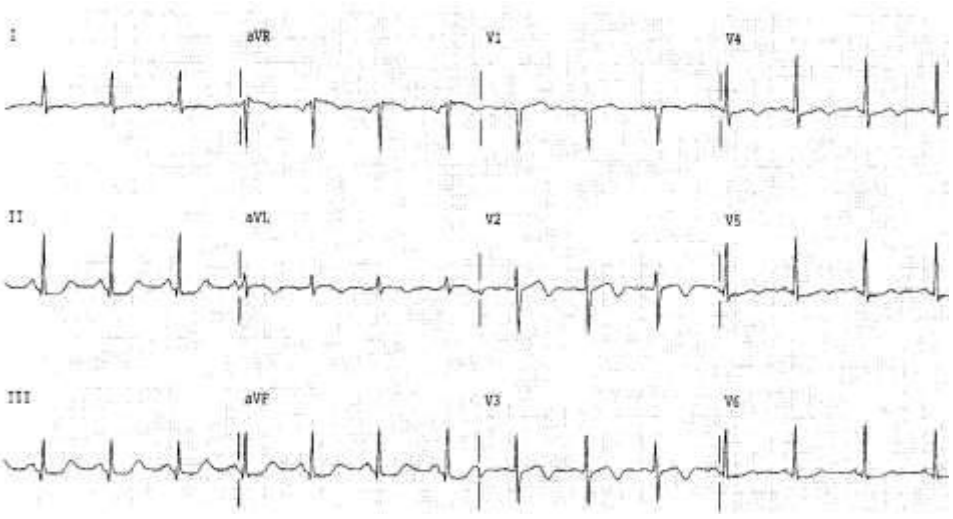
228

Normal V1-6: R Wave Progression

- The R wave becomes taller and the S wave becomes smaller as the electrode is moved from right to left
- This pattern is called R wave progression







231

Lateral Wall MIs

- Isolated MI frequently missed
- ST elevation may be < 1 mm
- ST elevation may only be in aVL
- Myocardium at risk and mortality benefit

- Lateral Wall
  - First diagonal branch of LAD
  - Obtuse marginal branches of circumflex
  - Note: Circumflex occlusion produces ST segment elevation on standard ECG less often than RCA or LAD occlusions

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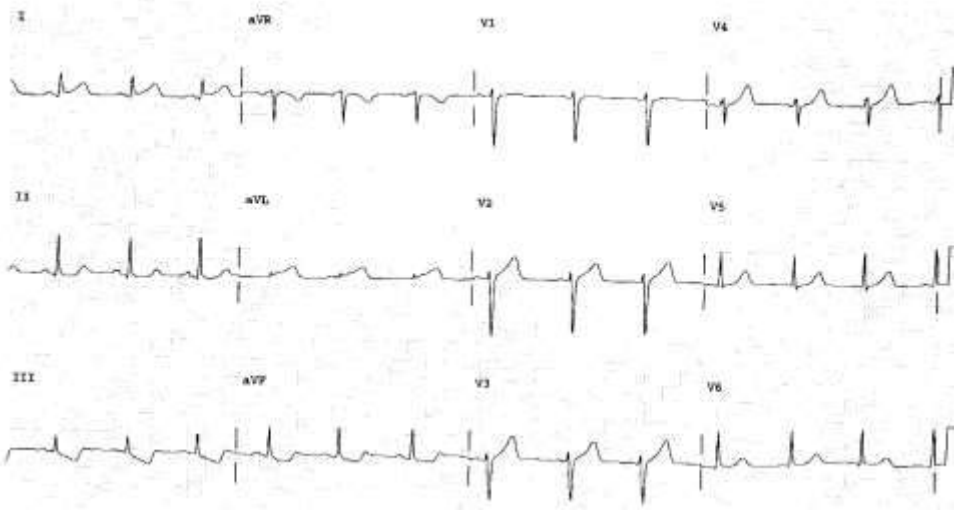
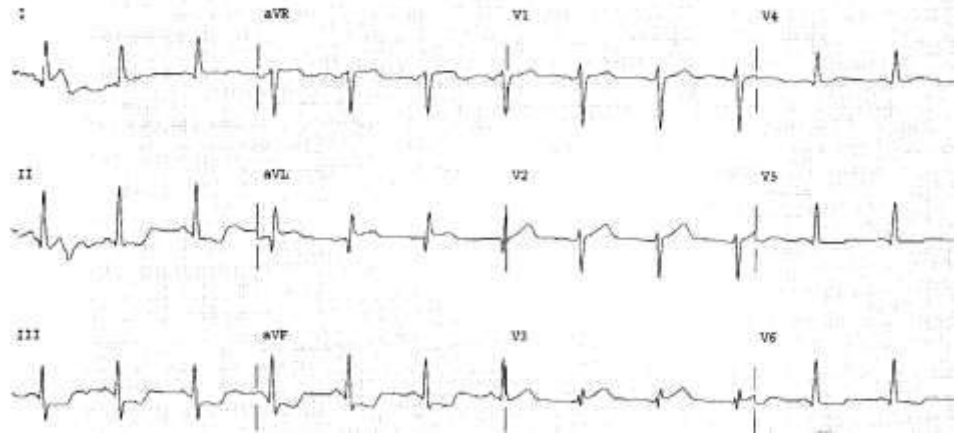

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

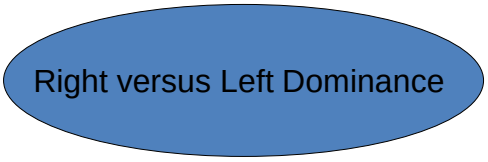
233


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

234

Inferior MI

- Proximal RCA = Inferior LV + RV
- Occlusion proximal to the marginal branch = Right ventricular MI
- Occlusion of posterior descending artery = Posterior MI



Inferior MIs

- Variations
  - Inferior posterior
  - Inferior with RV
  - Inferolateral (often with circumflex)
- Complications
  - Sinus Bradycardia, 2nd Degree HB Type I
  - Increased parasympathetic activity
  - Papillary muscle rupture with posterior wall involvement
  - RV failure with RV involvement

ST Changes in Inferior MI

- ST elevation leads II, III, aVF
  - Lead III > II (RCA occlusion)
  - Lead II > III (Circumflex occlusion)
- *ST depression in aVL*
- ST elevation ≥ 0.5mm in inferior leads should be considered abnormal until proven otherwise

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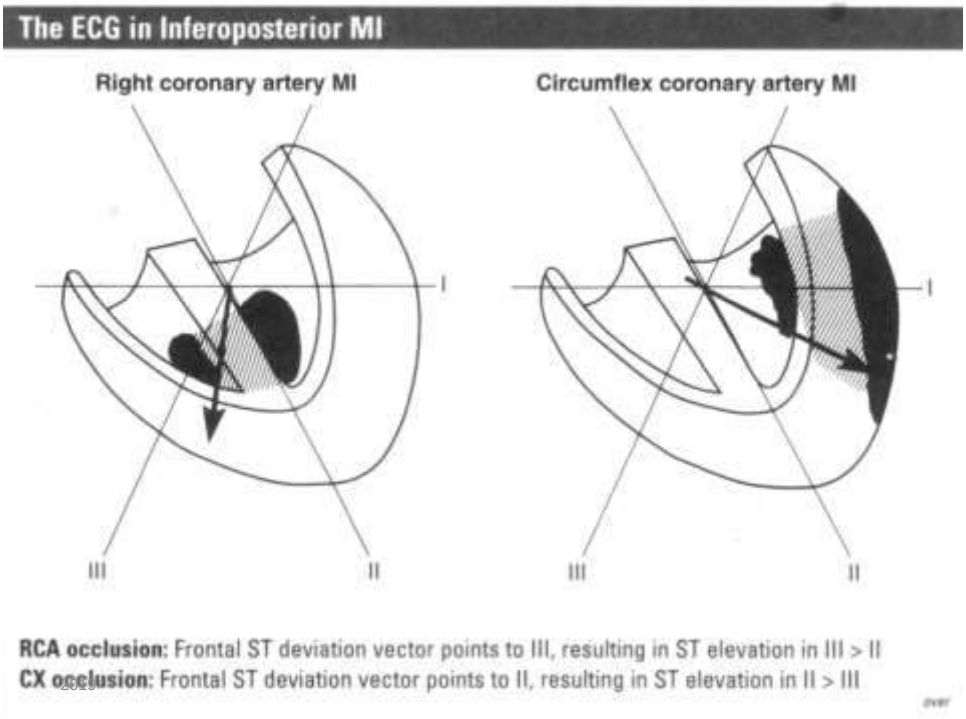
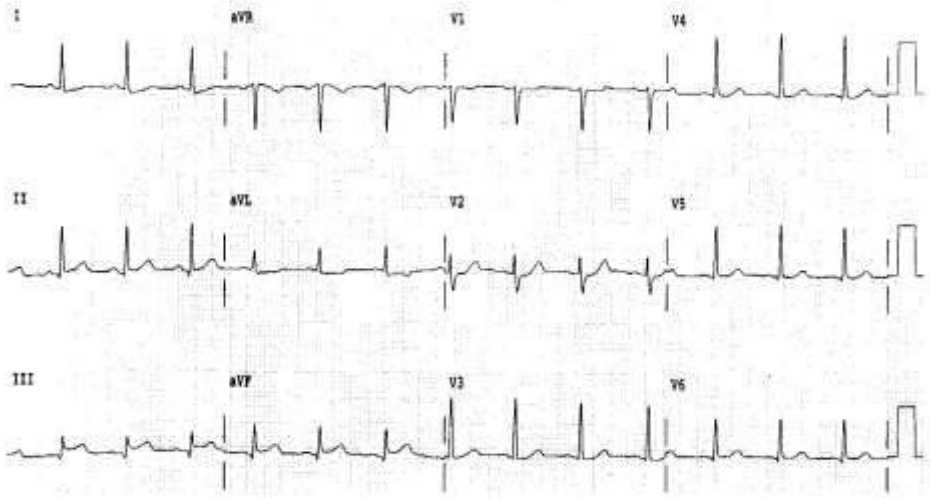
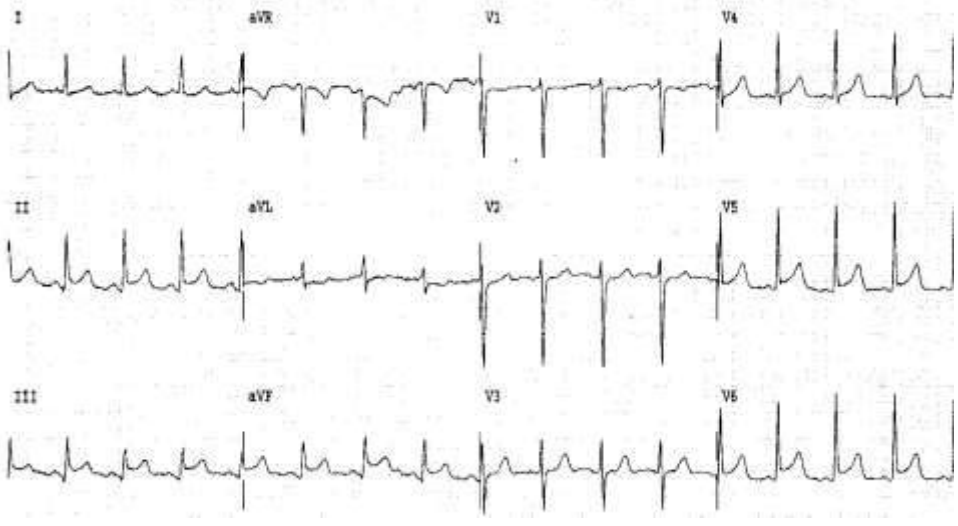


238


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

239


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

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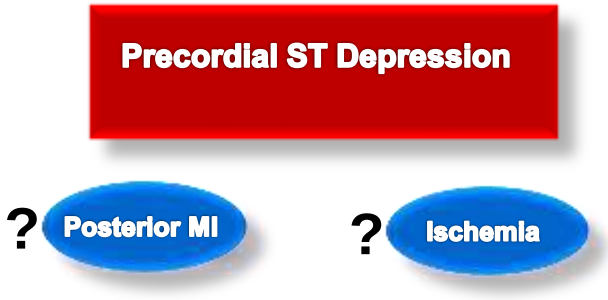
Posterior MI

- Note: MRI studies suggest that ECG evidence of posterior injury may actually reflect more anatomical injury of the lateral wall. However, ECG interpretation guidelines recommend we continue to refer to this type of MI as posterior.
- Coronary arteries and the posterior wall
  - RCA (responsible for Inferior / Posterior STEMIs)
    - Posterior descending coronary artery
  - Circumflex (responsible for isolated posterior STEMI)
    - PDA
    - Marginal Branch



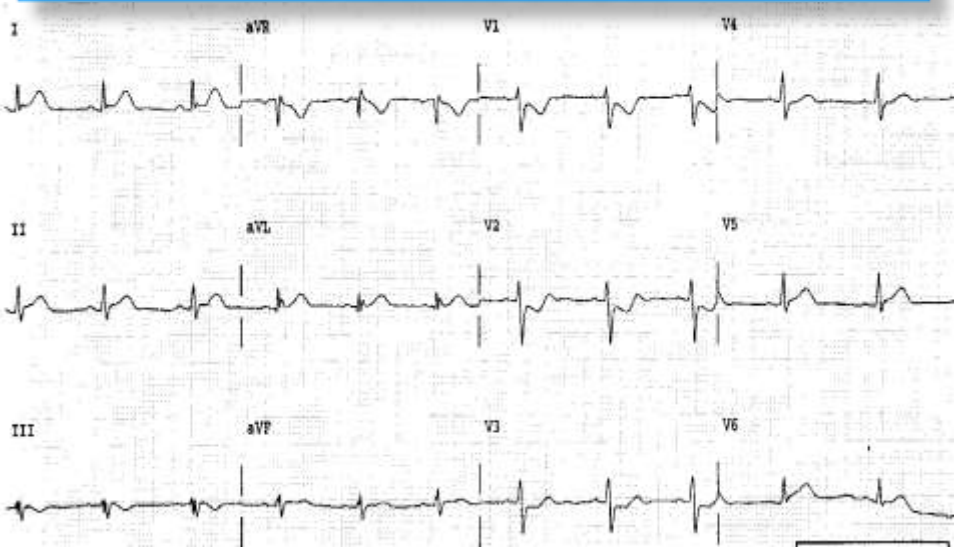
- Maximal ST depression ≥ 2 mm in V1 – V3 may be 90% specific for posterior MI
- T waves usually remain upright
- Persistent ST depression is more commonly due to posterior STEMI than LAD disease…….
- Anterior UA/ NonSTEMI is most likely when T wave inversion in present in V1-V4

Posterior MI

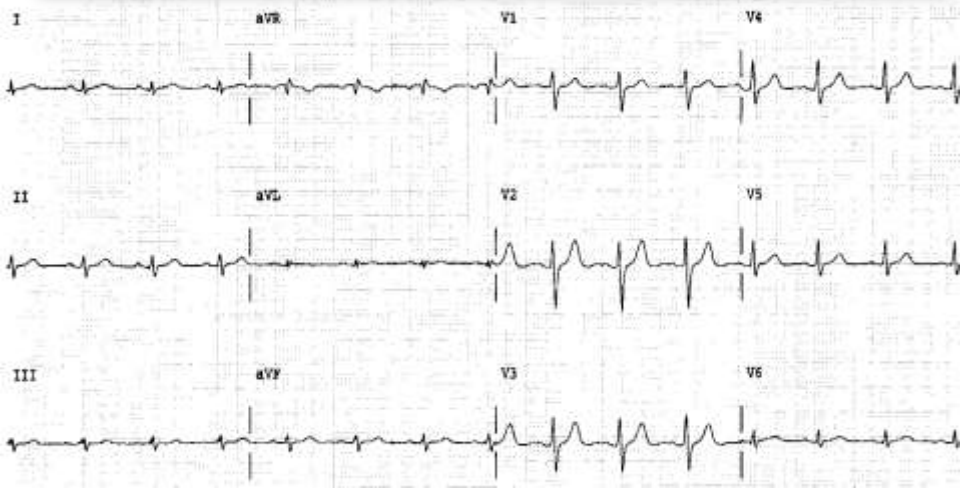
- Reperfusion is indicated if true posterior MI is confirmed
  - Even with absence of ST elevation on standard 12 lead ECG
  - 3.3% to 8.5% of MIs are isolated posterior STEMI (Smith et al., 2002)
- Non reperfused posterior MI
  - Tall R waves in V1-V3

Remember: Increased risk for papillary muscle ischemia or rupture. May hear new holosystolic murmur.

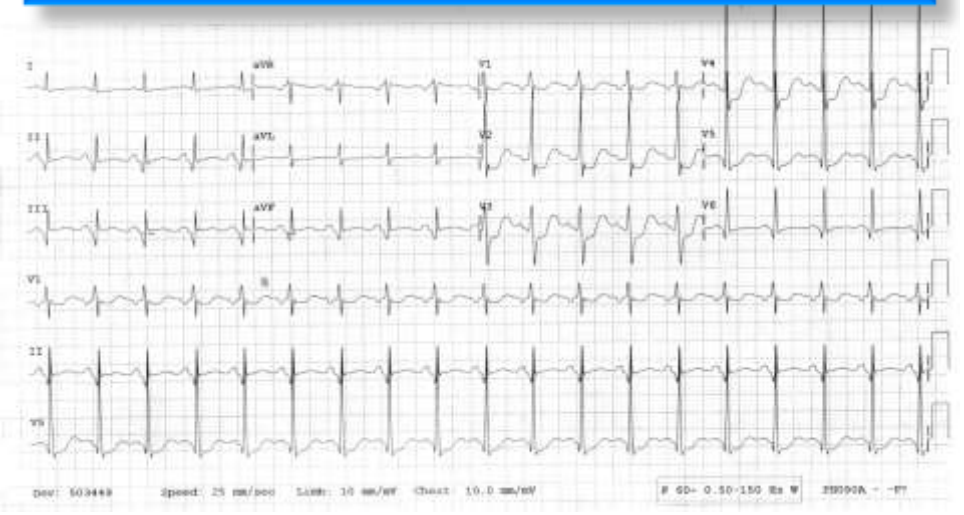
Practice ECG A



Practice ECG B



Practice ECG



RV Infarct

- Approximately 1/3 of Inferior MIs
  - Occlusion proximal to marginal branch of RCA

Recognition

- > 0.5 mm ST elevation in V4R
  - Men < 30 years of age (> 1.0 mm ST elevation in V4R)
- Suspect when elevation in V1 but not in V2
  - However, cannot rely on if there is simultaneous RV and posterior involvement
- Combination of elevation in V4R and V1 is very specific to RV infarct
- Reciprocal changes to RV injury may be seen in low lateral leads

RV Infarct

- Implications
  - Increased short term mortality
  - RV can recover well if patient survives acute phase
- RV Failure = Decreased LV Preload = Decreased Stroke Volume = Hypotension
- Clinical presentation
  - Hypotension
  - Clear lungs
  - Signs of RV failure (increased CVP; jugular venous pressure)

RV Infarct

- Pathophysiology of RV infarct is complex
  - Ventricle can be noncompliant or distensible
- Treatment of hypotension in RV infarct
  - Avoid diuretics and venous vasodilators
  - IV fluids to CVP of 15 mmHg and PAOP of 15 mmHg (Brenner & Tschopp, 2009)
  - Caution with too much fluid:
    - Distendability
    - Septal displacement
  - Need for inotrope when fluid loading not successful
  - Atrial fibrillation and bradyarrhythmias need treated

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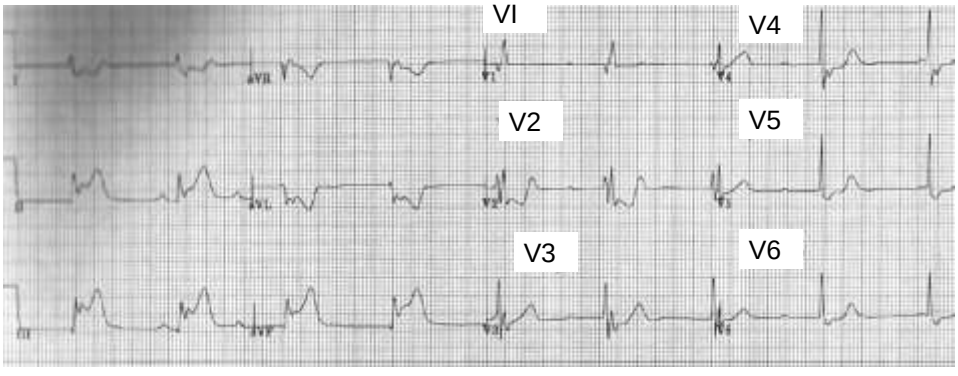
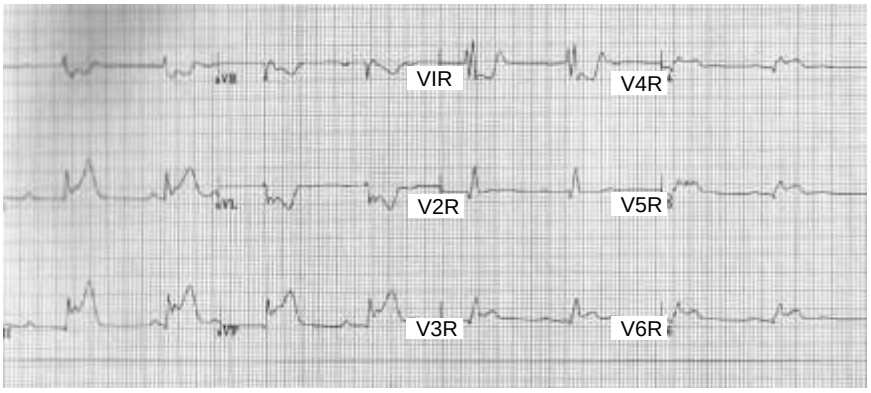
Practice ECG 1 of 2



250

Practice ECG 2 of 2



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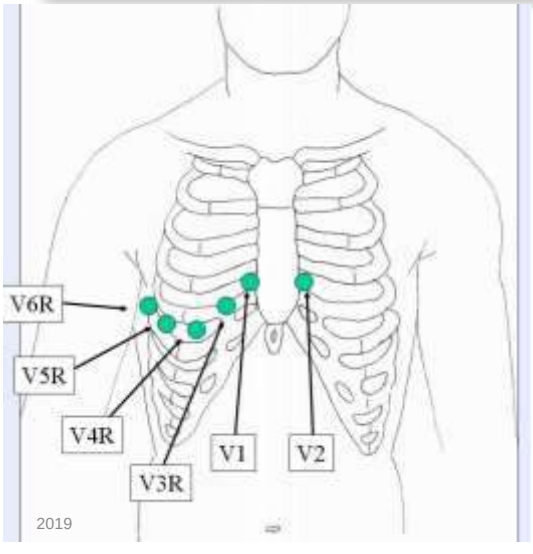
Right Sided ECG



Note: V1 and V2 may also be reversed with V1 on LSB and V2 on RSB.

If so, these are labeled V1R and V2R.

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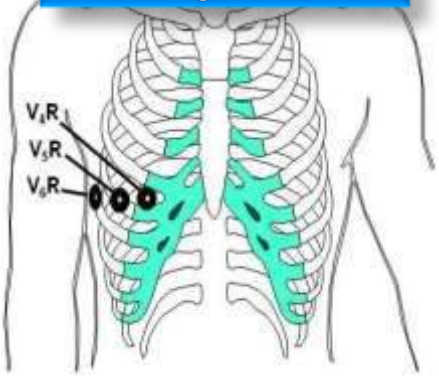
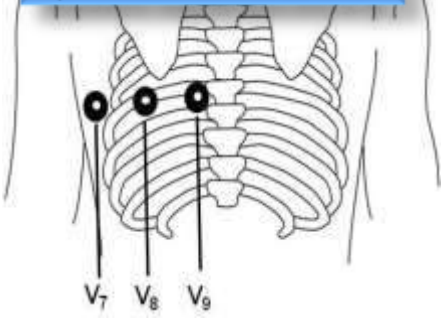
Right Ventricular Leads

May also record V_3R, V_4R, and V_5R. Mirror image of normal



Posterior Leads

V7: Posterior axillary line
V8: Under tip of scapula
V9: Same level at paraspinal border
All posterior leads the same level as V6



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Labeling the Additional Leads

Note: Verify that electronically recorded 12 leads are able to labeled correctly.

Original Chest Leads	New Chest Leads
V1	V4R
V2	V5R
V3	V6R
V4	V7
V5	V8
V6	V9

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Right Sided and Posterior Quick Look on Bedside Monitor

Right Sided Lead

- Place electrode in V4R Position
  - 5th ICS Right MCL
- Attach V monitoring lead (Brown Lead) to electrode
- Assure monitor lead selector is on V
- Run strip and clearly mark "V4 Right Chest Lead"

Posterior Lead

- Place electrode in V8 position
  - Under tip of left scapula same level as V6
- Attach V monitoring lead (Brown Lead) to electrode
- Assure monitor lead selector is on V
- Run strip and clearly mark "V8 Posterior Lead"

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Patient post cardiac arrest – on hypothermia.

