ECG Breakout Session

September 16<sup>th</sup>/17th, 2022 Danielle M Sparenga, DNP, ACNP-BC Mayo Clinic Phoenix, Arizona  No disclosures
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#### Learning Objectives

Review 6 step approach to 12 lead interpretation

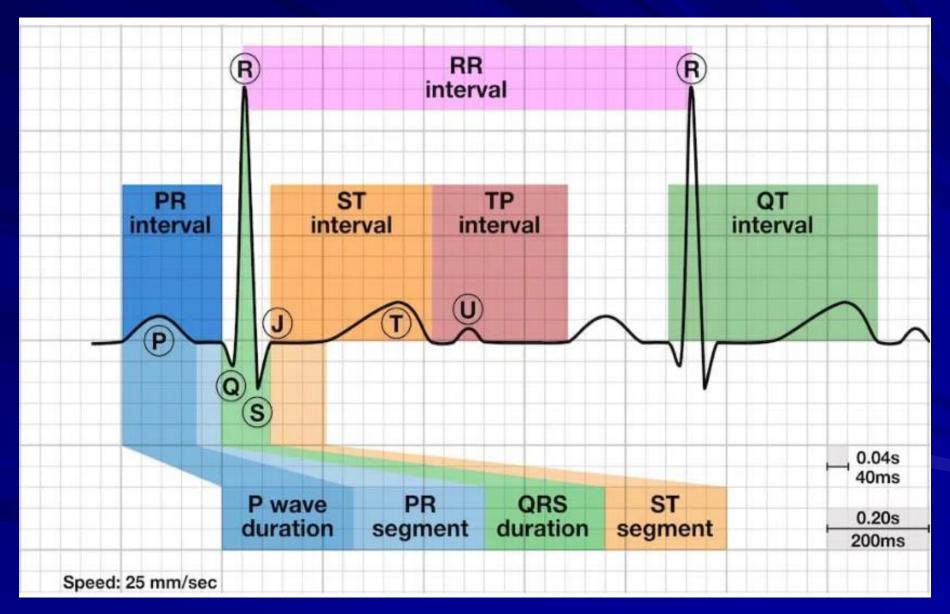
- –Name the rhythm
- -Name the abnormality
- Recognize common clinical ECG findings including ECG changes associated with myocardial ischemia & infarction
- Clinical ECG applications/findings in an acute setting

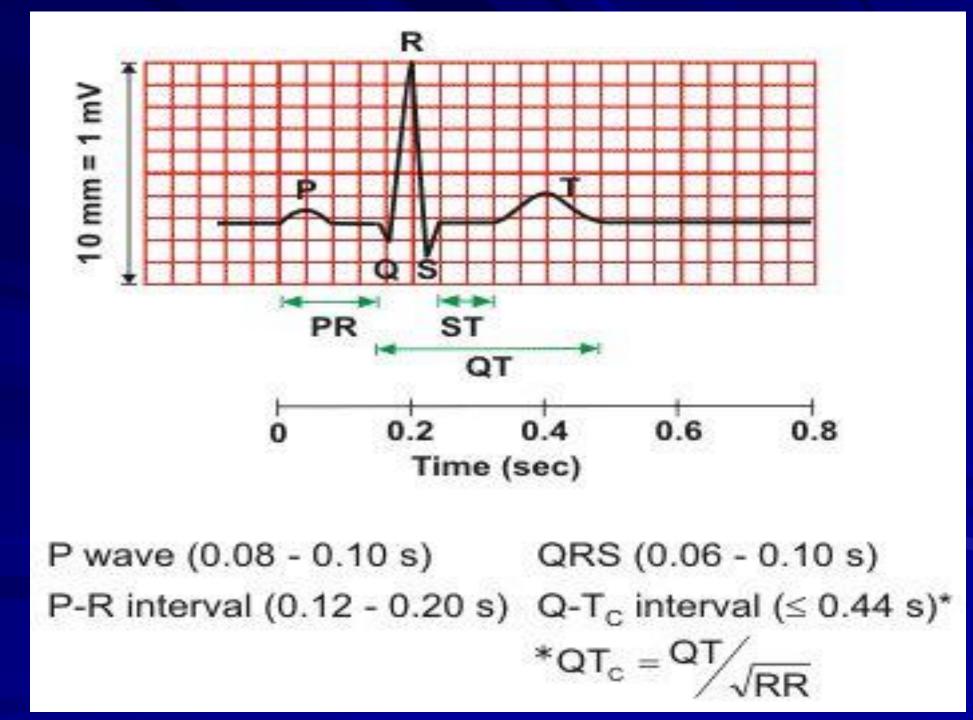


## Key To 12 Lead Interpretation 6 step method

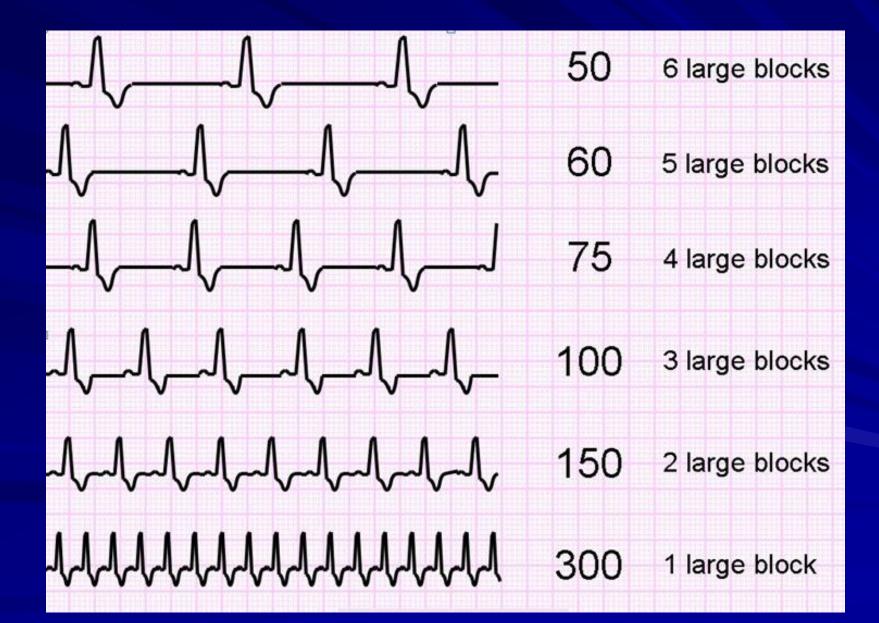
- Rate & Rhythm
- Axis determination
- Intervals (PR, QRS, QT)
- Morphology (BBB)
- Ischemia, Injury, Infarct
- Bonus step: Correlate to history and clinical presentation

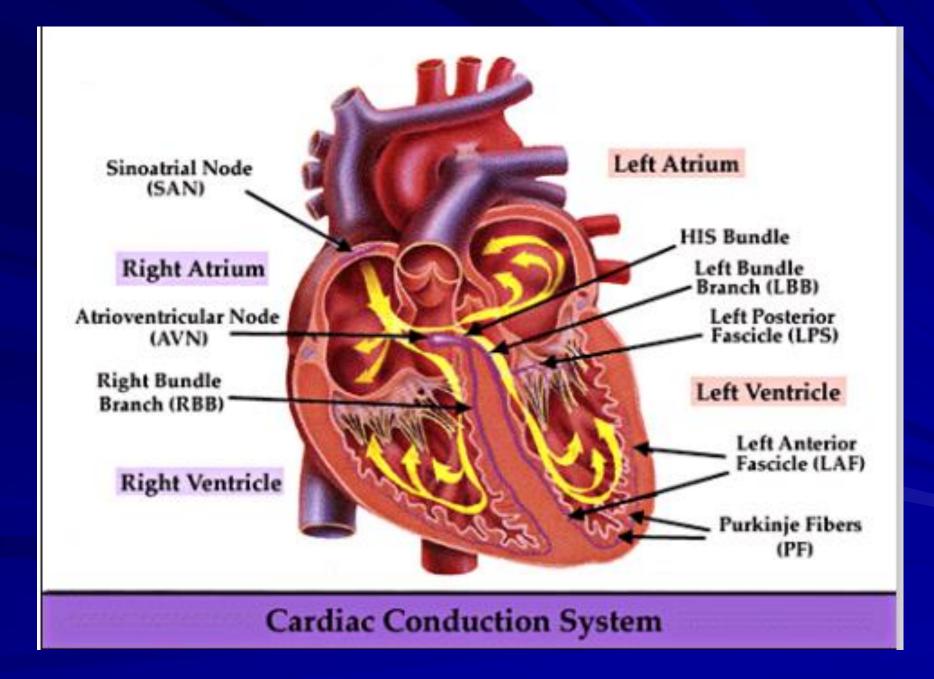
#### Intervals (PR, QRS, QT)

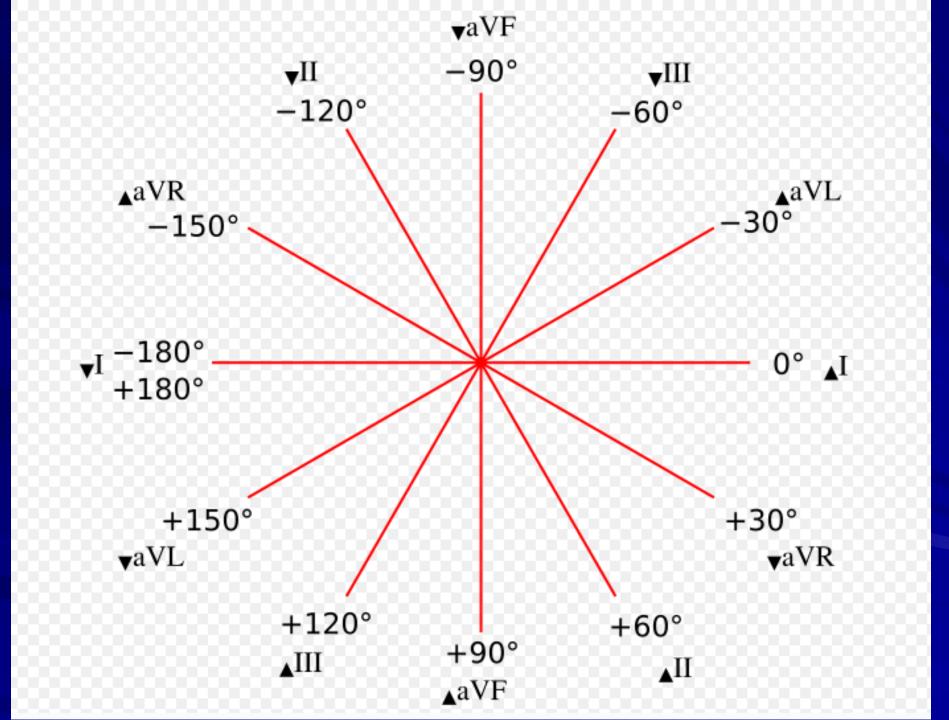




#### **Determining Heart Rate**

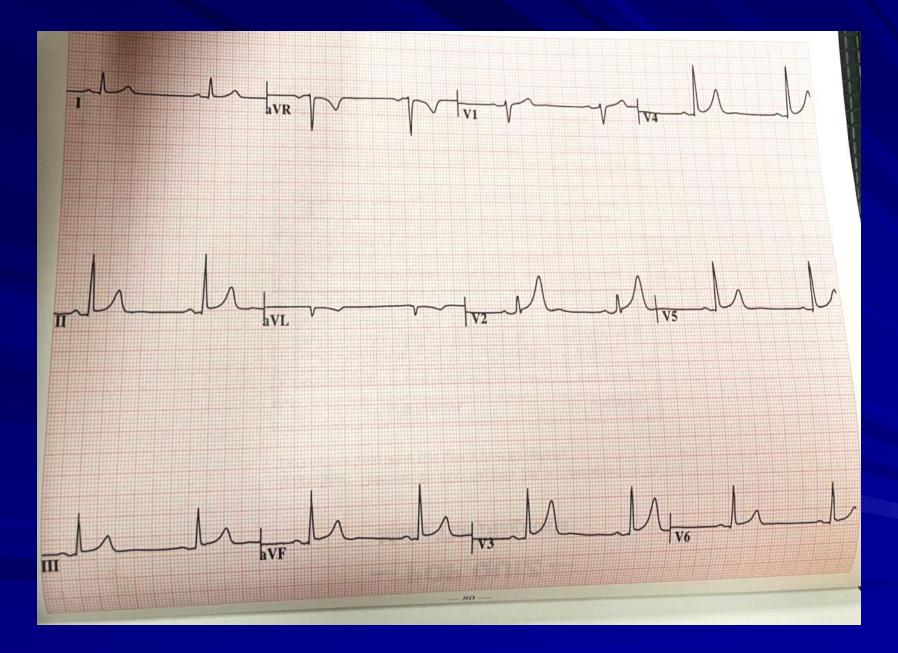








Axis		Lead I	aVF	Lead II	
Normal Axis (0° to 90°)		+	+		
Normal variant (0° to -30°)		+	-	+	
Left axis deviation 30° to -90°)	( -	+	-	-	
Right axis deviation >100°)	(	-	+		
Right superior axis to +180°)	(-90°	-	-		



