# ECG

### **Common Cases in Clinical Practice**

### Bader Almahdi, MD.

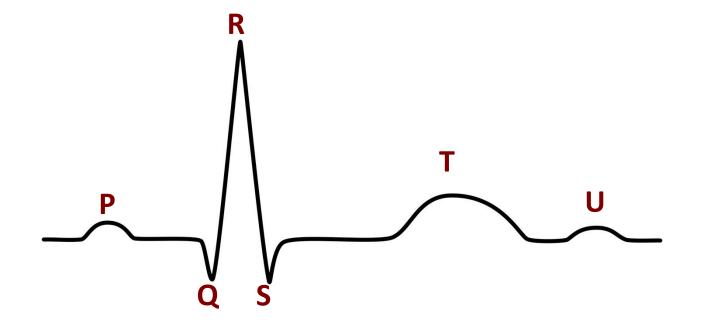
**Consultant Interventional Cardiologist** 

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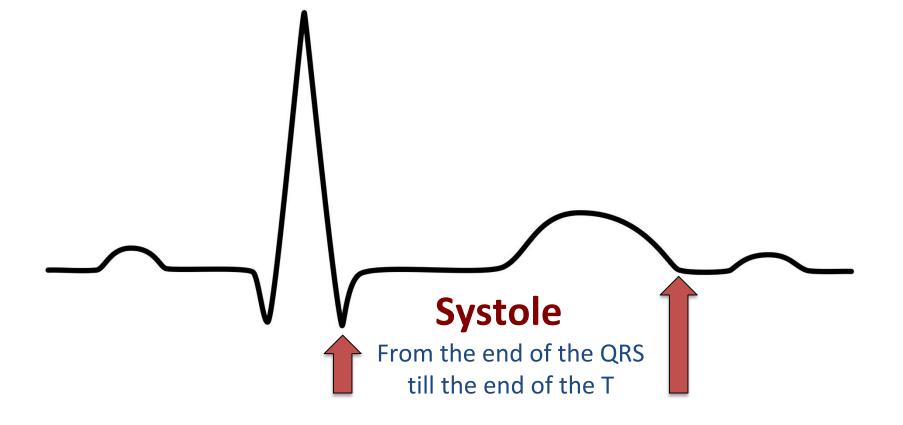
August 2021

### Mastering the Language

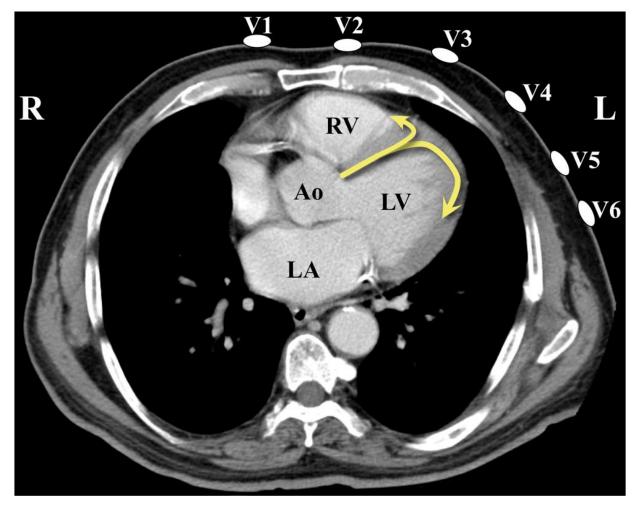
- PQRST is the letter sequence of the alphabets.
- They were given these letters to make them sound cool and prestigious!

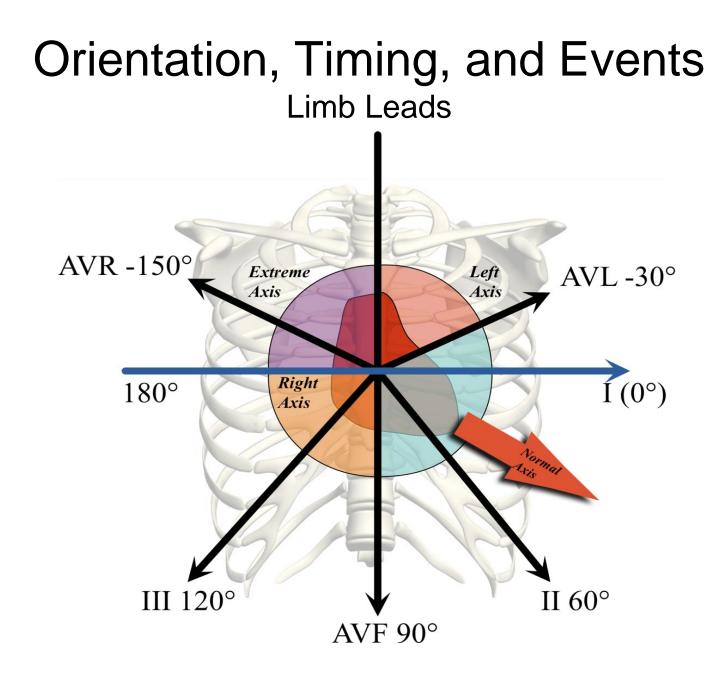


#### **Orientation, Timing, and Events** Electrical activity first. Mechanical activity later.



### Orientation, Timing, and Events Precordial Leads





### Orientation, Timing, and Events Limb Leads

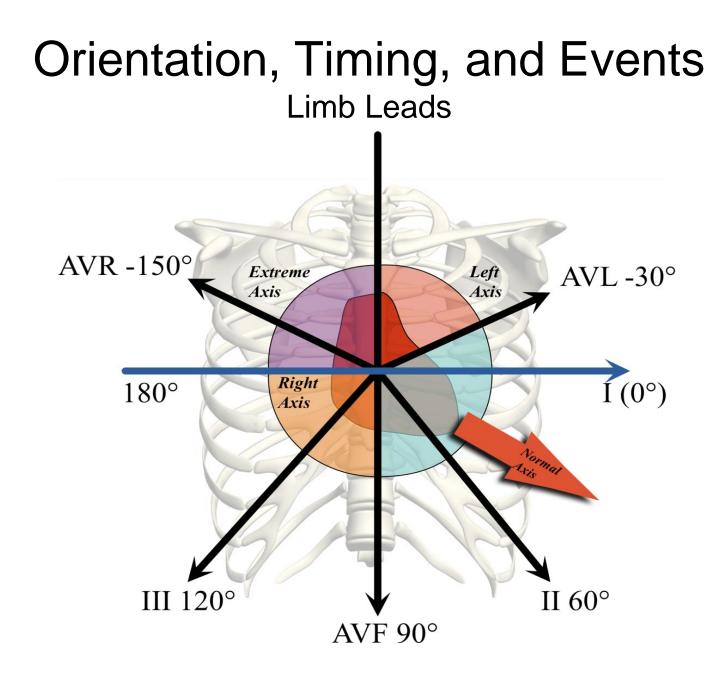
AVR -150°

eme

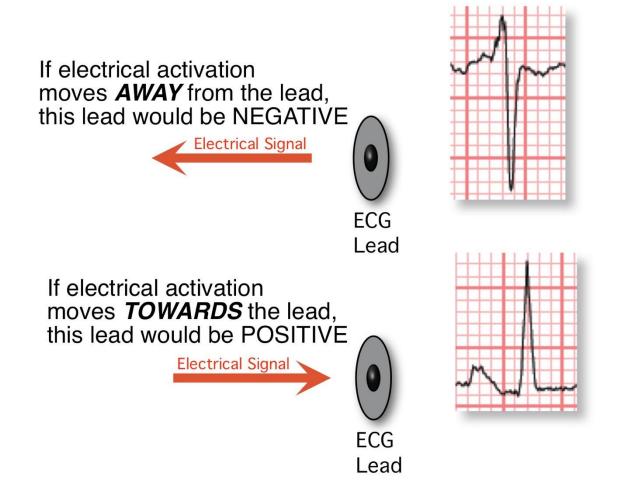
AVL -30°

Draw them NOW, Please

III 120° II 60° AVF 90°



## Lead Deflection



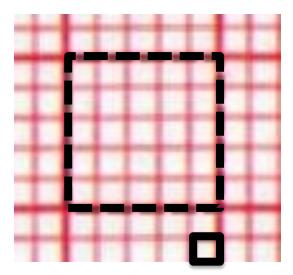
This rule applies to P wave as well as the QRS complex and T wave

### Orientation, Timing, and Events Squares



One large square= 200 ms (0.2 sec)

One small square= 40 ms (0.04 sec)

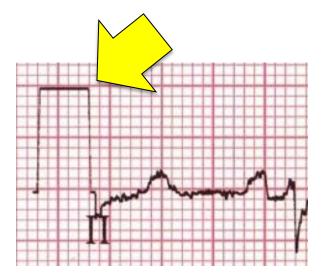


### Orientation, Timing, and Events The Standard

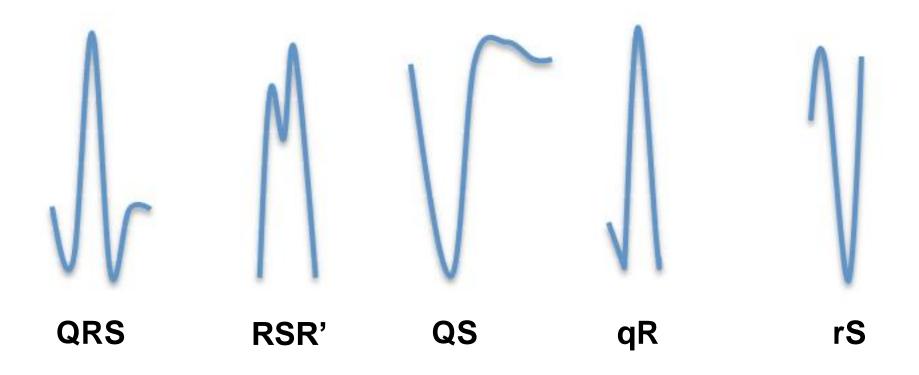
This rectangle at the margin.

