# ECG Interpretation in Clinical Practice

## **Presentation Objectives**

- To recognize the normal rhythm of the heart -"Normal Sinus Rhythm."
- To recognize the 13 most common rhythm disturbances.
- To recognize an acute myocardial infarction on a 12-lead ECG.

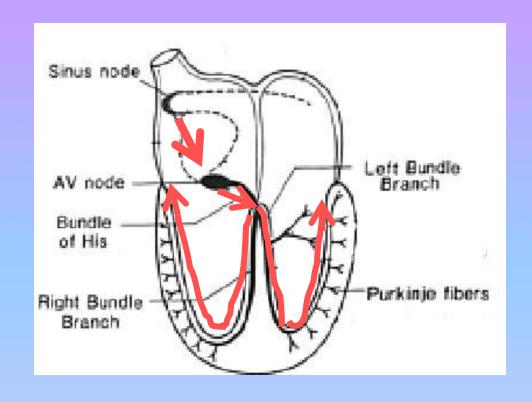
## The Learning Module

- ECG Basics
- How to Analyze a Rhythm
- Normal Sinus Rhythm
- Heart Arrhythmias
- Diagnosing a Myocardial Infarction
- Advanced 12-Lead Interpretation

# Normal Impulse Conduction

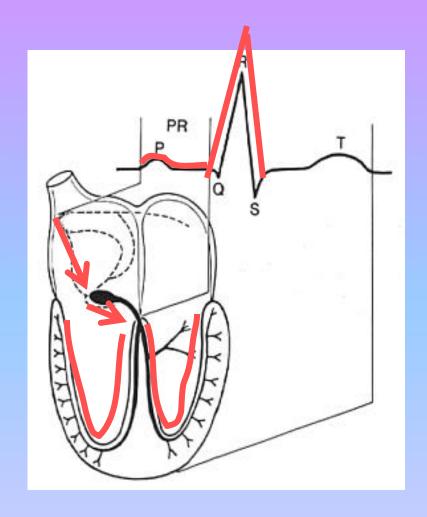
Sinoatrial node

AV node **Bundle of His Bundle Branches** Purkinje fibers

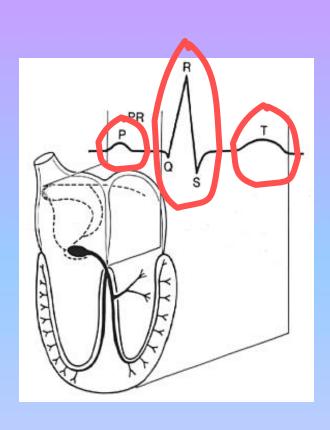


## Impulse Conduction & the ECG

Sinoatrial node AV node **Bundle of His Bundle Branches** Purkinje fibers



### The "PQRST"



- P wave Atrial depolarization
- QRS Ventricular depolarization
- T wave Ventricular repolarization

#### The PR Interval

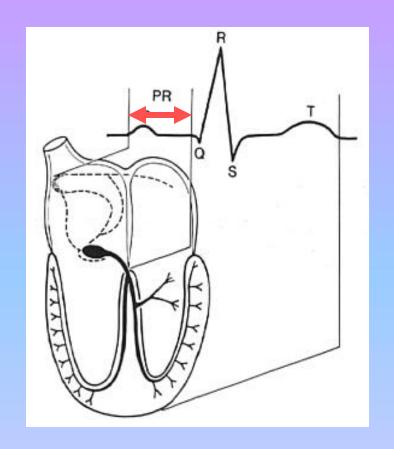
Atrial depolarization

+

delay in AV junction

(AV node/Bundle of His)

(delay allows time for the atria to contract before the ventricles contract)

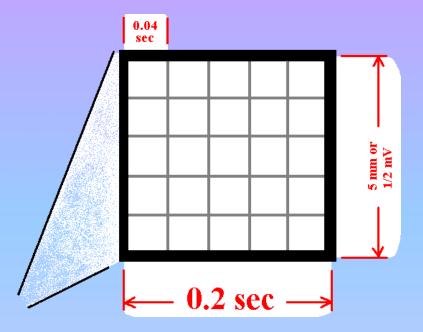


#### Pacemakers of the Heart

- SA Node Dominant pacemaker with an intrinsic rate of 60 - 100 beats/minute.
- AV Node Back-up pacemaker with an intrinsic rate of 40 - 60 beats/minute.
- Ventricular cells Back-up pacemaker with an intrinsic rate of 20 - 45 bpm.

## The ECG Paper

- Horizontally
  - One small box 0.04 s
  - One large box 0.20 s
- Vertically
  - One large box 0.5 mV





# The ECG Paper (cont)



This helps when calculating the heart rate.

NOTE: the following strips are not marked but all are 6 seconds long.

# Rhythm Analysis



- Step 1: Calculate rate.
- Step 2: Determine regularity.
- Step 3: Assess the P waves.
- Step 4: Determine PR interval.
- Step 5: Determine QRS duration.

#### Step 1: Calculate Rate



#### Option 1

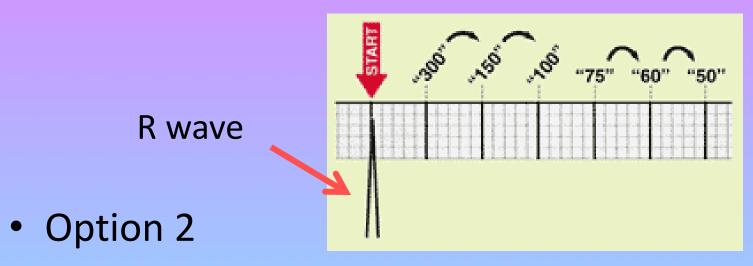
- Count the # of R waves in a 6 second rhythm strip,
   then multiply by 10.
- Reminder: all rhythm strips in this Module are 6 seconds in length.

#### Interpretation?

$$9 \times 10 = 90 \text{ bpm}$$

12

## Step 1: Calculate Rate



- Find a R wave that lands on a bold line.
- Count the # of large boxes to the next R wave. If the second R wave is 1 large box away the rate is 300, 2 boxes - 150, 3 boxes - 100, 4 boxes - 75, etc. (cont)

## Step 1: Calculate Rate

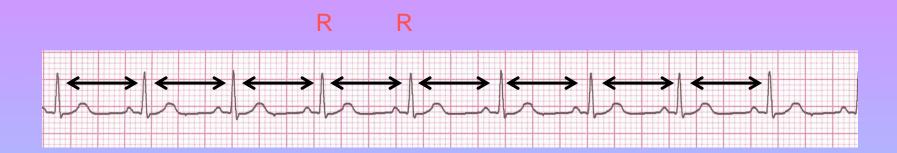


- Option 2 (cont)
  - Memorize the sequence:

Interpretation?

Approx. 1 box less than 100 = 95 bpm

## Step 2: Determine regularity



- Look at the R-R distances (using a caliper or markings on a pen or paper).
- Regular (are they equidistant apart)? Occasionally irregular? Regularly irregular? Irregularly irregular?

Interpretation?

Regular

#### Step 3: Assess the P waves



- Are there P waves?
- Do the P waves all look alike?
- Do the P waves occur at a regular rate?
- Is there one P wave before each QRS?

Interpretation?

Normal P waves with 1 P wave for every QRS

# Step 4: Determine PR interval



Normal: 0.12 - 0.20 seconds.

(3 - 5 boxes)

Interpretation?

0.12 seconds

# Step 5: QRS duration



• Normal: 0.04 - 0.12 seconds.

(1 - 3 boxes)

Interpretation?

0.08 seconds

## Rhythm Summary



Rate 90-95 bpm

Regularity regular

P waves normal

• PR interval 0.12 s

• QRS duration 0.08 s

Interpretation? Normal Sinus Rhythm

# Normal Sinus Rhythm (NSR)



- Etiology: the electrical impulse is formed in the SA node and conducted normally.
- This is the normal rhythm of the heart; other rhythms that do not conduct via the typical pathway are called arrhythmias.

#### **NSR Parameters**



Rate 60 - 100 bpm

Regularity regular

P waves normal

• PR interval 0.12 - 0.20 s

QRS duration
 0.04 - 0.12 s

Any deviation from above is sinus tachycardia, sinus bradycardia or an arrhythmia

# **Arrhythmia Formation**

Arrhythmias can arise from problems in the:

- Sinus node
- Atrial cells
- AV junction
- Ventricular cells

#### SA Node Problems

#### The SA Node can:

- fire too slow
- fire too fast

- Sinus Bradycardia
- Sinus Tachycardia

Sinus Tachycardia may be an appropriate response to stress.

#### **Atrial Cell Problems**

#### Atrial cells can:

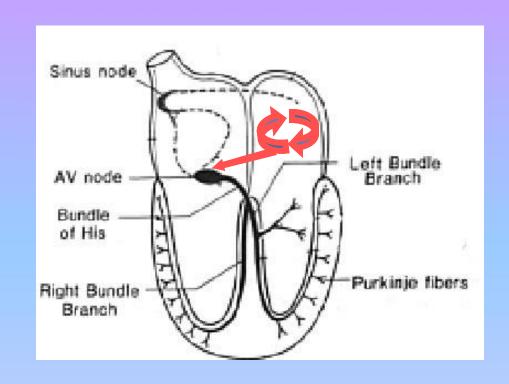
 fire occasionally from a focus

Premature Atrial Contractions (PACs)

• fire continuously due *Atrial Flutter* to a looping reentrant circuit

#### **Teaching Moment**

 A re-entrant pathway occurs when an impulse loops and results in selfperpetuating impulse formation.



#### **Atrial Cell Problems**

Atrial cells can also:

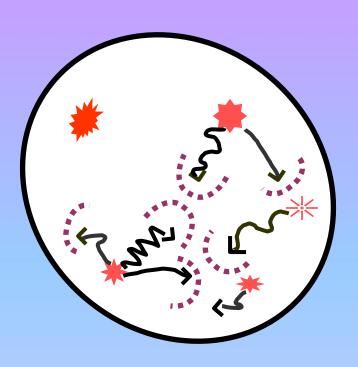
 fire continuously from multiple foci or fire continuously due to multiple micro re-entrant "wavelets" Atrial Fibrillation

Atrial Fibrillation

## **Teaching Moment**

Multiple micro reentrant "wavelets" refers to wandering small areas of activation which generate fine chaotic impulses. Colliding wavelets can, in turn, generate new foci of activation.

Atrial tissue



#### **AV Junctional Problems**

#### The AV junction can:

- fire continuously due to a looping reentrant circuit
- block impulses coming from the SA Node

Paroxysmal Supraventricular Tachycardia

**AV Junctional Blocks** 

#### Ventricular Cell Problems

#### Ventricular cells can:

- fire occasionally from 1 or more foci
- fire continuously from multiple foci
- fire continuously due to a looping reentrant circuit

Premature Ventricular Contractions (PVCs)

**Ventricular Fibrillation** 

Ventricular Tachycardia

# Arrhythmias

- Sinus Rhythms
- Premature Beats
- Supraventricular Arrhythmias
- Ventricular Arrhythmias
- AV Junctional Blocks

# Sinus Rhythms

- Sinus Bradycardia
- Sinus Tachycardia

## Rhythm #1



Rate?30 bpm

Regularity? regular

P waves? normal

PR interval? 0.12 s

QRS duration?
 0.10 s

Interpretation? Sinus Bradycardia

# Sinus Bradycardia



- Deviation from NSR
  - Rate

< 60 bpm

# Sinus Bradycardia



 Etiology: SA node is depolarizing slower than normal, impulse is conducted normally (i.e. normal PR and QRS interval).

# Rhythm #2



Rate?130 bpm

Regularity? regular

P waves? normal

PR interval? 0.16 s

QRS duration?
 0.08 s

Interpretation? Sinus Tachycardia

# Sinus Tachycardia



- Deviation from NSR
  - Rate

> 100 bpm

# Sinus Tachycardia



- Etiology: SA node is depolarizing faster than normal, impulse is conducted normally.
- Remember: sinus tachycardia is a response to physical or psychological stress, not a primary arrhythmia.

