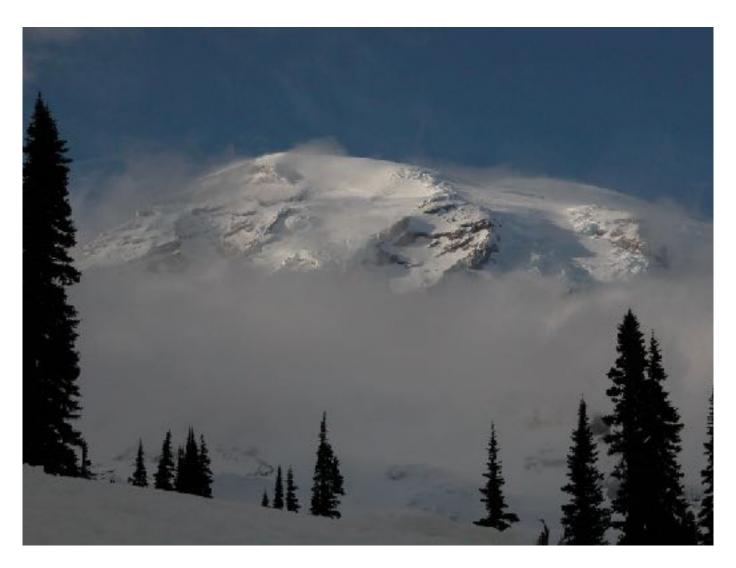
EKG Morphology Lecture



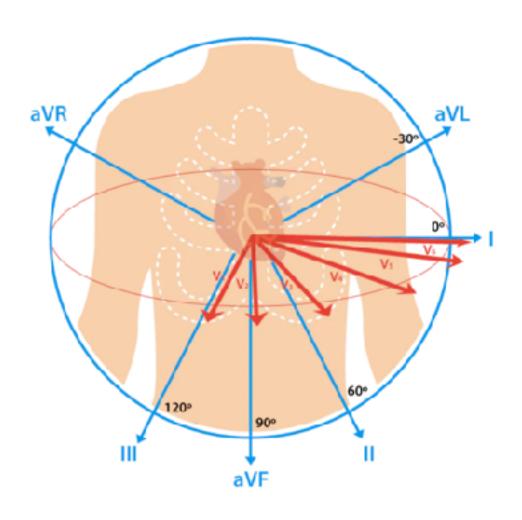
The "P's" to Practice by

Recap from last session: First 2 Steps

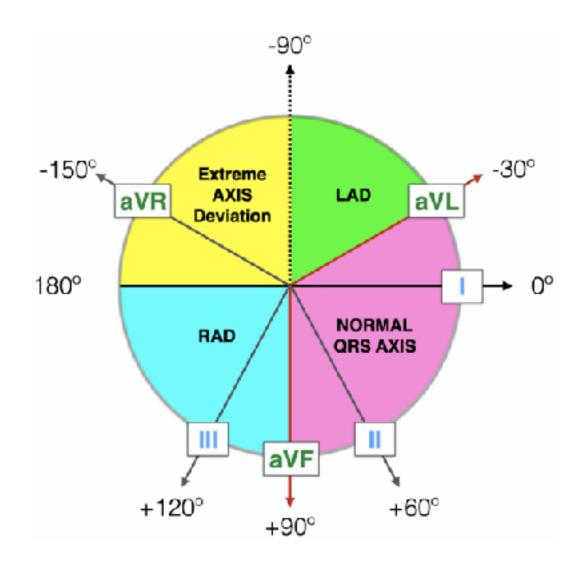
- Step 1: What is the heart rate?
 - What are the 2 methods we discussed?

- Step 2: What is the rhythm?
 - What are the 3 questions we ask to determine rhythm?

Step 3: Determining Axis



Normal vs Abnormal Axis



How do we determine axis?

+I & +avF

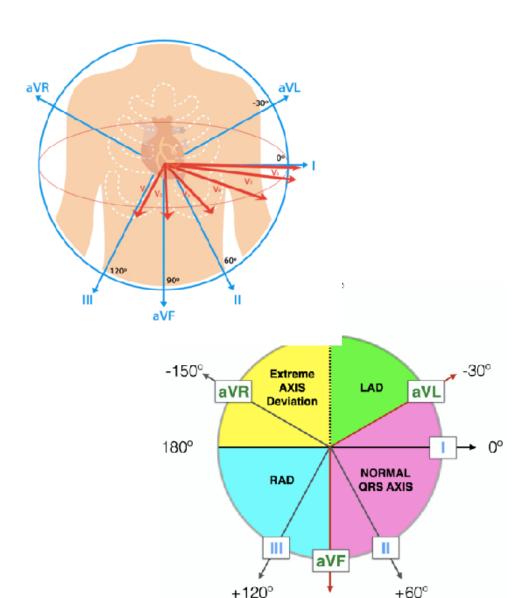
- Normal Axis
- "2 thumbs up"

+I & -avF

- Look at lead II
 - "Negative in II to be TRUE left axis deviation"
 - If +II, then normal axis