#### Bradycardia

Sinus Bradycardia Junctional Rhythm Heart blocks Atrial fibrillation with SVR \*\* Where are the P waves? \*\* Is the Patient symptomatic?

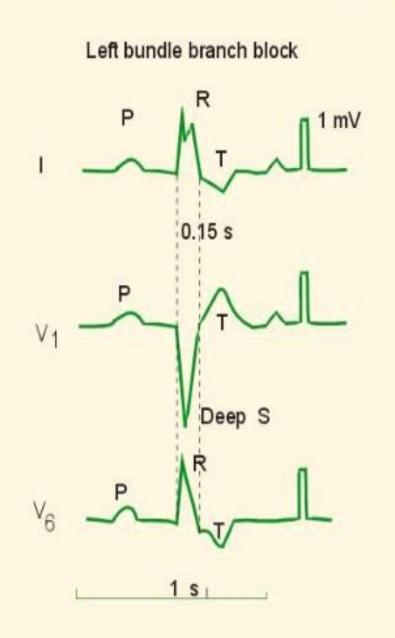
# **Junctional Rhythm**

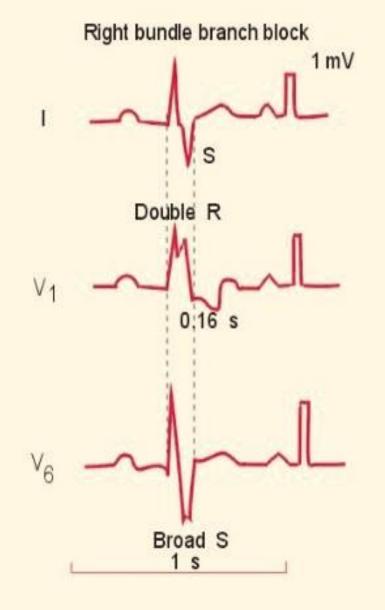




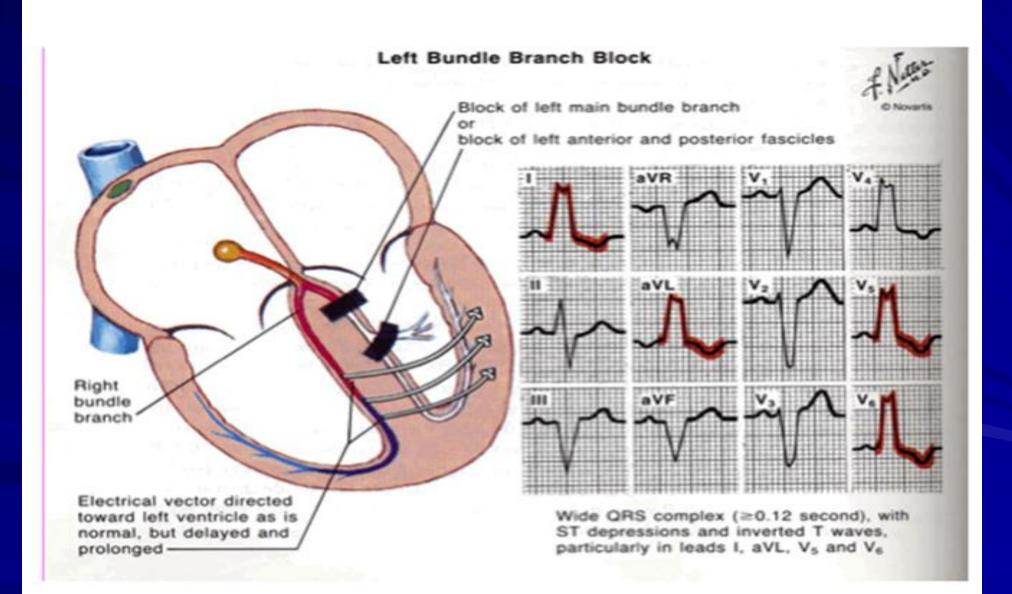
#### Narrow or Wide?

#### Bundle Branch Block

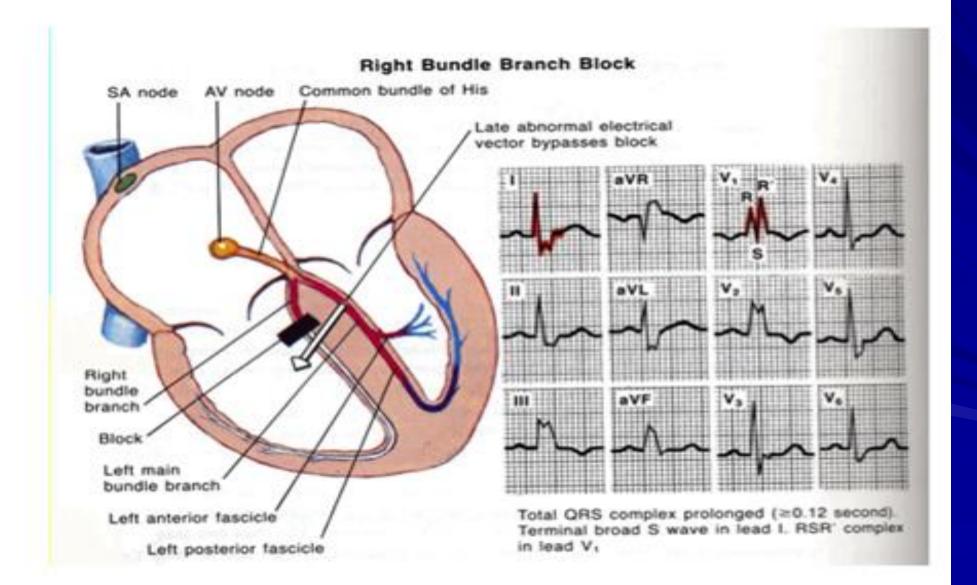




# Left bundle branch block



# Right bundle branch block



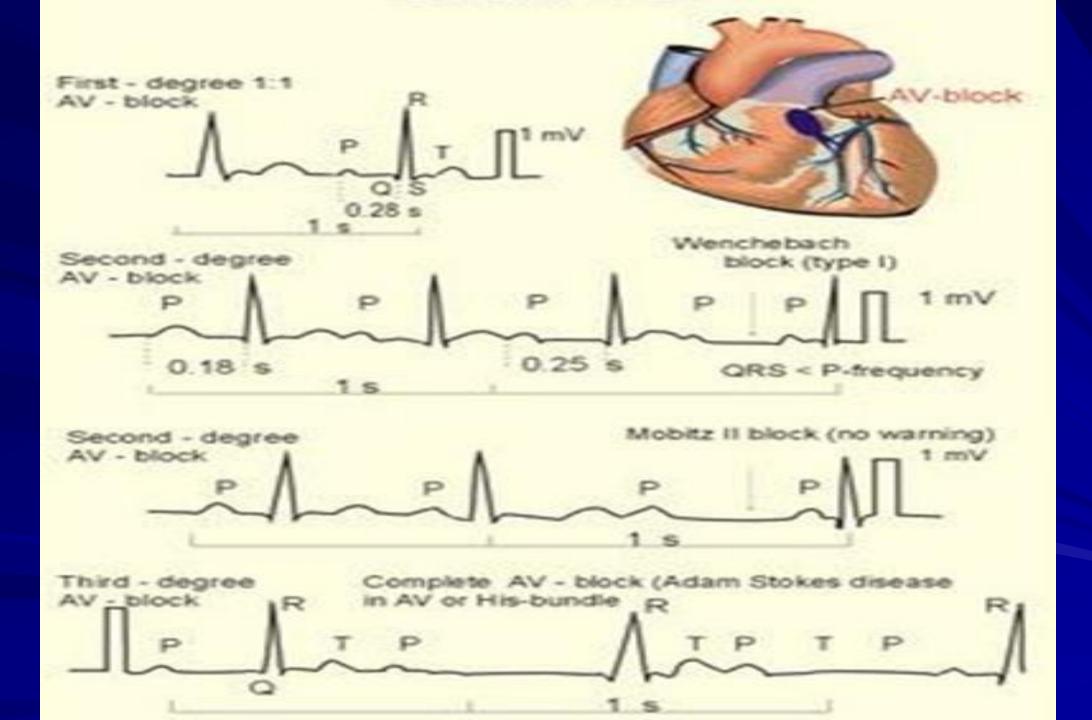
## Blocks

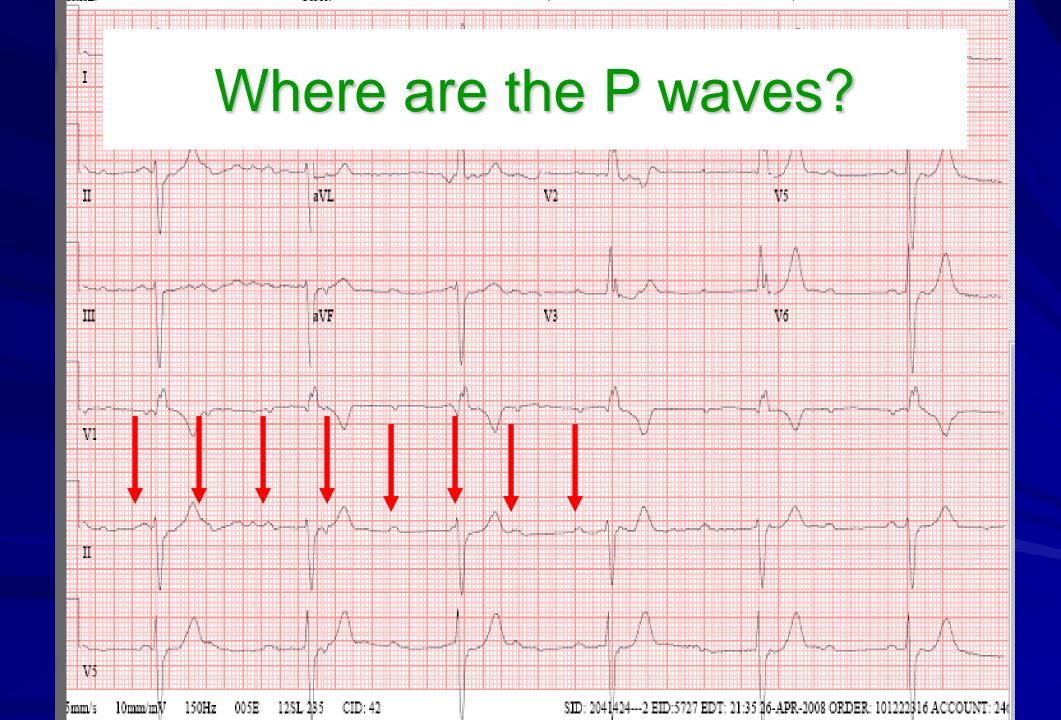
- 1<sup>st</sup> Degree
- 2<sup>nd</sup> Degree
  - Type I (Mobitz I) Wenchebach
  - Type II (Mobitz II)