It should be 10mm high.

It could be *doubled* using the machine settings if we want to see a "small" wave.



## ECG Wave Morphology



Draw them. Label them.

### Nomenclature According to Heart Rate

**Tachycardia**: Heart rate >100 bpm.

**Bradycardia**: Heart rate <60 bpm.

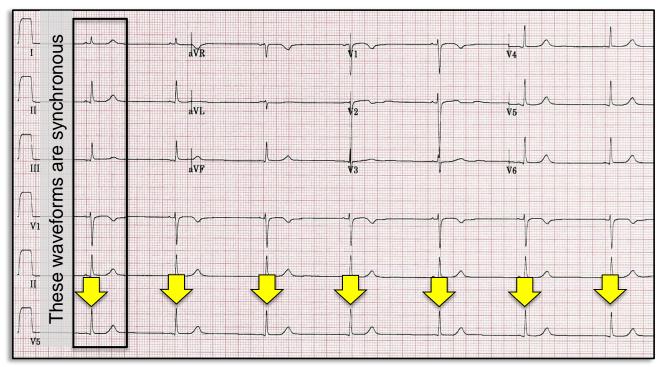
**Sinus rhythm**: P waves originate from the SA node.

Junctional (AV nodal) rhythm: Orchestrated by the AV node.

**Escape**: Cardiac activity "escaping" the control of a particular rhythm.

### Heart Rate: Clues

How to calculate the heart rate?

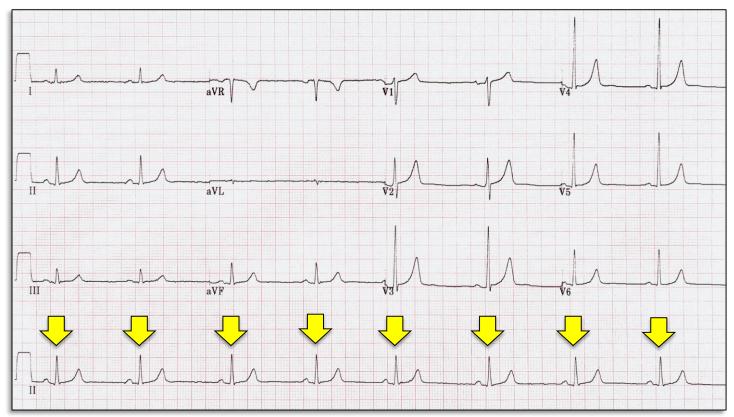


This 12-Lead ECG strip is recorded over 10 seconds

Count the R waves and multiply them by 6.

### Heart Rate: Exercise

Calculate the heart rate in this asymptomatic 28 year old male.



#### 8 R waves X 6 = 48 bpm.

Variable R-R interval with no change in PR interval.

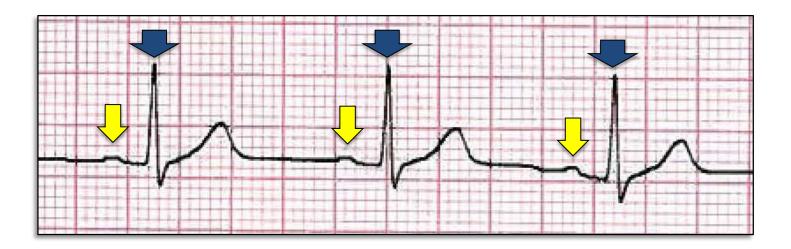
This is **sinus arrhythmia**. A sign of a good vagal tone. i.e. a healthy heart.

## AV association

#### Sinus rhythm

1. Upright P waves in the inferior leads.

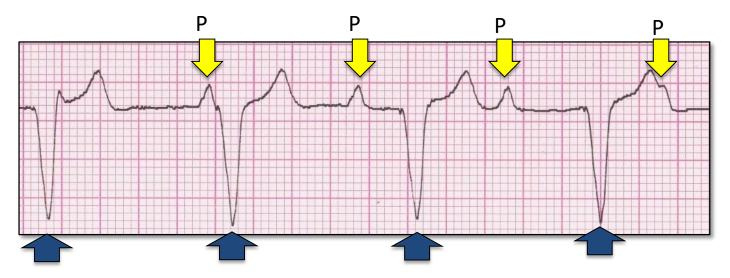
- 2. P waves comes first. QRS complexes later.
- 3. AV association: every P is followed by a QRS.



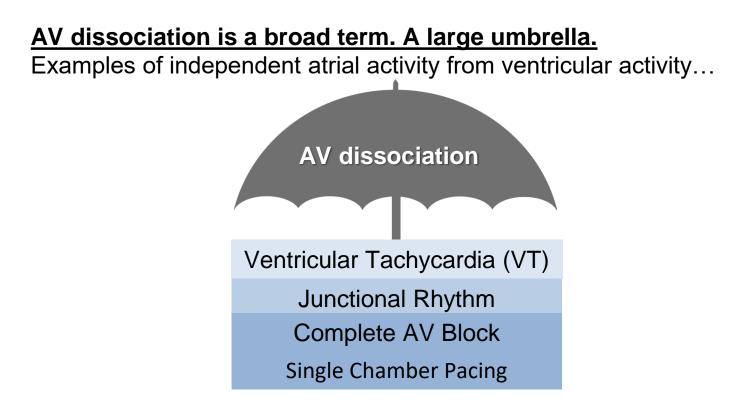
## AV dissociation

#### **AV dissociation**

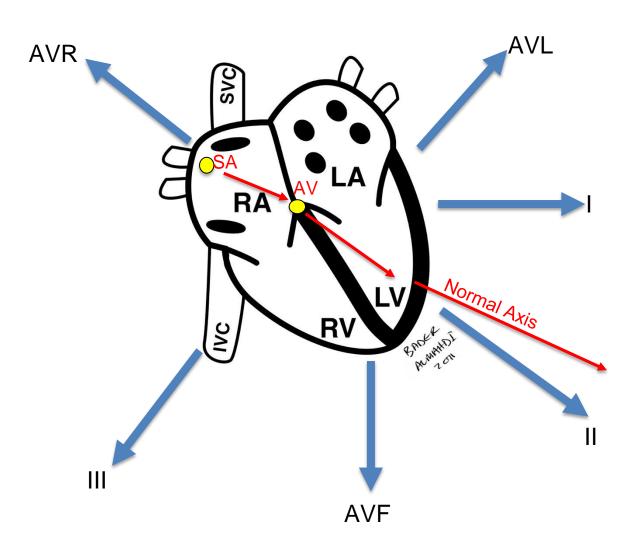
Atrial activity is independent from ventricular activity