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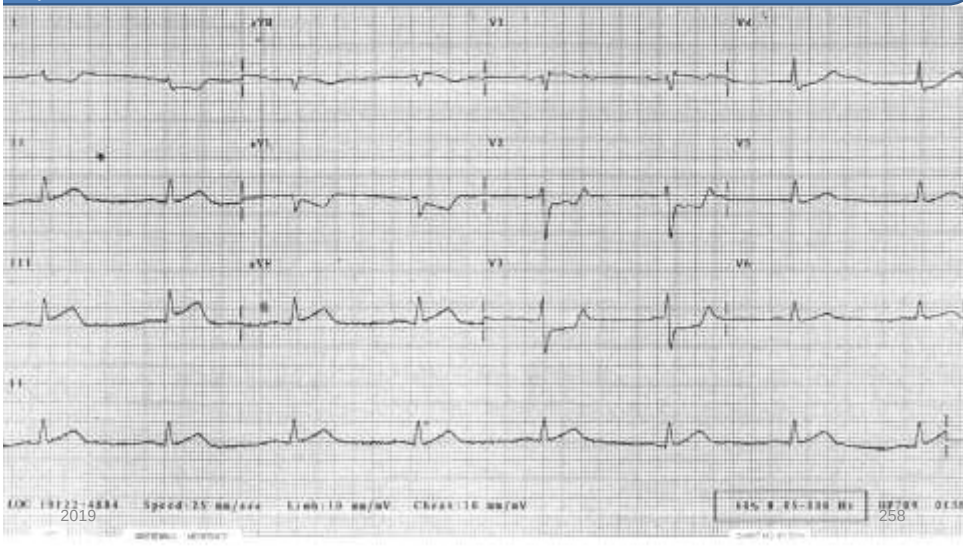
- Inferior STEMI: ST Elevation in II, III, aVF
- Reciprocal depression in Leads I and aVL
- ST Depression in V2 and V3: Reciprocal changes from Posterior STEMI
- Ideal patient for a 16 lead ECG to assess for injury to the right ventricle and posterior wall of the left ventricle.



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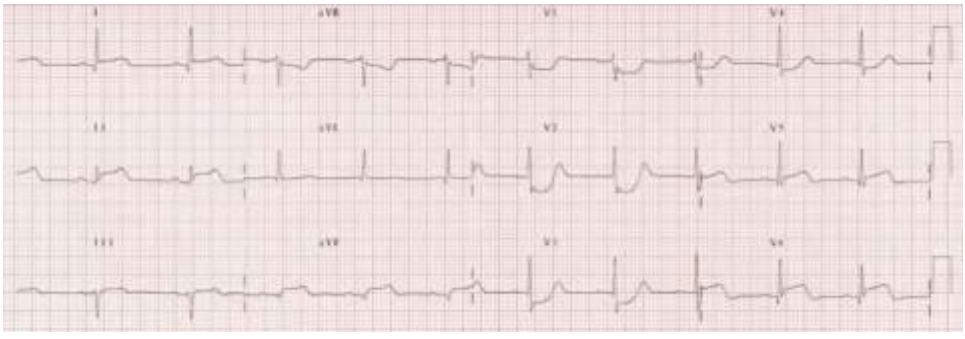
<u>Same Patient as Previous 12 Lead</u>:
Due to hypotension the point of care nurse used the V lead from bedside monitoring to record a V4R lead. This recording confirms RV injury and this knowledge was used to guide treatment.

Patient post cardiac arrest – on hypothermia.

ST segment elevation in lead V4R demonstrates high risk for high grade AV node block
(Wellen & Conover, 2006).

259



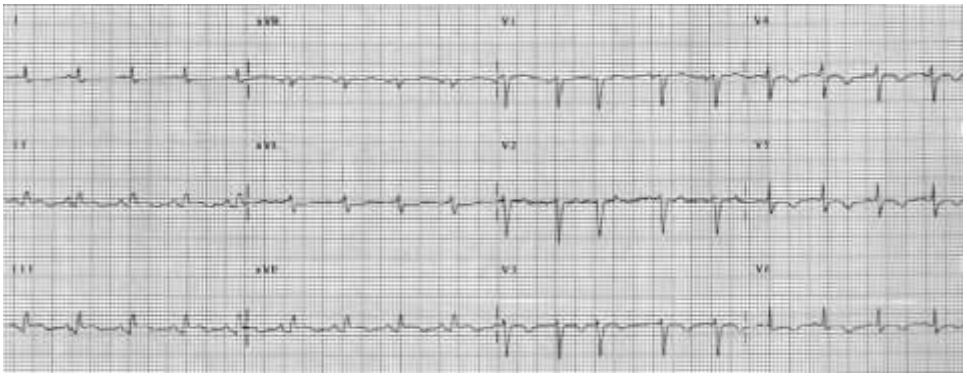
261

We become what we repeatedly do!!

Practice, Practice, Practice !!

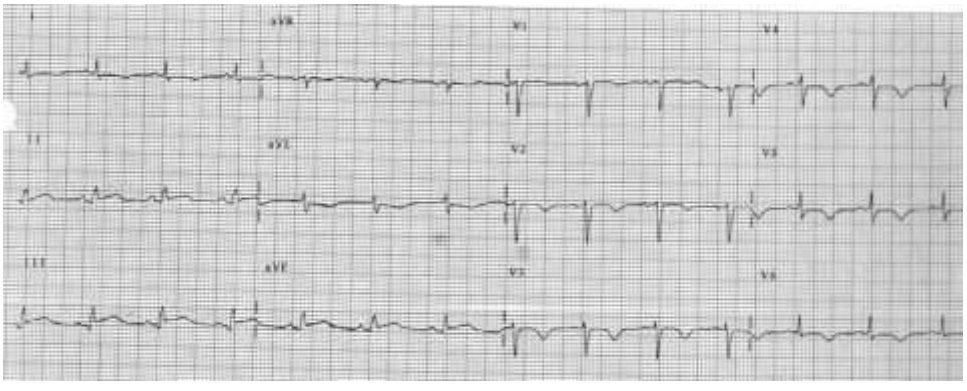
262

Practice ECG #1a



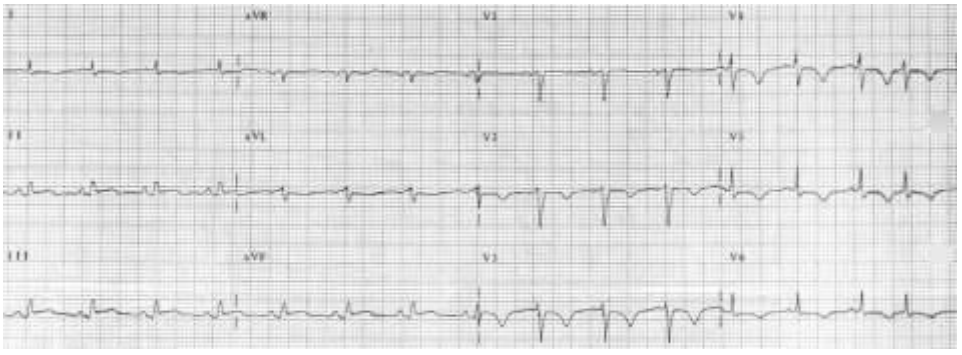
263

Practice ECG #1b



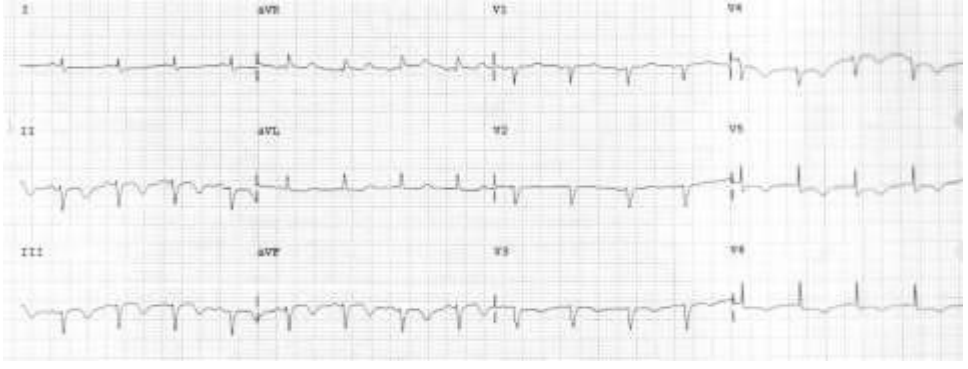
264

Practice ECG #1c



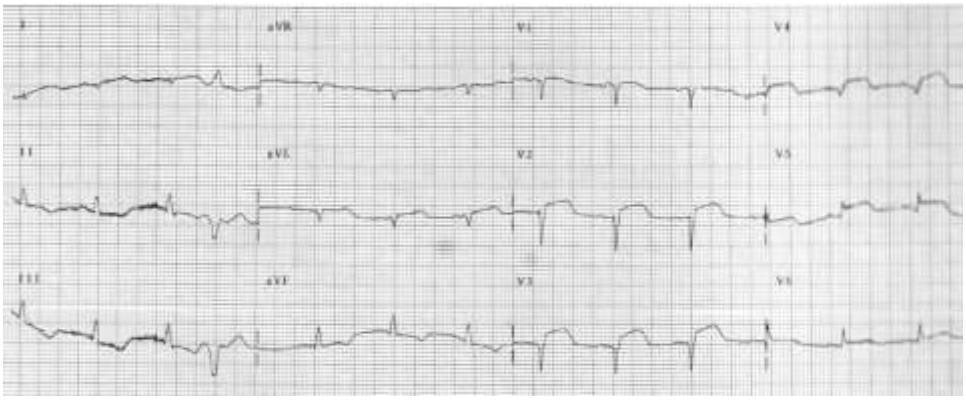
265

Practice ECG #1d



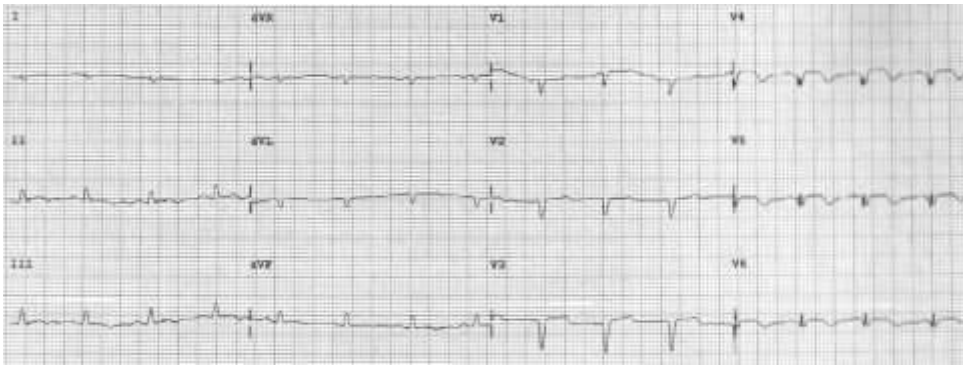
266

Practice ECG #1e



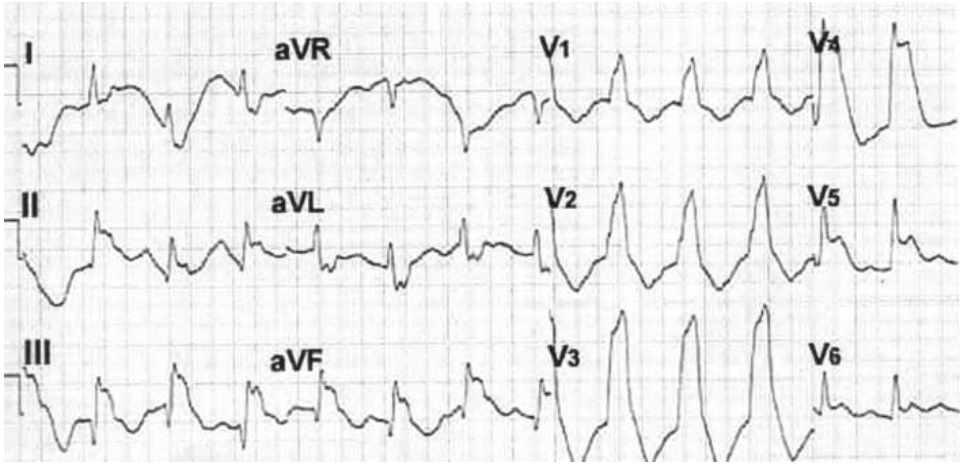
267

Practice ECG #1f



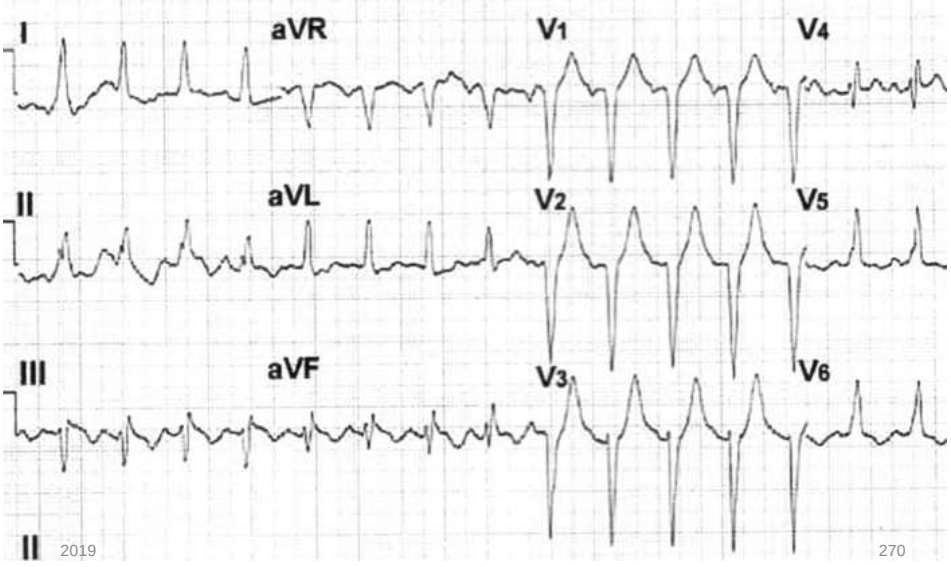
268

Practice ECG #2a



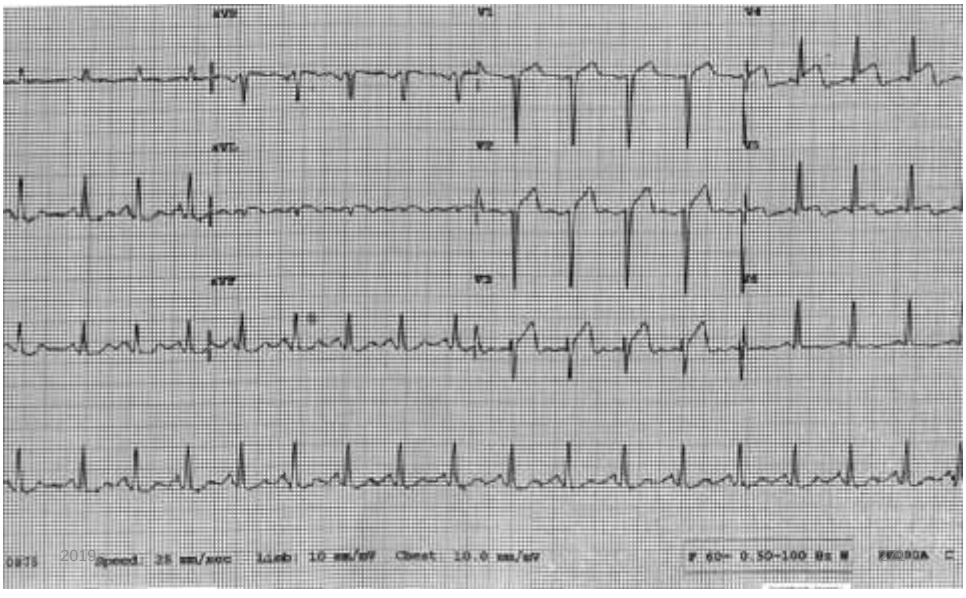
269

Practice ECG #2b



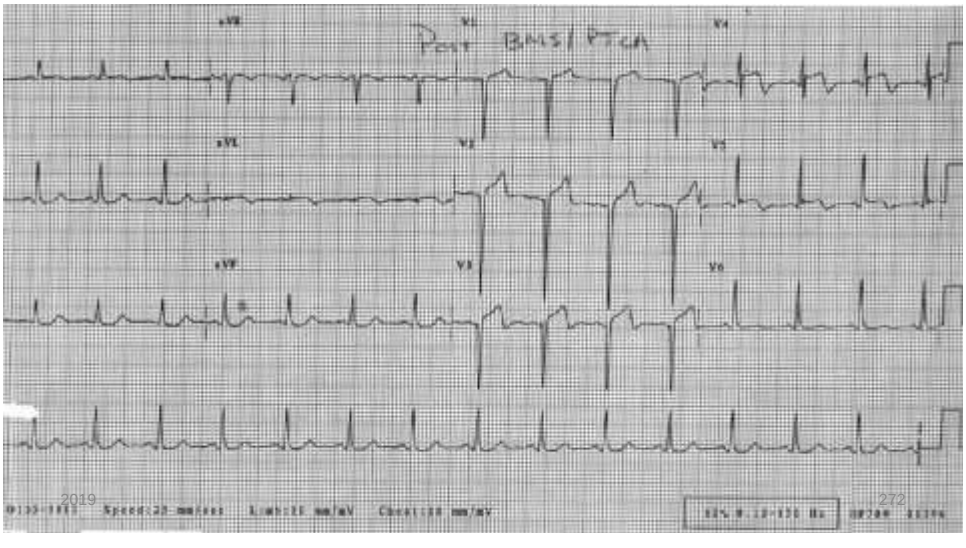
270

Practice ECG 3a



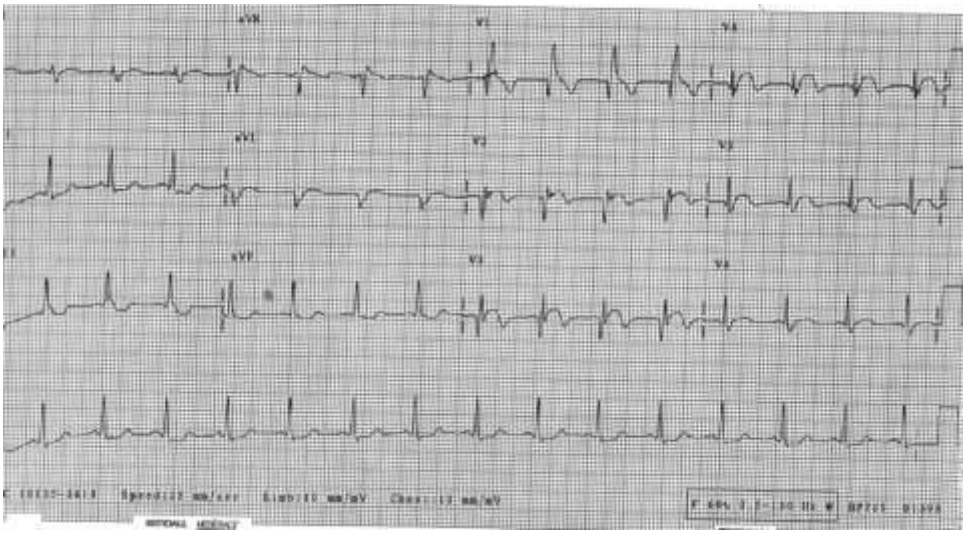
271

Practice ECG 3b



272

Practice ECG 3c



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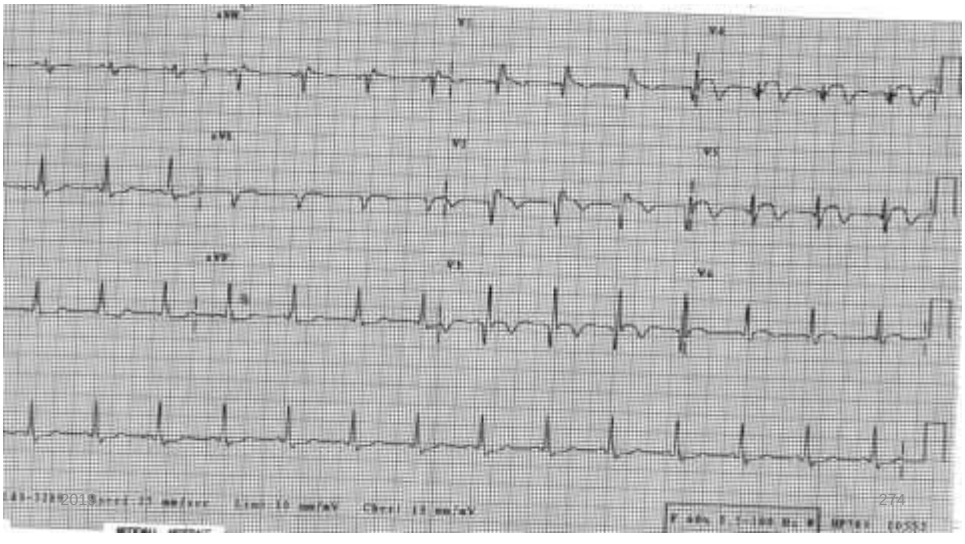
Practice ECG 3d



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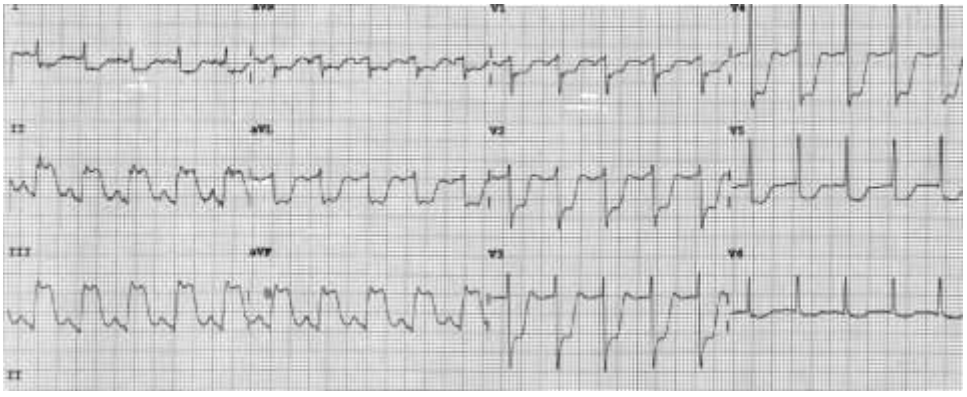
Practice ECG 4a
Day 1 5:30 am



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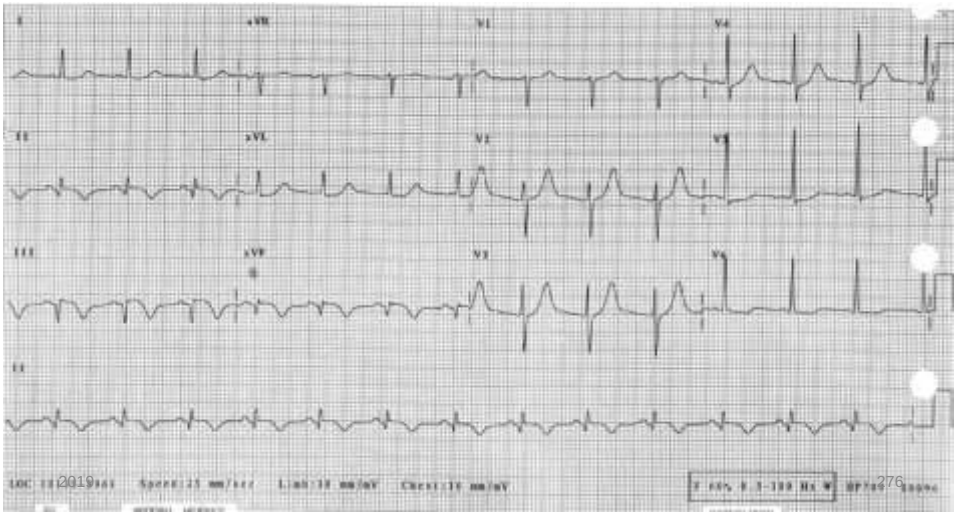
Practice ECG 4b
Day 2 8:30 am



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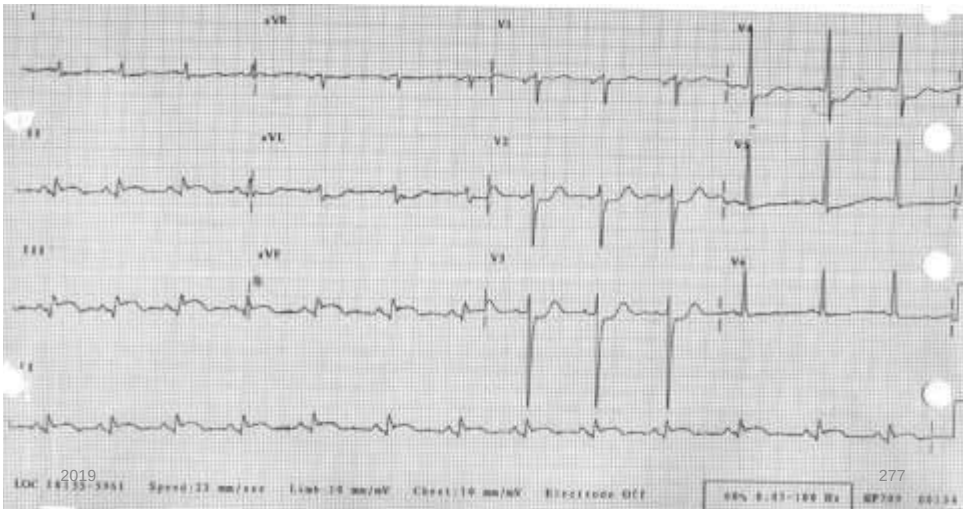
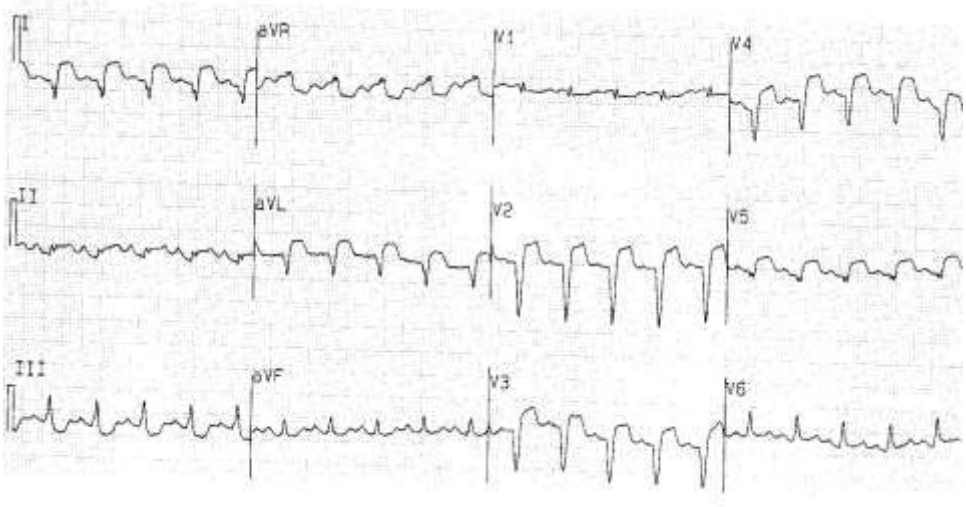
Practice 4c
Day 7 4:45 am



277

Practice ECG # 5



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Advocate and Also Be Aware

- Normal Variants
  - Early repolarization
  - Persistent juvenile T waves
- ECG Mimics
  - Hypertrophy
  - LBBB
  - Digitalis
  - Hyperkalemia
  - Adams – Stokes attack
  - Hypertrophic cardiomyopathy
  - Central nervous system disease
  - Intracranial bleed
  - Post extrasystolic T wave change
  - Blood glucose abnormalities
- Pain and ECG Mimics
  - Pericarditis / Myocarditis
  - Pulmonary Embolism
  - Aortic Dissection
- Pain Mimics
  - Aortic Dissection

Always correlate the ECG with the patient clinical picture!!