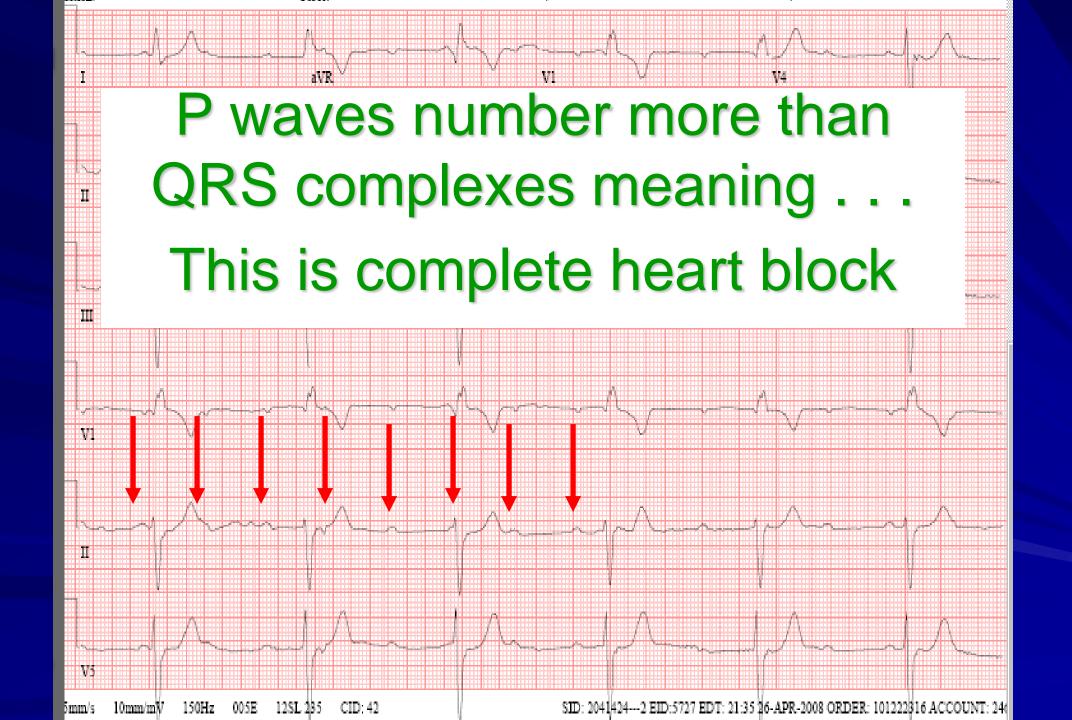
  - 2:1 AV block
- 3<sup>rd</sup> Degree (Complete Heart Block)

#### Heart Block Poem

- If the "R" is far from "P," then you have a FIRST DEGREE
- Longer, longer, longer, drop! Then you have a Wenkebach
- If some "P"s just don't get through, then you have MOBITZ II
- If "P"s and "Q"s just don't agree, then you have a THIRD DEGREE









#### 2:1

Feature	Mobitz Type I	Mobitz Type II		
QRS Duration	Narrow	Wide		
Response to maneuvers that increase heart rate & AV conduction (ie Atropine, Exercise)	Block Improves	Block worsens		
Response to maneuvers that reduce heart rate & AV conduction (ie carotid massage)	Block worsens	Block improves		
Develops during acute MI	Inferior MI	Anterior MI		
Other	Grouped beating	History of Syncope		
	O'Keefe, James H., et al., 2019			

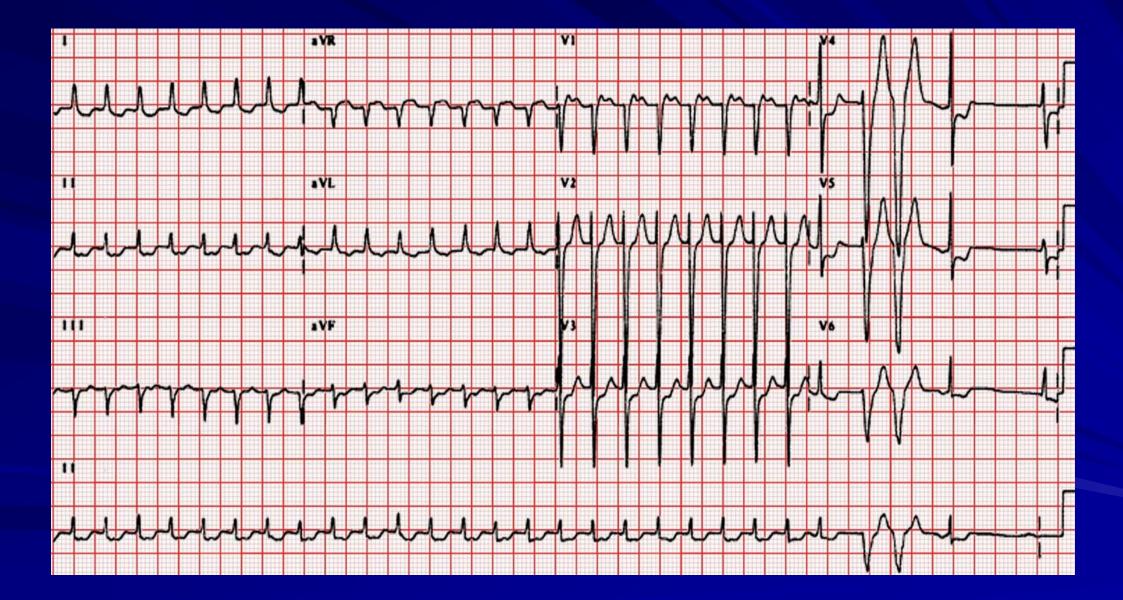
## Tachyarrhythmia

Tachyarrhythmia	Rate	QRS	Rhythm	P waves
Sinus tachycardia	Fast	Narrow	Regular	Yes
Supraventricular tachycardia or SVT (AVNRT)	Fast	Narrow	Regular	No
Atrial Fibrillation	Fast	Narrow	Irregular	No
Atrial Flutter	Fast	Narrow	Regular	Sawtooth
Ventricular tachycardia	Fast	Wide	Regular	No
Ventricular fibrillation	Fast	Wide	Irregular	No

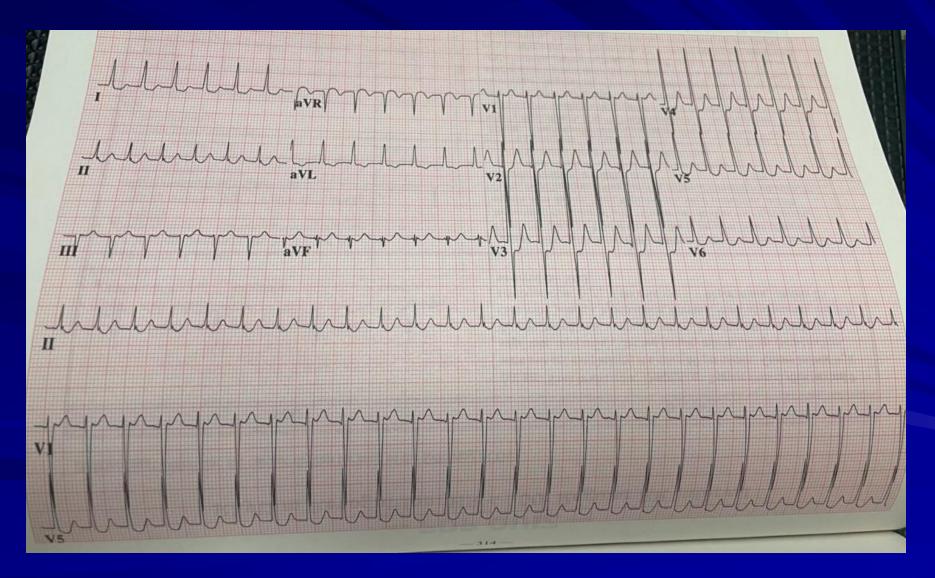
## SVT

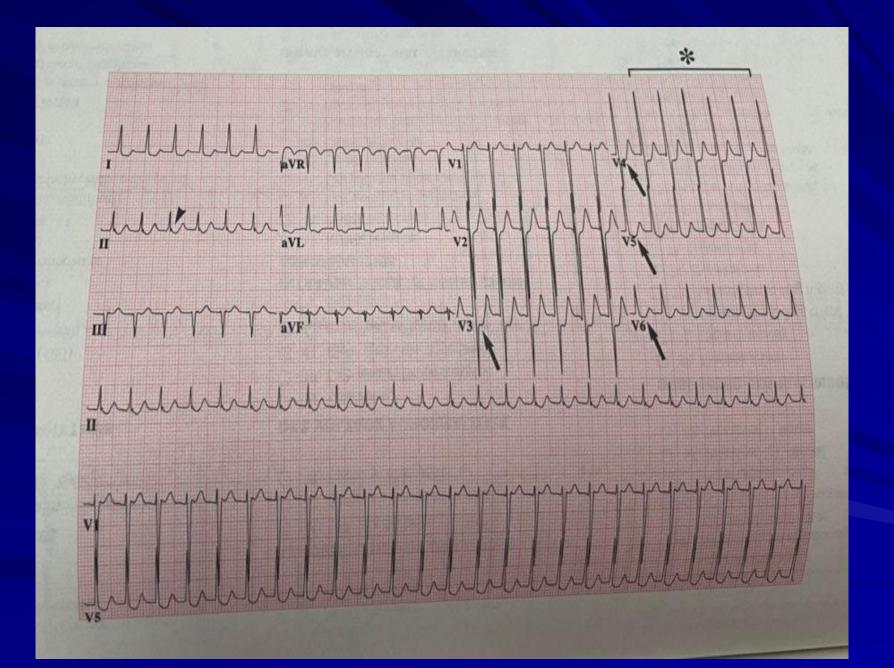
# Abnormally fast rhythm that starts in the atria

- Atrial tachycardia
- AV node re-entrant tachycardia
- Atrial Fibrillation
- Atrial Flutter
- Paroxysmal SVT

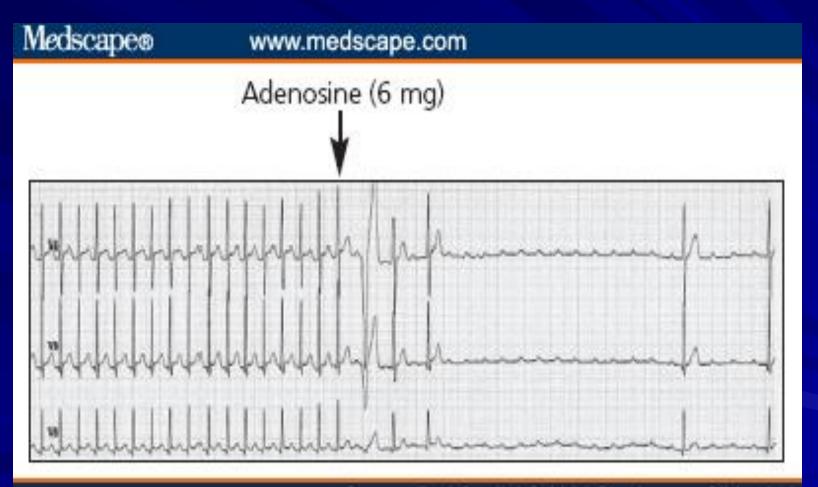


#### 71 yr old M presenting with palpitations









Source: Br J Cardiol @ 2004 Sherbourne Gibbs, Ltd.