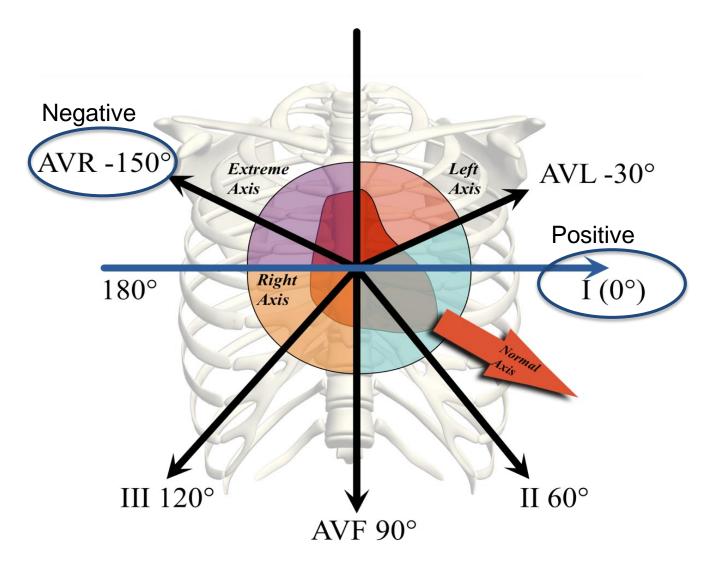
## AV dissociation

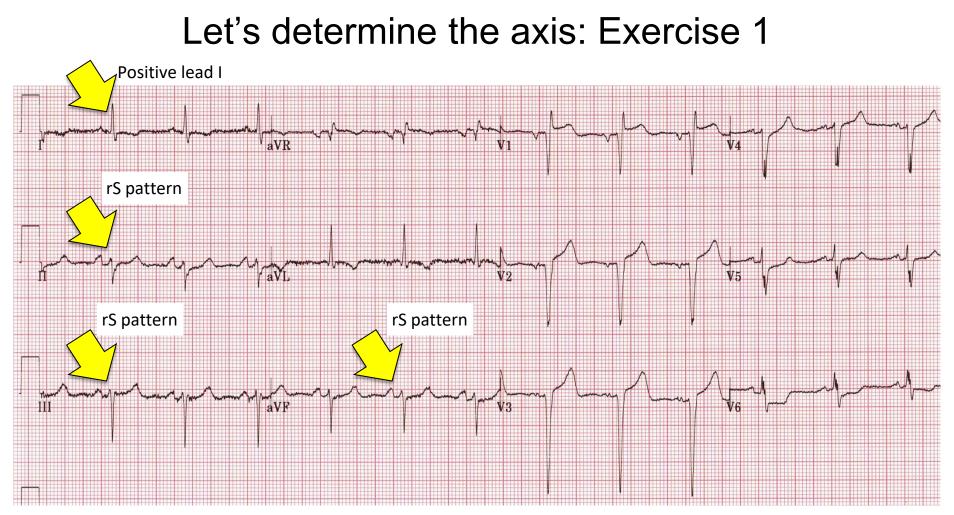


## Determining the Axis



### Putting Vectors into Perspective





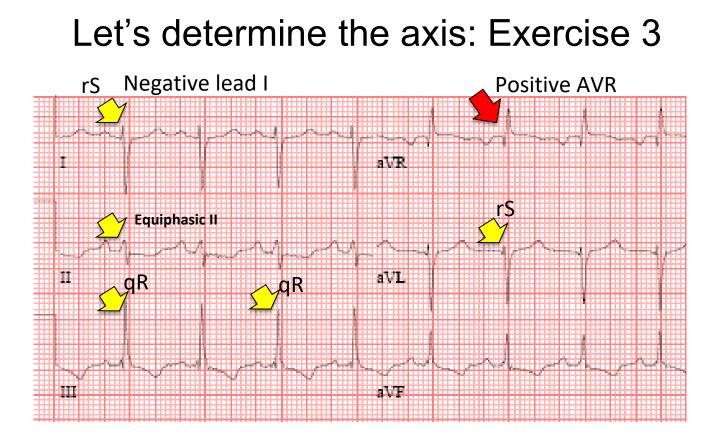
#### Left Axis Deviation



### Let's determine the axis: Exercise 2 Negative lead I aVR V1 **Positive AVR** QS pattern aVI V2 QS pattern QS pattern ΊΠ

Superior Axis. Extreme Axis. North West. No mans land.





Right Axis Deviation. The equiphasic lead has the axis perpendicular to it.



#### Common terminology

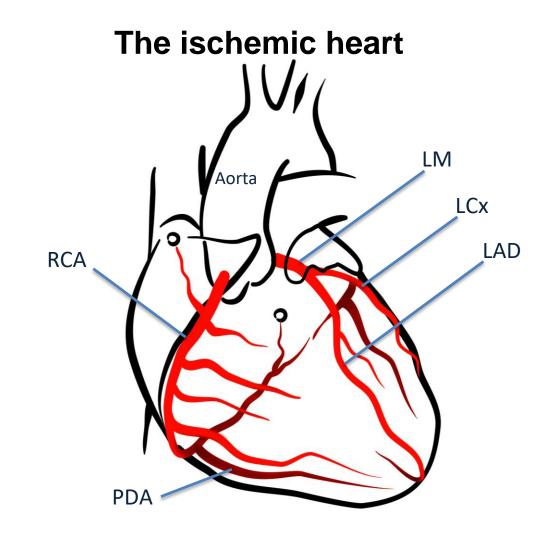
V1-V2: septal leads.

V3-V4: anterior leads.

I, AVL, V5-V6: lateral leads.

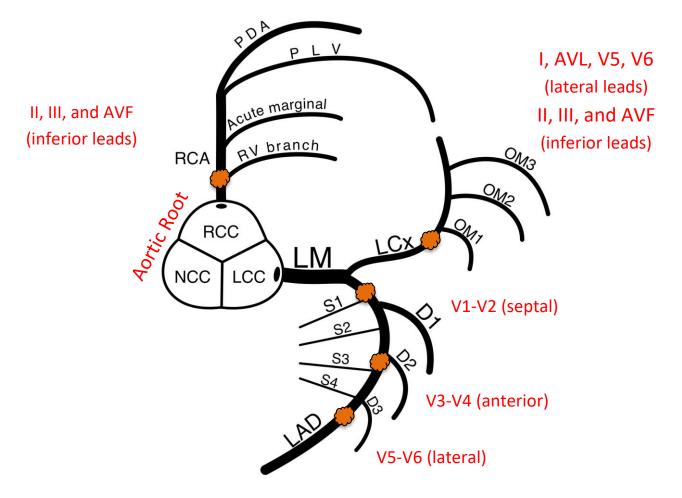
II, III, AVF: inferior leads.

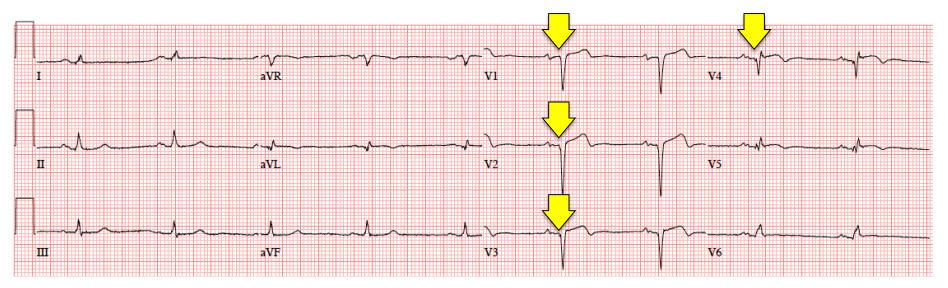
**Dominance**: the dominant vessel is the one which gives off the posterior descending artery (PDA). In 75% of the population, it is the RCA. Occlusion of the dominant vessel might cause complete AV block.



#### **The Ischemic Heart**

(Coronary Artery Layout, ST elevation MI)



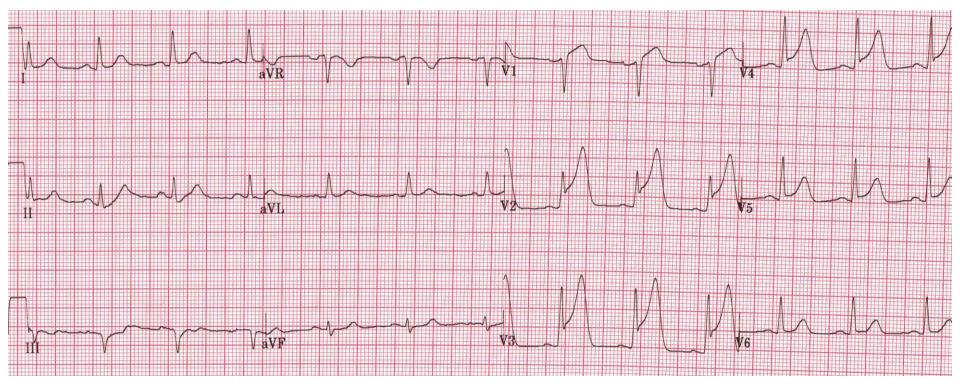


Q waves (QS pattern) in V1-4.

Evolved (Old) anterior wall MI.

R waves are lost. Damage has already been done.

Low voltage in limb and precordial leads.



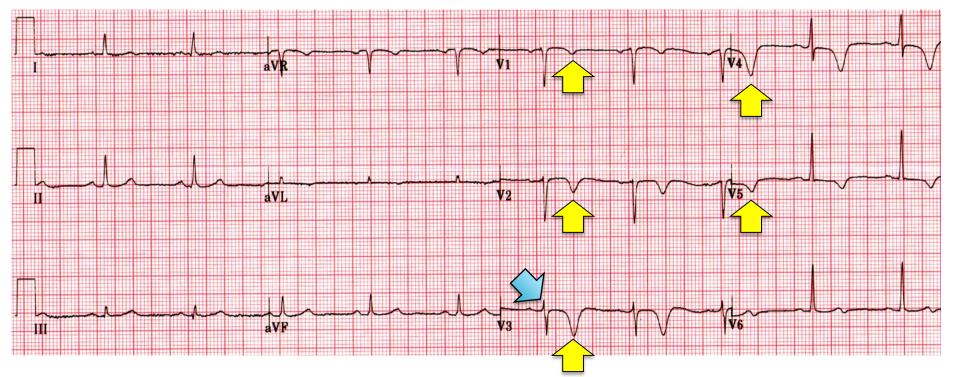
ST elevation in V1-V5.

Anteroseptal wall MI.

LAD territory. Thrombus most likely is at the proximal segment of the

LAD.

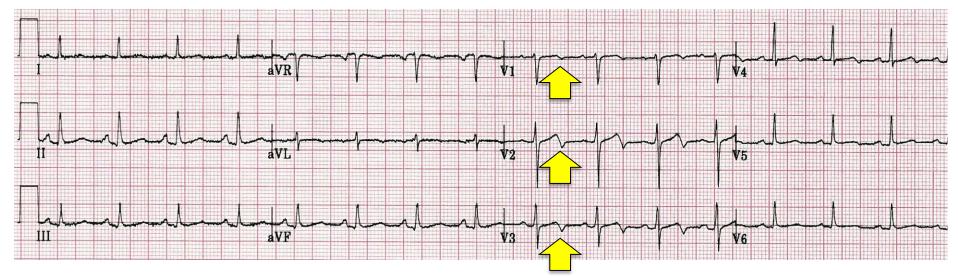
R waves not lost yet.