#### **Premature Beats**

- Premature Atrial Contractions (PACs)
- Premature Ventricular Contractions (PVCs)



- Rate?
- Regularity?
- P waves?
- PR interval?
- QRS duration?

70 bpm

occasionally irreg.

2/7 different contour

0.14 s (except 2/7)

0.08 s

Interpretation? NSR with Premature Atrial Contractions

### **Premature Atrial Contractions**



#### Deviation from NSR

These ectopic beats originate in the atria (but not in the SA node), therefore the contour of the P wave, the PR interval, and the timing are different than a normally generated pulse from the SA node.

### **Premature Atrial Contractions**



 Etiology: Excitation of an atrial cell forms an impulse that is then conducted normally through the AV node and ventricles.

## **Teaching Moment**

 When an impulse originates anywhere in the atria (SA node, atrial cells, AV node, Bundle of His) and then is conducted normally through the ventricles, the QRS will be narrow (0.04 - 0.12 s).





- Rate?
- Regularity?
- P waves?
- PR interval?
- QRS duration?

60 bpm

occasionally irreg.

none for 7th QRS

0.14 s

0.08 s (7th wide)

Interpretation? Sinus Rhythm with 1 PVC

### **PVCs**



#### Deviation from NSR

- Ectopic beats originate in the ventricles resulting in wide and bizarre QRS complexes.
- When there are more than 1 premature beats and look alike, they are called "uniform". When they look different, they are called "multiform".

### **PVCs**



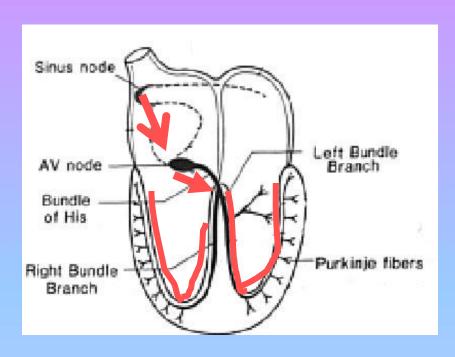
 Etiology: One or more ventricular cells are depolarizing and the impulses are abnormally conducting through the ventricles.

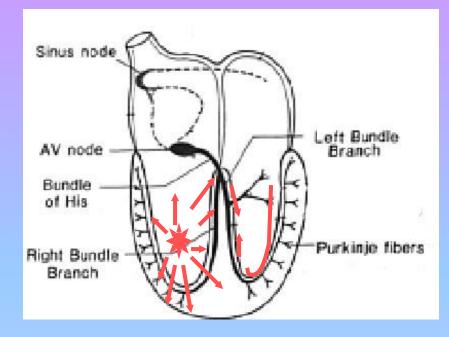
## Teaching Moment

 When an impulse originates in a ventricle, conduction through the ventricles will be inefficient and the QRS will be wide and bizarre.



### Ventricular Conduction





### **Normal**

Signal moves rapidly through the ventricles

### **Abnormal**

Signal moves slowly through the ventricles

## Arrhythmias

- Sinus Rhythms
- Premature Beats
- Supraventricular Arrhythmias
- Ventricular Arrhythmias
- AV Junctional Blocks

# Supraventricular Arrhythmias

- Atrial Fibrillation
- Atrial Flutter
- Paroxysmal Supraventricular Tachycardia



Rate?100 bpm

Regularity? irregularly irregular

P waves? none

PR interval? none

• QRS duration? 0.06 s

Interpretation? Atrial Fibrillation

### **Atrial Fibrillation**



#### Deviation from NSR

- No organized atrial depolarization, so no normal P waves (impulses are not originating from the sinus node).
- Atrial activity is chaotic (resulting in an irregularly irregular rate).
- Common, affects 2-4%, up to 5-10% if > 80 years old

### **Atrial Fibrillation**



• Etiology: Recent theories suggest that it is due to multiple re-entrant wavelets conducted between the R & L atria. Either way, impulses are formed in a totally unpredictable fashion. The AV node allows some of the impulses to pass through at variable intervals (so rhythm is irregularly irregular).



Rate?

70 bpm

Regularity?

regular

P waves?

flutter waves

PR interval?

none

QRS duration?

0.06 s

Interpretation? Atrial Flutter

### **Atrial Flutter**



#### Deviation from NSR

- No P waves. Instead flutter waves (note "sawtooth" pattern) are formed at a rate of 250 350 bpm.
- Only some impulses conduct through the AV node (usually every other impulse).

### **Atrial Flutter**



 Etiology: Reentrant pathway in the right atrium with every 2nd, 3rd or 4th impulse generating a QRS (others are blocked in the AV node as the node repolarizes).



- Rate?
- Regularity?
- P waves?
- PR interval?
- QRS duration?

74 → 148 bpm

Regular -> regular

Normal → none

 $0.16 s \rightarrow none$ 

0.08 s

Interpretation?

Paroxysmal Supraventricular Tachycardia (PSVT) 56

### **PSVT**



#### Deviation from NSR

 The heart rate suddenly speeds up, often triggered by a PAC (not seen here) and the P waves are lost.

### **PSVT**



- Etiology: There are several types of PSVT but all originate above the ventricles (therefore the QRS is narrow).
- Most common: abnormal conduction in the AV node (reentrant circuit looping in the AV node).

### Ventricular Fibrillation



 Etiology: The ventricular cells are excitable and depolarizing randomly.

 Rapid drop in cardiac output and death occurs if not quickly reversed

### **AV Nodal Blocks**

- 1st Degree AV Block
- 2nd Degree AV Block, Type I
- 2nd Degree AV Block, Type II
- 3rd Degree AV Block



Rate?60 bpm

Regularity? regular

P waves? normal

PR interval? 0.36 s

QRS duration?
 0.08 s

Interpretation? 1st Degree AV Block

# 1st Degree AV Block



- Deviation from NSR
  - -PR Interval > 0.20 s

# 1st Degree AV Block



• Etiology: Prolonged conduction delay in the AV node or Bundle of His.



- Rate?
- Regularity?
- P waves?
- PR interval?
- QRS duration?

50 bpm

regularly irregular

nl, but 4th no QRS

lengthens

0.08 s

Interpretation? 2nd Degree AV Block, Type I

# 2nd Degree AV Block, Type I



#### Deviation from NSR

 PR interval progressively lengthens, then the impulse is completely blocked (P wave not followed by QRS).

# 2nd Degree AV Block, Type I



 Etiology: Each successive atrial impulse encounters a longer and longer delay in the AV node until one impulse (usually the 3rd or 4th) fails to make it through the AV node.



- Rate?
- Regularity?
- P waves?
- PR interval?
- QRS duration?

40 bpm

regular

nl, 2 of 3 no QRS

0.14 s

0.08 s

Interpretation? 2nd Degree AV Block, Type II

# 2nd Degree AV Block, Type II



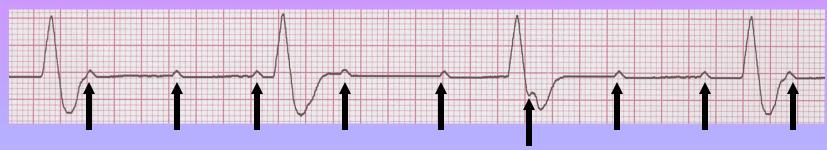
#### Deviation from NSR

Occasional P waves are completely blocked
 (P wave not followed by QRS).

# 2nd Degree AV Block, Type II



 Etiology: Conduction is all or nothing (no prolongation of PR interval); typically block occurs in the Bundle of His.



- Rate?
- Regularity?
- P waves?
- PR interval?
- QRS duration?

40 bpm

regular

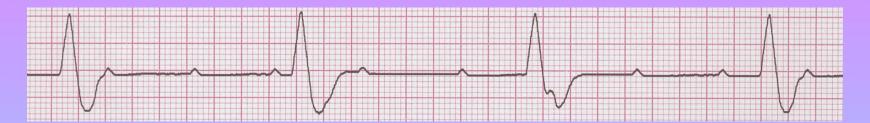
no relation to QRS

none

wide (> 0.12 s)

Interpretation? 3rd Degree AV Block

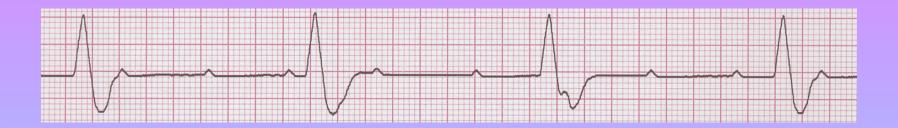
# 3rd Degree AV Block



#### Deviation from NSR

 The P waves are completely blocked in the AV junction; QRS complexes originate independently from below the junction.

## 3rd Degree AV Block



• Etiology: There is complete block of conduction in the AV junction, so the atria and ventricles form impulses independently of each other. Without impulses from the atria, the ventricles own intrinsic pacemaker kicks in at around 30 - 45 beats/minute.

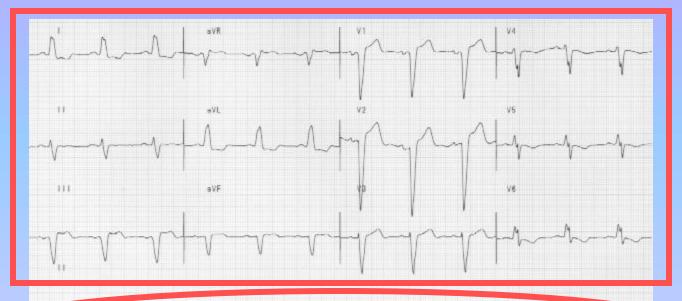
## Remember

 When an impulse originates in a ventricle, conduction through the ventricles will be inefficient and the QRS will be wide and bizarre.



# Diagnosing a MI

To diagnose a myocardial infarction you need to go beyond looking at a rhythm strip and obtain a 12-Lead ECG.



12-Lead ECG

Rhythm Strip

D 2/8/2021 10:52:5<del>7 PM 25nn/s 10mn/nV 4 989 011</del>

## The 12-Lead ECG

- The 12-Lead ECG sees the heart from 12 different views.
- Therefore, the 12-Lead ECG helps you see what is happening in different portions of the heart.
- The rhythm strip is only 1 of these 12 views.

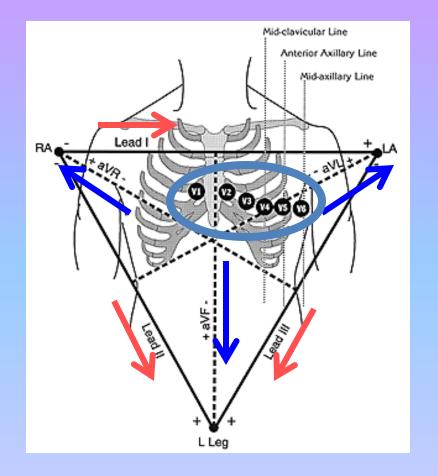
## The 12-Leads

### The 12-leads include:

-3 Limb leads (I, II, III)

-3 Augmented leads (aVR, aVL, aVF)

-6 Precordial leads
(V<sub>1</sub>- V<sub>6</sub>)

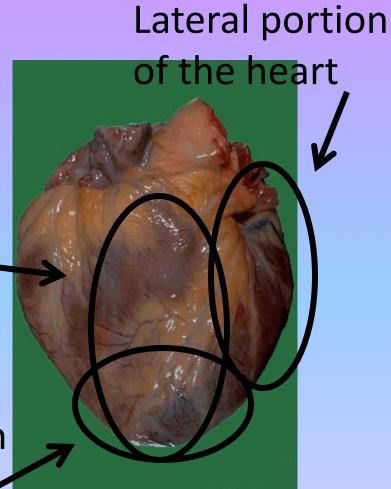


## Views of the Heart

Some leads get a good view of the:

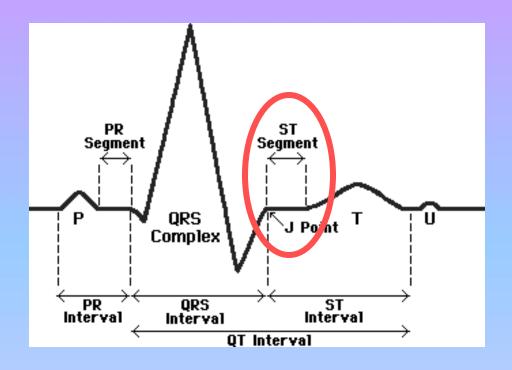
Anterior portion of the heart —

Inferior portion of the heart



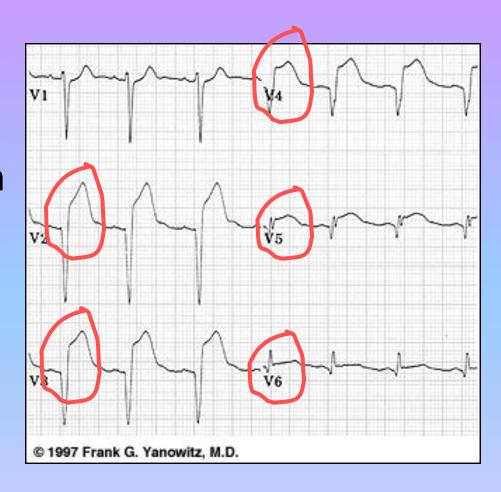
## ST Elevation

One way to diagnose an acute MI is to look for elevation of the ST segment.