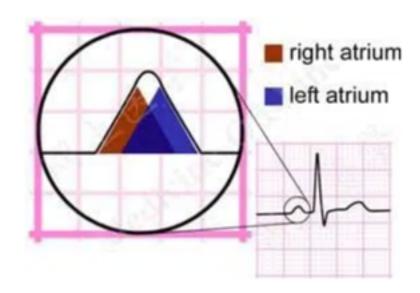
-l and +avF

Right axis deviation



P-wave Morphology

- Atria are small so voltage generated is also small
- Amplitude of p wave: usually < 0.25mV or 2.5mm (aka 2.5 small boxes)
- Atria depolarize from Right to Left



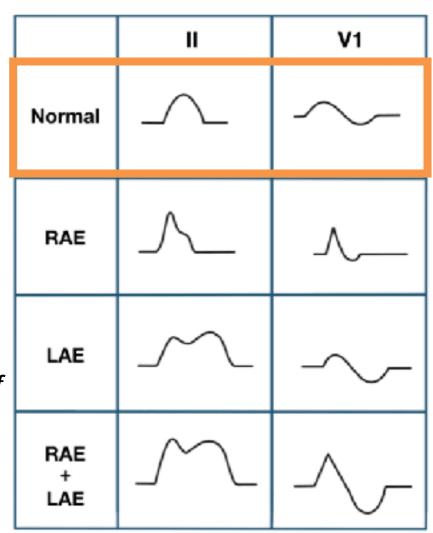
Leads for P-wave Morphology

Lead II

- Nearly parallel to flow of current through atria so records largest positive deflection
- Very sensitive to perturbation in atrial depolarization

Lead V1

- To right of sternum
- Oriented perpendicularly to flow of current
- Biphasic and allows easy separation of right and left atrial components



https://litfl.com/wp-content/uploads/2018/08/P-wave-morphology-LAE-RAE-BAE-Wagner-2007.png

P-wave Morphology

Right Atrial Enlargement

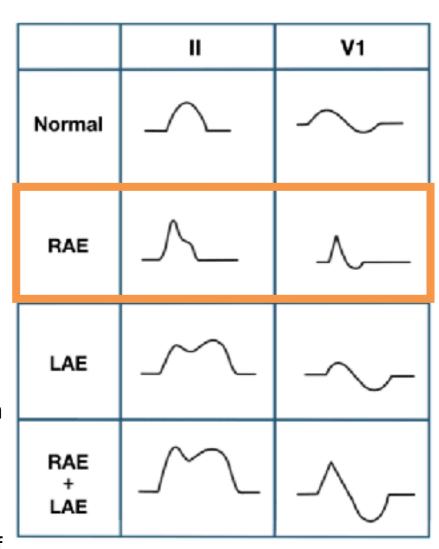
- Width doesn't change because terminal component of p wave is Left Atrial in origin
 - Prolonged depolarization of RA hidden by LA portion of p wave (eg while RA taking longer to depolarize don't see as LA is depolarizing at that time)

Lead II

 P wave amplitude TALLER (> 2.5mm/ small boxes) as parallel to vector which is larger given larger Right Atrium

Lead V1

 Peaked initial portion (reflects R-side of heart as R-side more anterior)



https://litfl.com/wp-content/uploads/2018/08/P-wave-morphology-LAE-RAE-BAE-Wagner-2007.png

P-wave Morphology

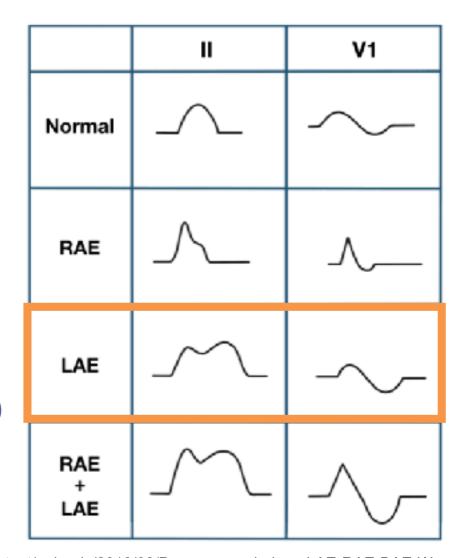
Left Atrial Enlargement

· Lead II

Increased duration of p-wave
 (>2.5 small boxes) since terminal
 portion of p wave represents Left
 Atrial depolarization (which will
 take longer time-wise as Left
 Atrium larger) >2.5 small boxes

Lead V1

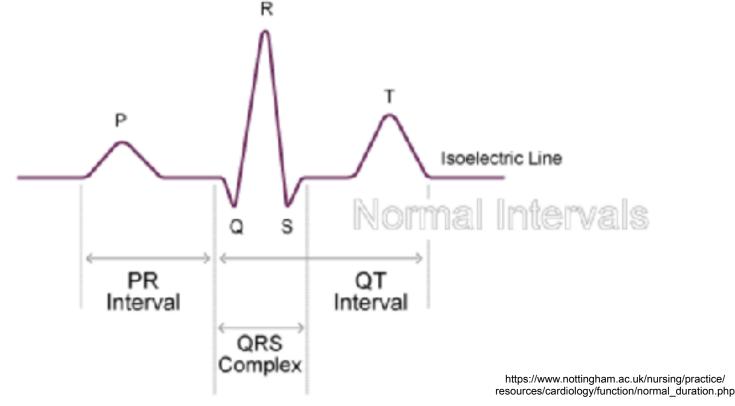
Will see drop >1mm (1 small box)
 below isoelectric line since V1
 overlies the R heart



PR Interval

Definition of PR Interval: Beginning of p-wave to beginning of QRS

Normal Duration: <0.2 seconds (< 1 big box)



What are the definitions of 1st, 2nd and 3rd degree AV blocks?