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ECG / Pain Mimics

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Abnormalities of Repolarization

- Abnormalities in ST segment, T Waves and QT interval reflect abnormalities in ventricular repolarization
- Coorelate with phase II and III of the action potential.

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Repolarization Changes

Primary Changes

- Disease processes: ischemia, myocarditis
- Drugs or toxins (digitalis, quinidine, QT interval prolonging drugs)
- Electrolyte abnormalities – especially Ca^{++} & K^{+}
- Abrupt change in heart rate, hyperventilation, changes in body position, catecholamines, sympathetic stimulation, temperature changes

Secondary Changes

- Result of changes in ventricular DEPOLARIZATION
  - Bundle branch blocks
  - Ventricular Hypertrophy

"WHENEVER THE VENTRICLE DEPOLARIZES ABNORMALLY IT ALSO REPOLARIZES ABNORMALLY"
C. JACOBSON

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Early Repolarization

- Precordial ST elevation in most adults
  - Up to 90%
- Early repolarization most common normal variant
  - Roughly 5-13% of population
  - African American men < 50 years of age
  - Athletes

? Increased Risk of Sudden Death

ST Elevation of Early Repolarization

- ST segments highest in V2-V3
- ST elevation up to 5 mm
- J point elevation
- Concavity, not convexity
- Tall peaked asymmetric T waves
- T waves are tall (not too tall) but not wide
- ST elevation < 0.5 m in lead V5 and V6
- T wave amplitude in V6 > V1
- No reciprocal ST depression
- Less common after age 55

ST Elevation of Early Repolorization

- Rarely seen in limb leads alone
- Right precordial early repolarization ("humpback")
- J wave (fishhook)

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- Proposed classification of ER
  - Type 1: associated with ER in the **lateral precordial leads**. This form is common among healthy male athletes and is thought to be largely benign.
  - Type 2: associated with ER in the **inferior or inferolateral leads** and is associated with a moderate level of risk.
  - Type 3: associated with ER **globally** in the inferior, lateral, and right precordial leads, and appears to be associated with the highest relative risk (though the absolute risk of sudden death remains small).

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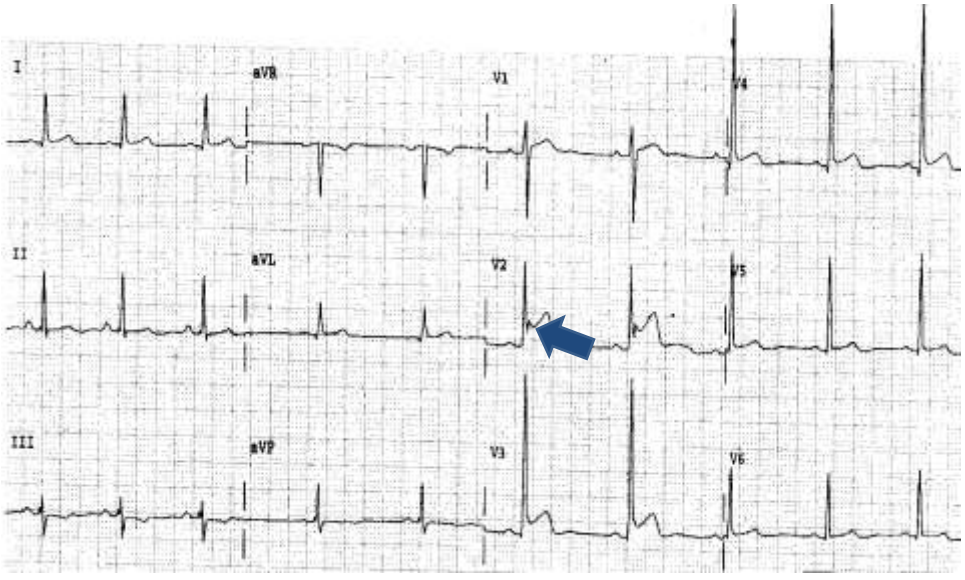
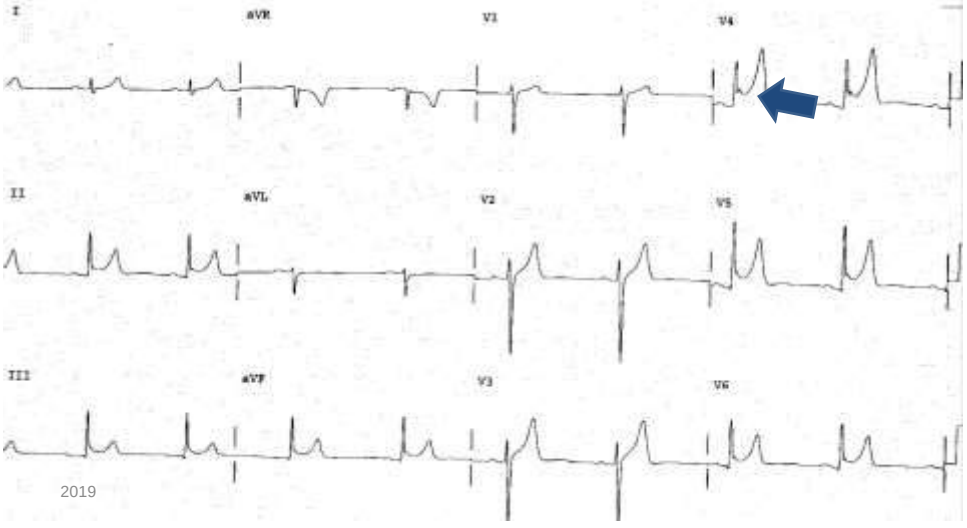


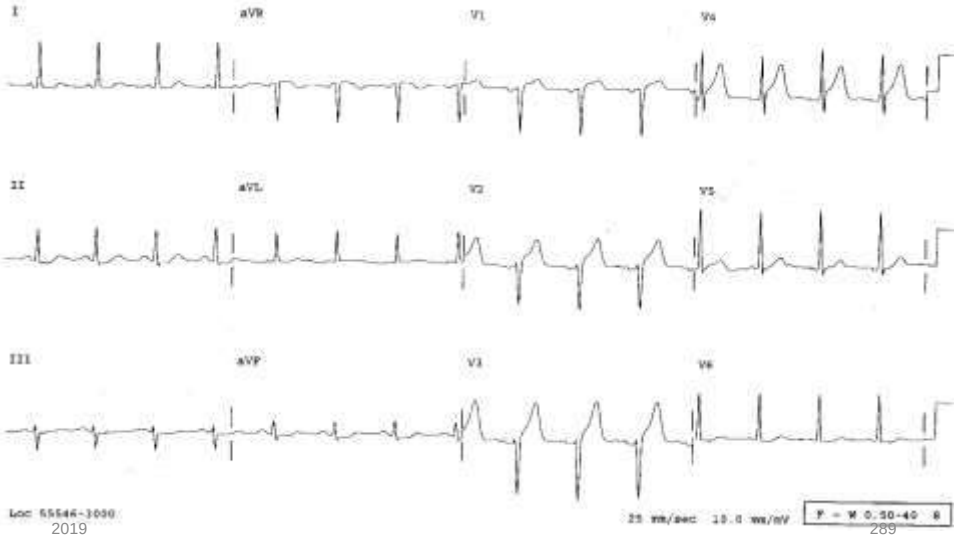
287

Early Repolarization

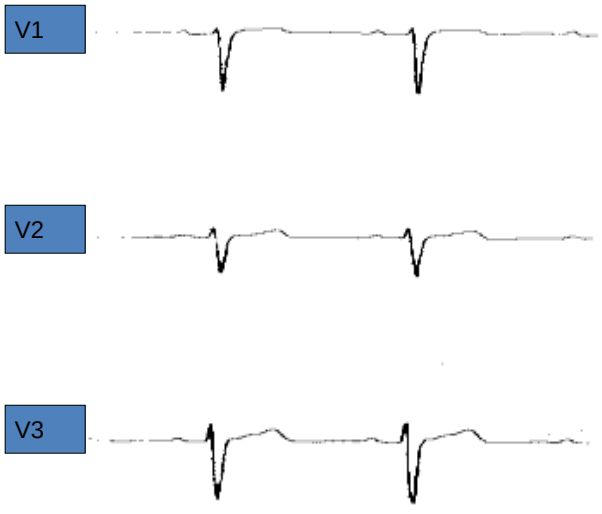


288

Case Study



289



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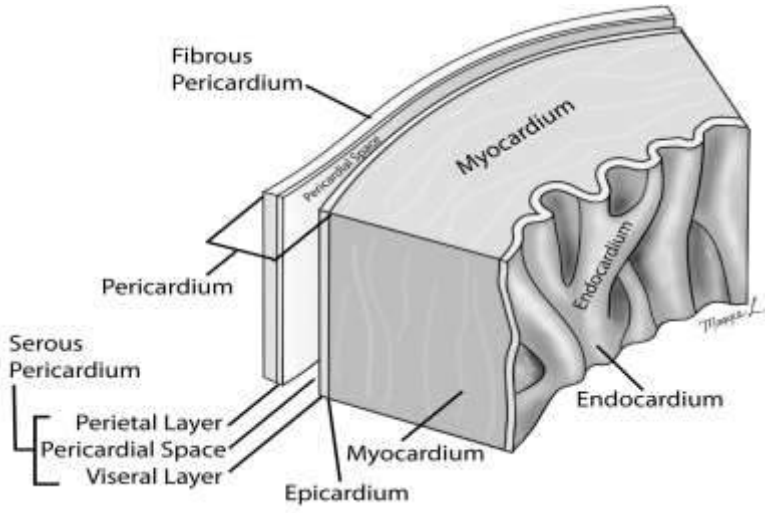
Pericarditis



Pericarditis: ECG Findings

- Mimics: anteroinferior; inferolateral; antero-infero-lateral MI
- ST Elevation
  - ST elevation typically greatest in II and V5 (also I and V6)
  - ST elevation may also be in V1 –V4; aVF, III and aVL (least)
  - Upwardly concave ST segments
  - ST elevation usually ≤ 5 mm

Pericarditis: ECG Findings

- Other ST changes
  - ST depression in aVR
  - Minimal depression V1, III, aVL may exist
- PR Segment depression
  - PR depression most common in II, aVF and V4 – V6
  - PR elevation > 0.5 mm in aVR
- Electrical Alternans
- Voltage changes with pericardial effusion or tamponade

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Stages of Pericarditis

- **Stage I**
  - ST elevation
  - More concave
  - Lasts up to 2 weeks
- **Stage II**
  - ST to baseline
  - Decrease T wave amplitude
  - Lasts from days to several weeks
- **Stage III**
  - T wave inversion
  - Starts at end of second to third week
- **Stage IV**
  - Gradual resolution
  - T wave may stay inverted up to 3 months

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Pericarditis

- Diffuse Pericarditis
  - Easiest to differentiate with both pain and ECG assessment
- Localized Pericarditis
  - May have reciprocal changes

Perimyocarditis
•Troponin
• Wall motion abnormalities

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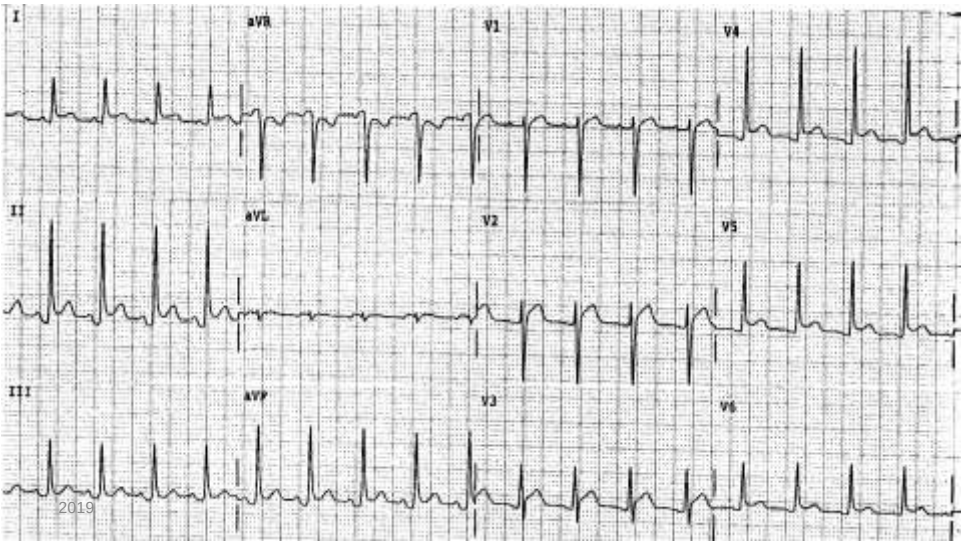
Classic Pericarditis



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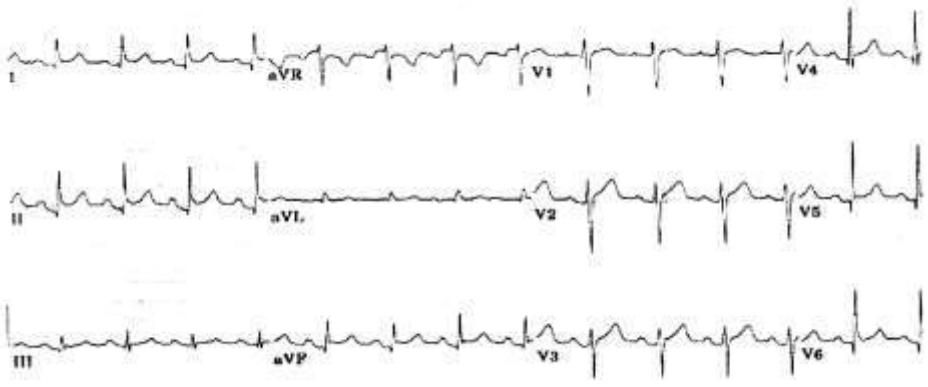
Pericarditis



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Pulmonary Embolism

- Obstruction of blood flow to one or more arteries of the lung by a thrombus (other emboli – fat, air, amniotic fluid) lodged in a pulmonary vessel
- 2nd most common cause of sudden death
- 3rd most common cause of death in hospitalized patient
  - 80% of unexpected hospital deaths
- Often recurrent

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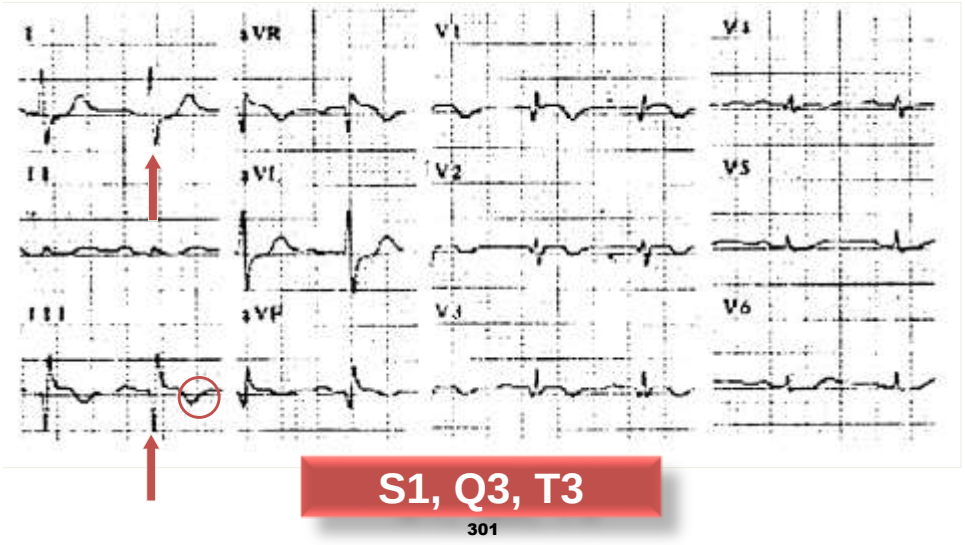
Massive PE

- Present in less than 5% of patients presenting with PE (Kucher, Rossi, De Rosa, & Goldhaber, 2006).
- Involves both the right and left pulmonary arteries or causes hemodynamic collapse
- Presenting systolic BP of < 90 mmHg
- Mortality rates range from 30% to 60% and most deaths occur within the first 1 to 2 hours (Ouellette et al., 2013; Wood, 2002).

ECG in PE – Acute Cor Pulmonale

- Changes in only 20% of pts
- Non specific
  - ST or atrial fibrillation
  - Small T wave inversion in limb and chest leads
- S1,Q3,T3
- RV hypertrophy
- Right axis deviation
- Other:
  - Large R waves in V_1 and V_2
  - Deep S waves in leads V_5 and V_6
  - Right atrial enlargement (tall P waves in lead II or dominant first ½ of P wave in V1)
  - Incomplete right bundle branch block (RBBB)
  - Delayed intrinsicoid deflection in leads V_1 and V_2

ECG in PE



301

Pain Mimics

Aortic Dissection

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Pathophysiology

- Intimal tear
- False channel
- Risk of rupture: outer wall
- Dissection – hematoma – occlusion of vessels
  - Ascending aorta
  - Descending aorta

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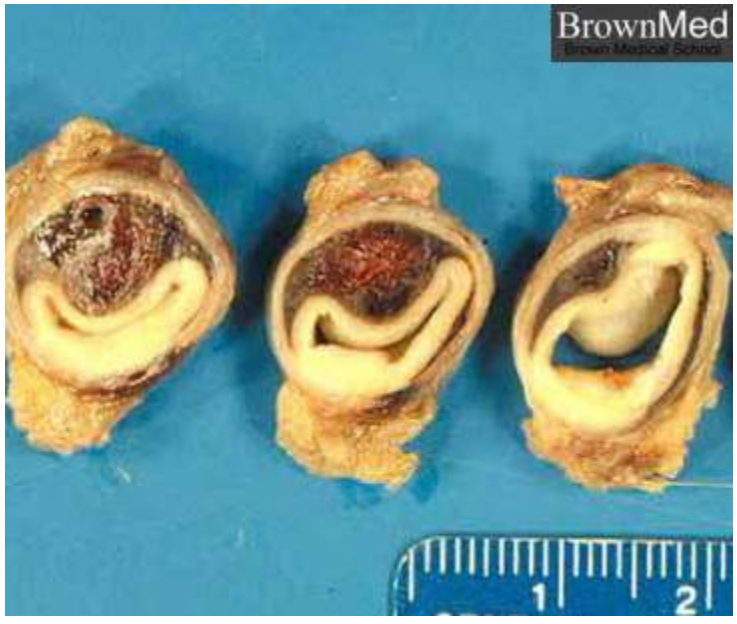

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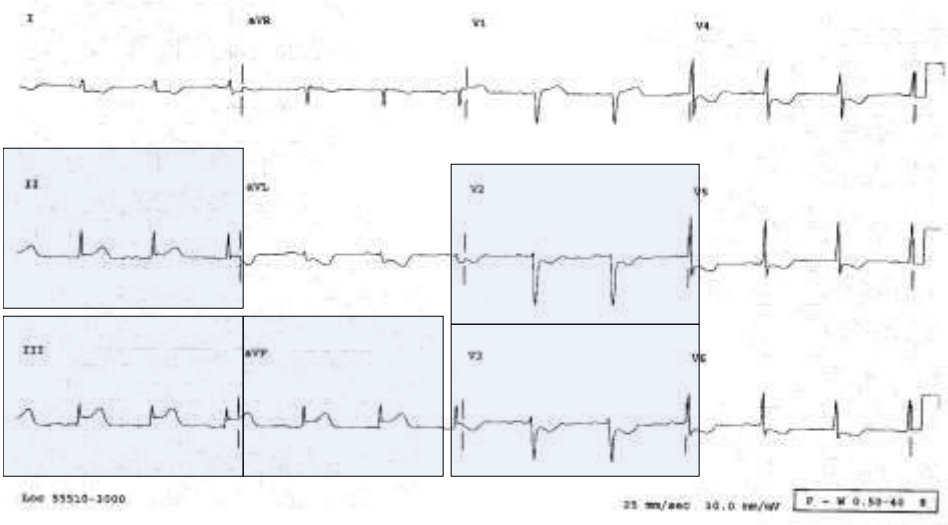
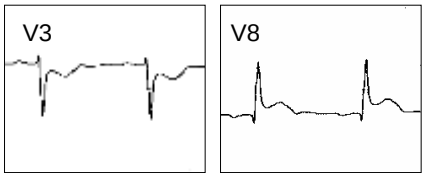
Complications of Dissection

- Aortic regurgitation from retrograde dissection involving aortic valve or from aortic dilatation.
- **MI from retrograde coronary artery dissection.**
- Cardiac tamponade from ascending aorta or aortic arch rupture.
- Intraplerual rupture from descending aortic dissection ruptures into intrapleural space – most commonly left sided.
- Retroperitoneal bleed from rupture of abdominal aorta dissection.
- Stroke from brachial artery compromise.
- Paraplegia, reduced blood flow to kidneys, bowels, and lower extremities from compromise of arterial branches.