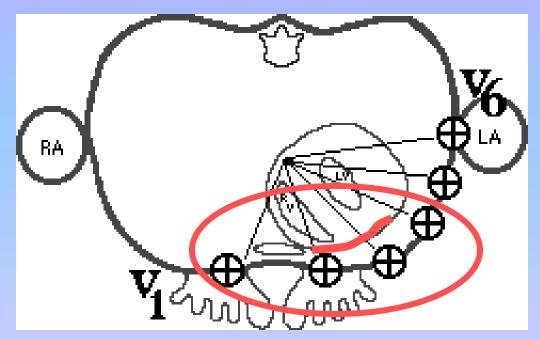
# ST Elevation (cont)

Elevation of the ST segment (greater than 1 small box) in 2 leads is consistent with a myocardial infarction.



## Anterior View of the Heart

The anterior portion of the heart is best viewed using leads  $V_1$ -  $V_4$ .

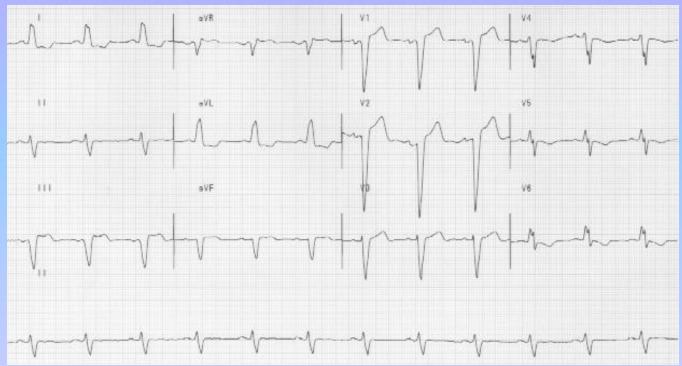


# **Anterior Myocardial Infarction**

If you see changes in leads  $V_1 - V_4$  that are consistent with a myocardial infarction, you can conclude that it is an anterior wall myocardial infarction.

# Putting it all Together

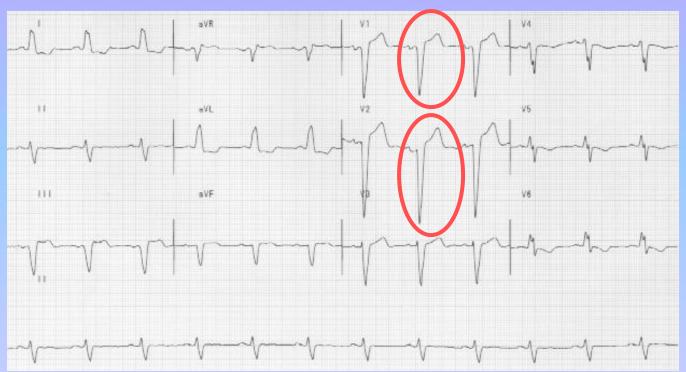
Do you think this person is having a myocardial infarction. If so, where?



88

# Interpretation

Yes, this person is having an acute anterior wall myocardial infarction.

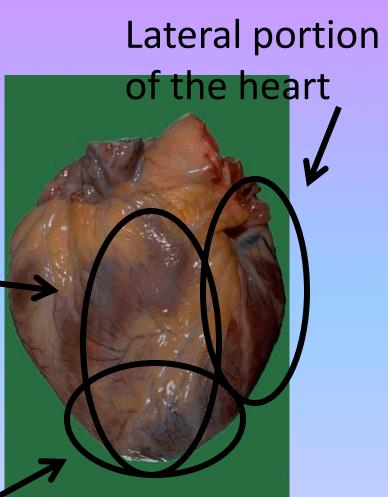


Now that you know where to look for an anterior wall myocardial infarction let's look at how you would determine if the MI involves the lateral wall or the inferior wall of the heart.

First, take a look again at this picture of the heart.

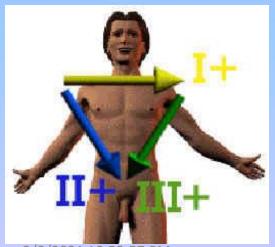
Anterior portion of the heart —

Inferior portion of the heart

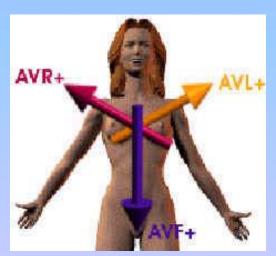


Second, remember that the 12-leads of the ECG look at different portions of the heart. The limb and augmented leads "see" electrical activity moving inferiorly (II, III and aVF), to the left (I, aVL) and to the right (aVR). Whereas, the precordial leads "see" electrical activity in the posterior to anterior direction.

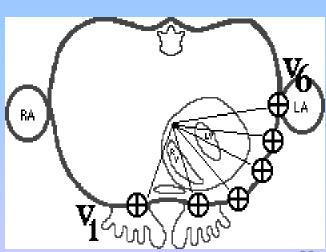
**Limb Leads** 



**Augmented Leads** 



**Precordial Leads** 

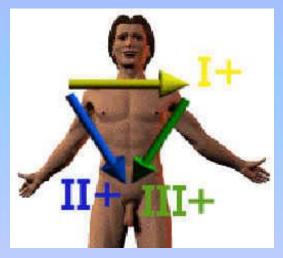


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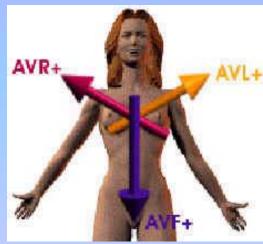
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Now, using these 3 diagrams let's figure where to look for a lateral wall and inferior wall MI.

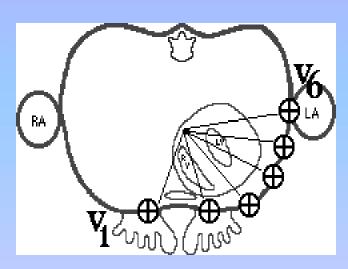
**Limb Leads** 



**Augmented Leads** 



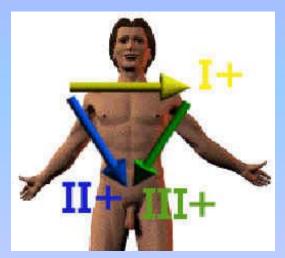
**Precordial Leads** 



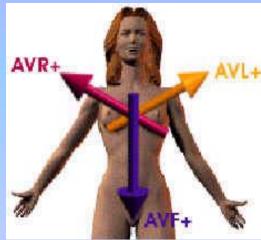
### **Anterior MI**

Remember the anterior portion of the heart is best viewed using leads  $V_1$ -  $V_4$ .

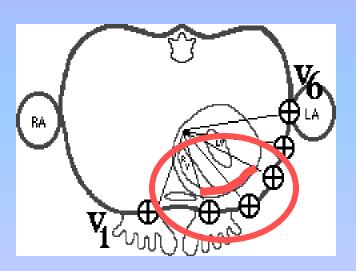
**Limb Leads** 



**Augmented Leads** 



**Precordial Leads** 

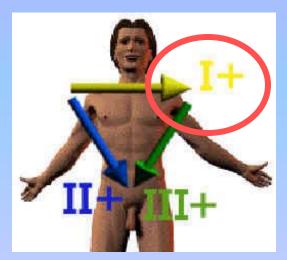


## Lateral MI

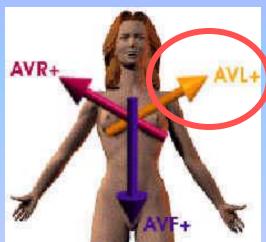
So what leads do you think the lateral portion of the heart is best viewed?

Leads I, aVL, and V<sub>5</sub>- V<sub>6</sub>

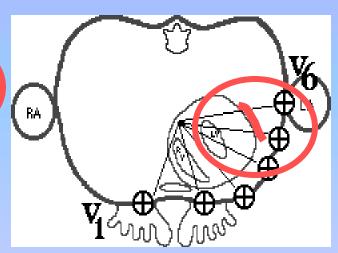
**Limb Leads** 



**Augmented Leads** 



**Precordial Leads** 

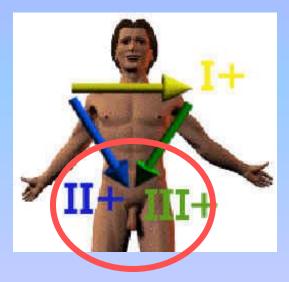


## Inferior MI

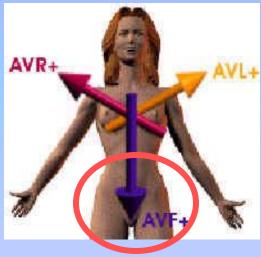
Now how about the inferior portion of the heart?

Leads II, III and aVF

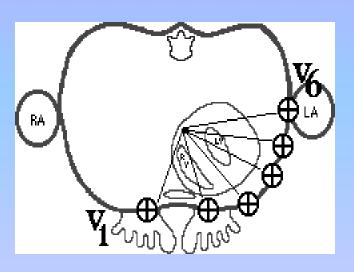
**Limb Leads** 



**Augmented Leads** 

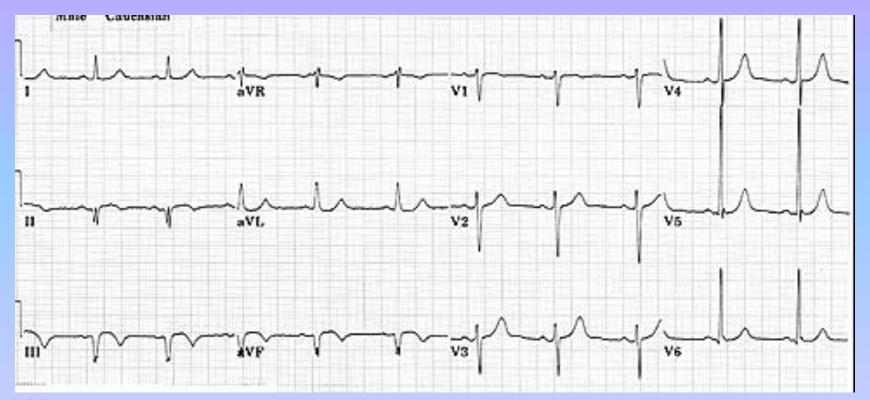


**Precordial Leads** 



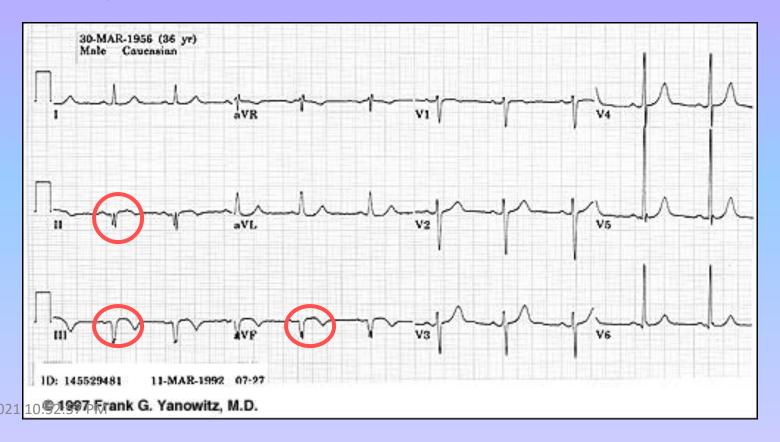
# Putting it all Together

Now, where do you think this person is having a myocardial infarction?



