Basics of ECG

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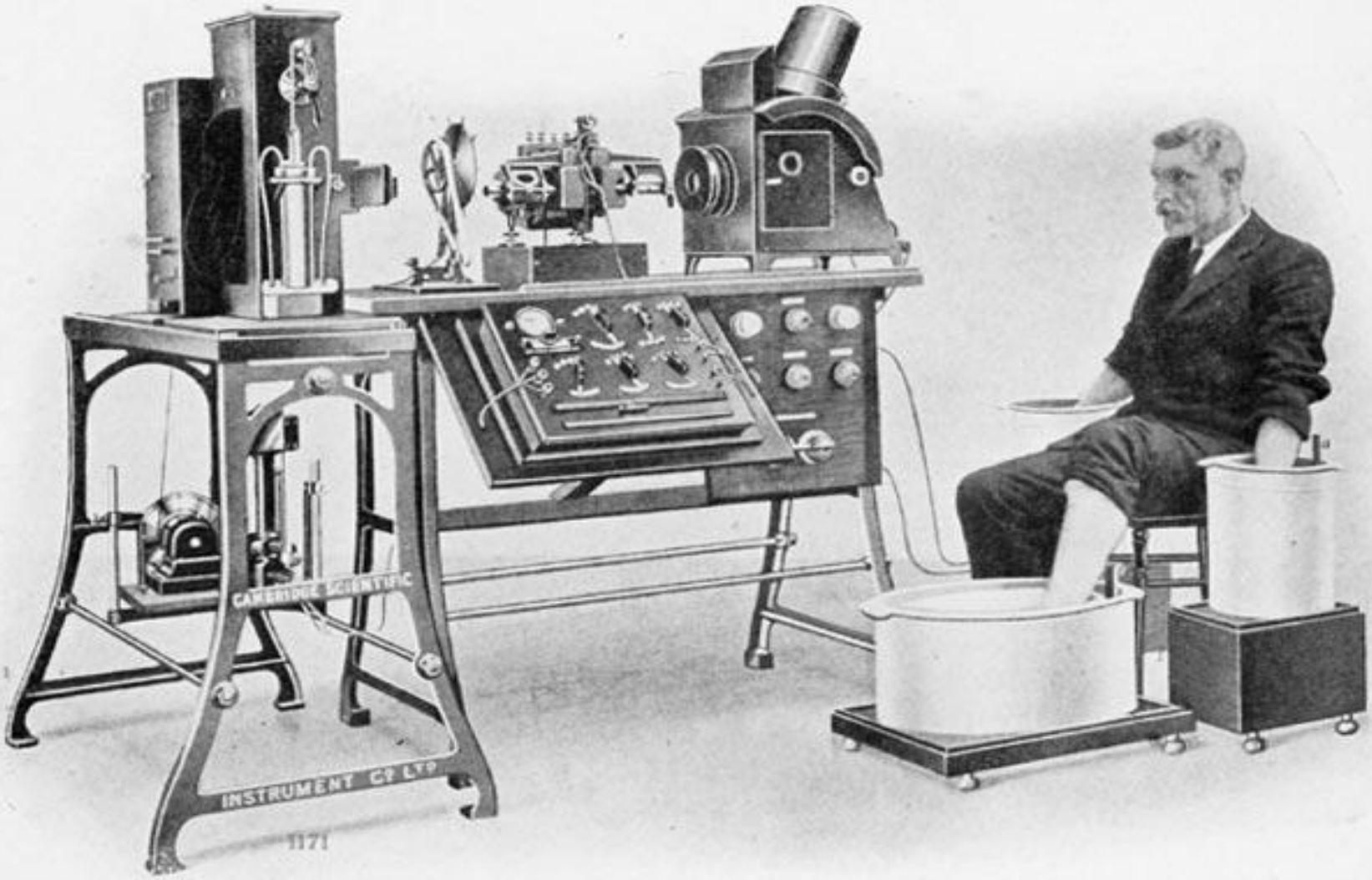
HISTORY

- 1842- Italian scientist Carlo Matteucci realizes that electricity is associated with the heart beat
- 1876- Irish scientist Marey analyzes the electric pattern of frog's heart
- 1895 - William Einthoven , credited for the invention of EKG
- 1906 - using the string electrometer EKG,

  William Einthoven diagnoses some heart problems

CONTD…

- 1924 - the noble prize for physiology or medicine is given to William Einthoven for his work on EKG
- 1938 -AHA and Cardiac society of great Britan defined and position of chest leads
- 1942- Goldberger increased Wilson's Unipolar lead voltage by 50% and made Augmented leads
- 2005- successful reduction in time of onset of chest pain and PTCA by wireless transmission of ECG on his PDA.



PHOTOGRAPH OF A COMPLETE ELECTROCARDIOGRAPH, SHOWING THE MANNER IN WHICH THE ELECTRODES ARE ATTACHED TO THE PATIENT, IN THIS CASE THE HANDS AND ONE FOOT BEING IMMERSED IN JARS OF SALT SOLUTION

MODERN ECG INSTRUMENT

What is an EKG?

- The electrocardiogram (EKG) is a representation of the electrical events of the cardiac cycle.
- Each event has a distinctive waveform
- the study of waveform can lead to greater insight into a patient's cardiac pathophysiology.

With EKGs we can identify

- Arrhythmias
- Myocardial ischemia and infarction
- Pericarditis
- Chamber hypertrophy
- Electrolyte disturbances (i.e. hyperkalemia, hypokalemia)
- Drug toxicity (i.e. digoxin and drugs which prolong the QT interval)

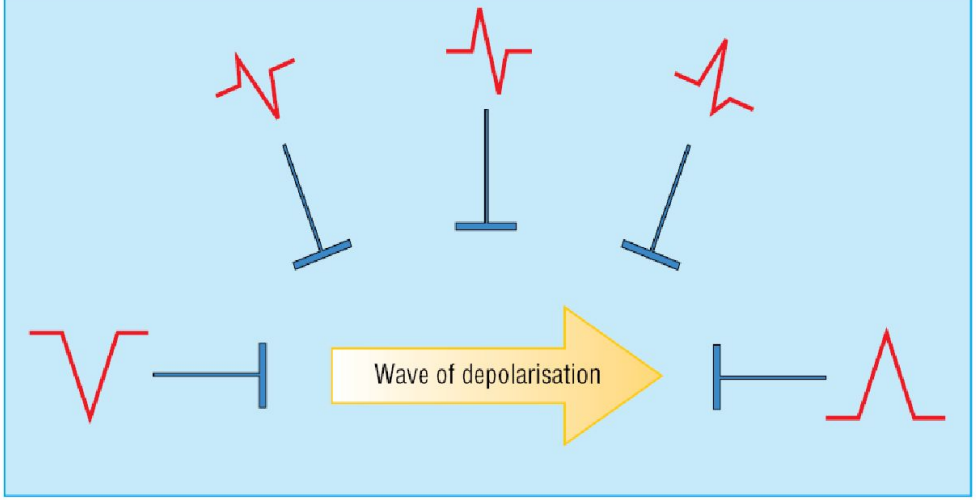
Depolarization

- Contraction of any muscle is associated with electrical changes called depolarization
- These changes can be detected by electrodes attached to the surface of the body

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.

- Standard calibration
  - 25 mm/s
  - 0.1 mV/mm
- Electrical impulse that travels towards the electrode produces an upright ("positive") deflection



Wave of depolarisation. Shape of QRS complex in any lead depends on orientation of that lead to vector of depolarisation

Impulse Conduction & the ECG

Sinoatrial node

↓

AV node

↓

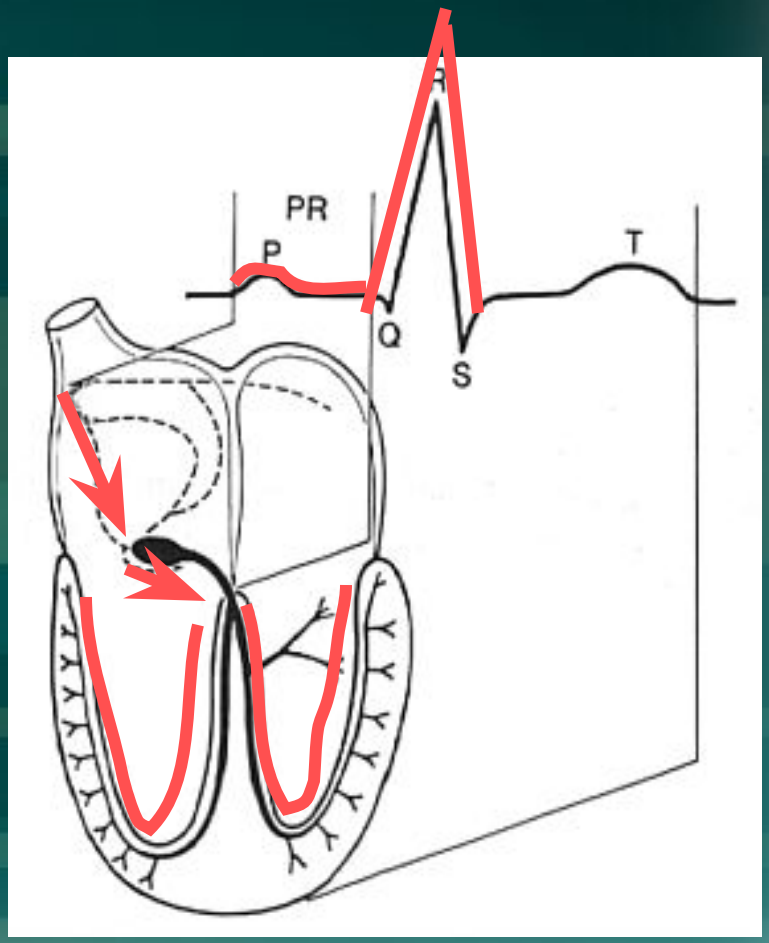
Bundle of His

↓

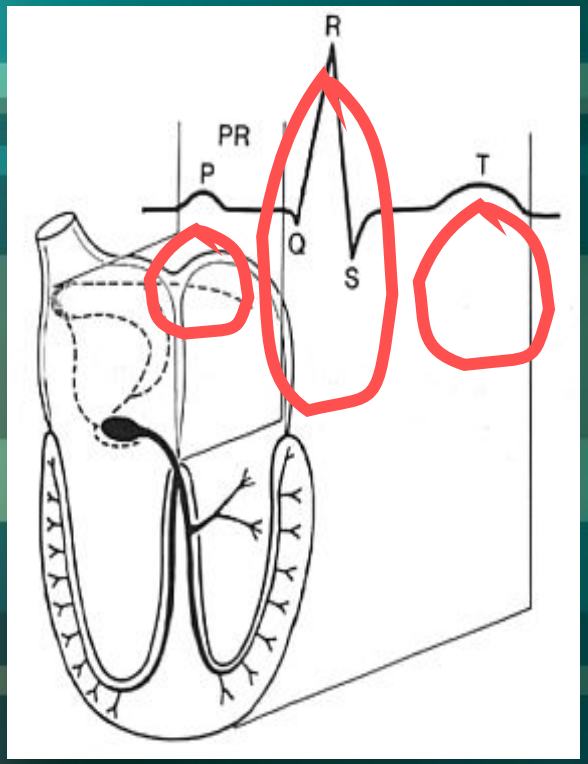
Bundle Branches

↓

Purkinje fibers



The "PQRST"



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

The PR Interval

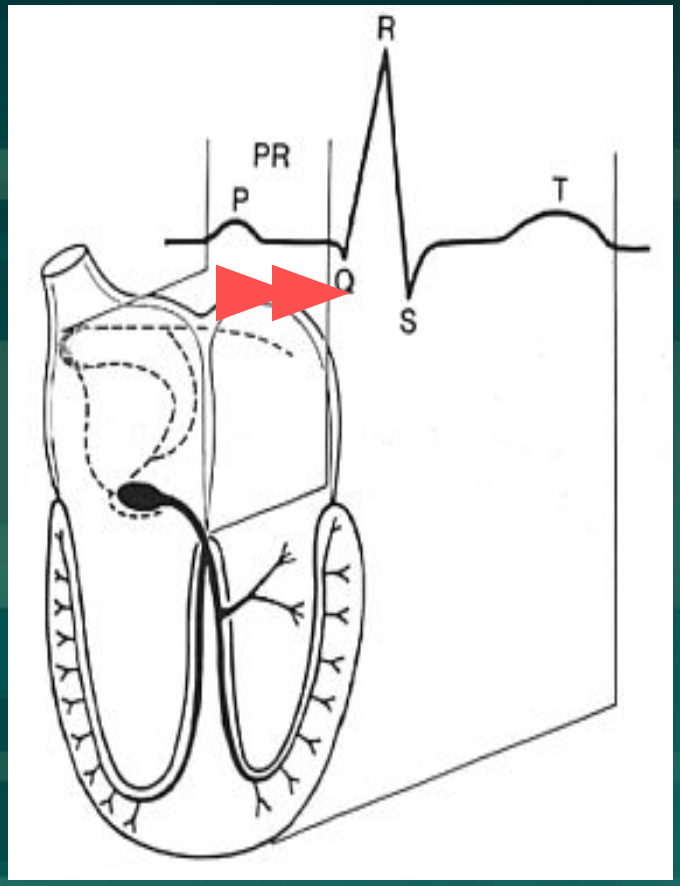
Atrial depolarization

+

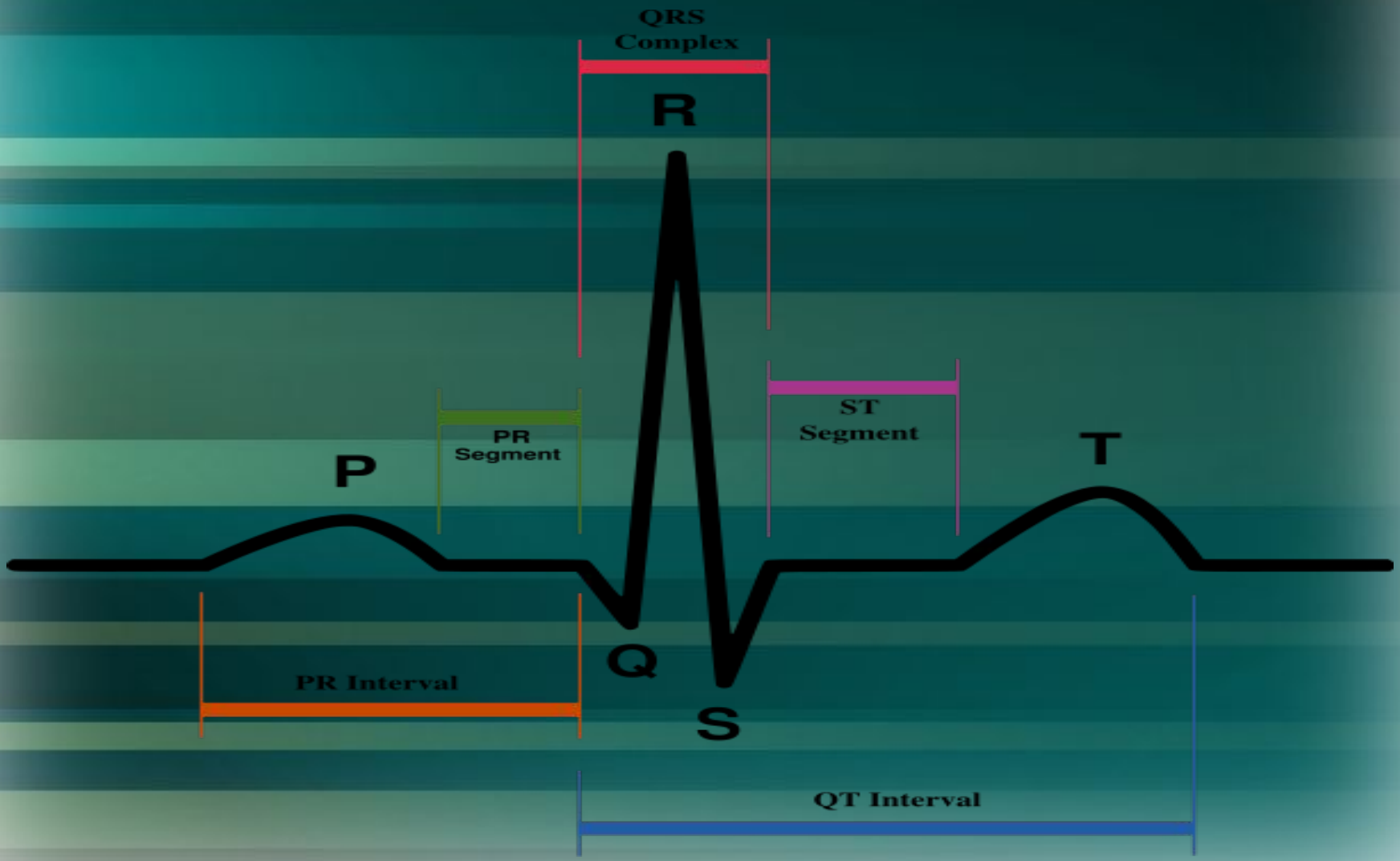
delay in AV junction

(AV node/Bundle of His)

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

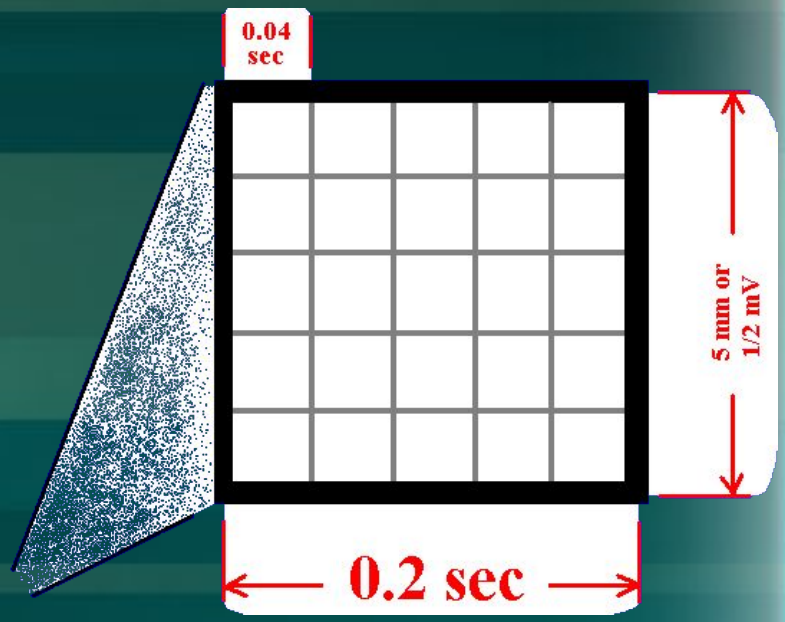


NORMAL ECG


QRS
Complex
R
PR
Segment
ST
Segment
P
T
PR Interval
Q
S
QT Interval

The ECG Paper

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



EKG Leads

which measure the difference in electrical potential between two points

1. Bipolar Leads: Two different points on the body

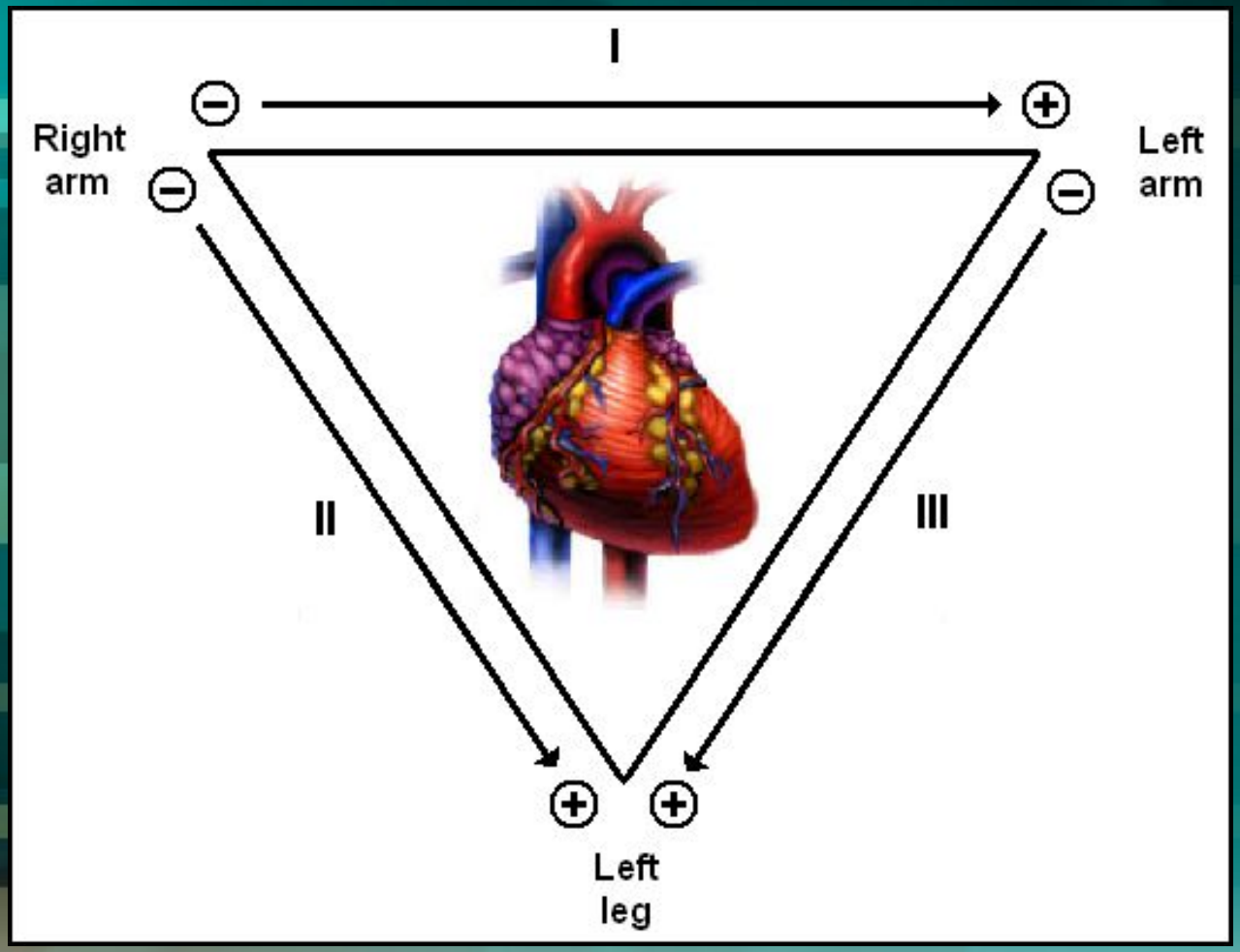
2. Unipolar Leads: One point on the body and a virtual reference point with zero electrical potential, located in the center of the heart

EKG Leads

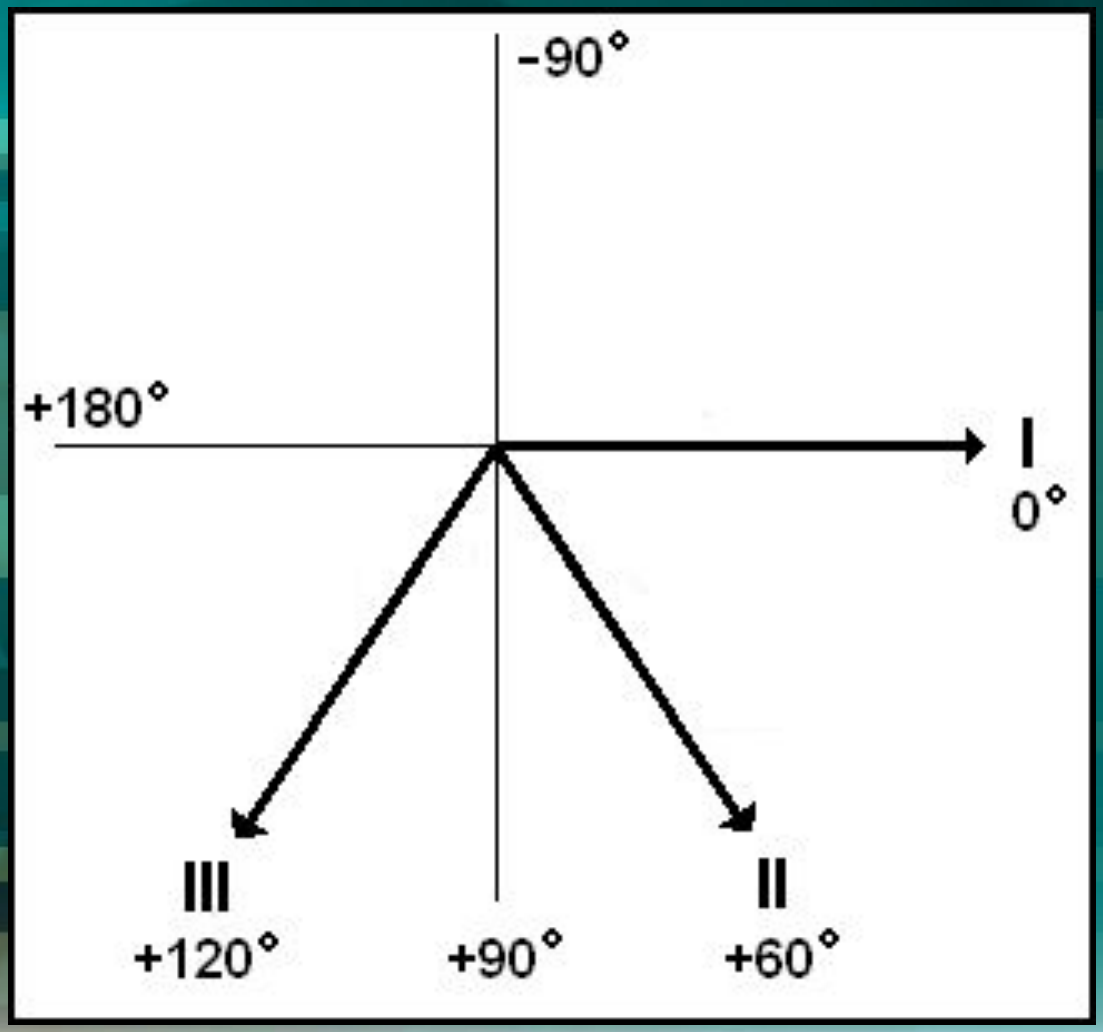
The standard EKG has 12 leads:

- 3 Standard Limb Leads
- 3 Augmented Limb Leads
- 6 Precordial Leads

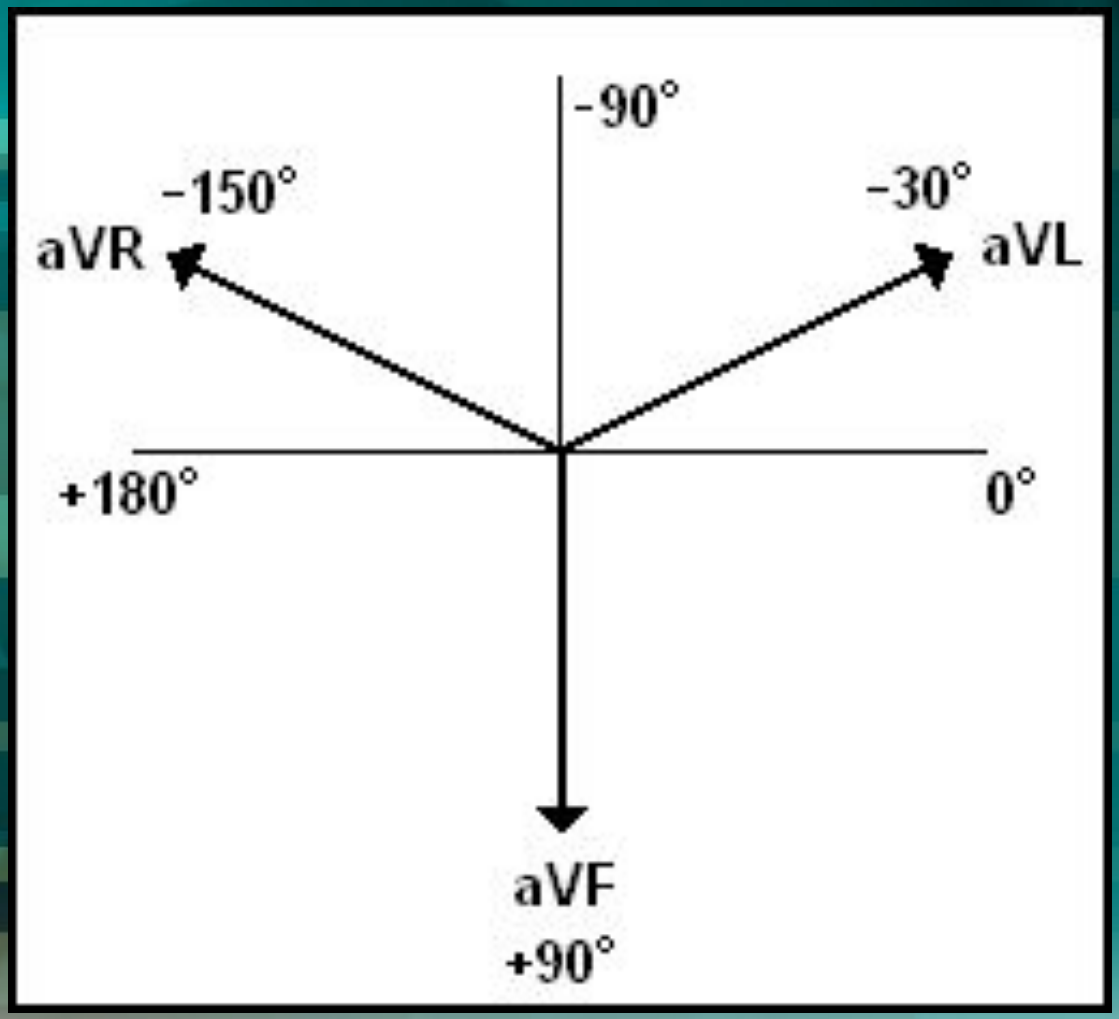
Standard Limb Leads



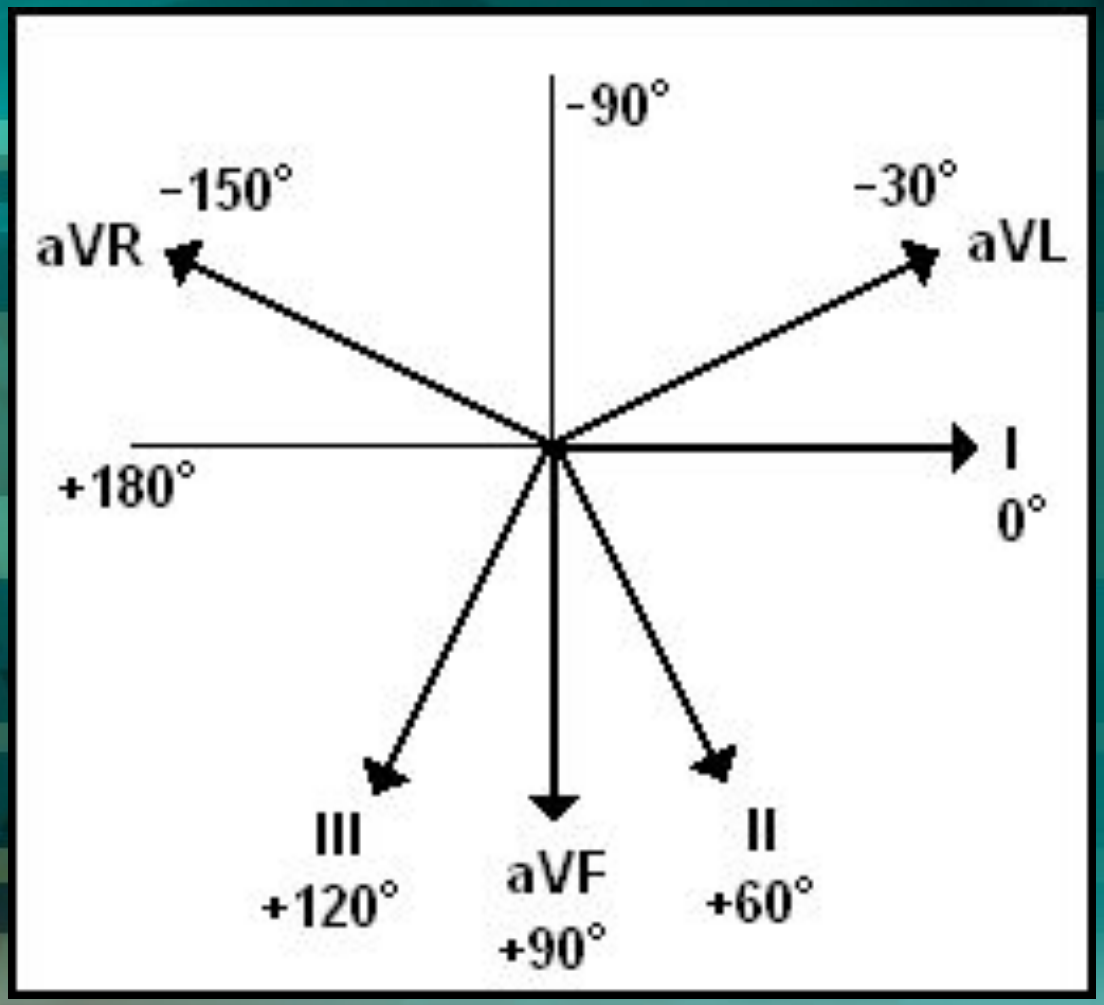
Standard Limb Leads



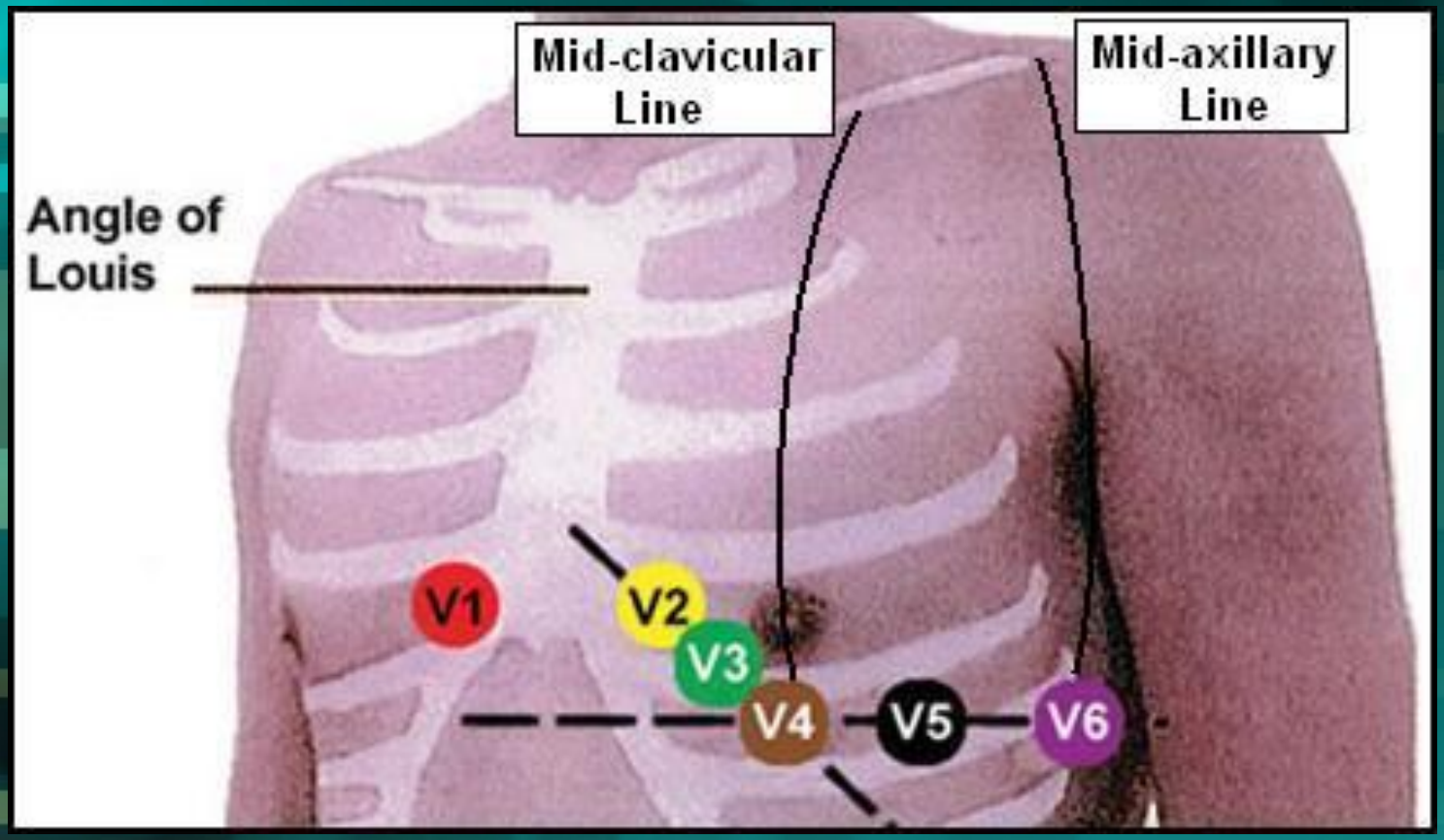
Augmented Limb Leads



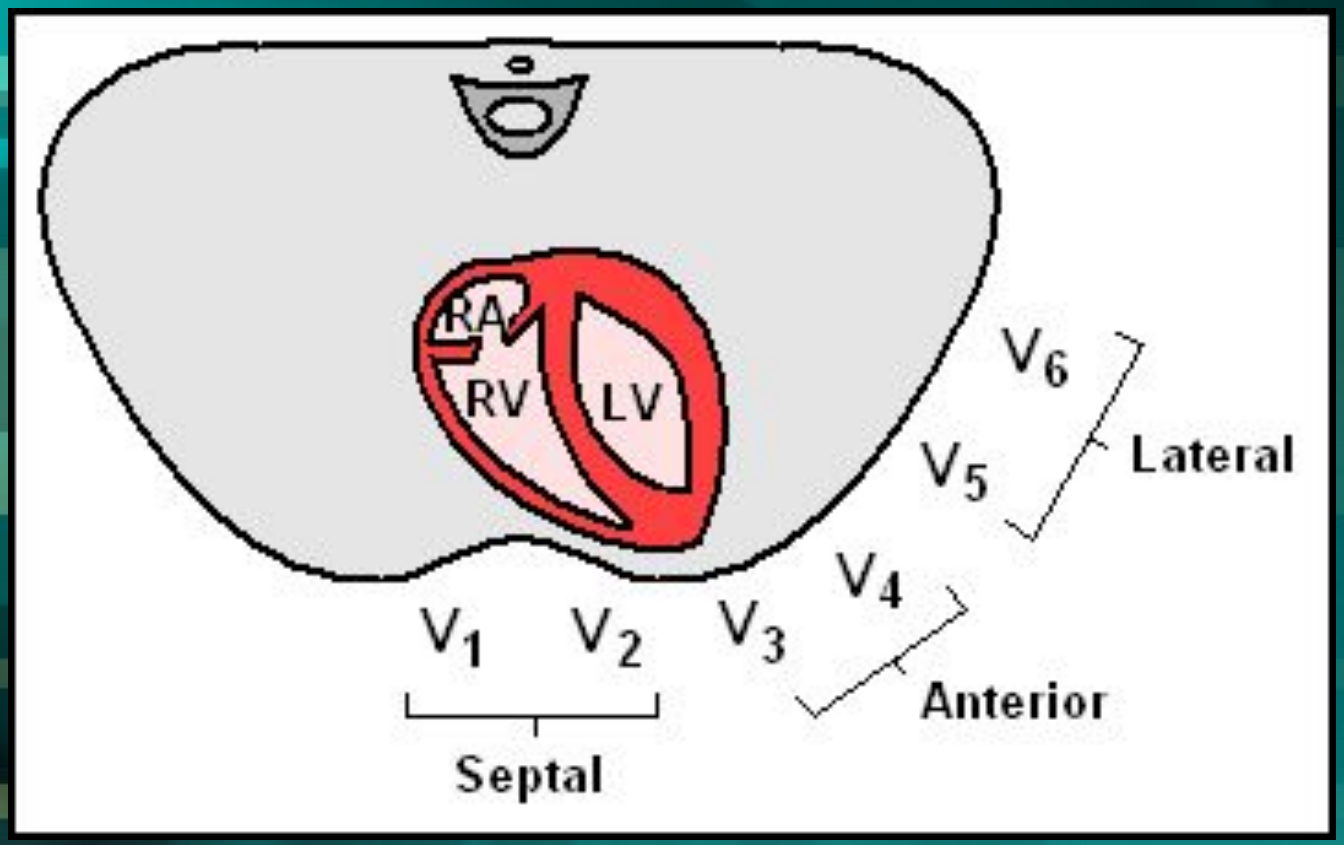
All Limb Leads



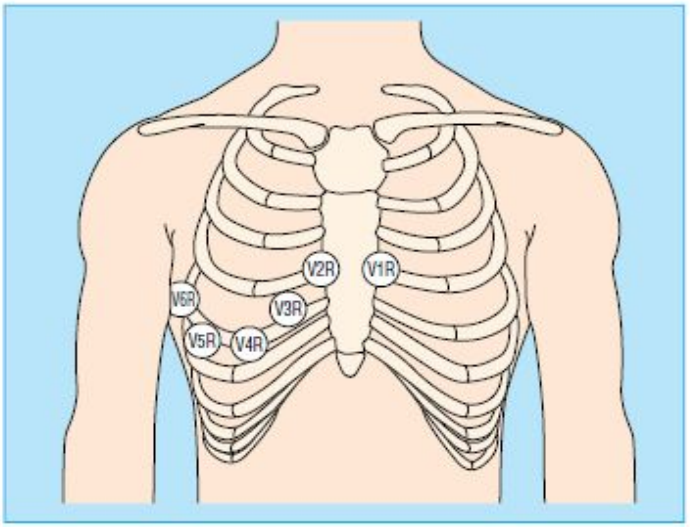
Precordial Leads



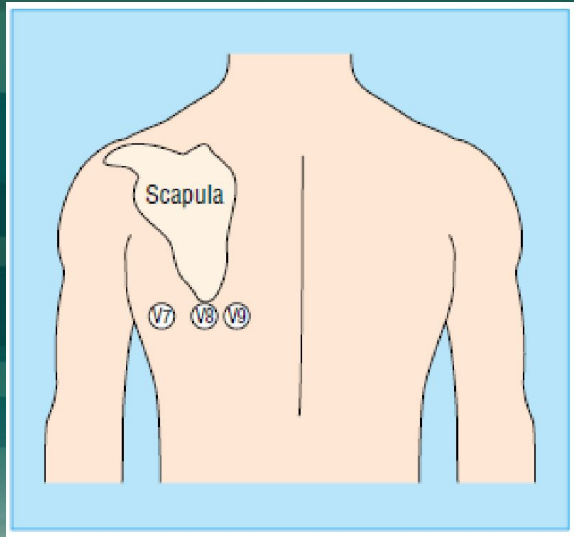
Precordial Leads



Right Sided & Posterior Chest Leads



Placement of right sided chest leads



Position of V7, V8, and V9 on posterior chest wall

Arrangement of Leads on the EKG

I	aVR	V_1	V_4
II	aVL	V_2	V_5
III	aVF	V_3	V_6

Anatomic Groups (Septum)

I Lateral	aVR None	V_1 Septal	V_4 Anterior
II Inferior	aVL Lateral	V_2 Septal	V_5 Lateral
III Inferior	aVF Inferior	V_3 Anterior	V_6 Lateral

Anatomic Groups (Anterior Wall)

I Lateral	aVR None	V_1 Septal	V_4 Anterior
II Inferior	aVL Lateral	V_2 Septal	V_5 Lateral
III Inferior	aVF Inferior	V_3 Anterior	V_6 Lateral

Anatomic Groups (Lateral Wall)

I Lateral	aVR None	V_1 Septal	V_4 Anterior
II Inferior	aVL Lateral	V_2 Septal	V_5 Lateral
III Inferior	aVF Inferior	V_3 Anterior	V_6 Lateral