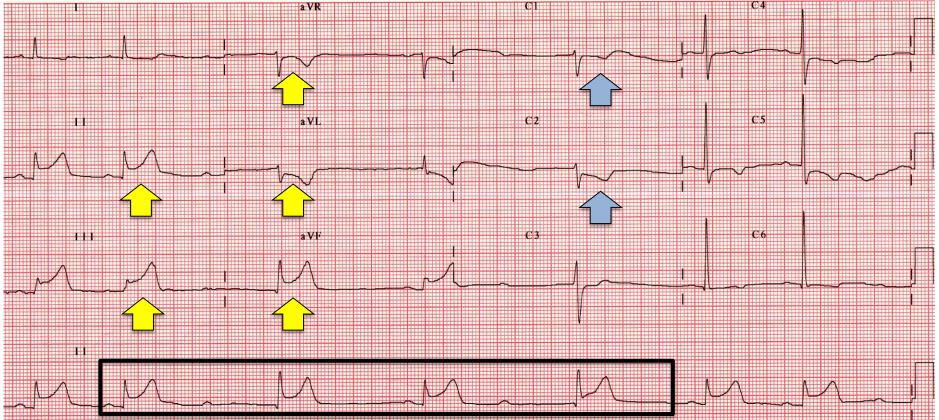
Deep, symmetrical T wave inversion in precordial leads.

#### Wellens' syndrome: proximal LAD disease

Poor R wave progression (R in V3 is <3mm) is also a sign of LAD disease.



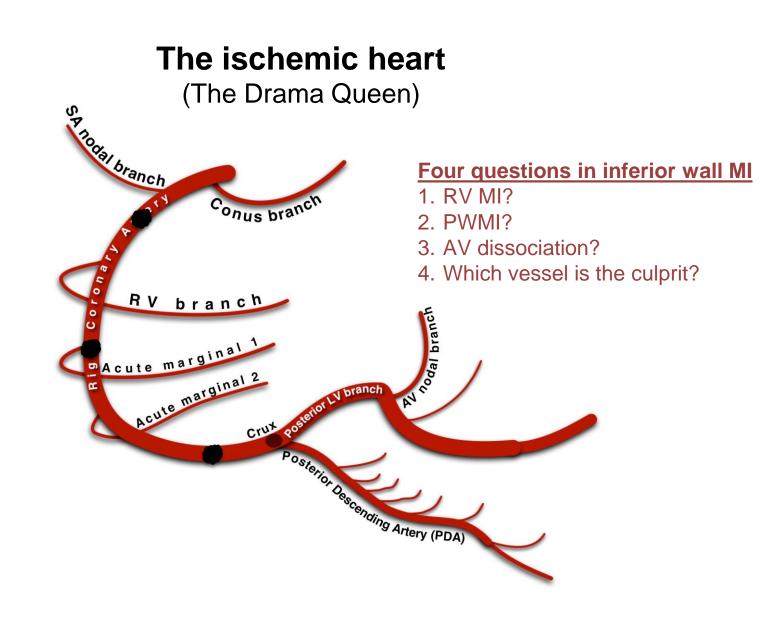
Biphasic T waves in precordial leads. **Another variant of Wellens syndrome** Proximal LAD disease



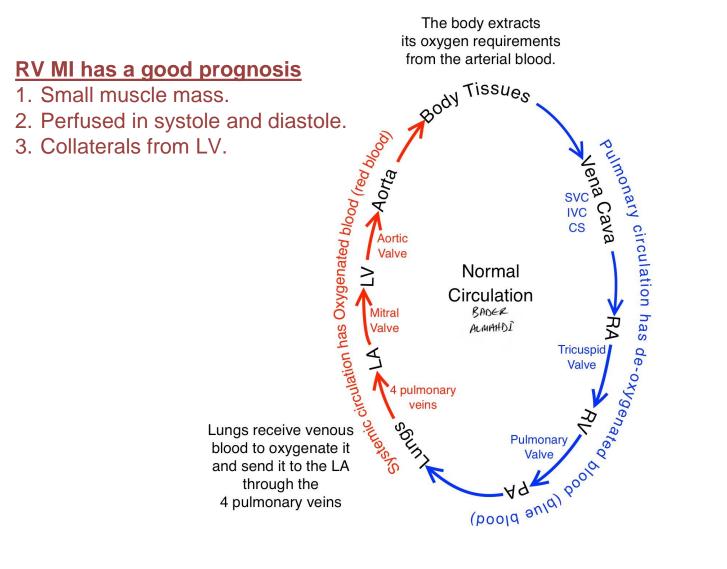
ST elevation in II, III, and AVF (inferior leads) with *reciprocal* changes in AVL and AVR. Posterior wall MI

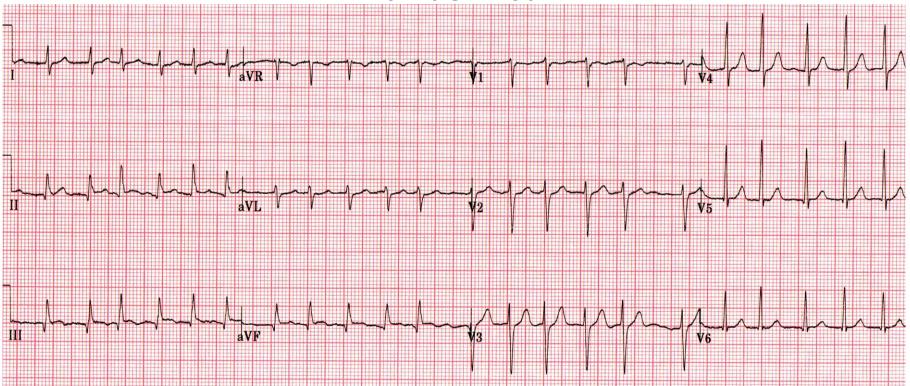
AV dissociation

Which coronary artery is the culprit? RCA versus LCx.



### **RV infarction** Explanation of signs and symptoms



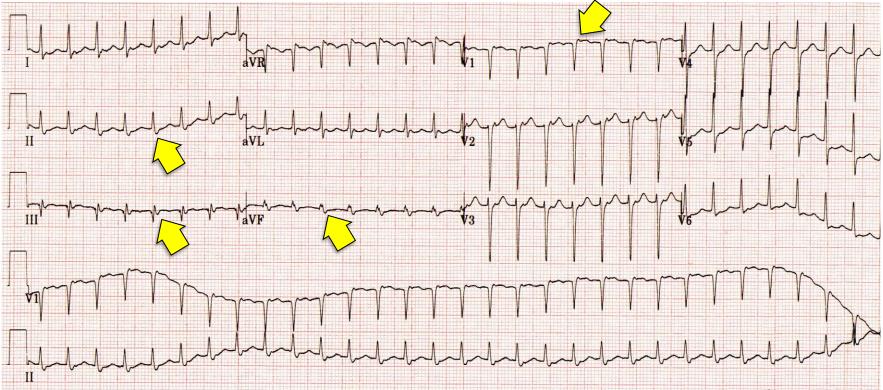


Tachycardia. Absent P waves. Irregular rhythm. Narrow QRS complexes.

Atrial fibrillation. The most common tachyarrhythmia.

The acute risk here is the formation of thrombus in the left atrium.

Chronically fast AF might lead to tachyarrhythmia-induced cardiomyopathy.

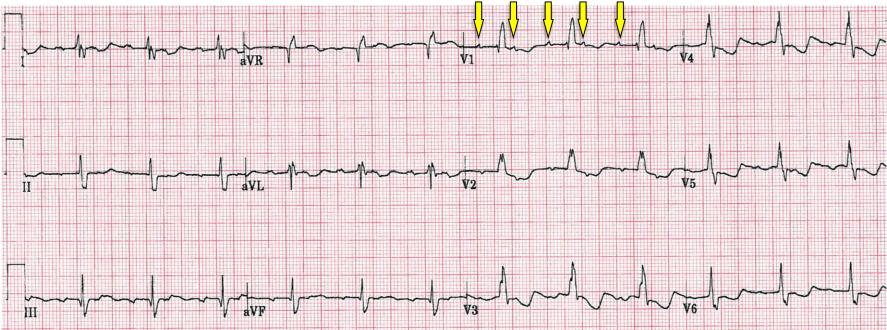


Tachycardia. Regular rhythm. Narrow complexes. No obvious P waves. Pseudo R in V1. Pseudo S in II, III, AVF.

#### AV nodal re-entry tachycardia (AVNRT)

Common arrhythmia in the young and healthy.