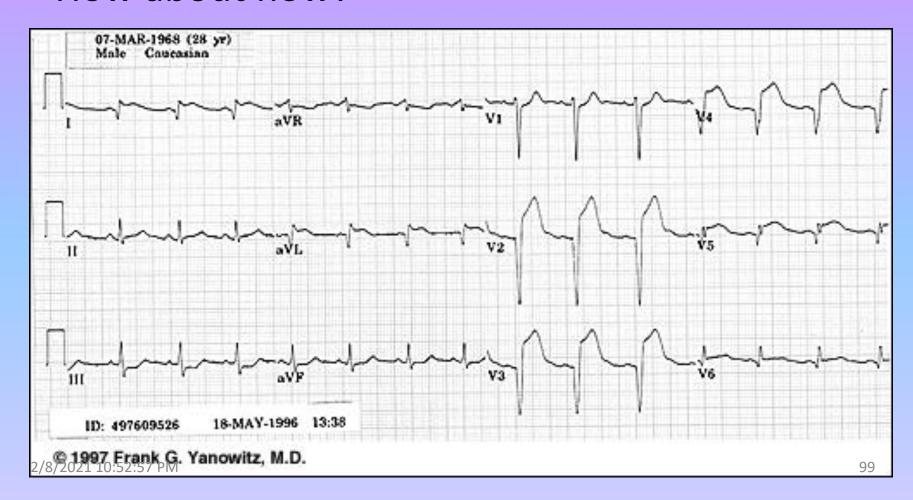
## Inferior Wall MI

This is an inferior MI. Note the ST elevation in leads II, III and aVF.



# Putting it all Together

### How about now?



### Anterolateral MI

This person's MI involves both the anterior wall ( $V_2$ - $V_4$ ) and the lateral wall ( $V_5$ - $V_6$ , I, and aVL)!



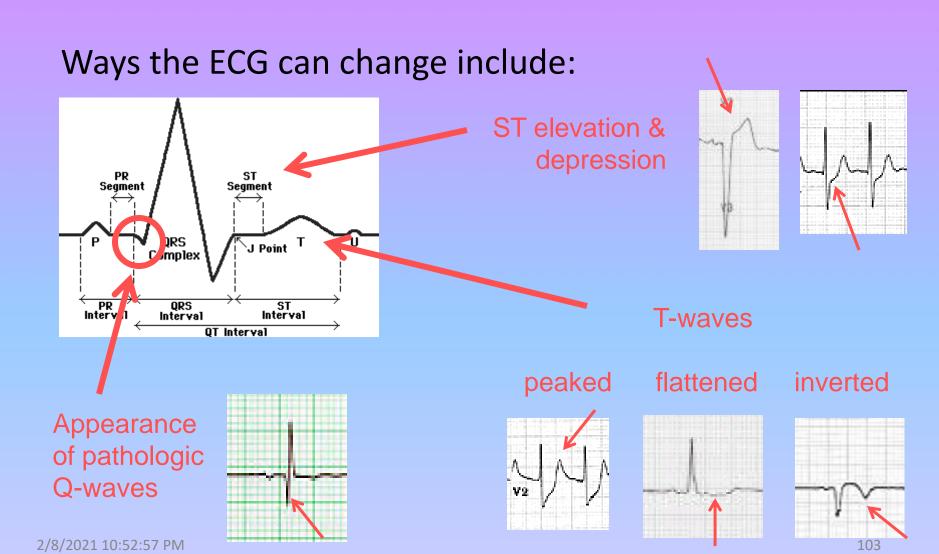
# ST Elevation and non-ST Elevation MIs

### ST Elevation and non-ST Elevation MIs

 When myocardial blood supply is abruptly reduced or cut off to a region of the heart, a sequence of injurious events occur beginning with ischemia (inadequate tissue perfusion), followed by necrosis (infarction), and eventual fibrosis (scarring) if the blood supply isn't restored in an appropriate period of time.

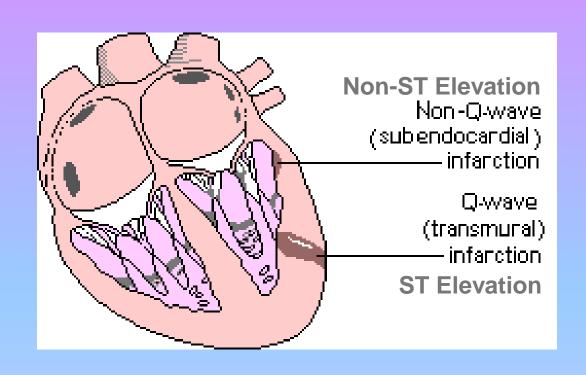
The ECG changes over time with each of these events...

# **ECG Changes**



## ECG Changes & the Evolving MI

There are two distinct patterns of ECG change depending if the infarction is:



- -ST Elevation (Transmural or Q-wave), or
- -Non-ST Elevation (Subendocardial or non-Q-wave)

## ST Elevation Infarction

The ECG changes seen with a ST elevation infarction are:

Before injury Normal ECG

Ischemia

ST depression, peaked T-waves, then Twave inversion

Infarction

ST elevation & appearance of **Q-waves** 

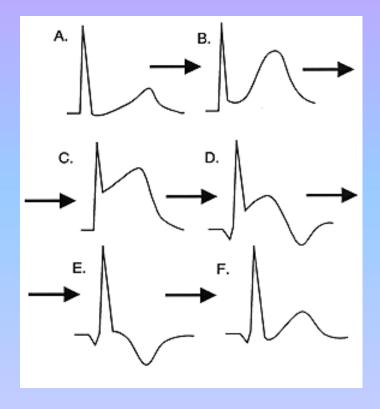
**Fibrosis** 

ST segments and T-waves return to normal, but Q-waves persist

## ST Elevation Infarction

### Here's a diagram depicting an evolving infarction:

- A. Normal ECG prior to MI
- B. Ischemia from coronary artery occlusion results in ST depression (not shown) and peaked T-waves
- C. Infarction from ongoing ischemia results in marked ST elevation
- D/E. Ongoing infarction with appearance of pathologic Q-waves and T-wave inversion
- F. Fibrosis (months later) with persistent Q- waves, but normal ST segment and T- waves



## ST Elevation Infarction

### Here's an ECG of an inferior MI:

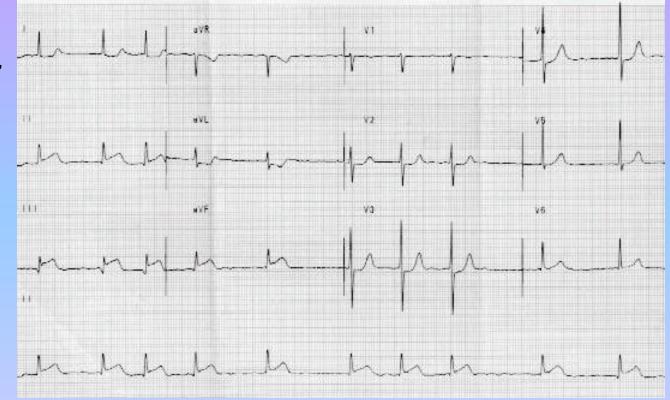
Look at the inferior leads (II, III, aVF).

#### Question:

What ECG changes do you see?

ST elevation and Q-waves Extra credit:

What is the



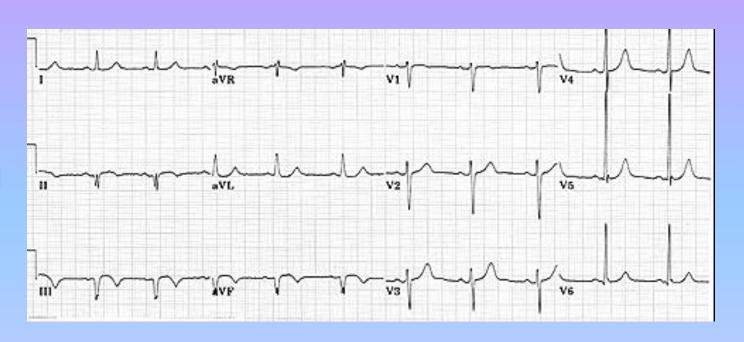
rhythm? Atrial fibrillation (irregularly irregular with narrow QRS)!

## Non-ST Elevation Infarction

### Here's an ECG of an inferior MI later in time:

Now what do you see in the inferior leads?

ST elevation, Q-waves and T-wave inversion



## Non-ST Elevation Infarction

The ECG changes seen with a non-ST elevation infarction are:

Before injury Normal ECG

Ischemia

ST depression & T-wave inversion

Infarction

ST depression & T-wave inversion

**Fibrosis** 

ST returns to baseline, but T-wave inversion persists

## Non-ST Elevation Infarction

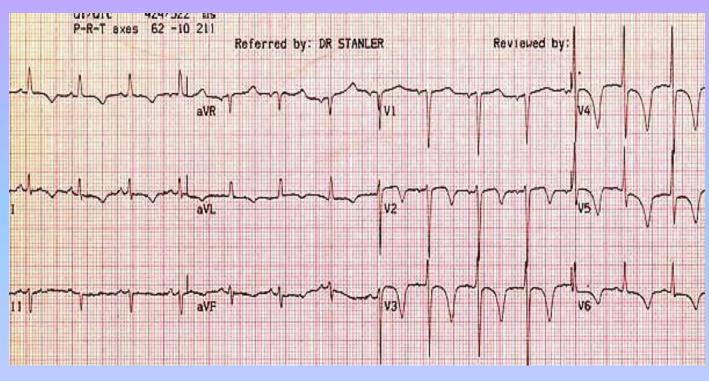
### Here's an ECG of an evolving non-ST elevation MI:

Note the ST depression and T-wave inversion in leads  $V_2$ - $V_6$ .

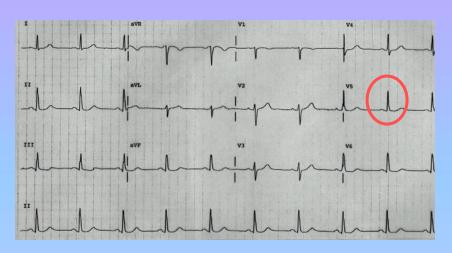
### Question:

What area of the heart is infarcting?

**Anterolateral** 



Compare these two 12-lead ECGs. What stands out as different with the second one?



**Normal** 



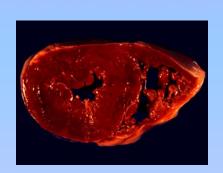
Left Ventricular Hypertrophy

Answer: The QRS complexes are very tall (increased voltage)

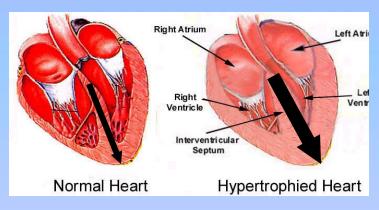
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Why is left ventricular hypertrophy characterized by tall QRS complexes?

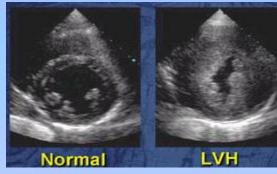
As the heart muscle wall thickens there is an increase in electrical forces moving through the myocardium resulting in increased QRS voltage.