Anatomic Groups (Inferior Wall)

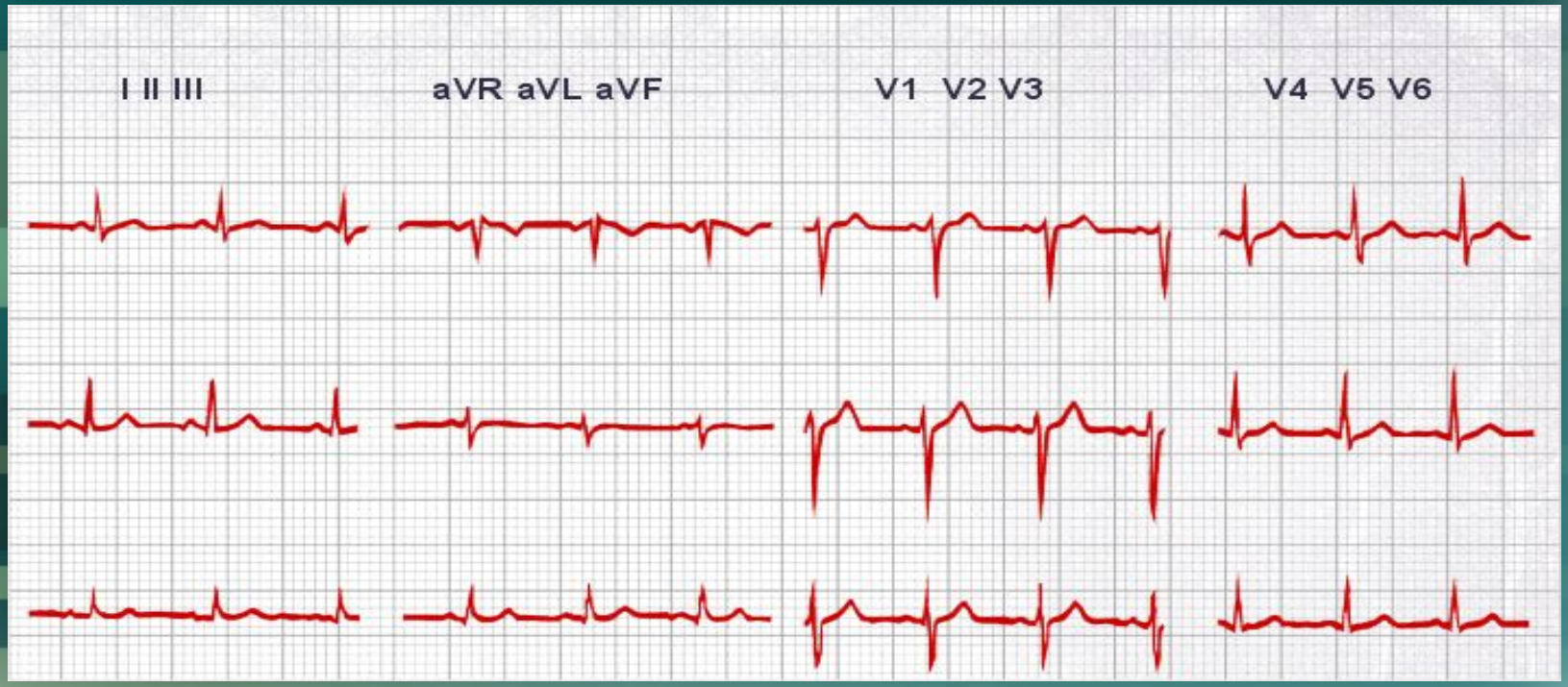
I Lateral	aVR None	V_1 Septal	V_4 Anterior
II Inferior	aVL Lateral	V_2 Septal	V_5 Lateral
III Inferior	aVF Inferior	V_3 Anterior	V_6 Lateral

Anatomic Groups (Summary)

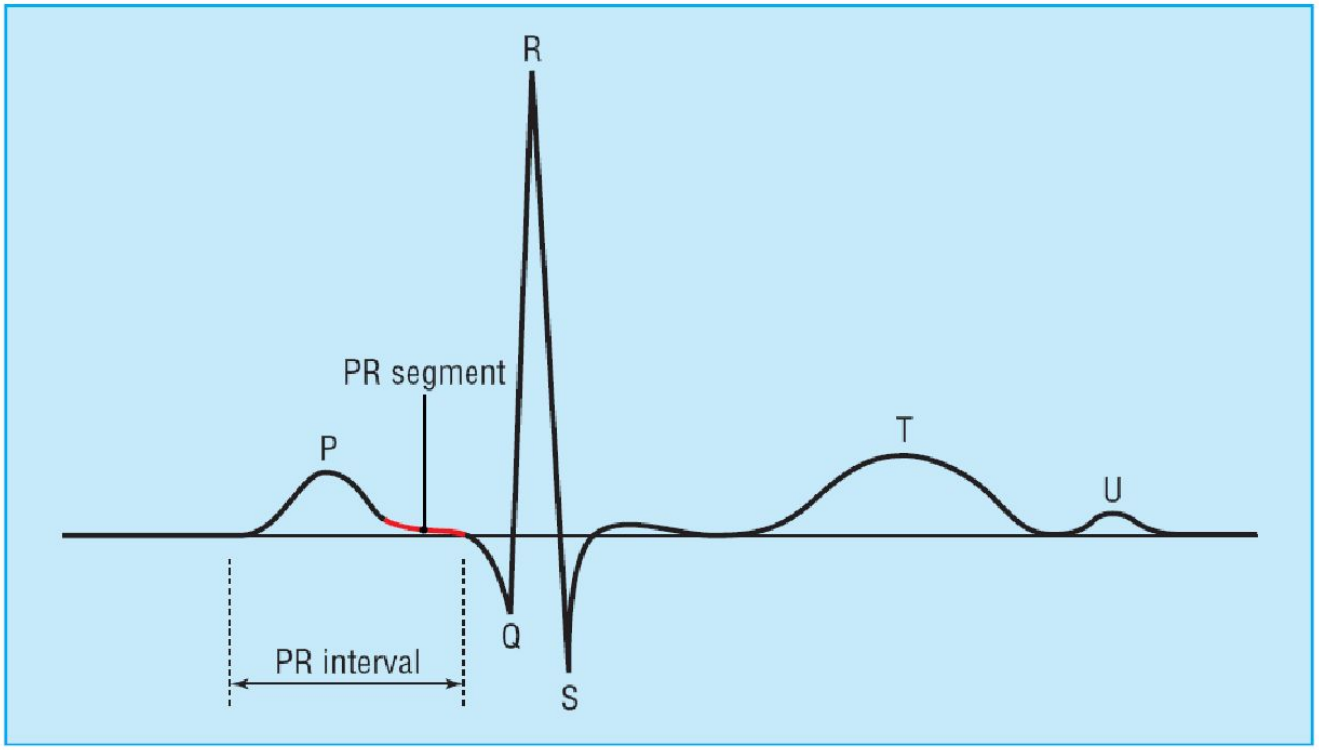
I Lateral	aVR None	V_1 Septal	V_4 Anterior
II Inferior	aVL Lateral	V_2 Septal	V_5 Lateral
III Inferior	aVF Inferior	V_3 Anterior	V_6 Lateral

ECG RULES

- Professor Chamberlains 10 rules of normal:-



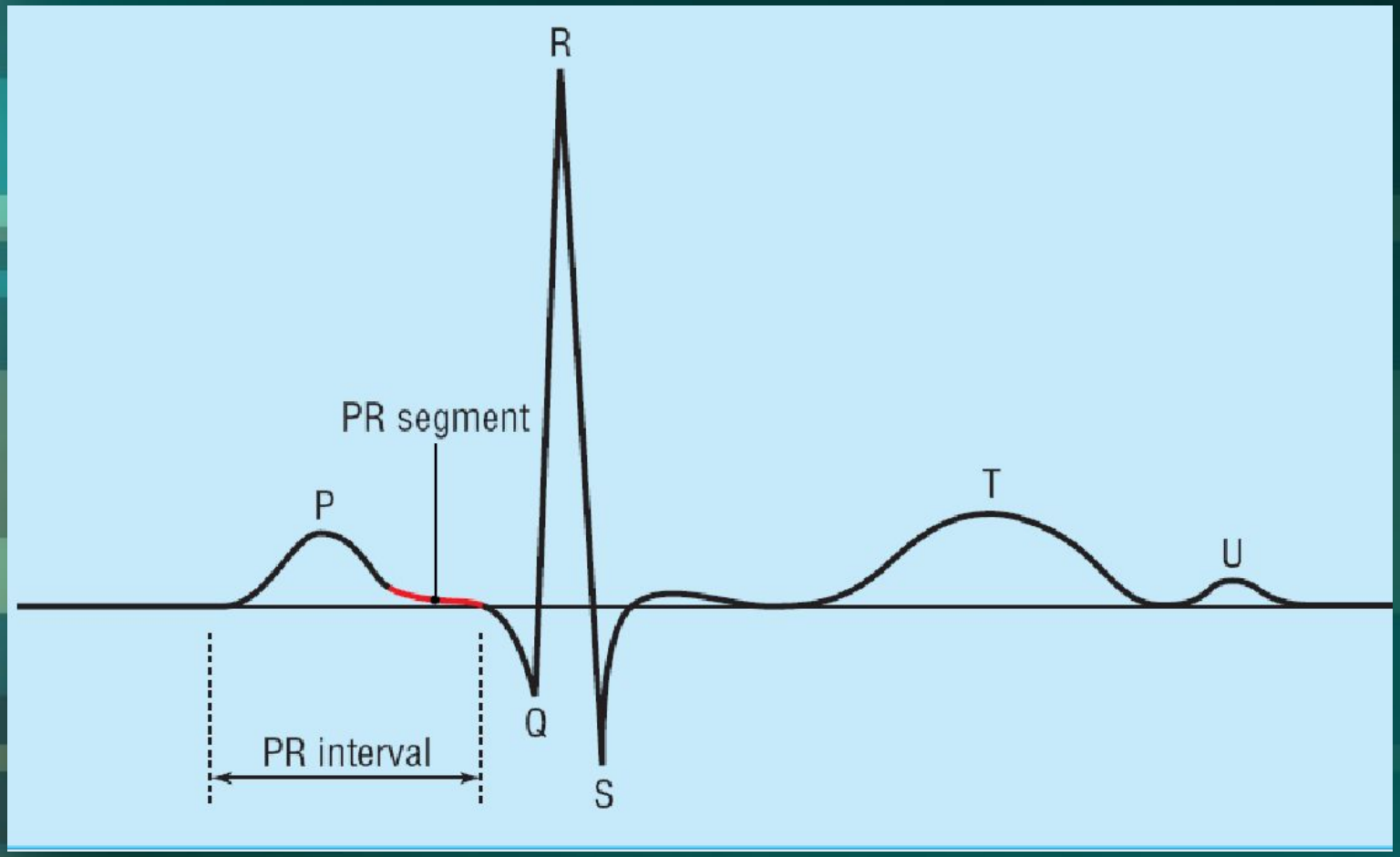
RULE 1



Normal duration of PR interval is 0.12-0.20 s (three to five small squares)

PR interval should be 120 to 200 milliseconds or 3 to 5 little squares

RULE 2



The width of the QRS complex should not exceed 110 ms, less than 3 little squares

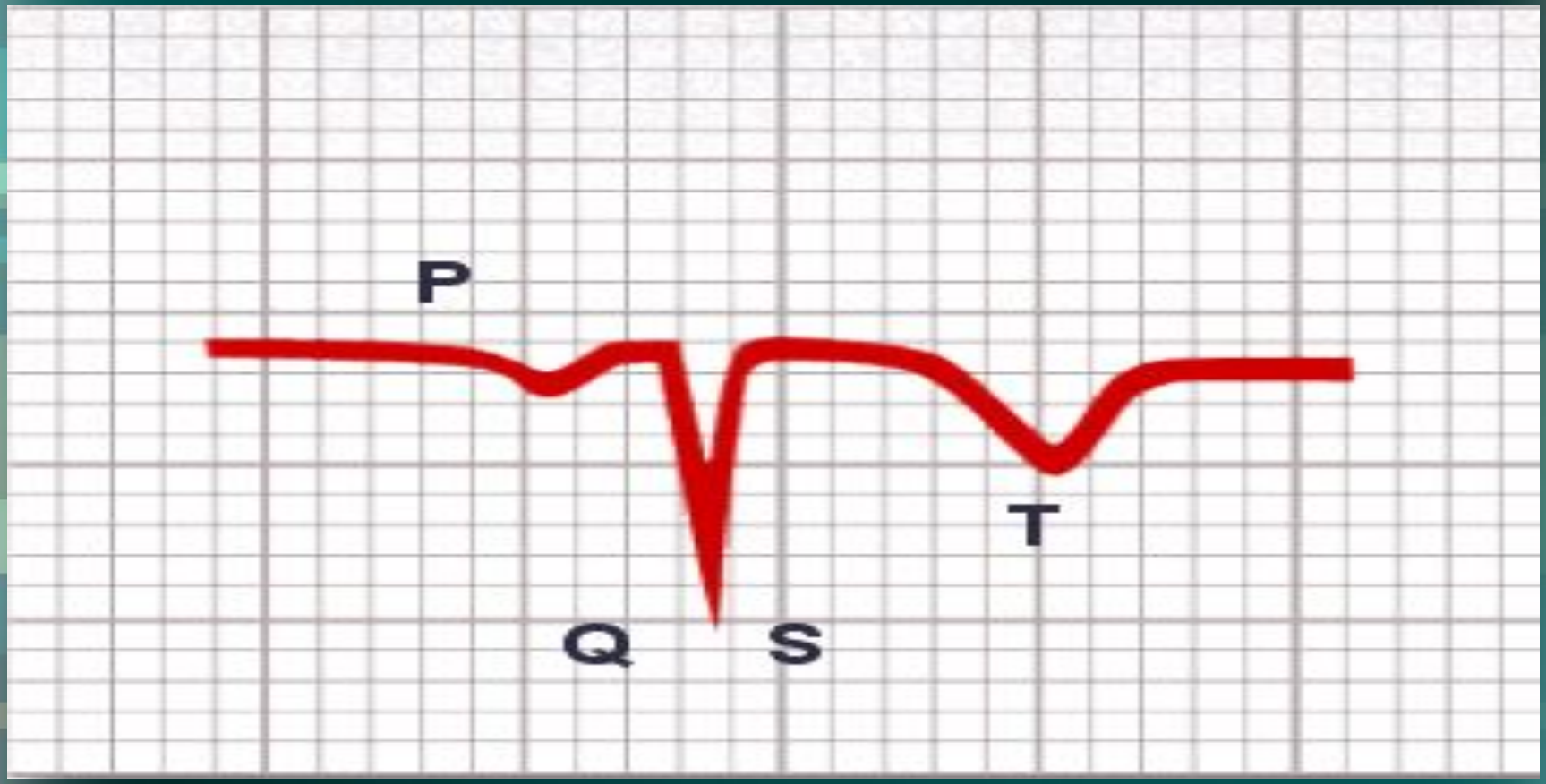
RULE 3

The QRS complex should be dominantly upright in leads I and II

RULE 4

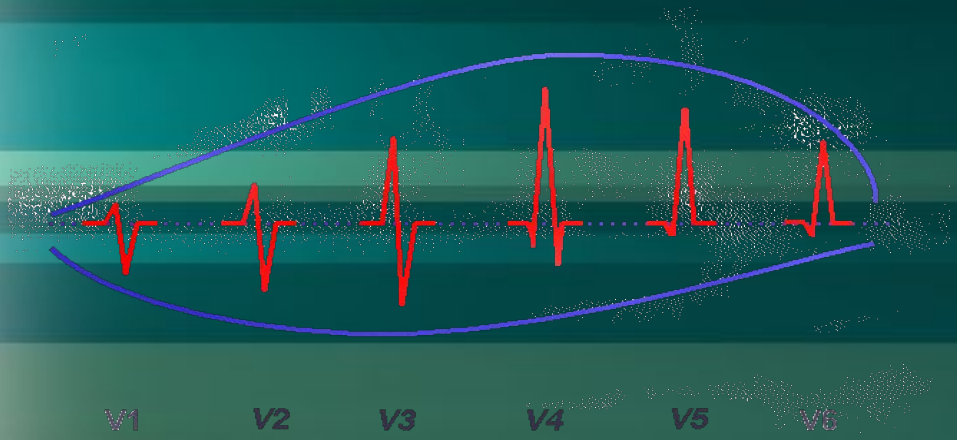
QRS and T waves tend to have the same general direction in the limb leads

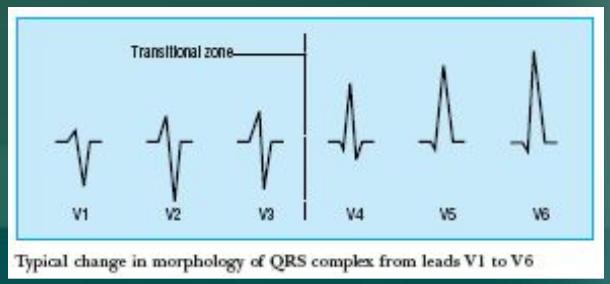
RULE 5



All waves are negative in lead aVR

RULE 6





Typical change in morphology of QRS complex from leads V1 to V6

The R wave must grow from V1 to at least V4

The S wave must grow from V1 to at least V3 and disappear in V6

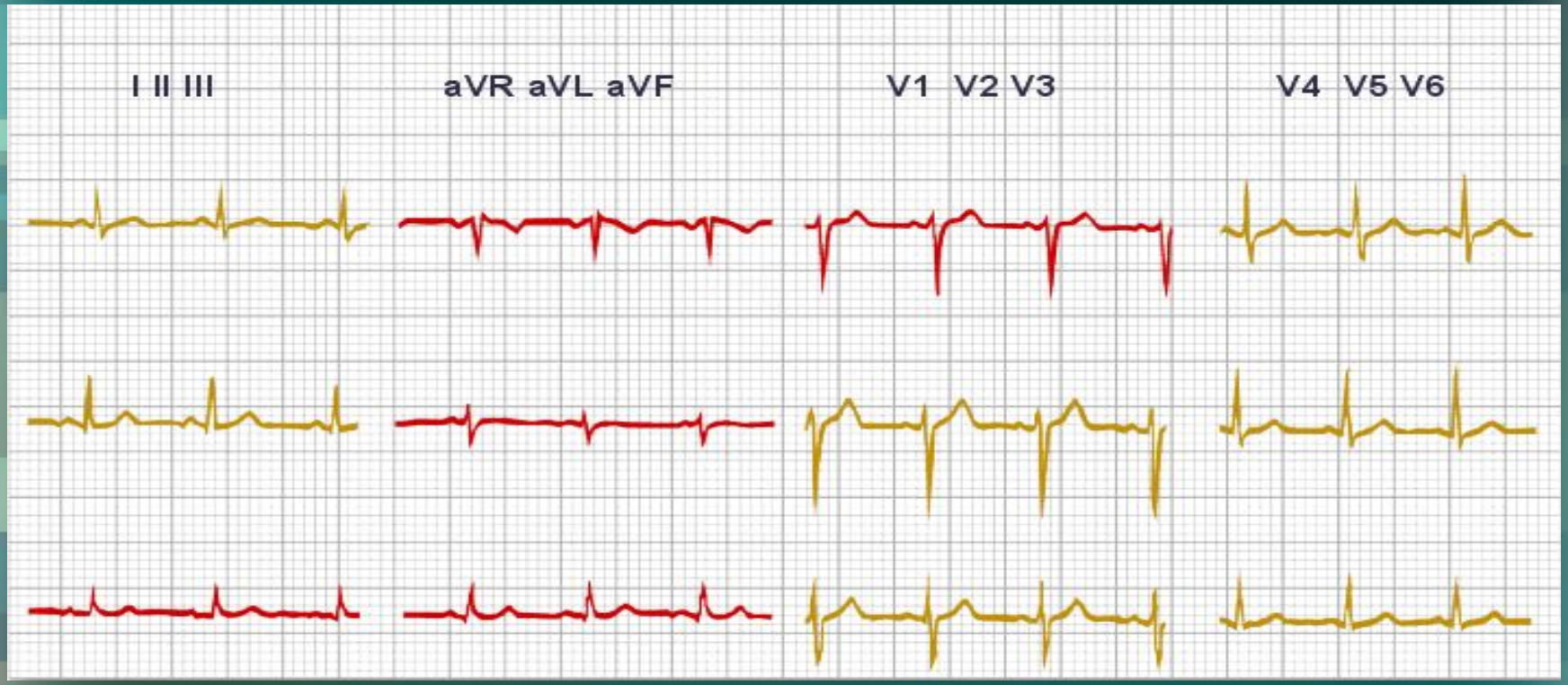
RULE 7

The ST segment should start isoelectric except in V1 and V2 where it may be elevated

RULE 8

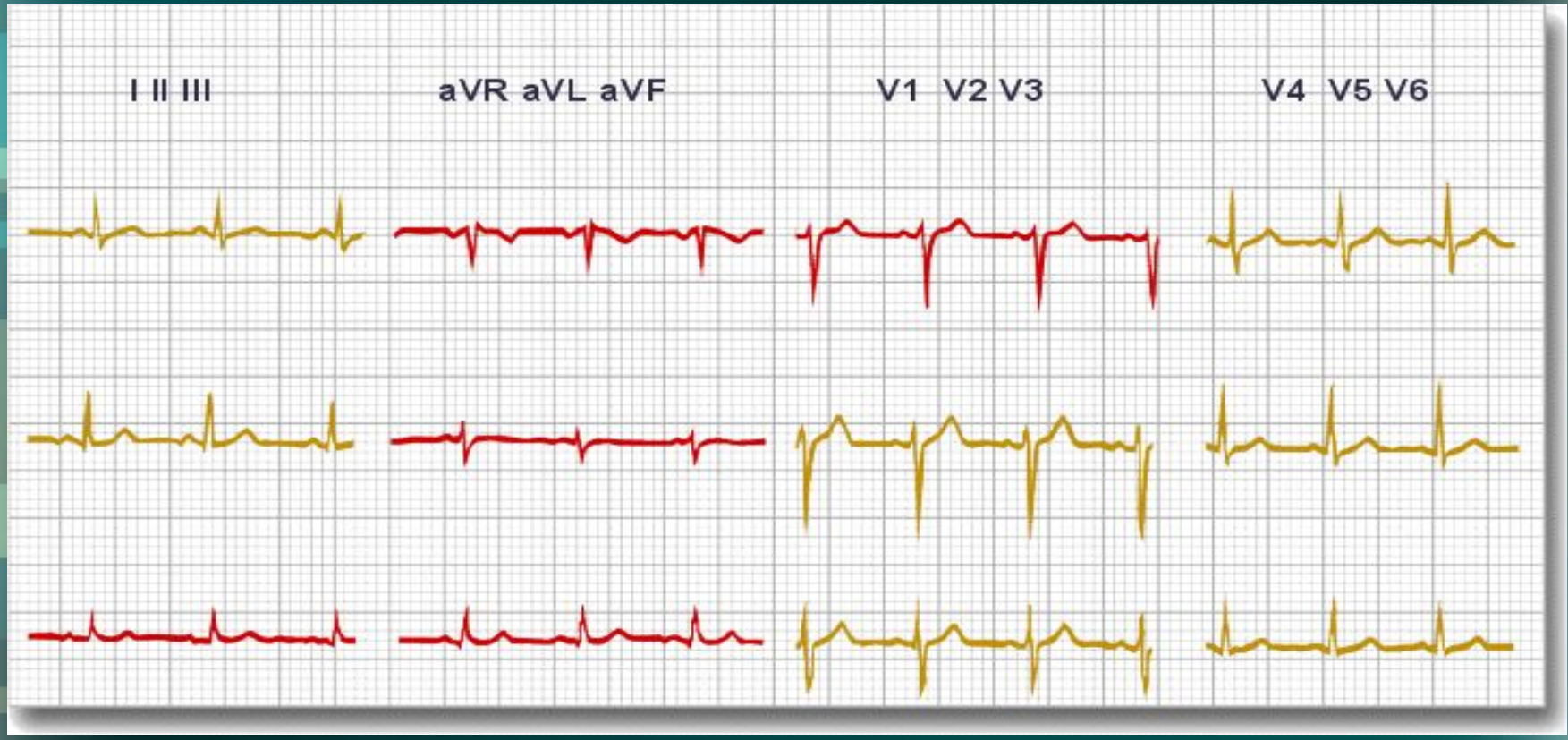
The P waves should be upright in I, II, and V2 to V6

RULE 9



There should be no Q wave or only a small q less than 0.04 seconds in width in I, II, V2 to V6

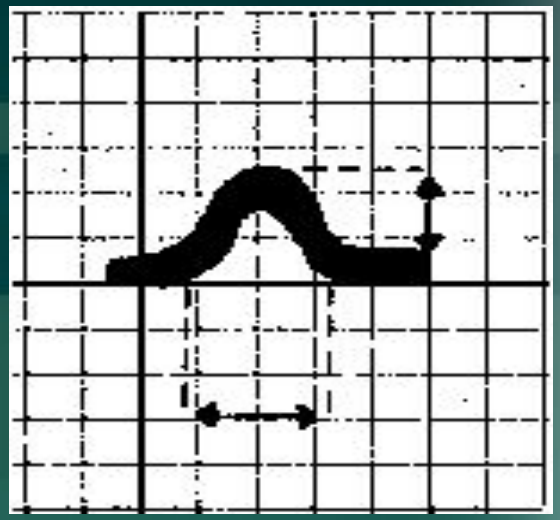
RULE 10



The T wave must be upright in I, II, V2 to V6

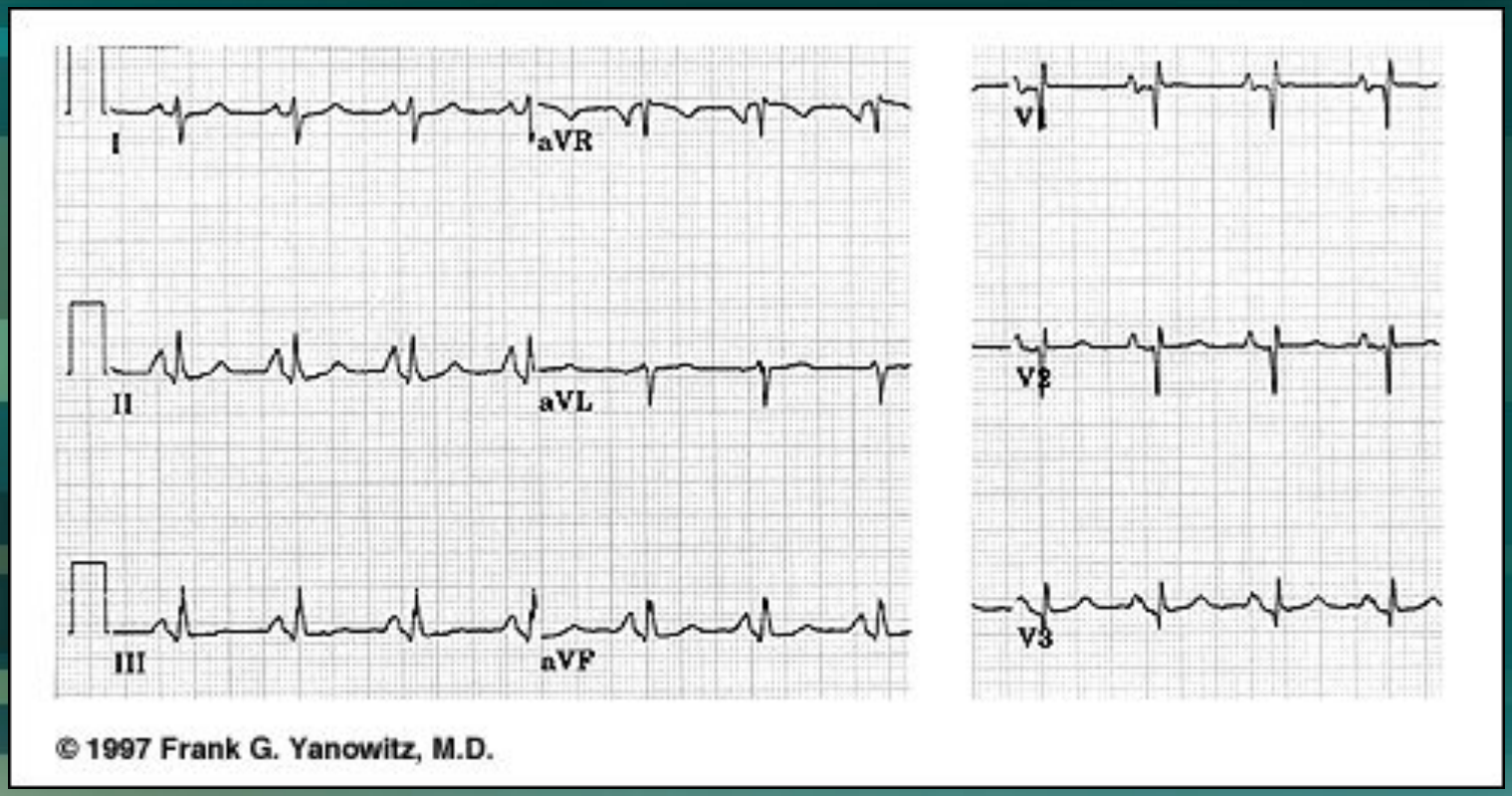
P wave

- Always positive in lead I and II
- Always negative in lead aVR
- < 3 small squares in duration
- < 2.5 small squares in amplitude
- Commonly biphasic in lead V1
- Best seen in leads II



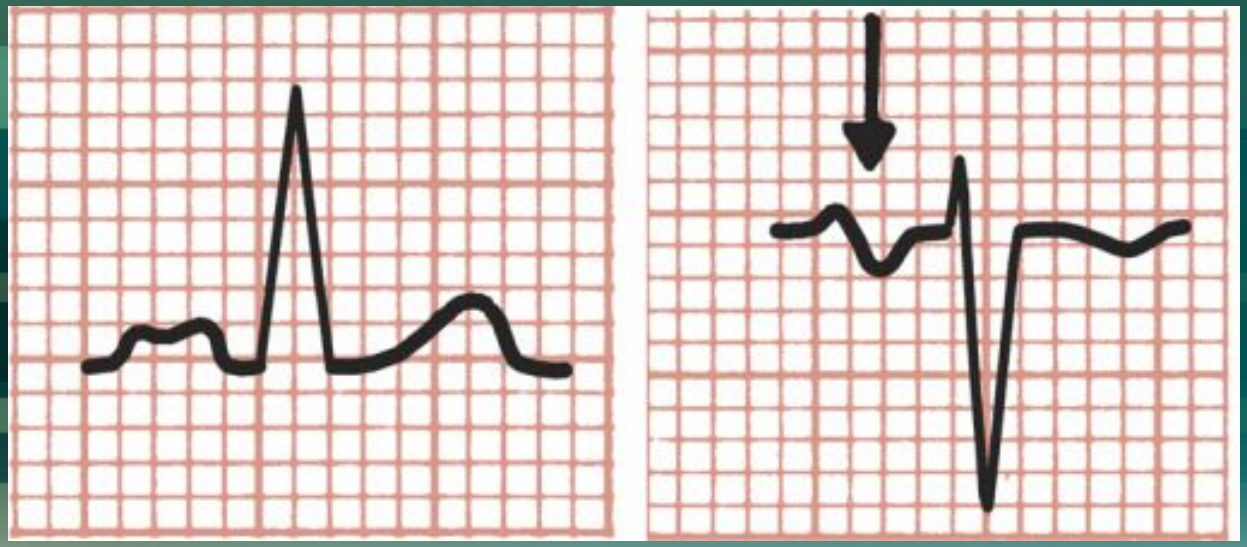
Right Atrial Enlargement

- Tall (> 2.5 mm), pointed P waves (P Pulmonale)



Left Atrial Enlargement

- Notched/bifid ('M' shaped) P wave (P 'mitrale') in limb leads



P Pulmonale

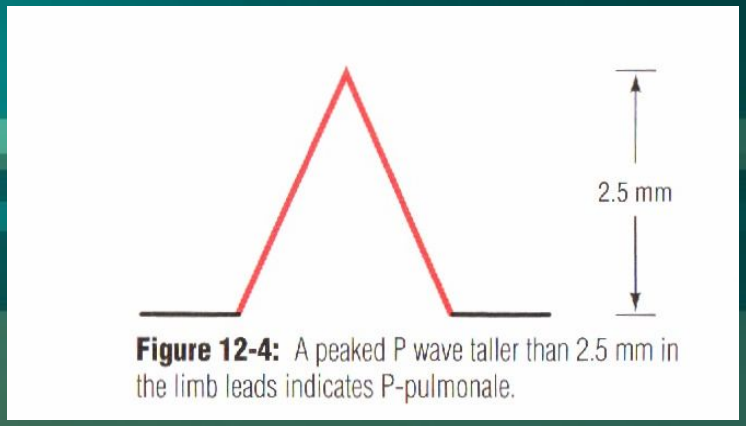
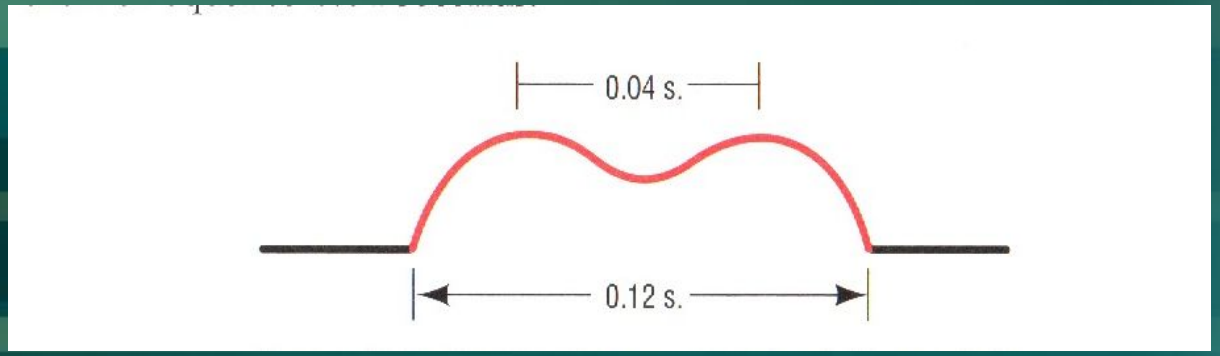
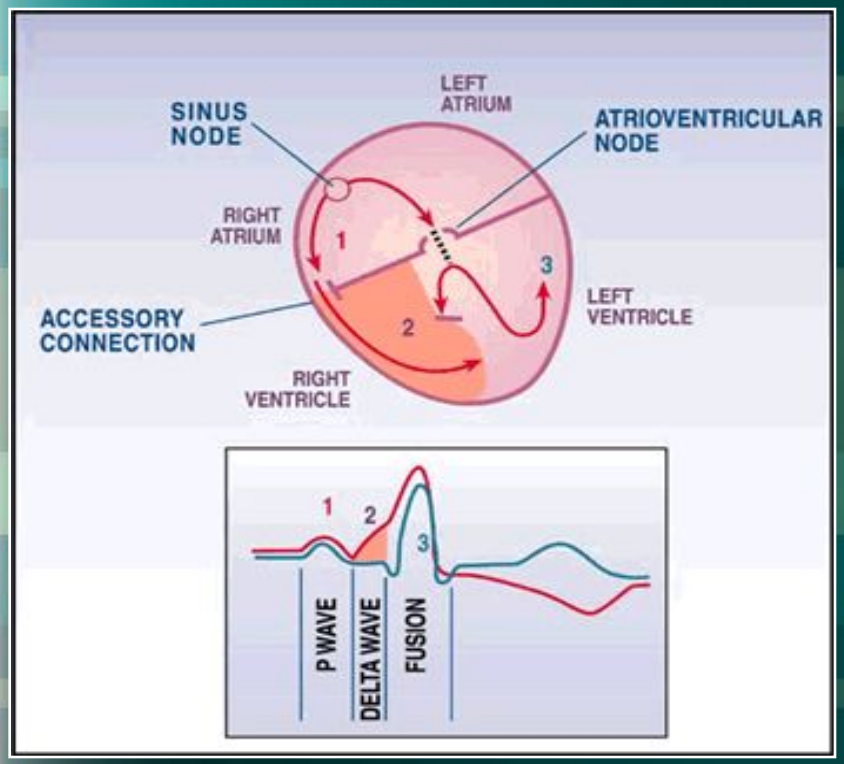


Figure 12-4: A peaked P wave taller than 2.5 mm in the limb leads indicates P-pulmonale.

P Mitrale



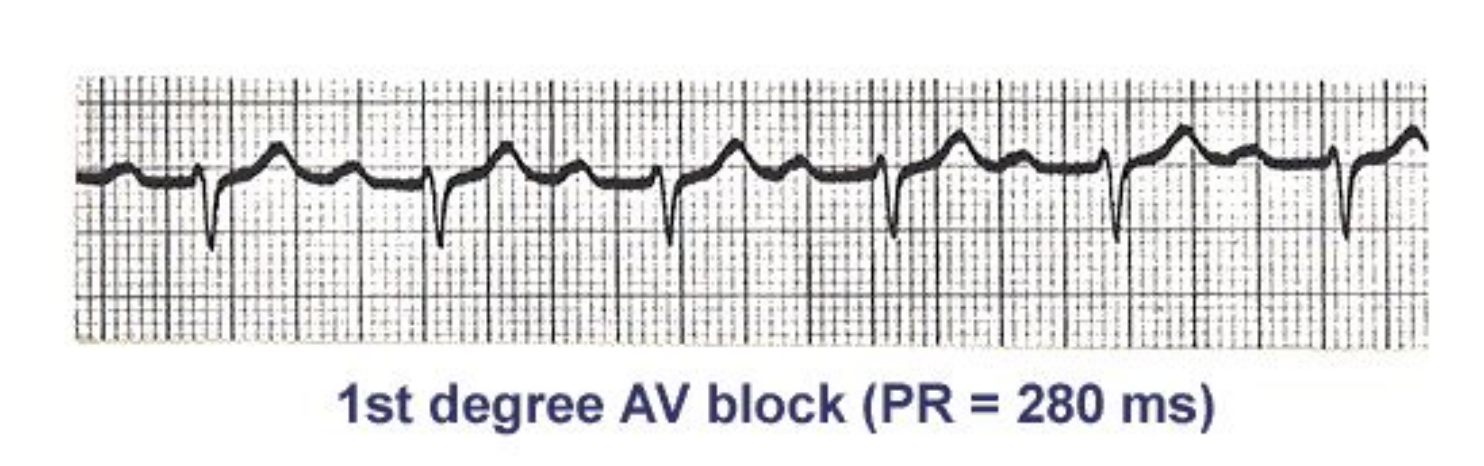
Short PR Interval



- WPW (Wolff-Parkinson-White) Syndrome
- Accessory pathway (Bundle of Kent) allows early activation of the ventricle (delta wave and short PR interval)

Long PR Interval

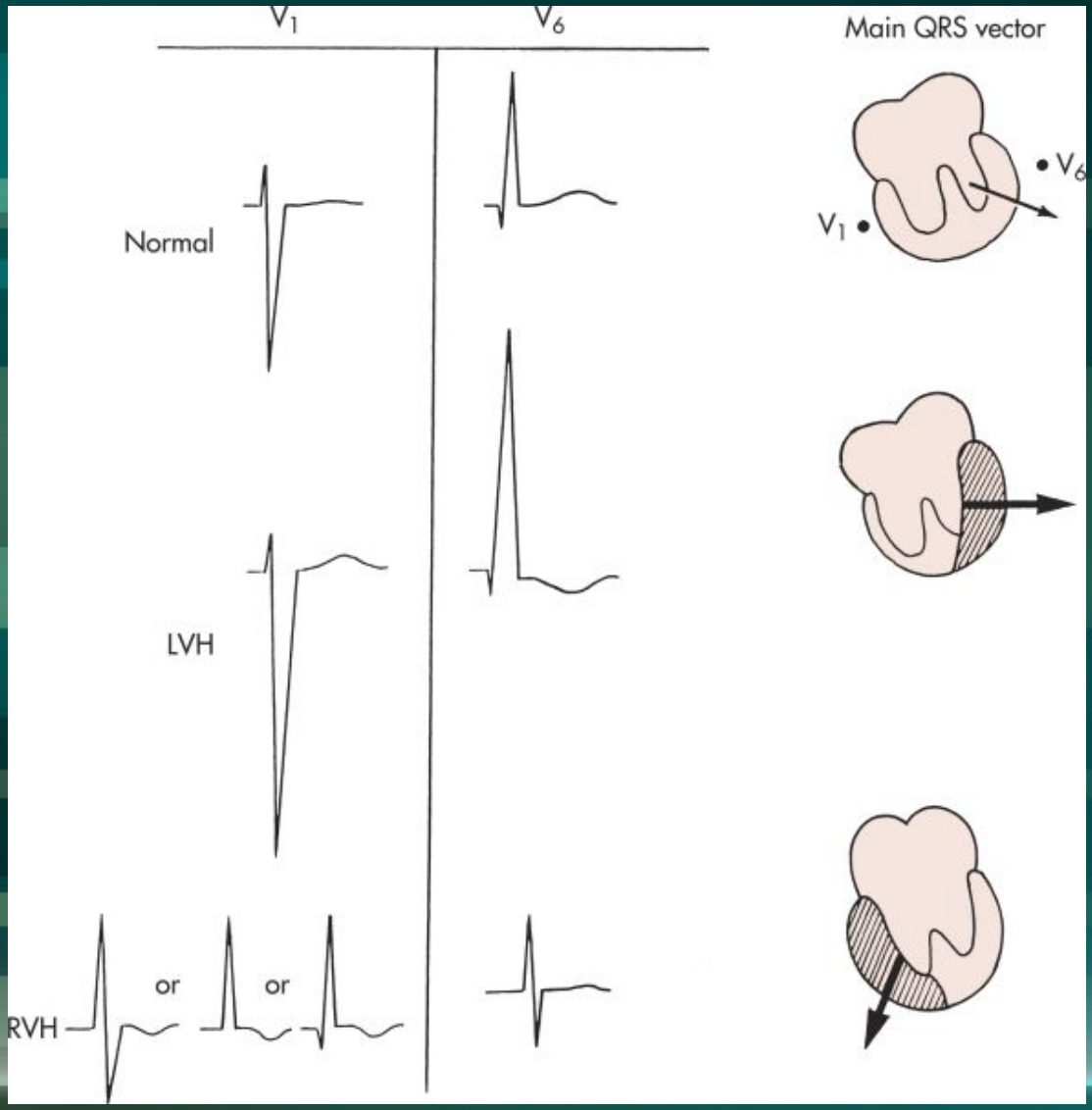
- First degree Heart Block



QRS Complexes

- Nonpathological Q waves may present in I, III, aVL, V5, and V6
- R wave in lead V6 is smaller than V5
- Depth of the S wave, should not exceed 30 mm
- Pathological Q wave > 2mm deep and > 1mm wide or > 25% amplitude of the subsequent R wave