305





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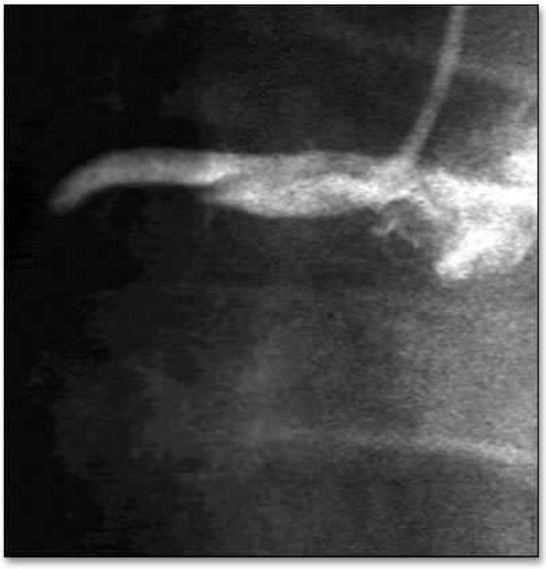
Occlusion of RCA by Aortic Dissection



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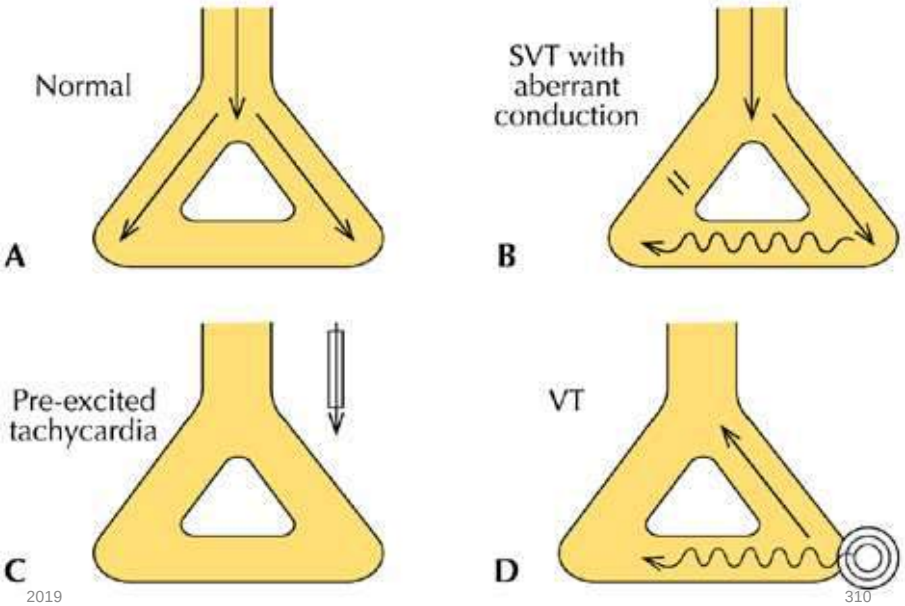
Advanced Concepts in 12 Lead ECG Interpretation Part 3

Differentiation of Wide Complex Tachycardias
WPW
High Risk Features for SCD
Torsades de Pointes

308

DIFFERENTIATING SUPRAVENTRICULAR & VENTRICULAR TACHYARRHYTHMIAS

309



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Ectopy VS Aberrancy

SVT with aberrancy	Ventricular tachycardia

• SVT with RBBB Aberration
• SVT with LBBB Aberration
	• VT with LBBB Pattern
	  – Right VT
	• VT with RBBB Pattern
	  – Left VT

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Acute Management of Wide Complex Tachycardias

- Wide complex tachycardia presumed to be VT if diagnosis is unclear
- DC cardioversion with sedation if hemodynamically unstable
- Don't assume VT cannot be well tolerated!
- The rate, size of the heart and presence of additional complications are often more important than the source of the tachycardia

✓Check the patient (need to defib?)
✓Check the blood pressure (need to cardiovert?)
✓Check the ECG (determine the rhythm)

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Criteria for Differentiating Ectopy from Aberrancy

- Patient history / assessment
- QRS Width
- Concordance
- AV Dissociation
- Axis
- Morphology

Note:
VT is much more common than supraventricular tachycardia with bundle branch aberration. In wide QRS tachycardias VT is the right answer up to 80% of the time. A wide complex tachycardia is always considered ventricular in origin if the diagnosis is uncertain

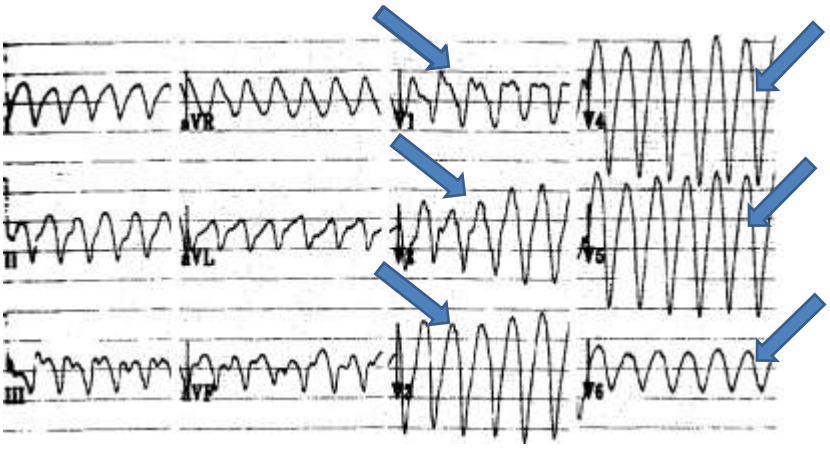
Patient History

- Acute ischemia / injury (Abnormal automaticity)
- **Post myocardial infarction / ischemic cardiomyopathy (Reentrant circuit within myocardium)**
- Non ischemic dilated cardiomyopathy (Bundle branch reentrant VT)

QRS Width

- The wider the QRS – VT is favored – However:
- SVT with LBBB will have a wider QRS than SVT with RBBB
- Other causes of SVT with wider than expected QRS: antidromic tachycardia and patients on Class I antiarrhythmics or amiodarone
- Not all VT is significantly wide
  - VT originating from septum more narrow than VT from free wall
  - If QRS more narrow than sinus rhythm = VT

Negative Concordance

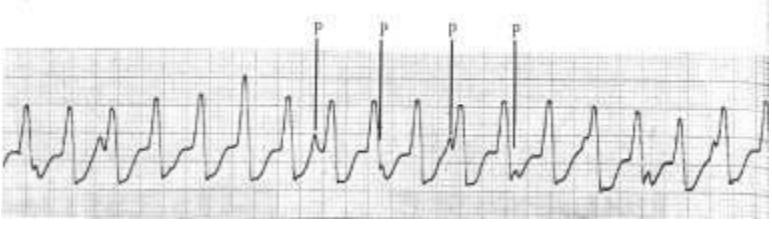


315

AV Dissociation

- Independent atrial and ventricular activity (AV dissociation) is diagnostic for ventricular ectopy

Only seen in 30% VTs



- Ventricular tachycardia may also have retrograde P waves (retrograde P waves do not confirm VT)

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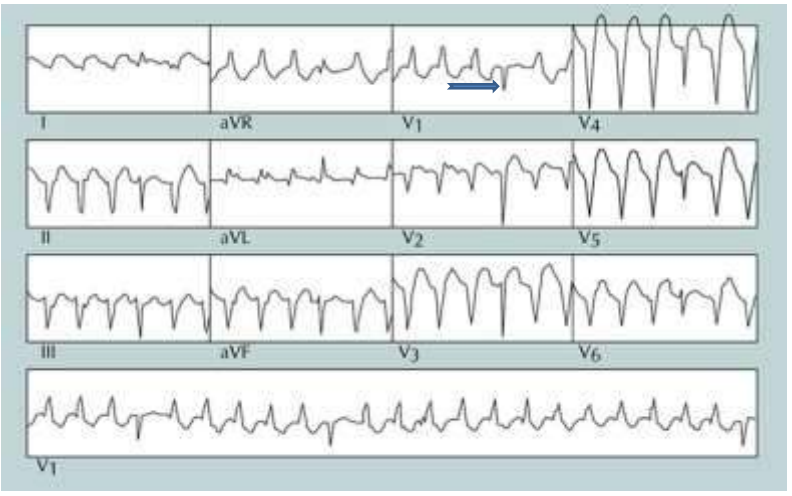
AV Dissociation: Fusion or Capture Beats



Axis

- **Right superior / extreme axis is strong indicator of ectopy**
- Right axis deviation confirms ectopy with LBBB pattern
- Ventricular tachycardia <u>rarely</u> occurs with normal axis

Morphology (Shape)

Ventricular Ectopy compared to Aberrancy (BBB)

Morphology Challenges:
BBB Reentrant VT
Idiopathic RVOT
Antidromic tachycardia

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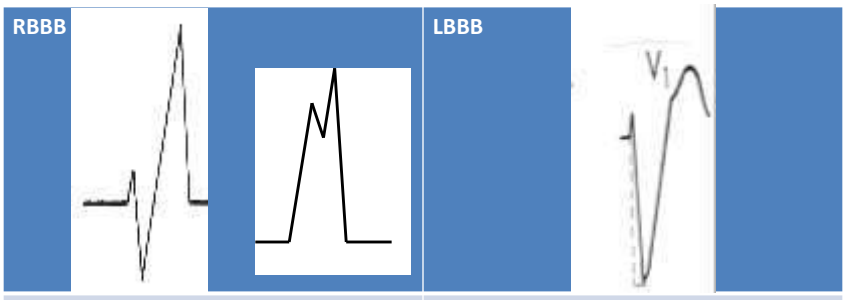
Comparison of Morphology in Lead V1



VT from Left Ventricle

VT from Right Ventricle

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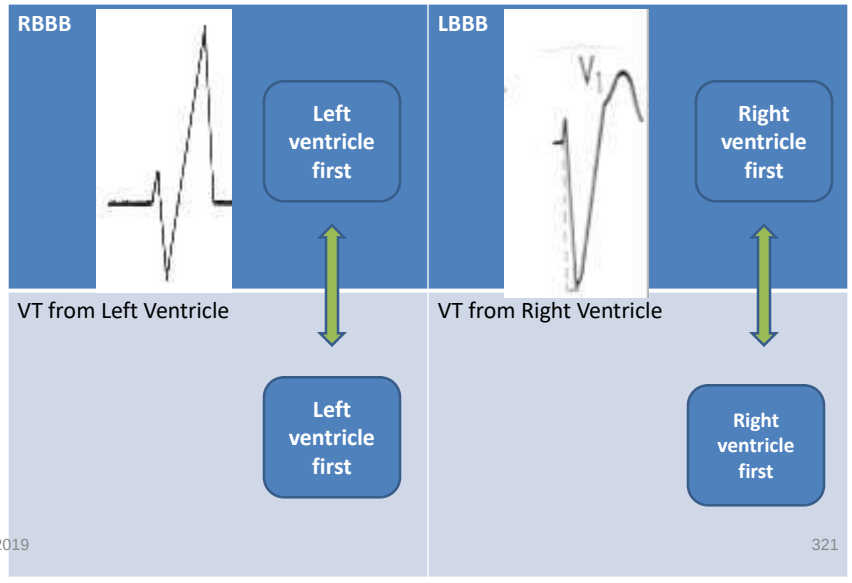
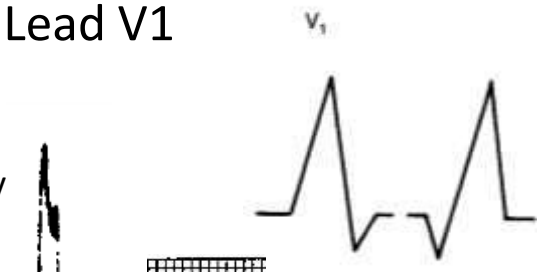
Comparison of Morphology in Lead V1



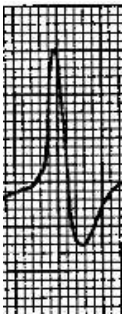
321

Left Ventricular Ectopy Lead V1

- Right Bundle Branch shaped
  - R wave with an early left peak (Rr')
  - R wave with a single peak
  - q wave followed by R wave



Can also be shape of RBBB



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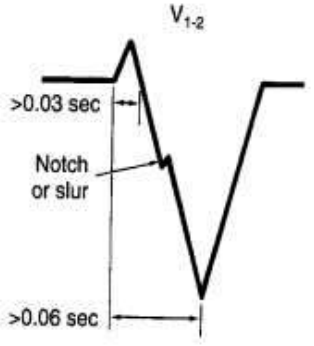
Right Ventricular Ectopy
Lead V1

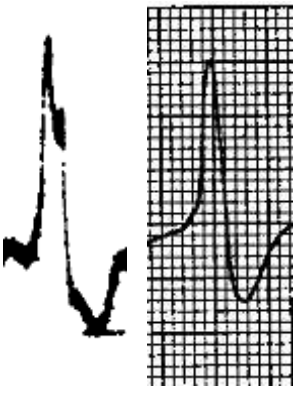
- LBBB shaped
  - Primarily negative wide rS complex
    - delay to the nadir
      - > 0.06 sec
    - r wave broader than 0.03 sec
    - Slurring on the down stroke



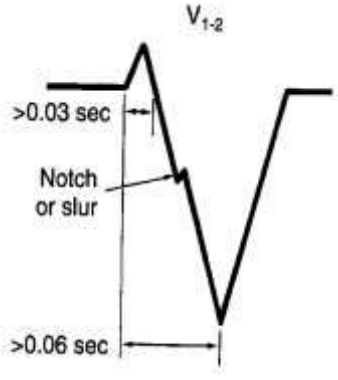
Note: LBBB shaped VT can come from RV or septum.
VT from RV includes: Idiopathic VT,
BB Reentrant VT,
Arrhythmogenic right ventricular dysplasia, VT from Brugada Syndrome

323

Lead V1 (VT Patterns)



VT with RBBB pattern or LVT



VT with LBBB pattern or RVT

2019 324

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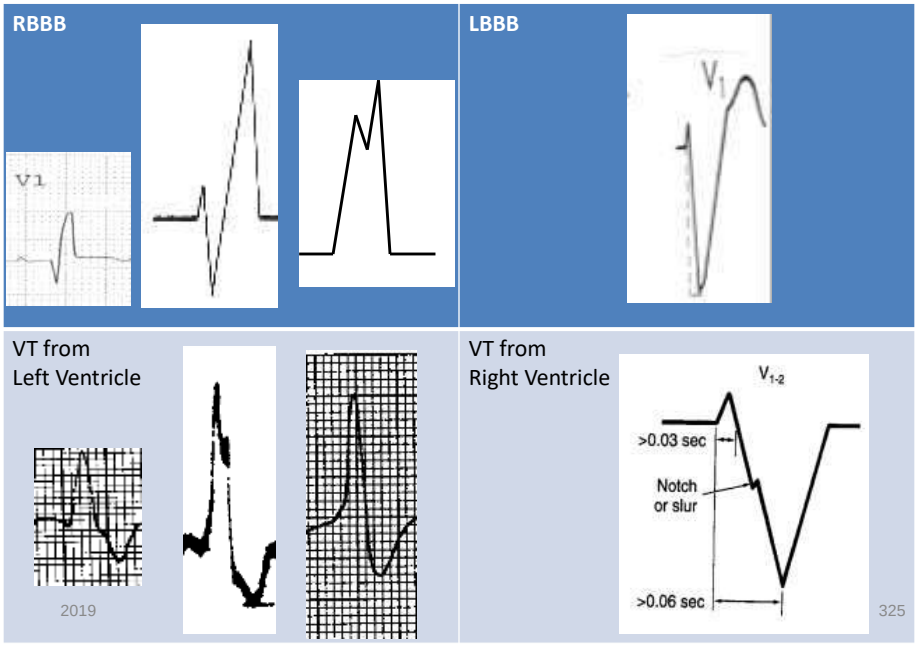
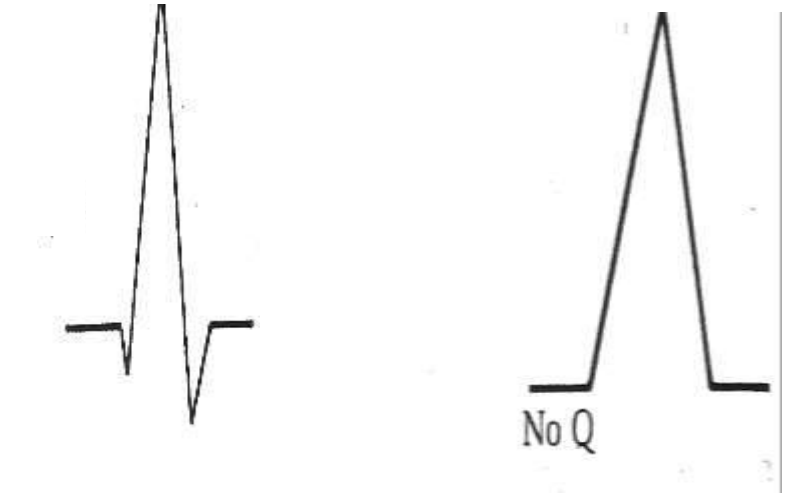
Comparison of Morphology in Lead V1



325

Bundle Branch Block Morphology in Lead V6



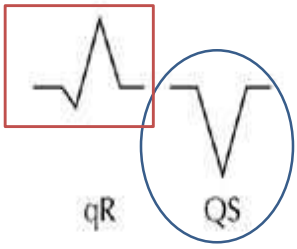
326

Ventricular Ectopy Morphology in Lead V6

Left Ventricular VT

- QS complex
- r wave followed by S wave with R:S ratio < 1

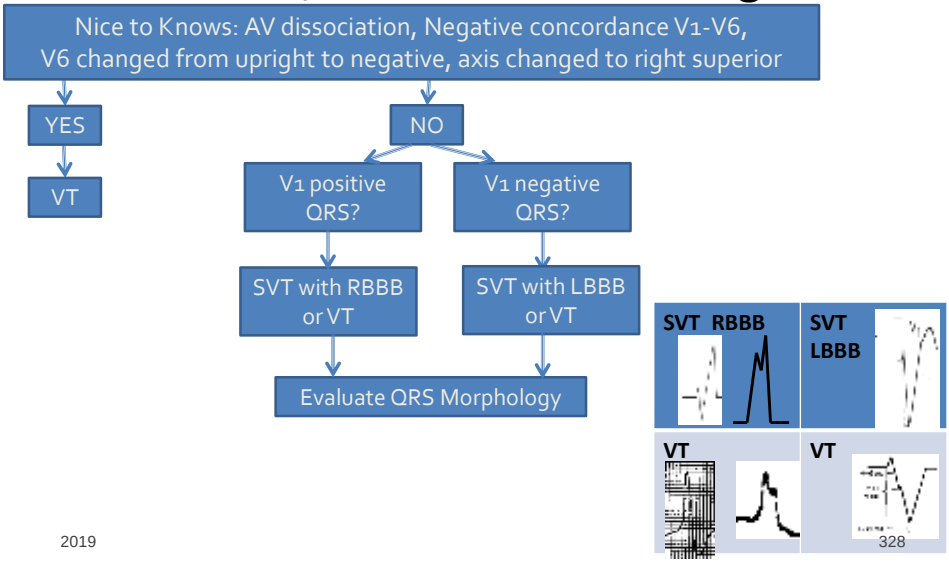
Right Ventricular Like VT



- Any Q Wave
- QS wave

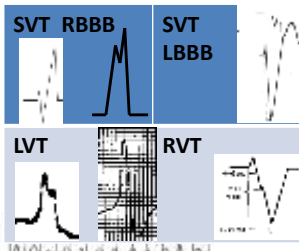
327

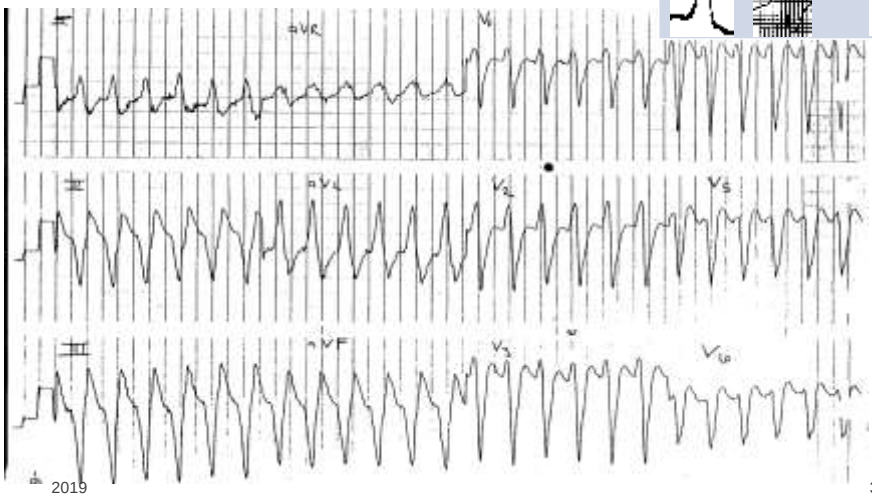
Methodology for Differentiation Using ECG / Bedside Monitoring



328

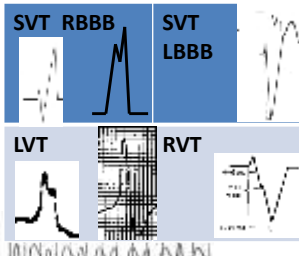
Practice ECG 1 – Poll ?

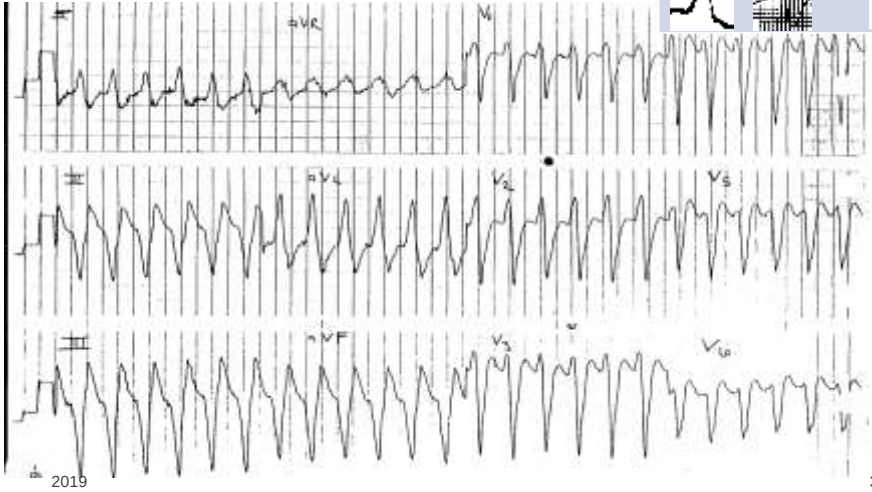




329

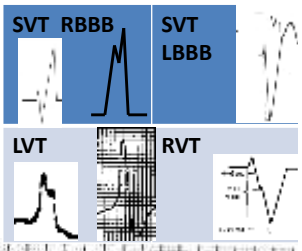
Practice ECG 1 – Poll ?

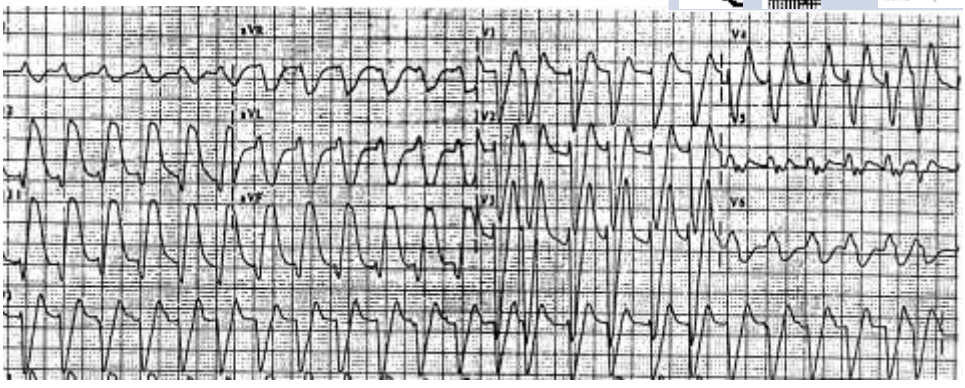




330

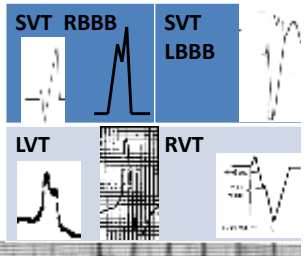
Practice ECG 2

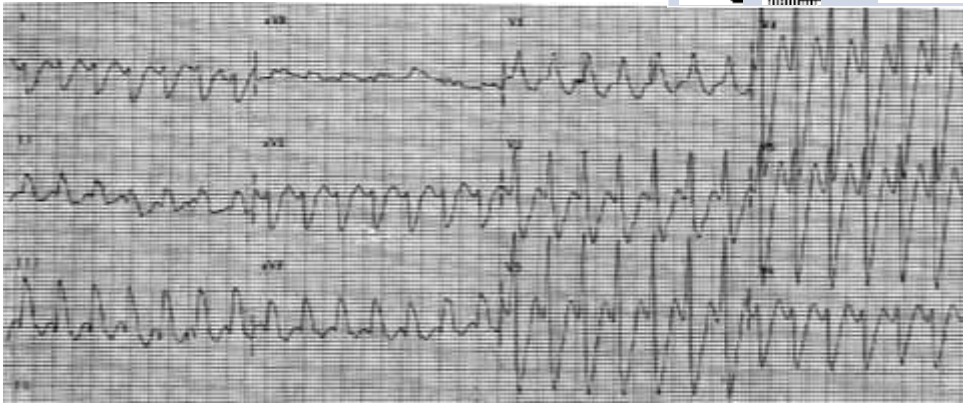




331

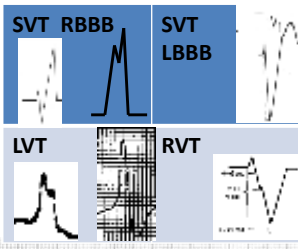
Practice ECG 3

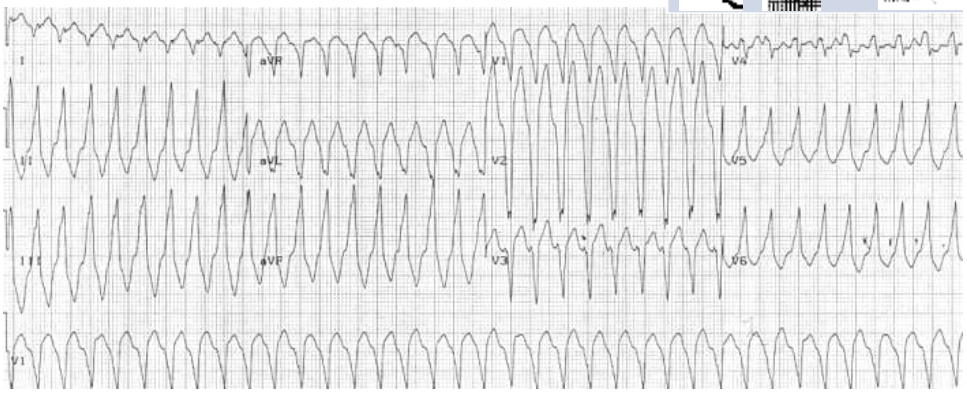




332

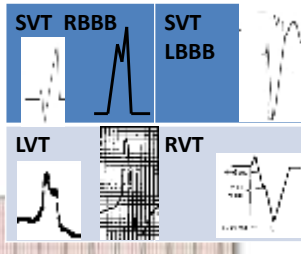
Practice ECG 4

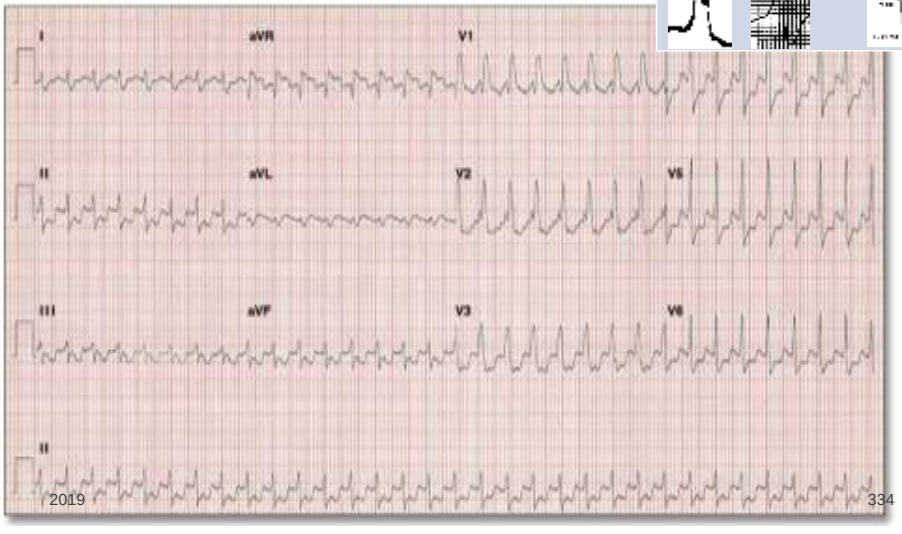




333

Practice ECG 5





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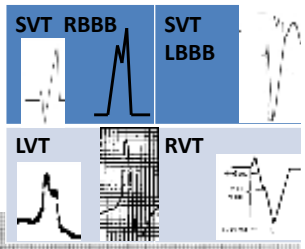
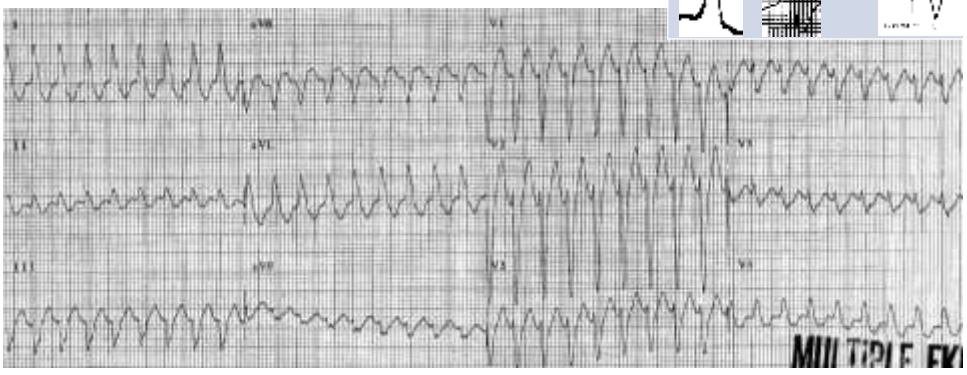
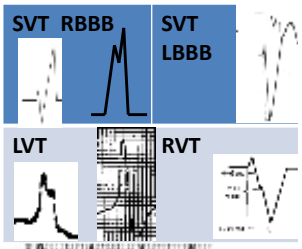
Practice ECG 6