46years Female Caucasian 62in 1441b€

88 bpm Vent. rate PR interval 102 ms QRS duration 82 ms QT/QTc 346/419 ms P-R-T saves 42 -76 57

ID: 30531482376

55 MD08

Rate **Rhythm** Axis

6-Jan-1998 16:22:48

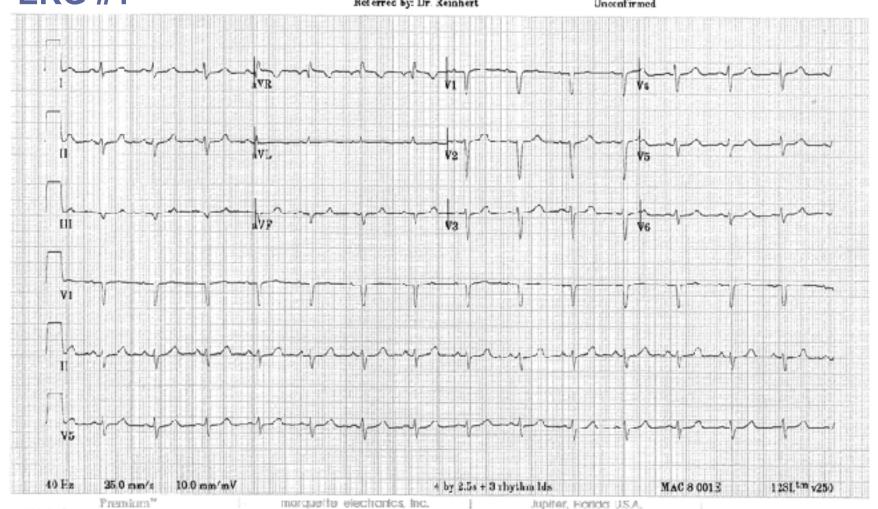
P wave morphology-?Atrial Enlargement PR interval