## Regular or Irregular?

#### Afib vs. Aflutter

Afib is regularly irregular meaning: Variation in R-R intervals that are not multiples of the P-P interval

- Atrial flutter has undulations ("F waves") that are regular
- Coarse AF vs Atrial Flutter
- Typical Atrial Flutter vs Atypical Atrial Flutter

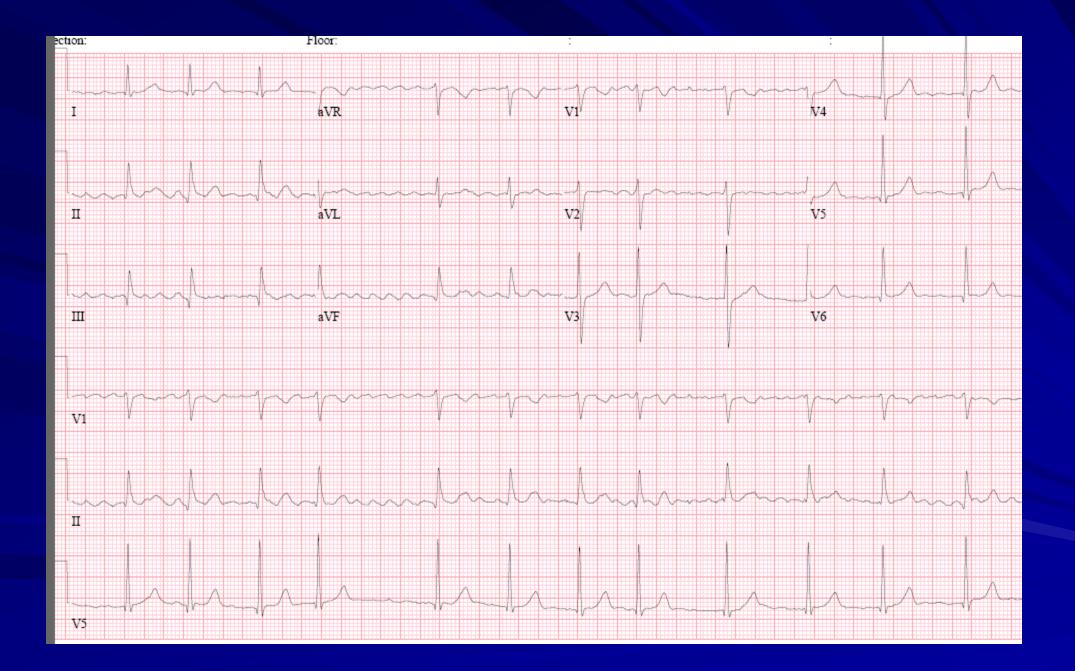


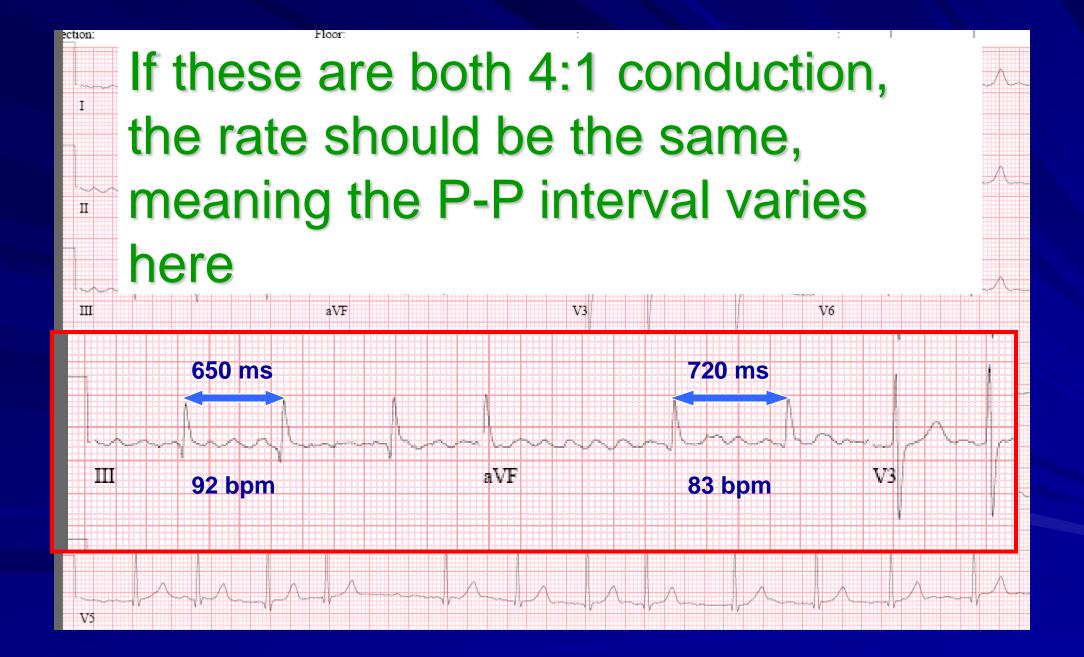
## **Clinic Case**

70 year old man Fatigue and weakness Palpitations No chest pain, dyspnea, syncope PMHx: HTN SHx: Tobacco, quit 1970. Prior 2ppd x10 years. 1 martini Q3 days

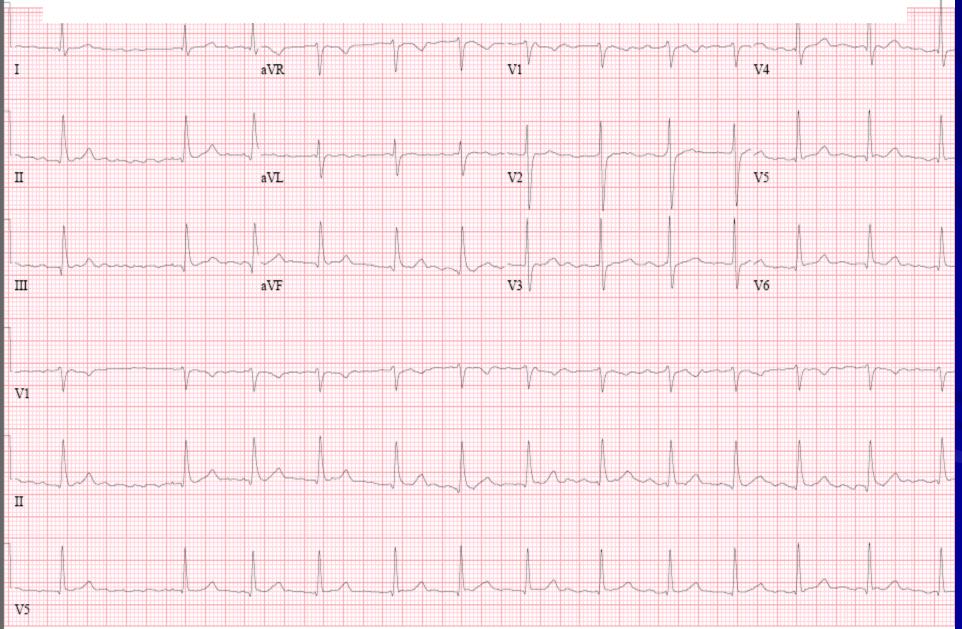
Metoprolol 100 mg BID
Warfarin per Coumadin Clinic
Levothyroxine 112 mcg daily
Zocor 40 mg daily
Coenzyme Q10
Prilosec 20 mg daily

#### FHx: none

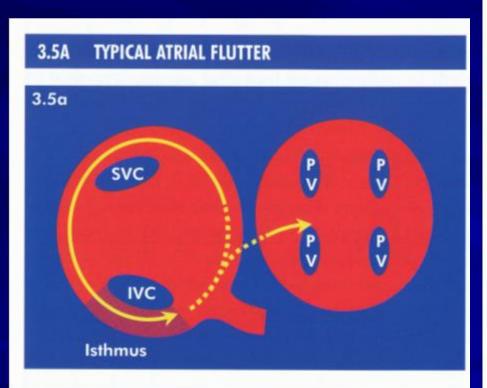


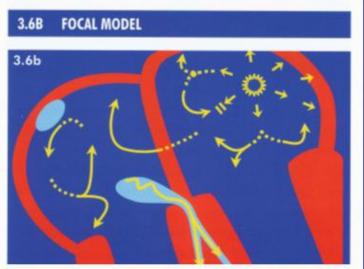


## Another ECG



## Models of Atrial Flutter and Fibrillation





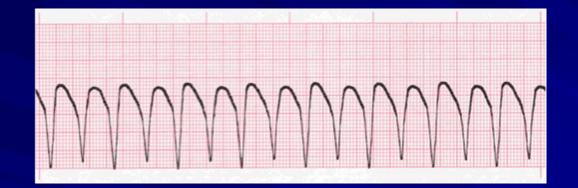
3.6A MULTIPLE WAVELET MODEL

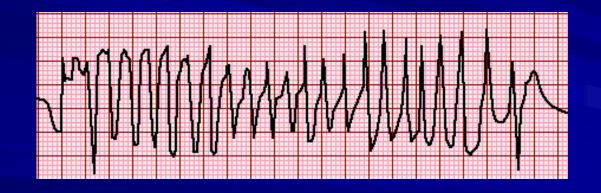


# Ventricular Tachycardia

- Monomorphic VT
- Polymorphic VT
- NSVT

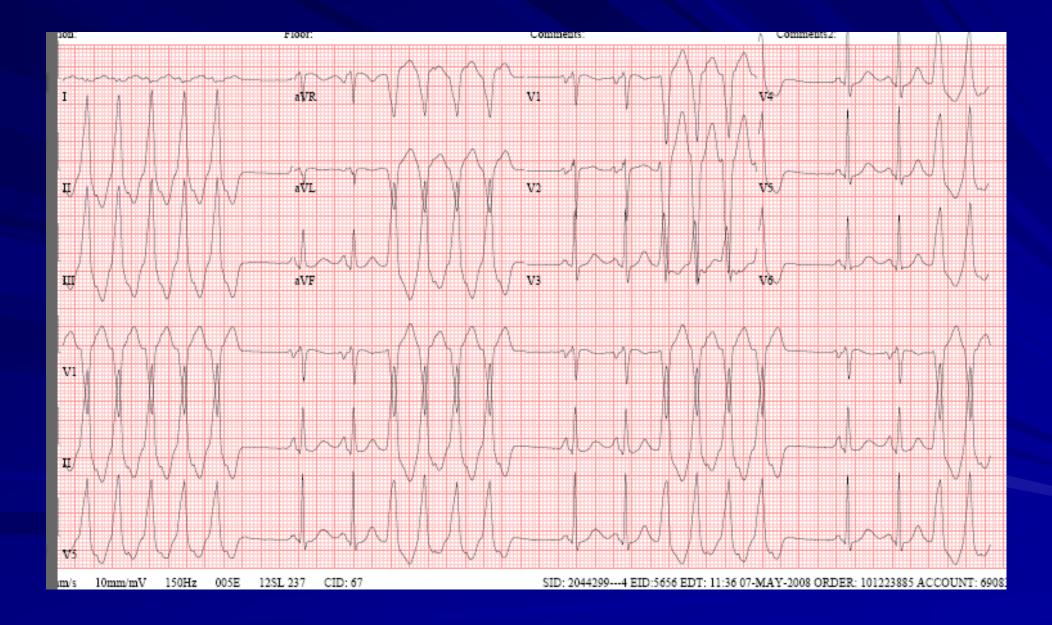
## Ventricular Tachycardia (VT)