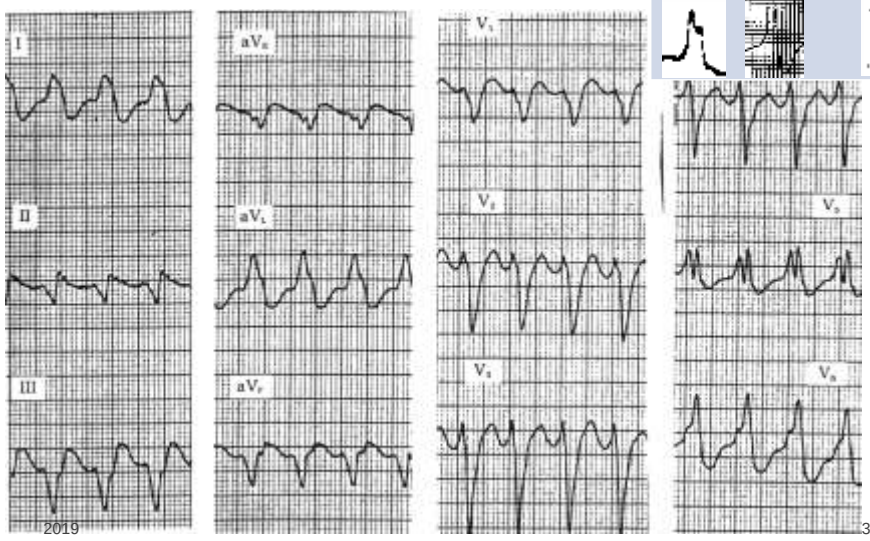




335

Practice ECG 7





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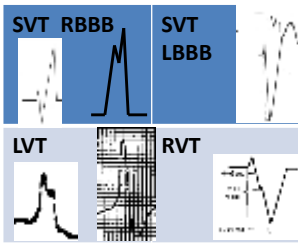
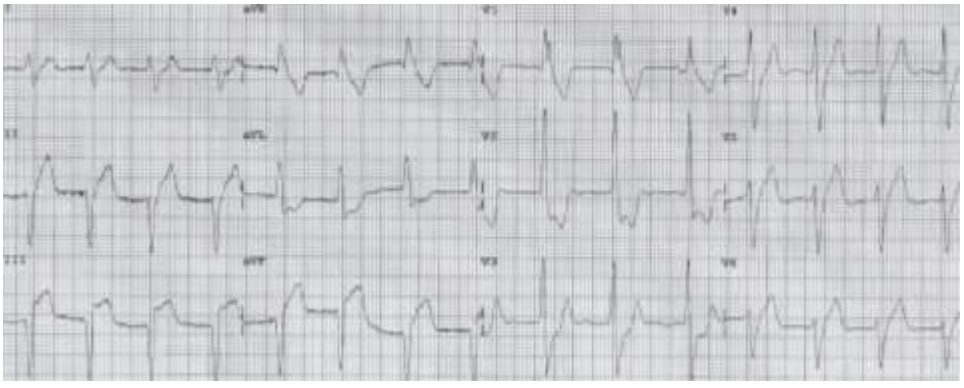
Practice ECG 8



12 Lead ECG Post Inferior STEMI on Arrival to CCU
Vital Signs Stable
12 lead ECG Interpretation:

- Atrial Fibrillation
- RBBB with Left Anterior Hemiblock ???



2019 337

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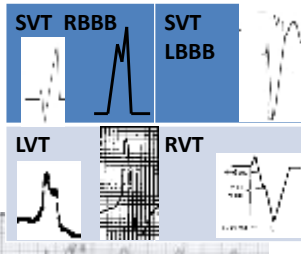
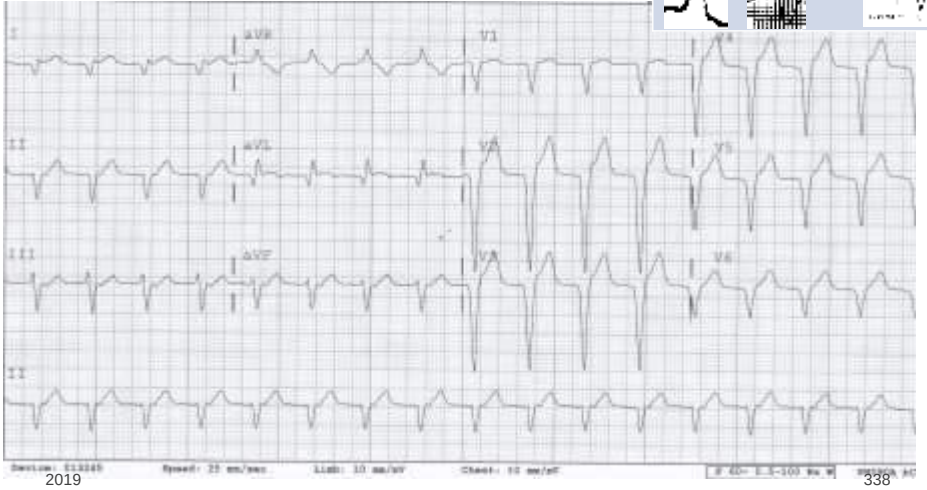
Practice ECG 9



12 Lead ECG Post Inferior STEMI on Arrival to CCU
Vital Signs Stable
12 lead ECG Interpretation: Non Specific Intraventricular Conduction Delay??



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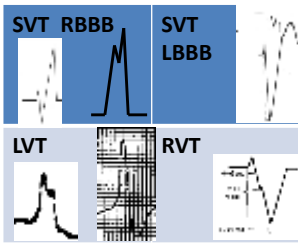
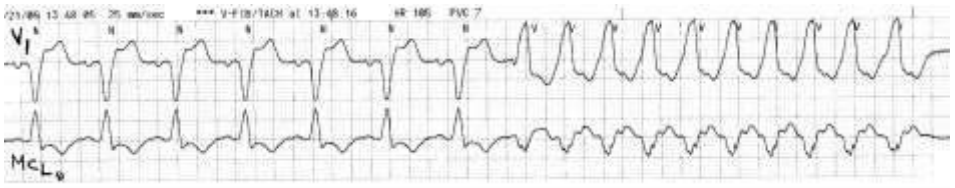
Linking to the Bedside Monitor Practice ECG 1



Dissociation or Negative Concordance
Extreme Axis or V6 Negative



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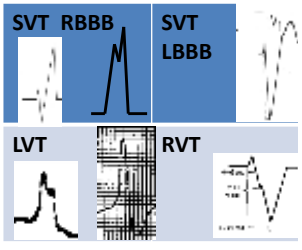
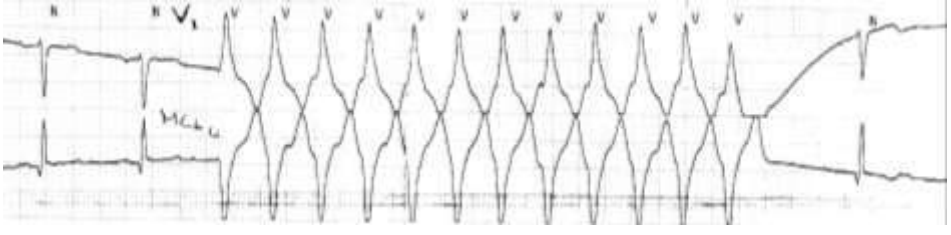
Linking to the Bedside Monitor Practice ECG 2



Dissociation or Negative Concordance
Extreme Axis or V6 Negative



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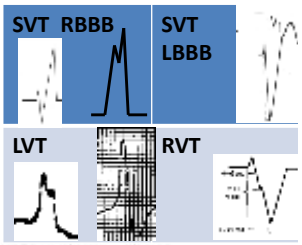
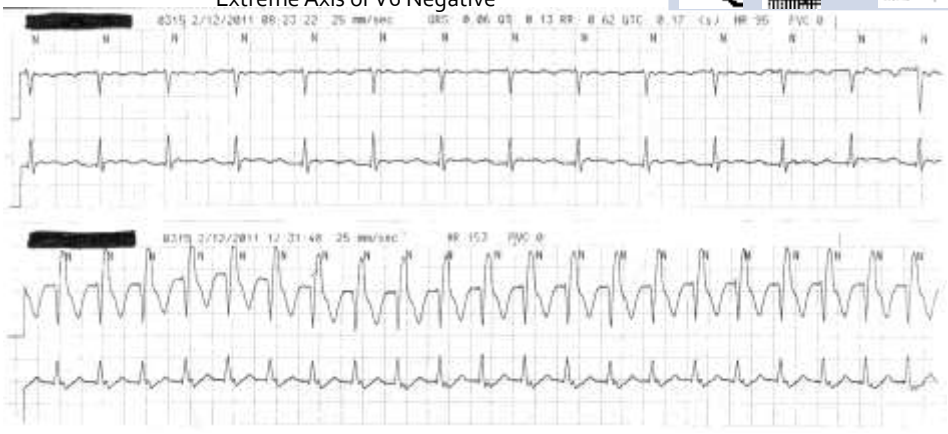
Linking to the Bedside Monitor Practice ECG 3



Dissociation or Negative Concordance
Extreme Axis or V6 Negative



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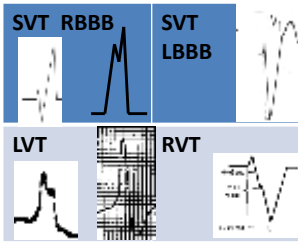
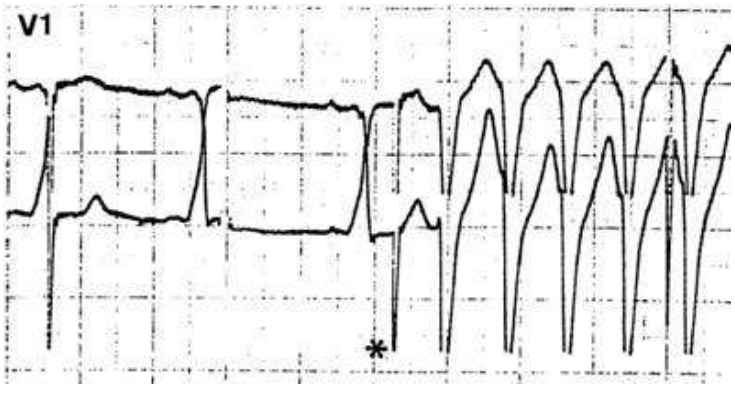
Linking to the Bedside Monitor Practice ECG 4



Dissociation or Negative Concordance
Extreme Axis or V6 Negative



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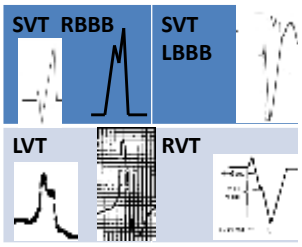
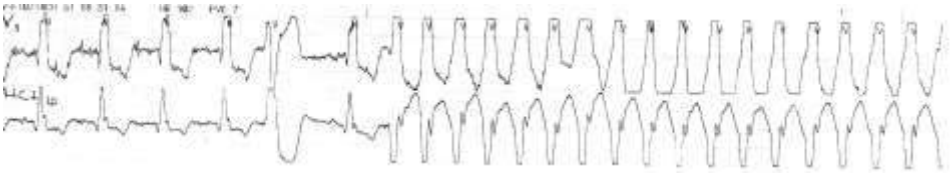
Linking to the Bedside Monitor Practice ECG 5



Dissociation or Negative Concordance
Extreme Axis or V6 Negative



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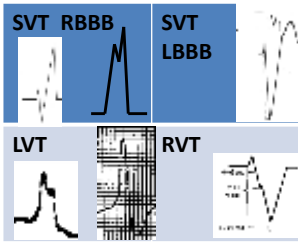
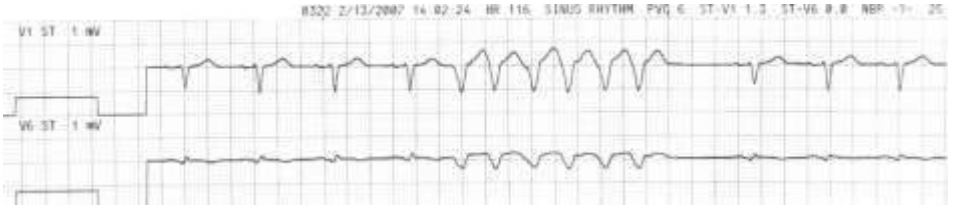
Linking to the Bedside Monitor Practice ECG 6



Dissociation or Negative Concordance
Extreme Axis or V6 Negative



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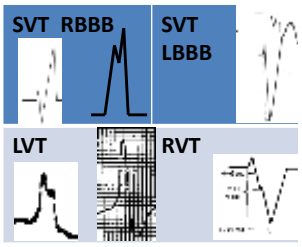
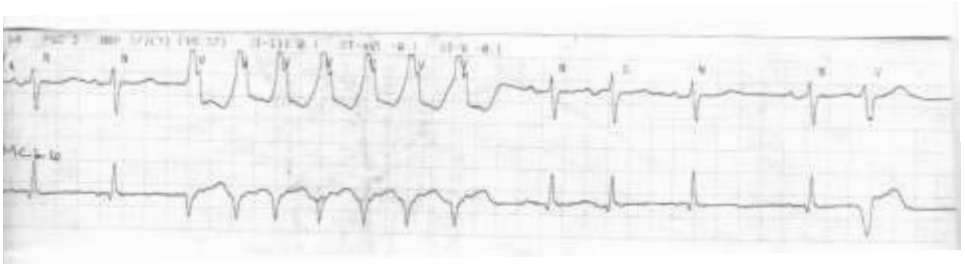
Linking to the Bedside Monitor Practice ECG 7



Dissociation or Negative Concordance
Extreme Axis or V6 Negative



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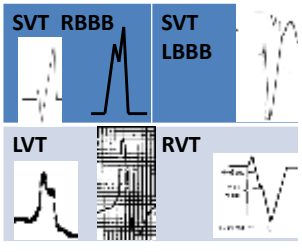
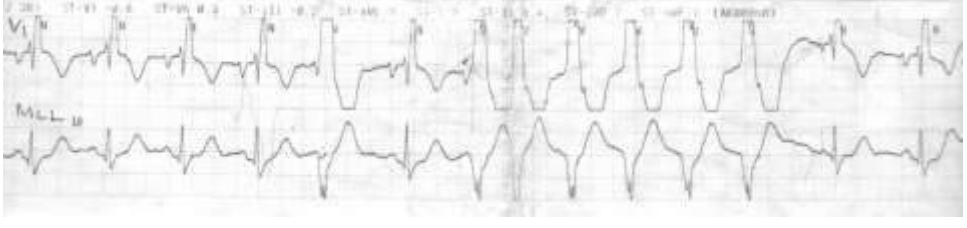
Linking to the Bedside Monitor Practice ECG 8



Dissociation or Negative Concordance
Extreme Axis or V6 Negative



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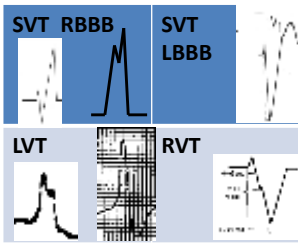
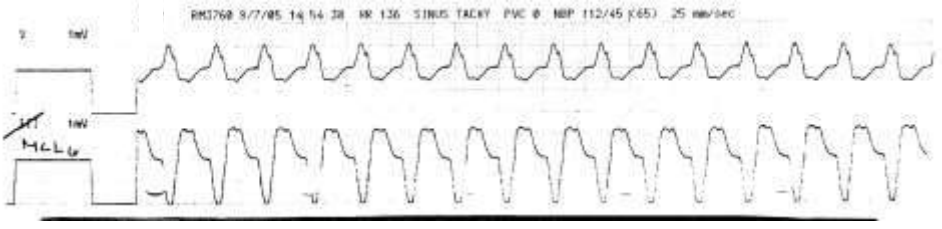
Case Study





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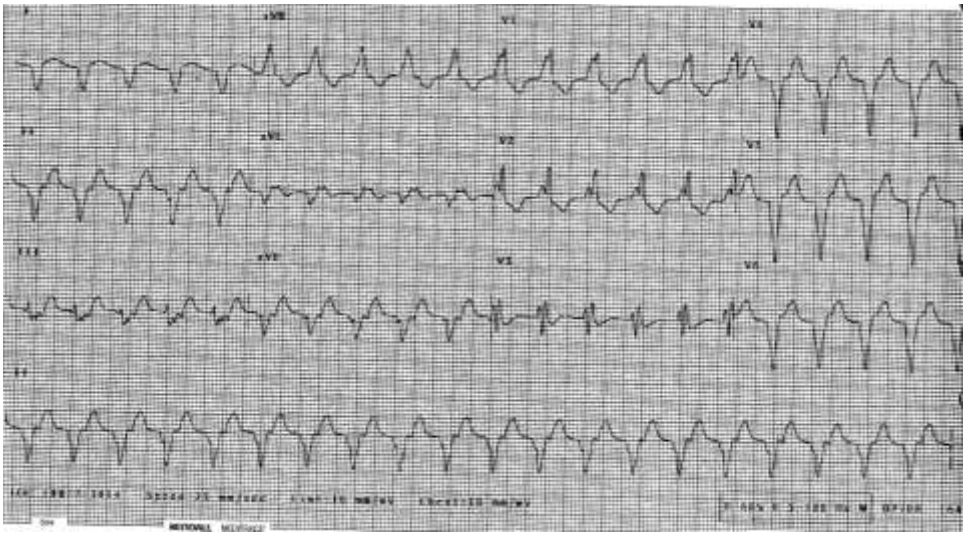
Case Study



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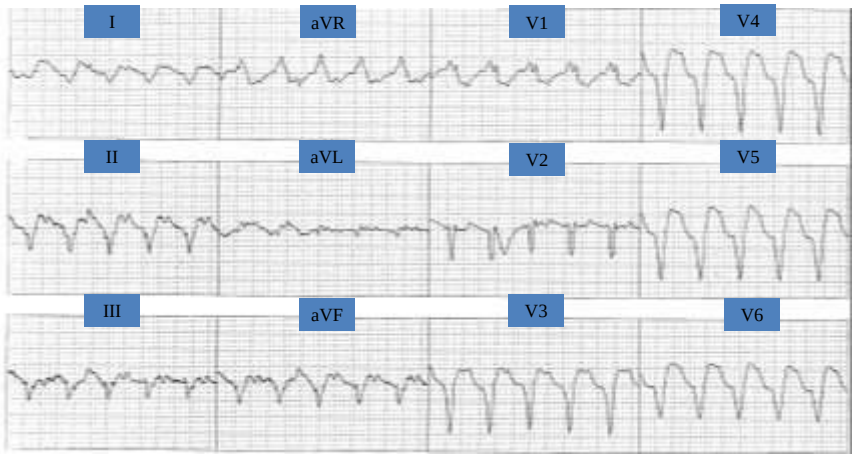
Case Study



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Morphology Challenges

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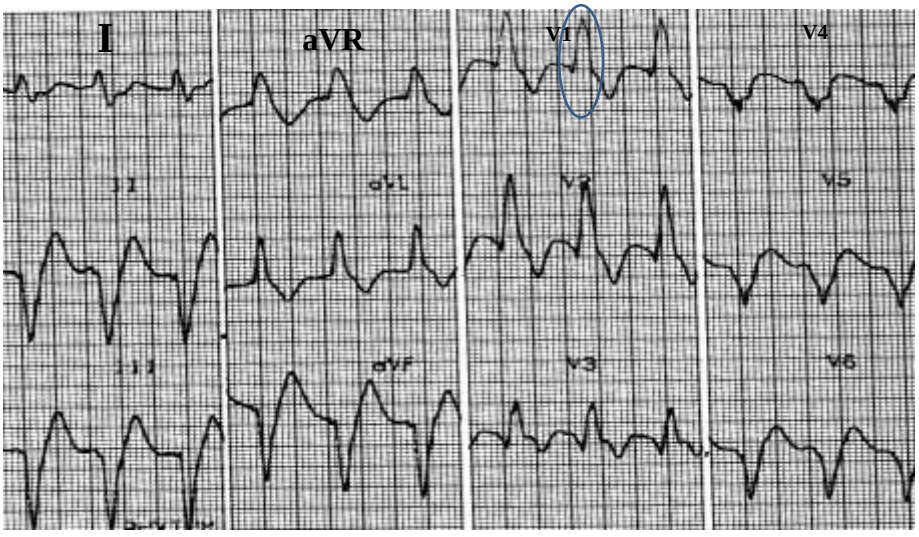

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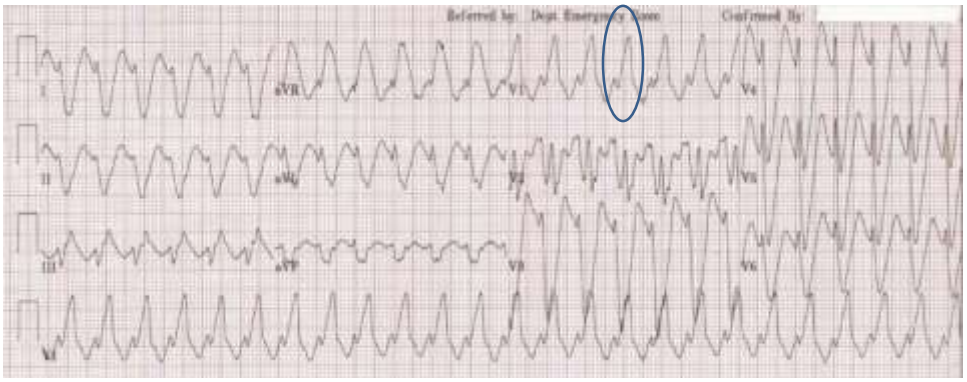

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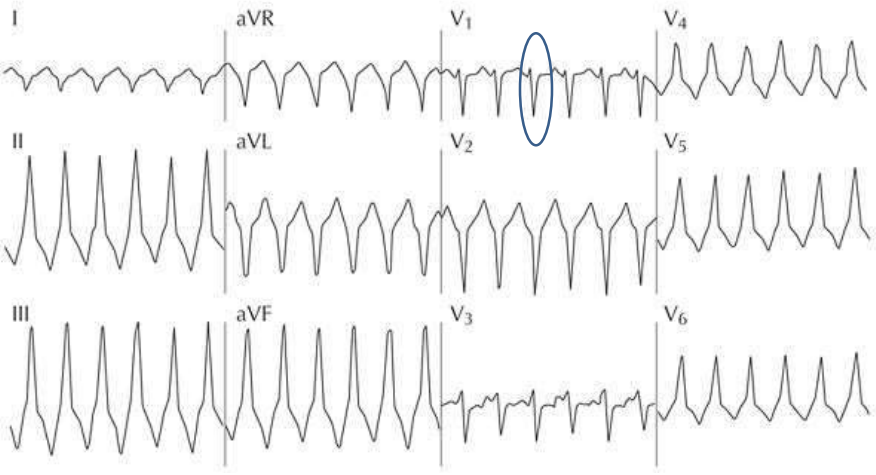