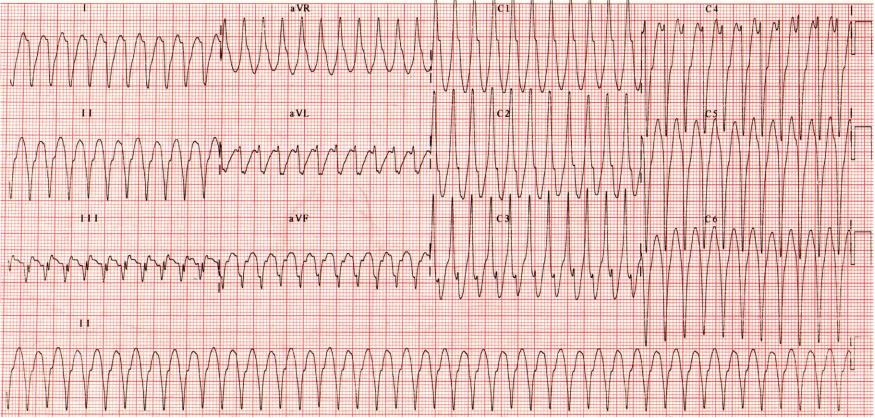
Aborted by Valsalva maneuver, IV adenosine/verapamil, or DC cardioversion.



Regular rhythm. Two P waves per QRS complex. Atrial rate>100/min.

Atrial tachycardia with 2:1 block.

This is common with digoxin toxicity.



Wide complex tachycardia. No visible P waves. Superior axis.

#### Ventricular tachycardia.

A medical emergency. If hemodynamically compromised. Then DC shock.

Slow heart rates are called "Bradyarrhythmias" on the ECG.

#### Classification of bradyarrhythmias depends on..

#### Are they normal or not?

E.g. athlete's heart. Good vagal tone.

#### Do they cause symptoms?

E.g. Fatigue, palpitations, syncope, presyncope.

#### Is the problem temporary?

Should I treat the underlying cause and that's it?

#### Is the problem permanent?

In other words, will patient require permanent pacing?

#### Where does the rhythm come from?

#### Sinus node?

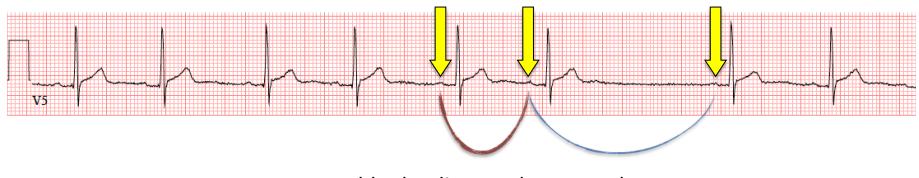
Sinus bradycardia. Sino-atrial exit block / sinus arrest. First / Second degree AV block.

#### AV node? (AV node lies at the AV junction)

Junctional escape rhythm. Accelerated junctional rhythm.

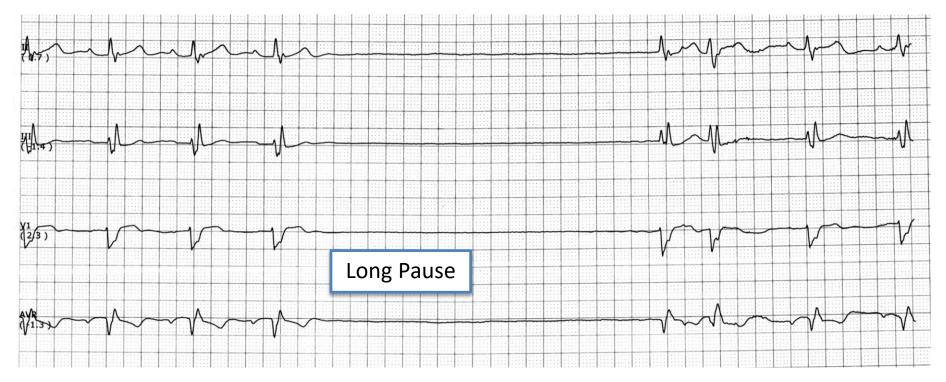
#### Ventricles?

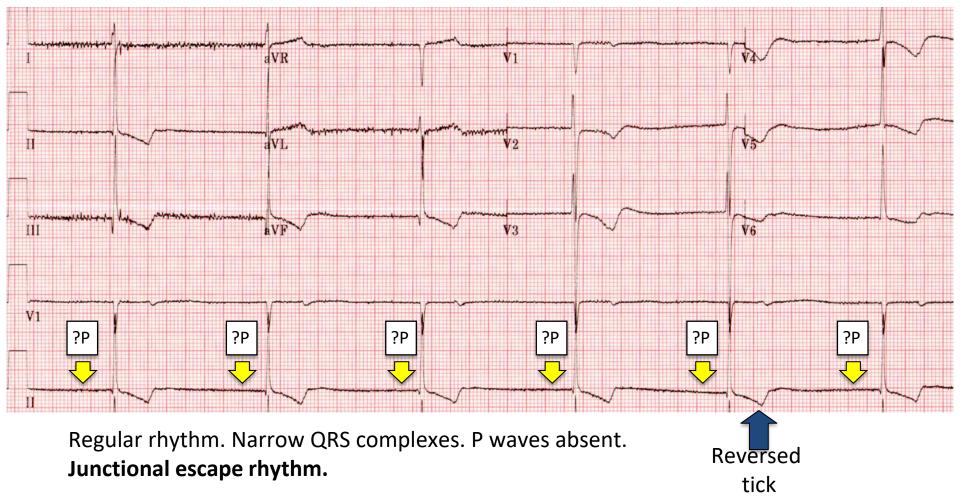
Complete AV block.

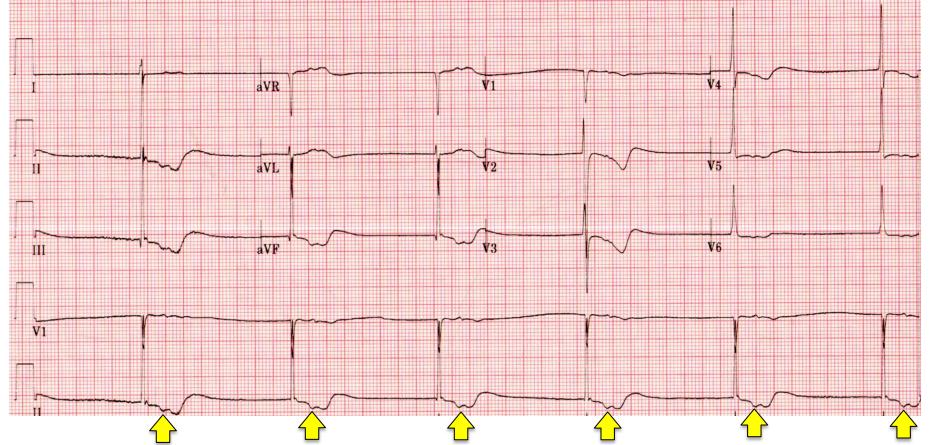


Double the distance between the two P waves

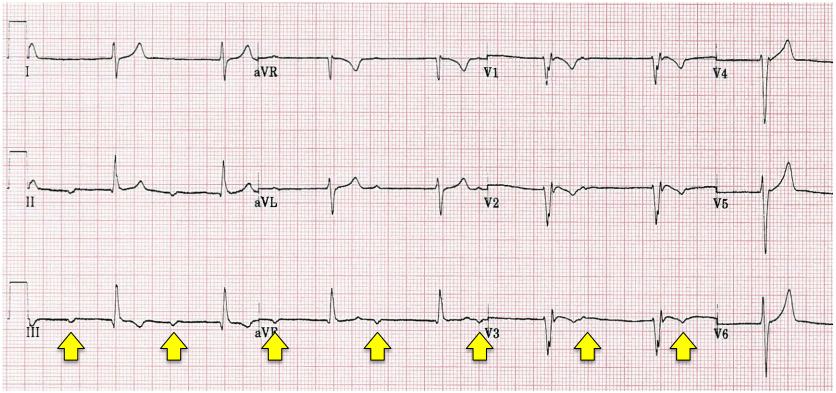
Sino-atrial exit block.





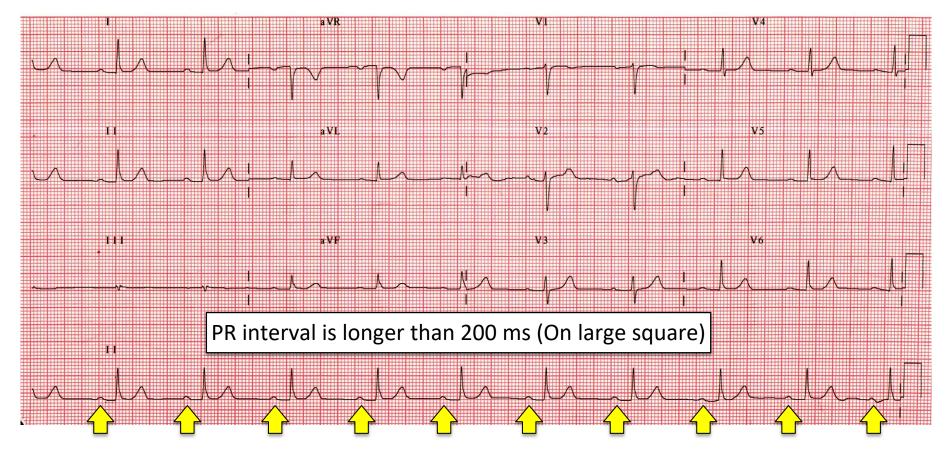


Retrograde inverted P waves. Narrow QRS complexes. Junctional escape rhythm.