Technician: SB

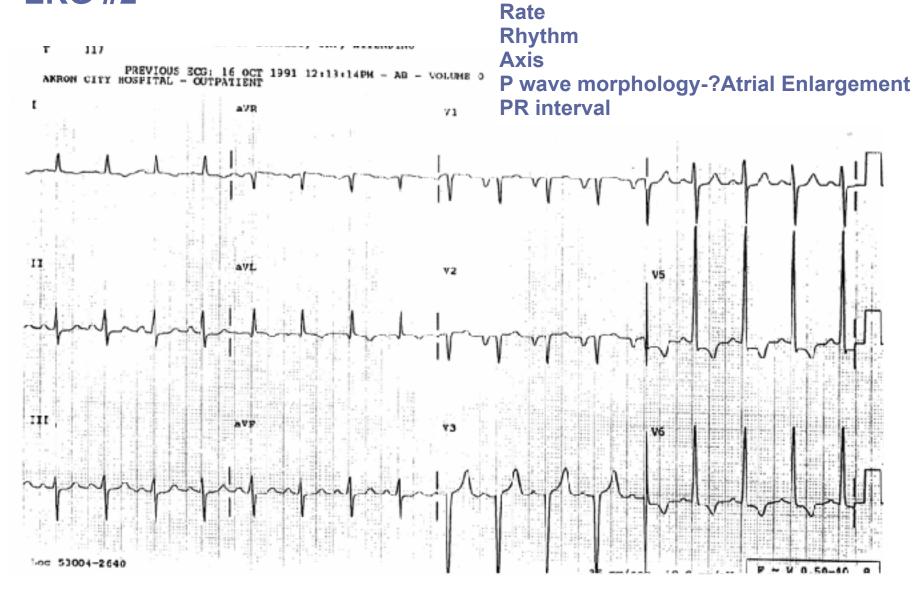
EKG #1

Referred by: Dr. Reinhert

Unconfirmed



EKG #2

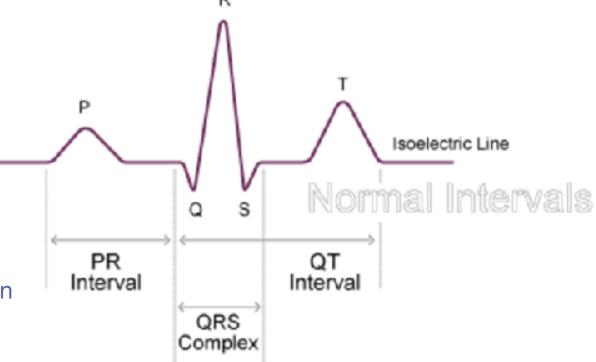


QRS Complex

- If 1st deflection is downward
 - Q wave

- The 1st deflection upward
 - R wave

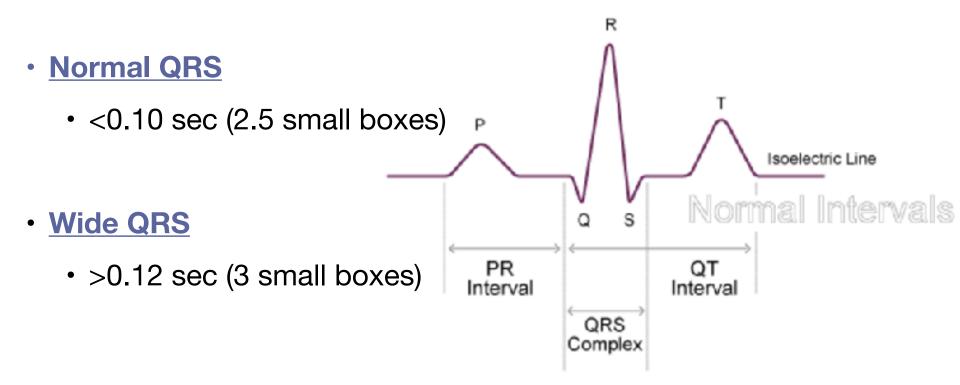
- The 1st downward deflection following an upward deflection
 - S wave



- If there is a 2nd upward deflection
 - · R' wave

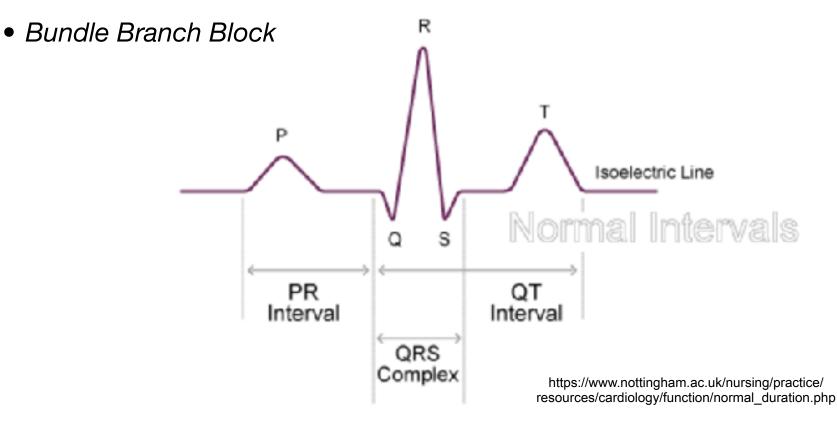
QRS Complex

 Most of what is seen on EKG represents Left Ventricle (since LV = 3x mass of RV)

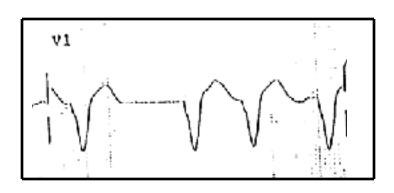


QRS Complex

- Causes of wide QRS
 - Beat originating in ventricle
 - Supra-ventricular beat conducted aberrantly



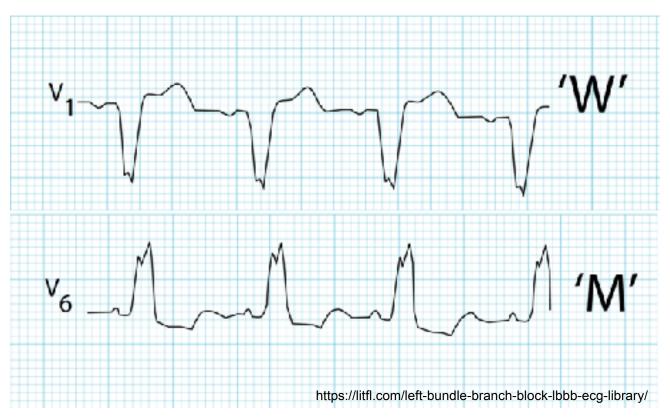
- Wide QRS: >0.12sec (3 small boxes)
- Lead V1: Deep S wave (qS or rS)
 - Why?
- "Left behind in a hole"





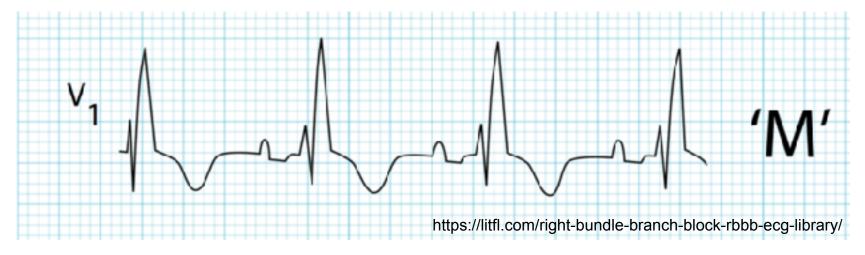
Lead V6:

• Overlies the LV so wider R wave because LV taking longer to depolarize



• Difficult to assess/interpret ST elevations in LBBB (can interpret in RBBB)

- Wide QRS: >0.12sec (3 small boxes)
- <u>Lead V1</u>: M shaped QRS ("bunny ears" / rSR')
 - Second R' represents RV finally depolarizing after LV has finished
 - Positive wave because V1 overlies the RV



<u>Simple rule</u>: if you see a wide QRS and a prominent R' wave in V1 it is a RBBB

R is for Right Bundle Branch Block

What would you expect to happen to "S" wave in lead V6 then?

- Wide QRS: >0.12sec (3 small boxes)
- Lead V6: Wide S at end of QRS
 - Late RV depolarization causes reciprocal late deep S waves
 - -V6 along midaxillary line and so RV depolarization opposite direction/negative





Left Ventricular Hypertrophy

- Determining LVH by EKG
 - Useful but imperfect tool for detecting LVH
 - Inexpensive and widely available
 - Limitations
 - Moderate sensitivity
 - So if not meeting criteria, doesn't rule out LVH
 - Higher specificity
 - If evidence of LVH on EKG by certain criteria, then higher likelihood LVH is actually present

EKG Findings of LVH

- Increased QRS Voltage
- Increased QRS duration ("kinda wide" not BBB)
- Leftward axis given larger LV mass
- Left atrial enlargement
- "Repolarization" abnormality in ST-T's

Left Ventricular Hypertrophy

- Two main voltage rules
 - S wave in V1 plus R wave in V5 or V6 ≥ 35mm (≥35 small boxes)
 - More sensitive

- R wave in aVL ≥ 11mm (≥11 small boxes)
 - More specific

Romhilt-Estes Point Score System

Criterion	Points
Any limb R wave or S wave ≥2.0 mV (20 mm)	3
OR S in V1 or S in V2 ≥3.0 mV (30 mm)	
OR R in V5 or R in V6 ≥3.0 mV (30 mm)	
ST-T wave changes typical of LVH	
Taking digitalis	1
Not taking digitalis	3
Left atrial abnormality	
P terminal force in V1 is 1 mm or more in depth with a duration 40 milliseconds (0.04 seconds)	3
Left axis deviation ≥-30°	2
QRS duration ≥90 milliseconds	1
Intrinsicoid deflection in V5 or V6 ≥50 milliseconds (0.05 seconds)*	1

A score of 5 or more indicates "definite" LVH; a score of 4 indicates "probable" LVH.

ECG: electrocardiogram; LVH: left ventricular hypertrophy.

* Intrinsicoid deflection is defined as interval between beginning of QRS interval and the peak of the R wave.

Q Waves

<u>Definition</u>: Considered Q wave if 1st deflection of QRS is downward

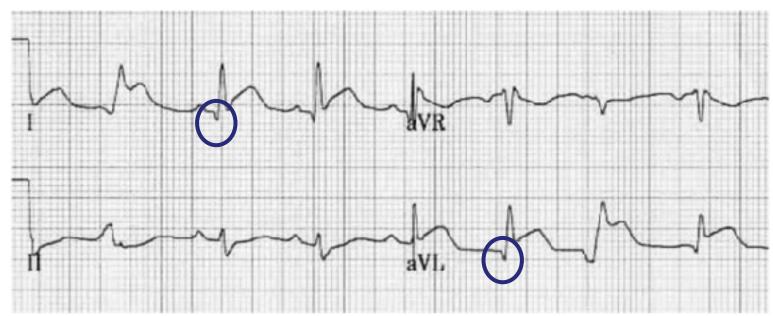
Normal Q-waves

 Ventricular septal depolarization moves L to R so left lateral leads record it as small initial negative deflection (Q wave) which can also be seen in inferior leads and can be normal

Pathologic Q-waves

- Indicate scar/infarct (develop within hours of MI)
- >1mm (>1 small box) wide
- >2mm (>2 small boxes) deep
- >25% of depth of QRS
- In contiguous leads
 - What are contiguous leads (lateral, inferior, anterior)?

Q Waves



https://litfl.com/q-wave-ecg-library/

Lateral Q waves (I, aVL) with ST elevation due to acute MI

ST Segment

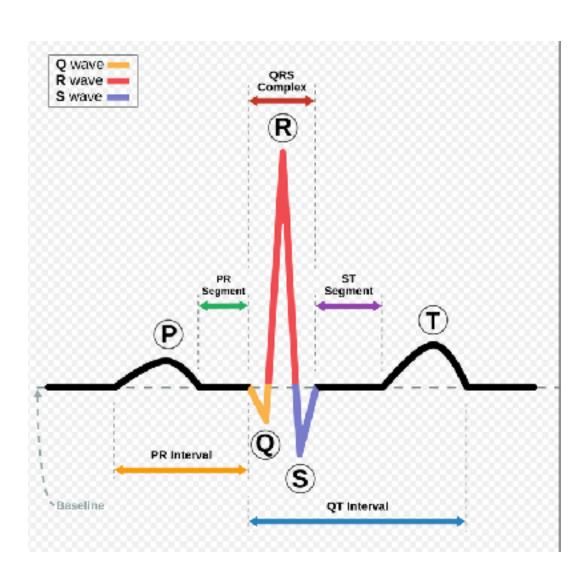
What are the reference points to determine if ST segment is elevated or depressed?