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The Not so Common:

Congenital and Acquired Long QT, Brugada, WPW, Arrhythmogenic Cardiomyopathy

Be on the Lookout

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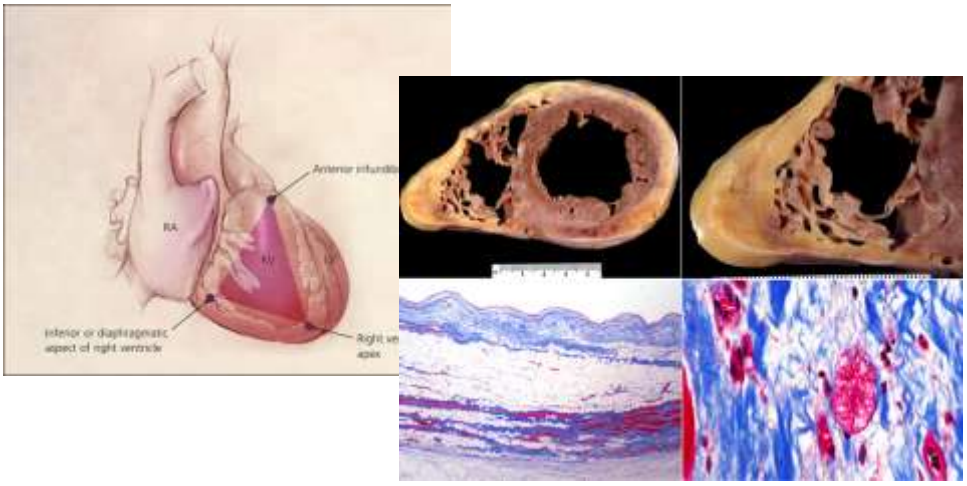
Arrhythmogenic Cardiomyopathy



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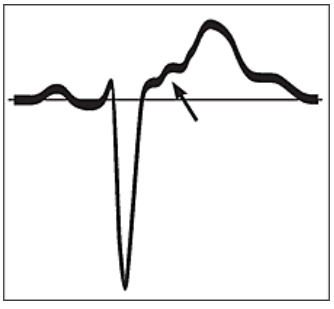
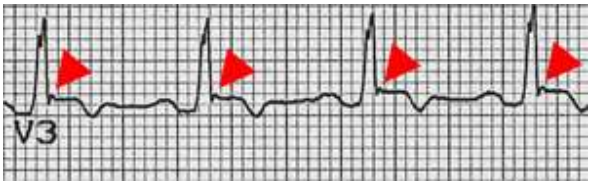
Arrhythmogenic Cardiomyopathy:
ECG Signs

- ECG
  - **T Wave inversion in leads V1-V6**
  - **Epsilon wave**
  - VT with LBBB pattern
  - Conduction delays through right bundle





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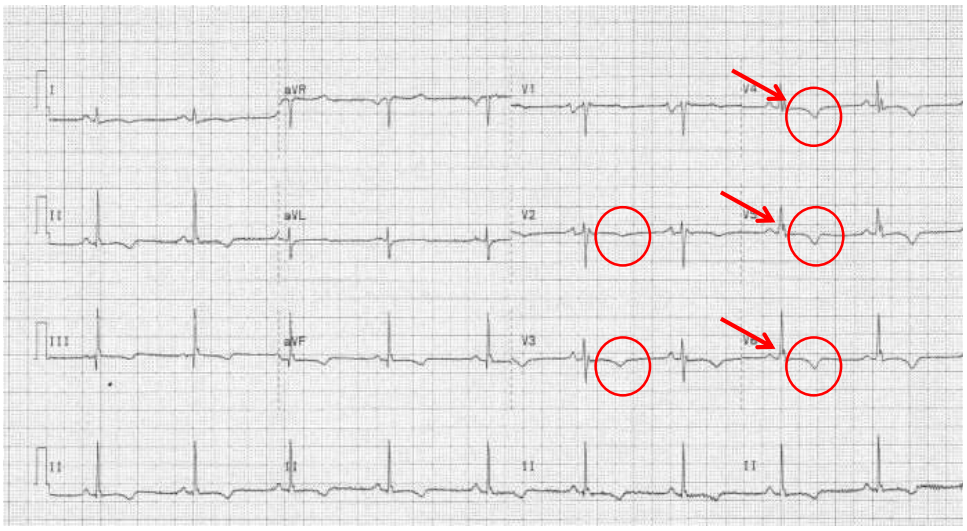

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QT Interval Evaluation

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Expected QTc Intervals

	1 to 15 Years	Adult Males	Adult Females
Normal	< .44 seconds	< .43 seconds	< .45 seconds
Borderline	.44 to .46 seconds	.43 to .45 seconds	.45 to .47 seconds
Prolonged	> .46 seconds	> .45 seconds	> .47 seconds

Source: Moss AJ, Robinson JL. Long QT Syndromes. Heart Dis Stroke. 1992;309-314

QTc .50 sec (500 msec) or more is dangerous and should be considered an ominous sign of impending Torsade's de Pointes.

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QT Interval

- Measured from beginning of QRS complex to the end of the T wave
- Reflects both ventricular depolarization (QRS) and ventricular repolarization (T wave)
- Used most specifically to reflect ventricular repolarization

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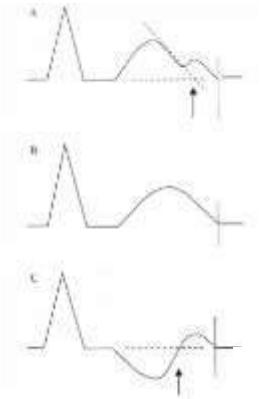
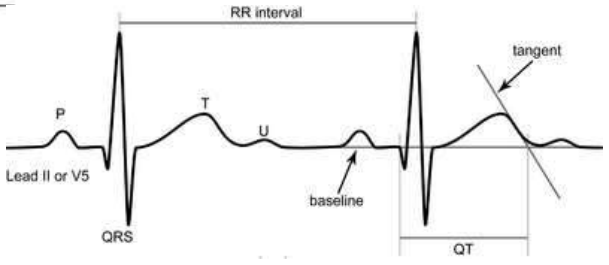


U Waves and
Biphasic T waves.



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Heart Rate Adjustment

- QT interval needs to be adjusted for HR
- QT does not adjust to HR on a beat to beat basis

- **Dynamic changes are most important**
- Abnormal findings are uncovered during abrupt changes in the R to R

- **Irregular heart rhythms (i.e. atrial fibrillation) remain a clinical challenge**

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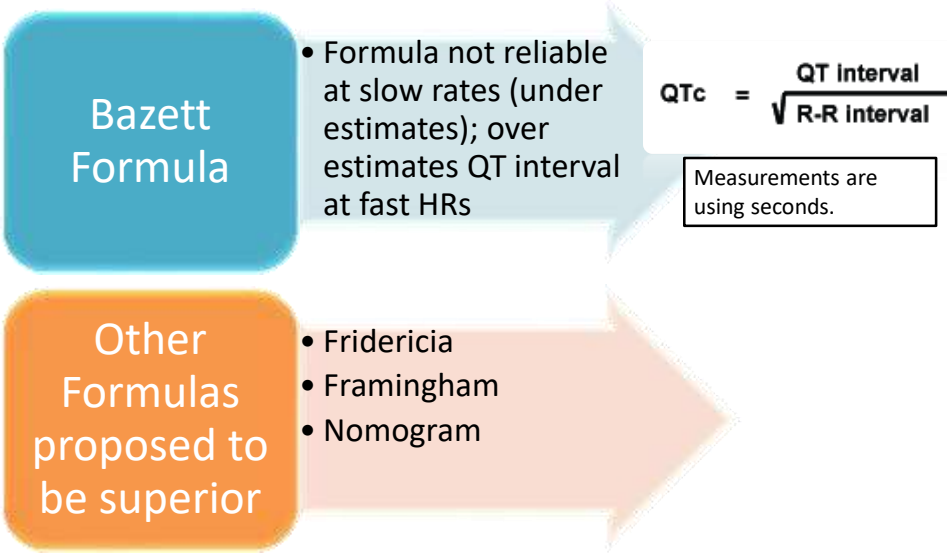

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QT in Practice (QTIP Study)

- QT prolongation > 500 msec common in hospitalized patients (ICU and progressive care) (24%)
- QT prolongation associated with longer length of stay
- QT prolongation associated with almost 3 times the odds for mortality

(Pickham et al., 2012, Critical Care Medicine)

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Cardiac Ion Channel Abnormalities

- Long QT Syndrome (LQTS)
- Brugada disease
- Idiopathic short QT
  - < 300 to 340 msec
- Diagnosed by family history and ECG

Note: Patients with heart failure can develop channelopathies

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LQTS

- QTc > 450 ms
- Genetic defect in either potassium (LQT1 or LQT2) or sodium (LQT3) channels
  - KCNQ1 (LQTS type 1), KCNQ2/HERG (LQTS type 2) and SCN5A (LQTS type 3)
  - Autosomal dominant trait
  - 1 in 2500
  - Delayed repolarization (1 and 2)
  - LQT1 and LQT2 = 95%
    - **Beta blockers (**Nadolol – Type 2)
  - LQT3 = 5%
    - Beta blockers may be harmful (Propanolol; other antiarrhythmics)
    - Events during sleep or episode of bradycardia
- QT prolongation important risk factor for SCD
  - Untreated mortality of 50%
    - QTc < 440 ms / < 5%
    - QTc 460 to 500 ms / 20%
    - QTc > 500 ms / 50%

Each Type of Congenital QT Looks Differently in Terms of T Wave Morphology

- **Interestingly – some acquired Torsade's may be preceded by T wave morphology looking like congenital LQTS**

- Long QT 1: wide, broad-based T waves
- Long QT 2: low amplitude, often notched T waves
- Long QT 3: long ST segment and tall, peaked T waves

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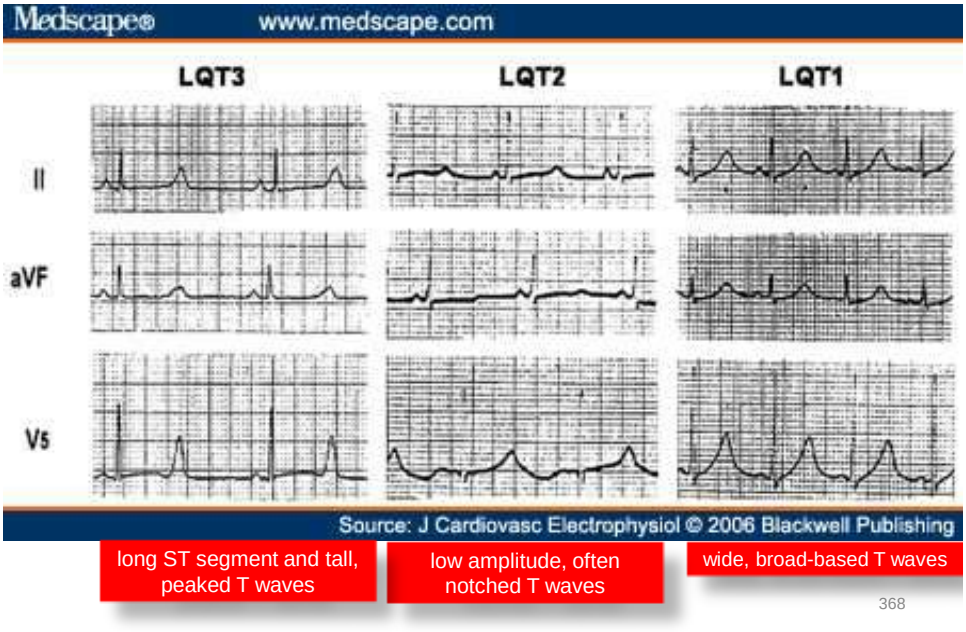
Resting ECG may not show QT prolongation.



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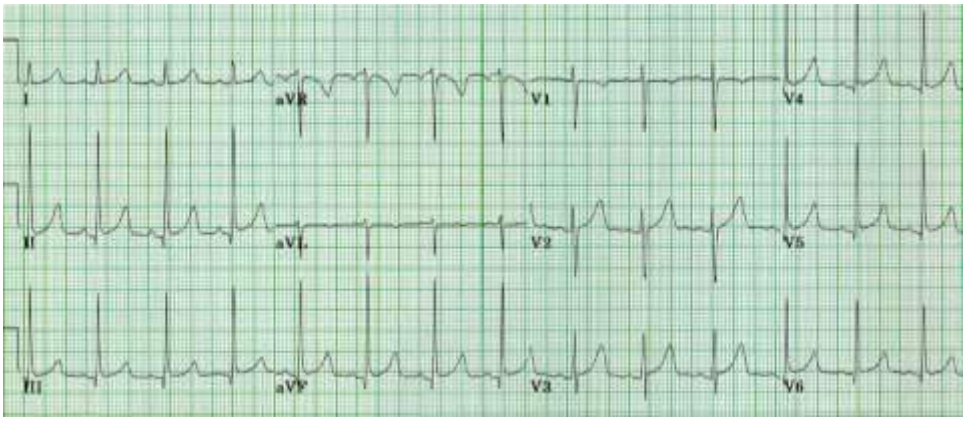
LQT1 episodes in response to exercise of exertion: particularly swimming
Prepuberty males more events than females; female adults more than adult males.



11 year old male LQT1 patient ECG showing a normal T wave pattern and average QTc of about 480 msec.

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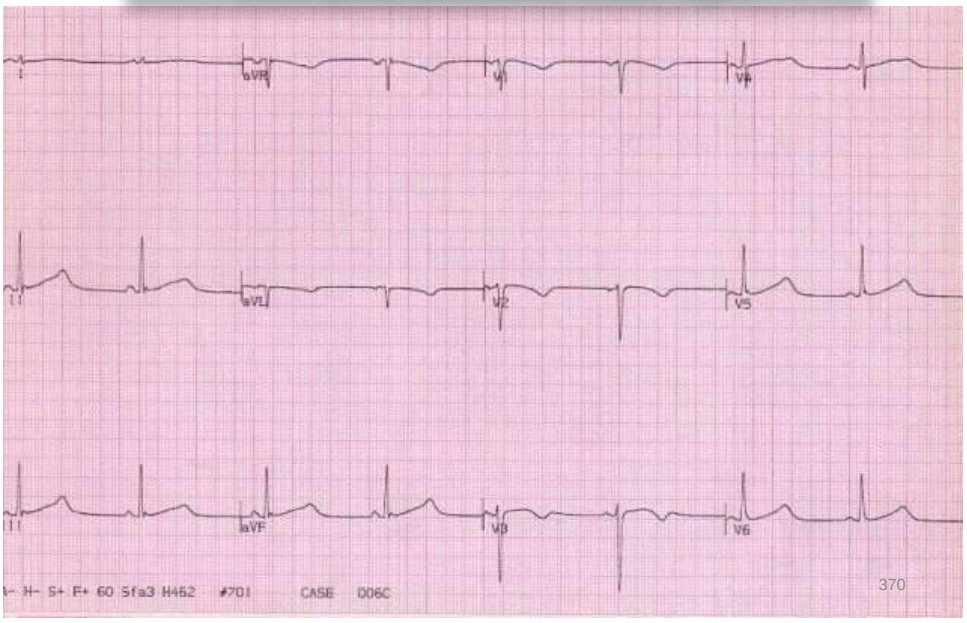
28 year old female with LQT2. Bifid T waves are evident in leads II, III, AVF and particularly V4.
Events typically associated with auditory triggers.



Brugada Syndrome

- Inherited ion channelopathy.
  - Disorder of increased sodium influx
  - Potassium and calcium can also be involved
- Autosomal dominant
  - Most common in Southeast Asian countries
  - 90% of patients are male
- Predispose to Syncope or sudden cardiac death (SCD)
  - Events occur more commonly at rest or during sleep
  - Events occur in 3rd or 4th decade of life
  - Increased risk for SCD
    - Syncopal episode
    - Early repolarization pattern on ECG
    - Family history of SCD
    - Asymptomatic patients at low risk for SCD
- Treatment
  - ICD
  - Quinidine (class 1) or isoproterenol for VT Storm

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Diagnosis of Brugada Syndrome

- 3 characteristic ECG patterns identified
- **If type 1**, 2 or 3 ECG findings are present one of the following must also be present to consider a diagnosis of the BS:
  - Documented ventricular fibrillation
  - Self-terminating polymorphic ventricular tachycardia
  - Family history of sudden cardiac death at < 45 years
  - Type 1 ST-segment elevation in family members
  - Electrophysiologic inducibility of VT
  - Unexplained syncope suggestive of a tachyarrhythmia
  - Nocturnal agonal respiration

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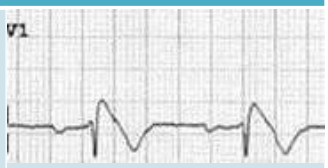
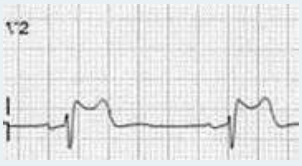
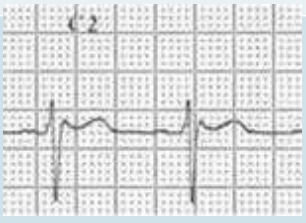
ECG Findings With Brugada Syndrome

Type 1	Coved ST elevation ST gradually descends to an inverted T wave Present in more than one right precordial lead V1-V3 Associated more ventricular events	
Type 2	T wave remains positive or biphasic The terminal portion of the ST-segment is elevated ≥ 1 mm Present in more than one right precordial lead V1-V3	
Type 3	T Wave is positive The terminal portion of the ST-segment is elevated < 1 mm Present in more than one right precordial lead V1-V3	

Source: Chaturvedi et al., 2011.

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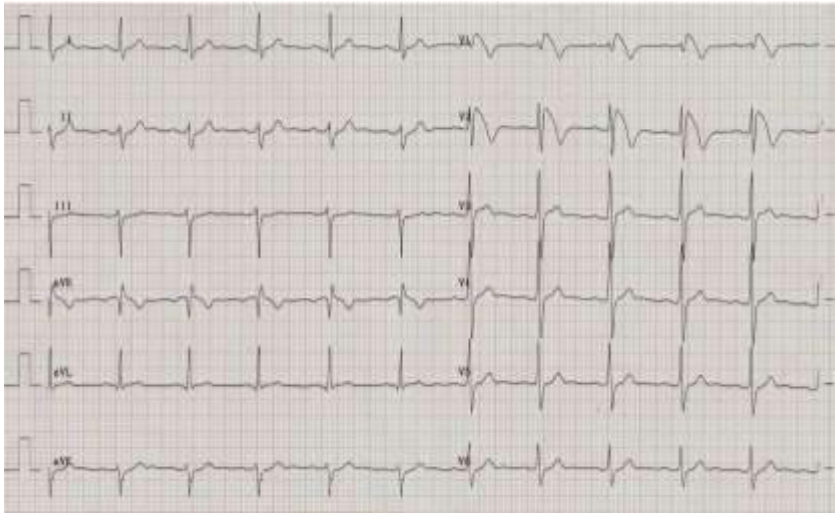

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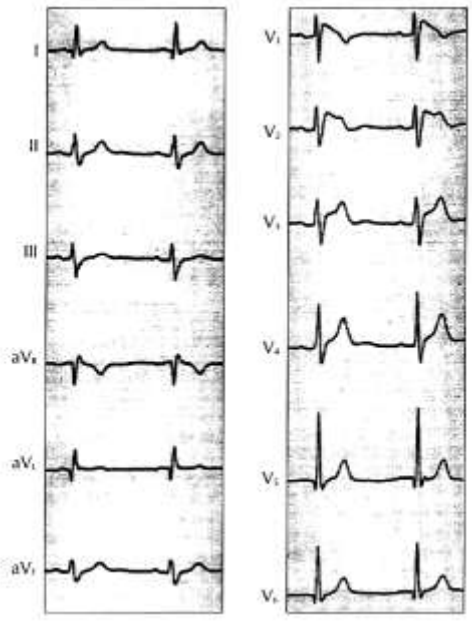

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Torsade's De Pointes

- Recognition of this life-threatening arrhythmia is important because it is not treated like other VTs
- Two groups: Acquired and congenital
- Acquired
  - Drugs prolonging repolarization
    - Most often as a result of blocking the potassium channel
  - Electrolyte abnormalities
    - Low potassium
    - Low magnesium
  - Severe bradycardias / pauses

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Dangers of Abnormal Repolarization

- Places of unequal repolarization can set up for reentrant tachyarrhythmias
- There can be the development of early after depolarizations

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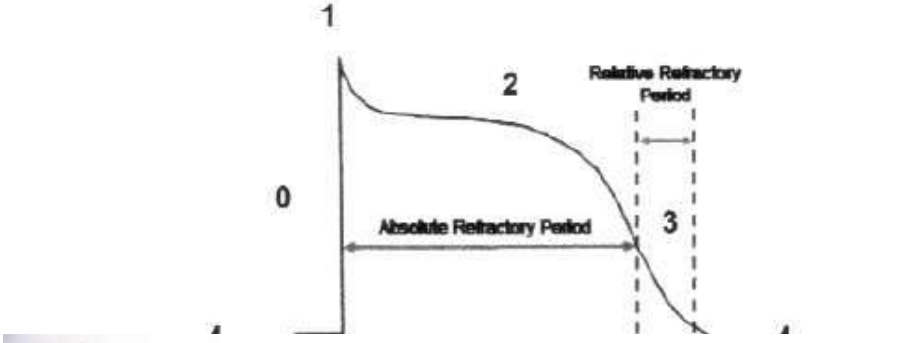

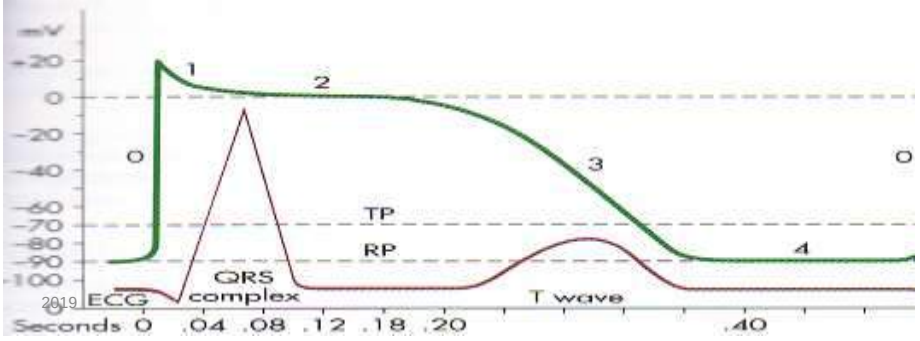

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What are Early After Depolarizations?

- Right after repolarization (or during) there is a transient sub threshold depolarization
  - Can occur during Phase II or III of the cardiac action potential
  - If an early after depolarization reaches threshold a second upstroke occurs and a **triggered** beat follows
  - The triggered beat may have its own after depolarization that reaches threshold – thus causing another triggered beat
- Thought to be etiology of Torsade's de Pointes
  - Acquired
  - Congenital

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More on Early After Depolarizations

- Precipitating Factors
  - Hypokalemia
  - Hypomagnesemia
  - Heightened sympathetic tone
  - Slow heart rate
  - Prolonged repolarization (QT interval)

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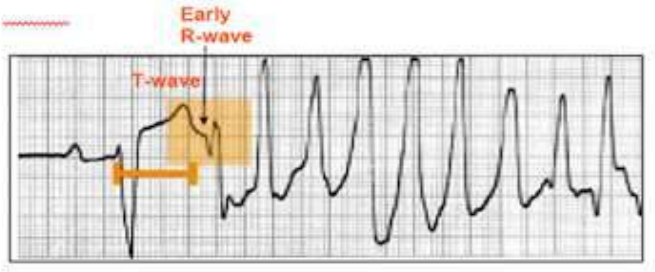

TdP initiated by R-on-T phenomenon caused by LQTS (QT=500ms).