QRS in LVH & RVH



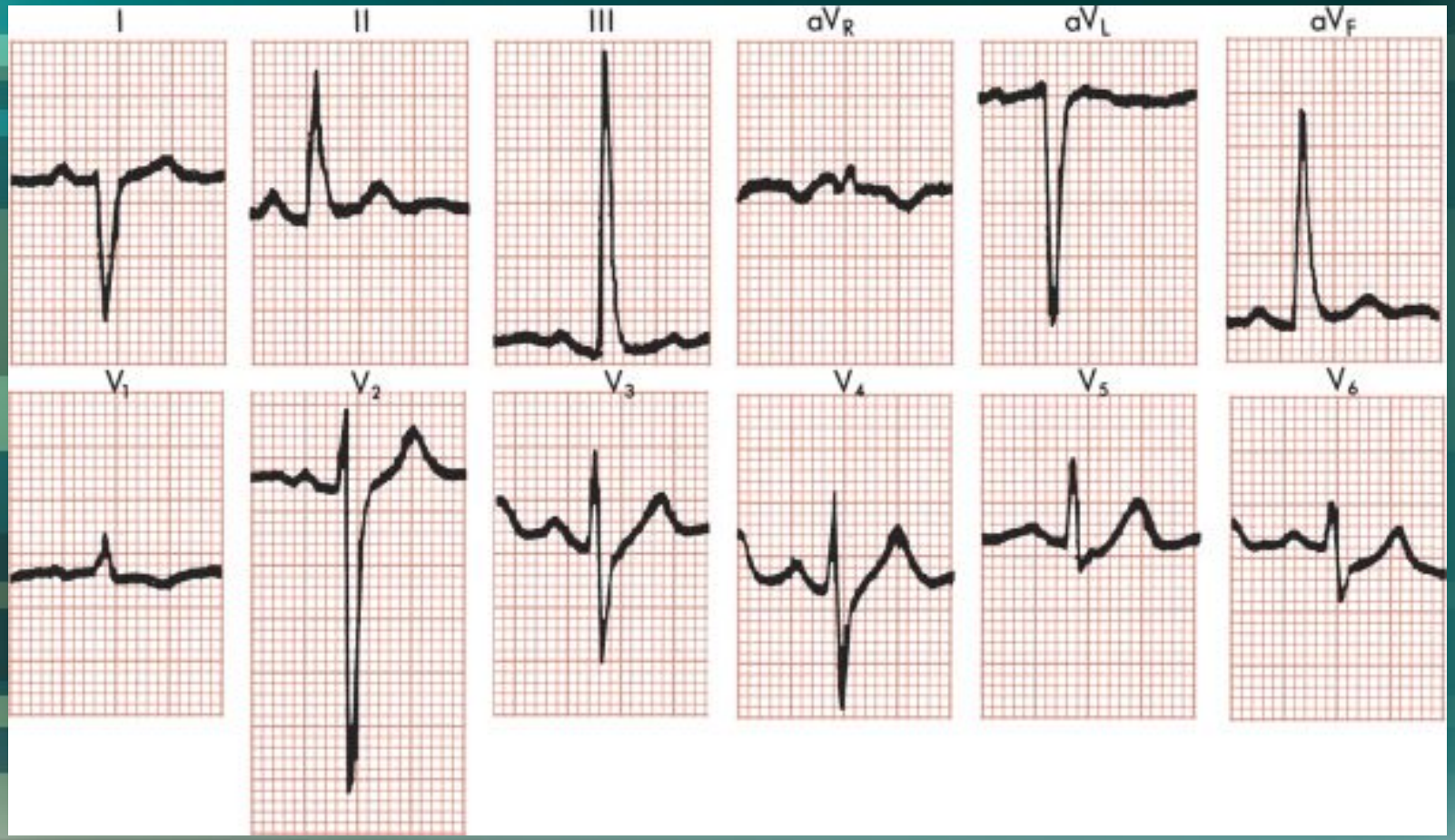
Conditions with Tall R in V1

Conditions associated with tall R wave in lead V1

- Right ventricular hypertrophy
- Posterior myocardial infarction
- Type A Wolff-Parkinson-White syndrome
- Right bundle branch block

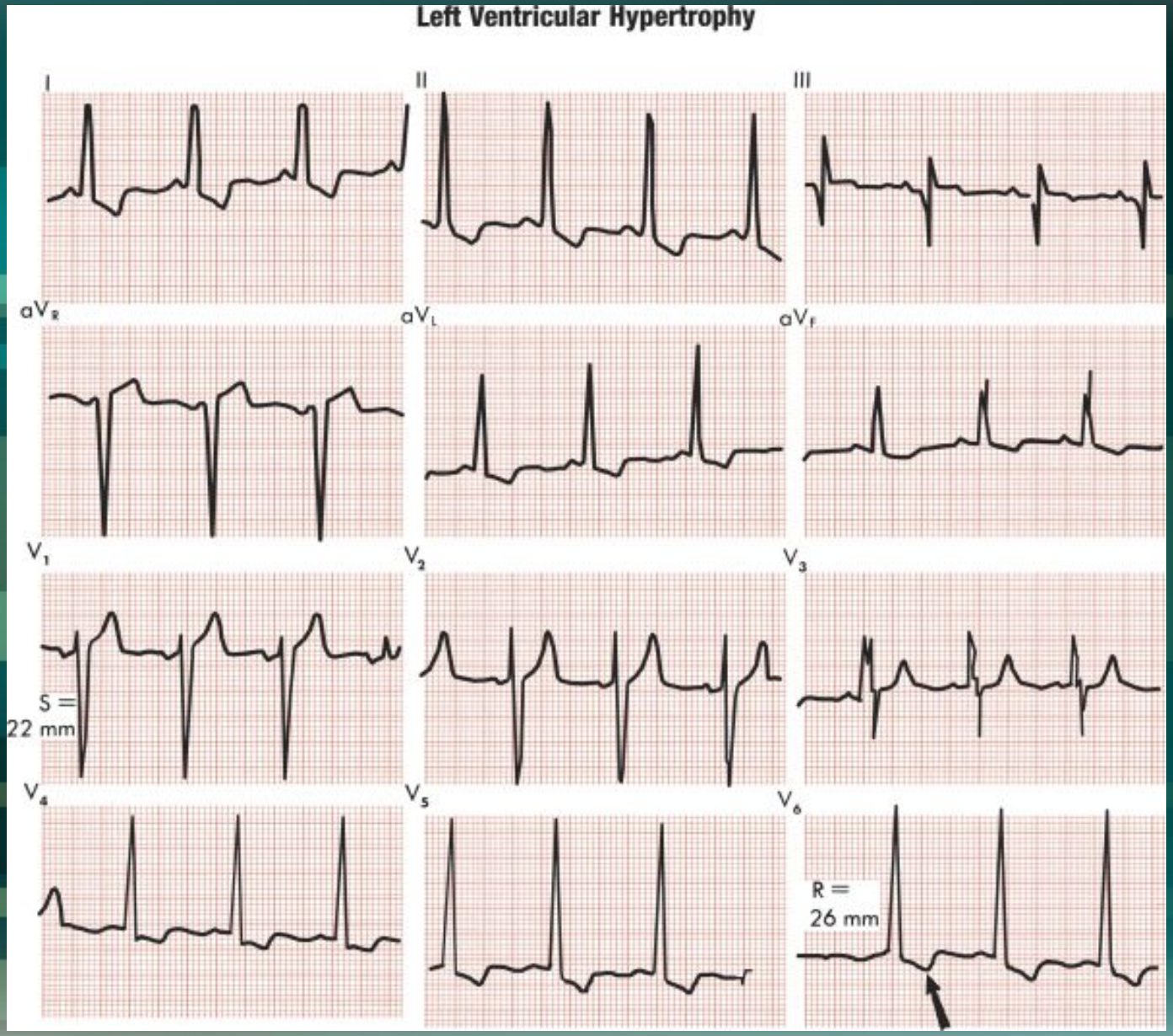
A tall R wave in lead V1 is normal in children and young adults

Right Atrial and Ventricular Hypertrophy



Left Ventricular Hypertrophy

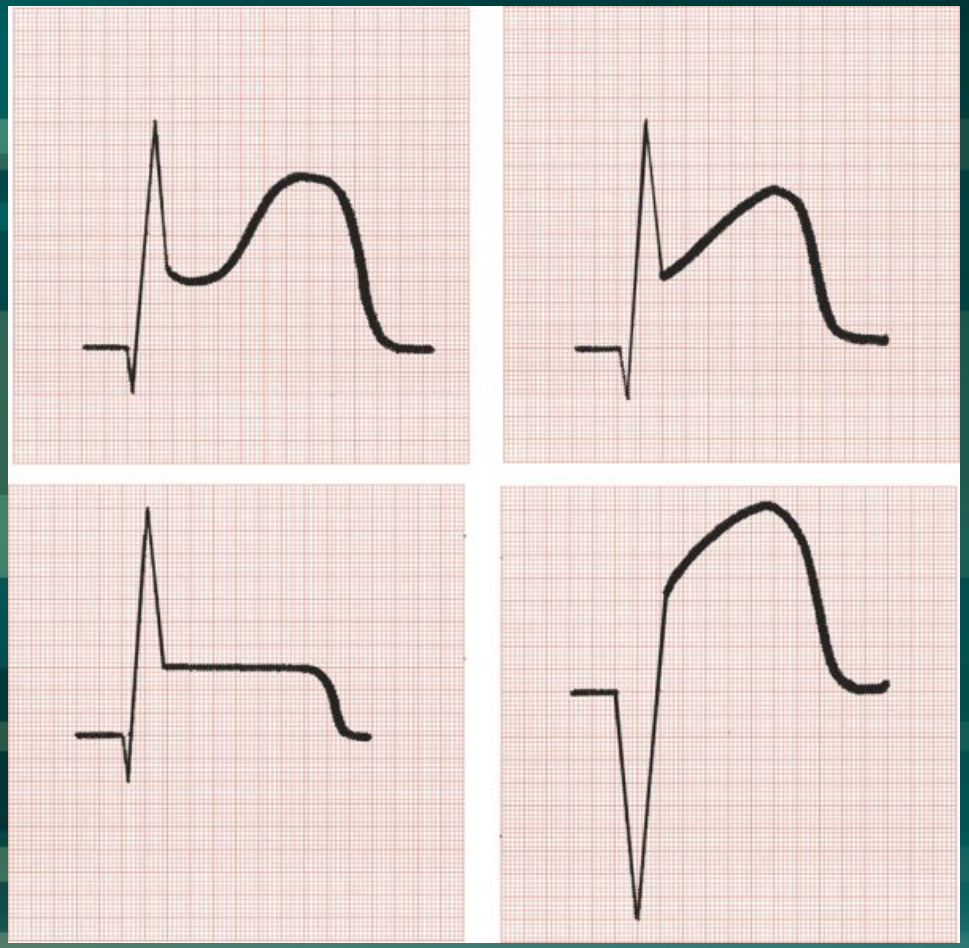
- Sokolow & Lyon Criteria
- S in V1+ R in V5 or V6 > 35 mm
- An R wave of 11 to 13 mm (1.1 to 1.3 mV) or more in lead aVL is another sign of LVH


Left Ventricular Hypertrophy
I
II
III
aVR
aVL
aVF
V1
V2
V3
S =
22 mm
V4
V5
V6
R =
26 mm

ST Segment

- ST Segment is flat (isoelectric)
- Elevation or depression of ST segment by 1 mm or more
- "J" (Junction) point is the point between QRS and ST segment

Variable Shapes Of ST Segment Elevations in AMI

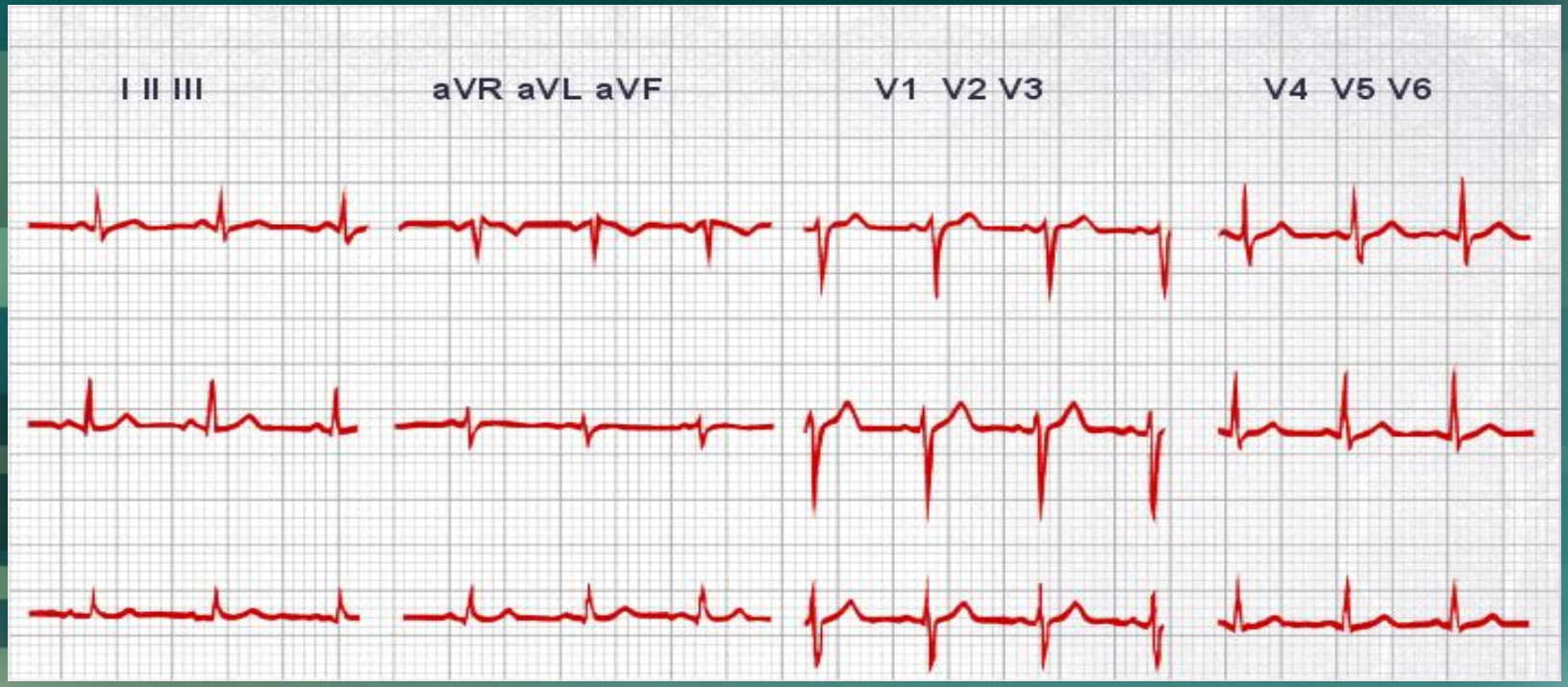


Goldberger AL. Goldberger: Clinical Electrocardiography: A Simplified Approach. 7th ed: Mosby Elsevier; 2006.

T wave

- Normal T wave is asymmetrical, first half having a gradual slope than the second
- Should be at least 1/8 but less than 2/3 of the amplitude of the R
- T wave amplitude rarely exceeds 10 mm
- Abnormal T waves are symmetrical, tall, peaked, biphasic or inverted.
- T wave follows the direction of the QRS deflection.

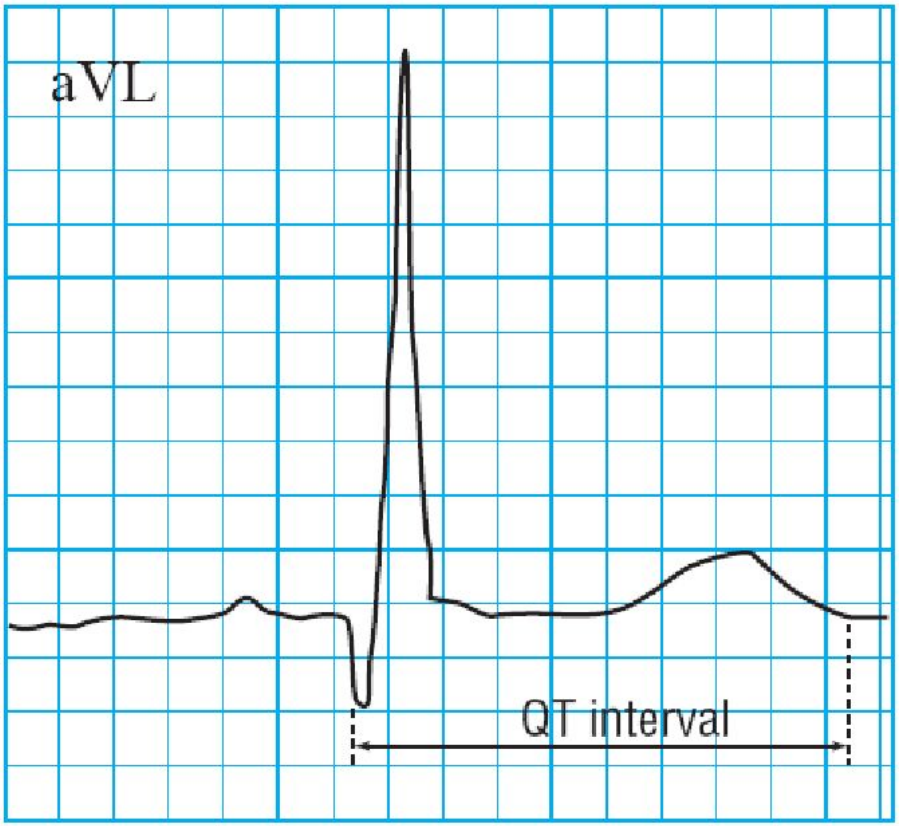
T wave



QT interval

1. Total duration of Depolarization and Repolarization
2. QT interval decreases when heart rate increases
3. For HR = 70 bpm, QT<0.40 sec.
4. QT interval should be 0.35 0.45 s,
5. Should not be more than half of the interval between adjacent R waves (RR interval).

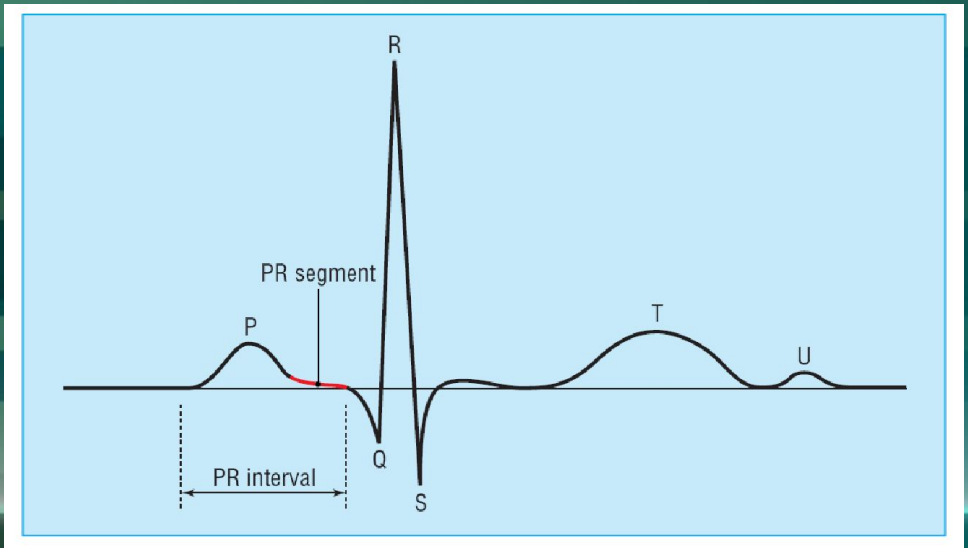
QT Interval



The QT interval is measured in lead aVL as this lead does not have prominent U waves (diagram is scaled up)

U wave

- U wave related to afterdepolarizations which follow repolarization
- U waves are small, round, symmetrical and positive in lead II, with amplitude < 2 mm
- U wave direction is the same as T wave
- More prominent at slow heart rates



Determining the Heart Rate

- Rule of 300/1500
- 10 Second Rule

Rule of 300

Count the number of "big boxes" between two QRS complexes, and divide this into 300. (smaller boxes with 1500)

for regular rhythms.

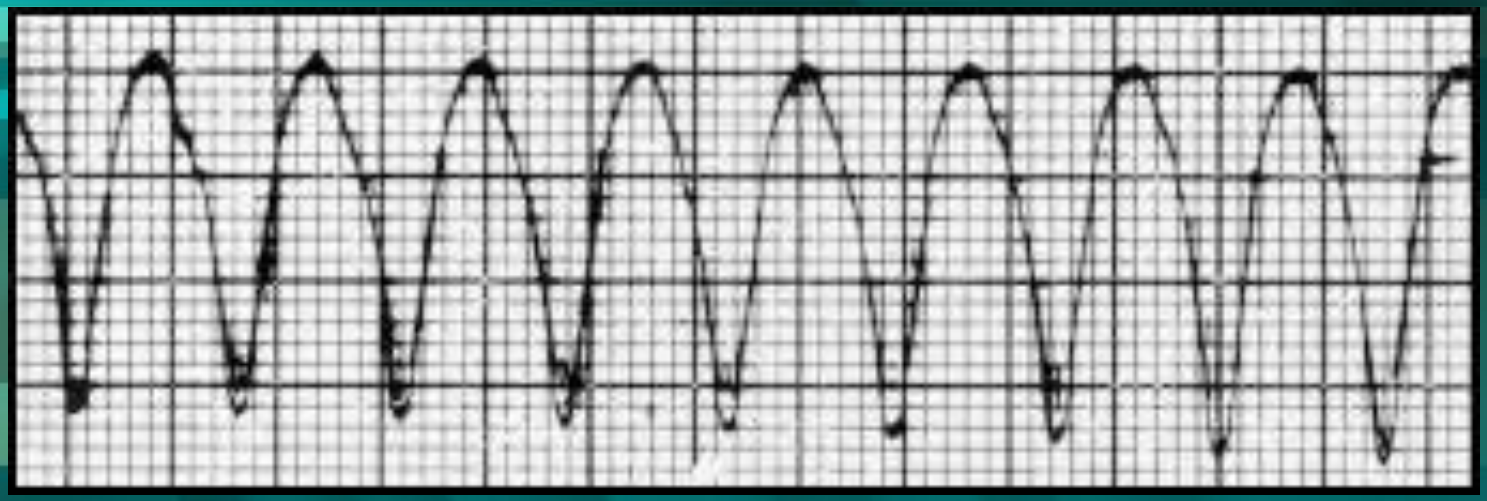
What is the heart rate?

(300 / 6) = 50 bpm

What is the heart rate?

(300 / ~ 4) = ~ 75 bpm

What is the heart rate?



(300 / 1.5) = 200 bpm

The Rule of 300

It may be easiest to memorize the following table:

No of big boxes	Rate
1	300
2	150
3	100
4	75
5	60
6	50

10 Second Rule

EKGs record 10 seconds of rhythm per page,

Count the number of beats present on the EKG

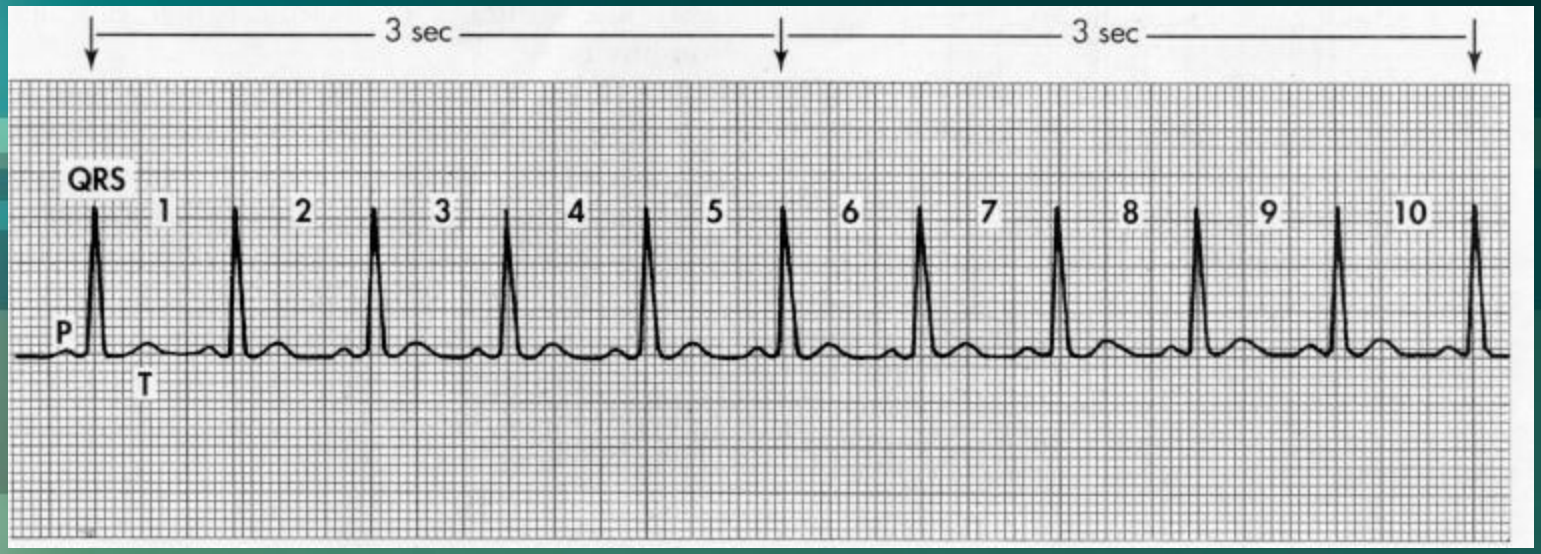
Multiply by 6

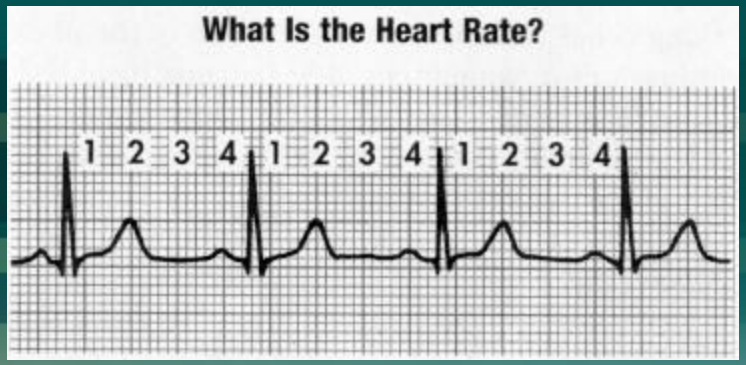
For irregular rhythms.

What is the heart rate?

33 x 6 = 198 bpm

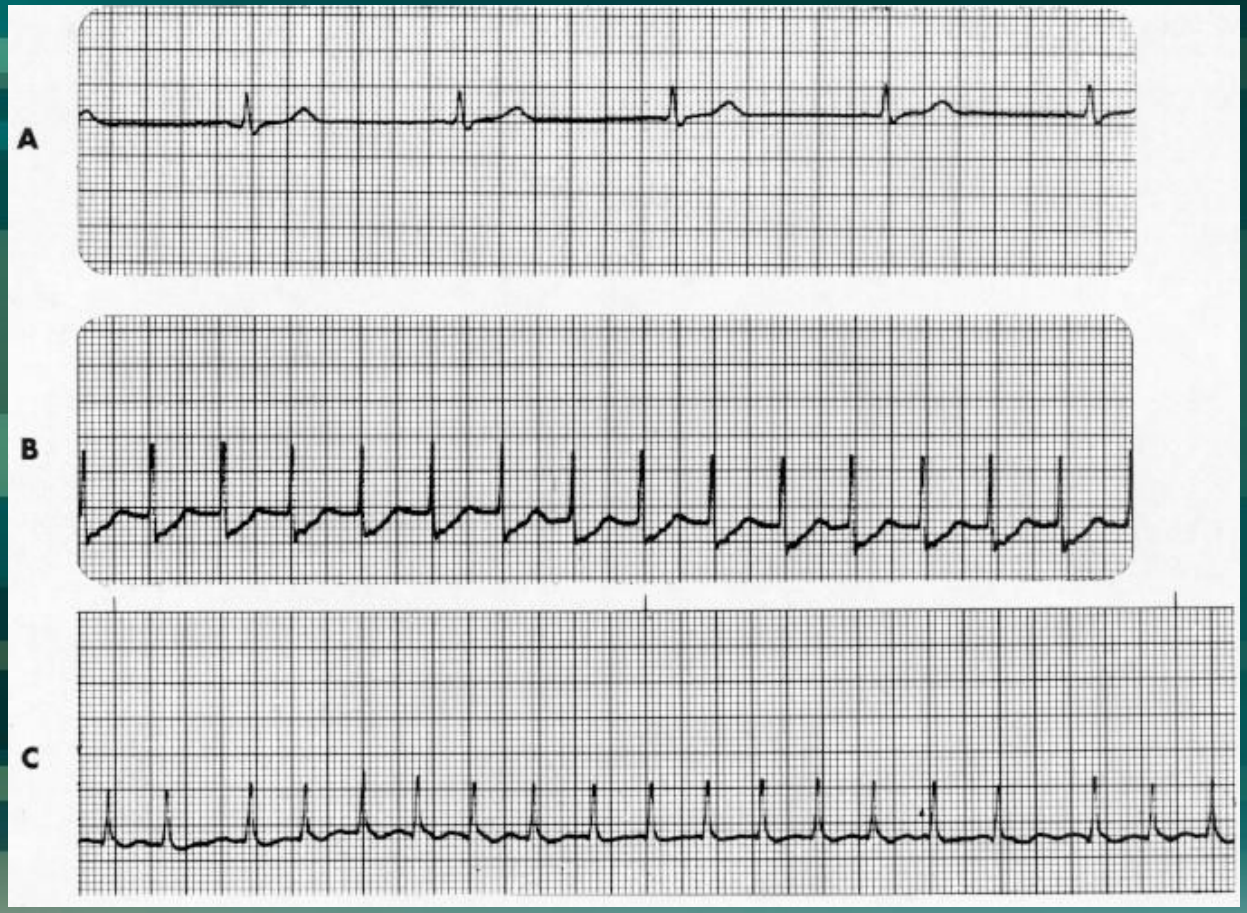
Calculation of Heart Rate





Question

- Calculate the heart rate



The QRS Axis

The QRS axis represents overall direction of the heart's electrical activity.

Abnormalities hint at:

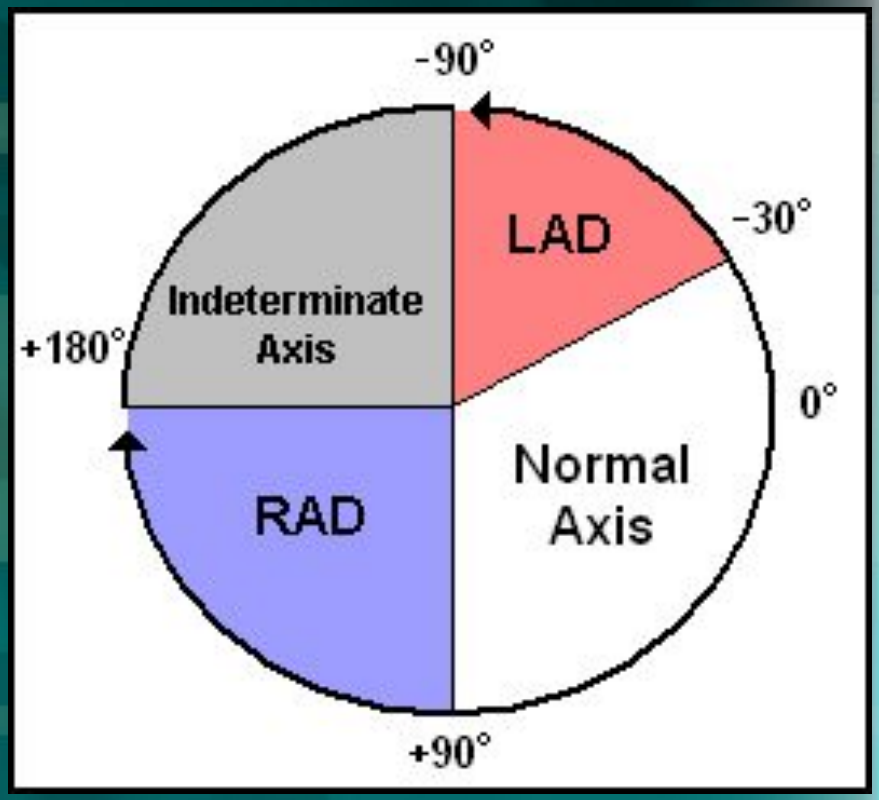
- Ventricular enlargement
- Conduction blocks (i.e. hemiblocks)

The QRS Axis

Normal QRS axis from -30° to +90°.

-30° to -90° is referred to as a left axis deviation (LAD)

+90° to +180° is referred to as a right axis deviation (RAD)



Determining the Axis

The Quadrant Approach

The Equiphasic Approach

Determining the Axis

Predominantly Positive

Predominantly Negative

Equiphasic

The Quadrant Approach

1. QRS complex in leads I and aVF
2. determine if they are predominantly positive or negative.
3. The combination should place the axis into one of the 4 quadrants below.

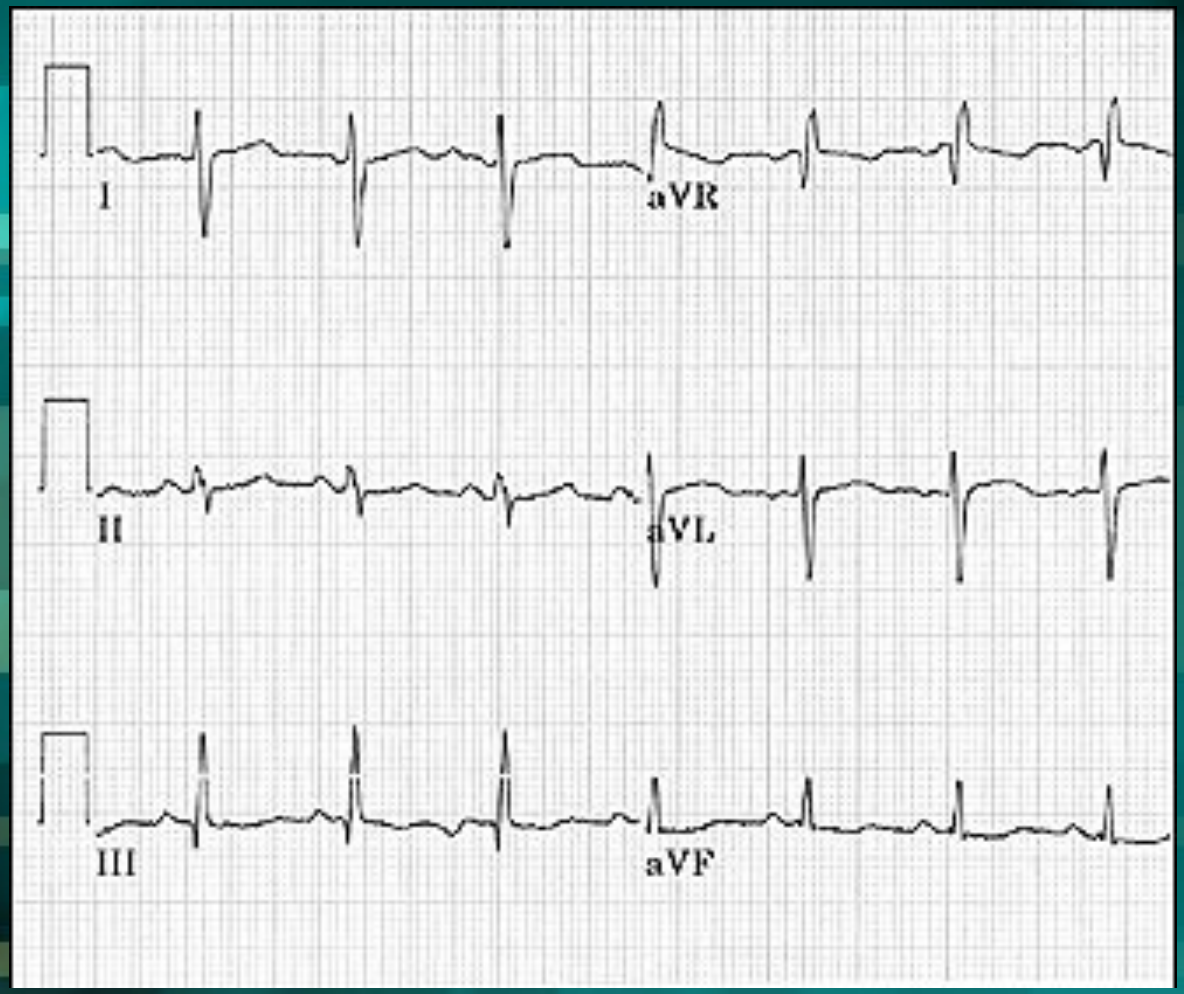
		Lead aVF: Positive	Lead aVF: Negative
Lead I	Positive	Normal Axis	LAD
	Negative	RAD	Indeterminate Axis

The Quadrant Approach

- When LAD is present,
- If the QRS in II is positive, the LAD is non-pathologic or the axis is normal
- If negative, it is pathologic.

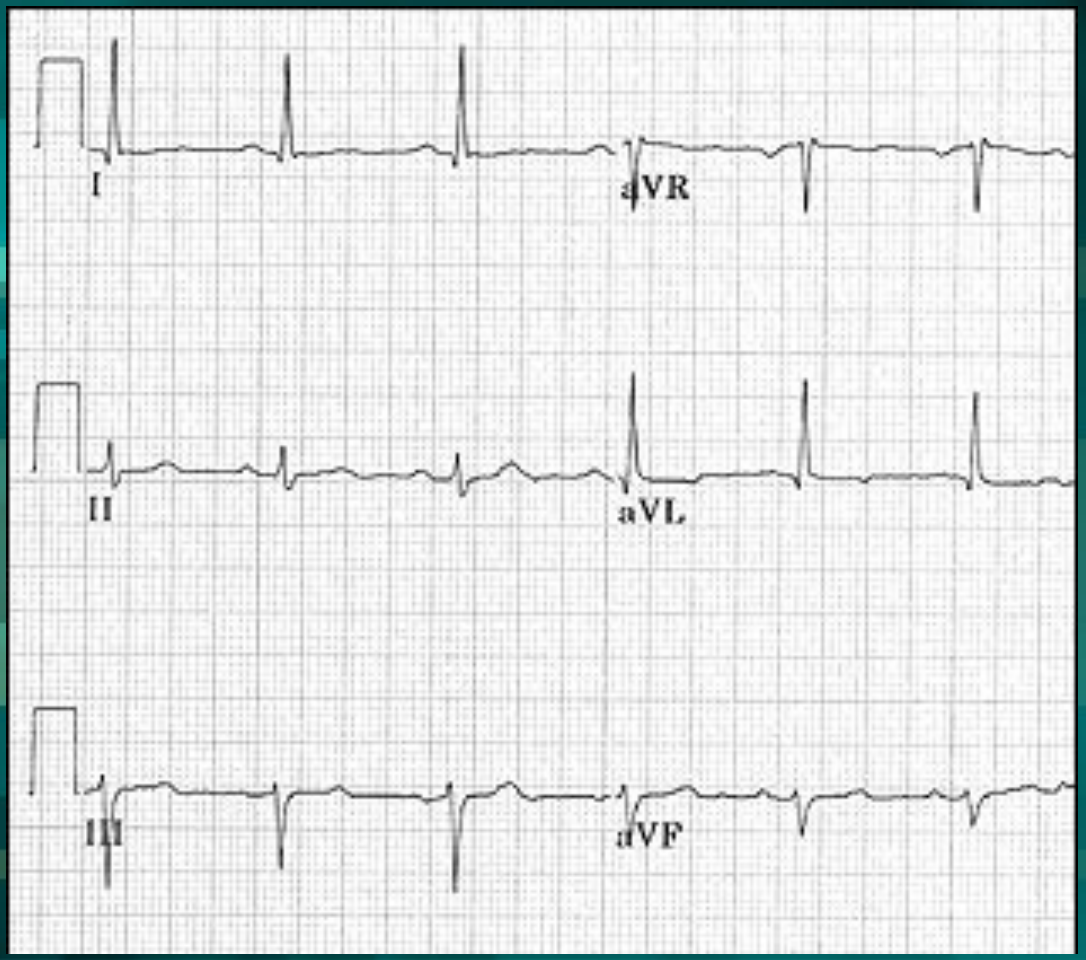
		Lead aVF: Positive	Lead aVF: Negative
Lead I	Positive	Normal Axis	LAD
	Negative	RAD	Indeterminate Axis

Quadrant Approach: Example 1



Negative in I, positive in aVF RAD

Quadrant Approach: Example 2

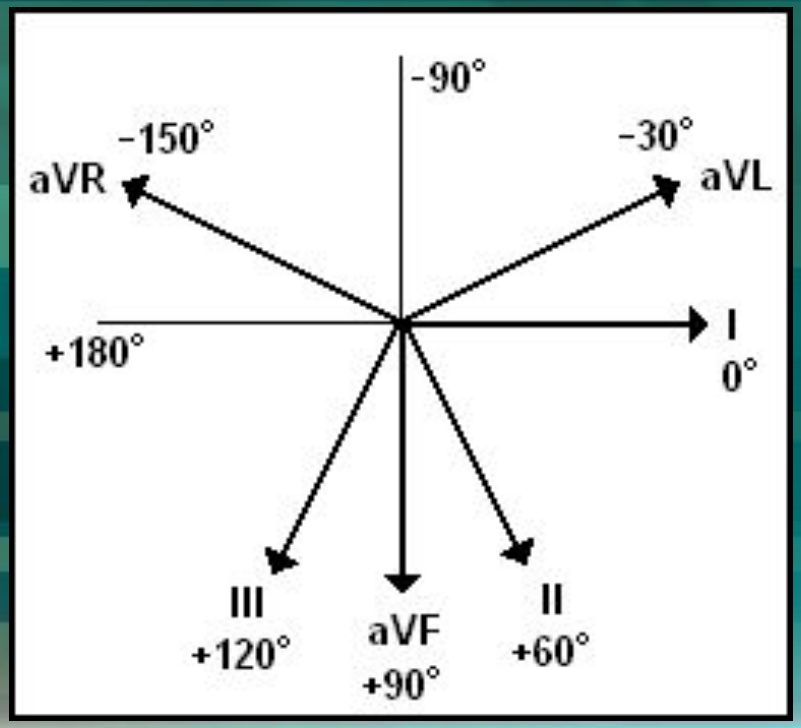


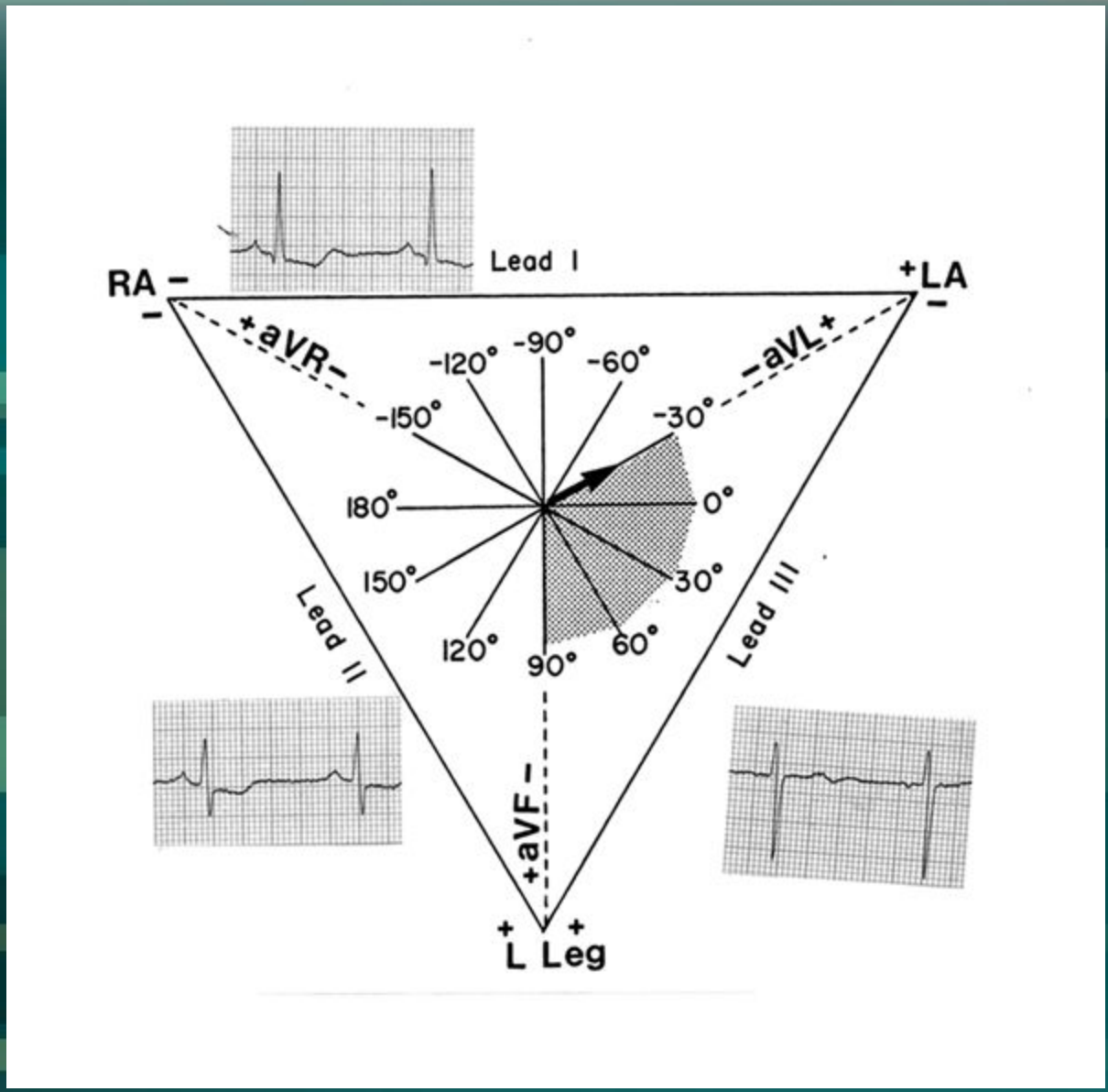
**Positive in I, negative in aVF Predominantly positive in II **