Causes of ST elevation

- Myocardial injury (infarcting, transmural)
- Pericarditis (if diffuse/all leads)

Causes of ST depression

- Myocardial Ischemia
- "Strain" pattern in LVH



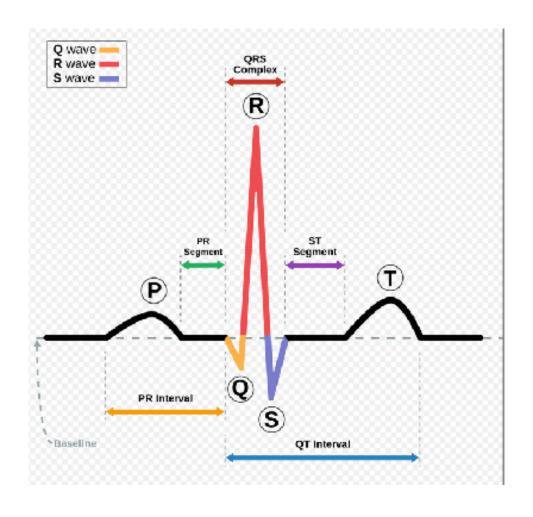
T Wave

Peaked

- Early ischemia (eg "hyper-acute T-waves")
- Hyperkalemia

Inverted

- Ischemia (also can see biphasic T-waves)
- "Strain" pattern (early repol) in LVH



QTc Interval

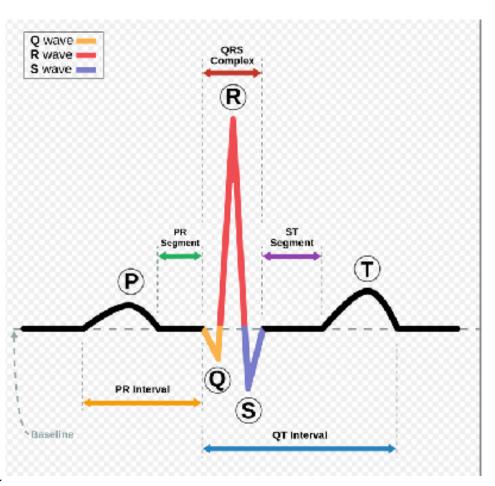
What is difference between QT-interval and QTc-interval?

Definition of prolonged QTc

 >460 borderline/prolonged (some differences between genders)

Causes of prolonged QTc

- Meds
 - Avoid if QTc already >500
- Electrolyte derangements
- Congenital conditions
- Brady-arrhythmias
 - Sinus node dysfunction
 - 2nd or 3rd degree AV block

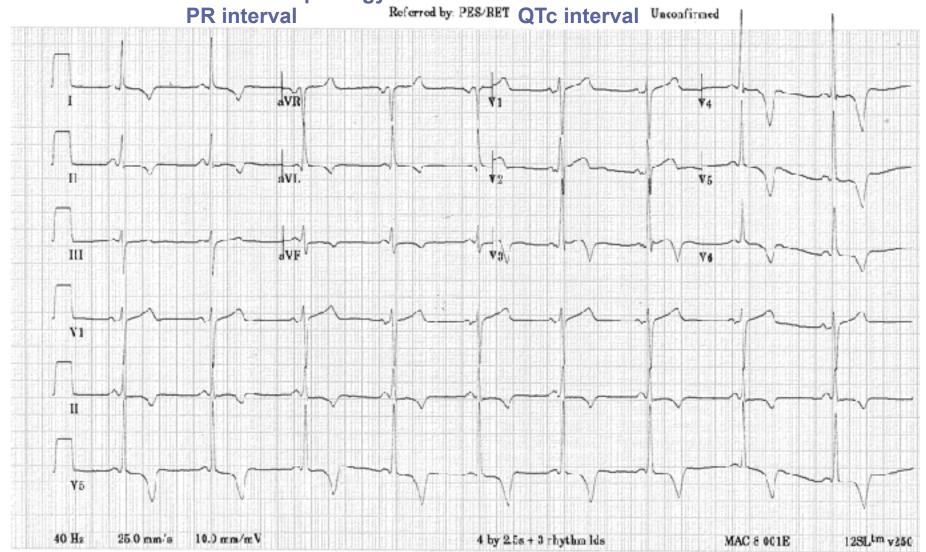


Is there a quick way to "eyeball" the QTc when looking at EKG without measuring/calculating?

