EKG 101

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Objectives

- Approach to reading an EKG
- Myocardial Ischemia
- Blocks
- Tachyarrhythmia and Bradyarrhythmia
- Other Miscellaneous EKGs

Before you interpret an EKG

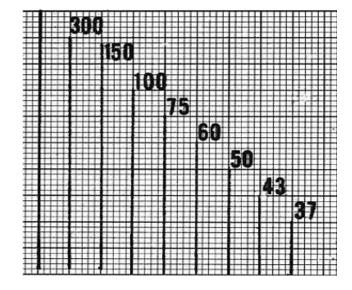
- One of the most important parts of EKG interpretation is comparing the current EKG with any previous EKGs available.
- Minor changes in between EKGs can have huge implications.
- Reading an EKG can be intimidating but the key is forming a system that works for you.
- Take a DEEP Breath!

Approach to Reading an EKG

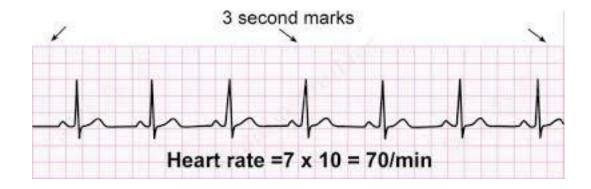
- Step 1: Rate
- Step 2: Rhythm
- Step 3: Axis
- Step 4: Intervals
- Step 5: P wave
- Step 6: QRS Complex
- Step 7: ST segment-T wave
- Step 8: Overall interpretation

Rate

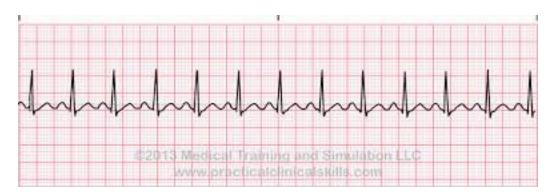
- Quick Estimate
 - "300, 150, 100, 75, 60, 50"

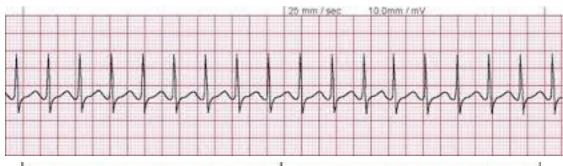


- Alternative Methods
 - Count the 6 second strip and multiply by 10
 - Count the number of beats on the EKG and multiply by 6



What is the Rate?

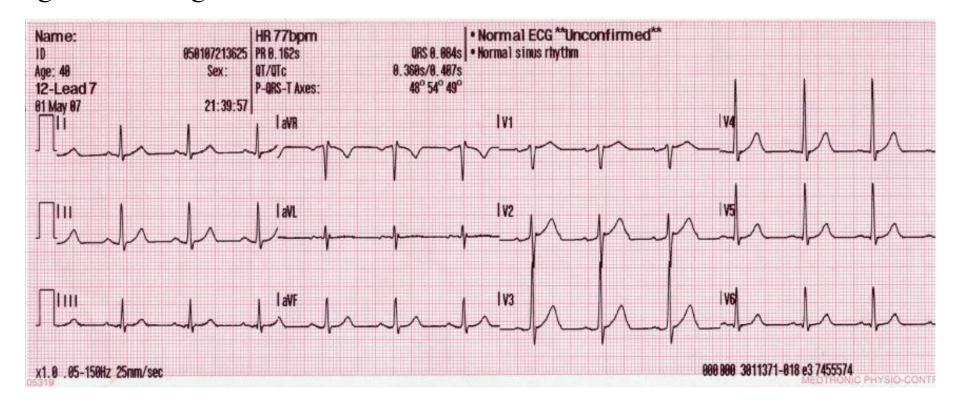




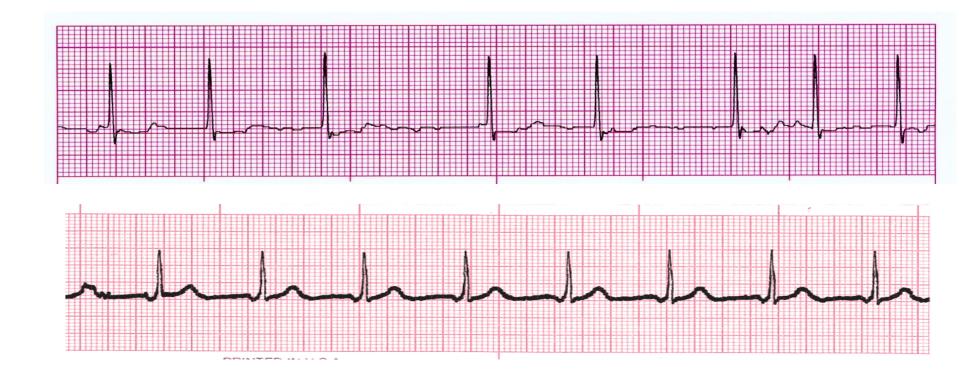


Rhythm

- P wave before every QRS?
- Every P waves followed by QRS?
- Regular Vs Irregular?