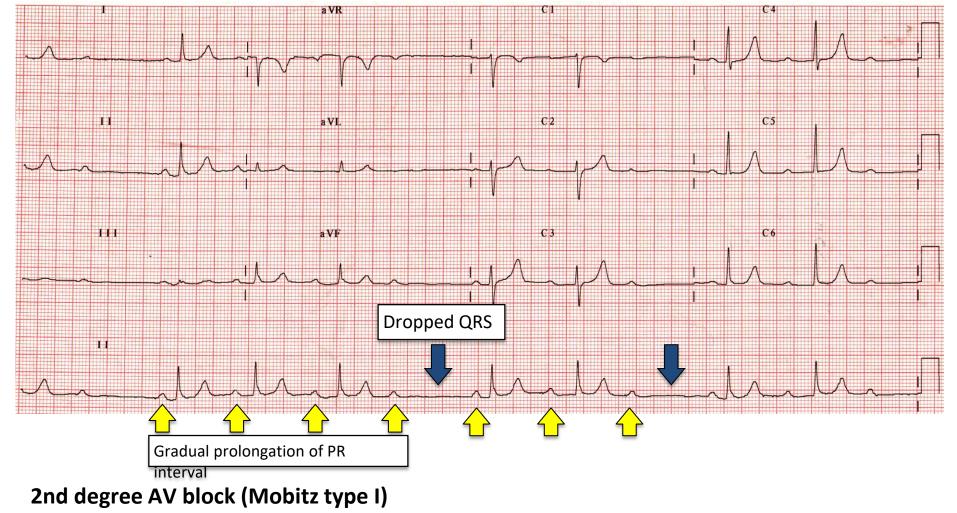


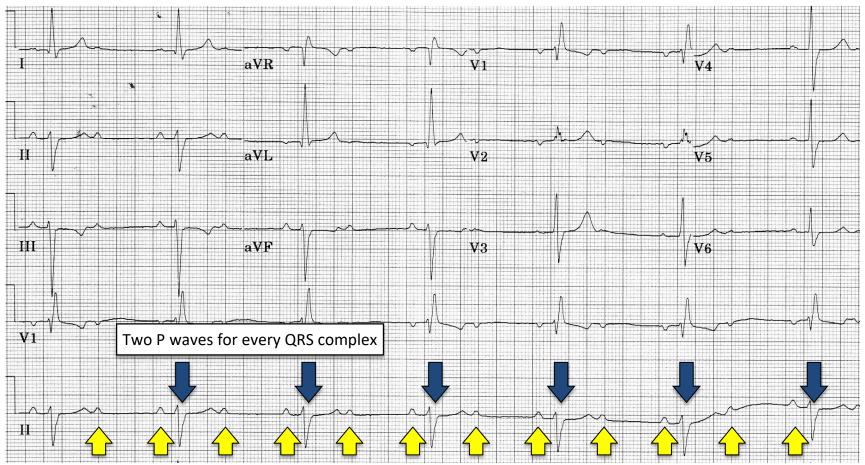
Inverted P waves. AV dissociation. Narrow QRS complexes. Junctional escape rhythm.

P waves in II, III, AVF are negative. Activation from down to up.



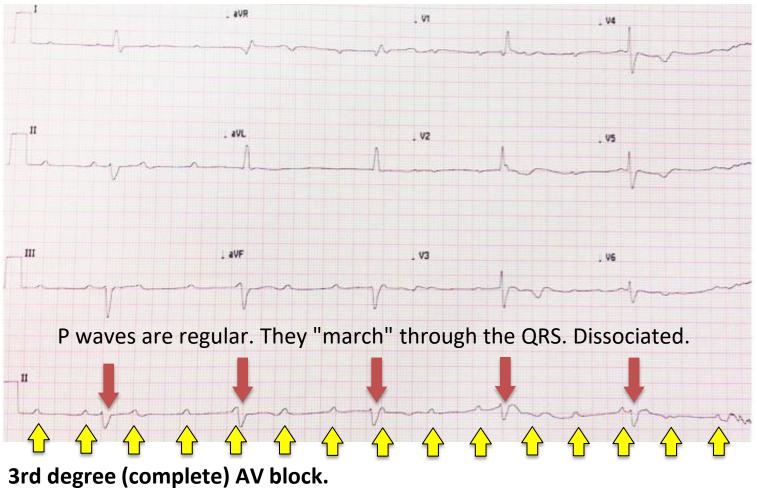
**1st degree AV block** 





#### 2nd degree AV block (Mobitz type II)

A serious arrhythmia. Might progress to complete AV block. Pacing required.



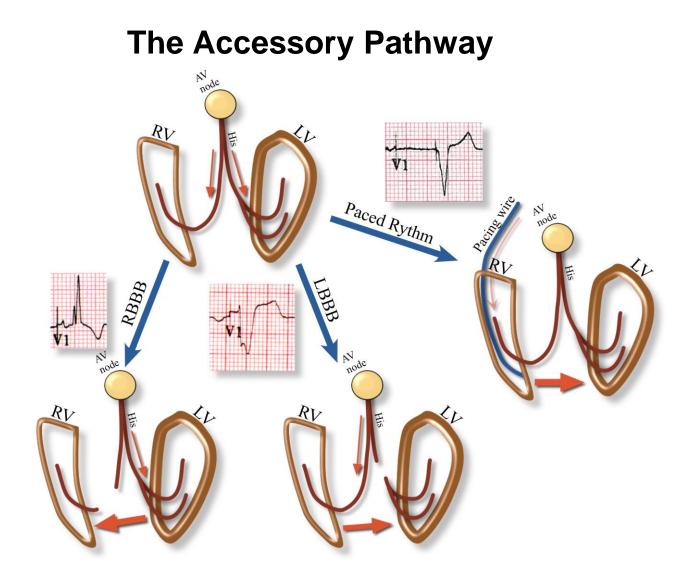
This requires urgent temporary pacing.

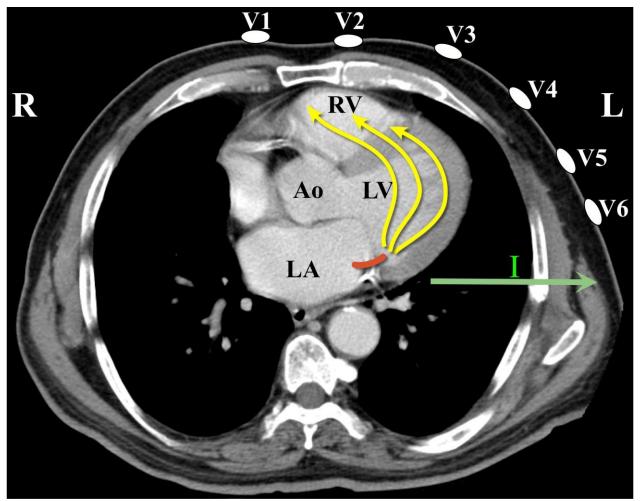
Accessory pathway in this context is *not* exclusive to WPW.

Activation starts at atrial level, then propagates through to activate both ventricles through the AV node.

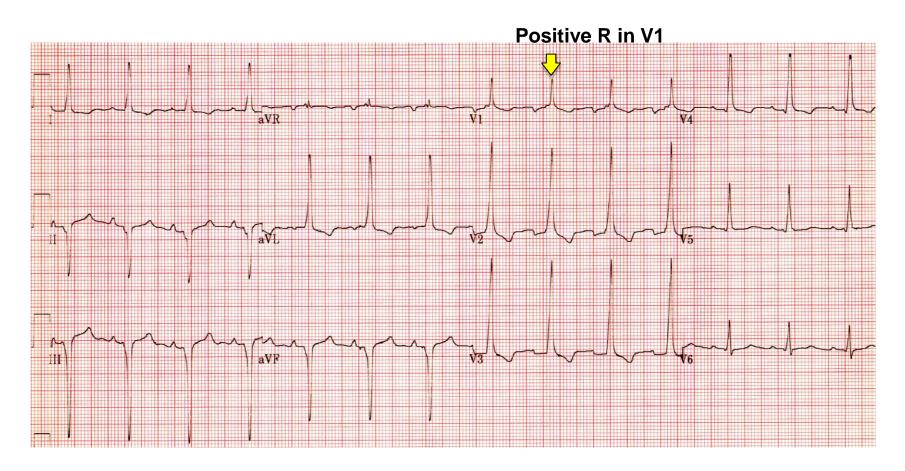
Bundle branch blocks Ventricular pacing Pre-excitation (e.g. WPW)

All share one mechanism... One chamber activates the other.