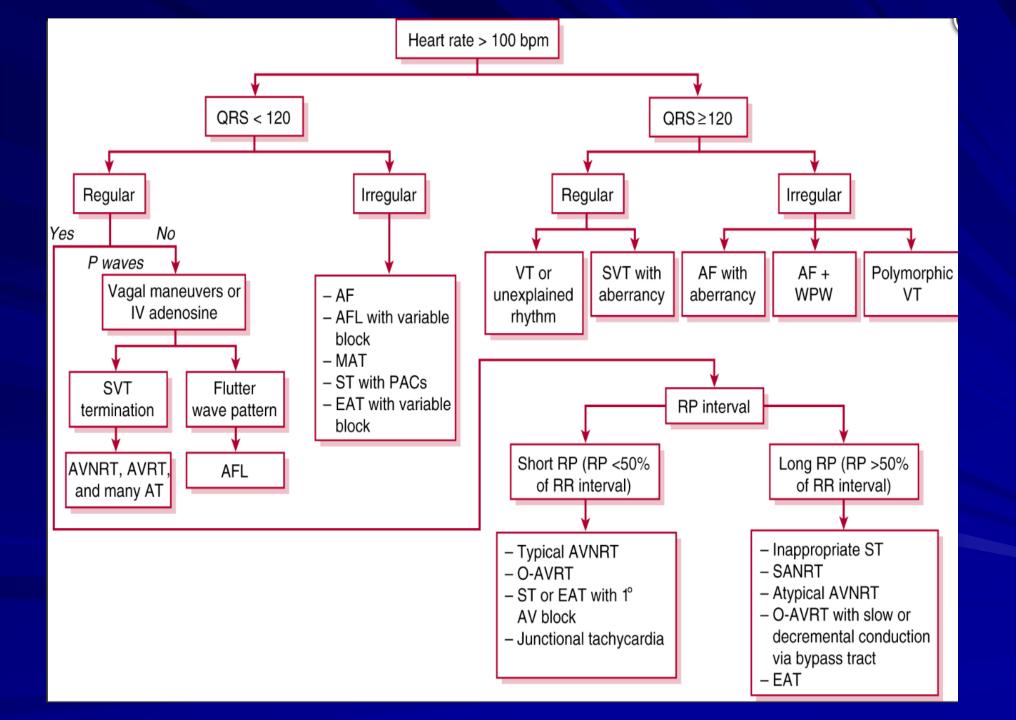




## **Criteria for VT**

Rapid succession of 3 or more ventricular premature complexes at ≥ 100 bpm
 RR interval is usually regular but may be irregular
 Abrupt onset and termination of arrhythmia is evident
 AV dissociation is common (P and QRS complexes at different rates)

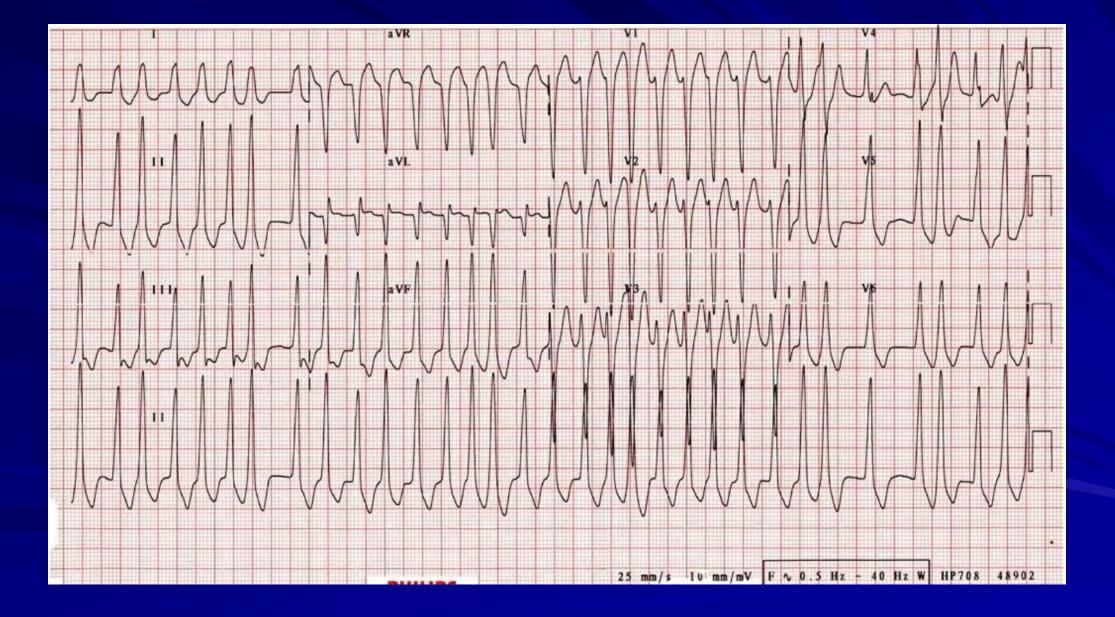




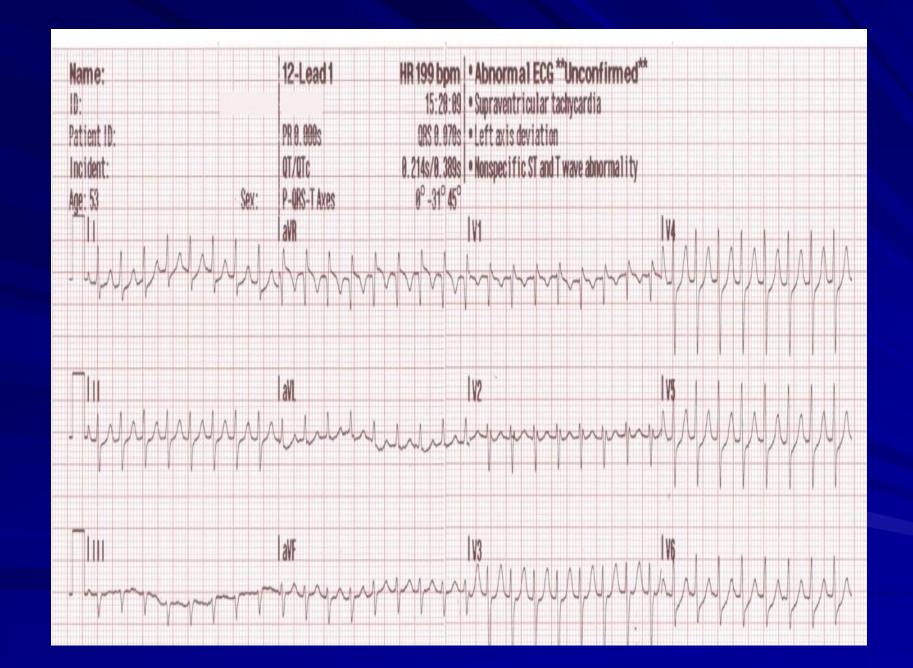




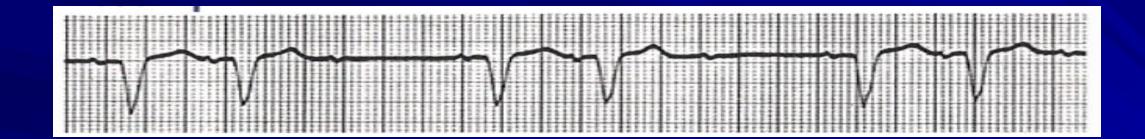
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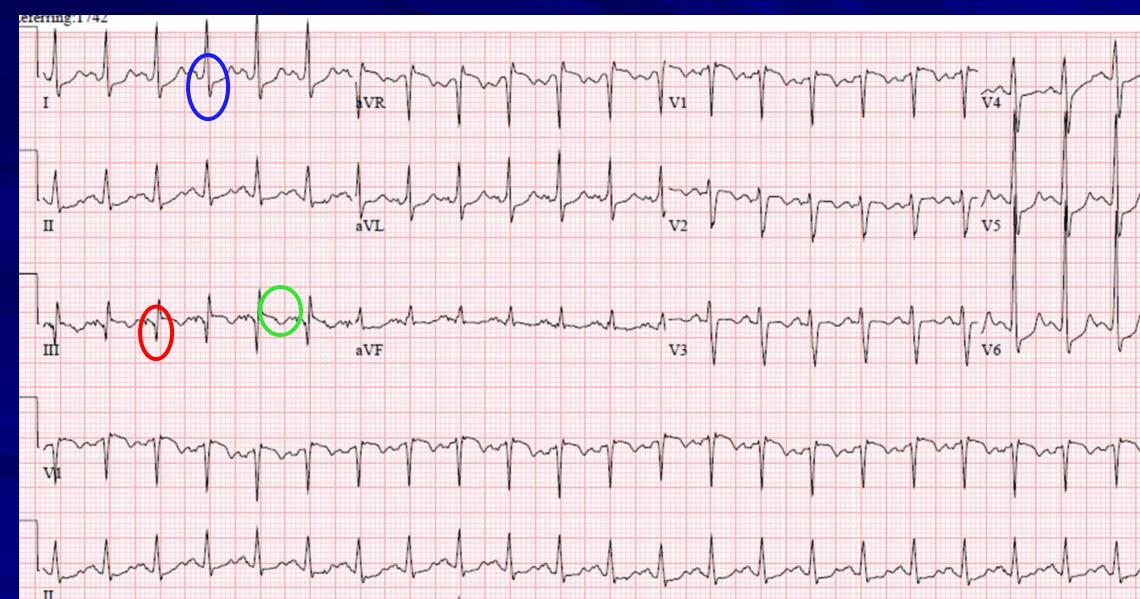
## ECG Findings in Pulmonary Embolism