Normal Axis (non-pathologic LAD)

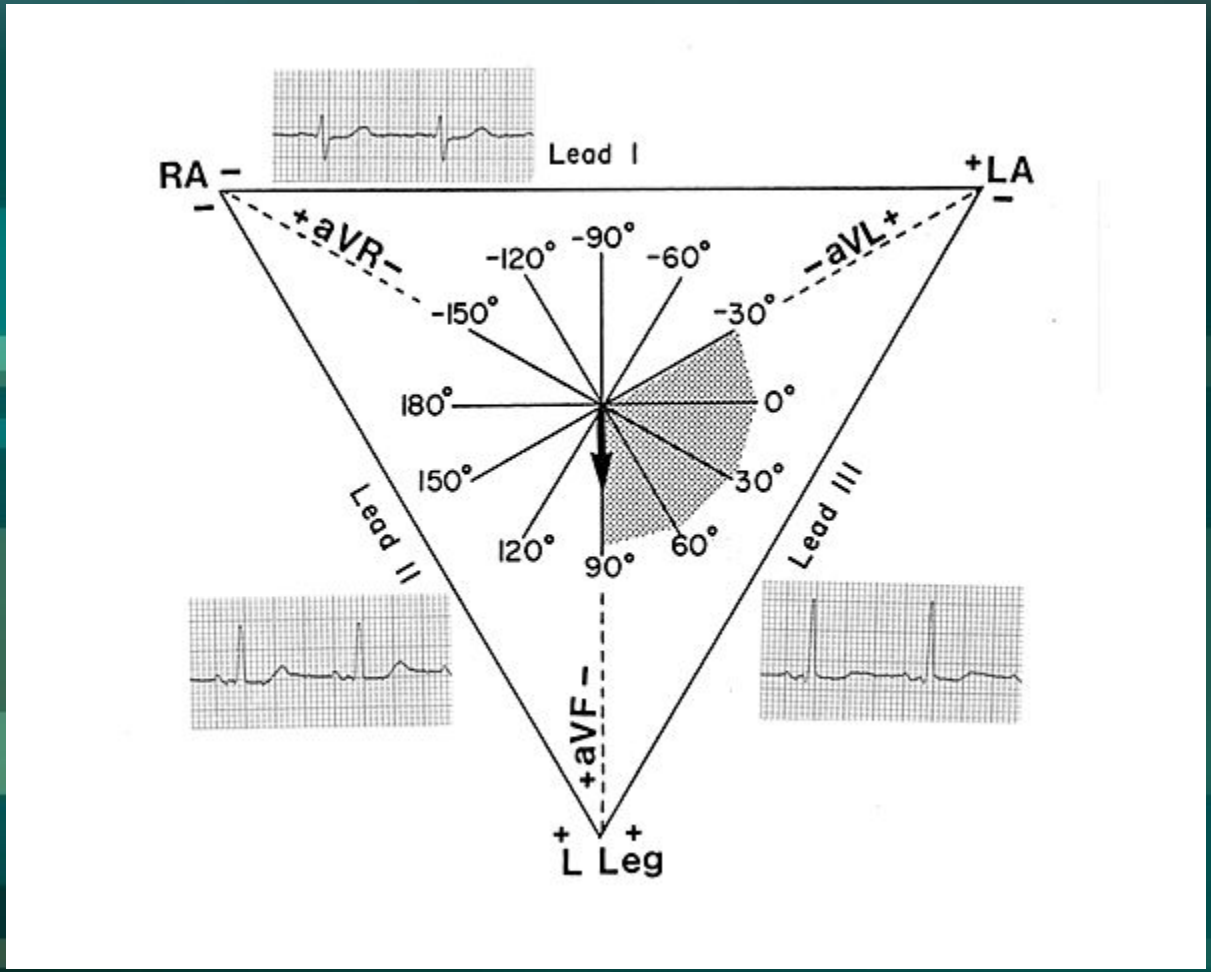
The Equiphasic Approach

1. Most equiphasic QRS complex.
2. Identified Lead lies 90° away from the lead
3. QRS in this second lead is positive or Negative

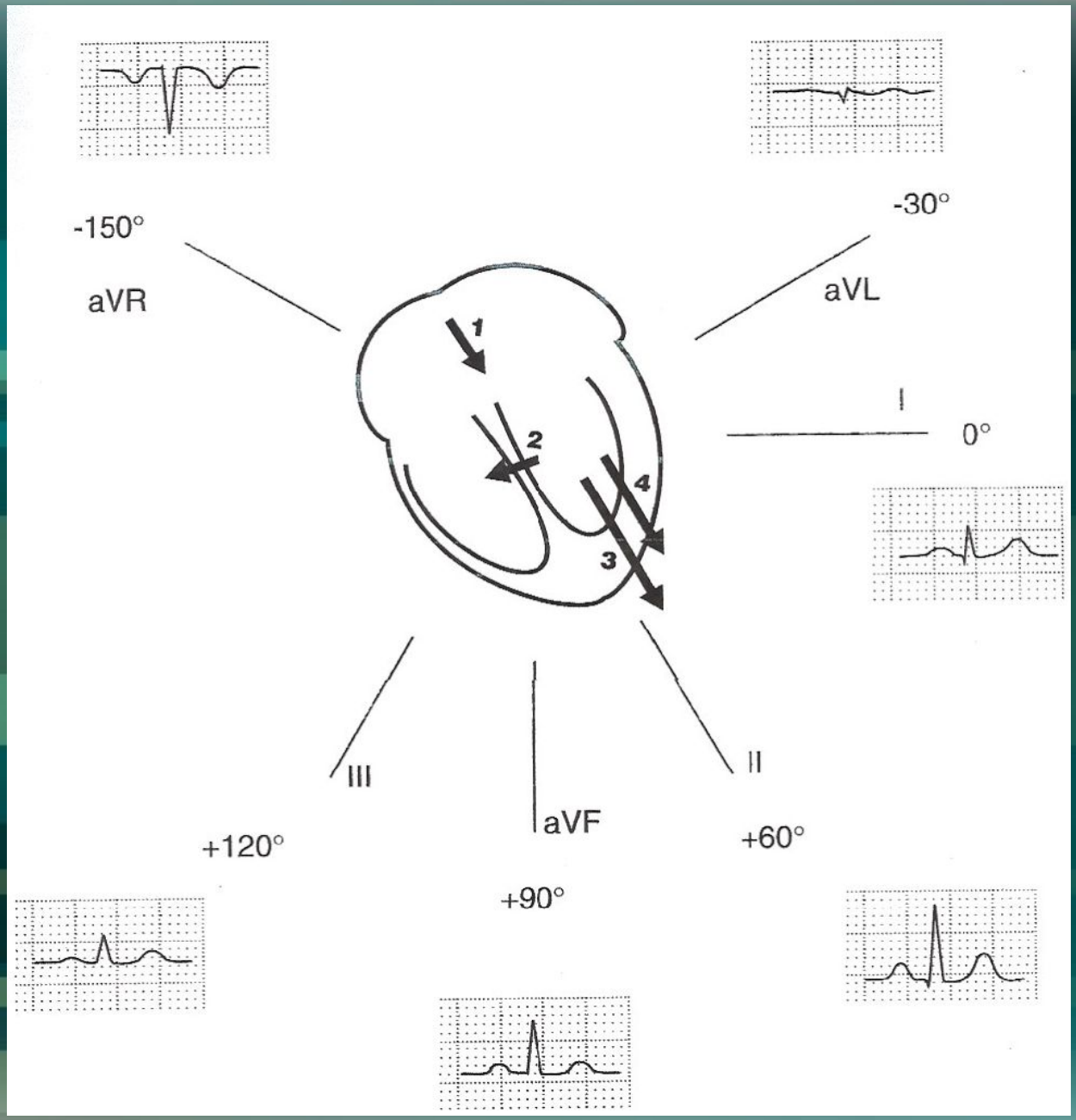




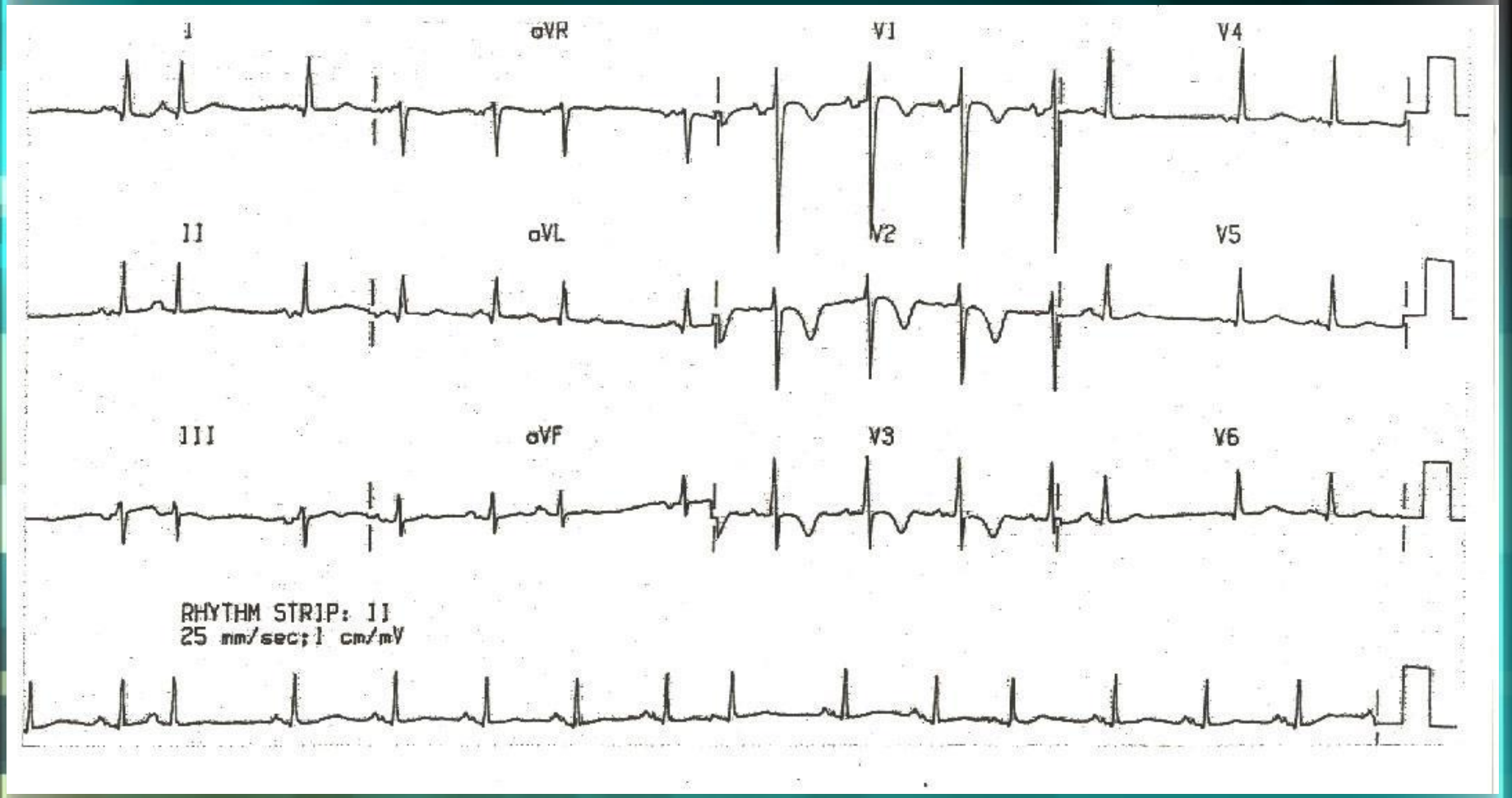
QRS Axis = -30 degrees



QRS Axis = +90 degrees-KH


-150°
aVR
-30°
aVL
I
0°
1
2
3
4
III
aVF
II
+120°
+90°
+60°

Equiphasic Approach



Equiphasic in aVF □ Predominantly positive in I □ QRS axis ≈ 0°

Thank You

BRADYARRYTHMIA

Dr Subroto Mandal, MD, DM, DC

Associate Professor, Cardiology

Classification

- Sinus Bradycardia
- Junctional Rhythm
- Sino Atrial Block
- Atrioventricular block

Impulse Conduction & the ECG

Sinoatrial node

↓

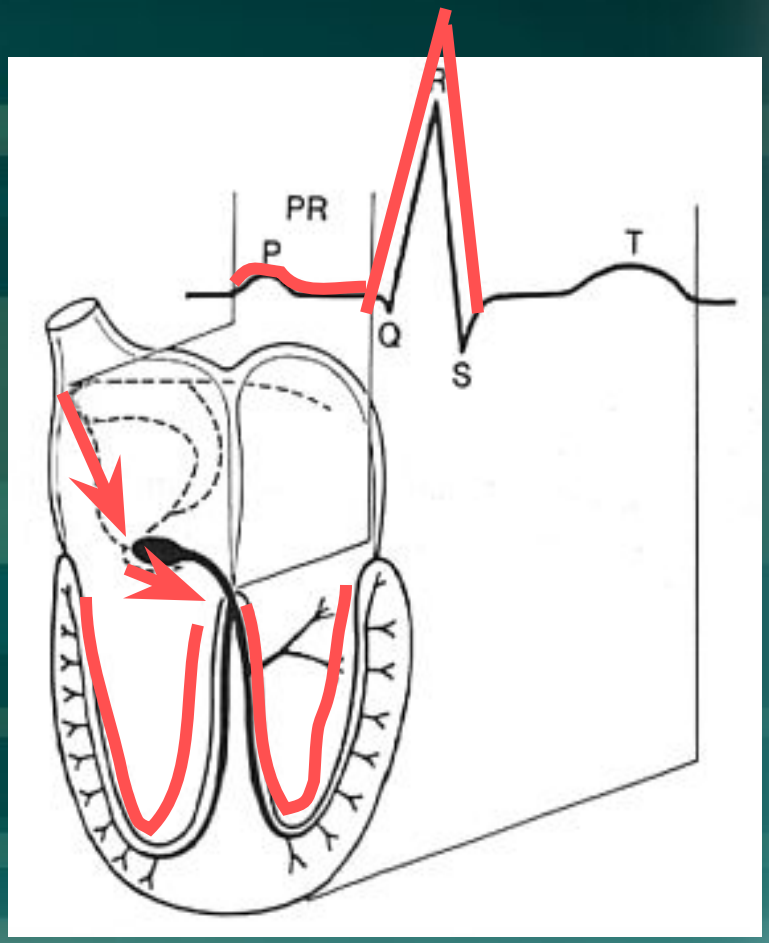
AV node

↓

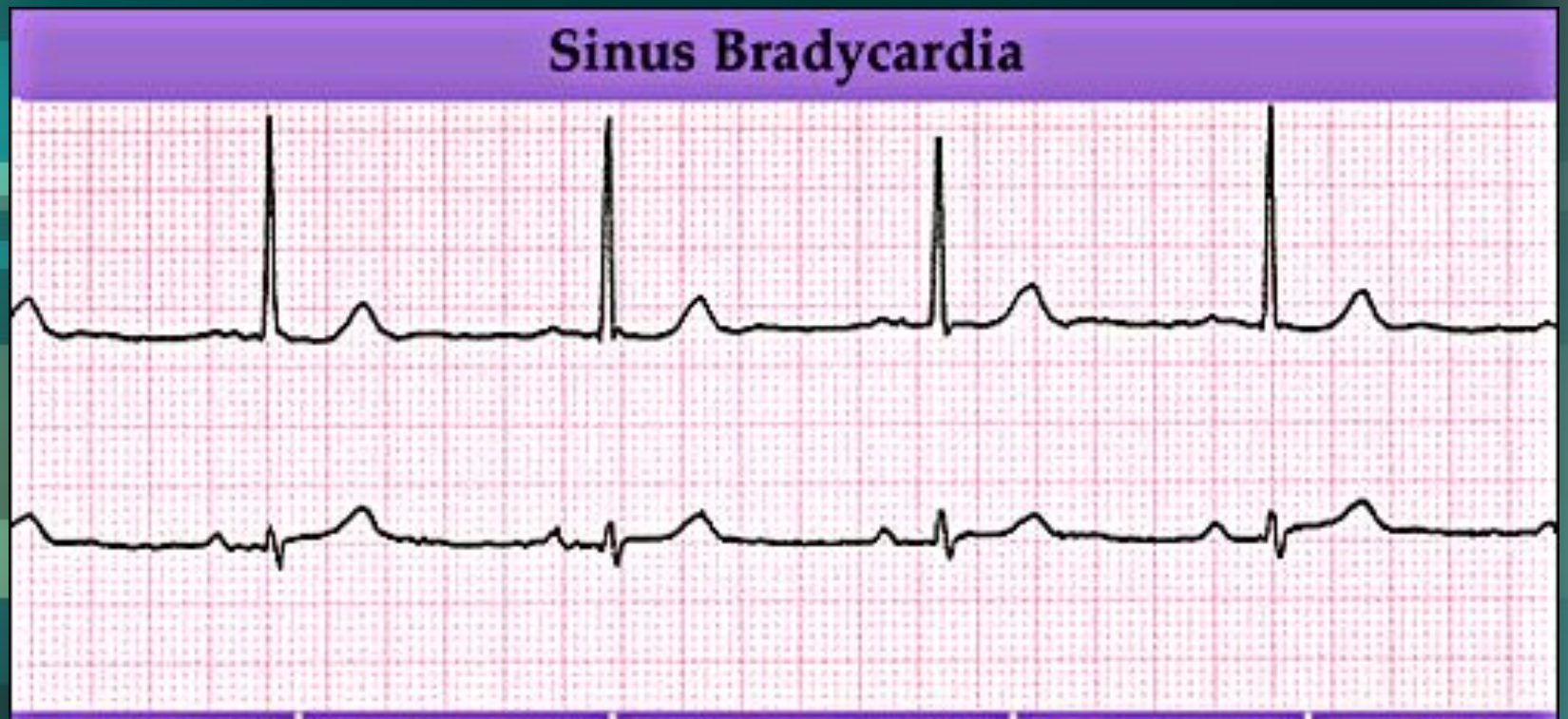
Bundle of His

↓

Bundle Branches



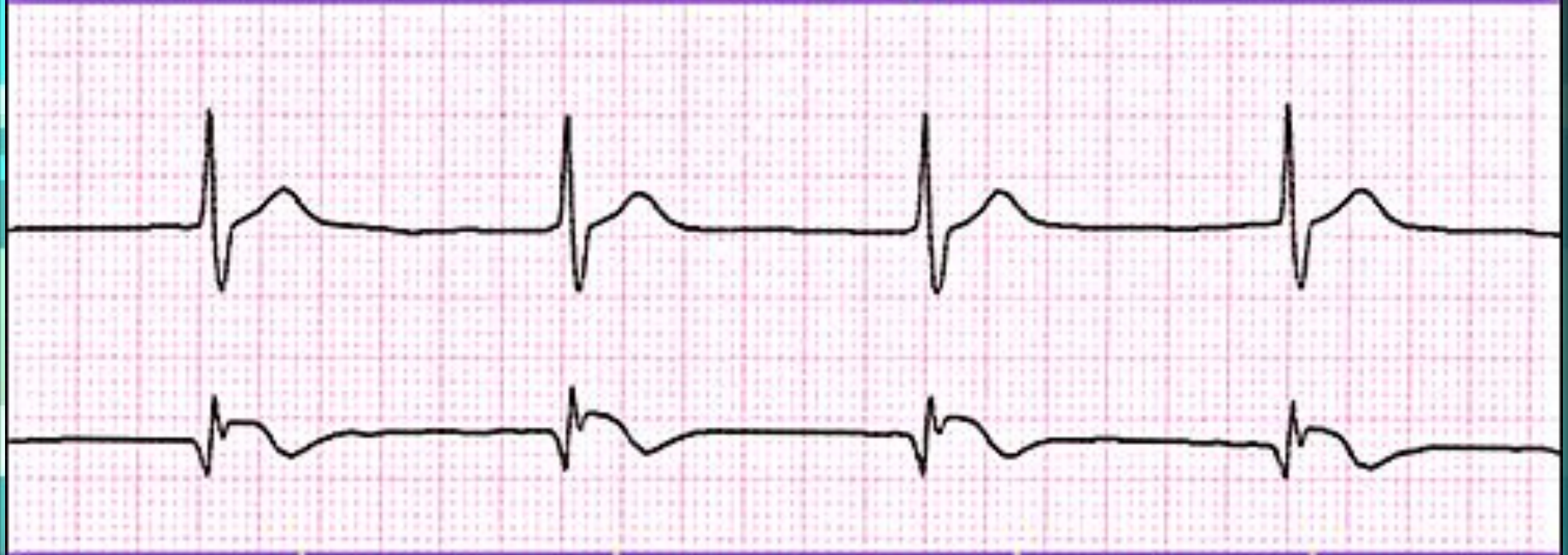
Sinus Bradycardia



Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
<60 bpm	Regular	Before each QRS, identical	.12 to .20	<.12

Junctional Rhythm

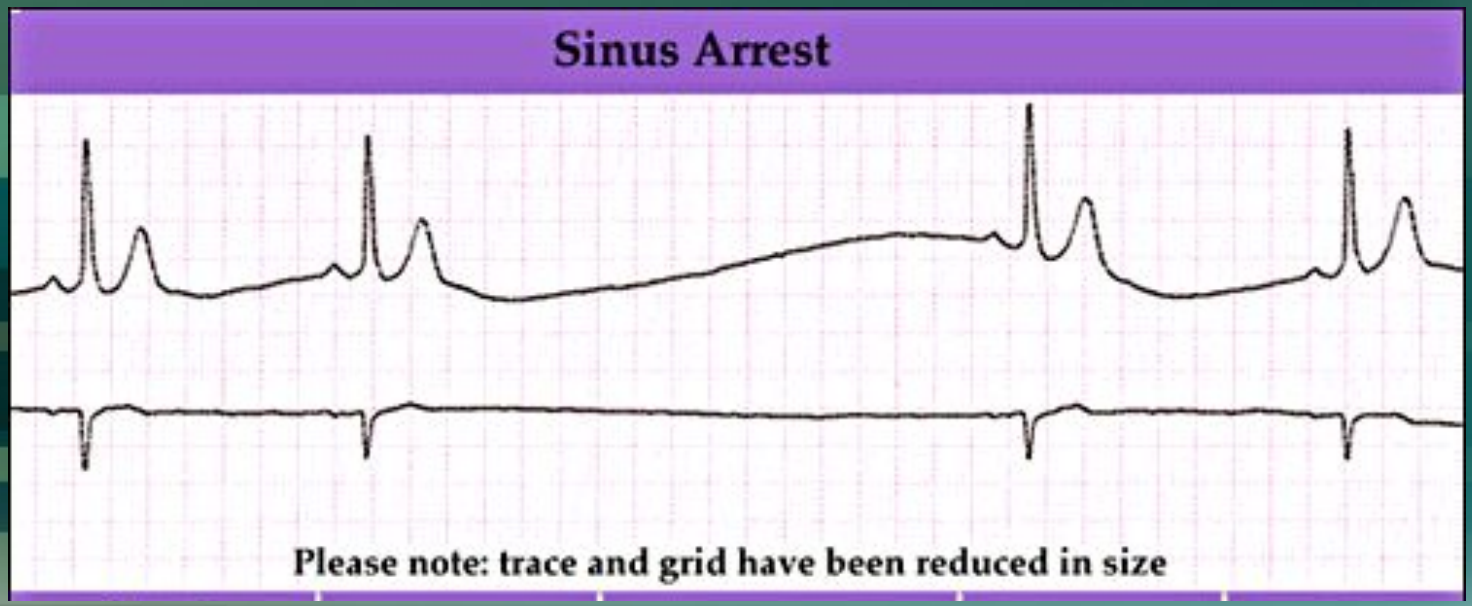
Junctional Rhythm



Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
40-60 bpm	Regular	Inverted, absent or after QRS	<.12	<.12

SA Block

- Sinus impulses is blocked within the SA junction
- Between SA node and surrounding myocardium
- Abscent of complete Cardiac cycle
- Occures irregularly and unpredictably
- Present :Young athletes, Digitalis, Hypokalemia, Sick Sinus Syndrome

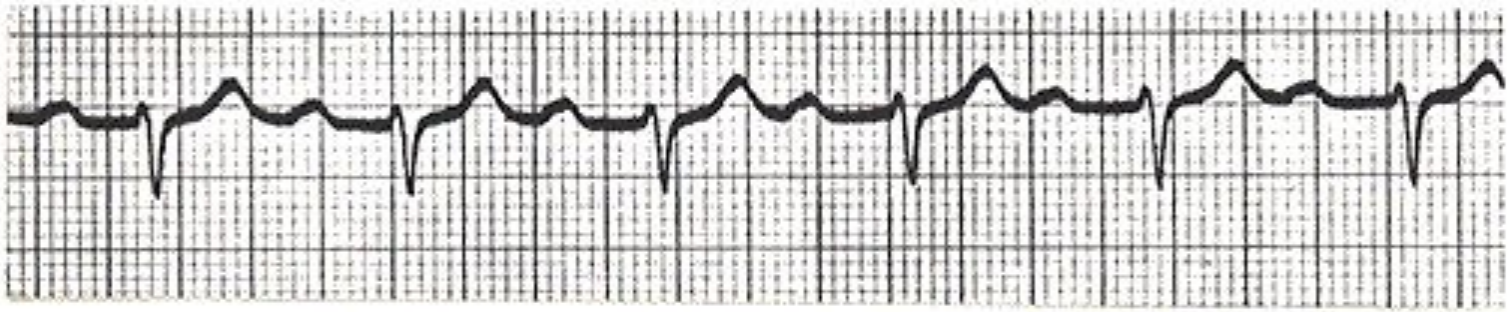


AV Block

- First Degree AV Block
- Second Degree AV Block
- Third Degree AV Block

First Degree AV Block

- **Delay in the conduction through the conducting system**
- **Prolong P-R interval**
- **All P waves are followed by QRS**
- **Associated with : AC Rheumati Carditis, Digitalis, Beta Blocker, excessive vagal tone, ischemia, intrinsic disease in the AV junction or bundle branch system.**

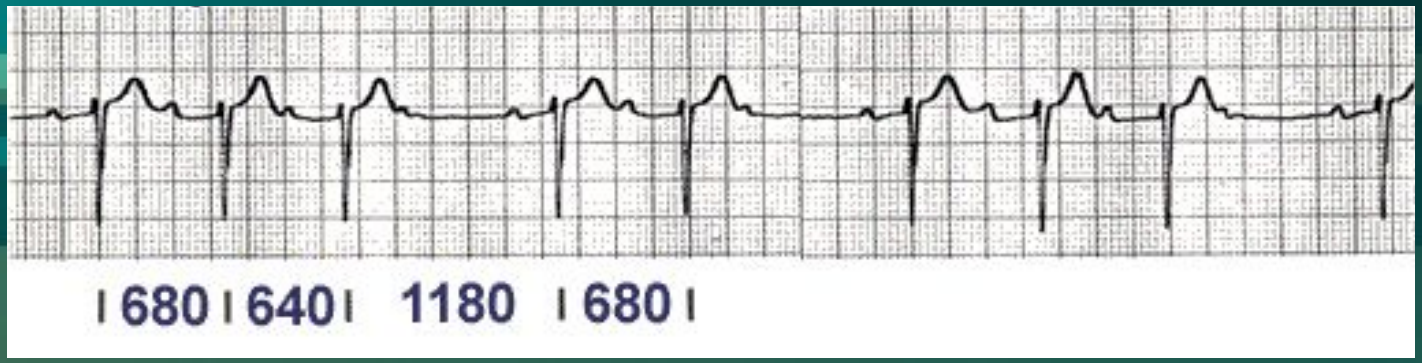


1st degree AV block (PR = 280 ms)

Second Degree AV Block

- Intermittent failure of AV conduction
- Impulse blocked by AV node
- Types:
- Mobitz type 1 (Wenckebach Phenomenon)
- Mobitz type 2

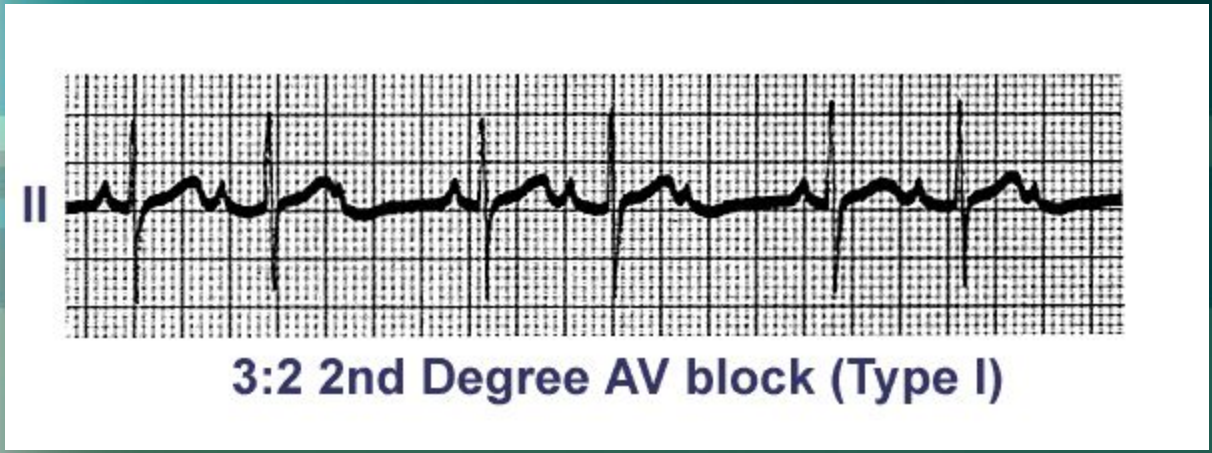
Mobitz type 1 (Wenckebach Phenomenon)



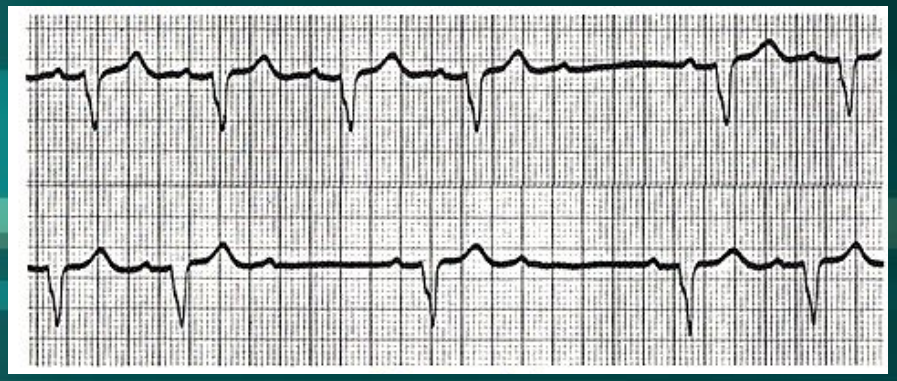
The 3 rules of "classic AV Wenckebach"

1. Decreasing RR intervals until pause;
2. Pause is less than preceding 2 RR intervals
3. RR interval after the pause is greater than RR prior to pause.

Mobitz type 1 (Wenckebach Phenomenon)

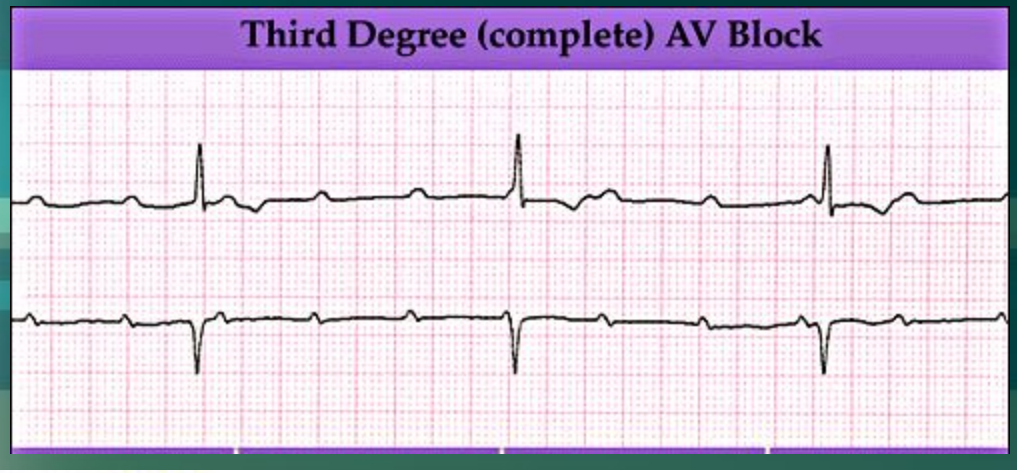


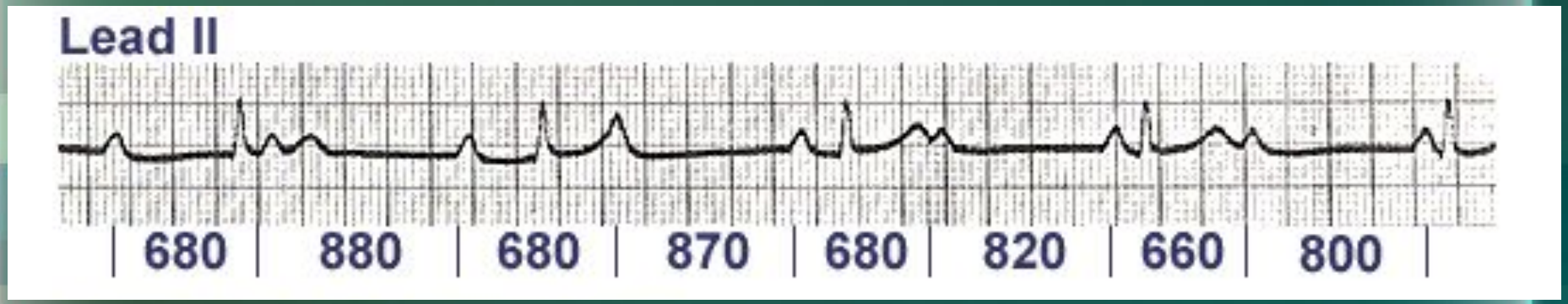
•Mobitz type 2



- Usually a sign of bilateral bundle branch disease.
- One of the branches should be completely blocked;
- most likely blocked in the right bundle
- P waves may blocked somewhere in the AV junction, the His bundle.

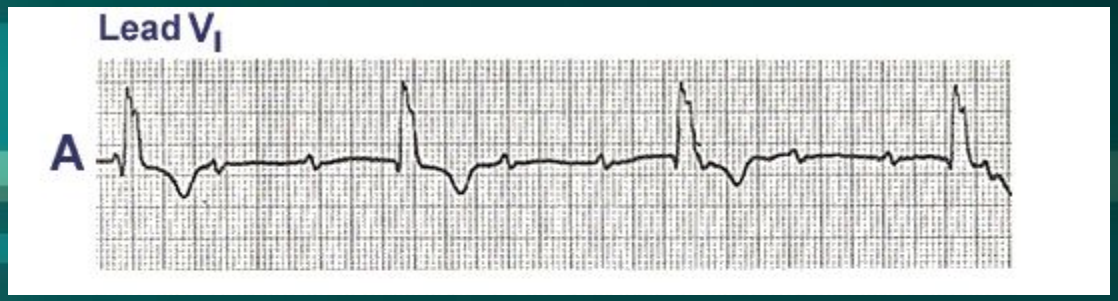
Third Degree Heart Block





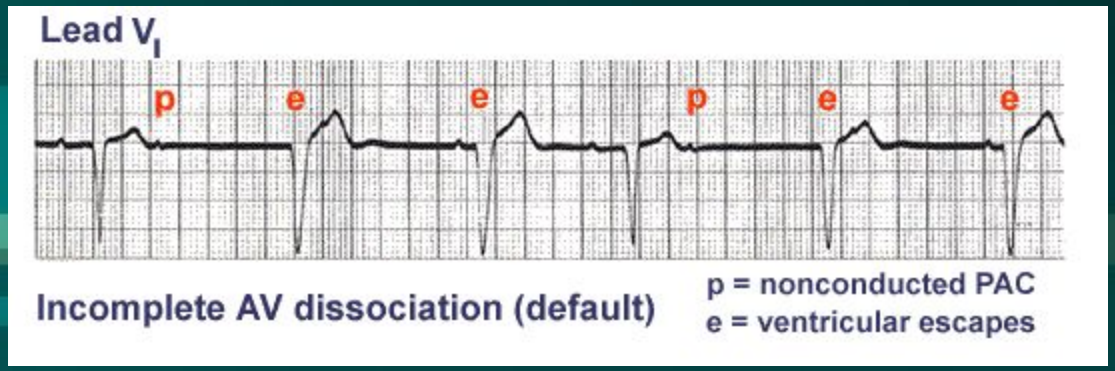
- CHB evidenced by the AV dissociation
- A junctional escape rhythm at 45 bpm.
- The PP intervals vary because of ventriculophasic sinus arrhythmia;

Third Degree Heart Block



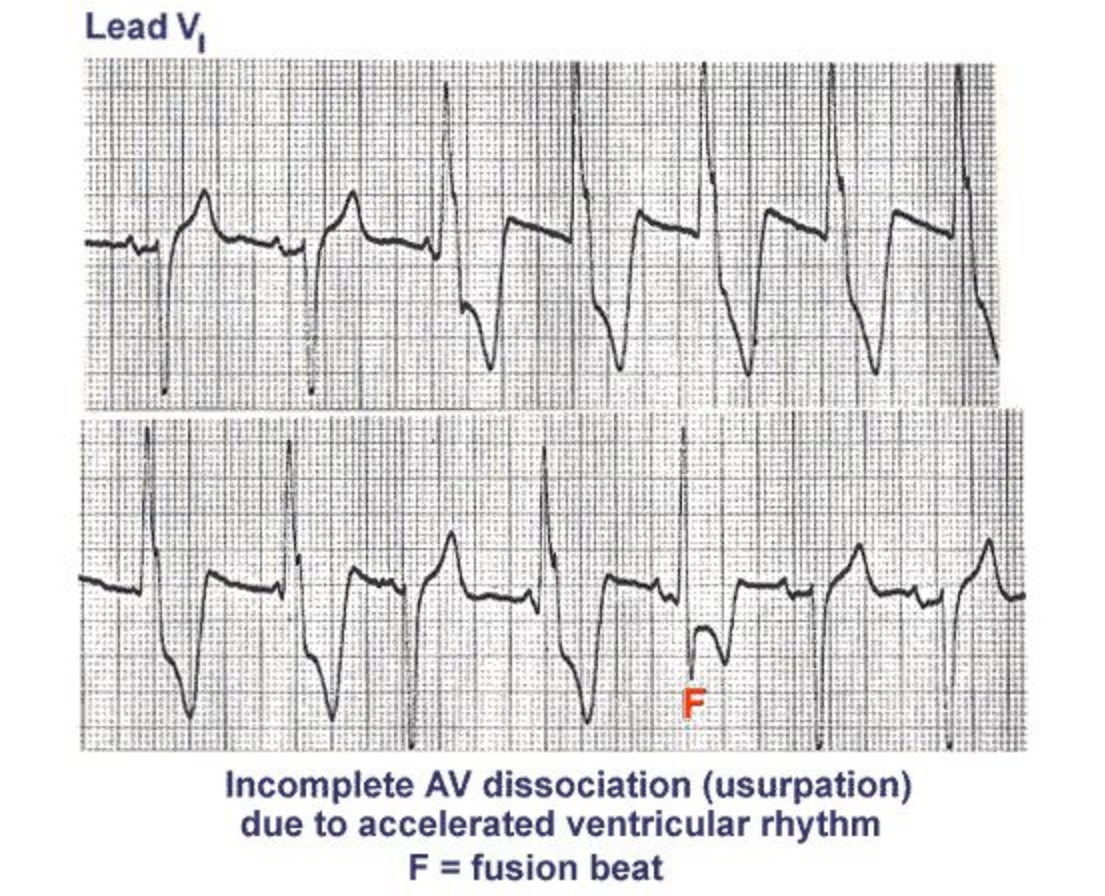
3rd degree AV block with a left ventricular escape rhythm, 'B' the right ventricular pacemaker rhythm is shown.

AV Dissociation



The nonconducted PAC's set up a long pause which is terminated by ventricular escapes;
Wider QRS morphology of the escape beats indicating their ventricular origin.