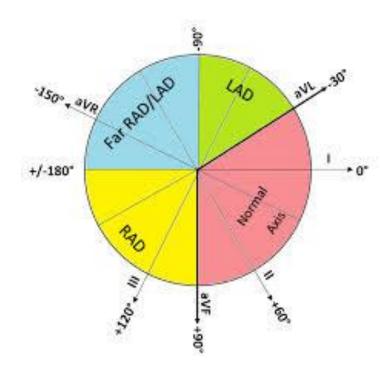


Is it Sinus?

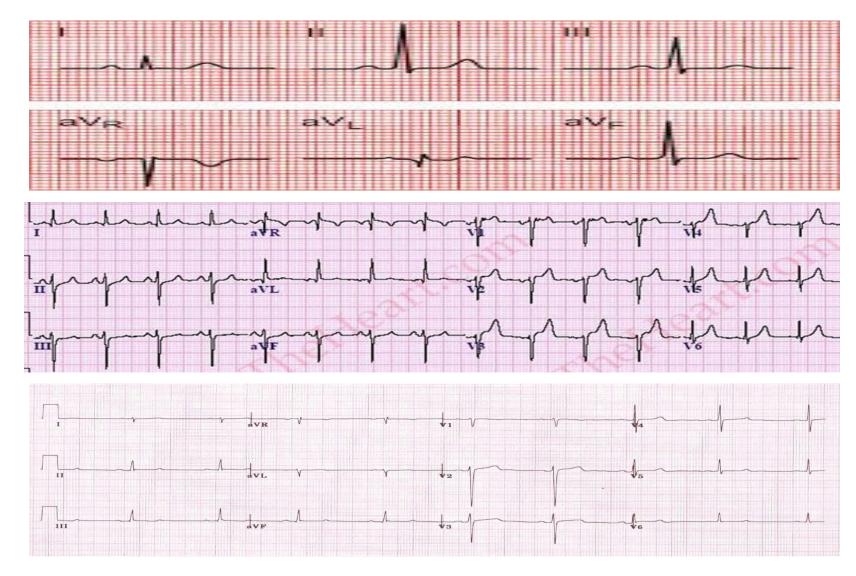


Axis

QRS deflection		Axis
Lead 1	<u>aVF</u>	
Positive	Positive	Normal
Positive	Negative	LAD
Negative	Positive	RAD
Negative	Negative	Extreme RAD or Extreme LAD



What is the Axis?



Intervals

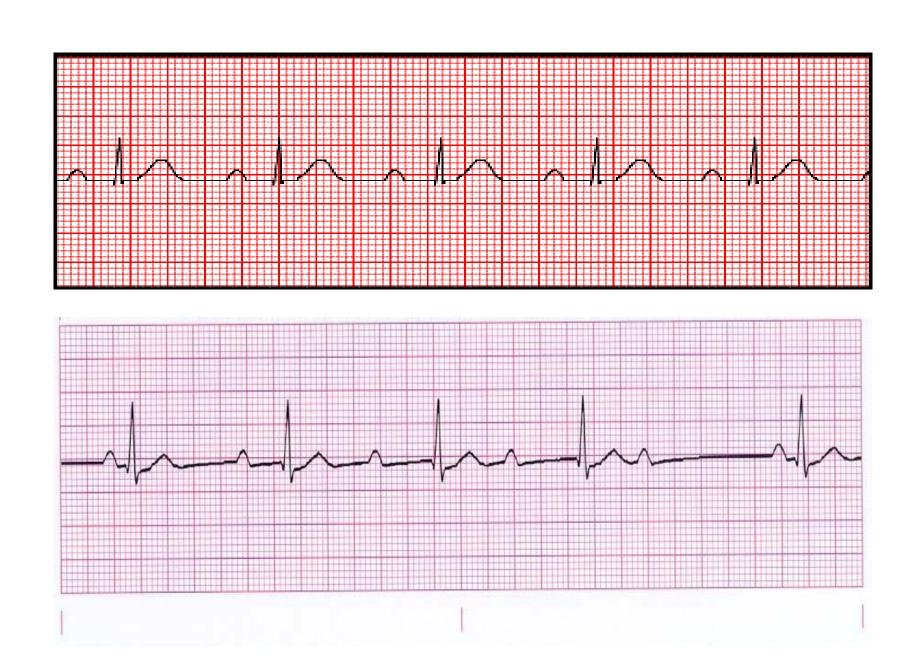
- PR
 - Normal PR interval is 0.20 sec (less than 1 large box)
- QRS
 - Normal QRS < 0.12sec (Less than 3 small Boxes)
- QT interval
 - 450 ms in men and 460 ms in women
 - Based on sex and The HR
 - With normal HR, usually less than Half of the RR interval
- QTc
 - Corrects for the HR
 - $QTm/\sqrt{(R-R)}$