#1 most common finding is... Tachycardia

## S1Q3T3

- S wave in Lead 1
- Q wave in Lead 3
- T wave inversion in Lead 3

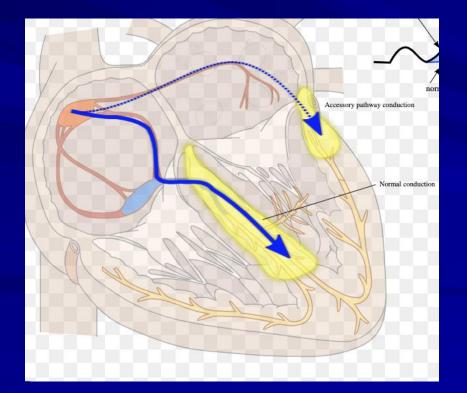
## S1Q3T3

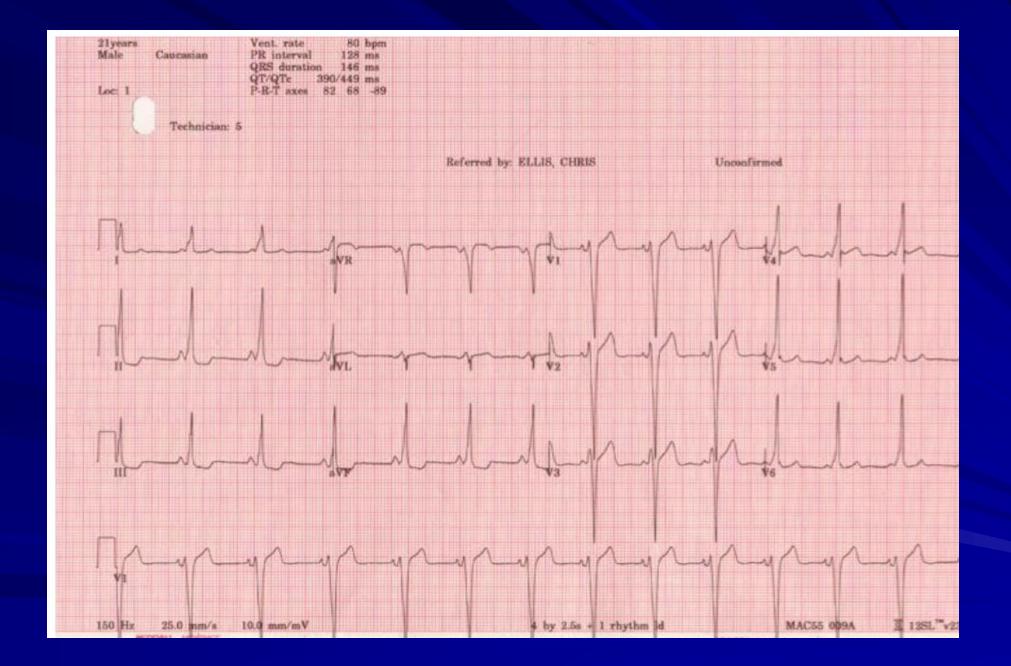


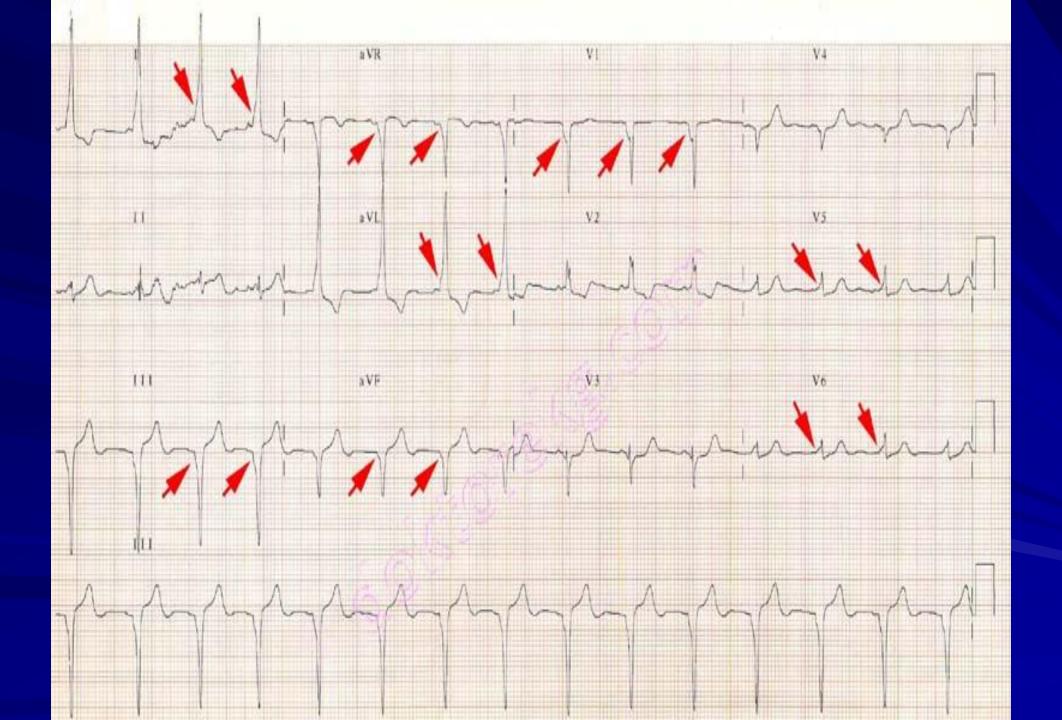
Wolff-Parkinson-White (WPW) Syndrome

#### Pre-excitation

### The Delta wave is a slurred upstroke in the QRS complex with a short PR interval.

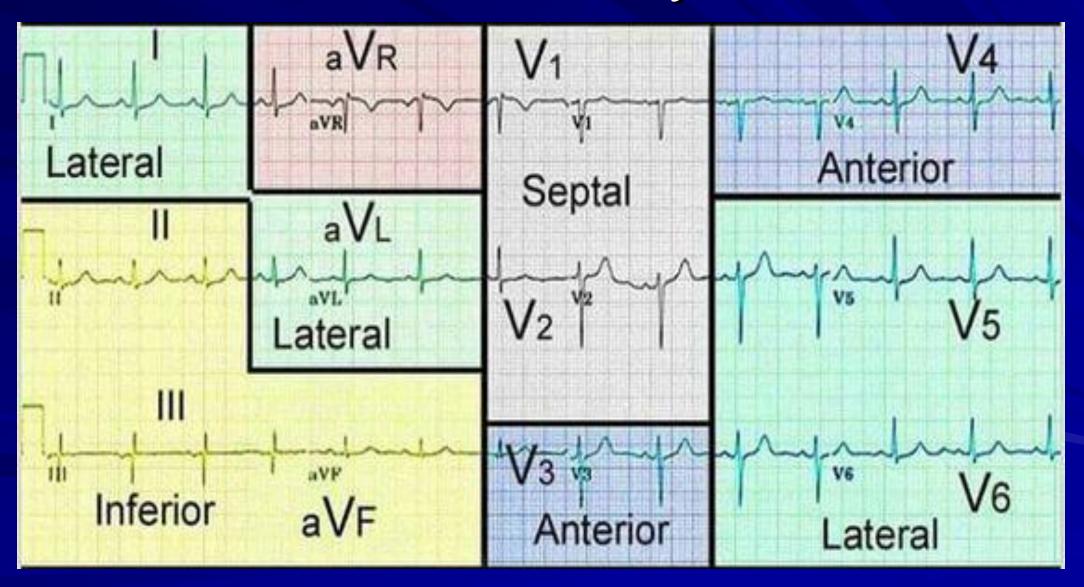




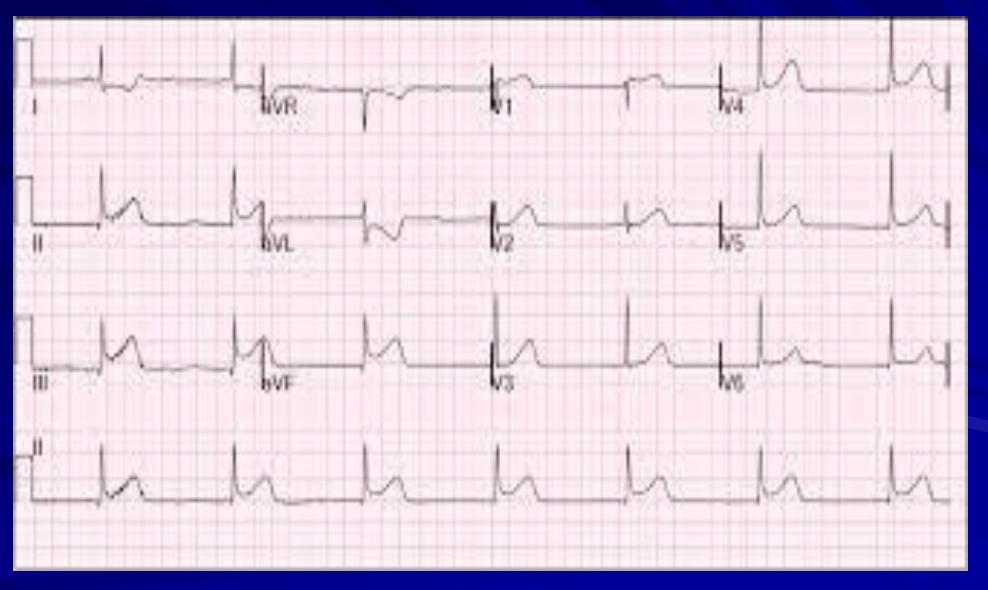


# Ischemia, Injury, Infarct

## STEMI Location by ECG



## Where is the STEMI?



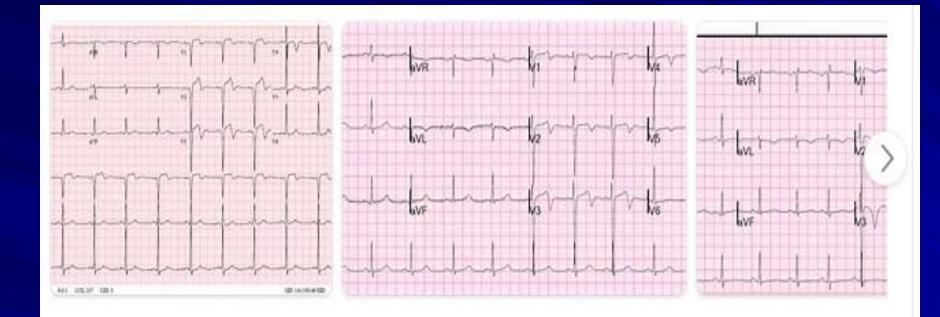
Location of Infarct (ST elevation MI)	Reciprocal changes* seen (ST depression)
Anterior (V1-V6)	Inferior leads (II, III, aVF)
Anteroseptal (V1-V2)	Inferior (II, III, aVF) or lateral leads (I, aVL, V5, V6
Lateral (I, aVL, V5-V6)	Inferior (II, III, aVF) In some cases leads V1-V2**
Inferior (II, III, aVF)	Lead I and aVL In some cases leads V1-V3**

\*Reciprocal changes are the same ST segment shifts as seen from a different angle or direction.

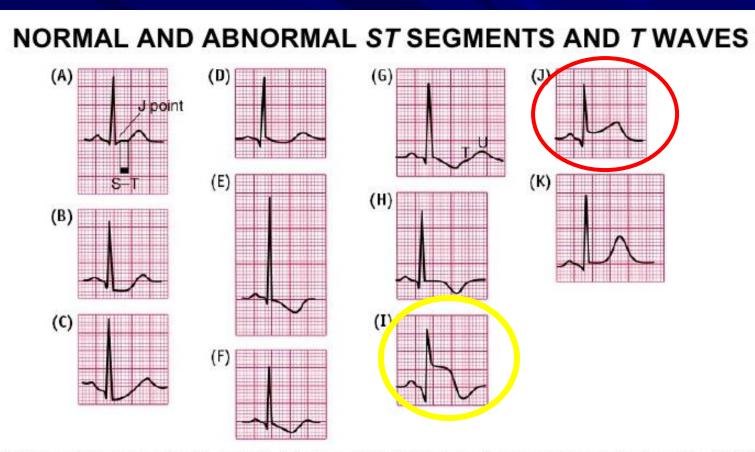
\*\*May be reciprocal, but more likely represents posterior MI.