AV Dissociation



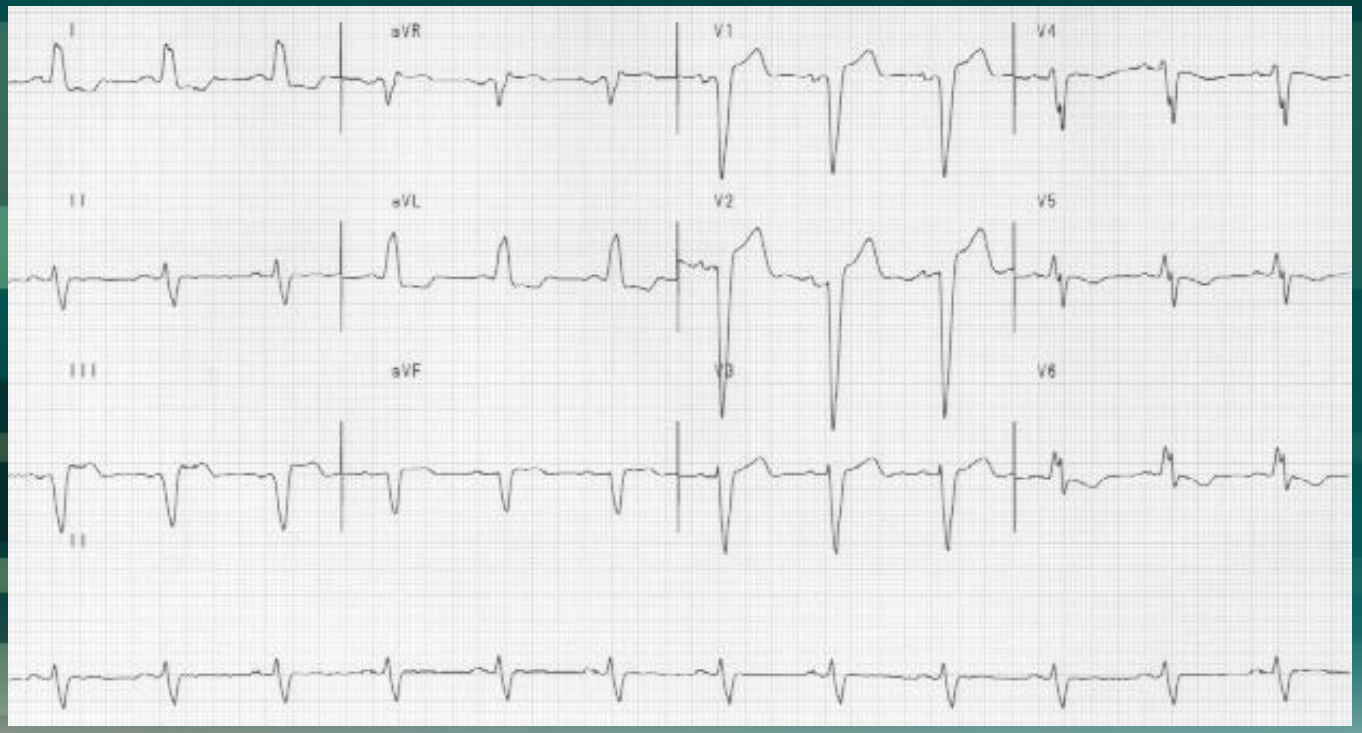
Incomplete AV dissociation (usurpation)
due to accelerated ventricular rhythm
F = fusion beat

Due to Accelerated ventricular rhythm

Thank You

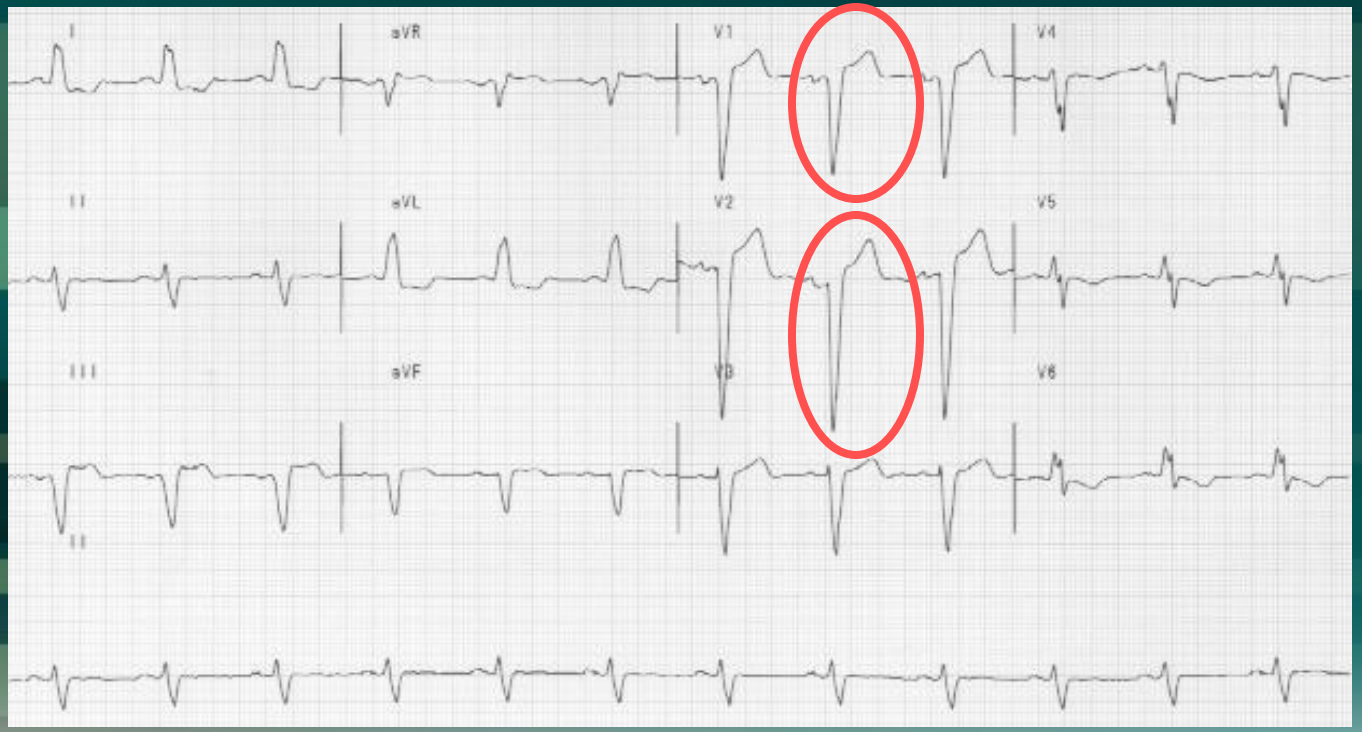
Putting it all Together

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



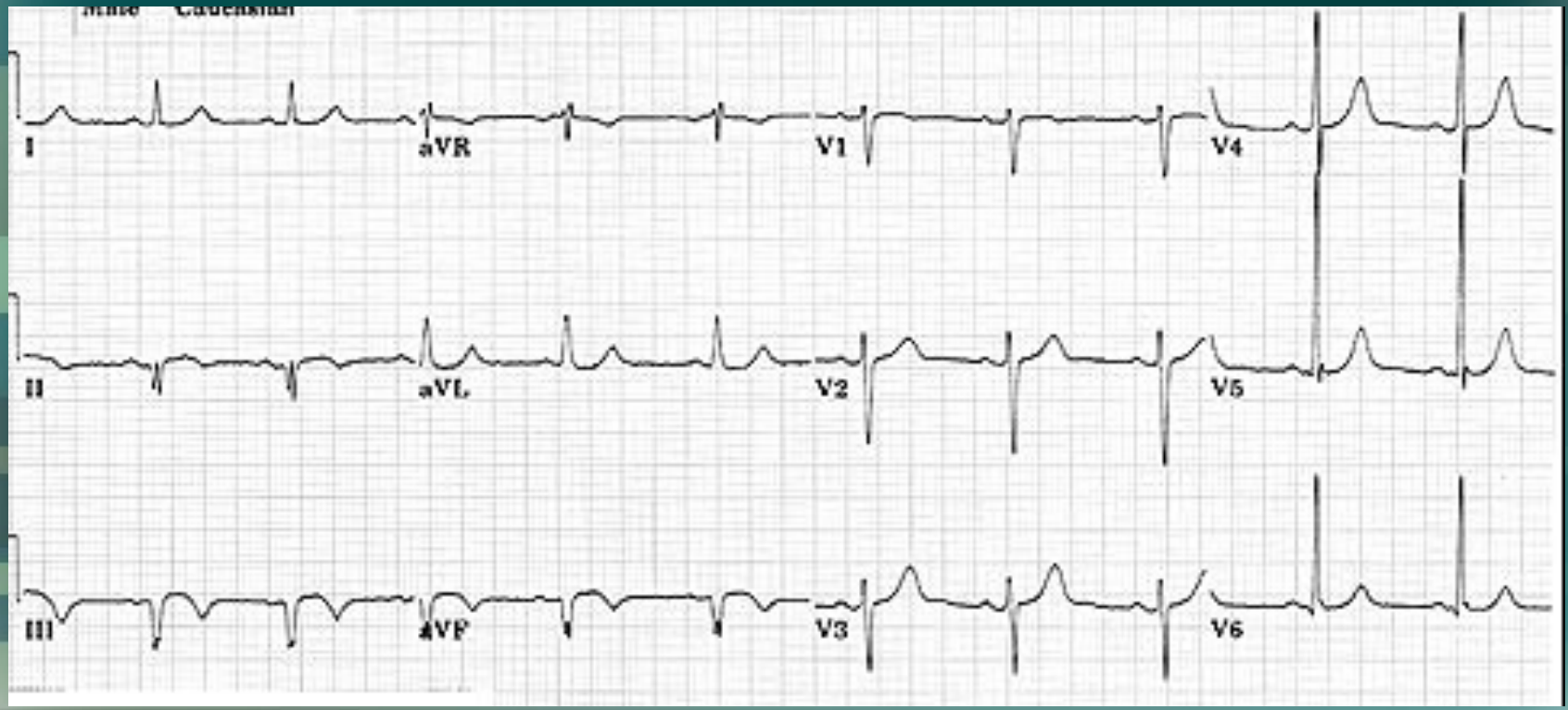
Interpretation

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



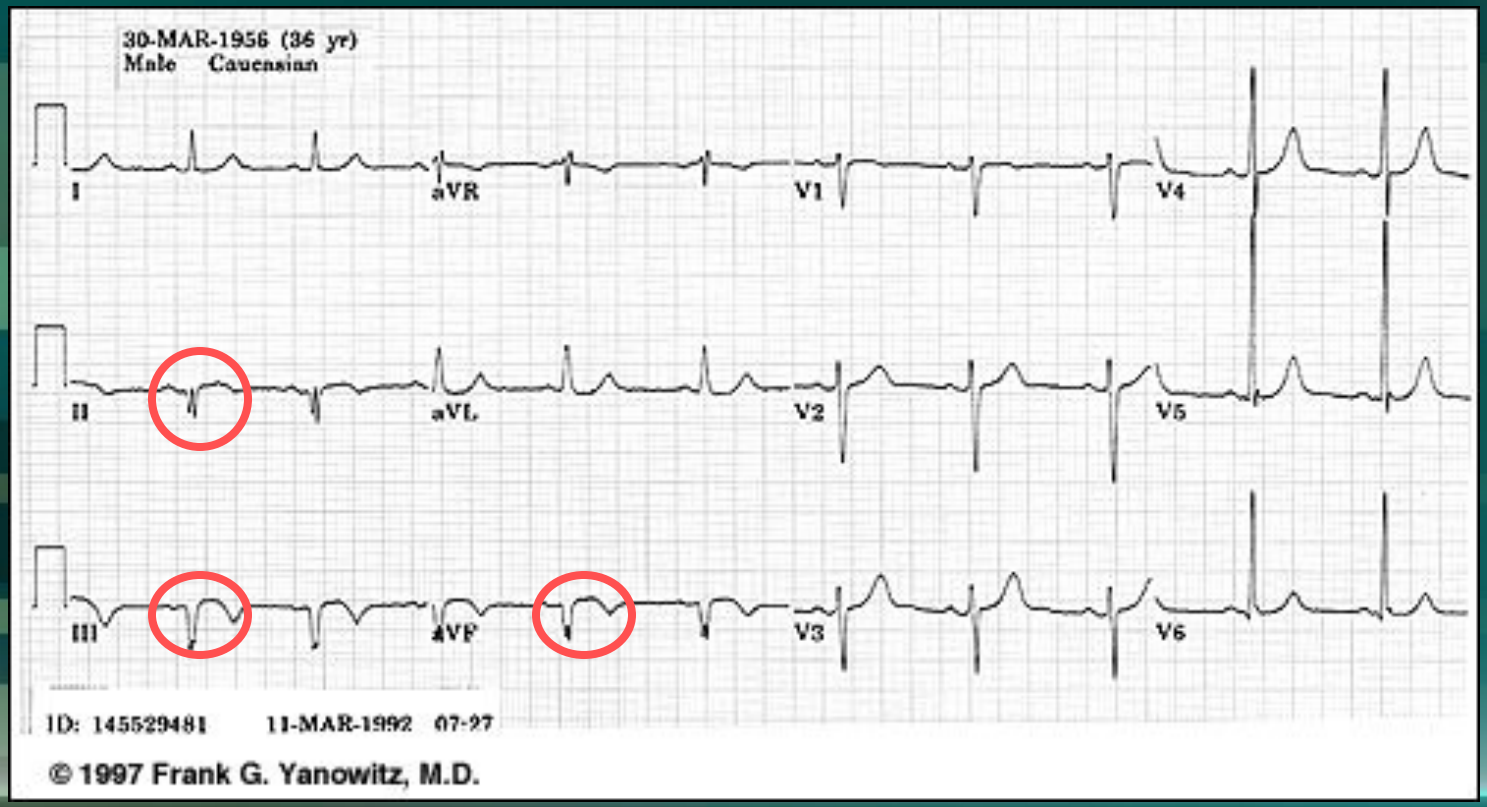
Putting it all Together

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



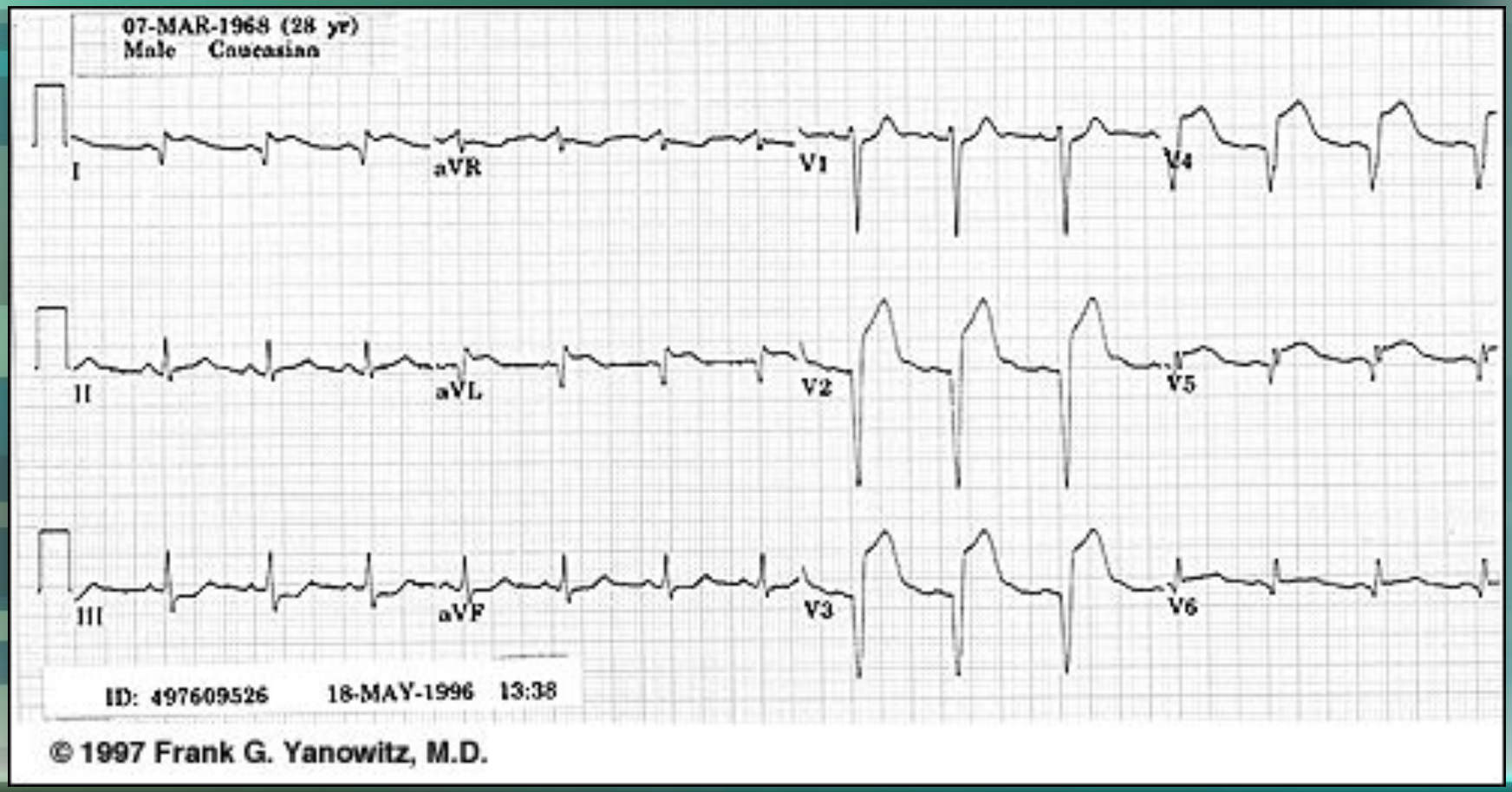
Inferior Wall MI

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



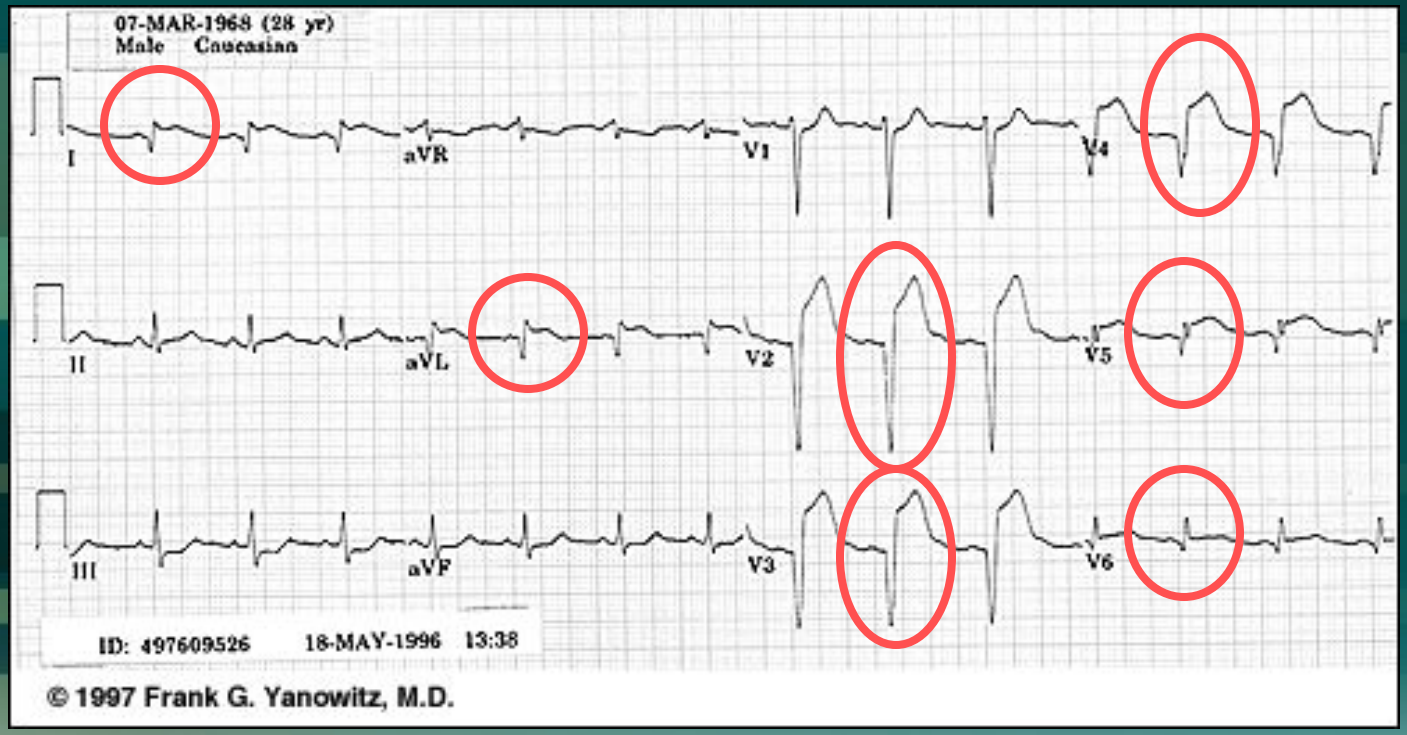
Putting it all Together

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)!



Rhythm #6

- Rate? 70 bpm
- Regularity? regular
- P waves? flutter waves
- PR interval? none
- QRS duration? 0.06 s

Interpretation? *Atrial Flutter*

Rhythm #7

- Rate? 74 □148 bpm
- Regularity? Regular □ regular
- P waves? Normal □ none
- PR interval? 0.16 s □ none
- QRS duration? 0.08 s

Interpretation? *Paroxysmal Supraventricular Tachycardia (PSVT)*

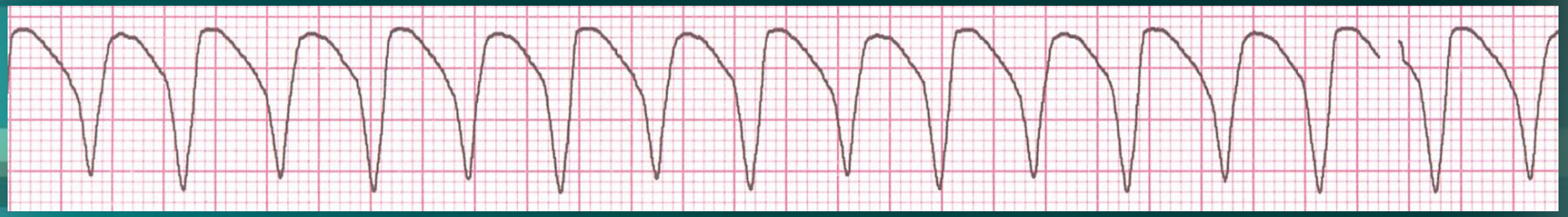
PSVT

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

Ventricular Arrhythmias

- *Ventricular Tachycardia*
- *Ventricular Fibrillation*

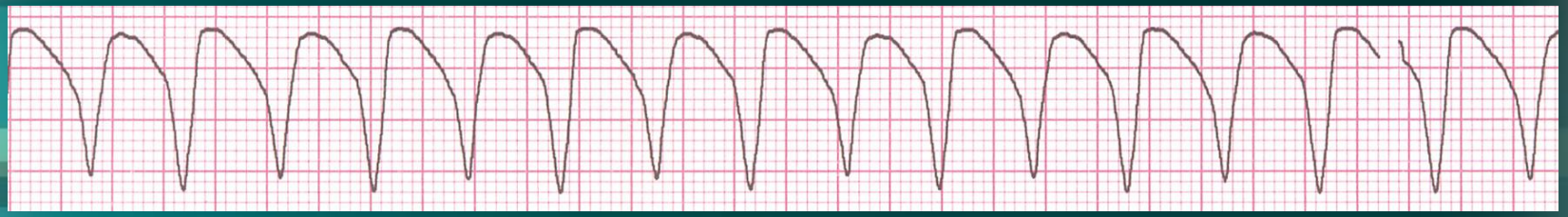
Rhythm #8



- Rate? 160 bpm
- Regularity? regular
- P waves? none
- PR interval? none
- QRS duration? wide (> 0.12 sec)

Interpretation? *Ventricular Tachycardia*

Ventricular Tachycardia



- Deviation from NSR
  - Impulse is originating in the ventricles (no P waves, wide QRS).

Rhythm #9

- Rate? none
- Regularity? irregularly irreg.
- P waves? none
- PR interval? none
- QRS duration? wide, if recognizable

Interpretation? *Ventricular Fibrillation*

Ventricular Fibrillation

- Deviation from NSR
  - Completely abnormal.

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 to a looping re-entrant circuit — *Atrial Flutter*

AV Junctional Problems

The AV junction can:

- fire continuously due to a looping re-entrant circuit — *Paroxysmal Supraventricular Tachycardia*
- block impulses coming from the SA Node — *AV Junctional Blocks*

Rhythm #1

- Rate? 30 bpm
- Regularity? regular
- P waves? normal
- PR interval? 0.12 s
- QRS duration? 0.10 s

Interpretation? *Sinus Bradycardia*

Rhythm #2

- Rate? 130 bpm
- Regularity? regular
- P waves? normal
- PR interval? 0.16 s
- QRS duration? 0.08 s

Interpretation? *Sinus Tachycardia*

Rhythm #3

- Rate? 70 bpm
- Regularity? occasionally irreg.
- P waves? 2/7 different contour
- PR interval? 0.14 s (except 2/7)
- QRS duration? 0.08 s

Interpretation? *NSR with Premature Atrial Contractions*

Premature Atrial Contractions

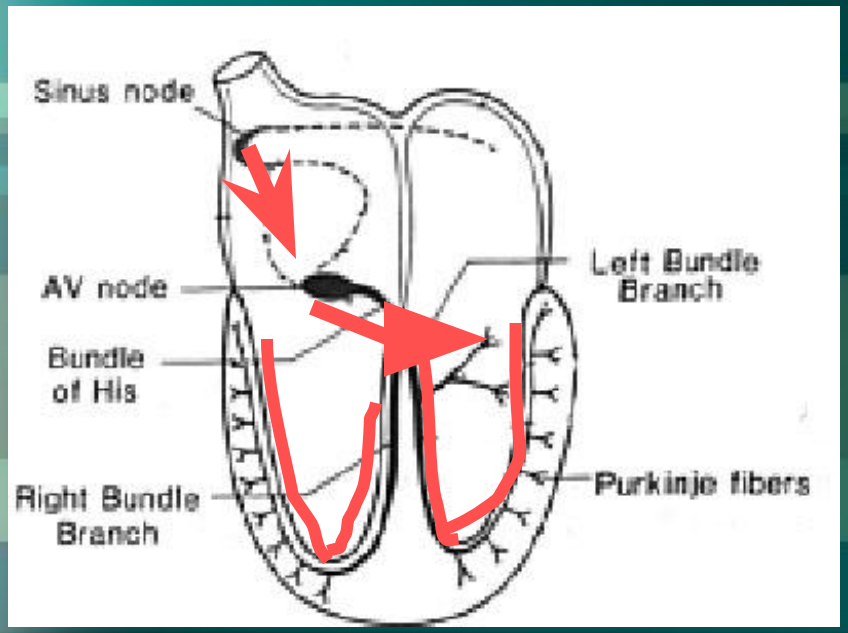
- Deviation from NSR
  - These ectopic beats originate in the atria (but not in the SA node), therefore the contour of the P wave, the PR interval, and the timing are different than a normally generated pulse from the SA node.

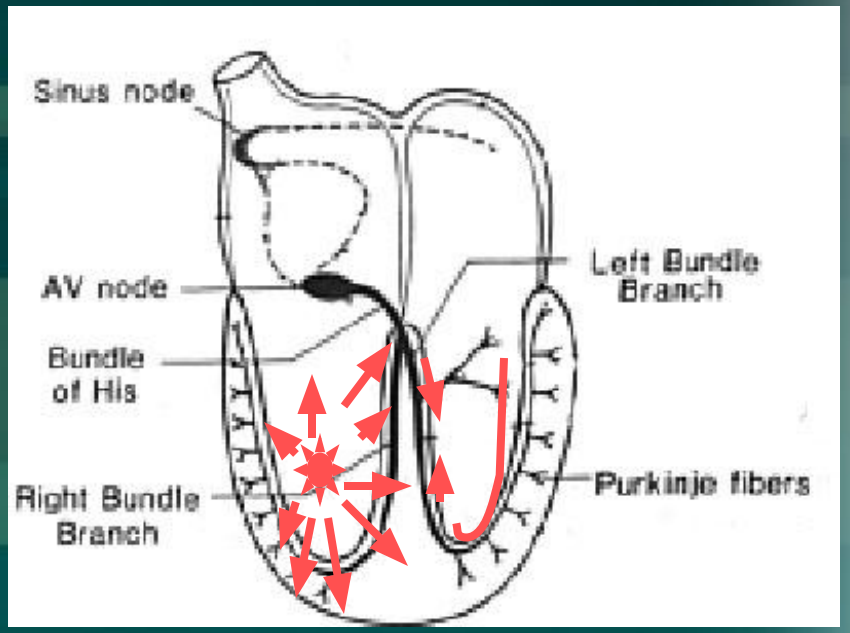
Rhythm #4

- Rate? 60 bpm
- Regularity? occasionally irreg.
- P waves? none for 7th QRS
- PR interval? 0.14 s
- QRS duration? 0.08 s (7th wide)

Interpretation? *Sinus Rhythm with 1 PVC*

Ventricular Conduction





Normal

Signal moves rapidly through the ventricles

Abnormal

Signal moves slowly through the ventricles

AV Nodal Blocks

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

Rhythm #10

- 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

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

Rhythm #11

- Rate? 50 bpm
- Regularity? regularly irregular
- P waves? nl, but 4th no QRS
- PR interval? lengthens
- QRS duration? 0.08 s

Interpretation? *2nd Degree AV Block, Type I*

Rhythm #12

- Rate? 40 bpm
- Regularity? regular
- P waves? nl, 2 of 3 no QRS
- PR interval? 0.14 s
- QRS duration? 0.08 s

Interpretation? *2nd Degree AV Block, Type II*

2nd Degree AV Block, Type II

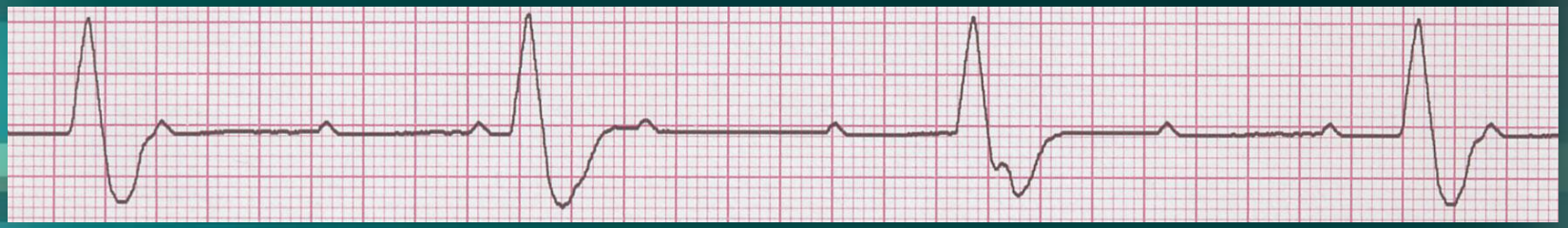
- Deviation from NSR
  - Occasional P waves are completely blocked (P wave not followed by QRS).

Rhythm #13

- Rate? 40 bpm
- Regularity? regular
- P waves? no relation to QRS
- PR interval? none
- QRS duration? 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.

Supraventricular Arrhythmias

- *Atrial Fibrillation*
- *Atrial Flutter*
- *Paroxysmal Supraventricular Tachycardia*

Rhythm #5

- 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

Rhythm #6

- Rate? 70 bpm
- Regularity? regular
- P waves? flutter waves
- PR interval? none
- QRS duration? 0.06 s

Interpretation? *Atrial Flutter*

Rhythm #7

- Rate? 74 □148 bpm
- Regularity? Regular □ regular
- P waves? Normal □ none
- PR interval? 0.16 s □ none
- QRS duration? 0.08 s

Interpretation? *Paroxysmal Supraventricular Tachycardia (PSVT)*

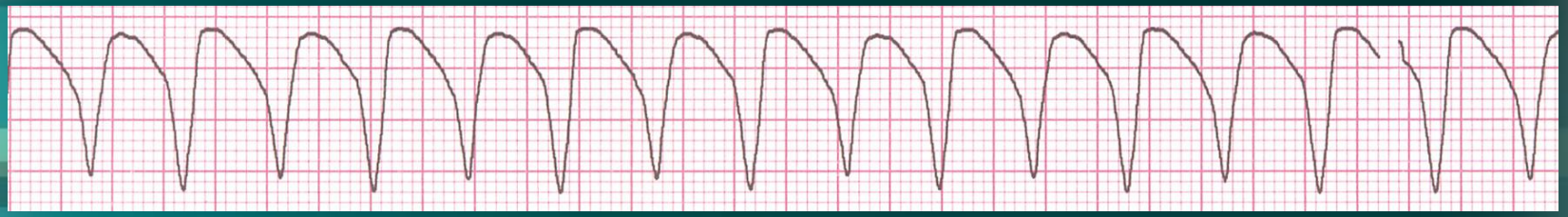
PSVT

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

Ventricular Arrhythmias

- *Ventricular Tachycardia*
- *Ventricular Fibrillation*

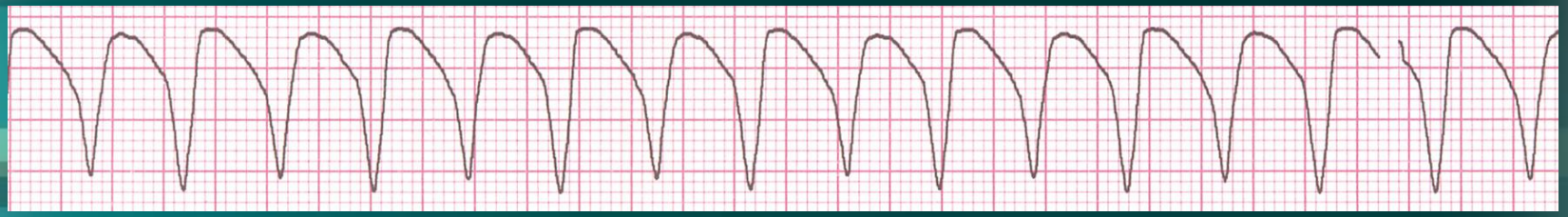
Rhythm #8



- Rate? 160 bpm
- Regularity? regular
- P waves? none
- PR interval? none
- QRS duration? wide (> 0.12 sec)

Interpretation? *Ventricular Tachycardia*

Ventricular Tachycardia



- Deviation from NSR
  - Impulse is originating in the ventricles (no P waves, wide QRS).

Rhythm #9

- Rate? none
- Regularity? irregularly irreg.
- P waves? none
- PR interval? none
- QRS duration? wide, if recognizable

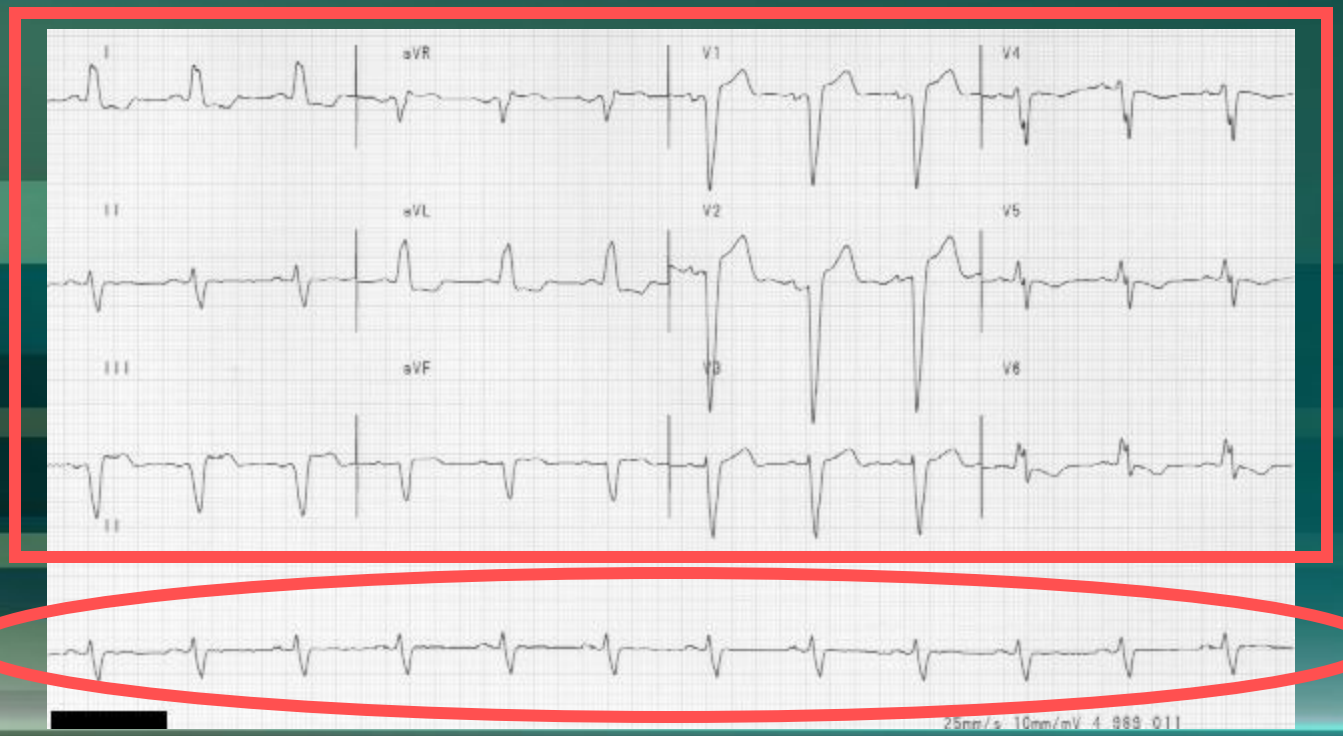
Interpretation? *Ventricular Fibrillation*

Ventricular Fibrillation

- Deviation from NSR
  - Completely abnormal.

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

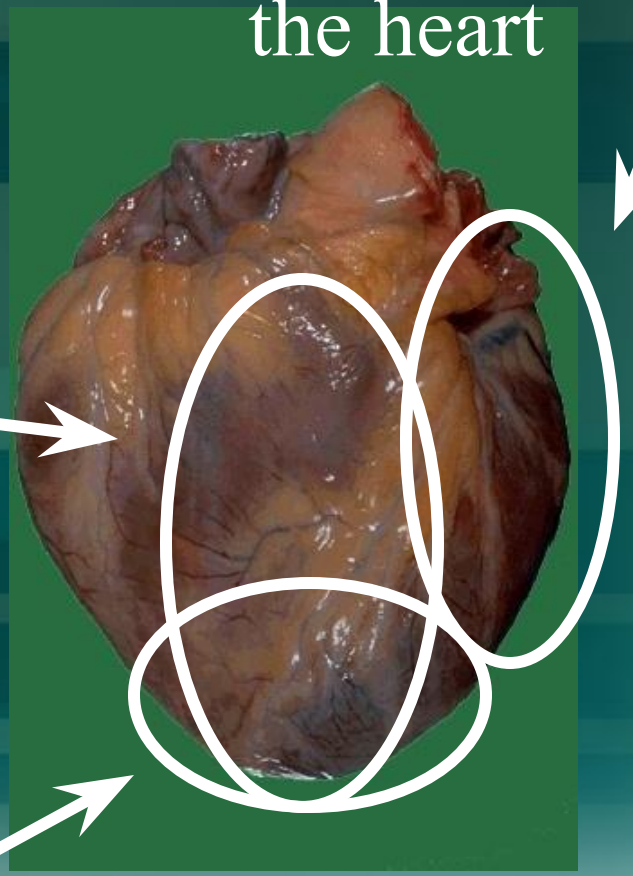
Views of the Heart

Some leads get a good view of the:

Lateral portion of the heart

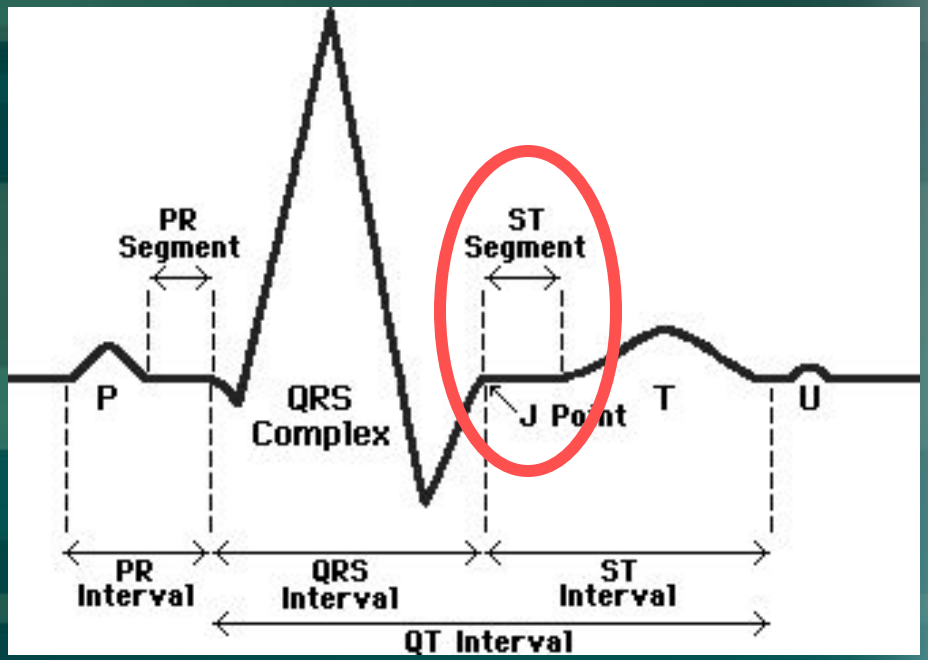
Anterior portion of the heart

Inferior portion of the heart



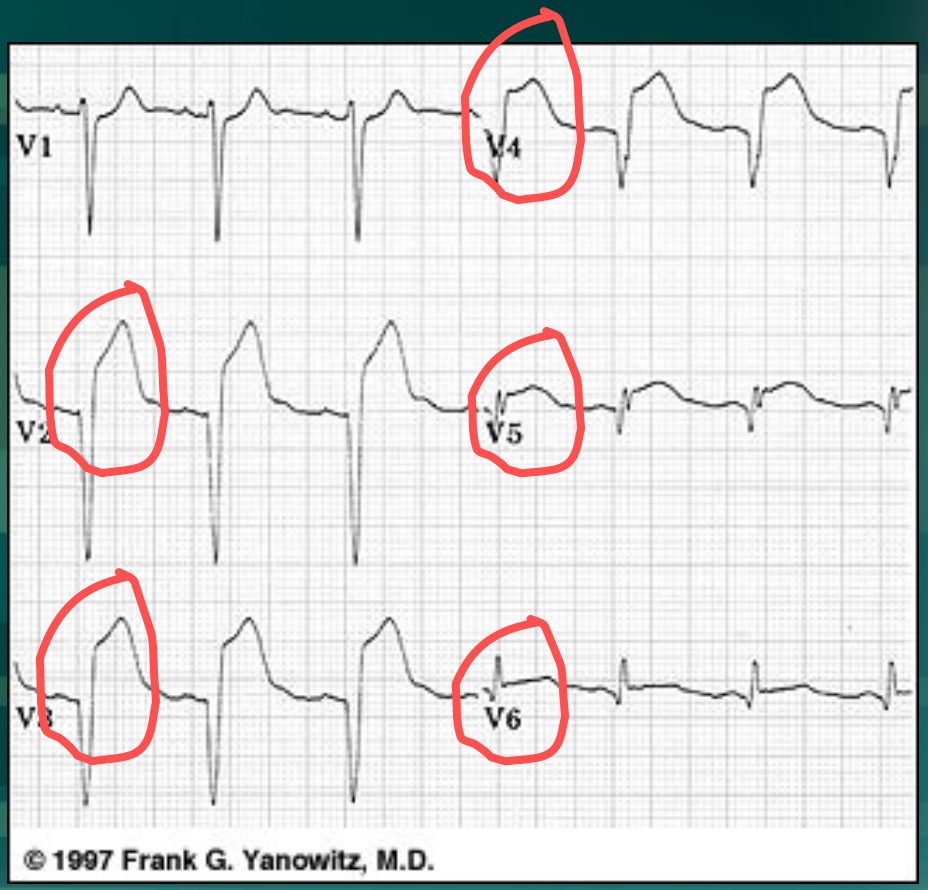
ST Elevation

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



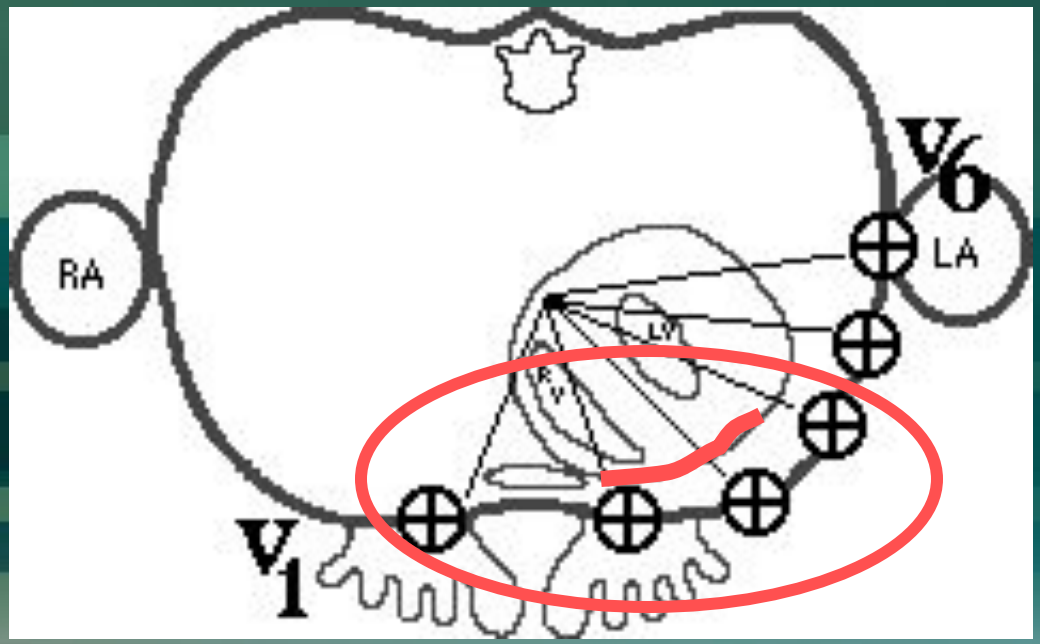
ST Elevation (cont)

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.