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More on Drugs that Prolong Repolarization (blocking of potassium channel efflux)

- www.QTdrugs.org
- www.torsades.org

- **Class Ia and Class III antiarrhythmics**
- Antihistamines
- Antibiotics
- Antipsychotics
- Antidepressants
- Sedatives
- Gastric motility agents
- Anticancer agents
- Opiate agonists

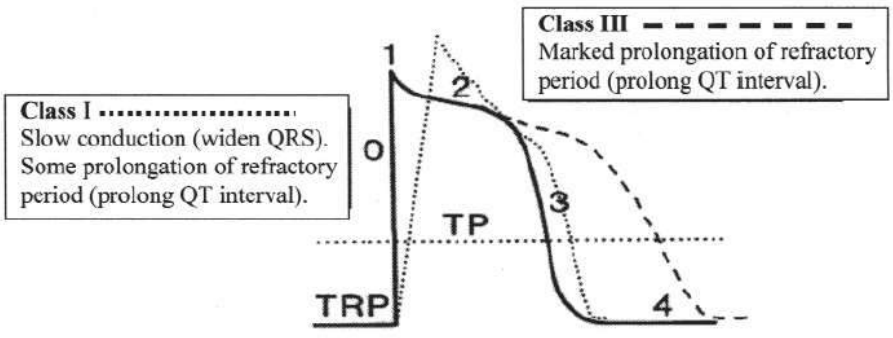
✓Risk
✓Possible Risk
✓Conditional

382

Other Risk Factors for Torsade's de Pointes

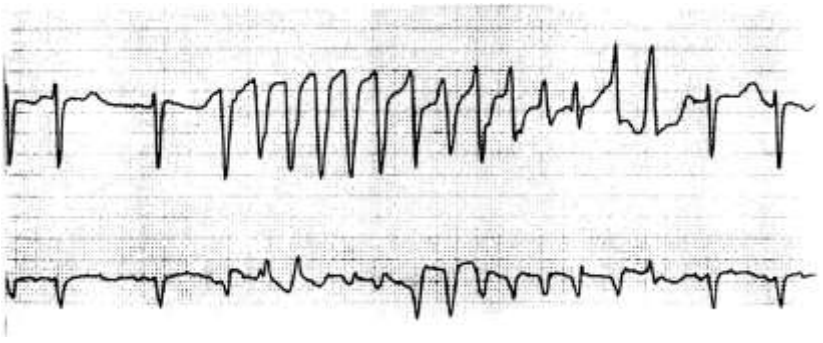
- Rapid (IV) administration of QT prolonging agent
- Renal or hepatic dysfunction
- Female gender (particularly for drug induced)
- Advanced age
- Anorexia
- Heart disease
- Poly pharmacy

383



384

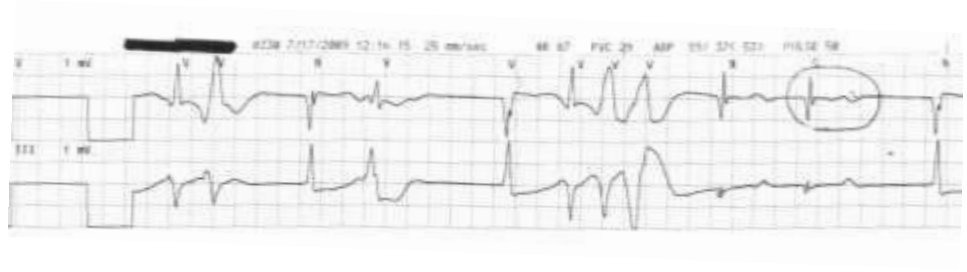
Simultaneous 2-lead ECG (leads II and V1) showing initiation and termination of torsade de pointes in patient in AF after ibutilide infusion.

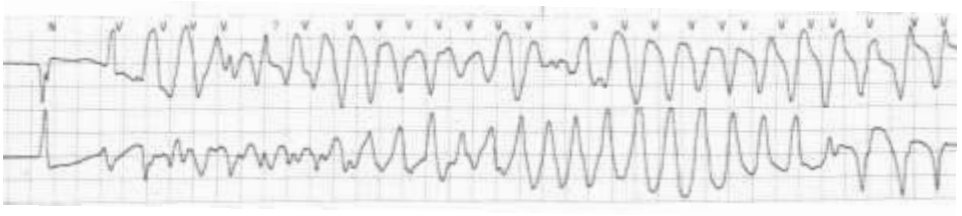


VanderLugt J T et al. Circulation. 1999;100:369-375

Warning signs for Torsades de Pointes

- Increase QTc from predrug baseline of 60 ms,
- Marked QTc interval prolongation .500 ms
- T-U wave distortion that becomes more exaggerated in the beat after a pause
- Visible (macroscopic) T-wave alternans
- New-onset ventricular ectopy, couplets
- Nonsustained polymorphic ventricular tachycardia initiated in the beat after a pause.





387

Torsade's de Pointes

- Class I
  - Discontinue offending drugs
    - Note: Class IA drug induced TdP usually appears soon after the initial administration of the drug
  - Correct electrolytes
    - Magnesium
    - Potassium
  - Increase HR
    - Isoproteronol
      - 2 mcg/min then titrate to HR of 100 beats per minute
    - Temporary pacing at rate of 100 to 110
    - Permanent pacing if bradycardia or CHB cannot be resolved.
- Defibrillation if sustained
  - However, continue to assess for and treat cause

Magnesium is considered treatment of choice.

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More on Magnesium in Torsade's de Pointes

- 2 Gm IV bolus over 1-2 minutes
  - Followed in 15 minutes by another bolus if necessary
  - May start continuous infusion at rate of 3-20 mg/min
- Benefit occurs without shortening of QT interval and in presence of normal Magnesium level

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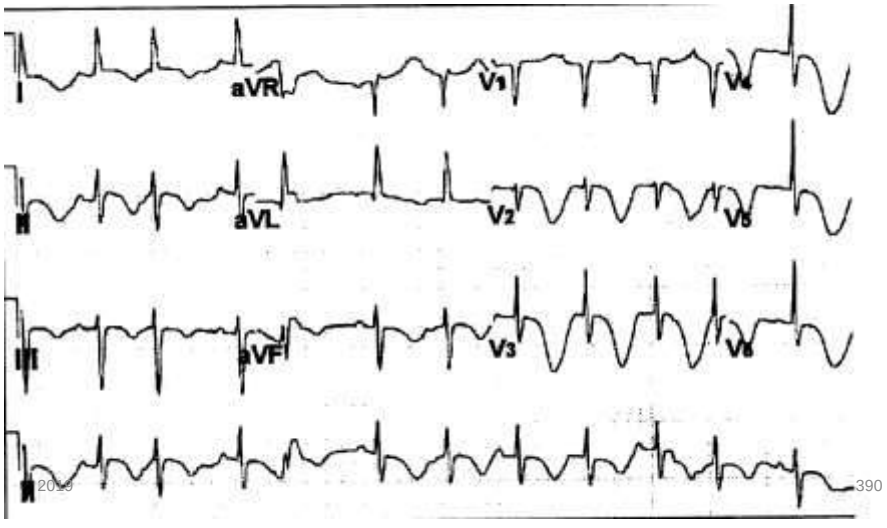
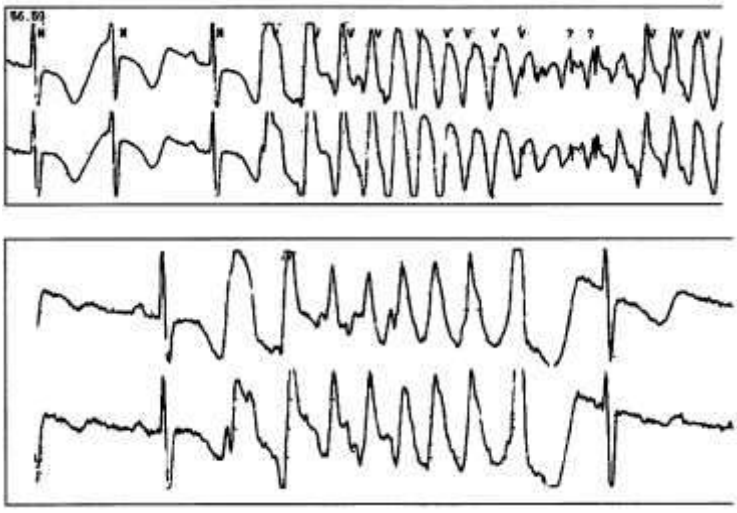
Case Example



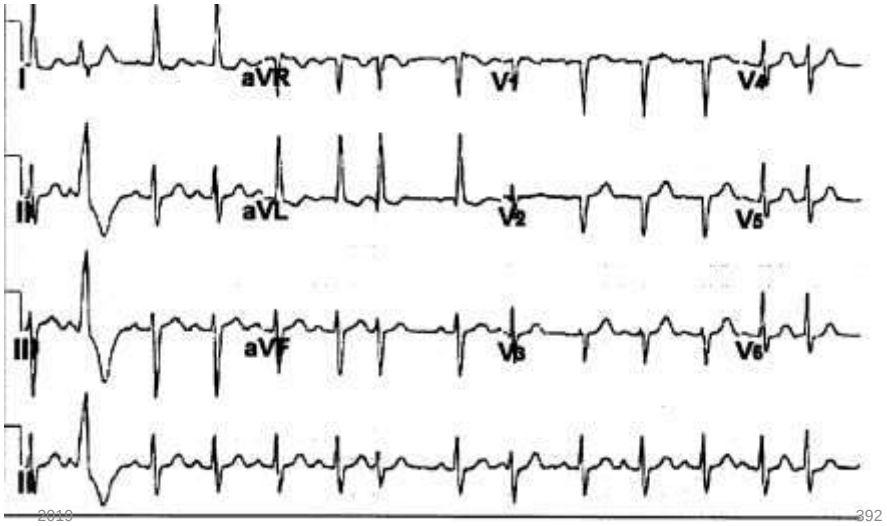
390

Case Example



391

Case Example



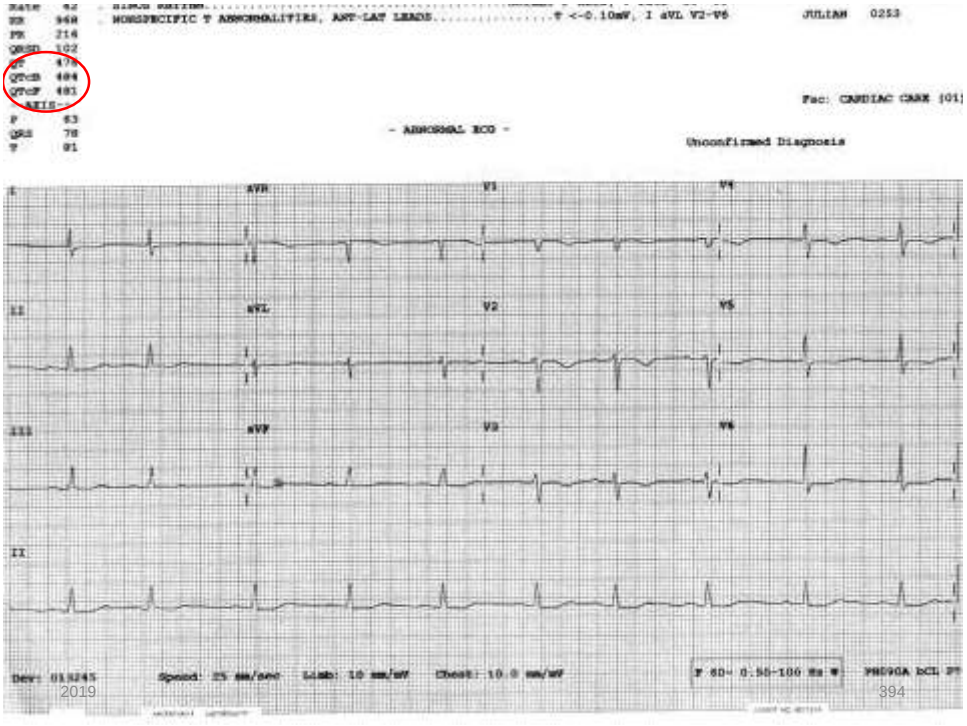
392

QT Interval Case Example

- Patient admitted for syncope after having motor vehicle crash while driving.
- Long standing history of paroxysmal atrial fibrillation – on dofetilide (Tykosin) for several years.
- Recent chemotherapy for breast CA resulting in a reduction of EF.
- Recent increase in carvedilol and lisinopril per general cardiology to improve EF.

- Next slide is admission ECG. Note the QTc interval..

393



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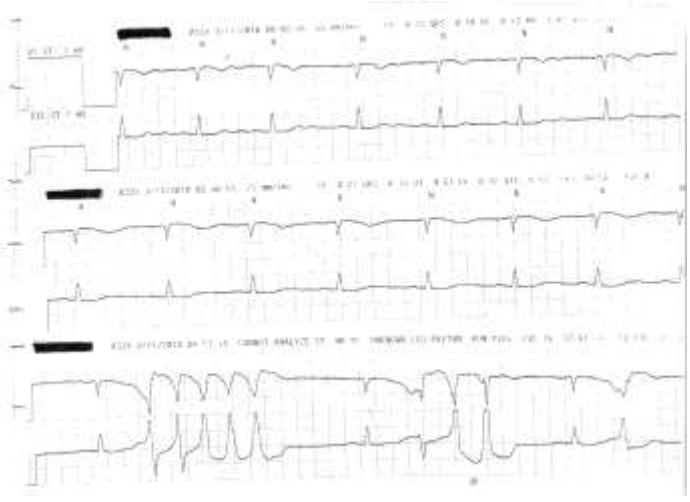
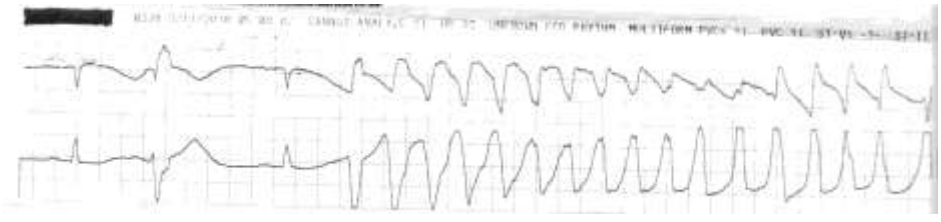


1. Strip 1: QTc consistent with admission ECG.
2. Strip 2: Marked QTc prolongation when patient asleep.
3. Initial run of ventricular tachycardia initiated by PVC firing at end of T wave,

395



Same patient with sustained Torsades de Pointes. Treated effectively with 2 grams IV Magnesium (magnesium level was normal at baseline). Magnesium is the drug of choice to stabilize the cardiac membrane. Dofetilide (Tikosyn) was also discontinued.

Note: Although the patient had been on dofetilide (Tikosyn) for several years, the recent change in ejection fraction and increase in beta blocker therapy increased her risk for Torsades de Pointes.

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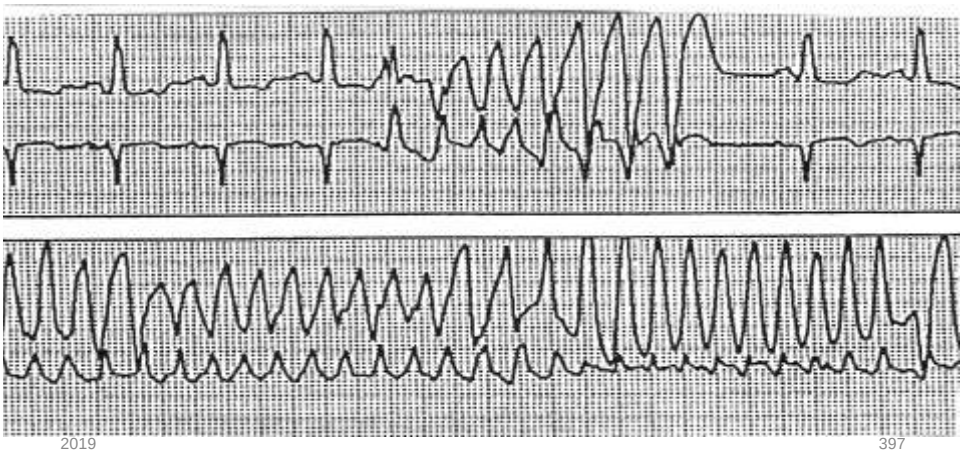
Polymorphic VT with normal QT:

- Seen frequently in ischemic conditions
  - Think revascularization
  - Think beta blockers



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Special Considerations: Polymorphic VT (normal QT)

- DC cardioversion with sedation when unstable
- IV beta-blockers if ischemia suspected
  - Improve mortality
- IV amiodarone in absence of abnormal repolarization
  - Amiodarone better than placebo
  - Magnesium not better than placebo
- Urgent angiography to exclude ischemia
- Lidocaine may be reasonable if ischemia suspected
- Check electrolytes
- Consider any other potential reversible cause

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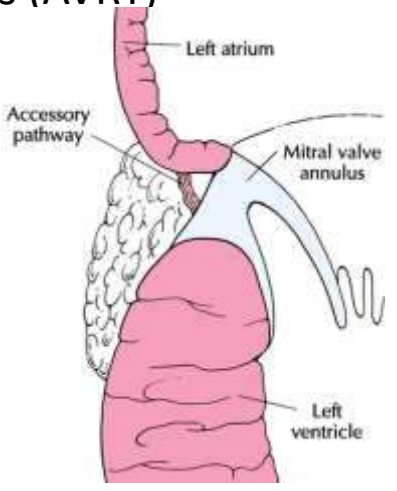
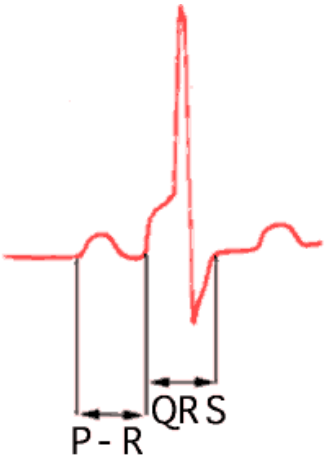
Atrioventricular Reciprocating Tachycardias (AVRT)

- Requires the presence of a bypass tract or accessory pathway
- Most common: Kent bundles in "Wolf Parkinson White" Syndrome
- Left lateral free wall, right lateral free wall, and posterior septum



Concept of Pre-excitation

- Termed Pre-excitation because some conduction occurs via the Kent bundles in addition to the normal pathway; because conduction via the Kent bundles is faster than via the AV node the ventricles are pre-excited
- **This produces a "delta wave" on the EKG**
- Fusion beat
  - Short PR
  - Wider than normal QRS



WOLFF-PARKINSON-WHITE (WPW) SYNDROME

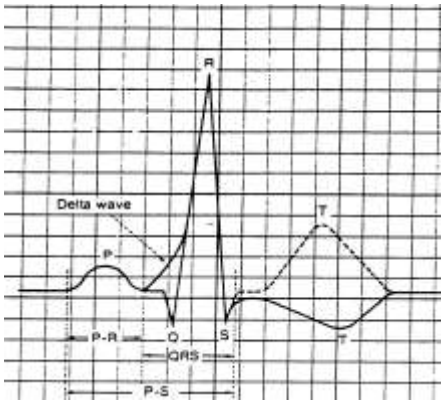
Evidence of Pre-excitation in SR

Can cause dyssynchrony and LV dysfunction from premature septal activation

Known SVT or symptoms of SVT

401

Delta Wave of Pre-excitation Syndrome

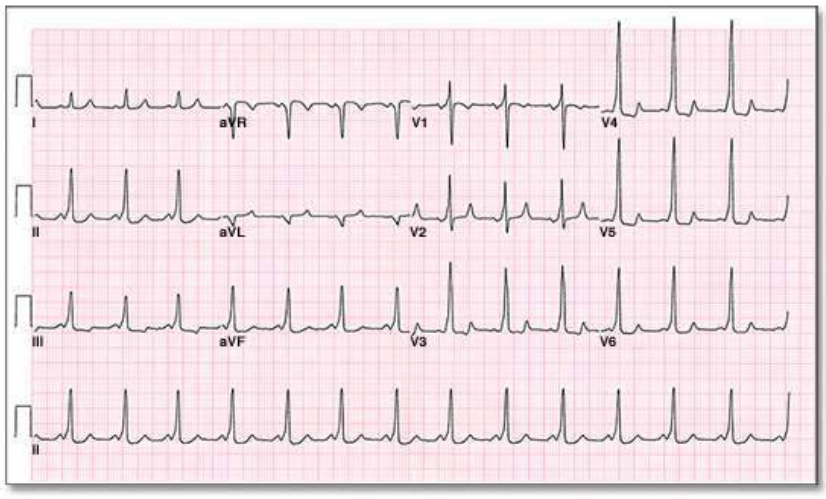


✓60 to 70% of WPW shows evidence in SR

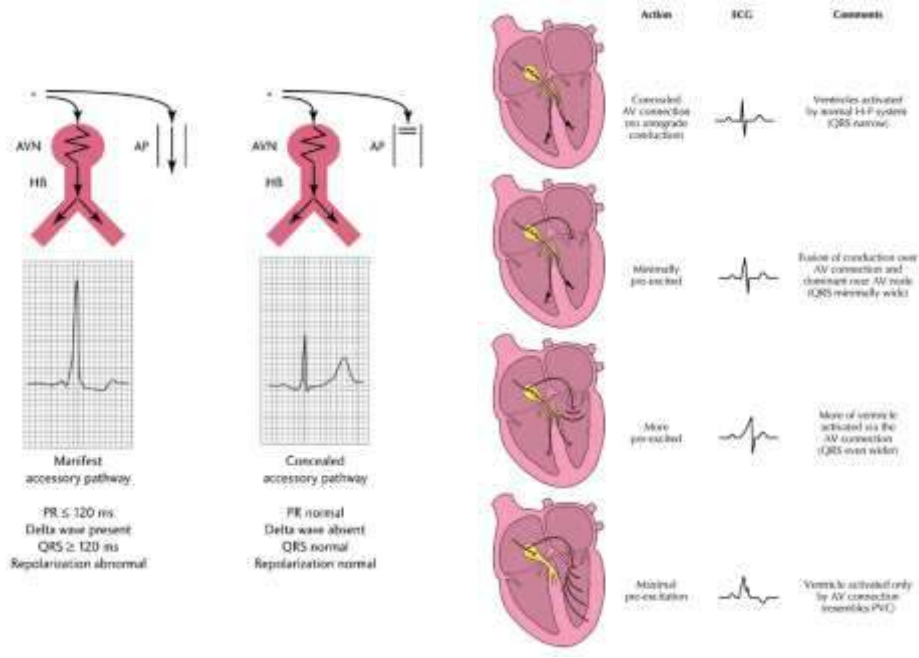
Left sided accessory pathway:
Positive delta wave in V1

Right sided accessory pathway:
Negative delta wave in V1

402


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

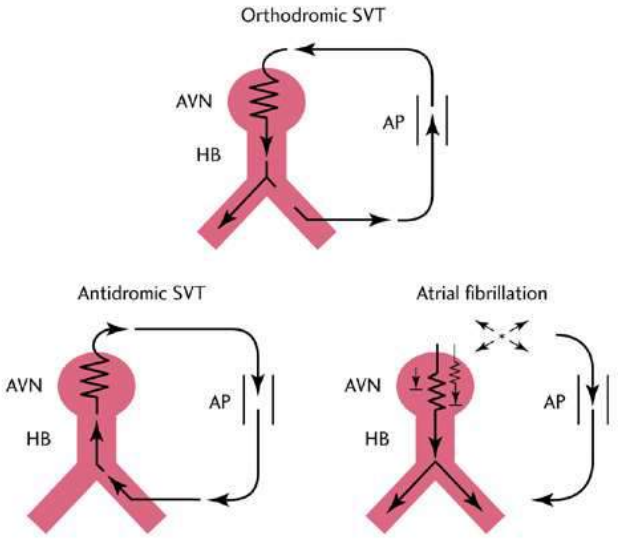
403


AVN
AP
HB
Manifest
accessory pathway
PR ≤ 120 ms
Delta wave present
QRS ≥ 120 ms
Repolarization abnormal
AVN
AP
HB
Concealed
accessory pathway
PR normal
Delta wave absent
QRS normal
Repolarization normal
Action
ECG
Comments

404

Arrhythmias of WPW

(AVRT or CMT)



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Atrioventricular Reentrant Tachycardia (AVRT)

- **Orthodromic**
  - 90 to 95%
  - Traveling down the AV junction and up an accessory pathway
  - Sequential depolarization
  - Narrow because travel via the AV node
  - More common than antidromic tachycardia
- **Antidromic**
  - 5%
  - Activation of the ventricles is initiated by impulses descending via an accessory pathway
  - Ventricular depolarization begins at an ectopic site in the myocardium and returns via the AV node

Presence of pre-excitation on 12 lead and paroxysmal palpitations.

406

Orthodromic Tachycardia

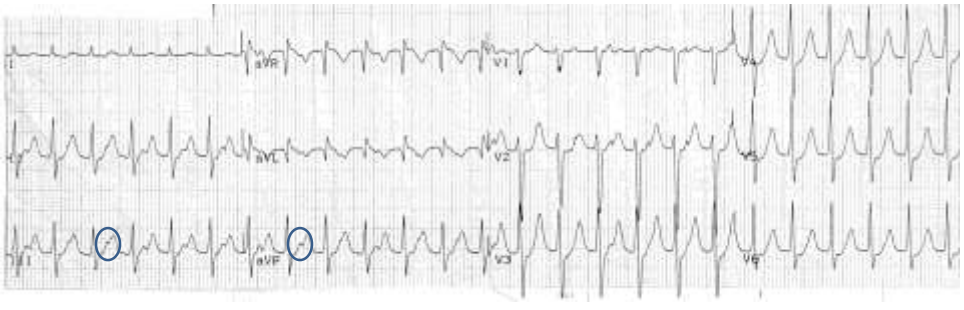
This WPW tachycardia can mimic AVNRT (most common form of SVT).

In AVNRT ‘p’ waves are usually buried in the QRS.

In orthodromic tachycardia the ‘p’ wave is typically distinct from the QRS.

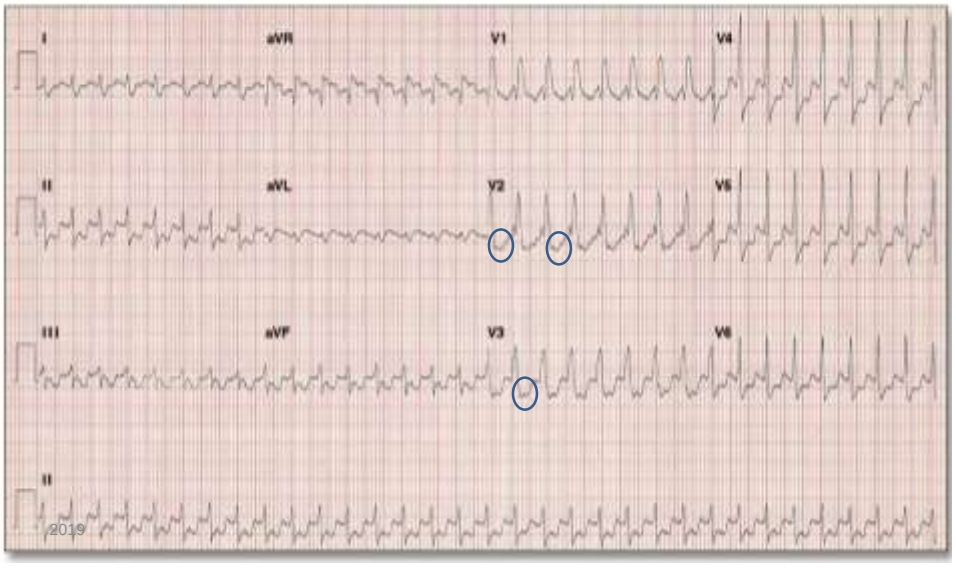
Short RP interval

407



408

62 year old male presenting to ED 4 hours after onset of palpitations. BP 110/72, pale and anxious.