LVH

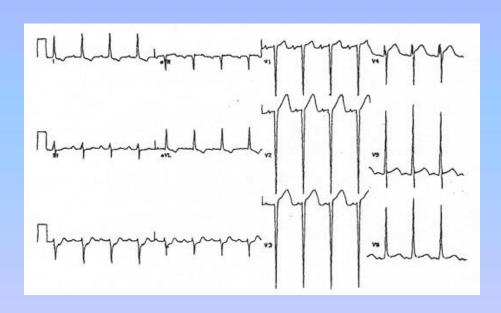


Increased QRS voltage

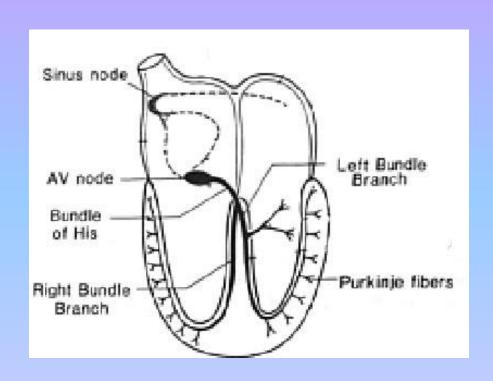


**ECHOcardiogram** 

- Criteria exists to diagnose LVH using a 12-lead ECG.
  - For example:
    - The R wave in V5 or V6 plus the S wave in V1 or V2 exceeds 35 mm.
- However, for now, all you need to know is that the QRS voltage increases with LVH.



Turning our attention to bundle branch blocks...



Remember normal impulse conduction is

SA node →

AV node →

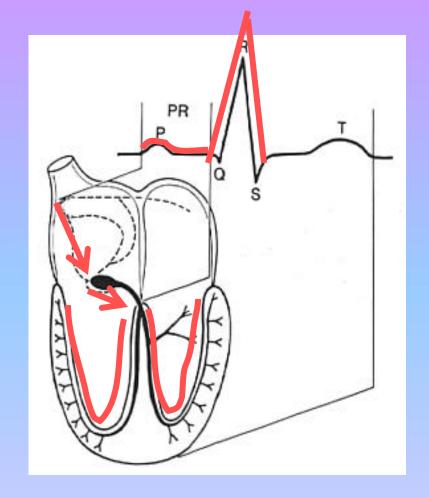
Bundle of His →

Bundle Branches →

Purkinje fibers

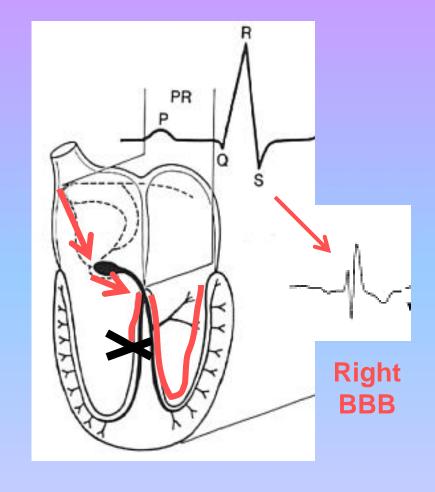
# Normal Impulse Conduction

Sinoatrial node AV node **Bundle of His Bundle Branches** Purkinje fibers



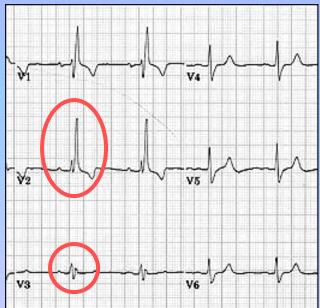
So, depolarization of the Bundle Branches and Purkinje fibers are seen as the QRS complex on the ECG.

Therefore, a conduction block of the Bundle Branches would be reflected as a change in the QRS complex.



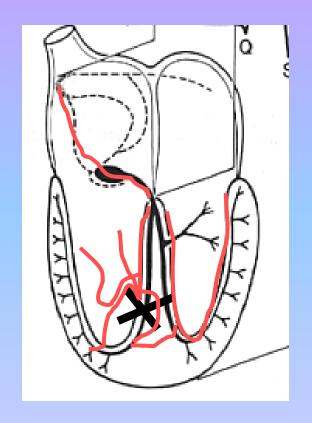
With Bundle Branch Blocks you will see two changes on the ECG.

- 1. QRS complex widens (> 0.12 sec).
- 2. QRS morphology changes (varies depending on ECG lead, and if it is a right vs. left bundle branch block).



#### Why does the QRS complex widen?

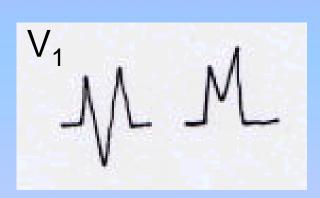
When the conduction pathway is blocked it will take longer for the electrical signal to pass throughout the ventricles.



# Right Bundle Branch Blocks

What QRS morphology is characteristic?

For RBBB the wide QRS complex assumes a unique, virtually diagnostic shape in those leads overlying the right ventricle ( $V_1$  and  $V_2$ ).



"Rabbit "Ears"



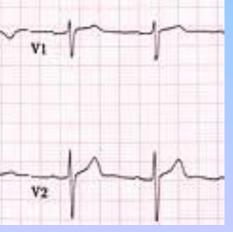
### Left Bundle Branch Blocks

What QRS morphology is characteristic?

For LBBB the wide QRS complex assumes a characteristic change in shape in those leads opposite the left ventricle (right ventricular

leads -  $V_1$  and  $V_2$ ).

Normal



Broad, deep S waves

# Reading 12-Lead ECGs

- The 12-Lead ECG contains information that will assist you in making diagnostic and treatment decisions in your clinical practice. In previous modules you learned how to read and interpret parts of the ECG. Now, we will bring all that you have learned together so that you can systematically read and interpret a 12-lead ECG.
- The information will be divided into two modules, VII a and VII
   b.

# Reading 12-Lead ECGs

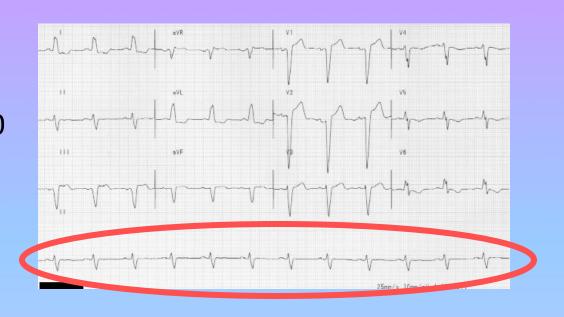
The best way to read 12-lead ECGs is to develop a step-by-step approach (just as we did for analyzing a rhythm strip). In these modules we present a 6-step approach:

- 1. Calculate RATE
- 2. Determine RHYTHM
- 3. Determine QRS AXIS
- 4. Calculate INTERVALS
- 5. Assess for HYPERTROPHY
- 6. Look for evidence of INFARCTION

• In Module II you learned how to calculate the rate. If you need a refresher return to that module.

• There is one new thing to keep in mind when determining the rate in a 12-lead ECG...

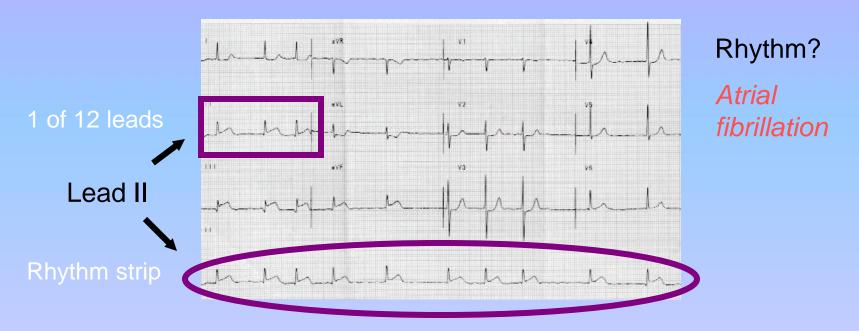
If you use the rhythm strip portion of the 12lead ECG the total length of it is always 10 seconds long. So you can count the number of R waves in the rhythm strip and multiply by 6 to determine the beats per minute.



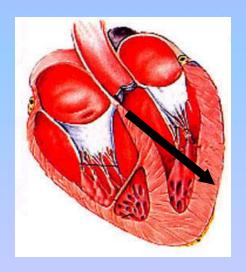
Rate? 12 (R waves) x 6 = 72 bpm

- In Module II you learned how to systematically analyze a rhythm by looking at the rate, regularity, P waves, PR interval and QRS complexes.
- In Modules III, IV and V you learned how to recognize Normal Sinus Rhythm and the 13 most common rhythm disturbances.
- If you need a refresher return to these modules.

Tip: the rhythm strip portion of the 12-lead ECG is a good place to look at when trying to determine the rhythm because the 12 leads only capture a few beats.



Axis refers to the mean QRS axis (or vector) during ventricular depolarization. As you recall when the ventricles depolarize (in a normal heart) the direction of current flows leftward and downward because most of the ventricular mass is in the left ventricle. We like to know the QRS axis because an abnormal axis can suggest disease such as pulmonary hypertension from a pulmonary embolism.



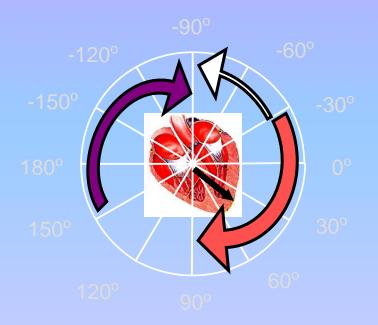
The QRS axis is determined by overlying a circle, in the frontal plane. By convention, the degrees of the circle are as shown.

The normal QRS axis lies between -30° and +90°.

A QRS axis that falls between -30° and -90° is abnormal and called left axis deviation.

A QRS axis that falls between +90° and +150° is abnormal and called right axis deviation.

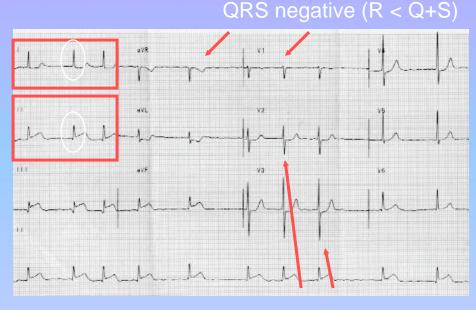
A QRS axis that falls between +150° and -90° is abnormal and called superior right axis deviation.



We can quickly determine whether the QRS axis is normal by looking at leads I and II.

If the QRS complex is overall positive (R > Q+S) in leads I and II, the QRS axis is normal.

In this ECG what leads have QRS complexes that are negative? equivocal?



QRS equivocal (R = Q+S)

How do we know the axis is normal when the QRS complexes are positive in leads I and II?

The answer lies in the fact that each frontal lead corresponds to a location on the circle.

#### **Limb leads**

$$I = +0^{\circ}$$

$$II = +60^{\circ}$$

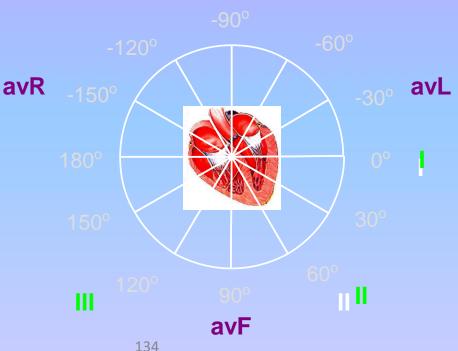
$$III = +120^{\circ}$$

#### **Augmented leads**

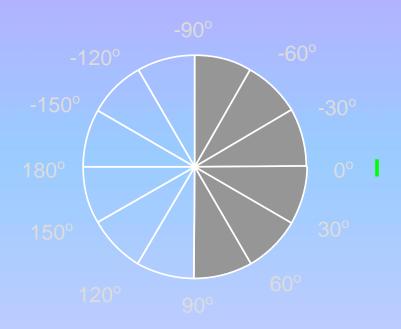
$$avL = -30^{\circ}$$

$$avF = +90^{\circ}$$

$$avR = -150^{\circ}$$



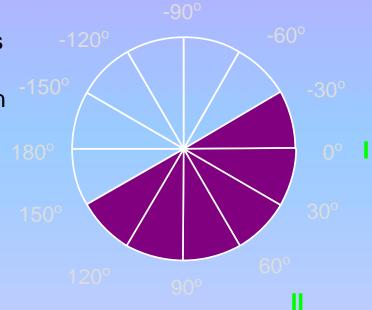
Since lead I is orientated at  $0^{\circ}$  a wave of depolarization directed towards it will result in a positive QRS axis. Therefore any mean QRS vector between -90° and +90° will be positive.