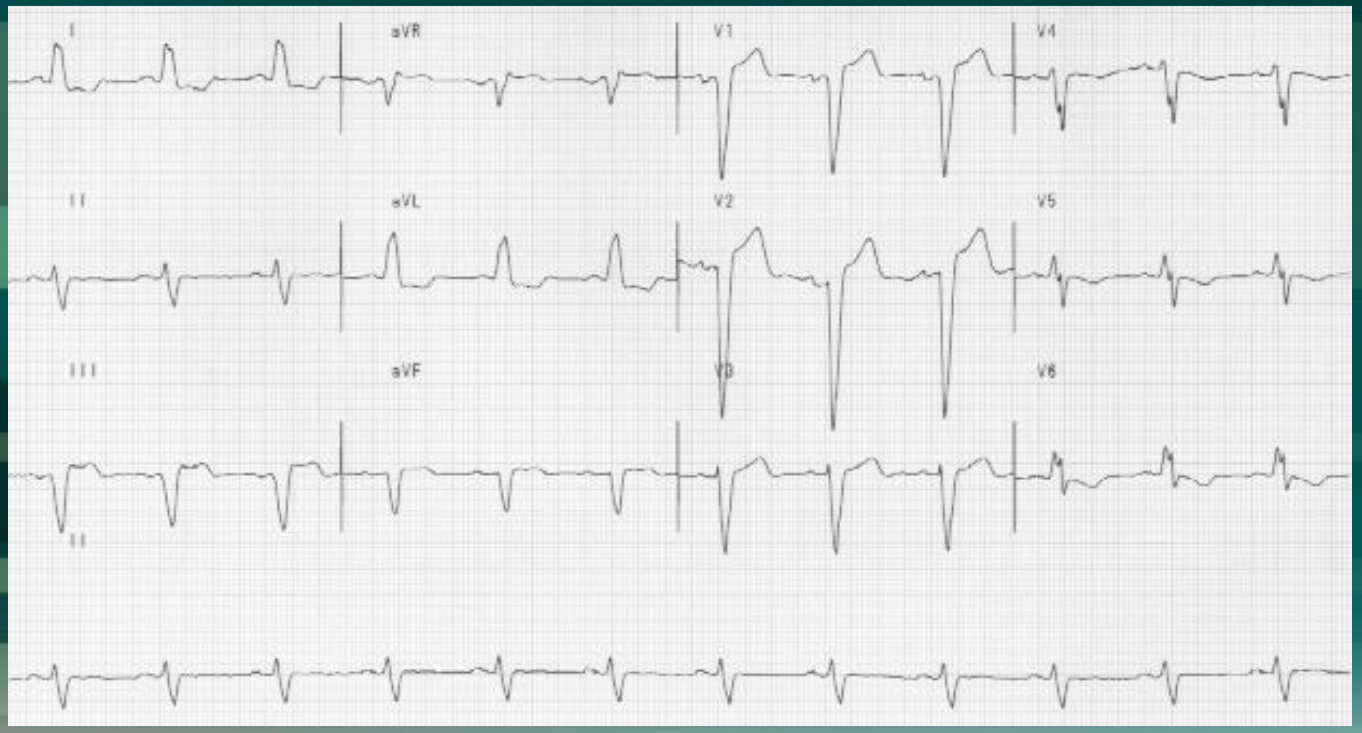


Anterior Myocardial Infarction

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.

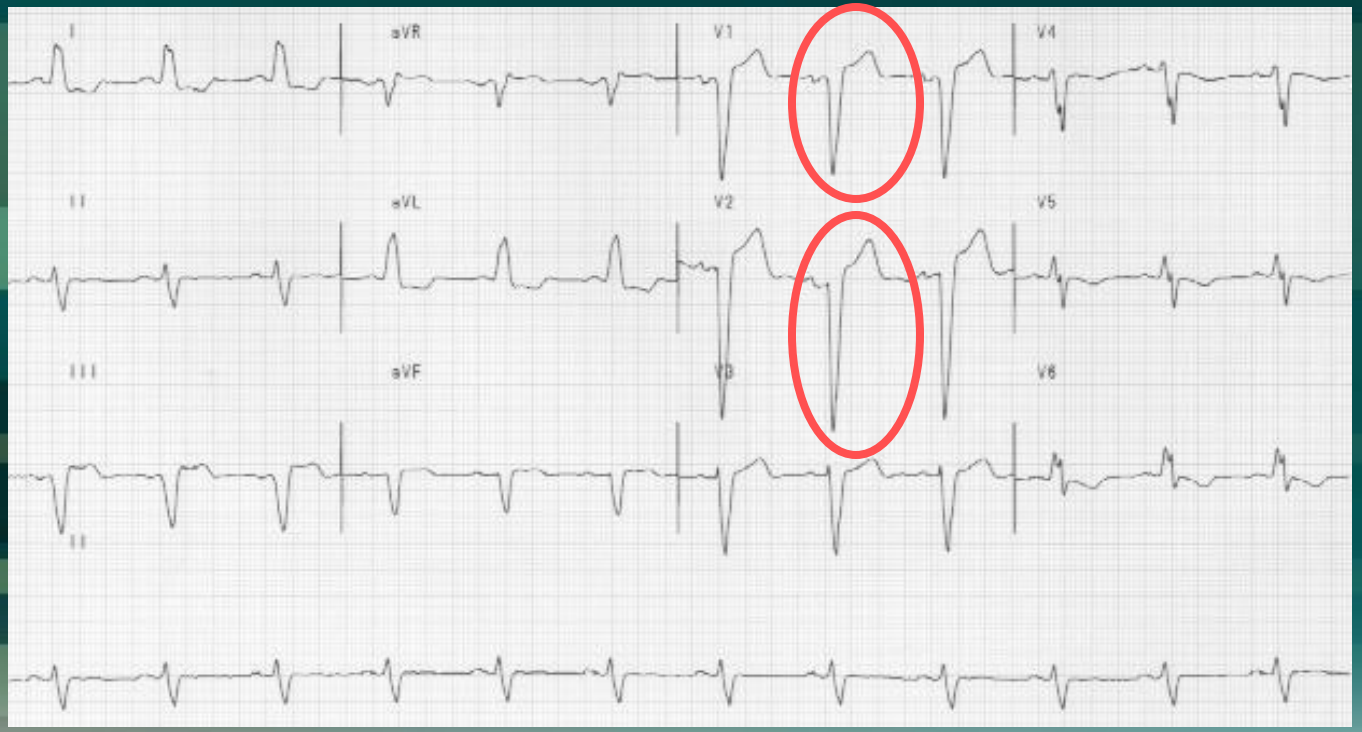
Putting it all Together

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



Interpretation

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

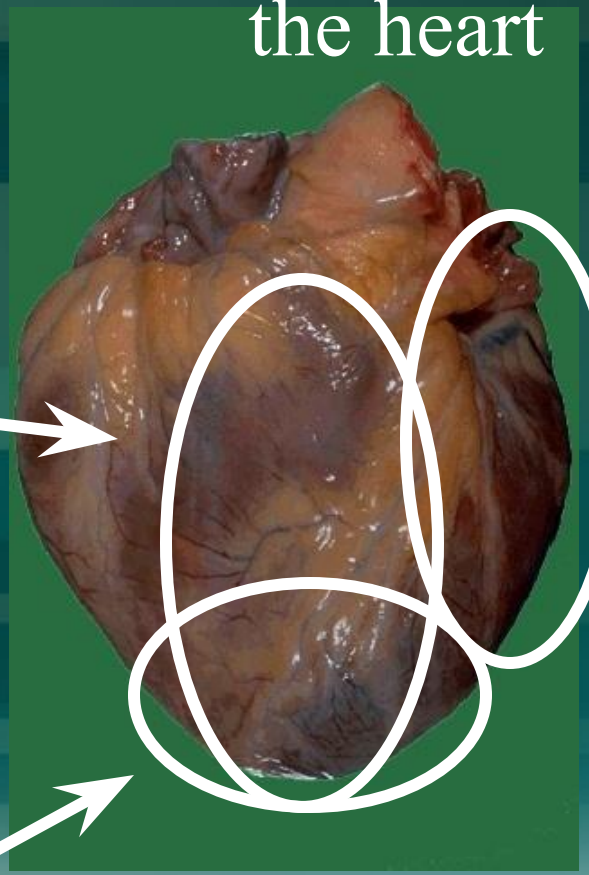


Other MI Locations

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.

Other MI Locations

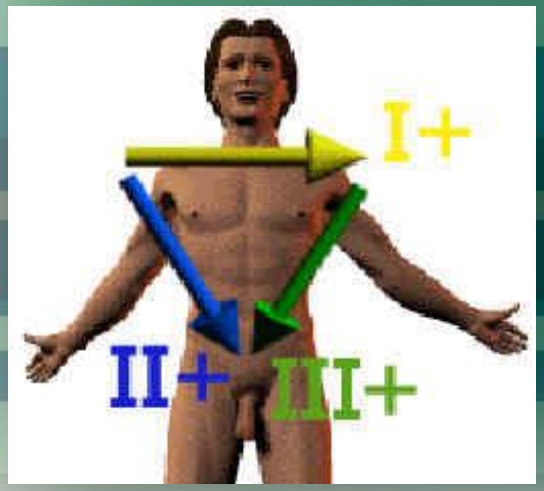
First, take a look again at this picture of the heart.



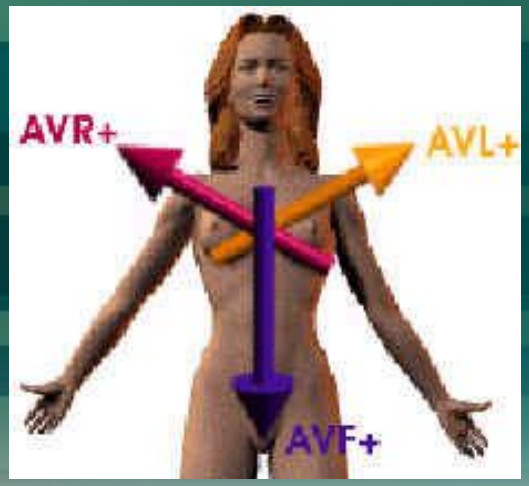
Other MI Locations

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.

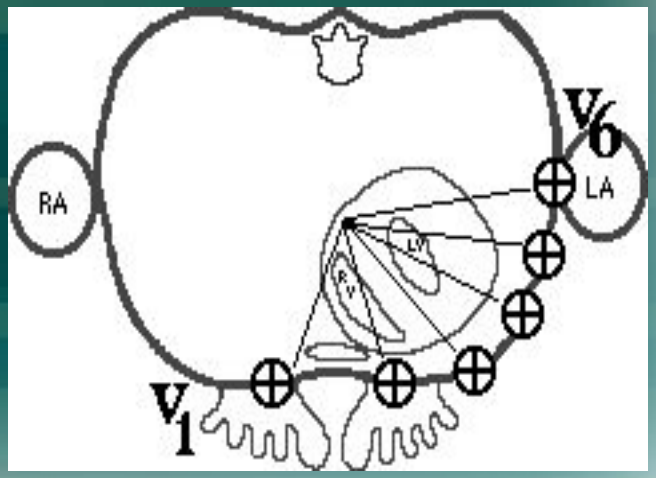
Limb Leads



Augmented Leads



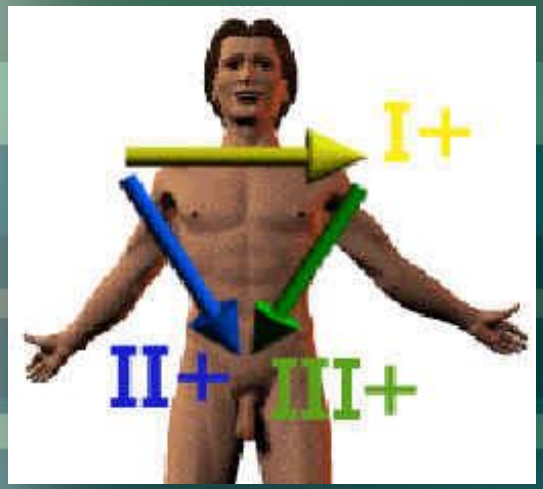
Precordial Leads



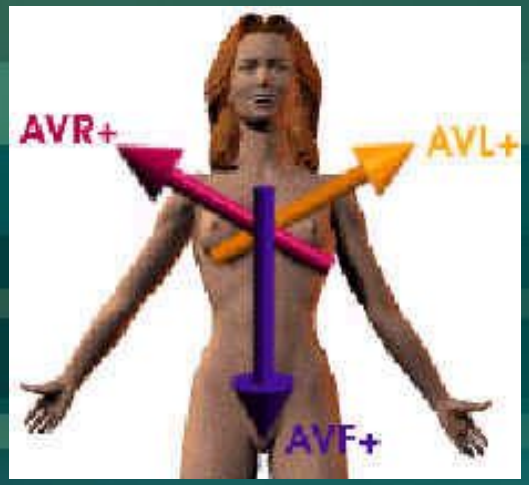
Other MI Locations

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

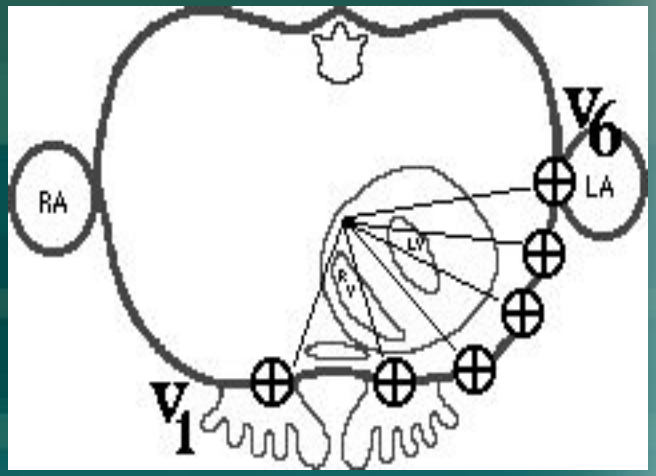
Limb Leads



Augmented Leads



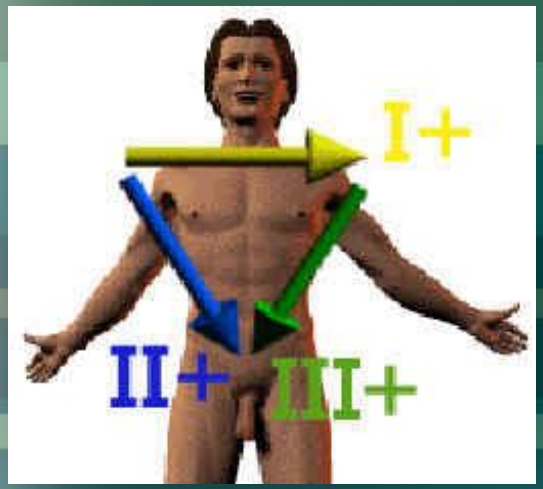
Precordial Leads



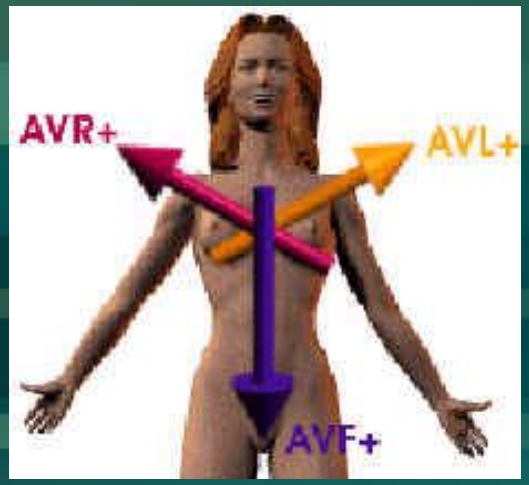
Anterior MI

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

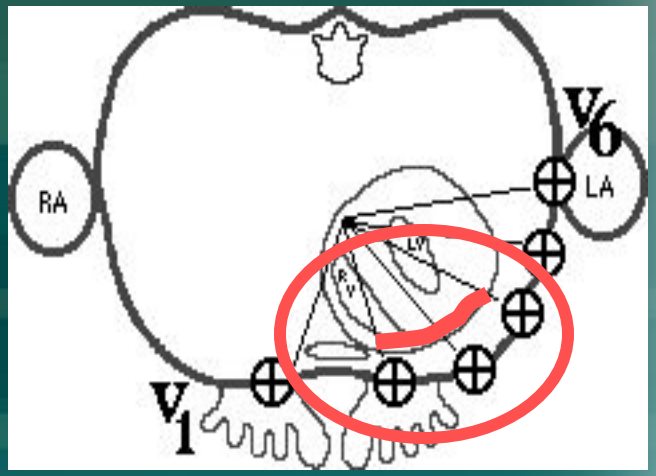
Limb Leads



Augmented Leads



Precordial Leads

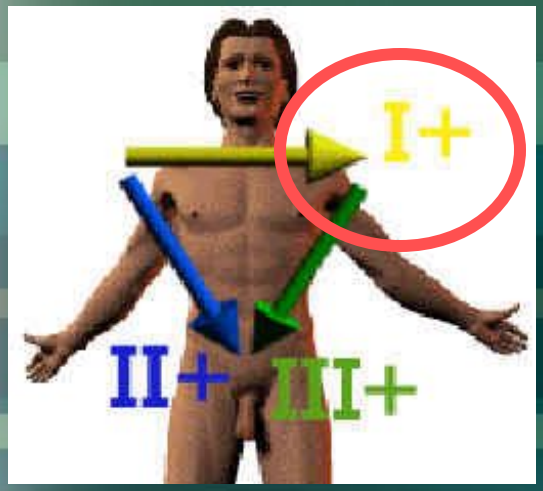


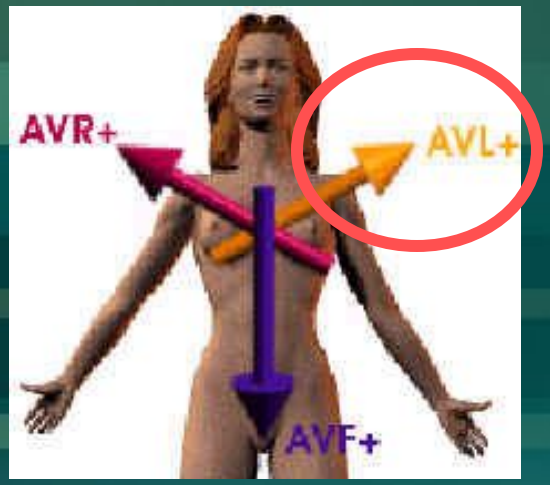
Lateral MI

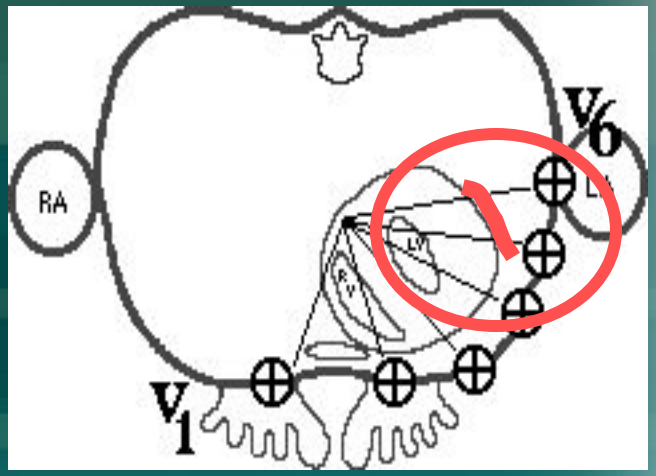
So what leads do you think the lateral portion of the heart is best viewed?

Leads I, aVL, and V_5- V_6

Limb Leads **Augmented Leads** **Precordial Leads**





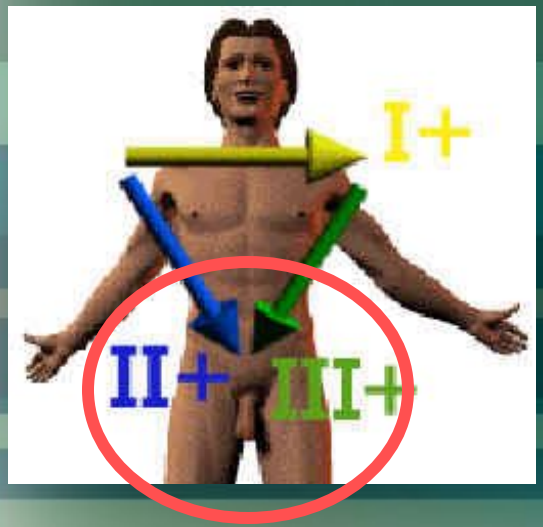


Inferior MI

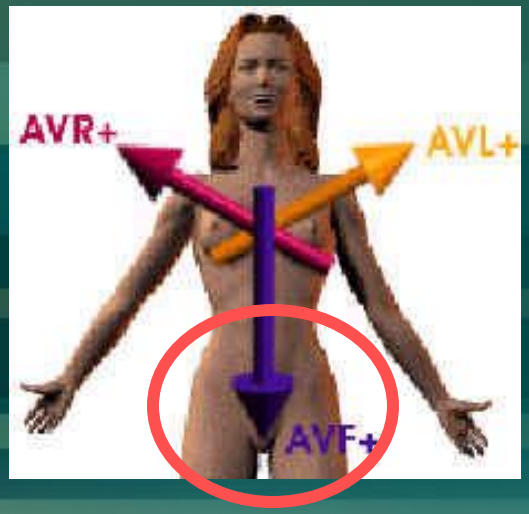
Now how about the inferior portion of the heart?

Leads II, III and aVF

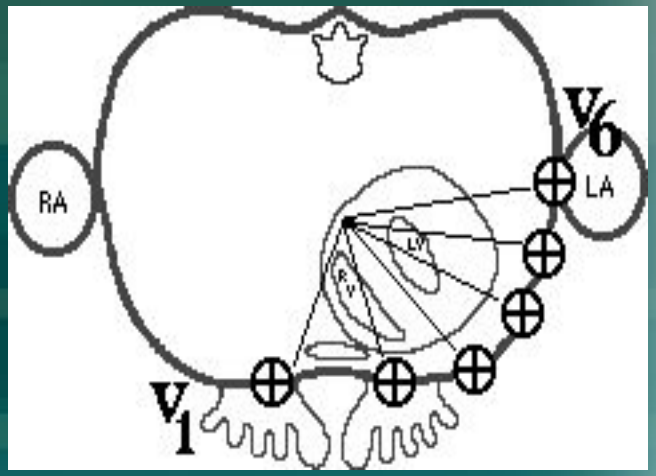
Limb Leads



Augmented Leads

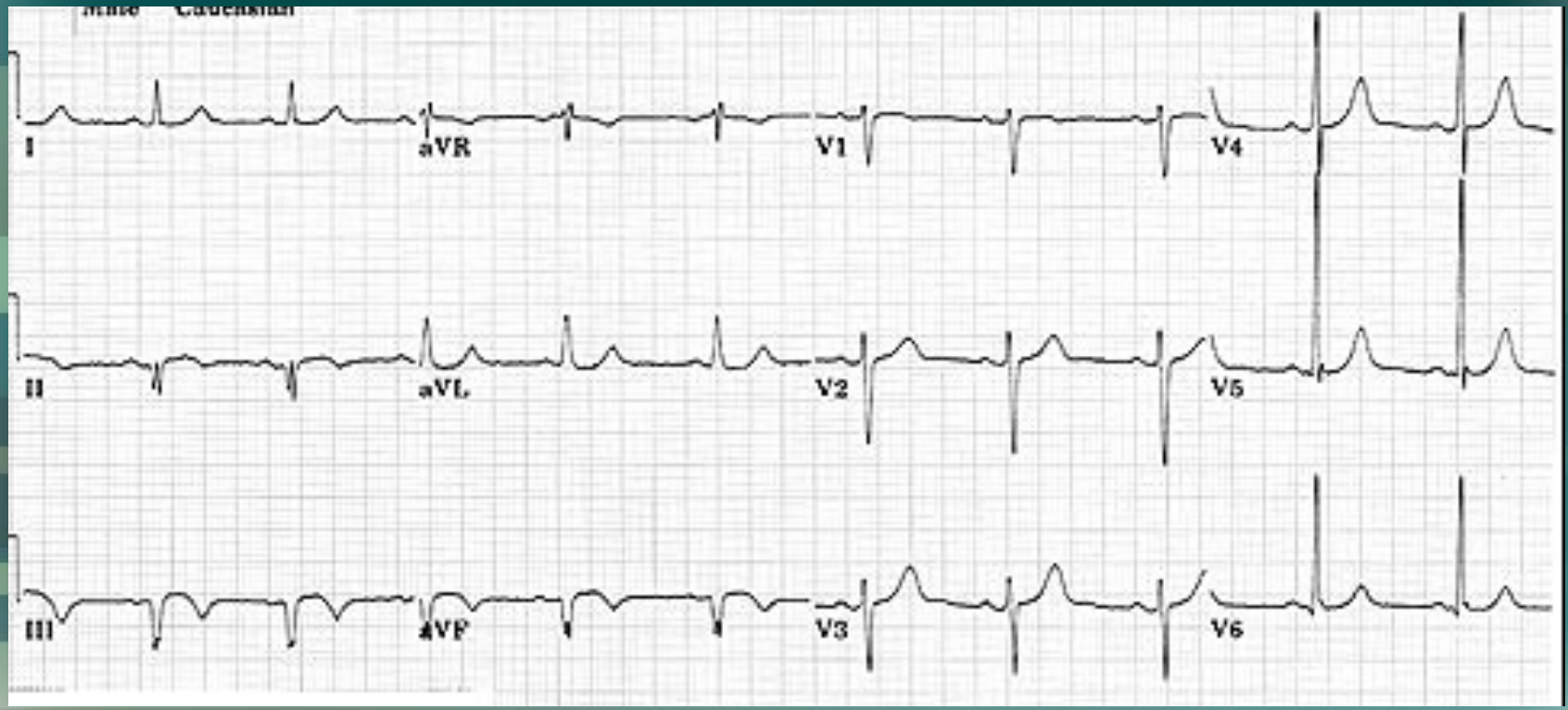


Precordial Leads



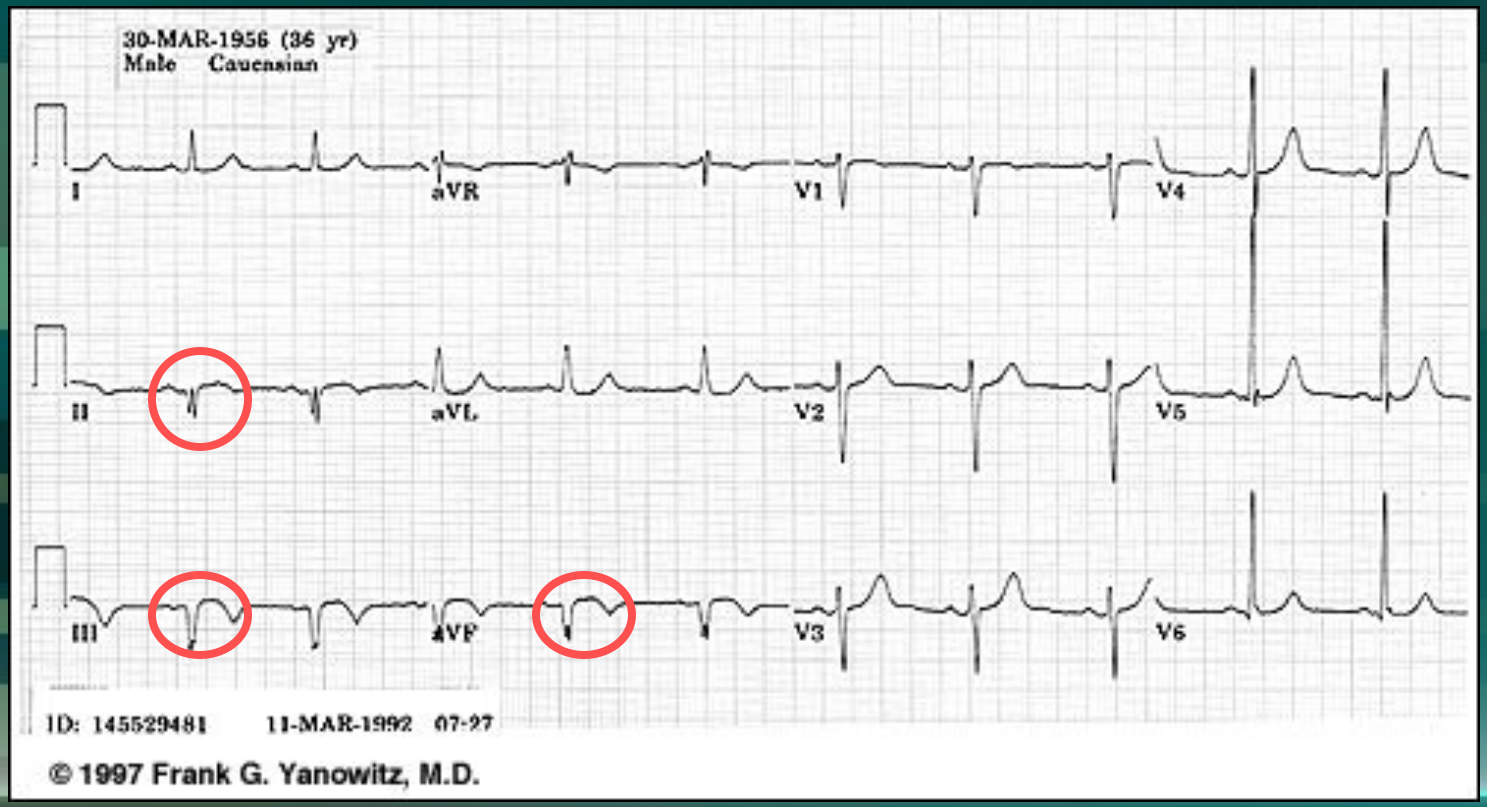
Putting it all Together

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



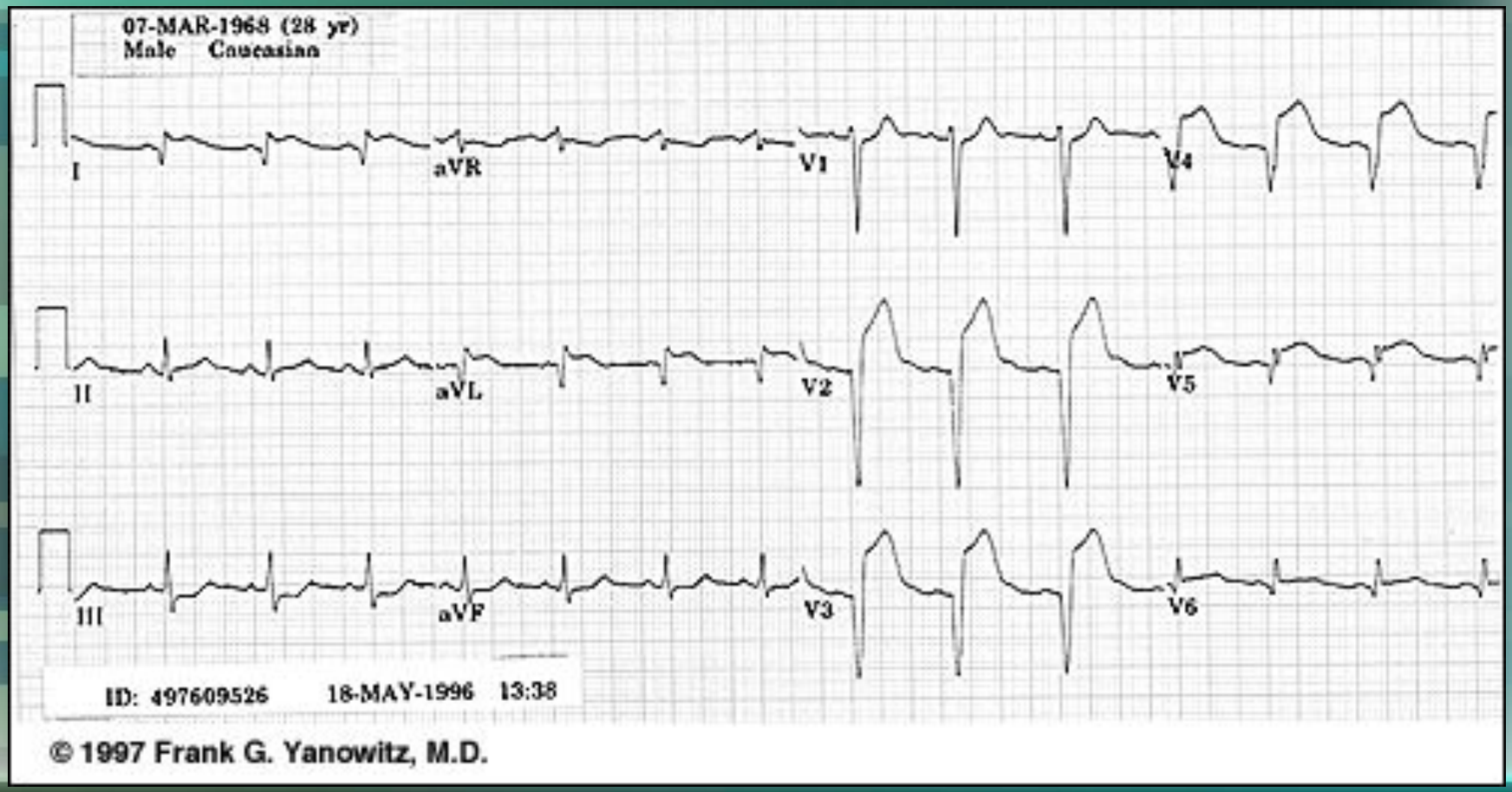
Inferior Wall MI

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



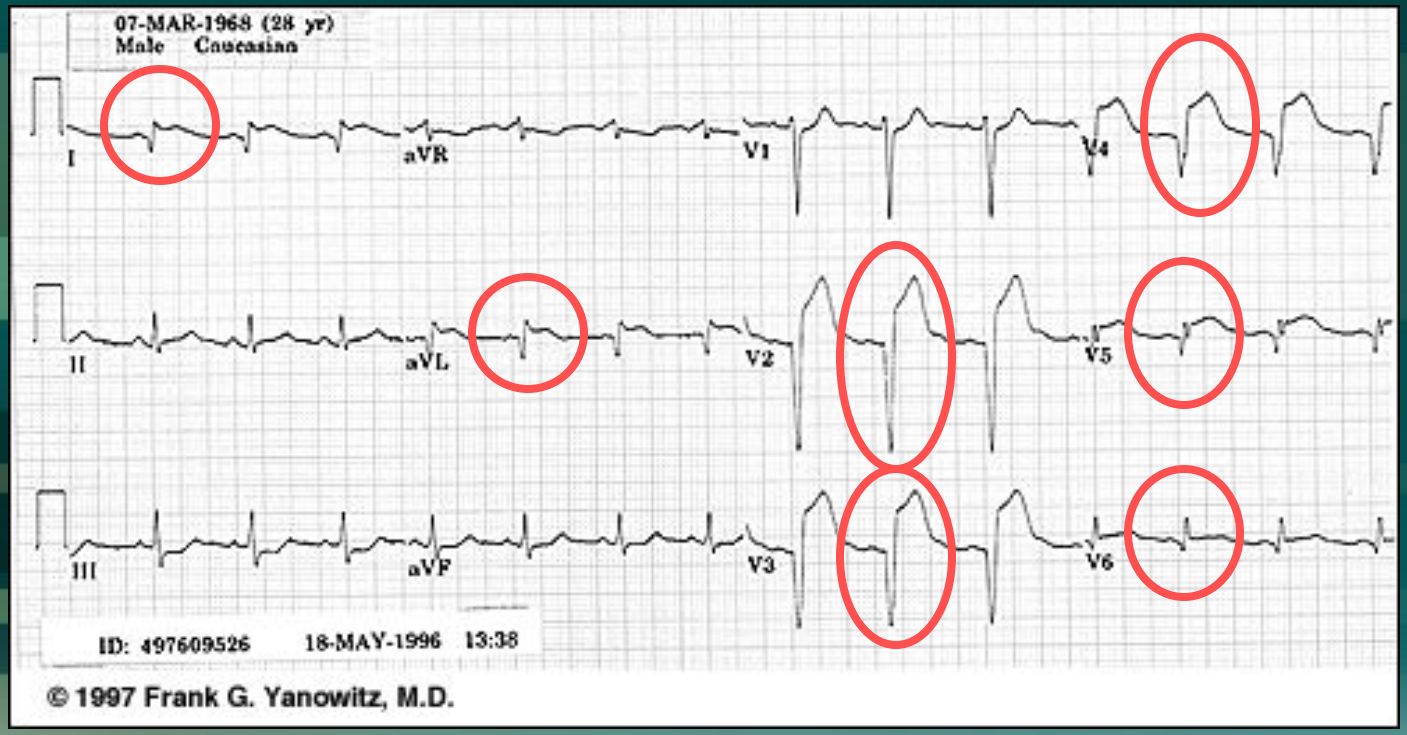
Putting it all Together

How about now?



Anterolateral MI

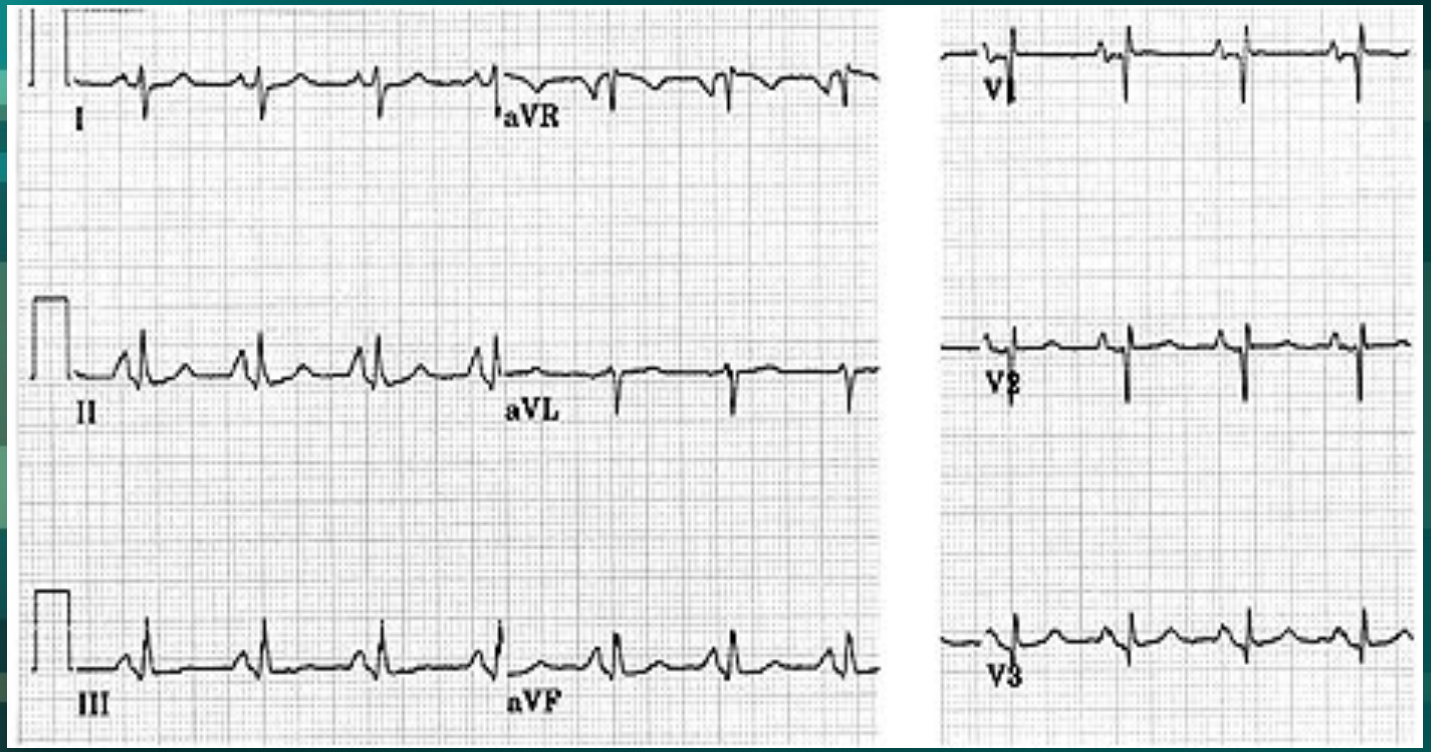
This person's MI involves both the anterior wall (V_2-V_4) and the lateral wall (V_5-V_6, I, and aVL)!



RIGHT ATRIAL ENLARGEMENT

Right atrial enlargement

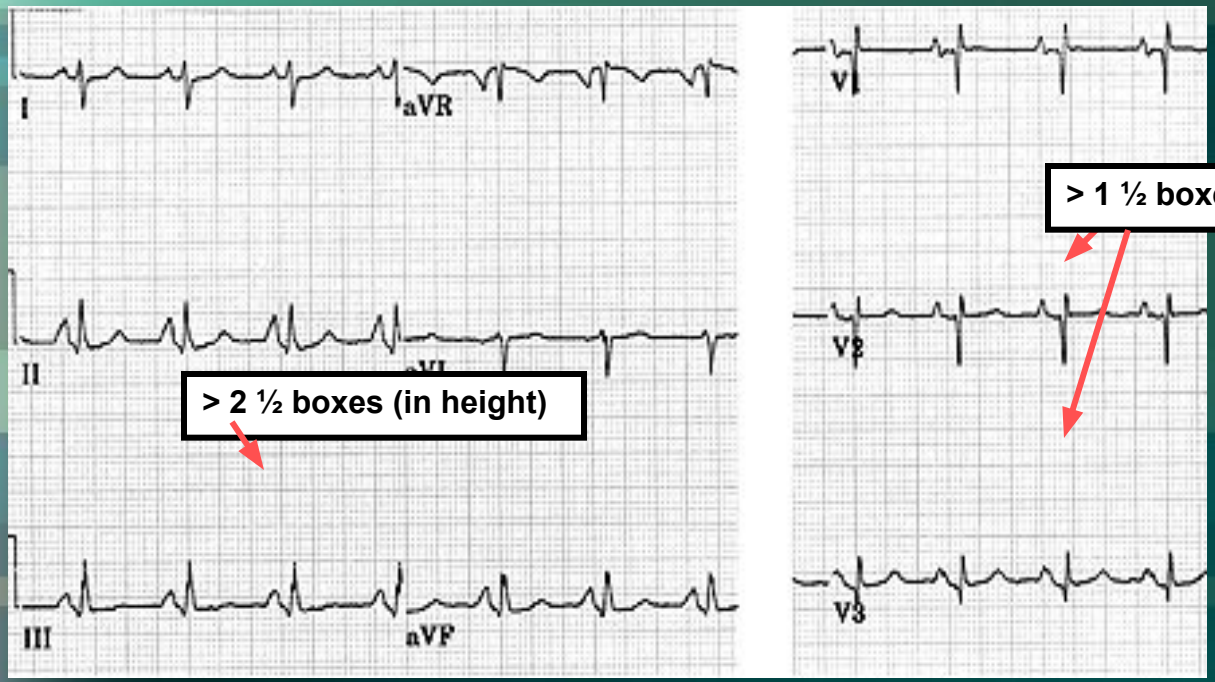
– Take a look at this ECG. What do you notice about the P waves?