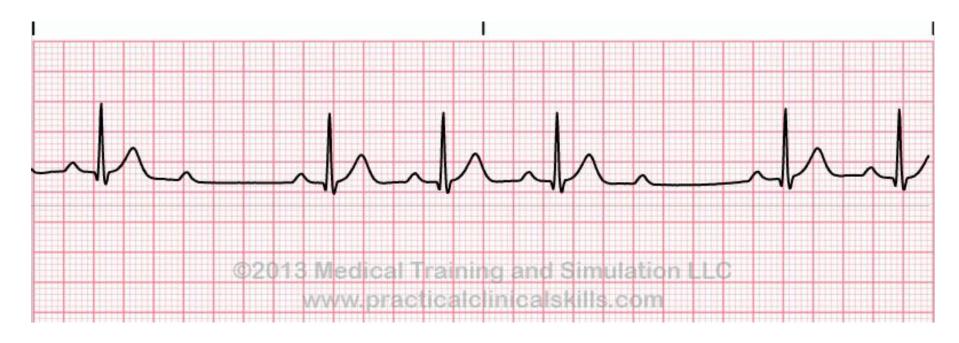
Causes of QT prolongation

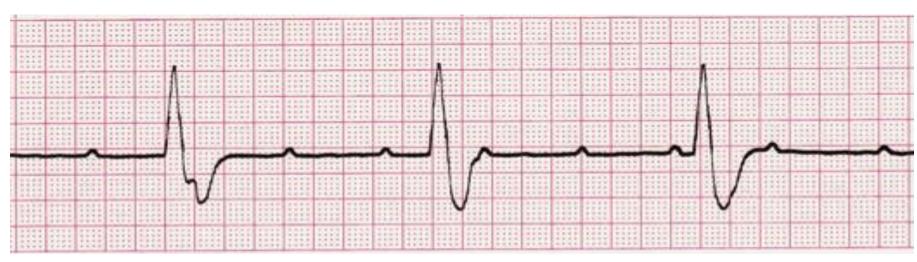
- Drugs (Na channel blockers), Antipsychotics
- Hypocalcemia
- Hypomagnesemia
- Hypokalemia
- Hypothermia
- AMI
- Congenital
- Increased ICP

Blocks

- AV blocks
 - First degree block
 - PR interval fixed and > 0.2 sec
 - Second degree block, Mobitz type 1
 - PR gradually lengthened, then drop QRS
 - Second degree block, Mobitz type 2
 - PR fixed, but drop QRS randomly
 - Type 3 block
 - PR and QRS dissociated

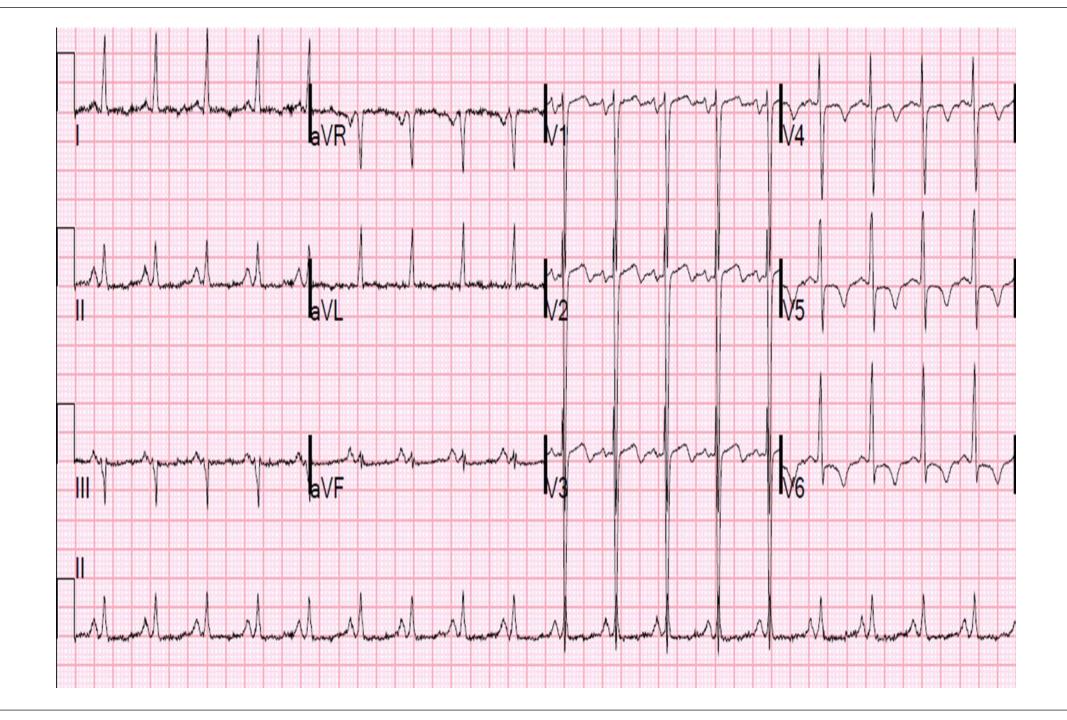






P Wave

- Upright In Lead II→ Sinus rhythm
- The P wave can also help with atrial enlargement
 - L Atrial Enlargement
 - Lead II: Bifid P wave with total P wave duration of >110ms
 - Lead V1: Biphasic P wave with terminal negative portion > 1mm deep
 - R Atrial Enlargement
 - Lead II: Peaked P waves >2.5mm
 - Lead I: Peaked P wave >1.5mm