Since lead I is orientated at  $0^{\circ}$  a wave of depolarization directed towards it will result in a positive QRS axis. Therefore any mean QRS vector between -90° and +90° will be positive.

Similarly, since lead II is orientated at 60° a wave of depolarization directed towards it will result in a positive QRS axis.

Therefore any mean QRS vector between -30° and +150° will be positive.

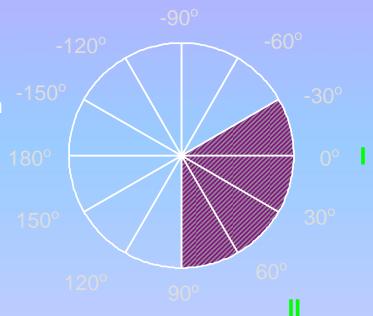


Since lead I is orientated at  $0^{\circ}$  a wave of depolarization directed towards it will result in a positive QRS axis. Therefore any mean QRS vector between -90° and +90° will be positive.

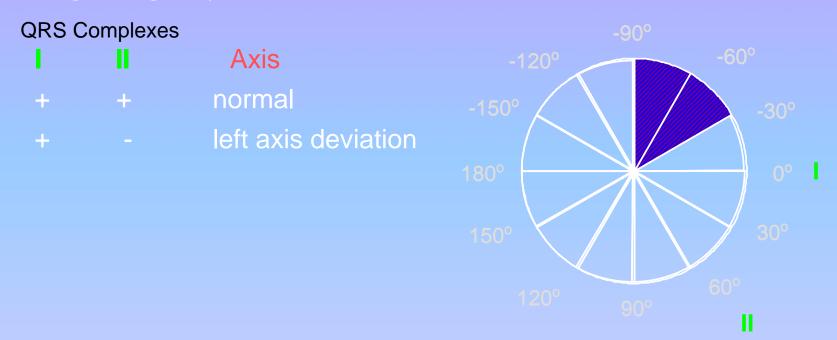
Similarly, since lead II is orientated at 60° a wave of depolarization directed towards it will result in a positive QRS axis.

Therefore any mean QRS vector between -30° and +150° will be positive.

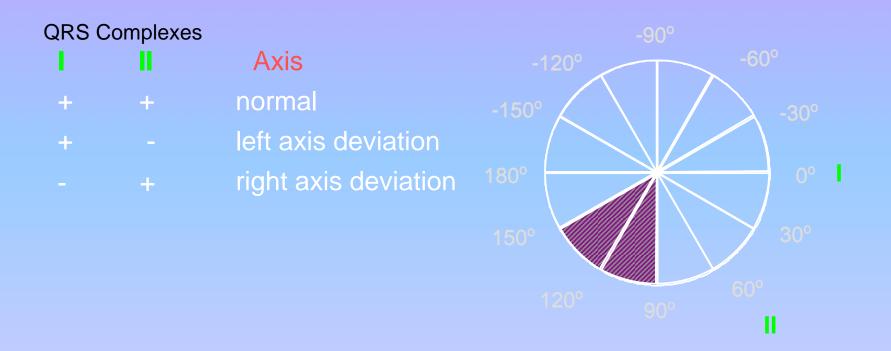
Therefore, if the QRS complex is positive in both leads I and II the QRS axis must be between -30° and 90° (where leads I and II overlap) and, as a result, the axis must be normal.



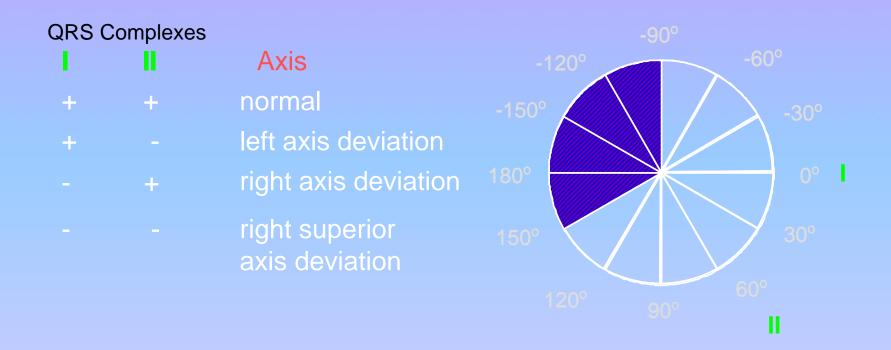
Now using what you just learned fill in the following table. For example, if the QRS is positive in lead I and negative in lead II what is the QRS axis? (normal, left, right or right superior axis deviation)



... if the QRS is negative in lead I and positive in lead II what is the QRS axis? (normal, left, right or right superior axis deviation)

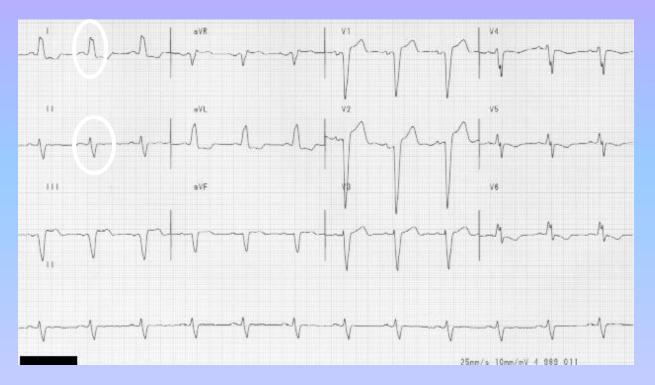


... if the QRS is negative in lead I and negative in lead II what is the QRS axis? (normal, left, right or right superior axis deviation)



Is the QRS axis normal in this ECG?

No, there is left axis deviation.



The QRS is positive in I and negative in II.

#### To summarize:

- The normal QRS axis falls between -30° and +90° because ventricular depolarization is leftward and downward.
- Left axis deviation occurs when the axis falls between  $-30^{\circ}$  and  $-90^{\circ}$ .
- Right axis deviation occurs when the axis falls between  $+90^{\circ}$  and  $+150^{\circ}$ .
- Right superior axis deviation occurs when the axis falls between between  $+150^{\circ}$  and  $-90^{\circ}$ .
- A quick way to determine the QRS axis is to look at the QRS complexes in leads I and II.

QRS C	omplexes	Avia
•		Axis
+	+	normal
+		left axis deviation
-		right axis deviation
-		right superior axis deviation

#### To summarize VII a:

- 1. Calculate RATE
- 2. Determine RHYTHM
- 3. Determine QRS AXIS
  - Normal
  - Left axis deviation
  - Right axis deviation
  - Right superior axis deviation

#### In VII b we will cover the next 3 steps:

- Calculate RATE
- Determine RHYTHM
- 3. Determine QRS AXIS
- 4. Calculate INTERVALS
- 5. Assess for HYPERTROPHY
- 6. Look for evidence of INFARCTION

# Reading 12-Lead ECGs

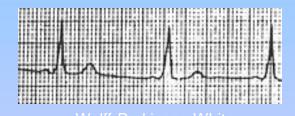
In Module VII a we introduced a 6 step approach for analyzing a 12-lead ECG and covered the first 3 steps. In this module we will cover the last 3 steps.

- 1. Calculate RATE
- 2. Determine RHYTHM
- 3. Determine QRS AXIS
- 4. Calculate INTERVALS
- Assess for HYPERTROPHY
- 6. Look for evidence of INFARCTION

- Intervals refers to the length of the PR and QT intervals and the width of the QRS complexes. You should have already determined the PR and QRS during the "rhythm" step, but if not, do so in this step.
- In the following few slides we'll review what is a normal and abnormal PR, QRS and QT interval. Also listed are a few common causes of abnormal intervals.

#### PR interval

< 0.12 s	0.12-0.20 s	> 0.20 s
High catecholamine states Wolff-Parkinson-White	Normal	AV nodal blocks

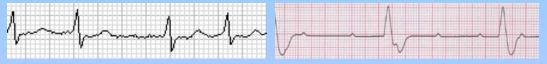




1st Degree AV Block

#### QRS complex

≤ 0.10 s	0.10-0.12 s	> 0.12 s
Normal	Incomplete bundle branch block	Bundle branch block PVC Ventricular rhythm



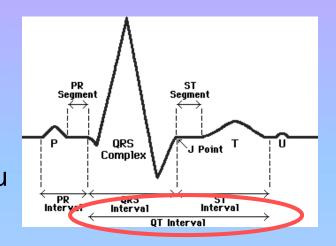
Incomplete bundle branch block

3<sup>rd</sup> degree AV block with ventricular escape rhythm

Remember: If you have a BBB determine if it is a right or left BBB. If you need a refresher see **Module VI**.

#### **QT** interval

The duration of the QT interval is proportionate to the heart rate. The faster the heart beats, the faster the ventricles repolarize so the shorter the QT interval. Therefore what is a "normal" QT varies with the heart rate. For each heart rate you need to calculate an adjusted QT interval, called the "corrected QT" (QTc):

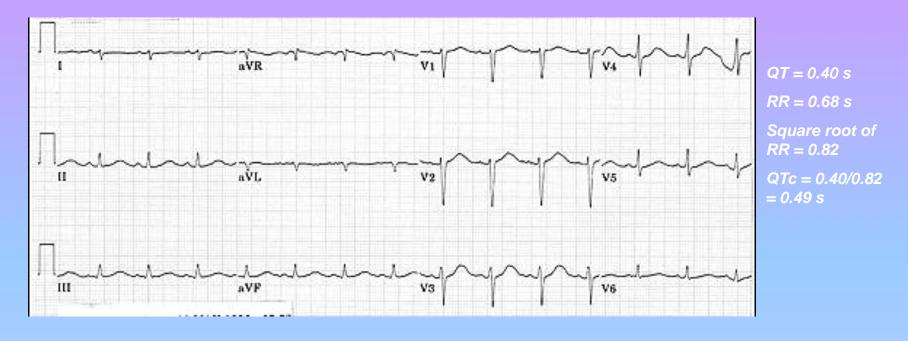


QTc = QT / square root of RR interval

#### QTc interval

< 0.44 s	> 0.44 s	Long QT
Normal	Long QT	
		Torsades de Pointes

A prolonged QT can be very dangerous. It may predispose an individual to a type of ventricular tachycardia called Torsades de Pointes. Causes include drugs, electrolyte abnormalities, CNS disease, post-MI, and congenital heart disease.



PR interval?

QRS width?

QTc interval?

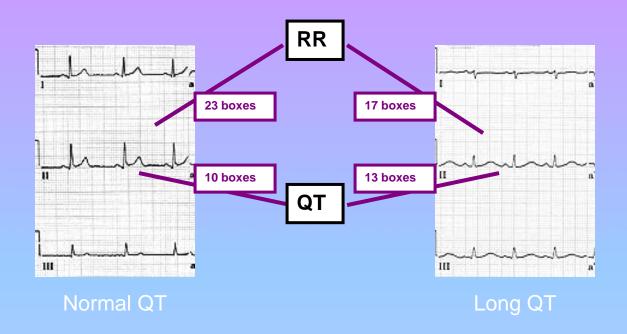
0.16 seconds

0.08 seconds

0.49 seconds

Interpretation of intervals? Normal PR and QRS, long QT

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Tip: Instead of calculating the QTc, a quick way to estimate if the QT interval long is to use the following rule:

A QT > half of the RR interval is probably long.