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:
  - II P > 2.5 mm, or
  - V1 or V2 P > 1.5 mm

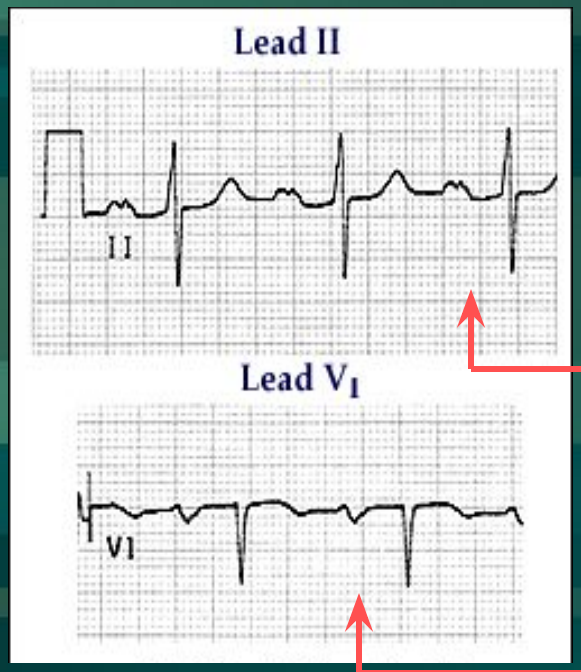


Remember 1 small box in height = 1 mm

A cause of RAE is RVH from pulmonary hypertension.

Left atrial enlargement

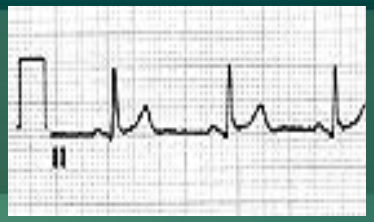
- Take a look at this ECG. What do you notice about the P waves?

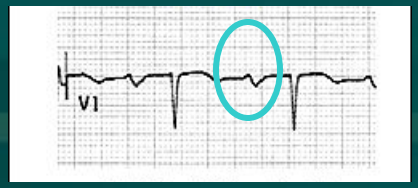


The P waves in lead II are notched and in lead V1 they have a deep and wide negative component.

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





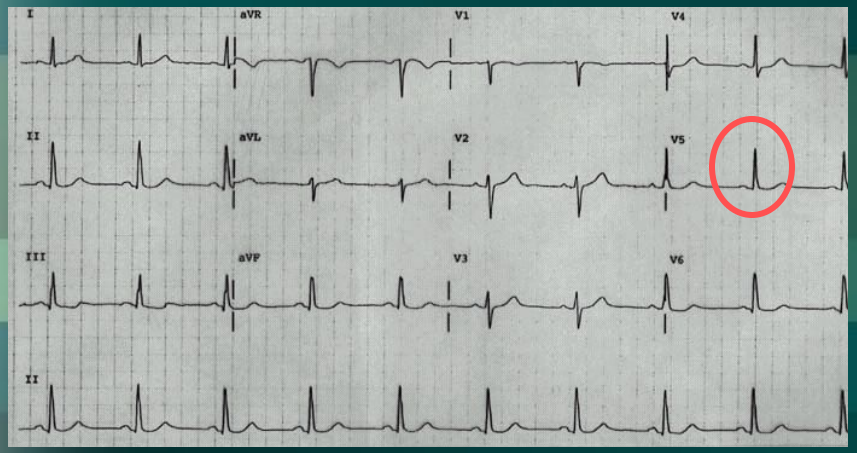
Normal *LAE*

A common cause of LAE is LVH from hypertension.

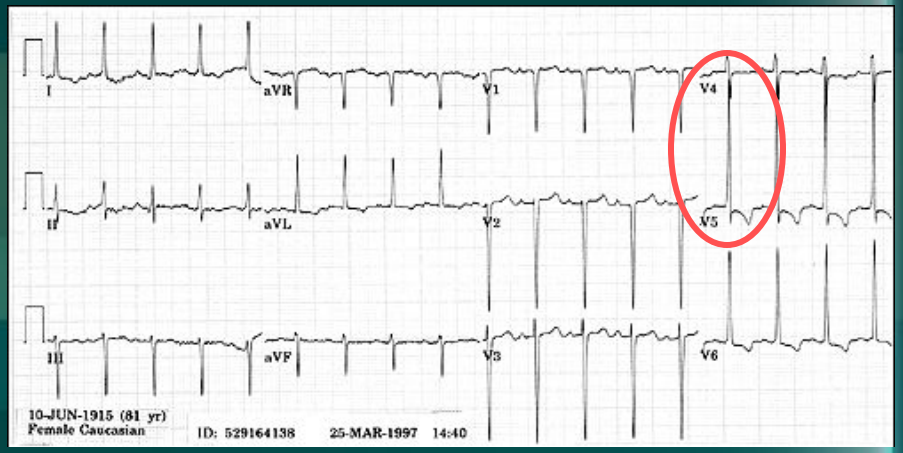
Left Ventricular Hypertrophy

Left Ventricular Hypertrophy

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



Normal



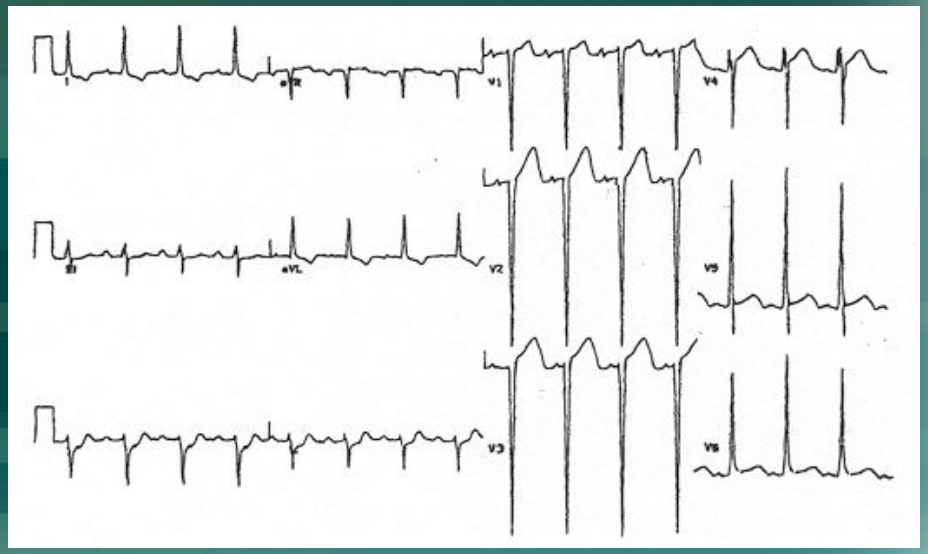
Left Ventricular Hypertrophy

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

Left Ventricular Hypertrophy

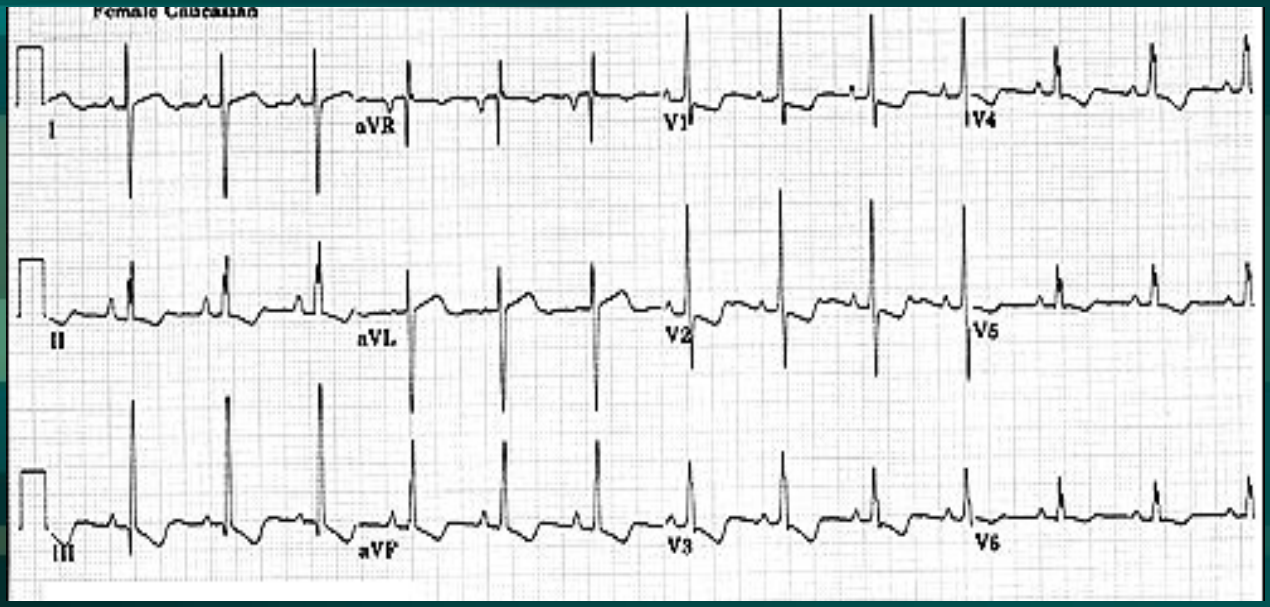
- Criteria exists to diagnose LVH using a 12-lead ECG.
  - For example:
    - The R wave in V5 or V6 plus the S wave in V1 or V2 exceeds 35 mm.

- However, for now, all you need to know is that the QRS voltage increases with LVH.



Right ventricular hypertrophy

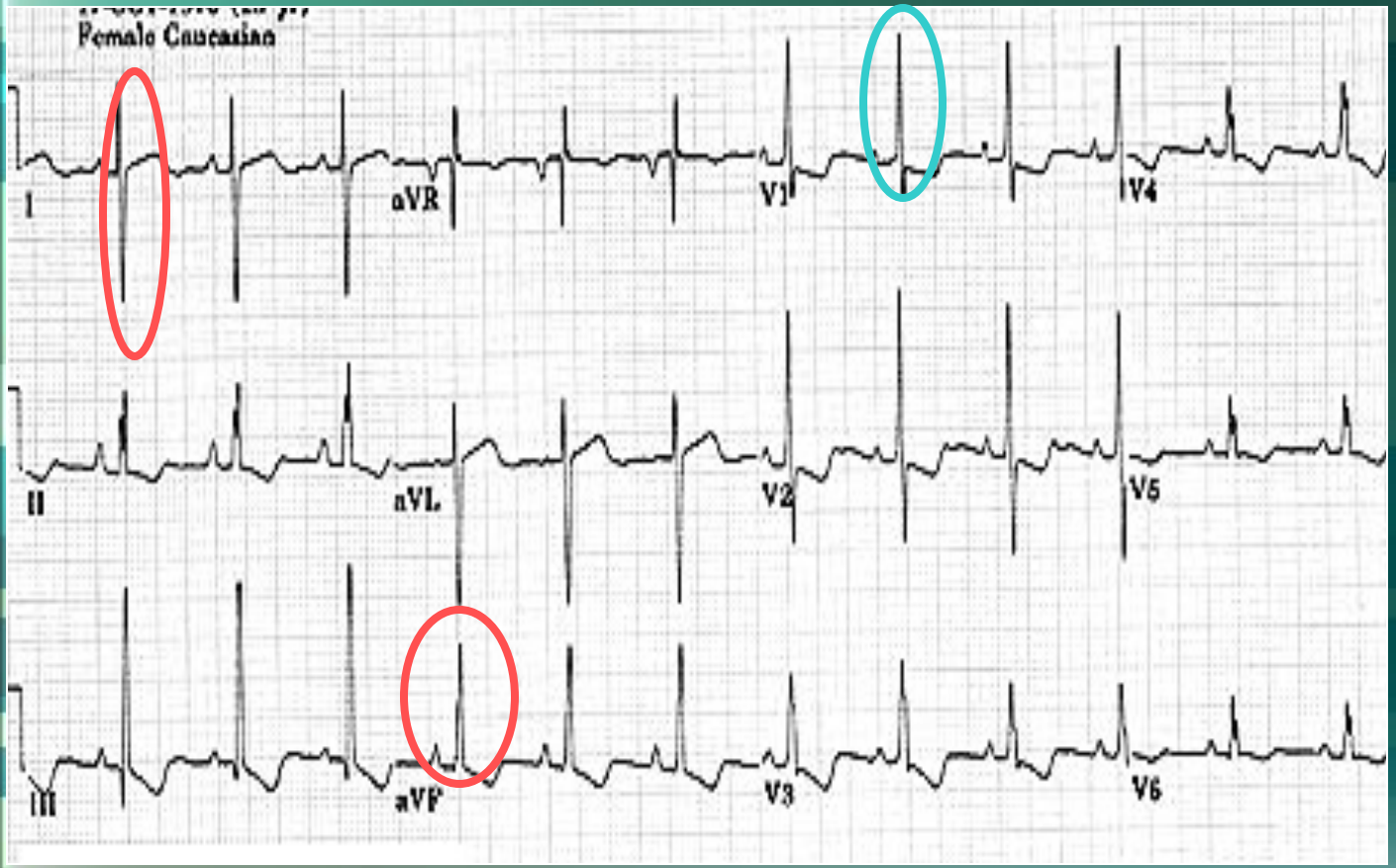
– Take a look at this ECG. What do you notice about the axis and QRS complexes over the right ventricle (V1, V2)?



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

Right ventricular hypertrophy

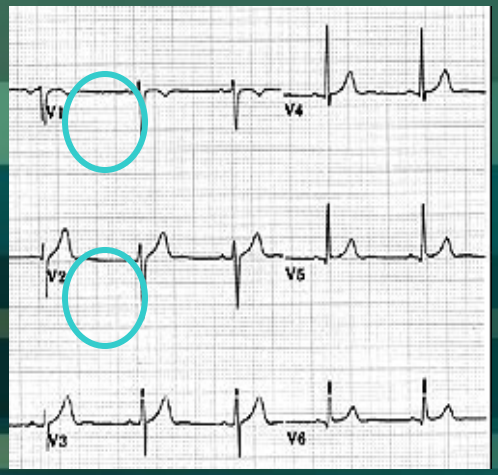
- To diagnose RVH you can use the following criteria:
  - Right axis deviation, and
  - V1 R wave > 7mm tall



A common cause of RVH is left heart failure.

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.

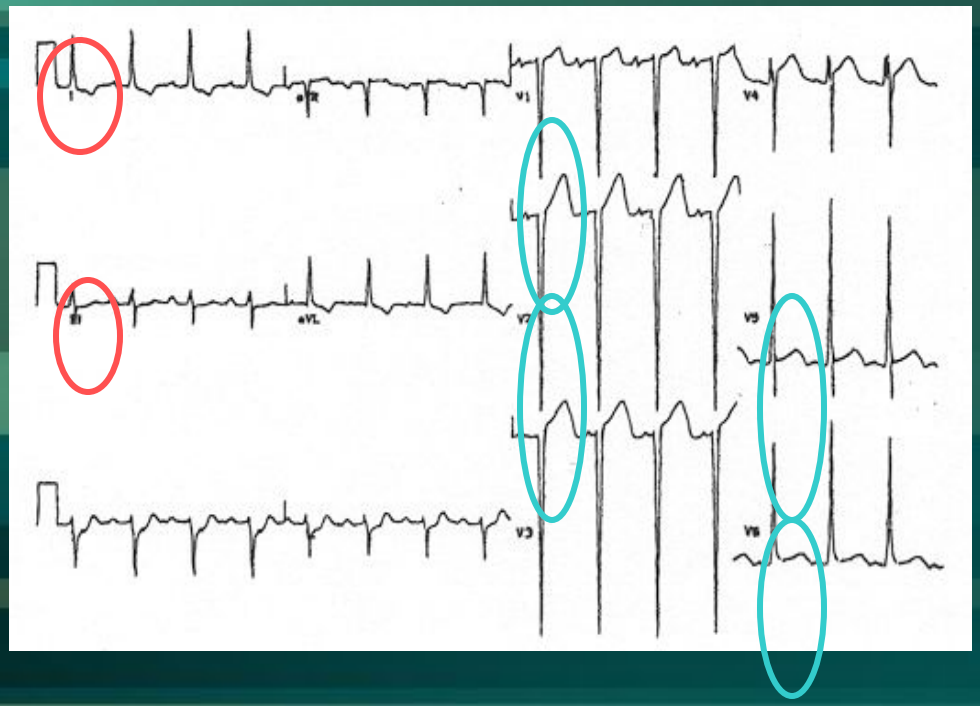


Normal

RVH

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)?

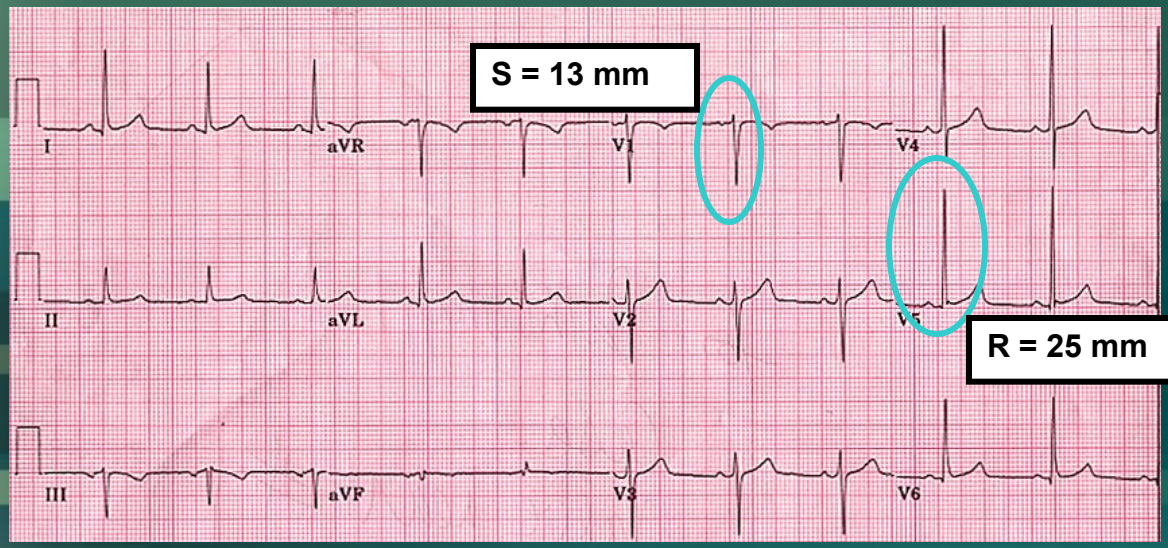


The deep S waves seen in the leads over the right ventricle are created because the heart is depolarizing left, superior and posterior (away from leads V1, V2).

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.

Bundle Branch Blocks

Normal Impulse Conduction

Sinoatrial node

↓

AV node

↓

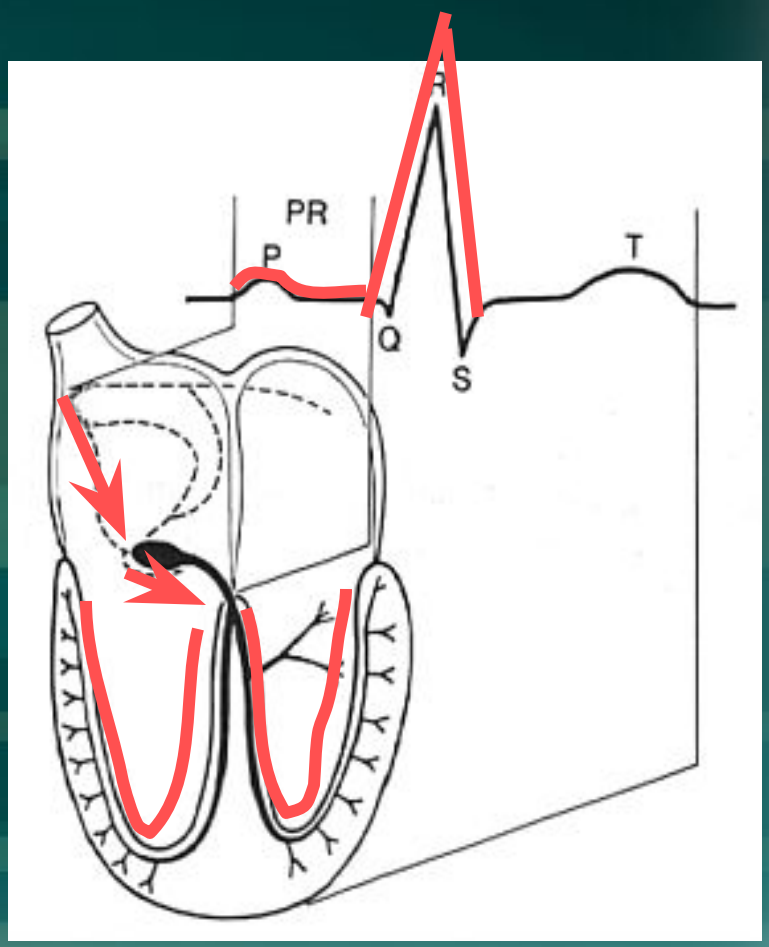
Bundle of His

↓

Bundle Branches

↓

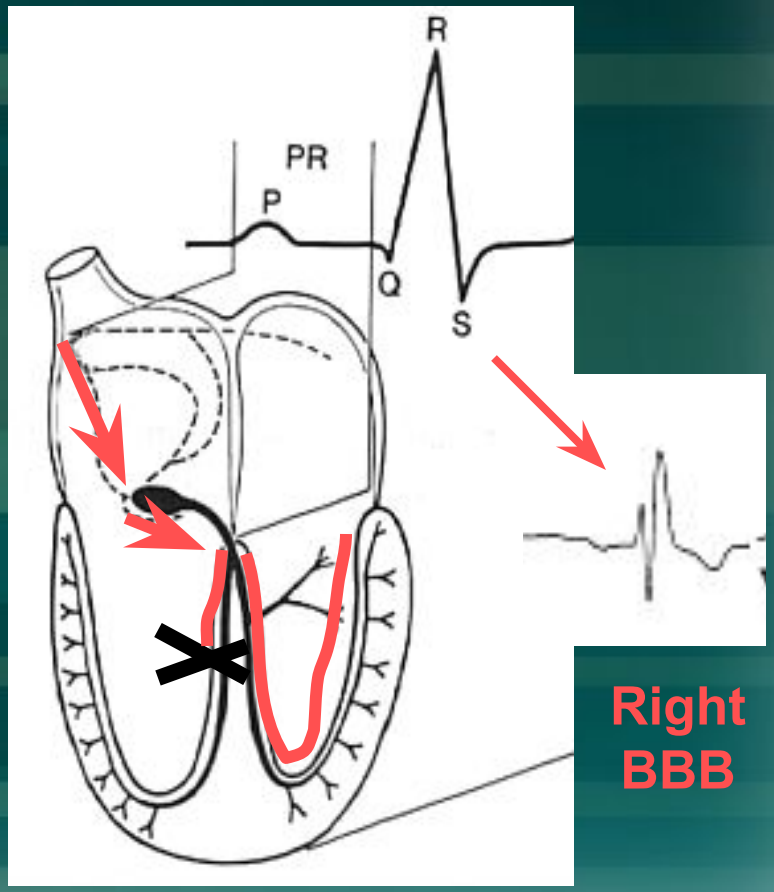
Purkinje fibers



Bundle Branch Blocks

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

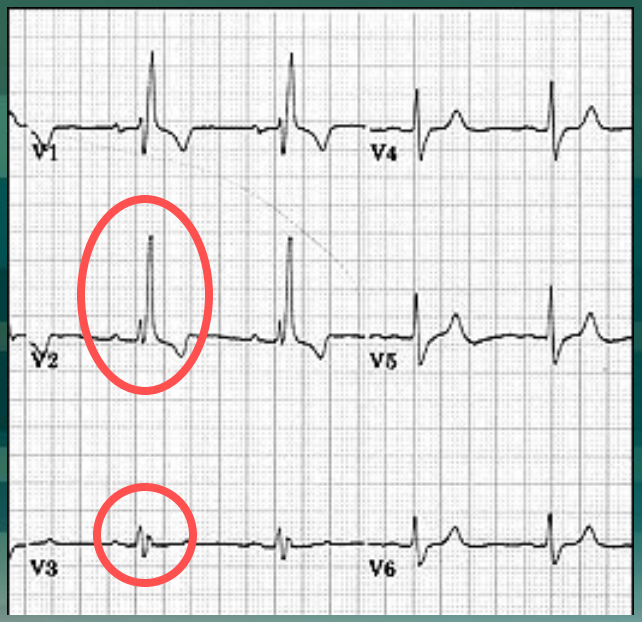
Therefore, a conduction block of the Bundle Branches would be reflected as a change in the QRS complex.



Bundle Branch Blocks

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).**



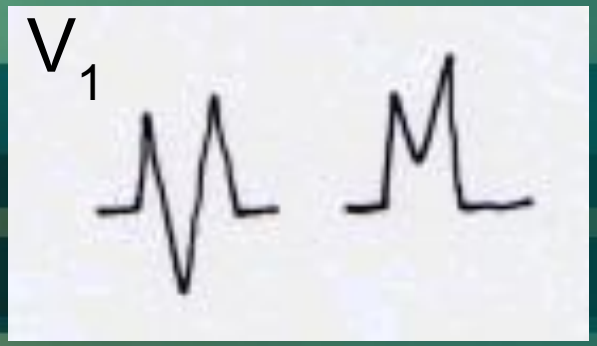
RBBB vs LBBB

- RBBB in V1

Right Bundle Branch Blocks

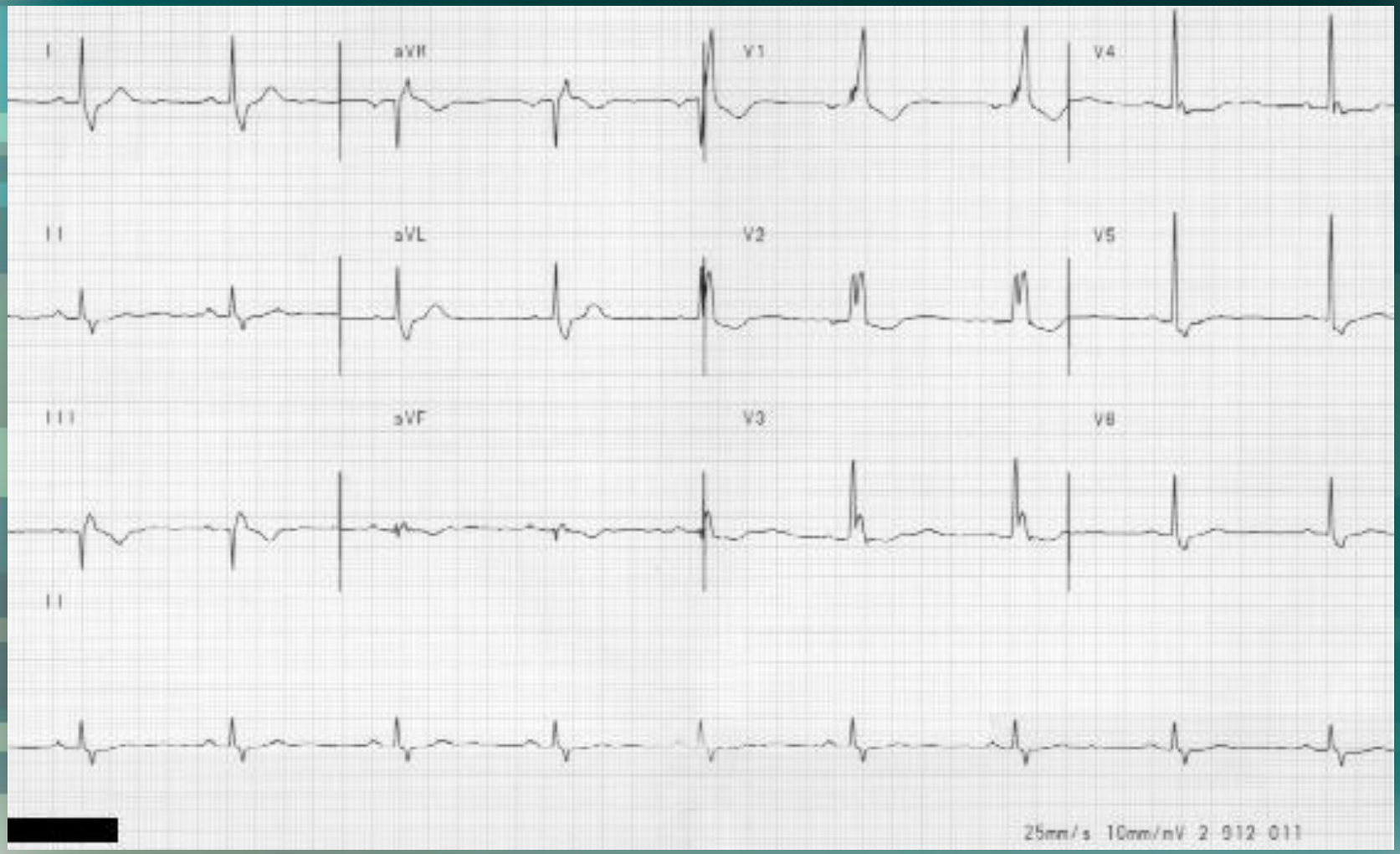
What QRS morphology is characteristic?

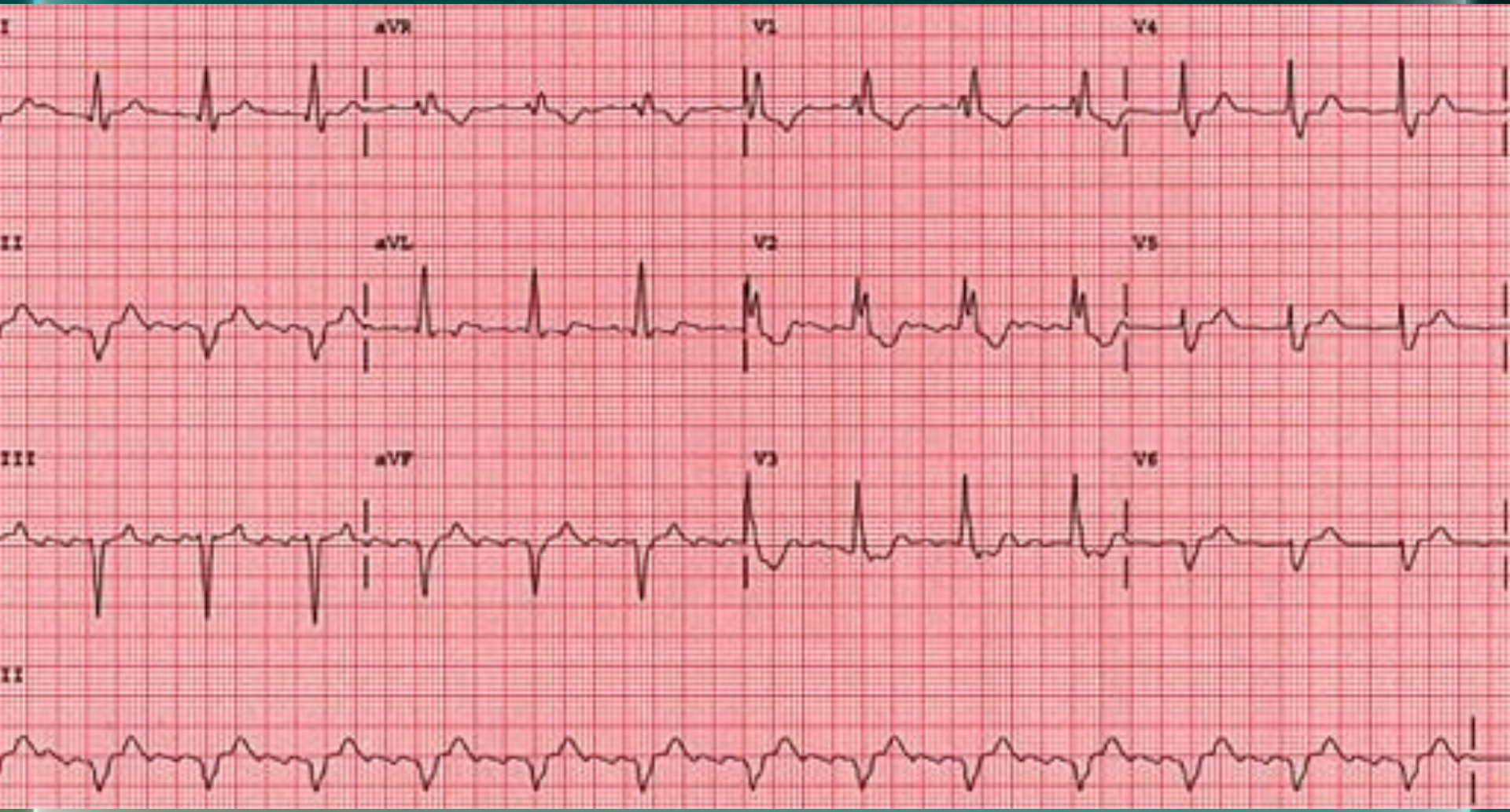
For RBBB the wide QRS complex assumes a unique, virtually diagnostic shape in those leads overlying the right ventricle (V_1 and V_2).



"Rabbit Ears"

RBBB



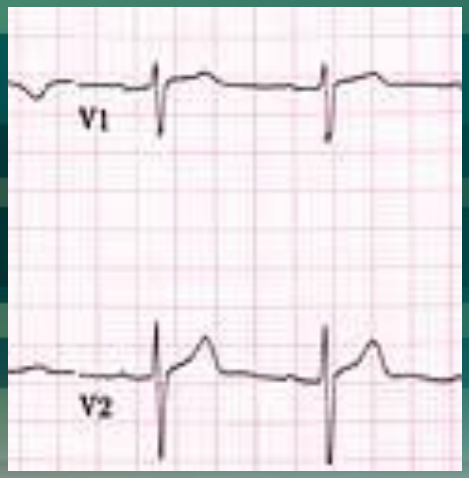

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

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

RBBB vs. LBBB

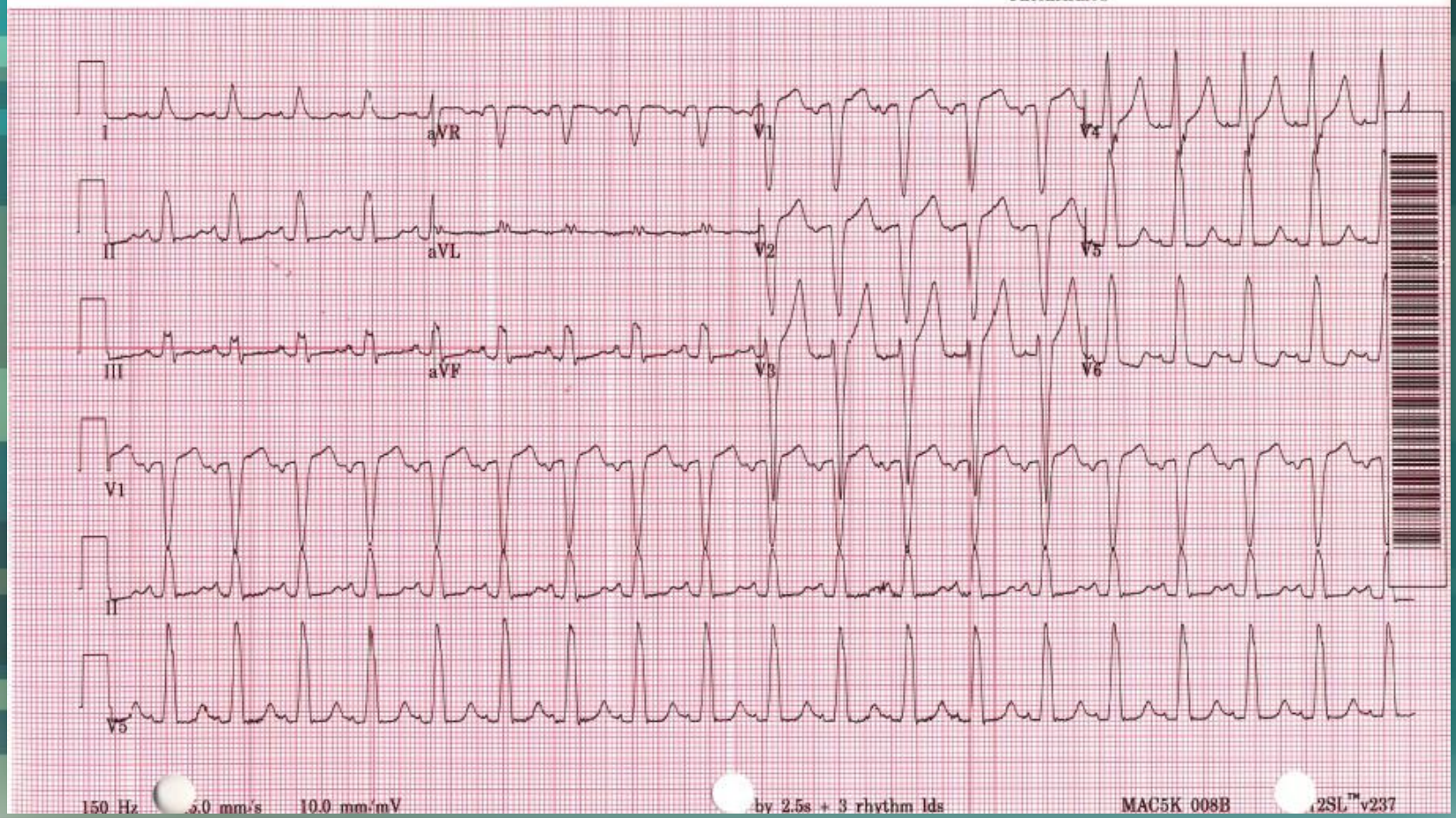
- LBBB in V1

19-Jul-2007 6:45:56

58years

Vent. rate	116 bpm	Sinus tachycardia
PR interval	132 ms	Possible Left atrial enlargement
QRS duration	132 ms	Left bundle branch block
QT/QTc	382/530 ms	Abnormal ECG
P-R-T axes	69 48 70	

Unconfirmed



Bundle Branch Block

- V1

BBB Recognition

- Wide QRS
  - ≥ 120ms
- Supraventricular rhythm

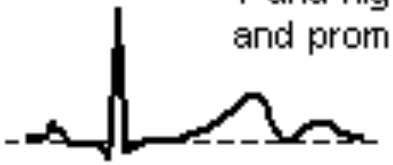
severe hypokalemia

depressed and sagging ST, low T and high U. longer PR interval and prominent P wave

hyperkalemia + hypocalcemia

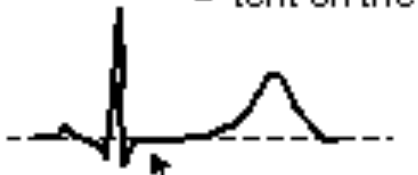
long QT + peaked T = 'tent on the desert'

hypokalemia



U wave, long PT interval

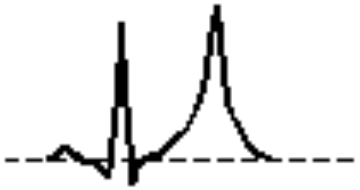
hypocalcemia



long QT

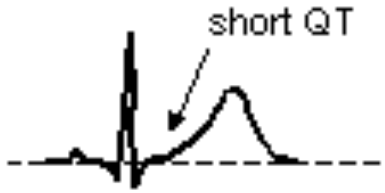
normal

hyperkalemia



Tall, peaked and narrow T

short QT



hypercalcemia

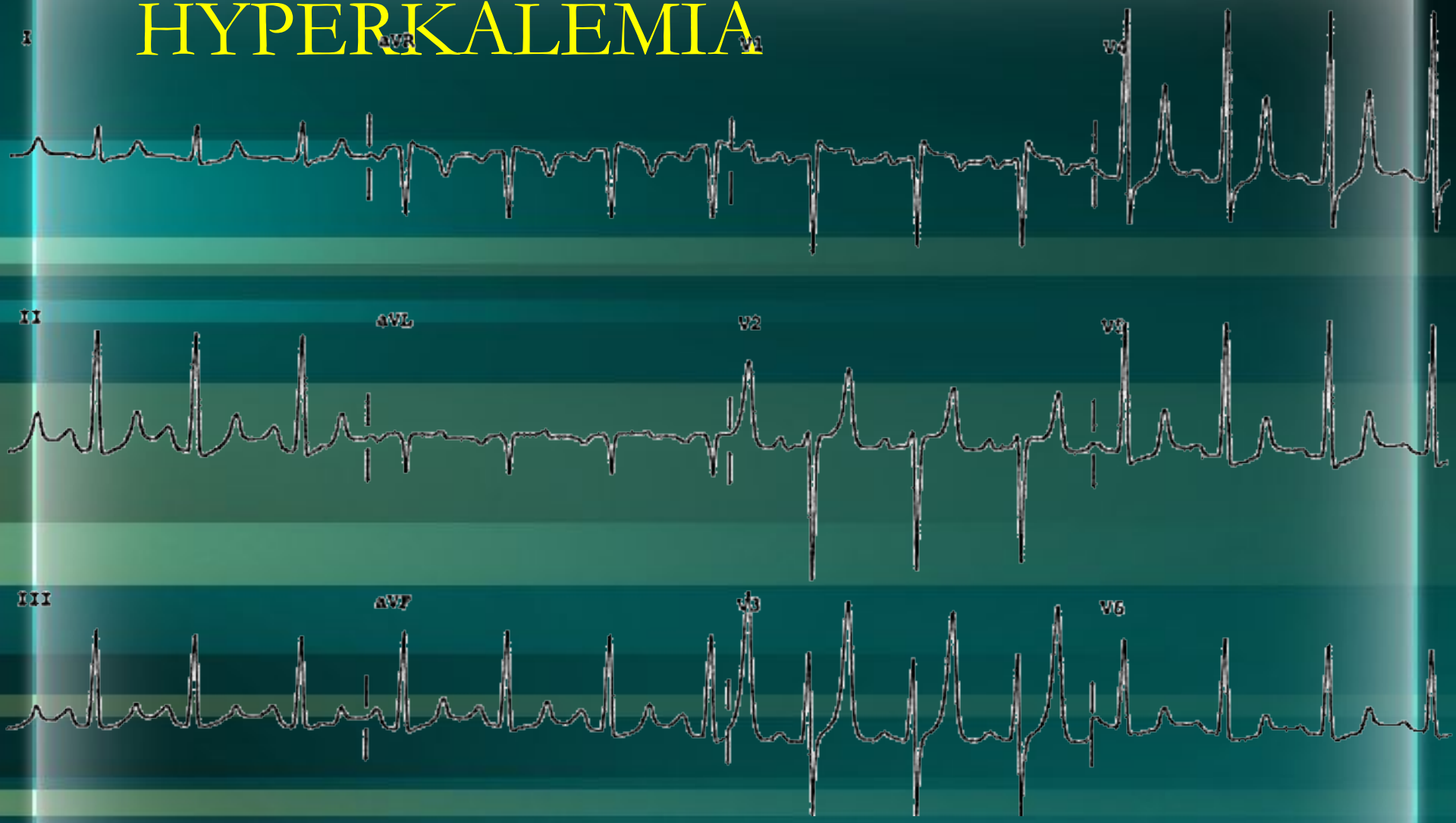
severe hyperkalemia

flattening of P wave. wide QRS and tall T = 'sine wave'