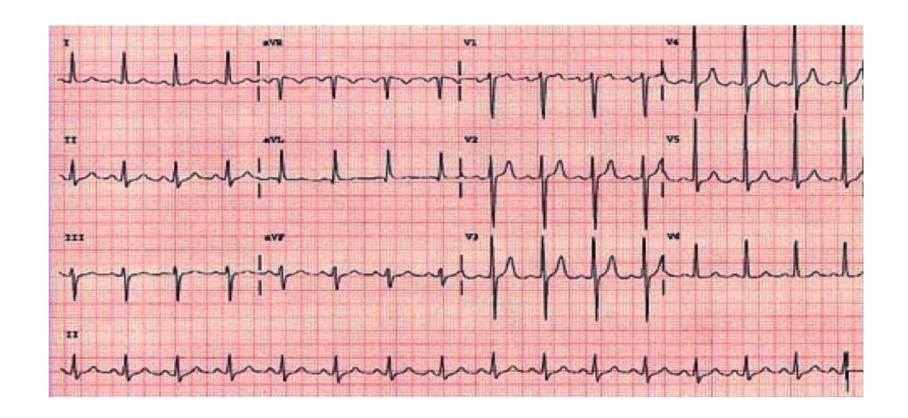


QRS Complex

- Dr. Mohan's 4 things to look for in a QRS complex
 - Amplitude (Helps with LVH)
 - Duration (Bundle Branch)
 - Q waves (Old MIs)
 - R wave progression

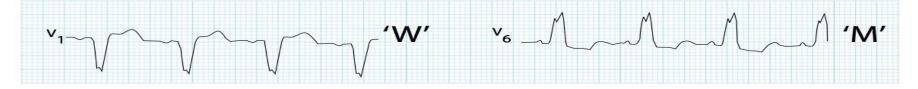
Amplitude

- Add the larger S wave of V1 or V2 in mm, to the larger R wave of V5 or V6.
- Sum is > 35mm = LVH

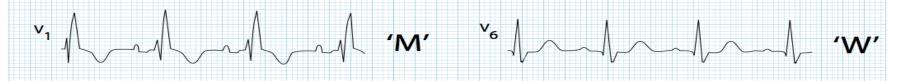


Duration

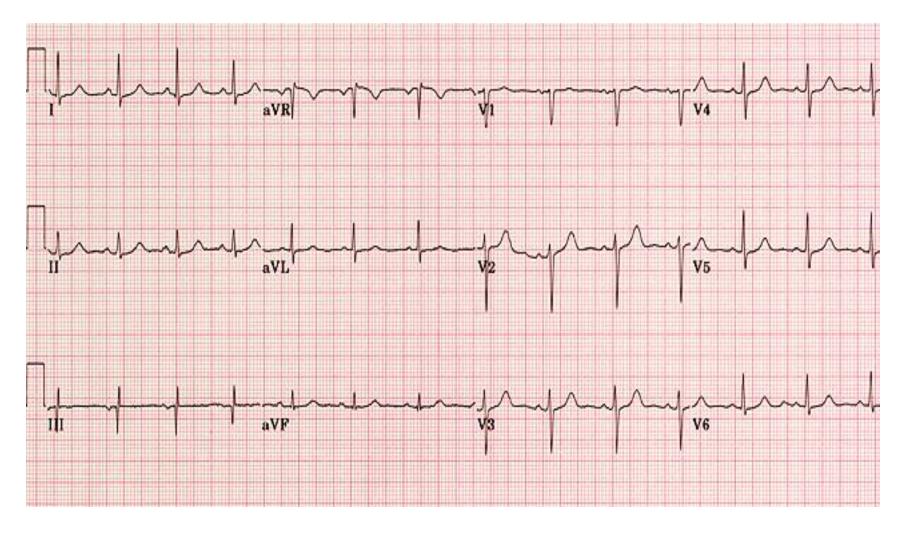
- Normal Duration < 0.12 sec
- If prolonged, have to think about RBBB or LBBB
 - LBBB
 - Dominant S wave in V1 and Broad monophasic R wave in lateral leads (I, aVL, V5-V6)



- RBBB
 - RSR' pattern in V1-3 ('M-shaped' QRS complex) and Wide, slurred S wave in the lateral leads (I, aVL, V5-6)