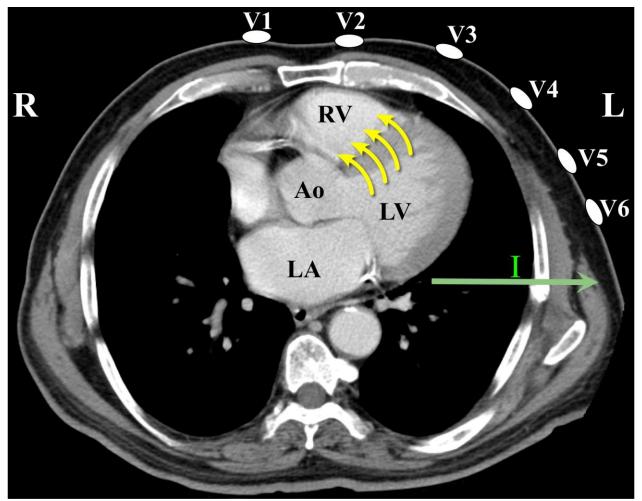




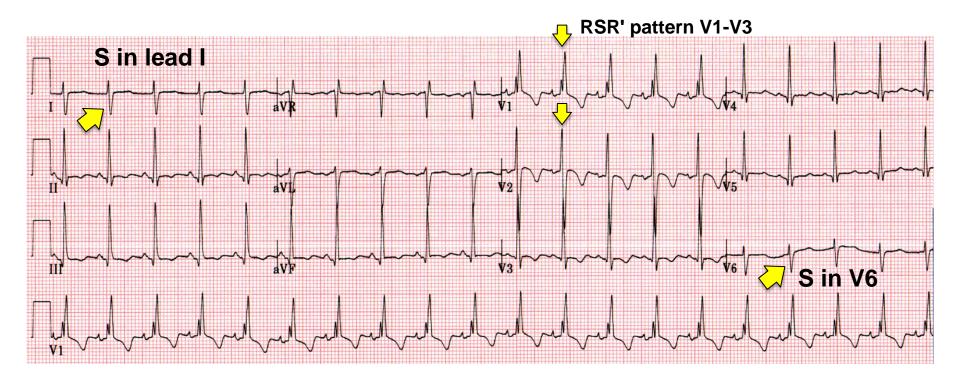
## Left Accessory Pathway (WPW)



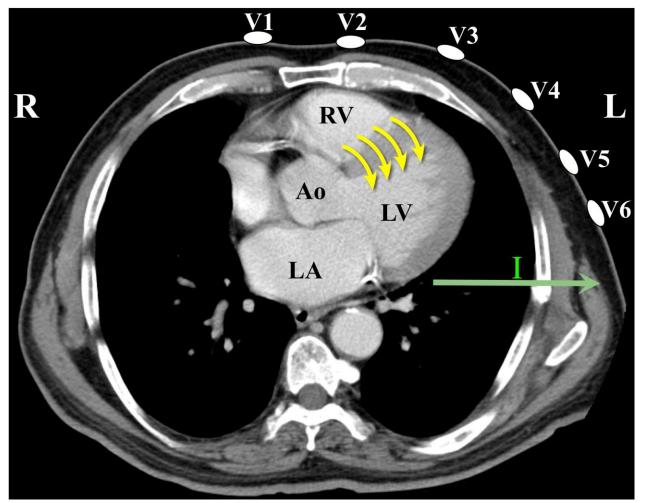
Left Accessory Pathway (WPW)



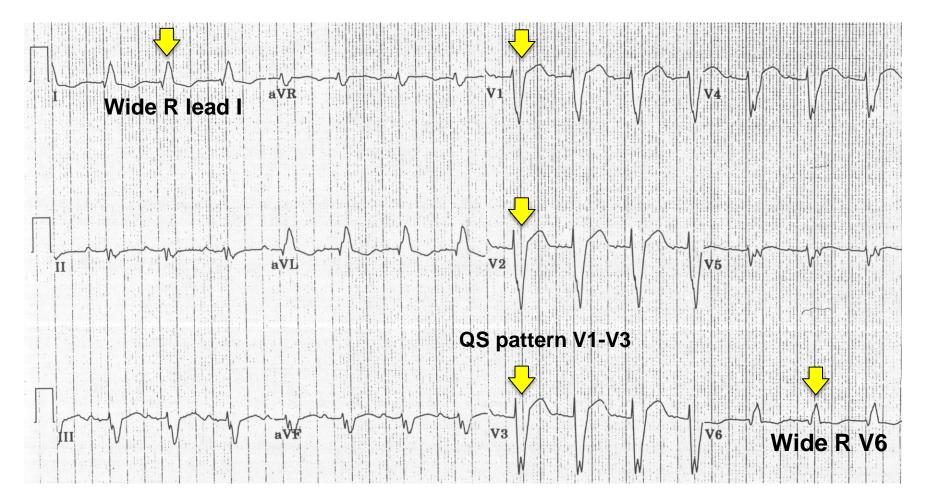
# **RBBB (LV activates RV)**



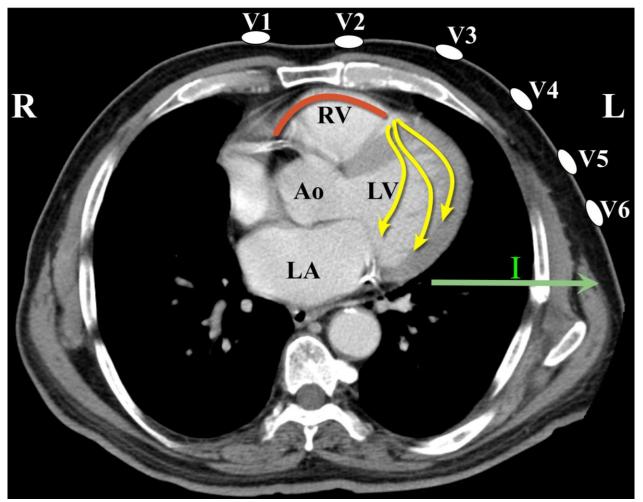
**RBBB (LV activates RV)** 



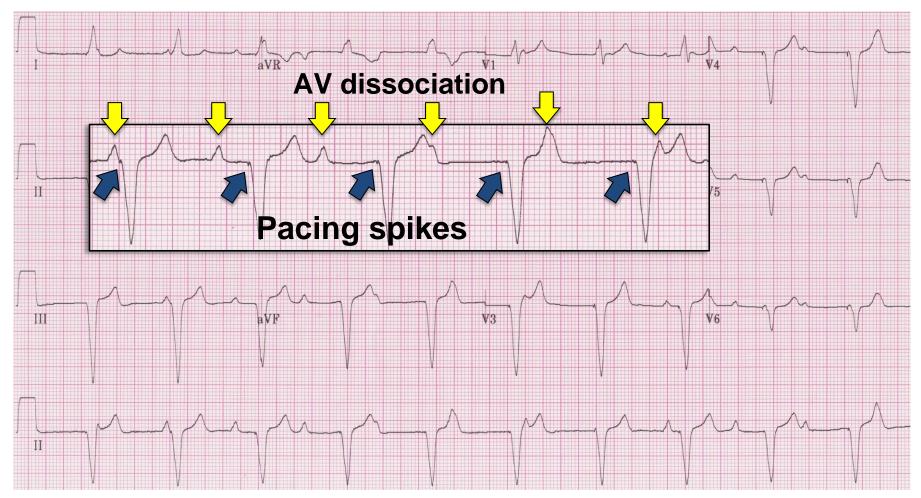
# LBBB (RV activates LV)



LBBB (RV activates LV)

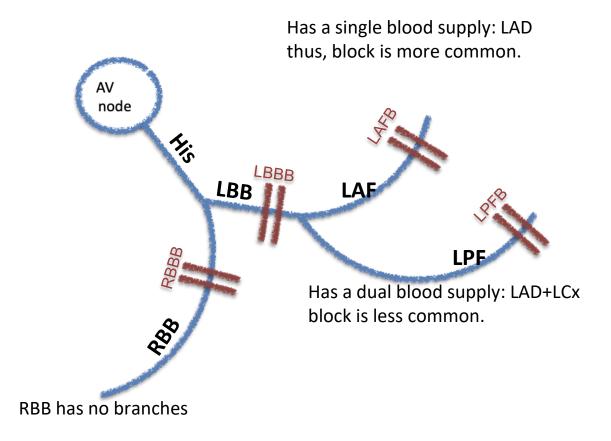


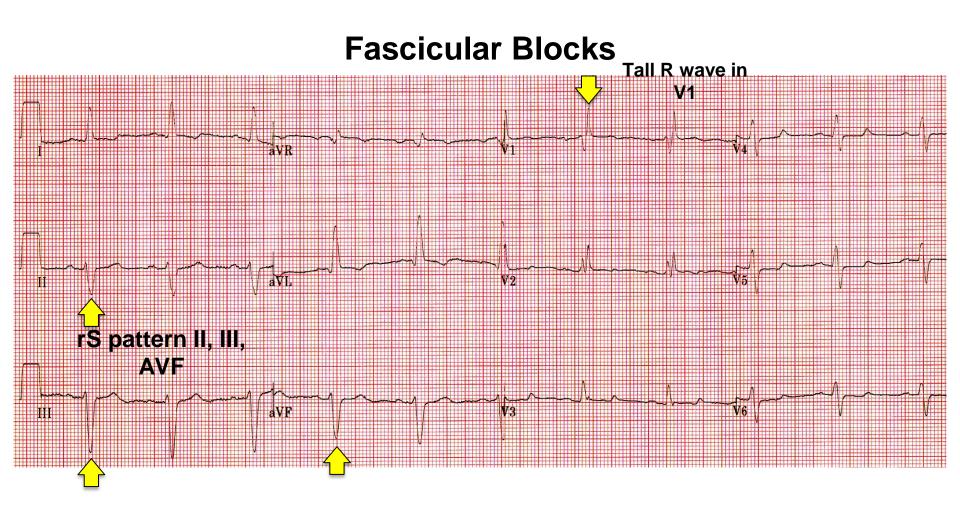
# **RV** pacing (**RV** activates **LV**)