EKG #3

Rate
Rhythm
Axis
P wave morphology

QRS- Wide or Narrow? BBB? LVH? Q waves present/pathologic? ST segment T waves

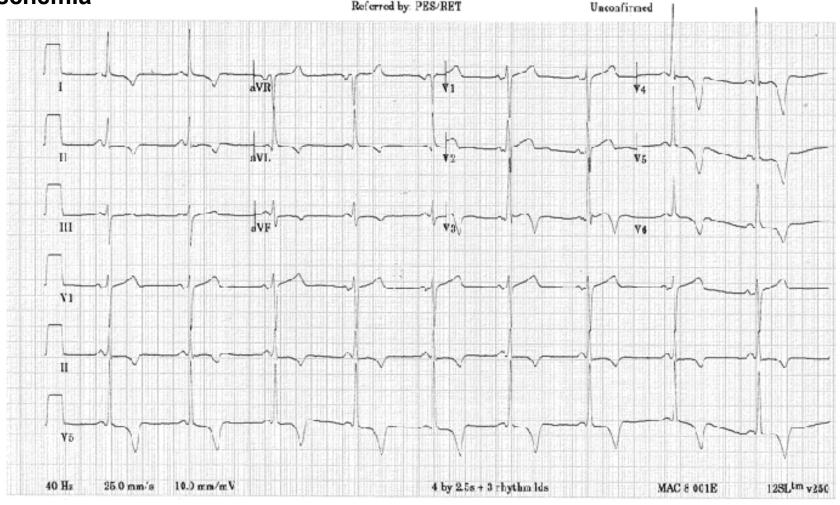


EKG #3: Rate ~50's / Reg Rhythm / P causing QRS = yes / P waves upright in lead II = yes // NSR / Axis Normal / P morphology normal / PR interval normal / Narrow QRS/no BBB / Voltage? +LVH as aVL>11 and S in V1 + R in V5 >35 / Q- ST – T wave = Diffuse changes including t-wave inversions/ STD's

SUMMARY: Normal Sinus Rhythm, LVH with diffuse ST/T-wave changes consistent with "Strain/Repolarization Abnormalities" but can't rule out ischemia

Referred by: PES/RET

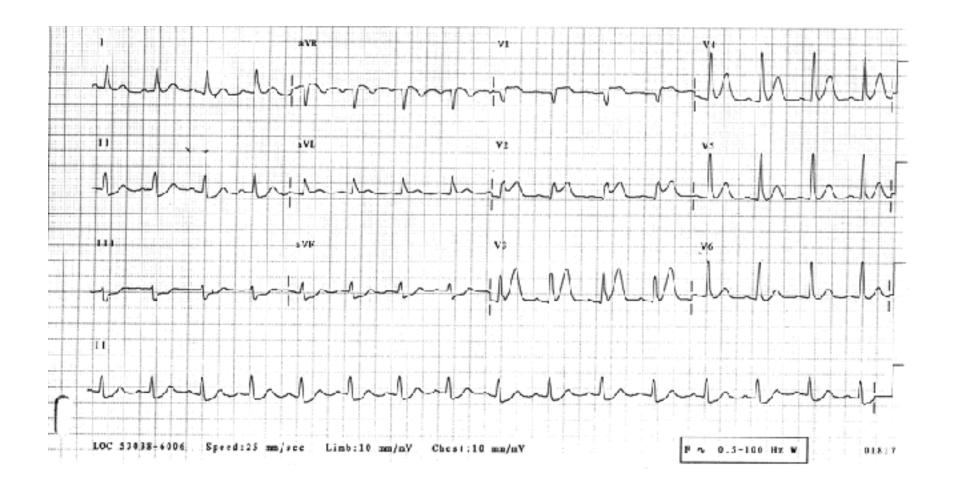
Unconfirmed



EKG #4

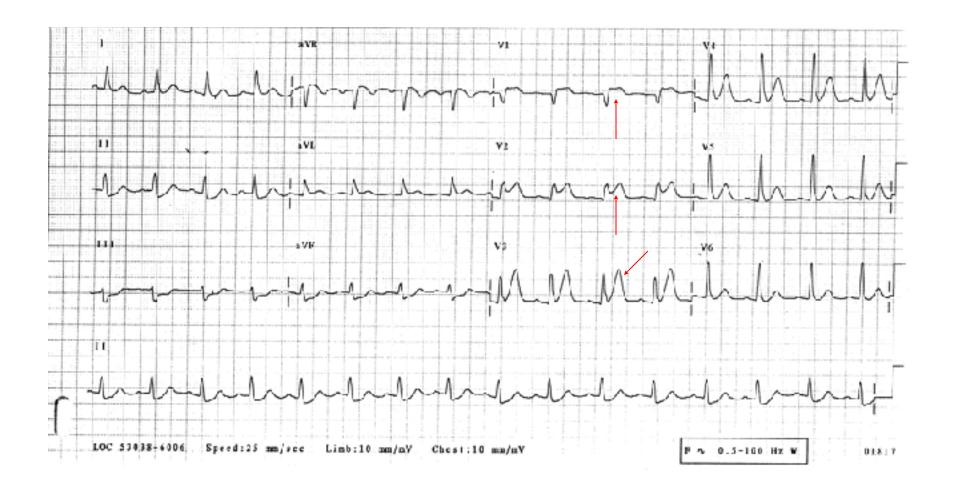
Rate
Rhythm
Axis
P wave morphology
PR interval