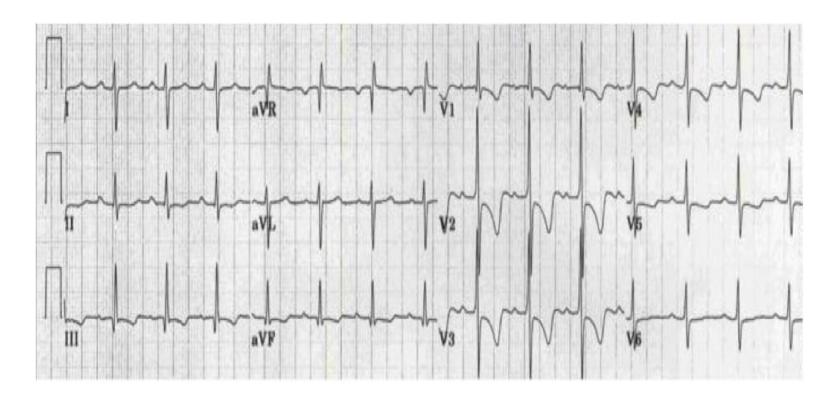


R wave progression



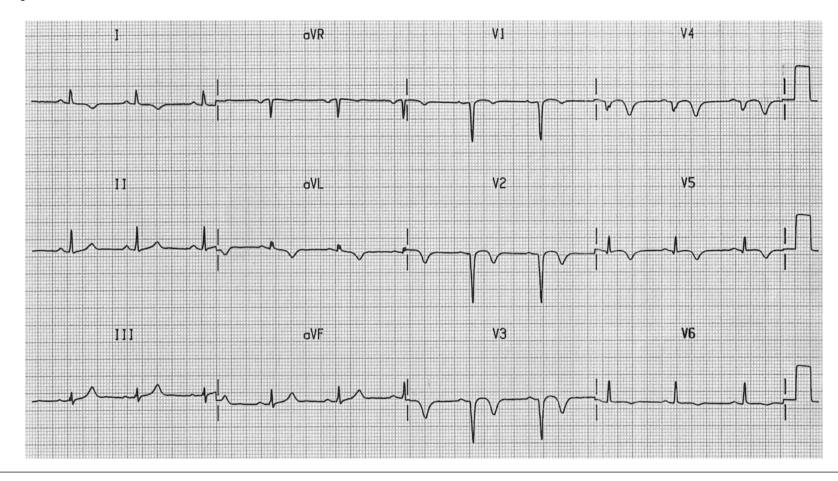
R wave Progression

- Usual Transition between V3-V4
- Early Progression
 - 3 major causes: RBBB, RVH and Posterior MI



R Wave progression

- Late R wave Progression
 - 3 Major causes: LVH, LBBB and Anterior MI



ST segment and T waves

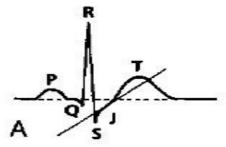
• Non-ST-elevation acute coronary syndrome (NSTEACS) encompasses two main entities: UA and NSTEMI

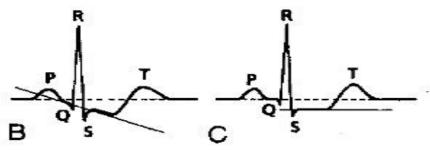
• Two main ECG abnormalities seen with NSTEACS: ST depression and T wave flattening or inversion

• True Transmural ischemia is evidenced by a STEMI on the EKG.

ST Depression

• ST depression can be either upsloping, downsloping, or horizontal (see diagram below).





- Horizontal or downsloping ST depression ≥ 0.5 mm at the J-point in ≥ 2 contiguous leads indicates myocardial ischemia.
- ST depression ≥ 1 mm is more specific and conveys a worse prognosis.
- ST depression \geq 2 mm in \geq 3 leads is associated with a high probability of NSTEMI and predicts significant mortality (35% mortality at 30 days).
- Upsloping ST depression is non-specific for myocardial ischemia.

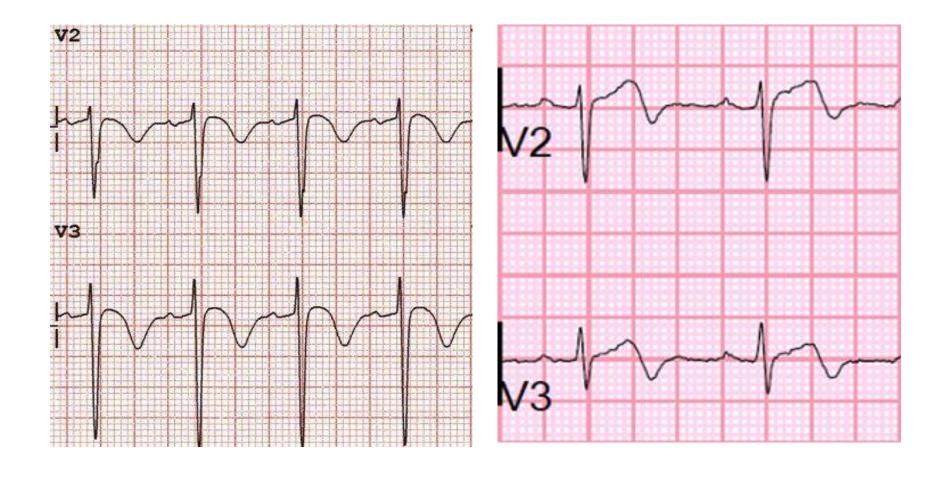
Distribution of ST Depression

- ST depression due to subendocardial ischemia is usually widespread typically present in leads I, II, V4-6 and a variable number of additional leads.
- ullet A pattern of widespread ST depression plus ST elevation in aVR > 1 mm is suggestive of a LM occlusion.
- ST depression localized to a particular territory (esp. inferior or high lateral leads only) is more likely to represent reciprocal change due to STEMI. The corresponding ST elevation may be subtle and difficult to see, but should be sought.

T wave Inversion

- At least 1 mm deep
- Present in ≥ 2 continuous leads that have dominant R waves (R/S ratio ≥ 1)
- Dynamic not present on old ECG or changing over time
- Wellens' syndrome is a pattern of inverted or biphasic T waves in V2-4 (in patients presenting with ischemic chest pain) that is highly specific for critical Stenosis of the left anterior descending artery.