Posterior wall MI may be missed on typical ECG!
 High degree of suspicion if ST depression in V1-V3

# Wellen's Sign



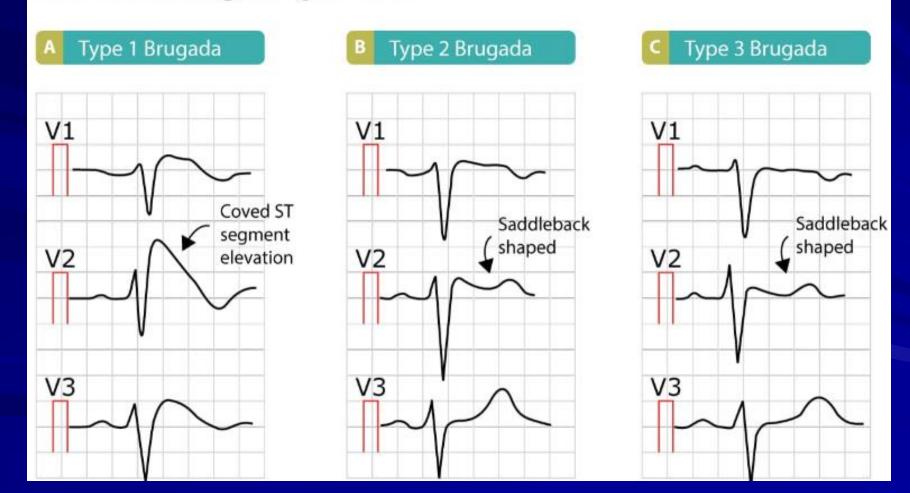
Wellens' ECG (sometimes referred to as Wellens syndrome or sign) is an ECG manifestation of **proximal left anterior descending stenosis** in patients with acute coronary syndrome. It is characterized by symmetrical, often deep (>2 mm), T wave inversions in the anterior precordial leads.

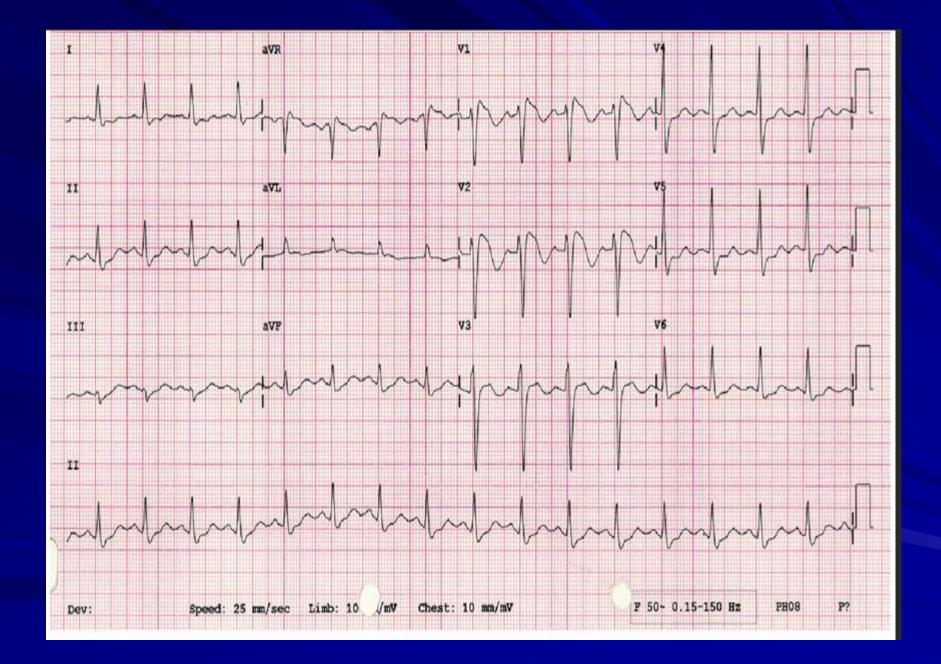


(A) Normal ST segment with J point. (B) Horizontal ST depression in myocardial ischaemia. (C) ST segment sloping upwards in sinus tachycardia. (D) ST sagging in digitalis therapy. (E)
Asymmetrical T wave inversion associated with ventricular hypertrophy. (F) Similar pattern sometimes seen without voltage changes in hypertrophy – 'strain'. (G) ST sagging and prominent U waves of hypokalaemia. (H) Symmetrically inverted T wave of myocardial ischaemia or infarction.
(I) ST elevation in acute myocardial infarction. (J) ST elevation in acute pericarditis. (K) Peaked T wave in hyperkalaemia.