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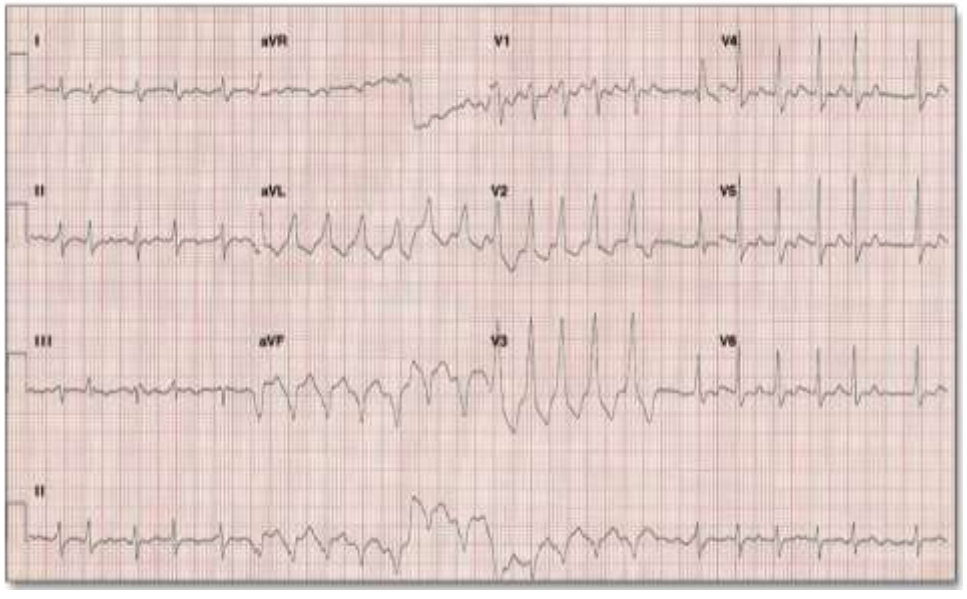

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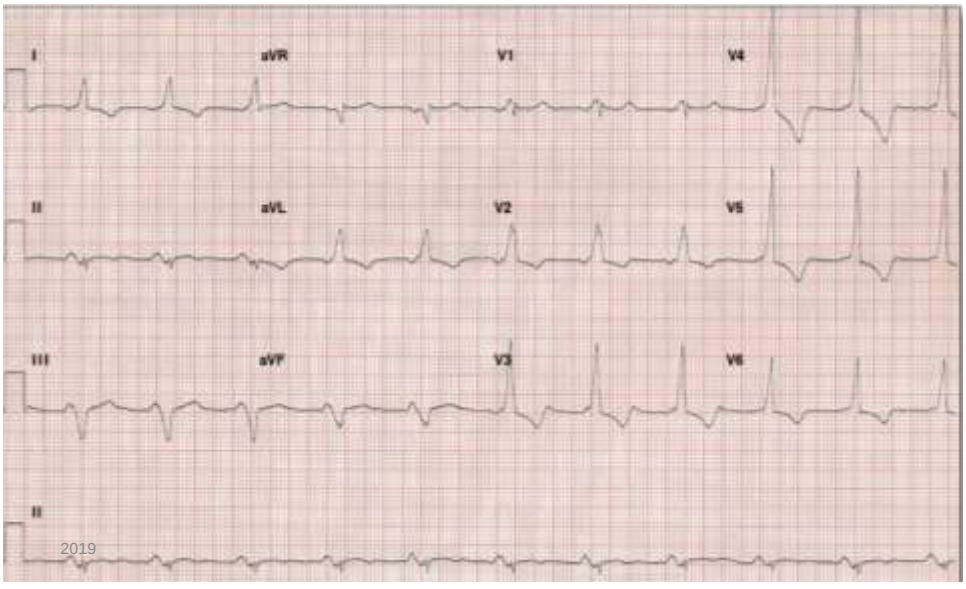

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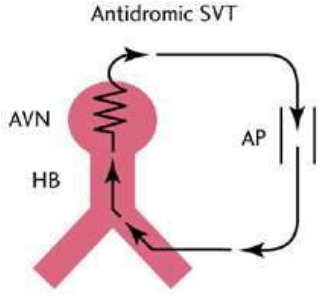
Antidromic Tachycardia

- The less common form of atrioventricular reentrant tachycardia
- The path of tachycardia passes from the atrium to the ventricle via the accessory pathway (Kent bundles) and returns to the atrium via the AV node
- The QRS complex is wide because antegrade conduction bypasses the AV node
- Antidromic tachycardia is very difficult to distinguish from ventricular tachycardia because ventricular depolarization begins where the accessory pathway enters the ventricle
    - Negative concordance will not be antidromic tachycardia



412 2019

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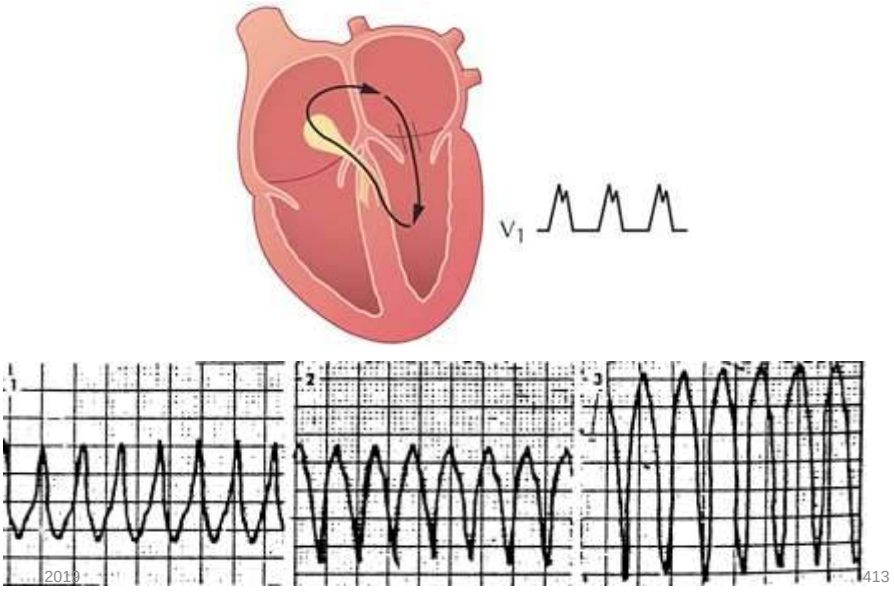
Antidromic Tachycardia



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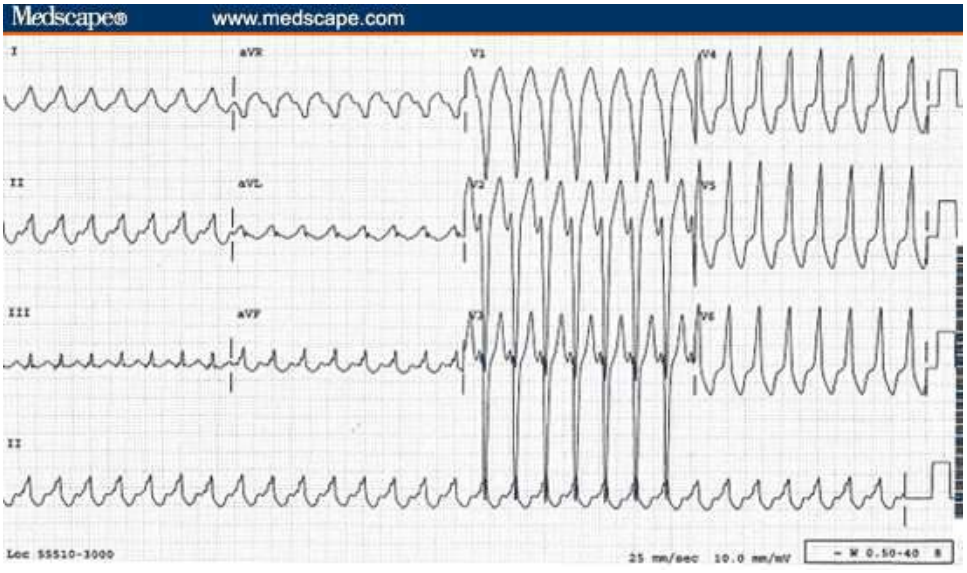

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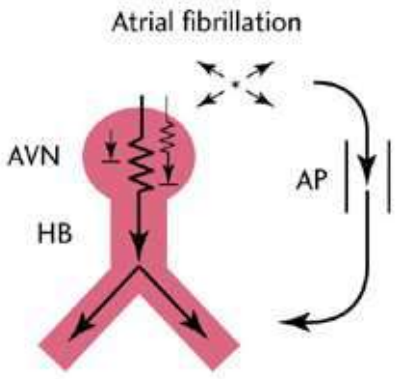
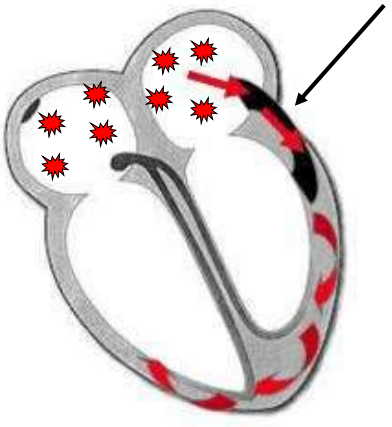
WPW and Atrial Fibrillation

- Mechanism of Action
  - Development of Atrial Fibrillation in WPW
    - 10-32% of patients
  - Refractory period of accessory pathway is short



415

AF in WPW



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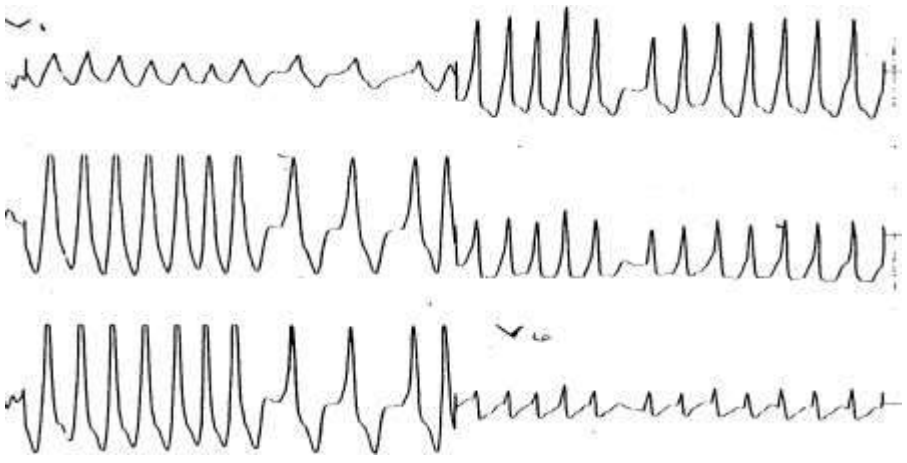

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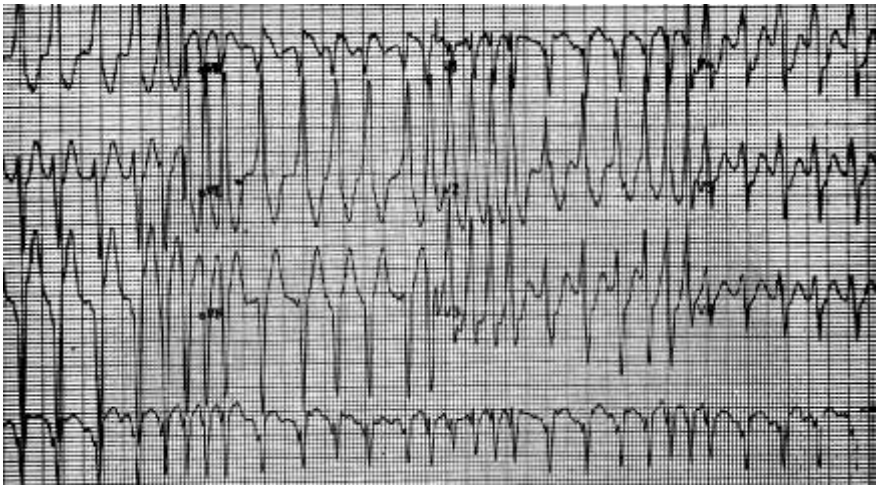

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Treatment for WPW Tachycardias

- AV Reentrant (orthodromic)
  - Can target AV node or accessory pathway
- AV Reentrant (antidromic)
- Atrial Fib with antegrade conduction over accessory pathway
  - Cannot slow conduction through AV node

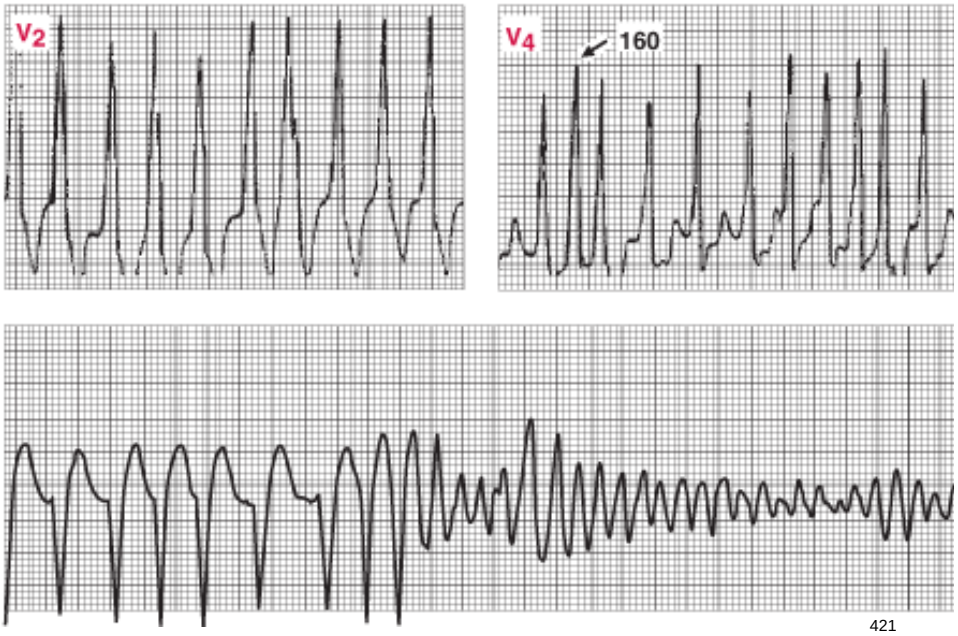
Slow conduction over accessory pathway:
- Procainamide
- Flecainide
- Sotalol
- Propofenone
- Ibutelide

- **Lethal Outcome After Intravenous Administration of Amiodarone in Patient with Atrial Fibrillation and Ventricular Preexcitation**

- MUJOVIĆ NEBOJŠA M.D.[1],
- SIMIĆ DRAGAN M.D.[1],
- ANTONIJEVIĆ NEBOJŠA M.D.[1] and
- ALEMPIJEVIĆ TAMARA M.D.[2]

- **Journal of Cardiovascular Electrophysiology**
- Volume 22, Issue 9, pages 1077–1078, September 2011

- Article first published online: 18 FEB 2011
- DOI: 10.1111/j.1540-8167.2011.02013.x



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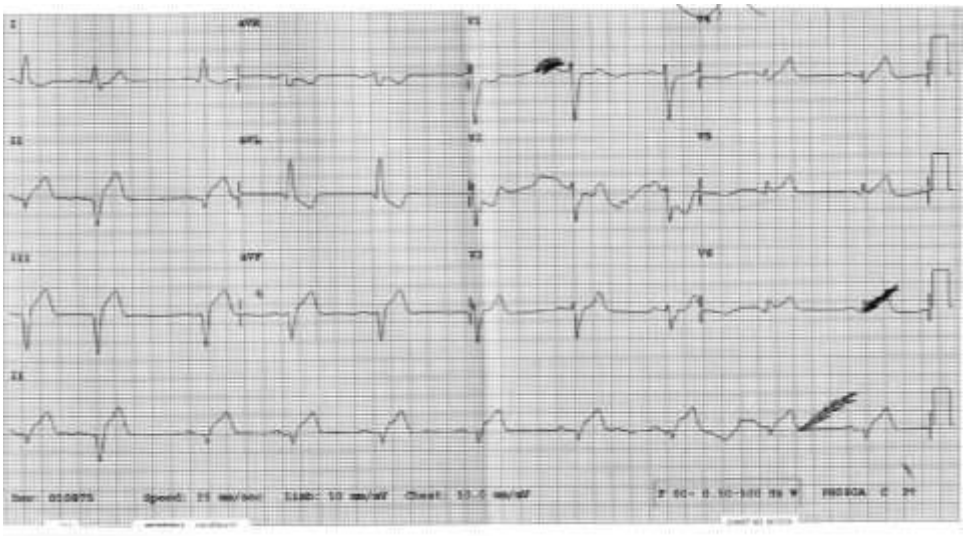
421

LET'S PULL IT ALL TOGETHER!!

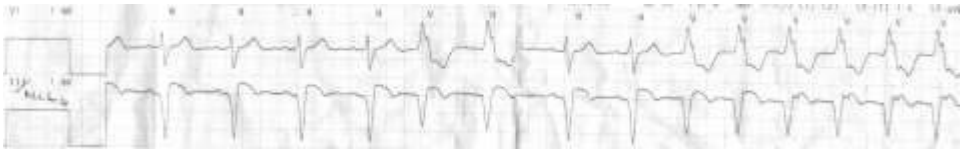
422

Final Case Study: ECG #1



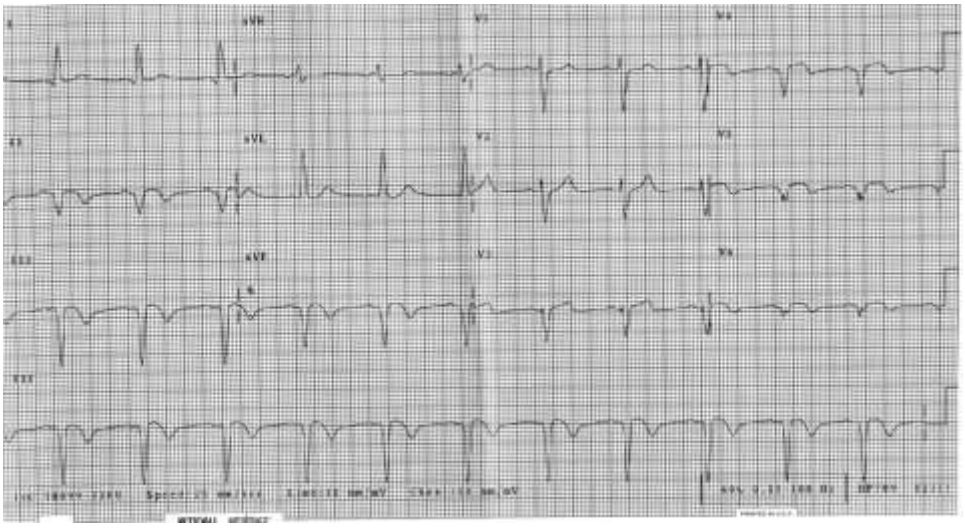
423

Final Case Study: ECG #2



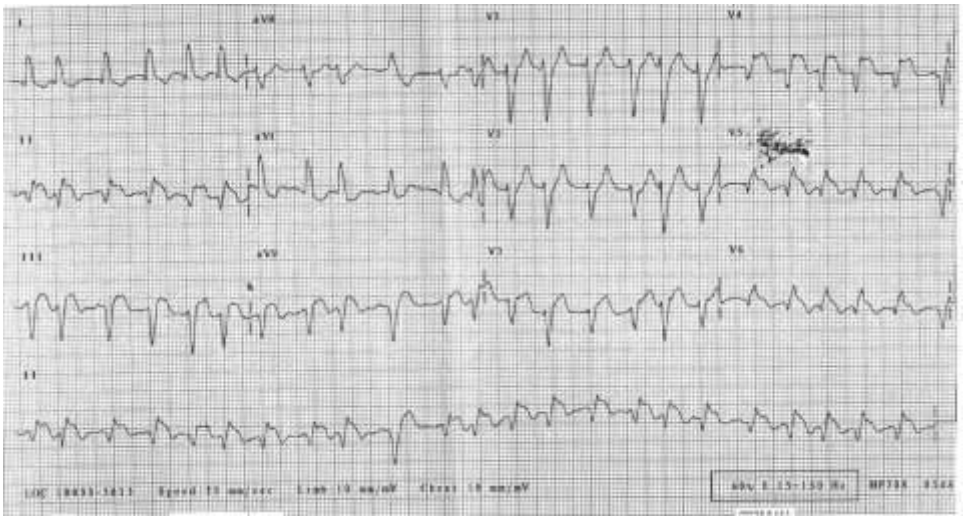
424

Final Case Study: ECG #3



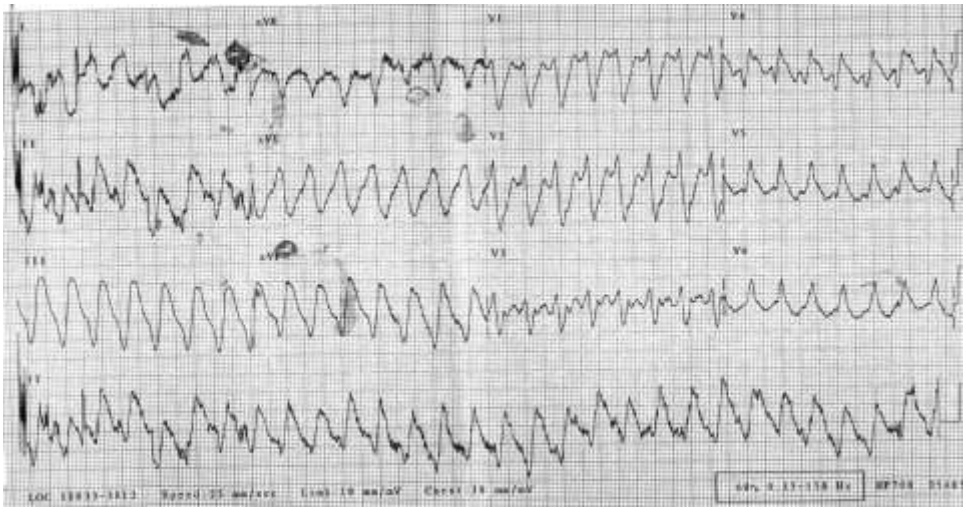
425

Final Case Study: ECG #4



426

Final Case Study: ECG #5



427

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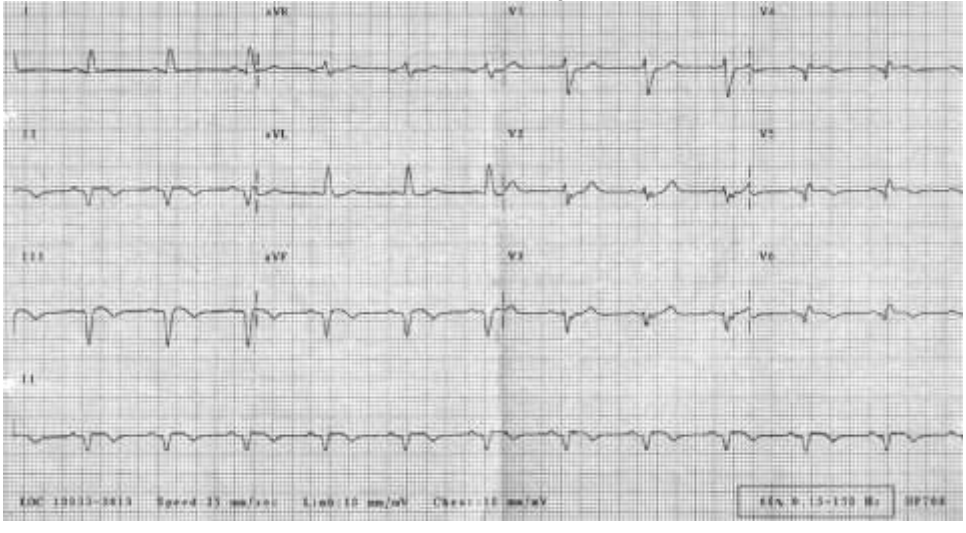

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Class Lesson Summary !

The 12 lead ECG in an inexpensive, noninvasive, risk free diagnostic tool, that can provide you with an amazing amount of information about your patient's heart.

However: Always treat the patient not the ECG or rhythm strip.

429

A Final Thought

Practice with joy. Positively impact every patient and family on their journey and provide safe passage by meeting them where they are, connecting with them in a meaningful way, and delivering care with wisdom and intention.

- Karen and Cindy

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THANK YOU!!!

Enjoy NTI!

BE THE BEST THAT YOU CAN BE EVERY DAY.
YOUR PATIENTS ARE COUNTING ON IT!

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karen@cardionursing.com

Handouts will be available for download at www.cardionursing.com by end of NTI.

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