Wellens T Waves



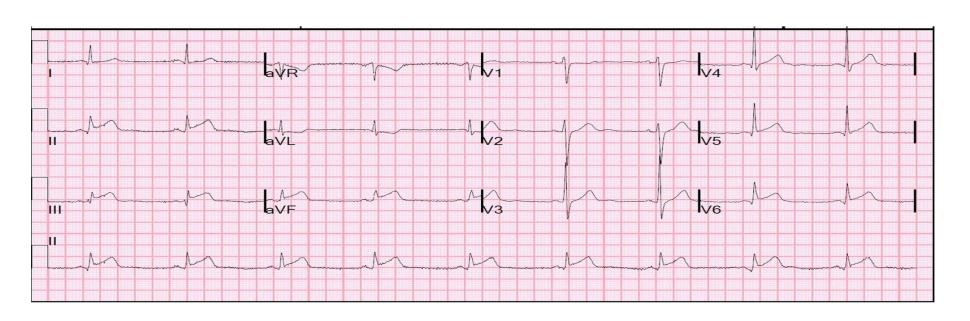
STEMI

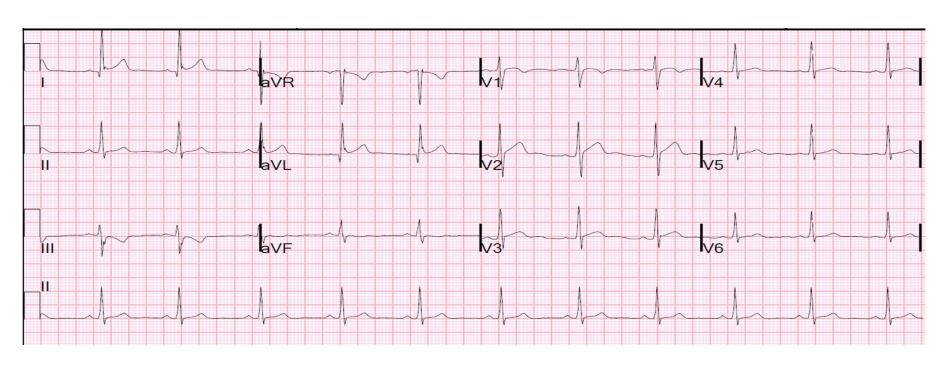
- ≥2 mm of ST segment elevation in 2 contiguous precordial leads in men (1.5 mm for women)
- ≥1mm in other leads (2 contiguous)
- 2 Other Categories considered to be STEMI even though there might not be true ST elevations: New LBBB and Posterior MI

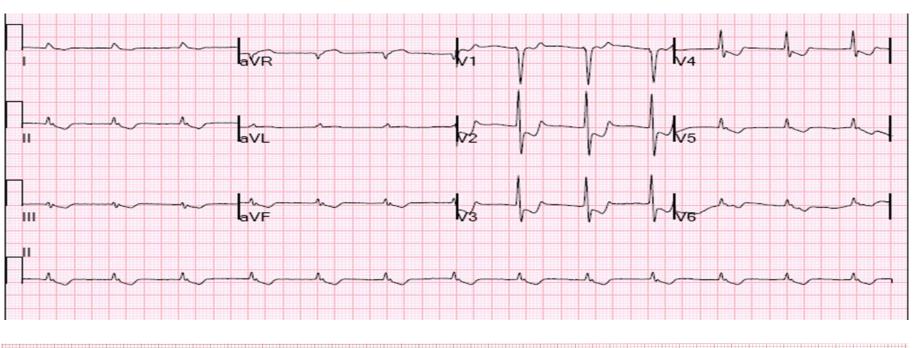
SITE	FACING	RECIPROCAL
INFERIOR	II, III, aVF	I, aVL EMS12Lead.com
HIGH LATERAL	I, aVL	II, III, aVF
ANTERIOR	V1, V2, V3, V4	NONE
POSTERIOR	NONE	V1, V2, V3, V4

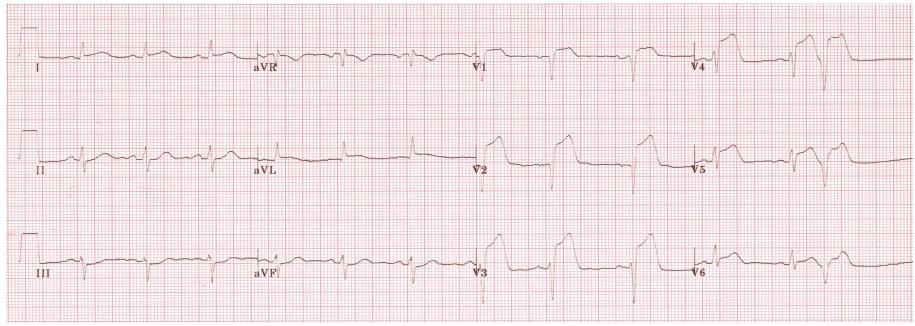
STEMI

- **Anterior STEMI:** ST elevation in the precordial leads + I and aVL (LAD territory)
- **Posterior STEMI**: reciprocal ST depressions in V1-V3 (ST elevation in post leads), may have component of inferior ischemia as well (ST elevations in II, III and aVF) Often occurs w/ inferior MI (L Cx)
- Inferior STEMI: ST elevation in II, III and aVF (+ ST elevation in R-sided precordial leads), reciprocal changes in I and aVL (R coronary or L Cx)