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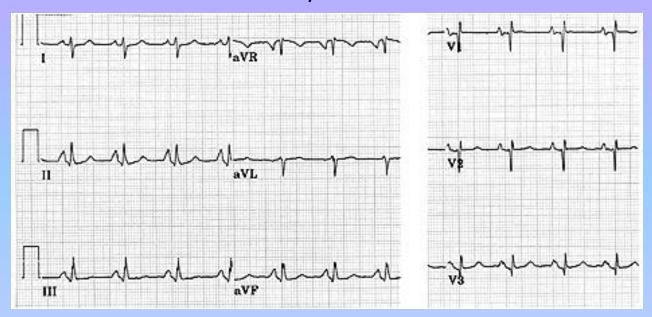
In this step of the 12-lead ECG analysis, we use the ECG to determine if any of the 4 chambers of the heart are enlarged or hypertrophied. We want to determine if there are any of the following:

- Right atrial enlargement (RAE)
- Left atrial enlargement (LAE)
- Right ventricular hypertrophy (RVH)
- Left ventricular hypertrophy (LVH)

- In Module VI we introduced the concept of left ventricular hypertrophy. As you remember the QRS voltage increases with LVH and is characterized by tall QRS complexes in certain leads. Similarly for right ventricular hypertrophy we look at the QRS complexes for changes in voltage patterns.
- With right and left atrial enlargement we analyze the P wave (since the P wave represents atrial depolarization). Here we also look for changes in voltage patterns.
- Note: as mentioned in Module VI criteria exists to diagnose LVH, the same goes for RAE, LAE and RVH. In the following slides we will be presenting criteria you can use. However other criteria exists and as a reference you might find it useful to carry a copy of Tom Evans' ECG Interpretation Cribsheet.

#### Right atrial enlargement

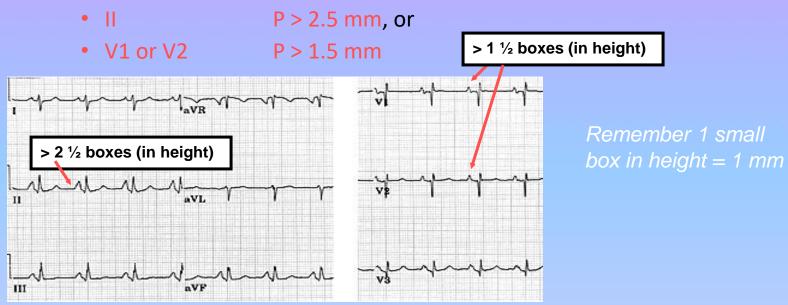
— Take a look at this ECG. What do you notice about the P waves?



The P waves are tall, especially in leads II, III and avF.
Ouch! They would hurt to sit on!!

#### Right atrial enlargement

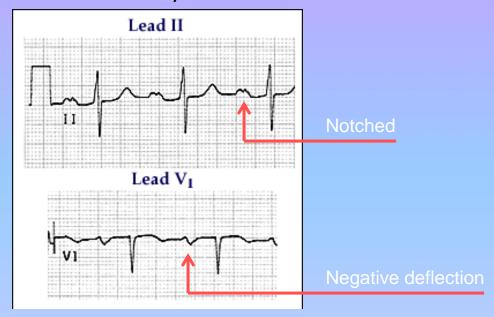
To diagnose RAE you can use the following criteria:



A cause of RAE is RVH from pulmonary hypertension.

#### Left atrial enlargement

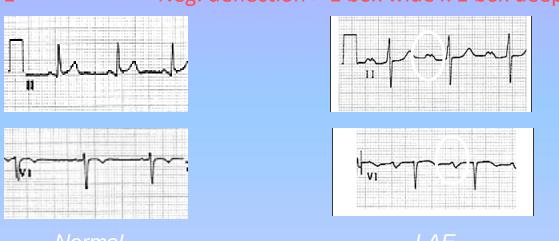
— Take a look at this ECG. What do you notice about the P waves?



The P waves in lead II are notched and in lead V1 they have a deep and wide negative component.

#### Left atrial enlargement

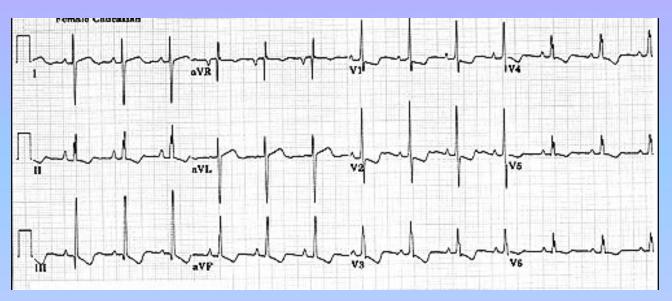
- To diagnose LAE you can use the following criteria:
  - II > 0.04 s (1 box) between notched peaks, or
  - V1 Neg. deflection > 1 box wide x 1 box deep



A common cause of LAE is LVH from hypertension.

#### Right ventricular hypertrophy

Take a look at this ECG. What do you notice about the axis and QRS complexes over the right ventricle (V1, V2)?

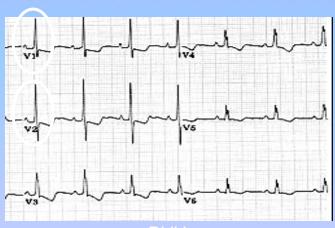


There is right axis deviation (negative in I, positive in II) and there are tall R waves in V1, V2.

#### Right ventricular hypertrophy

- Compare the R waves in V1, V2 from a normal ECG and one from a person with RVH.
- Notice the R wave is normally small in V1, V2 because the right ventricle does not have a lot of muscle mass.
- But in the hypertrophied right ventricle the R wave is tall in V1, V2.





RVH

#### Right ventricular hypertrophy

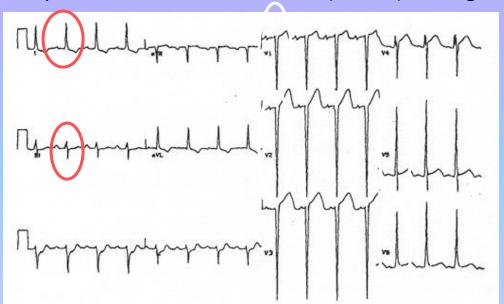
To diagnose RVH you can use the following criteria:

Right axis deviation, and R wave > 7mm tall Pemal Caucasian αVŘ nVL.

A common cause of RVH is left heart failure.

#### Left ventricular hypertrophy

– Take a look at this ECG. What do you notice about the axis and QRS complexes over the left ventricle (V5, V6) and right ventricle (V1, V2)?

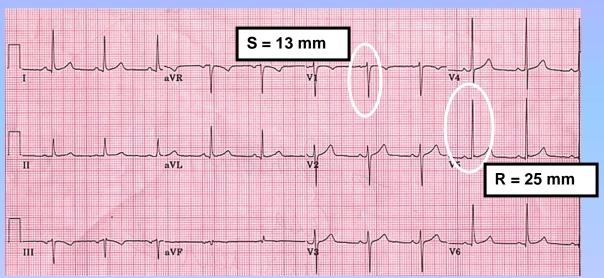


The deep S waves seen in the leads over the right ventricle are created because the heart is depolarizing left, superior and posterior (away from leads V1, V2).

There is left axis deviation (positive in I, negative in II) and there are tall R waves in V5, V6 and deep S waves in V1, V2.

#### Left ventricular hypertrophy

- To diagnose LVH you can use the following criteria\*:
  - R in V5 (or V6) + S in V1 (or V2) > 35 mm, or
  - avL R > 13 mm



\* There are several other criteria for the diagnosis of LVH.

A common cause of LVH is hypertension.

A 63 yo man has longstanding, uncontrolled hypertension. Is there evidence of heart disease from his hypertension? (Hint: There a 3 abnormalities.)



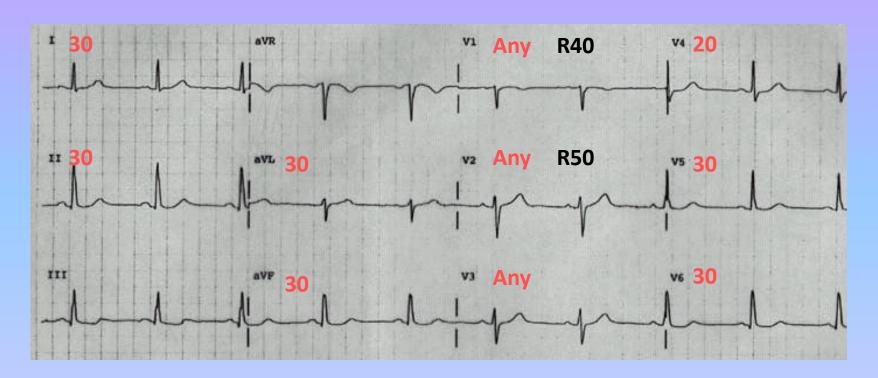
Yes, there is left axis deviation (positive in I, negative in II), left atrial enlargement (> 1 x 1 boxes in V1) and LVH (R in V5 = 27 + S in V2 = 10  $\rightarrow$  > 35 mm).

- When analyzing a 12-lead ECG for evidence of an infarction you want to look for the following:
  - Abnormal Q waves
  - ST elevation or depression
  - Peaked, flat or inverted T waves
- These topics were covered in Modules V and VI where you learned:
  - ST elevation (or depression) of 1 mm in 2 or more contiguous leads is consistent with an AMI
  - There are ST elevation (Q-wave) and non-ST elevation (non-Q wave)
     MIs

Tip: One way to determine if Q waves (and R waves) are abnormal is by looking at the width and using the following mantra (read red downwards):

Any	Any Q wave in V1	
Any	Any Q wave in V2	
Any	Any Q wave in V3	
20	A Q wave <u>&gt;</u> 20 msec in V4	(i.e. 0.02 sec or ½ width of a box)
30	A Q wave <b>&gt;</b> 30 msec in V5	
30	A Q wave $\geq$ 30 msec in V6	
30	A Q wave > 30 msec in I	
30	A Q wave > 30 msec in avL	
30	A Q wave > 30 msec in II	
30	A Q wave ≥ 30 msec in avF	
R40	A R wave > 40 msec in V1	
R50	A R wave > 50 msec in V2	
	7	

This mantra corresponds to the ECG in the following way:



- 1. Calculate RATE
- 2. Determine RHYTHM
- 3. Determine QRS AXIS
  - Normal
  - Left axis deviation
  - Right axis deviation
  - Right superior axis deviation

- 1. Calculate RATE
- 2. Determine RHYTHM
- 3. Determine QRS AXIS
- 4. Calculate INTERVALS
  - PR
  - QRS
  - QT

- 1. Calculate RATE
- 2. Determine RHYTHM
- 3. Determine QRS AXIS
- 4. Calculate INTERVALS
- 5. Assess for HYPERTROPHY
  - Right and left atrial enlargement
  - Right and left ventricular hypertrophy

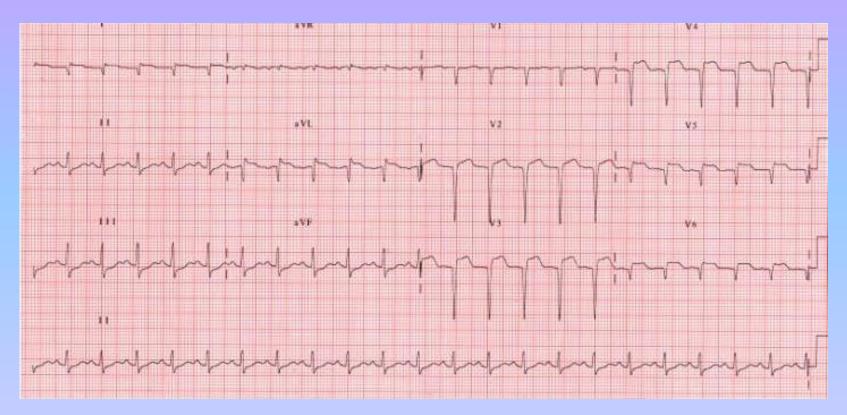
- 1. Calculate RATE
- 2. Determine RHYTHM
- 3. Determine QRS AXIS
- 4. Calculate INTERVALS
- 5. Assess for HYPERTROPHY
- Look for evidence of INFARCTION
  - Abnormal Q waves
  - ST elevation or depression
  - Peaked, flat or inverted T waves

#### To summarize:

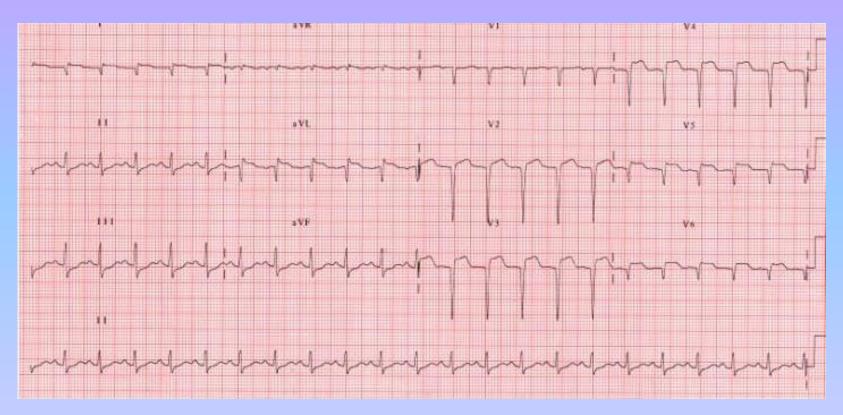
- 1. Calculate RATE
- 2. Determine RHYTHM
- 3. Determine QRS AXIS
- 4. Calculate INTERVALS
- 5. Assess for HYPERTROPHY
- 6. Look for evidence of INFARCTION

Now to finish this module lets analyze a 12-lead ECG!