QRS- Wide or Narrow? BBB? LVH? Q waves present/pathologic? ST segment T waves QTc interval



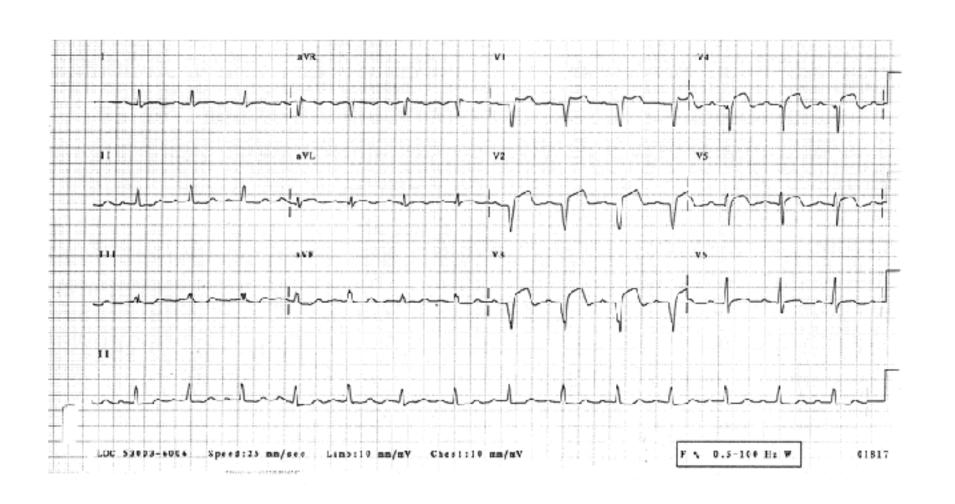
EKG #4: Rate 96 / Reg Rhythm / P causing QRS = yes / P waves upright in lead II = yes // NSR / Axis Normal / P morphology OK / PR interval normal / Narrow QRS/no BBB / Voltage? – No LVH / Q- ST – T wave = ST Depression inferior leads II, III, avF, ST ELEVATION V1-V2 with Q's and HYPERACUTE T V3-V4

<u>SUMMARY:</u> Normal Sinus Rhythm, Acute Anterior wall MI (early) with reciprocal changes in inferior leads



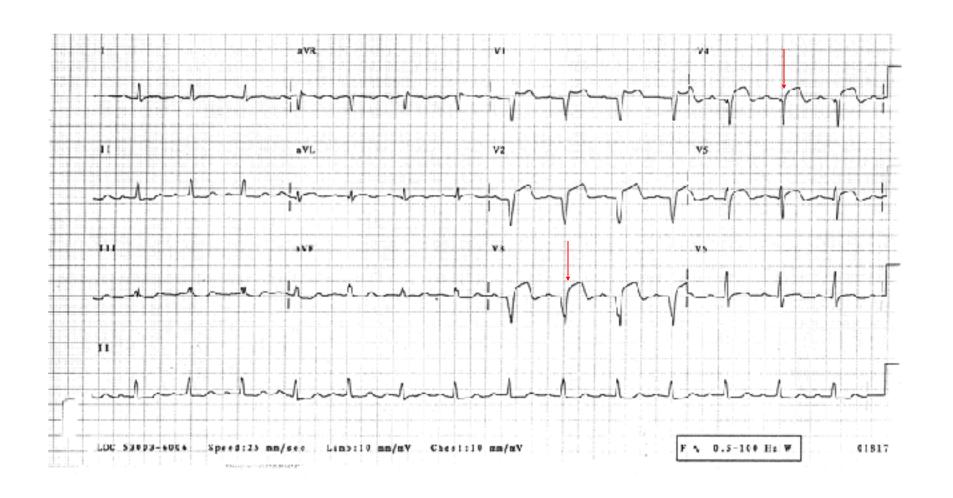
SAME PATIENT AS THE PRIOR EKG.

HOW HAS THE EKG CHANGED?



EKG #4 Second Version:

-Progression of the acute anterior MI with Q waves developing now in V3-V4 as well as ST elevation to replace the hyperacute T waves anteriorly



The characteristic ECG findings in Wolff-Parkinson-White syndrome are:

- Short PR interval (< 120ms)
- Broad QRS (> 100ms)
- A slurred upstroke to the QRS complex (the delta wave)



Delta wave: Premature excitation of the ventricles causes a slurred upstroke to the QRS

Delta wave



 Note that the remainder of the QRS remains normal — conduction still occurs through the AV node and this is the dominant pathway. On arrival to the ventricles, such conduction cancels out any pre-excitation that has occurred via an accessory pathway

EKG Assignment

- Be sure to do all the steps on the cards (show your work/write out your thought process)
 - Would add assessing for QTc prolongation to the steps at the end
- Write out your work for all the steps AND your summary statement/EKG interpretation on each individual EKG
- Useful website: life in the fast lane