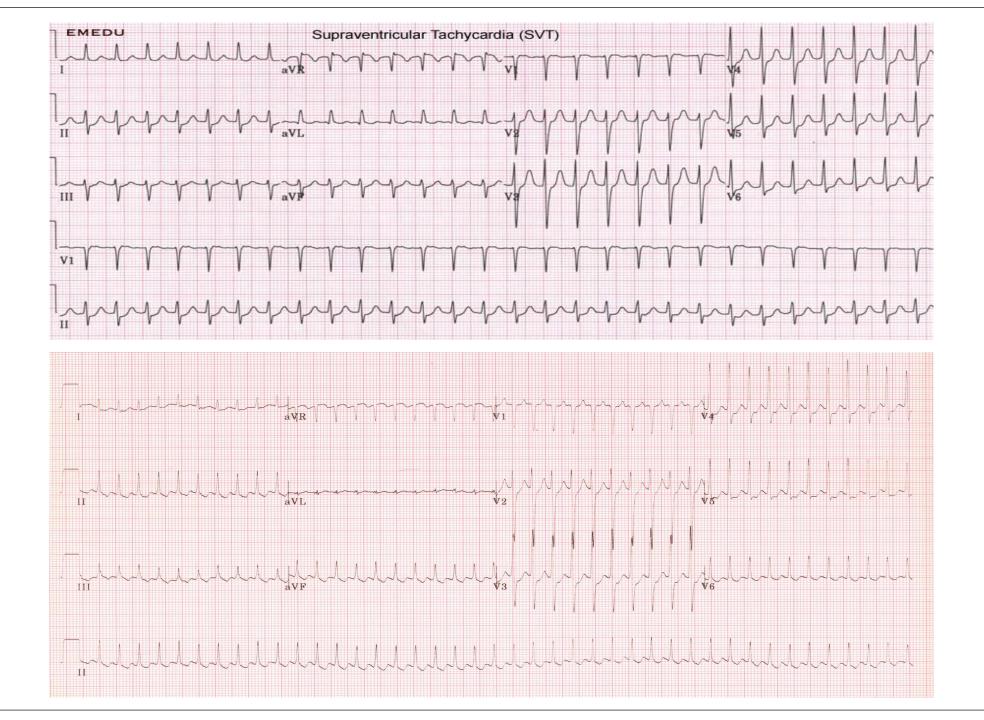




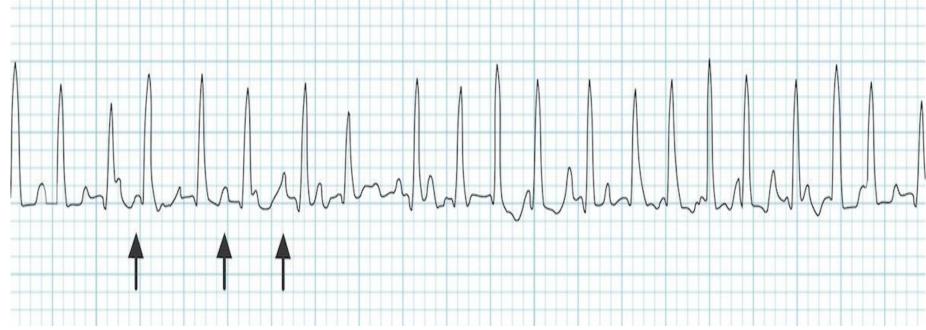


Tachycardia

- Narrow Complex Vs. Wide Complex
- Regular Vs. Irregular
- Regular Narrow Complex Tachycardia: Sinus Tachycardia, Atrial Tachycardia, A flutter, SVT, AVNRT
- Irregular Narrow Complex Tachycardia: A Fib, A flutter with Variable Block, MAT etc
- Regular Wide complex Tachycardia: VT, V-Flutter, Tachycardia with aberrancy, Hyperkalemia
- Irregular Wide Complex Tachycardia: Torsades, V-Fib Etc.







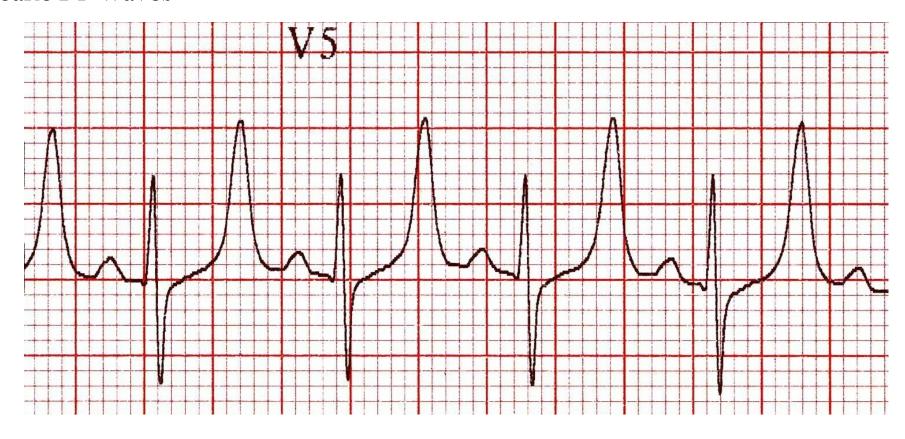


Bradycardia

- Narrow Vs Wide Complex
- Regular Vs. Irregular
- Regular narrow complex bradycardia: Sinus, Junctional, Complete AV block (junctional escape), A-flutter with high degree block.
- Irregular narrow complex bradycardia: Sinus, A-fib with slow ventricular response, A-flutter with variable block, Type I and Type II second degree block.
- Regular wide complex bradycardia: Idioventricular rhythm, Complete AV block (ventricular escape), Regular bradycardias with aberrancy or bundle branch block
- Irregular wide complex bradycardia: Type 1 and type 2 second degree blocks, Irregular bradycardias with bundle branch block.