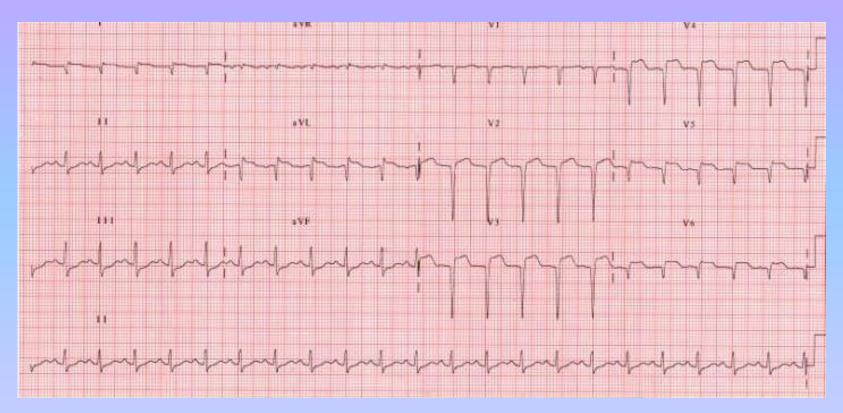
A 16 yo young man ran into a guardrail while riding a motorcycle. In the ED he is comatose and dyspneic. This is his ECG.



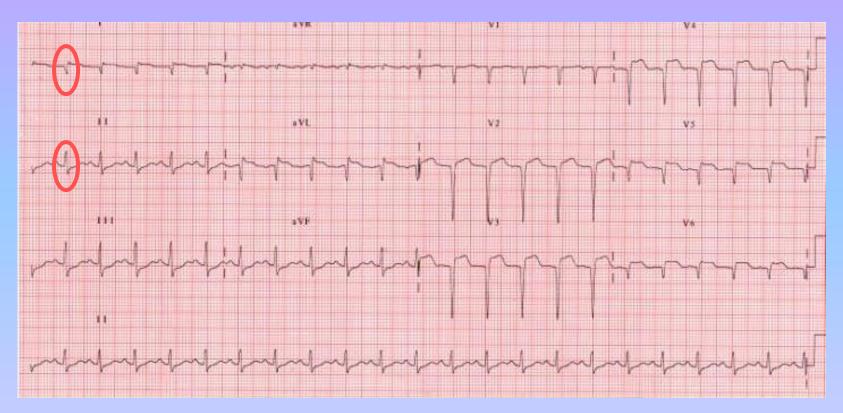
What is the rate? Approx. 132 bpm (22 R waves x 6)



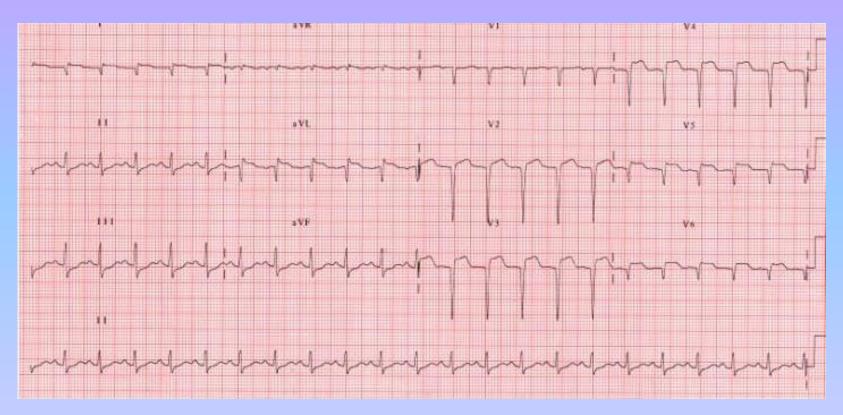
What is the rhythm? Sinus tachycardia



What is the QRS axis? Right axis deviation (- in I, + in II)

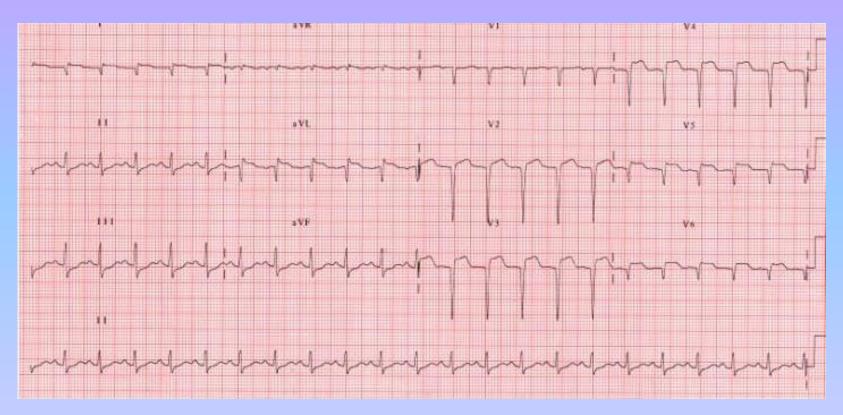


What are the PR, QRS PR = 0.12 s, QRS = 0.08 s, QTc = 0.482 s and QT intervals?

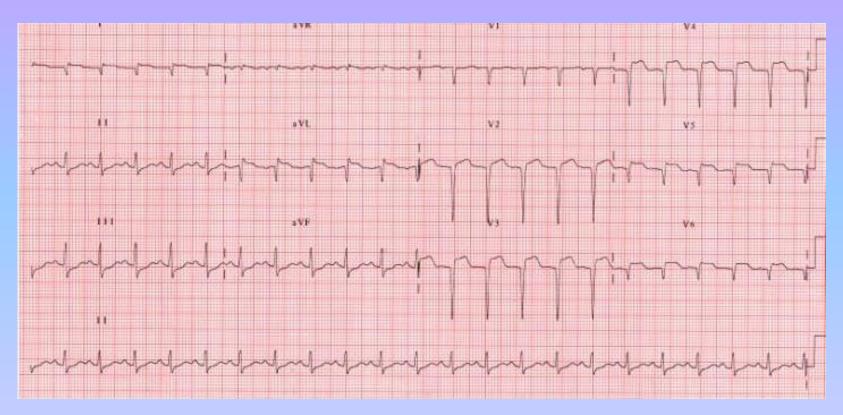


Is there evidence of atrial enlargement?

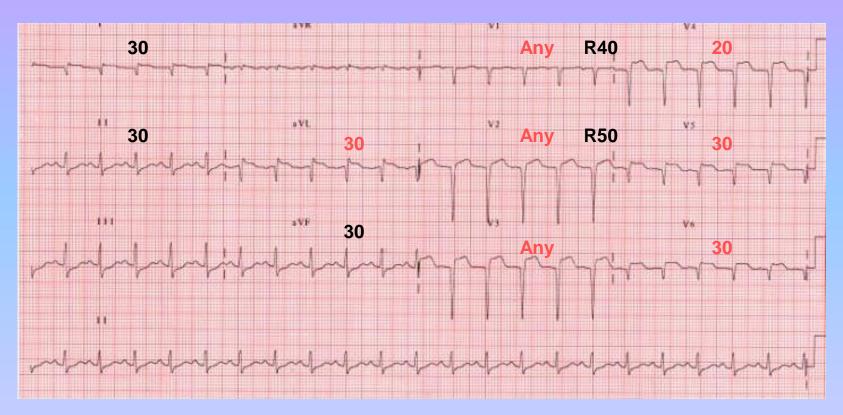
No (no peaked, notched or negatively deflected P waves)



Is there evidence of No (no tall R waves in V1/V2 or V5/V6) ventricular hypertrophy?

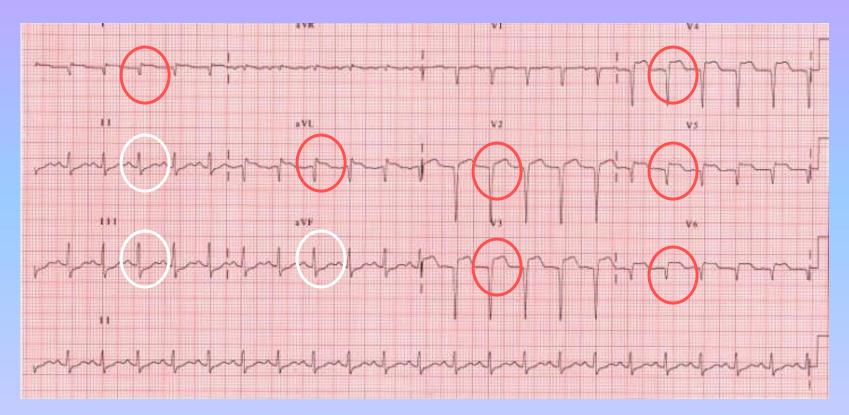


Infarct: Are there abnormal Yes! In leads V1-V6 and I, avL Q waves?

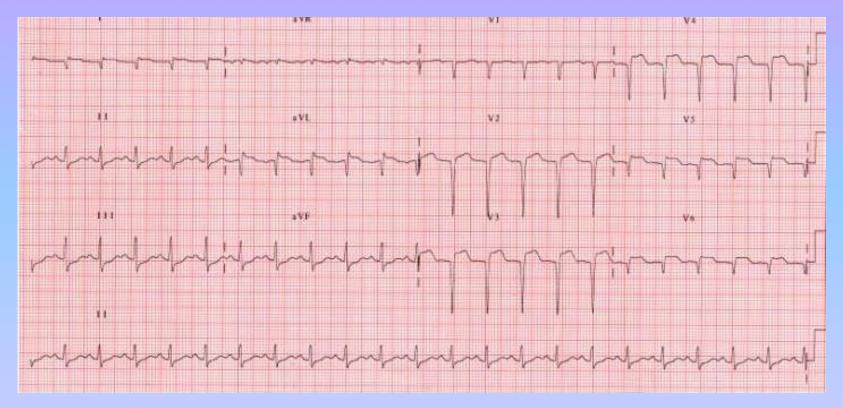


Infarct: Is the ST elevation or depression?

Yes! Elevation in V2-V6, I and avL. Depression in II, III and avF.



Infarct: Are there T wave No changes?



ECG analysis: Sinus tachycardia at 132 bpm, right axis deviation, long QT, and evidence of ST elevation infarction in the anterolateral leads (V1-V6, I, avL) with reciprocal changes (the ST depression) in the inferior leads (II, III, avF).

This young man suffered an acute myocardial infarction after blunt trauma. An echocardiogram showed anteroseptal akinesia in the left ventricle with severely depressed LV function (EF=28%). An angiogram showed total occlusion in the proximal LAD with collaterals from the RCA and LCX.