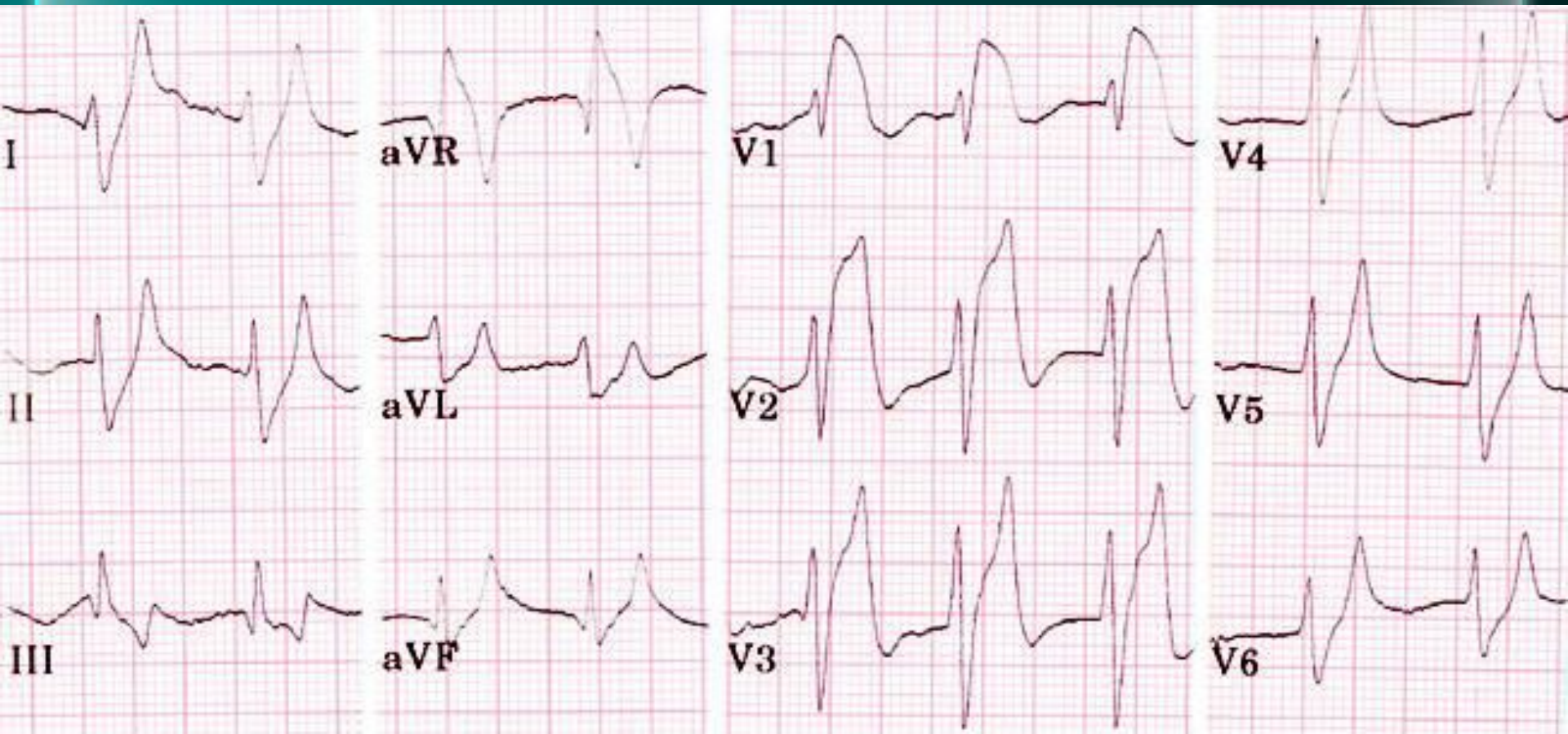
cf) ischemic T

tall T but base is wider than hyperkalemia

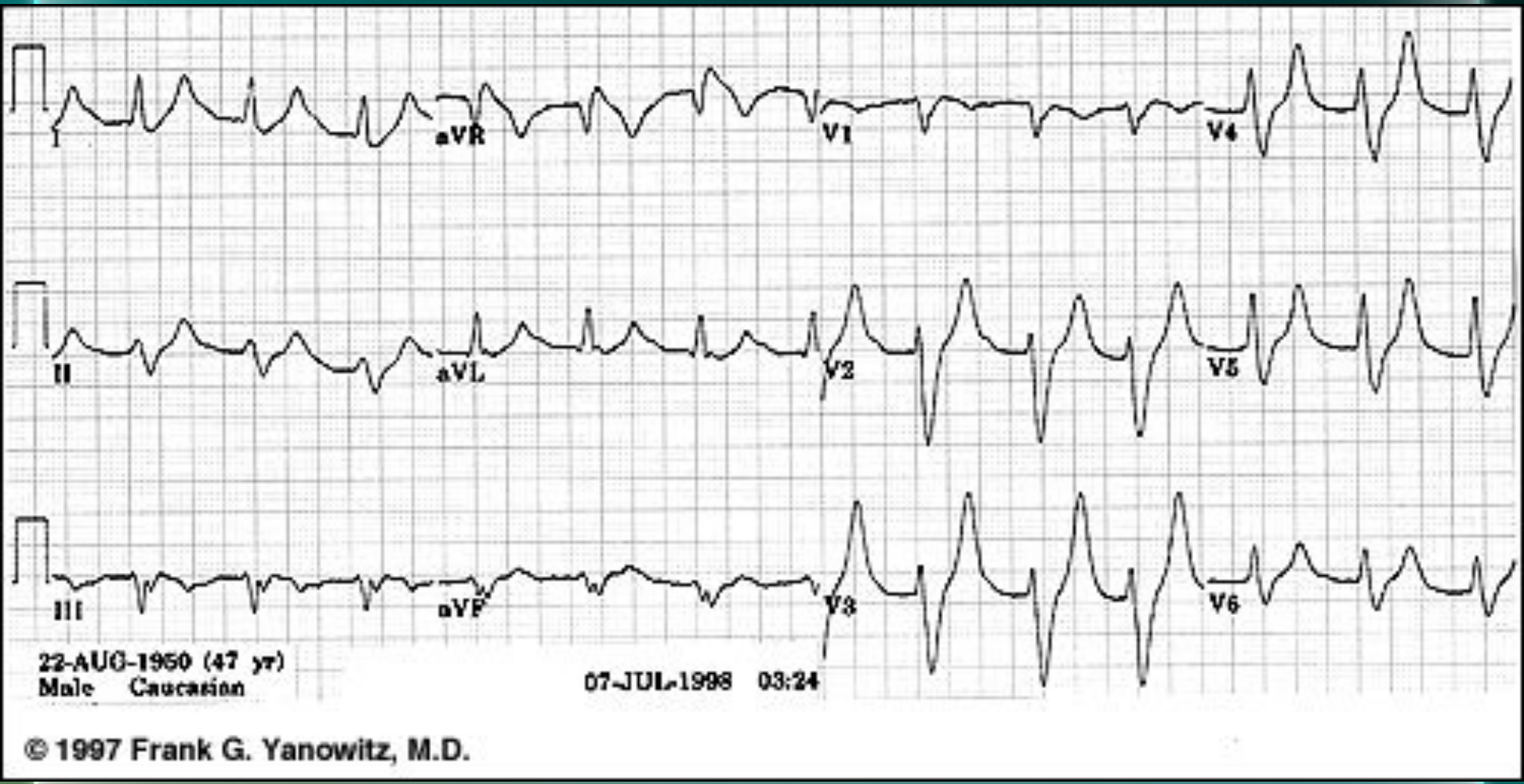
HYPERKALEMIA



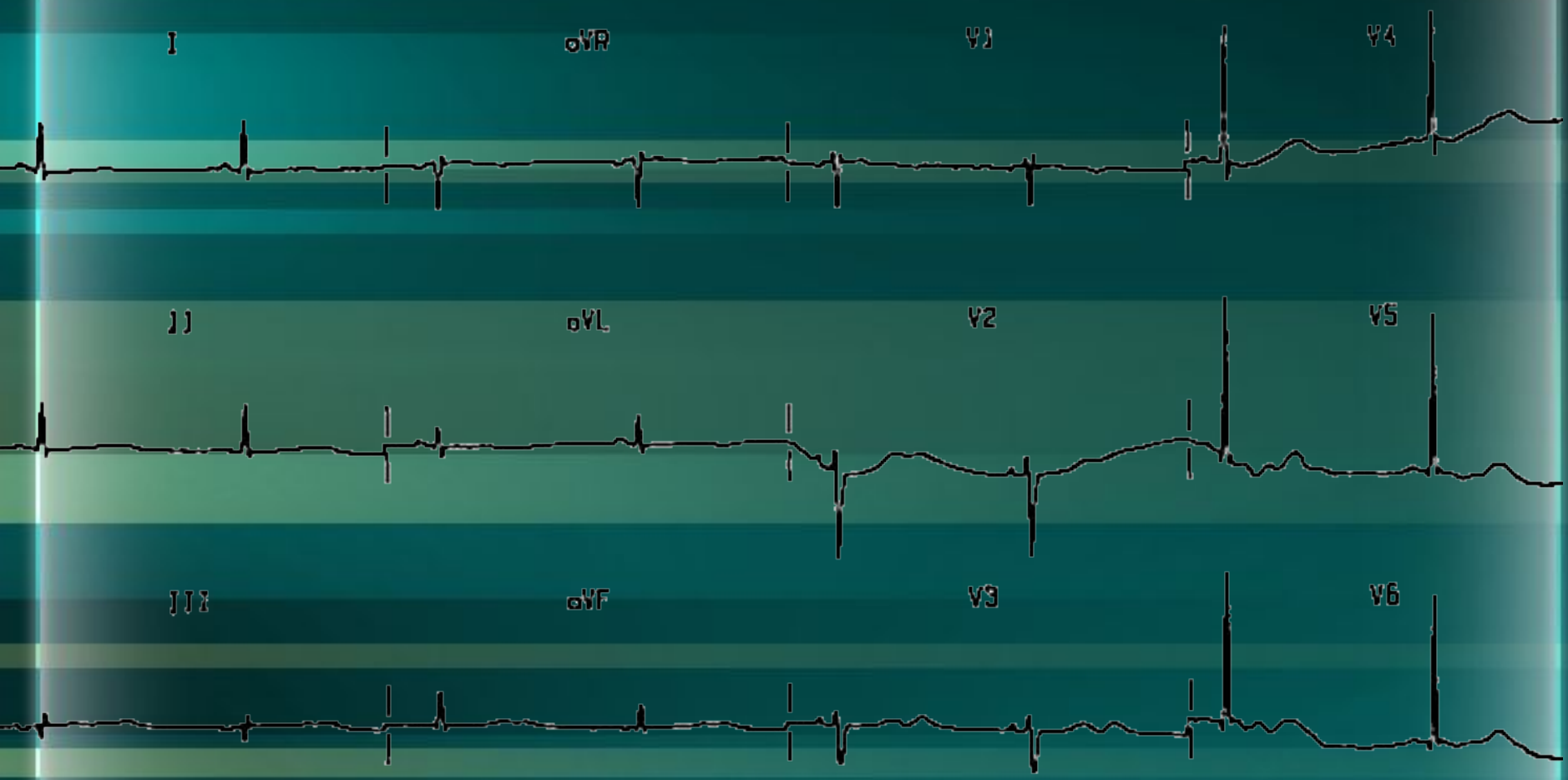
HYPERKALEMIA


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

SEVERE HYPERKALEMIA



HYPOKALEMIA



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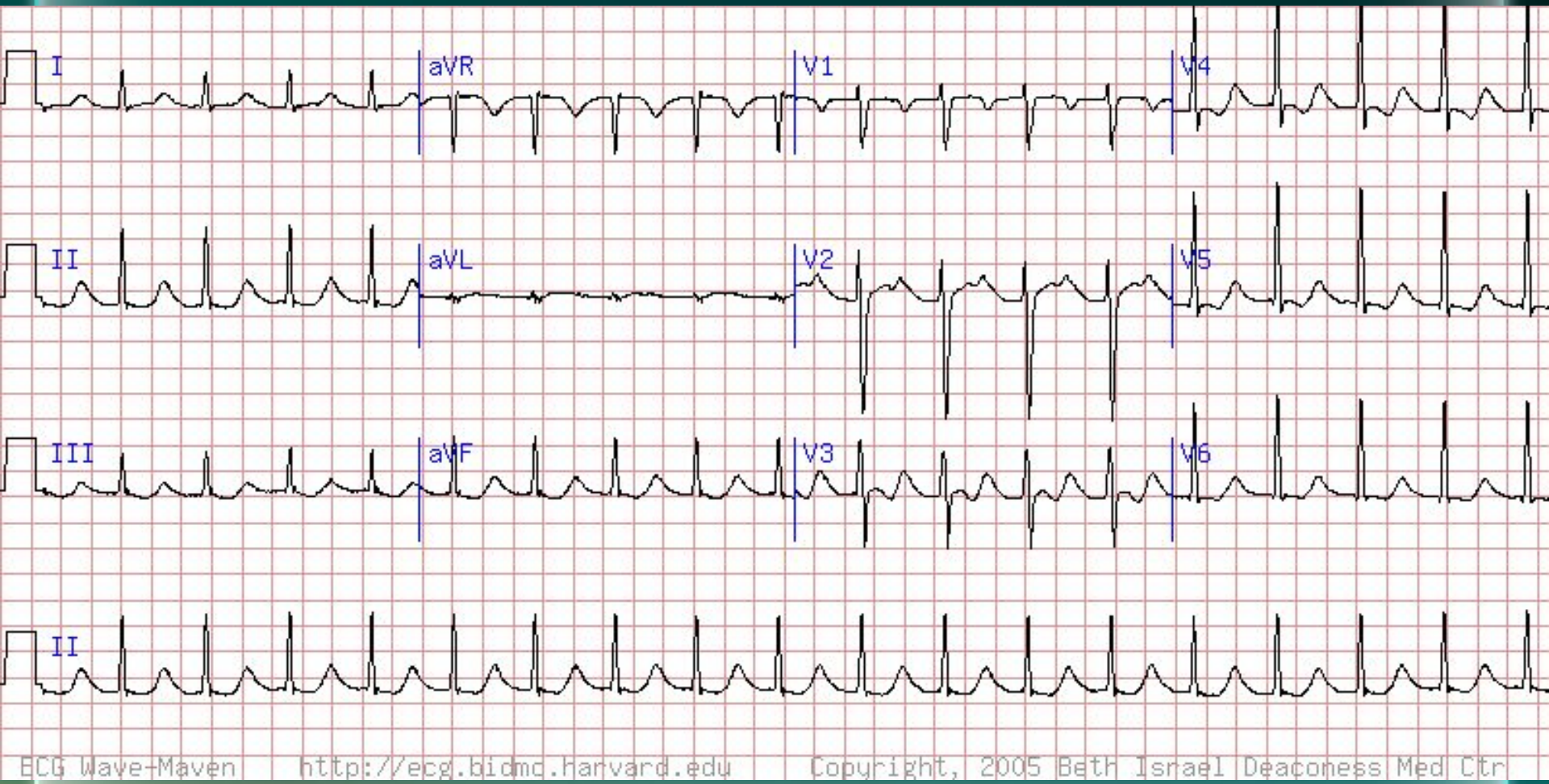
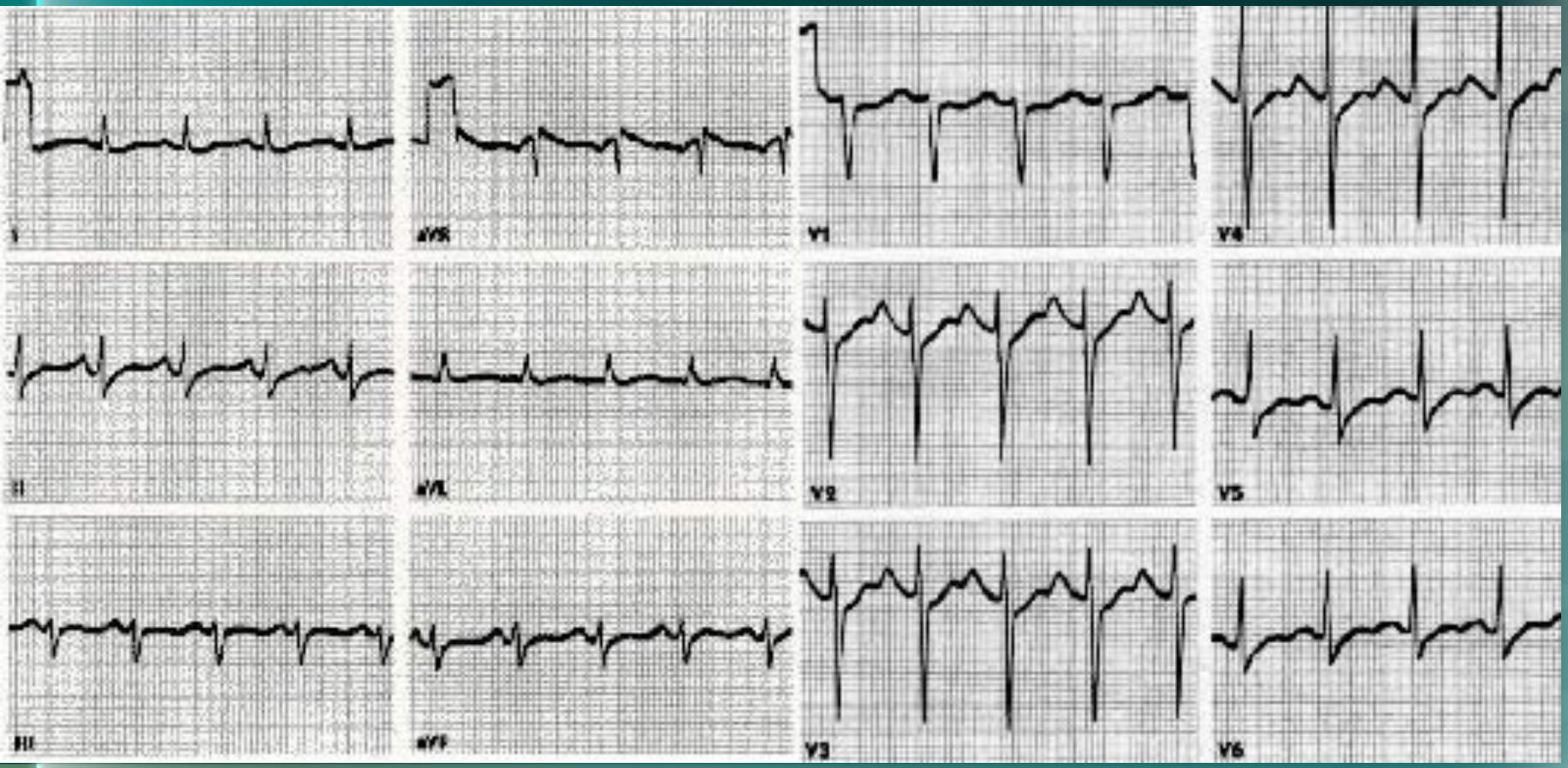
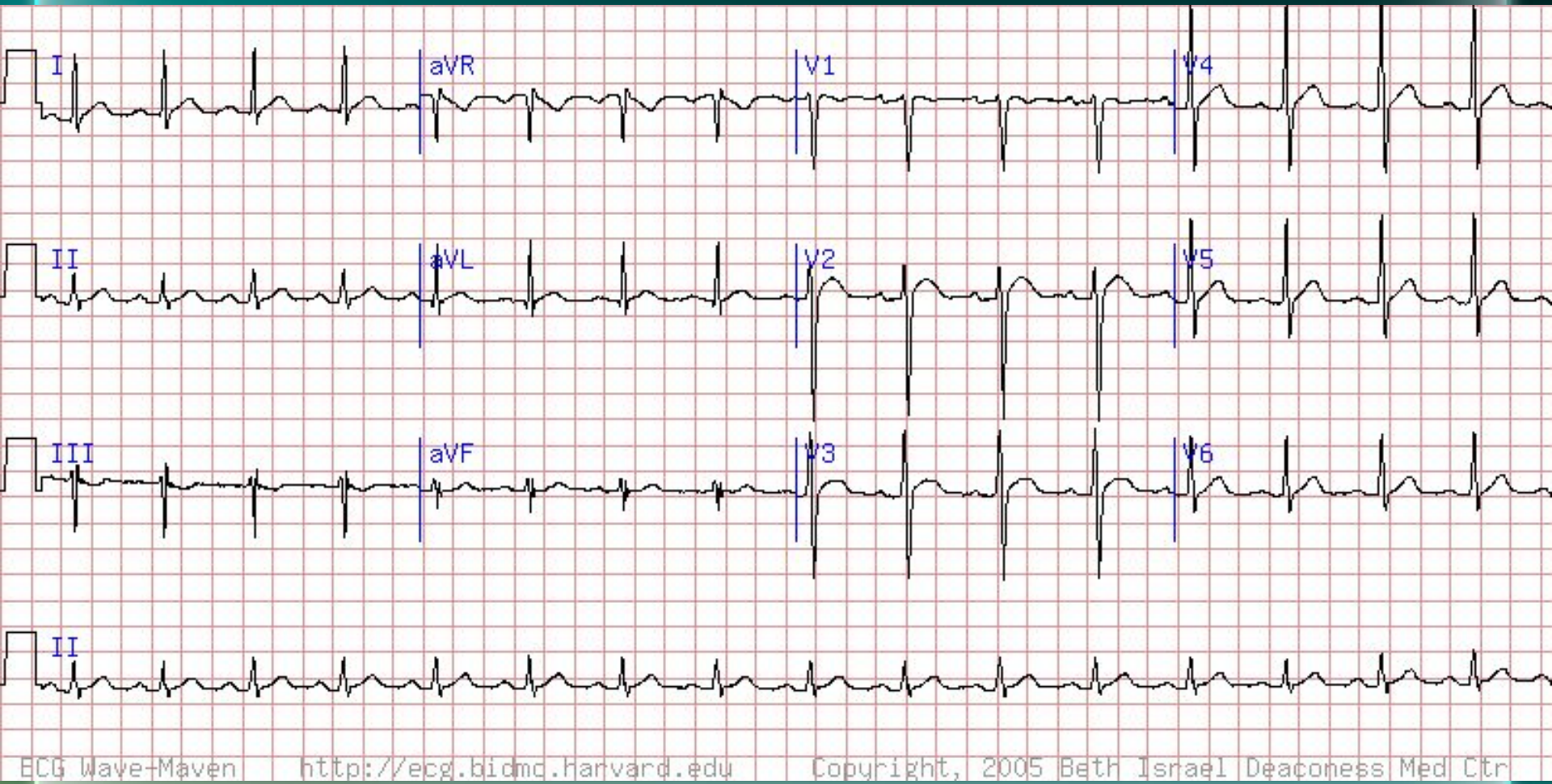


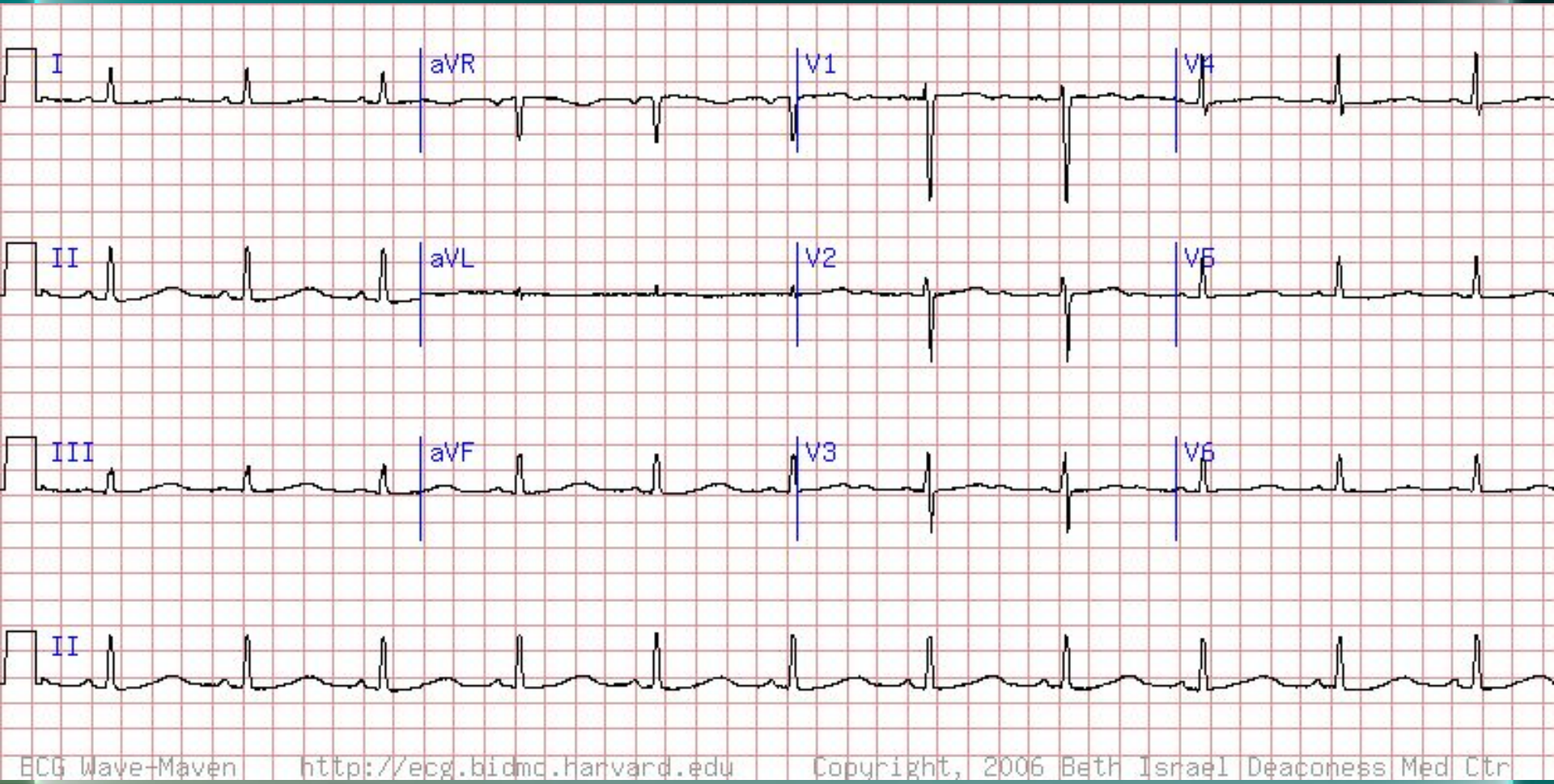
HYPOKALEMIA



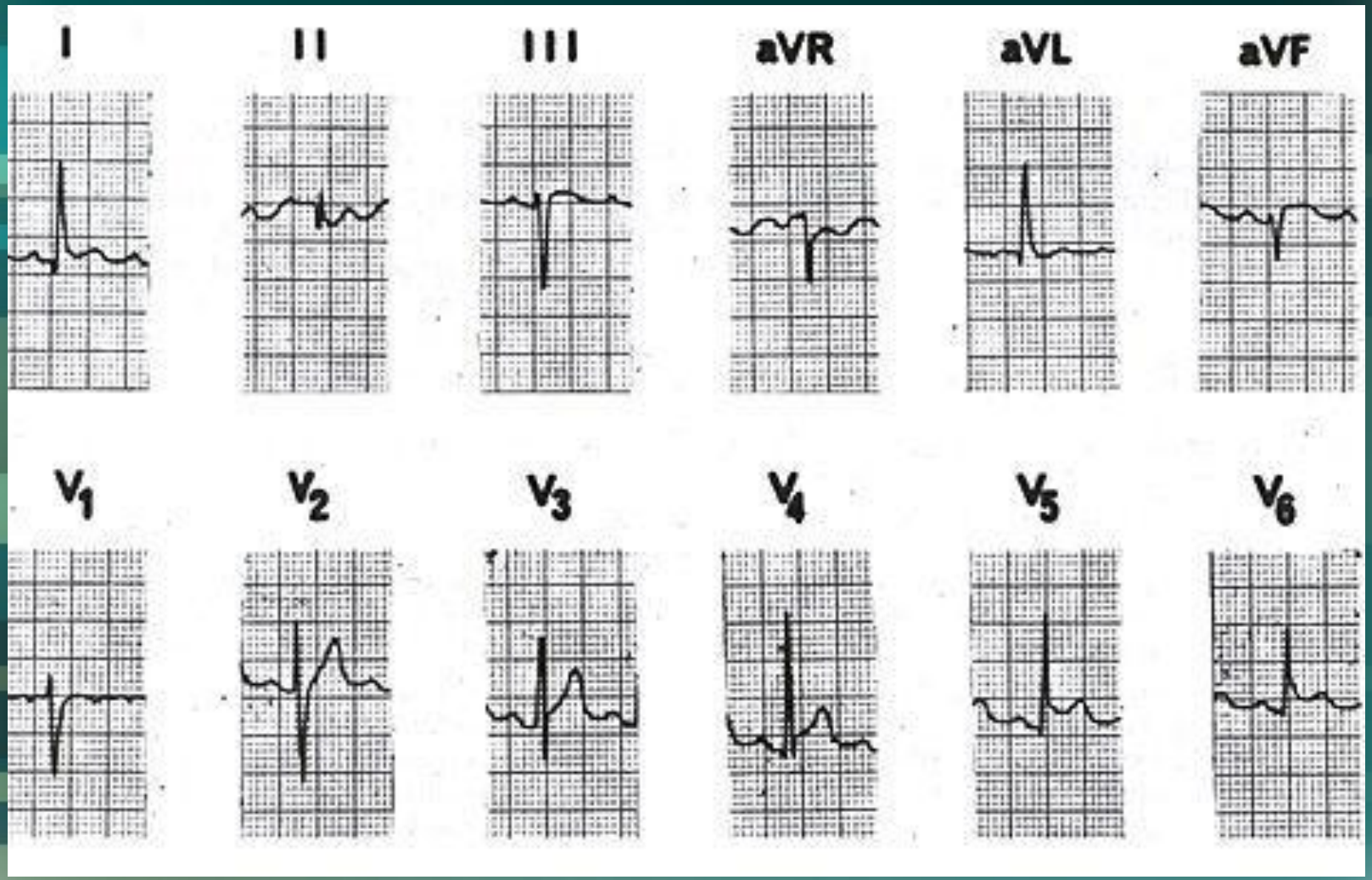
HYPERCALCEMIA



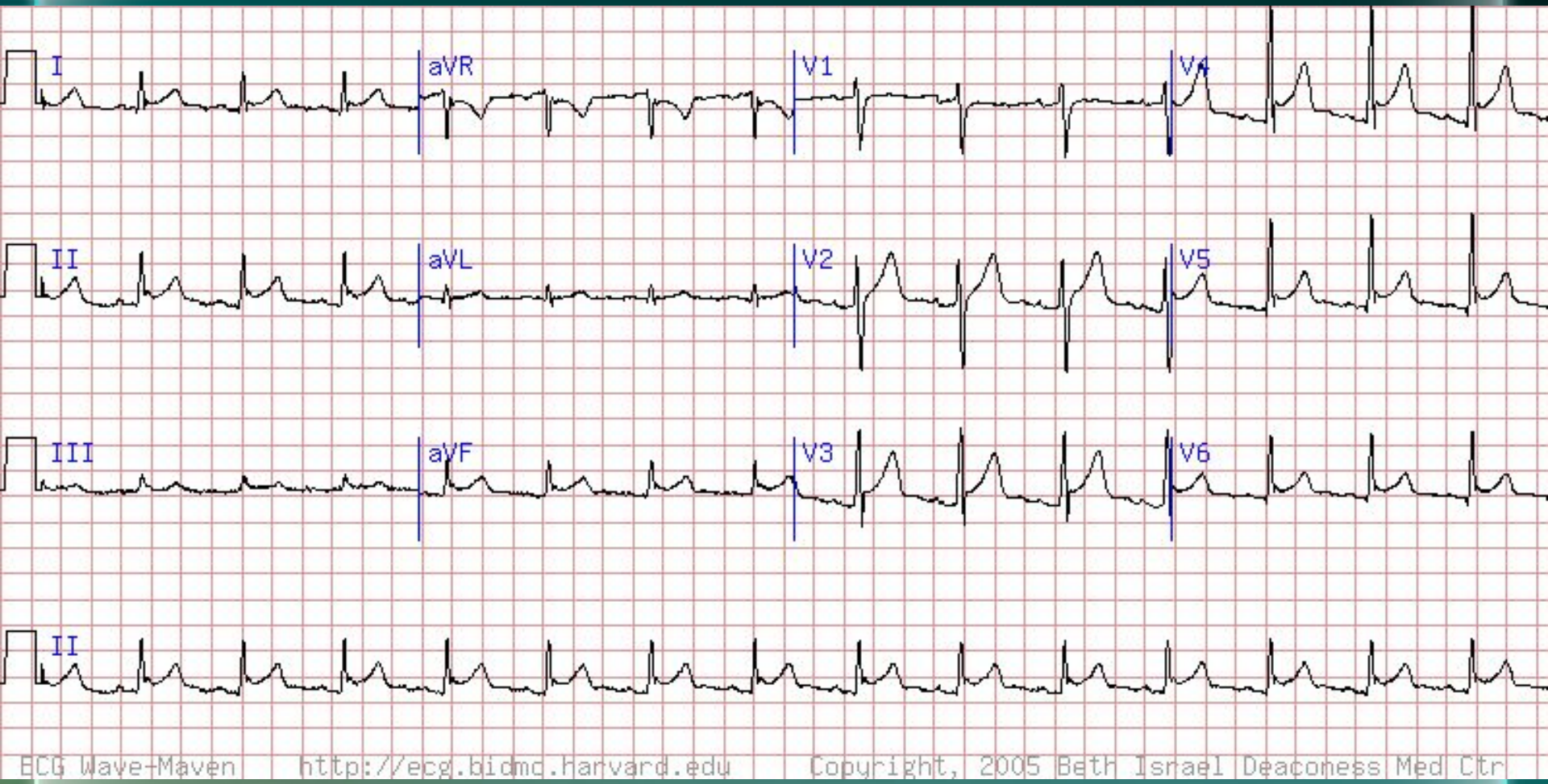
HYPOCALCEMIA



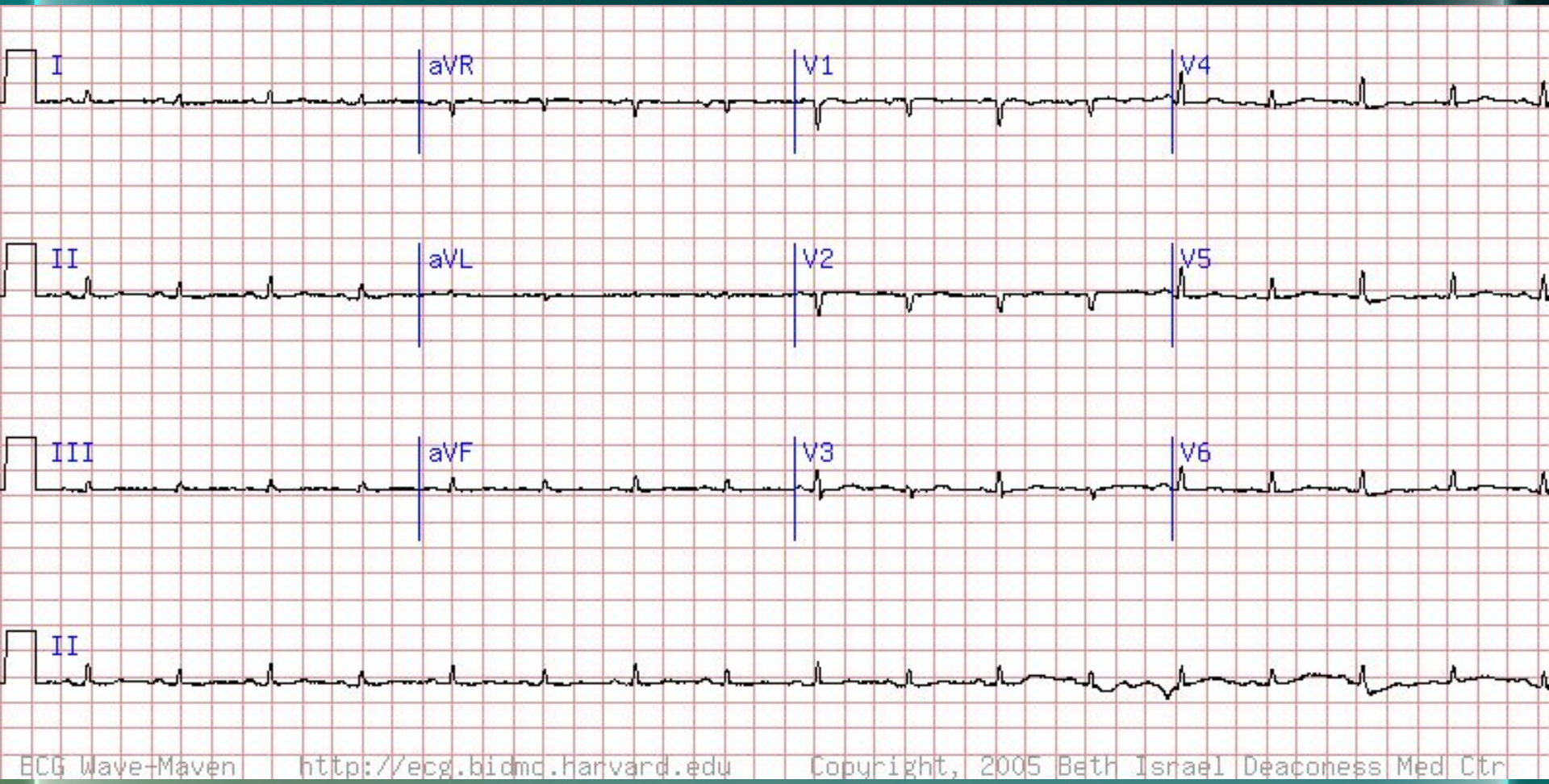
ACUTE PERICARDITIS



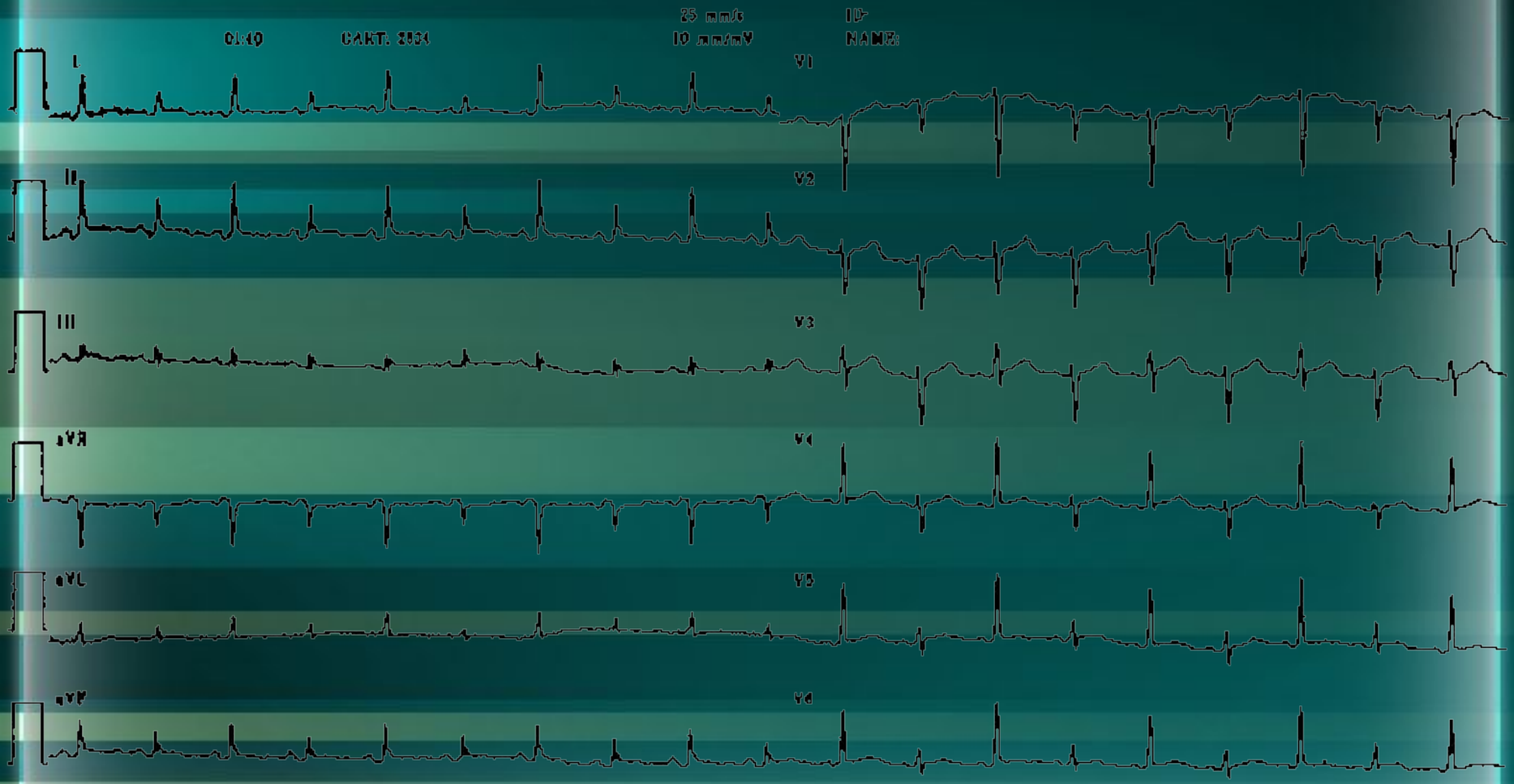
ACUTE PERICARDITIS



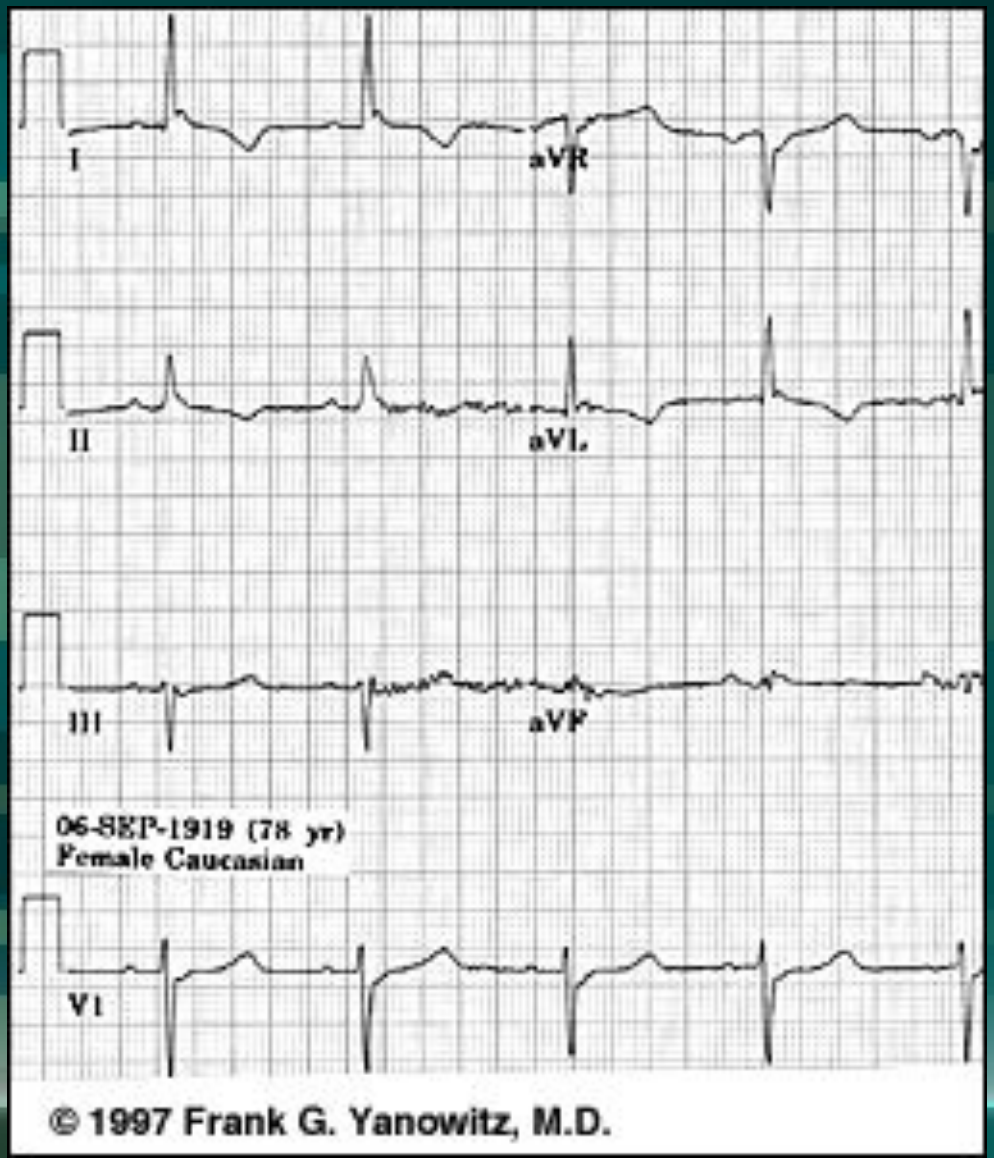
CARDIAC TAMPONADE



PERICARDIAL EFFUSION-Electrical alterans



HYPOTHERMIA-OSBORNE WAVE



HYPOTHERMIA-
Giant Osborne waves