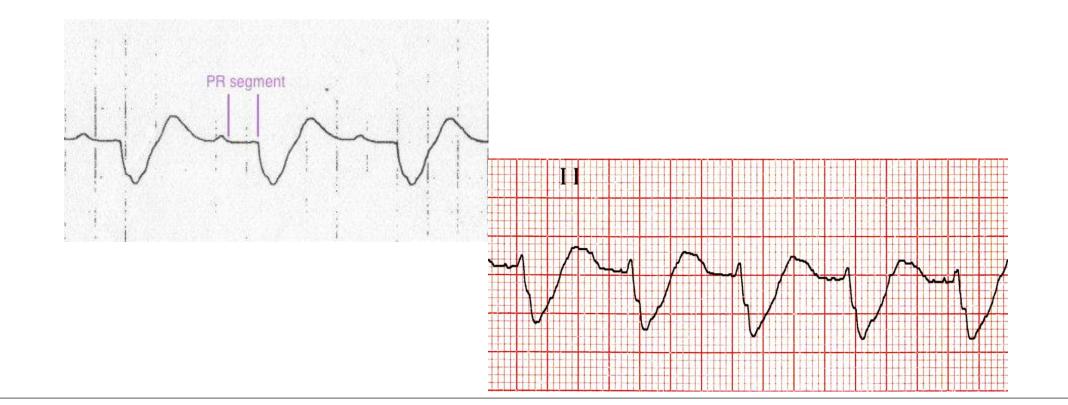
Hyperkalemia

- \bullet > 5.5 mEq/L is associated with repolarization abnormalities
- Peaked T waves



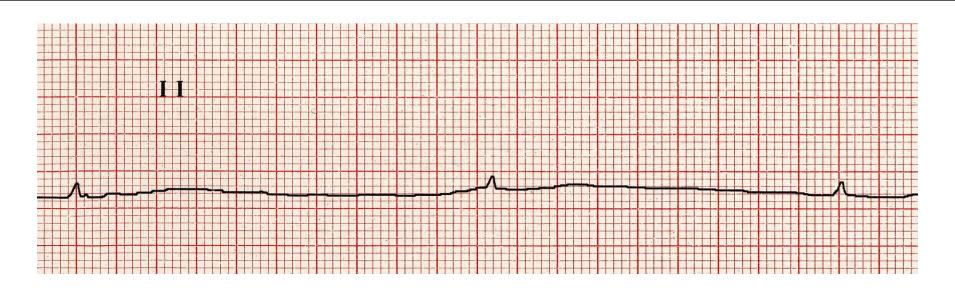
Progression of Hyperkalemia

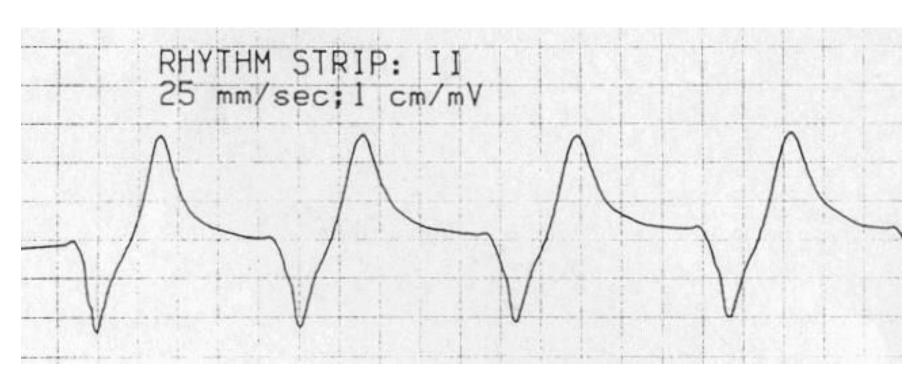
- > 6.5 mEq/L is associated with progressive paralysis of the atria
- P wave widens and flattens, PR segment lengthens, P waves eventually disappear



Hyperkalemia Continued

- > 7.0 mEq/L is associated with conduction abnormalities and bradycardia.
- Prolonged QRS interval with bizarre QRS morphology, High-grade AV block with slow junctional and ventricular escape rhythm, Any kind of conduction block (bundle branch blocks, fascicular blocks), Sinus bradycardia or slow AF, Development of a sine wave appearance (a pre-terminal rhythm)





Hyperkalemia Continued

- > 9.0 mEq/L causes cardiac arrest.
 - Asystole
 - Ventricular fibrillation
 - PEA with bizarre, wide complex rhythm