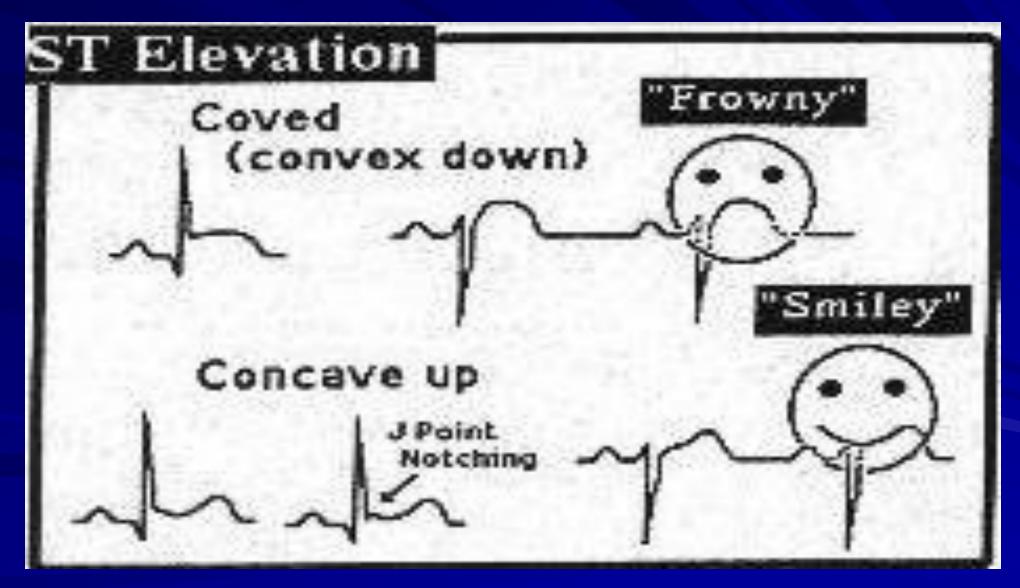
# Brugada 1,2,3

#### The ECG in Brugada syndrome

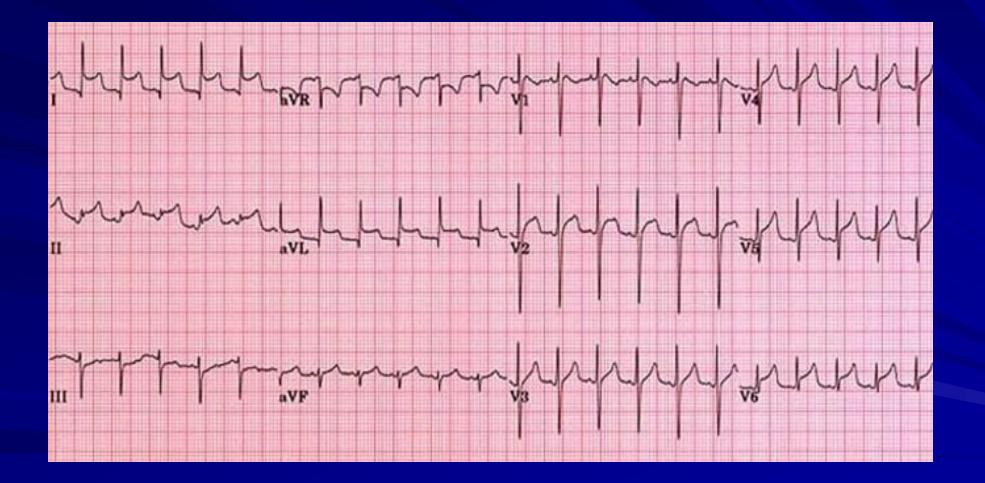




## Pericarditis



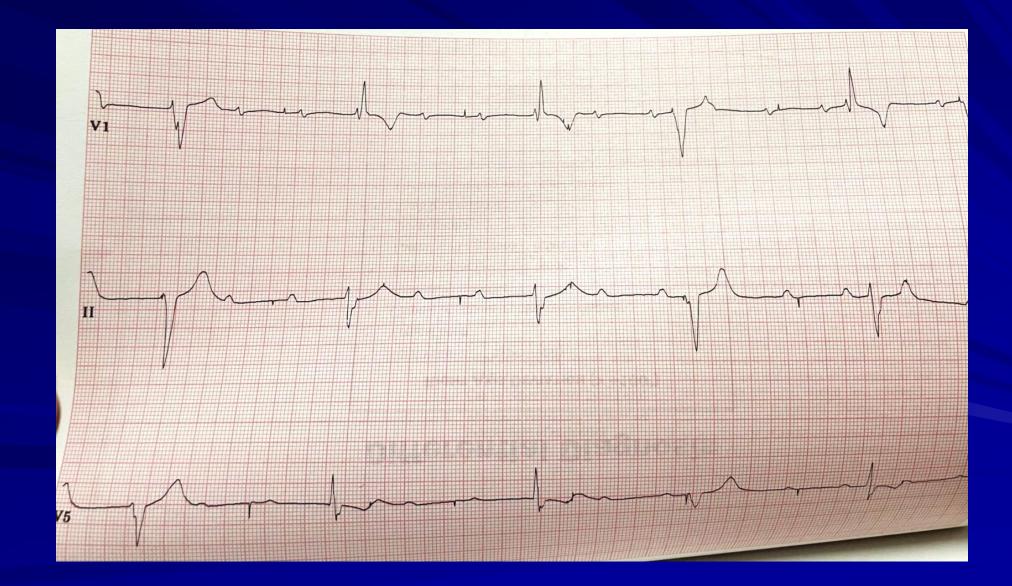
## **STEMI or Pericarditis?**

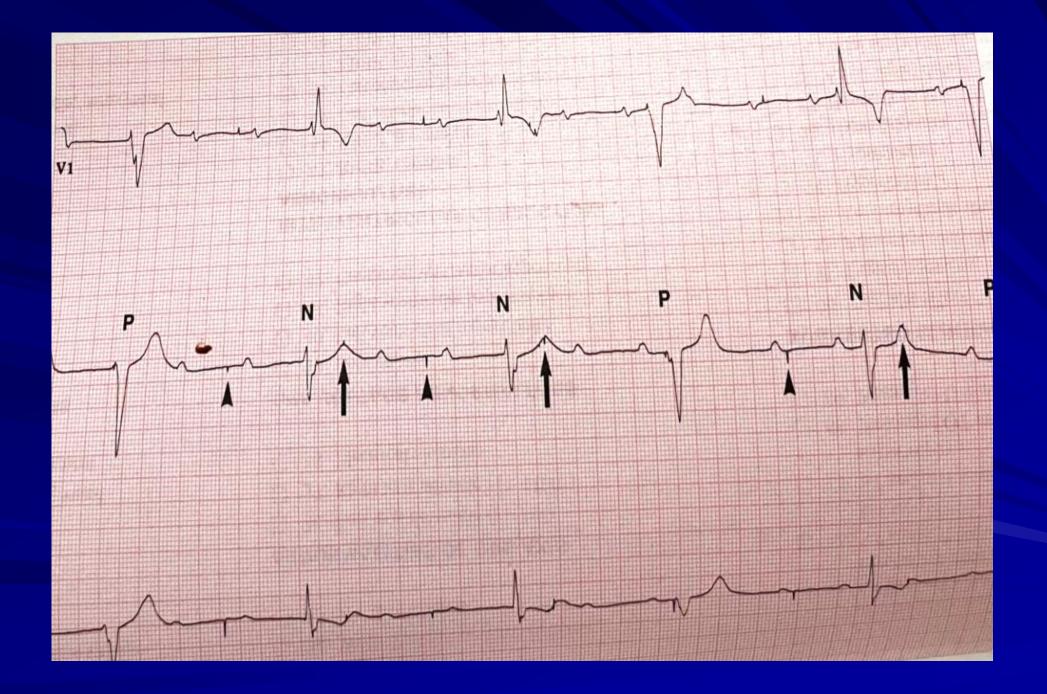


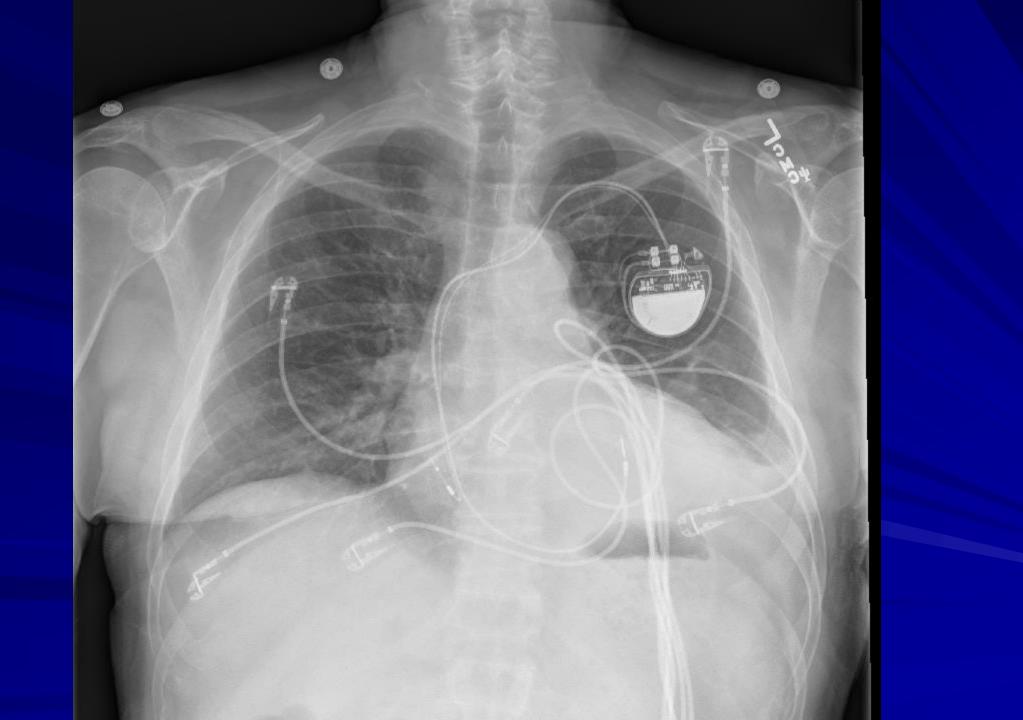
Correlate to history and clinical presentation

Pacer spikes?
Pt presenting symptoms & labs
Aberrancy vs VT- Remember the basics

## 54 yr old F presenting with lightheadedness







## Take home pearls

Its like a free-throw; the same every time Rate of 150, sometimes we clinically may not be sure, yet. How do we help ourselves better understand the rhythm? SLOW IT DOWN. Where are the P waves? Compare arrhythmia ECG with NSR ECG (if able)

When you feel like giving up, look at this picture and remember just how strong the human spirit really is.

## References

- O'Keefe, James H., et al. The Complete Guide to ECGS: A Comprehensive Study Guide to Improve ECG Interpretation Skills. Jones & Bartlett Learning, 2019.
- Chan TC, Vilke GM, Pollack M, Brady WJ. Electrocardiographic manifestations: pulmonary embolism. Journal of Emergency Medicine. 2001 Oct;21(3):263–70.
- Ullman E, Brady WJ, Perron AD, Chan T, Mattu A. Electrocardiographic manifestations of pulmonary embolism. American Journal of Emergency Medicine. 2001 Oct;19(6):514–9.
- Life in the Fastlane. Retrieved on July, 12, 2017 from https://lifeinthefastlane.com/ecg-library/basics/delta-waves/



# 5 ECG Practice Questions

Austin, Texas Sept 2022

What is the most common ECG finding for Pulmonary Embolus (PE)?

A. S1Q3T3 Sign (S wave in lead I, Q wave & inverted T wave lead B. Tachycardia C. Lateral T wave inversion D. Bradycardia

## **Answer: B**

Answer: B. Tachycardia is the most common ECG finding in PE. The S1Q3T3 is a sign of acute cor pulmonale (acute pressure and volume overload of the right ventricle because of pulmonary hypertension) and reflects right ventricular strain. However, this finding is present in 15% to 25% of patients ultimately diagnosed with PE. T wave inversion is occasionally present, but is typically anterior V1-V4.

References

- Chan TC, Vilke GM, Pollack M, Brady WJ. Electrocardiographic manifestations: pulmonary embolism. Journal of Emergency Medicine. 2001 Oct;21(3):263–70.
- Ullman E, Brady WJ, Perron AD, Chan T, Mattu A. Electrocardiographic manifestations of pulmonary embolism. American Journal of Emergency Medicine. 2001 Oct;19(6):514–9.

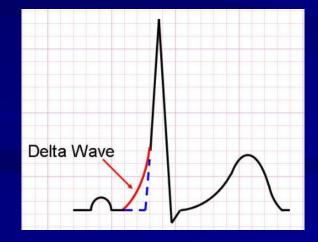
What prominent ECG finding below is characteristic of Wolff-Parkinson-White (WPW) Syndrome:



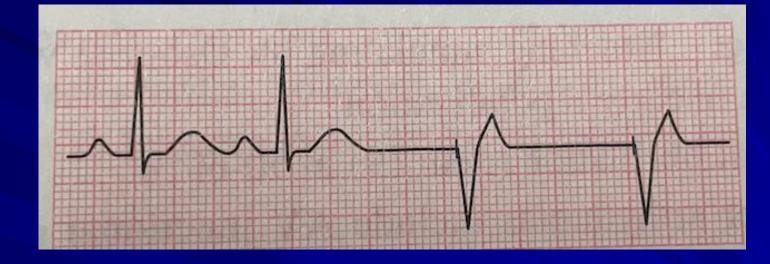
A. Prolonged QT IntervalB. Delta WaveC. Long PR intervalD. Peaked T waves

## Answer: B

Answer: B. The Delta wave is a slurred upstroke in the QRS complex often associated with a short PR interval. It is most commonly associated with pre-excitation syndrome such as WPW. The other common findings are Short PR Interval and a broad QRS.



## What sense can you make of this ECG



- Ventricular demand pacing, oversensing
- Ventricular demand pacing, normal function
- Ventricular demand pacing, under sensing
- Ventricular demand pacing, lead fractured

## Answer: B

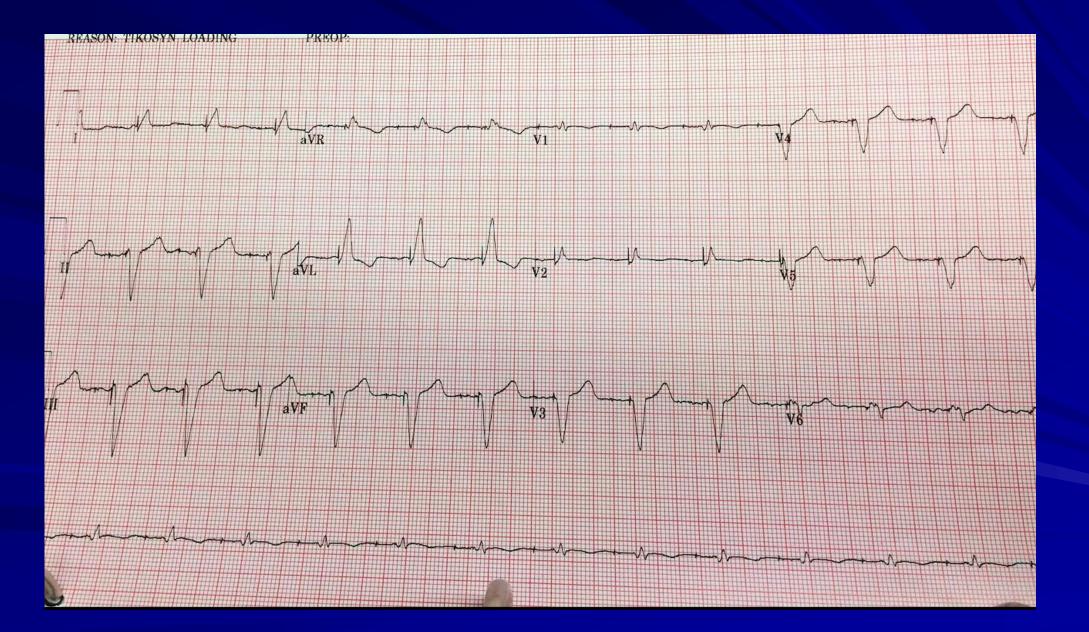
- Answer: B Ventricular demand pacing, normal function. Pacemaker stimulus followed by a QRS complex of different morphology than intrinsic QRS. A VVI pacemaker senses and paces only in the ventricle and is oblivious to native atrial activity. Appropriately sensed ventricular activity resets pacemaker timing clock. After an interval of time with no sensed ventricular activity, a ventricular paced beat is delivered and a new cycle begins. A spontaneous QRS arising before the end of the V-V interval is sensed and the ventricular output of the pacemaker is inhibited. A new timing cycle begins.
- O'Keefe, James H., et al. The Complete Guide to ECGS: A Comprehensive Study Guide to Improve ECG Interpretation Skills. Jones & Bartlett Learning, 2019.

Pt is a 65 year old male with known coronary artery disease s/p PCI to LAD, ischemic cardiomyopathy s/p dual chamber ICD placement and paroxysmal atrial fibrillation. APP noticed an increase In atrial fibrillation burden from 1% to 30% with last ICD interrogation. Pt presents as a direct admit for Tikosyn loading. Why does the patient need to be monitored in the hospital for the first 5 doses of Tikosyn?

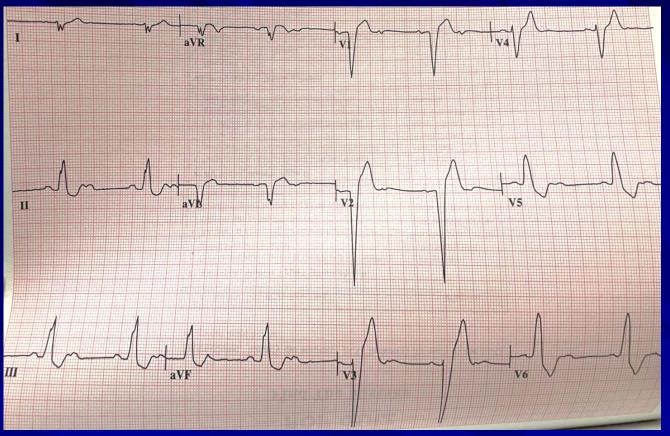
- QRS monitoring; risk of polymorphic ventricular tachycardia
- QRS monitoring; risk of monomorphic ventricular tachycardia
- QTc monitoring; risk of polymorphic ventricular tachycardia
- QTc monitoring; risk of monomorphic ventricular tachycardia

### Answer: C

### Answer: C QTc monitoring; risk of polymorphic ventricular tachycardia



51 year-old F presenting with orthopnea and paroxysmal nocturnal dyspnea. What rhythm do you see here that may be the culprit of her symptoms?



- Mobitz Type I (Wenckeback)
- Mobitz Type II
- 2:1 AV block
- Sinus bradycardia with pre-atrial complex (PACs)

## Answer: A

Answer: A. ECG shows sinus bradycardia with predominantly 2:1 AV block (arrowheads mark P waves), LBBB with secondary ST-T changes and right axis deviation. Close inspection reveals Mobitz Type I – the third P wave conducts at a normal PR interval, the fourth P wave at a prolonged PR interval, and the fifth P wave (hidden in the T wave) is blocked. The Q waves and ST elevation in leads V1-V3 are most likely due to LBBB, rather than acute anteroseptal MI (LBBB often results in a pseudoinfarct patter).

## Questions