**RV** pacing (**RV** activates **LV**)

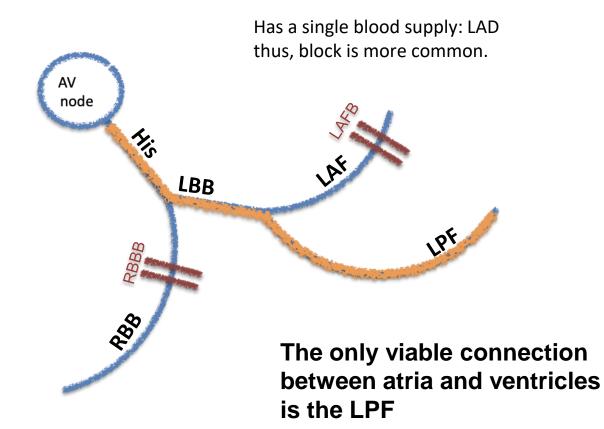
# **Fascicular Blocks**

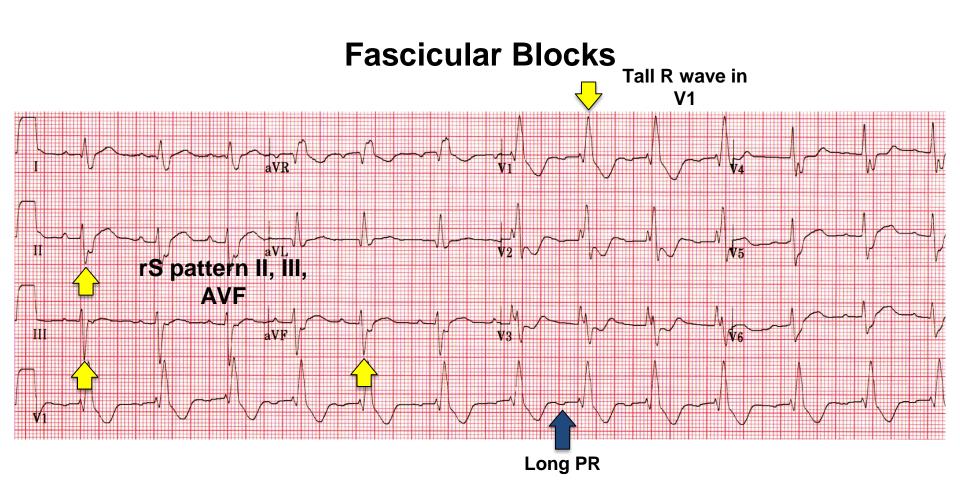




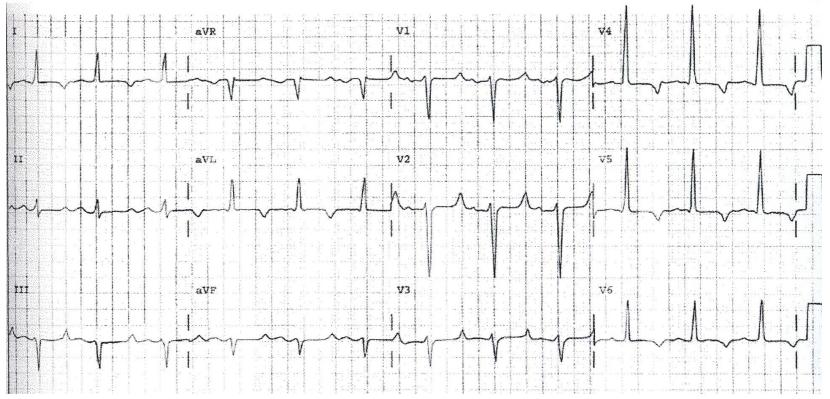
### **RBBB+LAFB = Bifascicular Block**

# **Bifascicular Block**



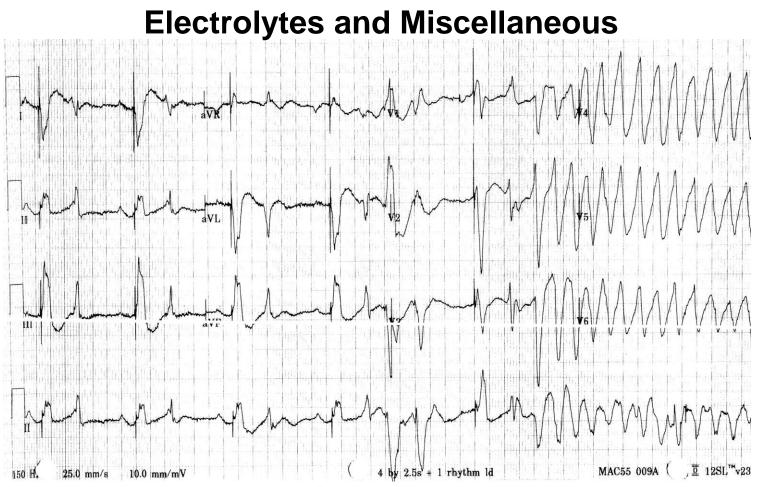


Long PR+RBBB+LAFB = Trifascicular Block If newly discovered with history of syncope, then permanent pacing is essential.



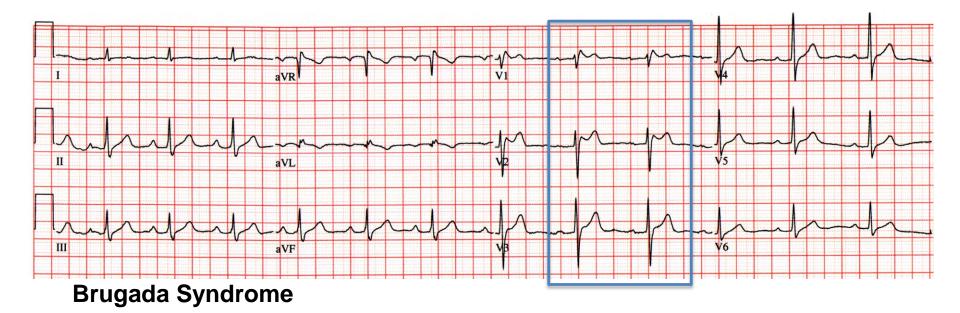
Prolonged QT interval.

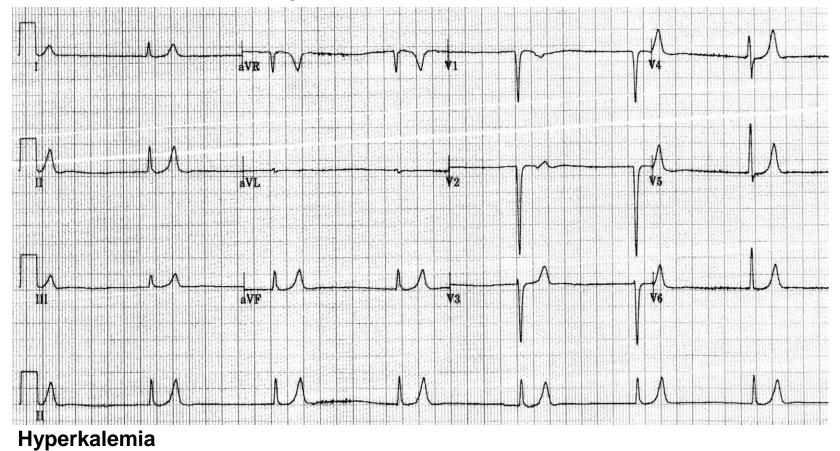
Know the 5 hypo sisters.

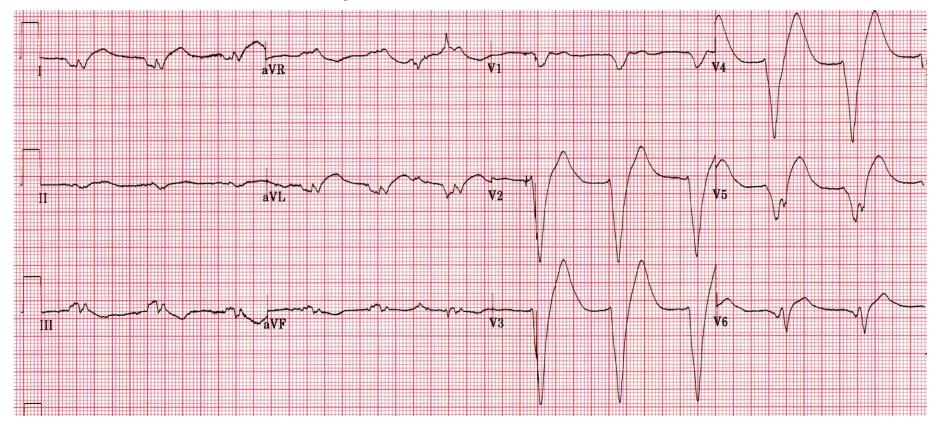


#### **Torsade de Pointes**

Polymorphic VT secondary to long QT interval







Accelerated idioventricular rhythm

# Well. That's it!

I wish you all the best. If you have any queries...

Please contact me.

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