

Electrocardiography

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Interventional Electrophysiologist

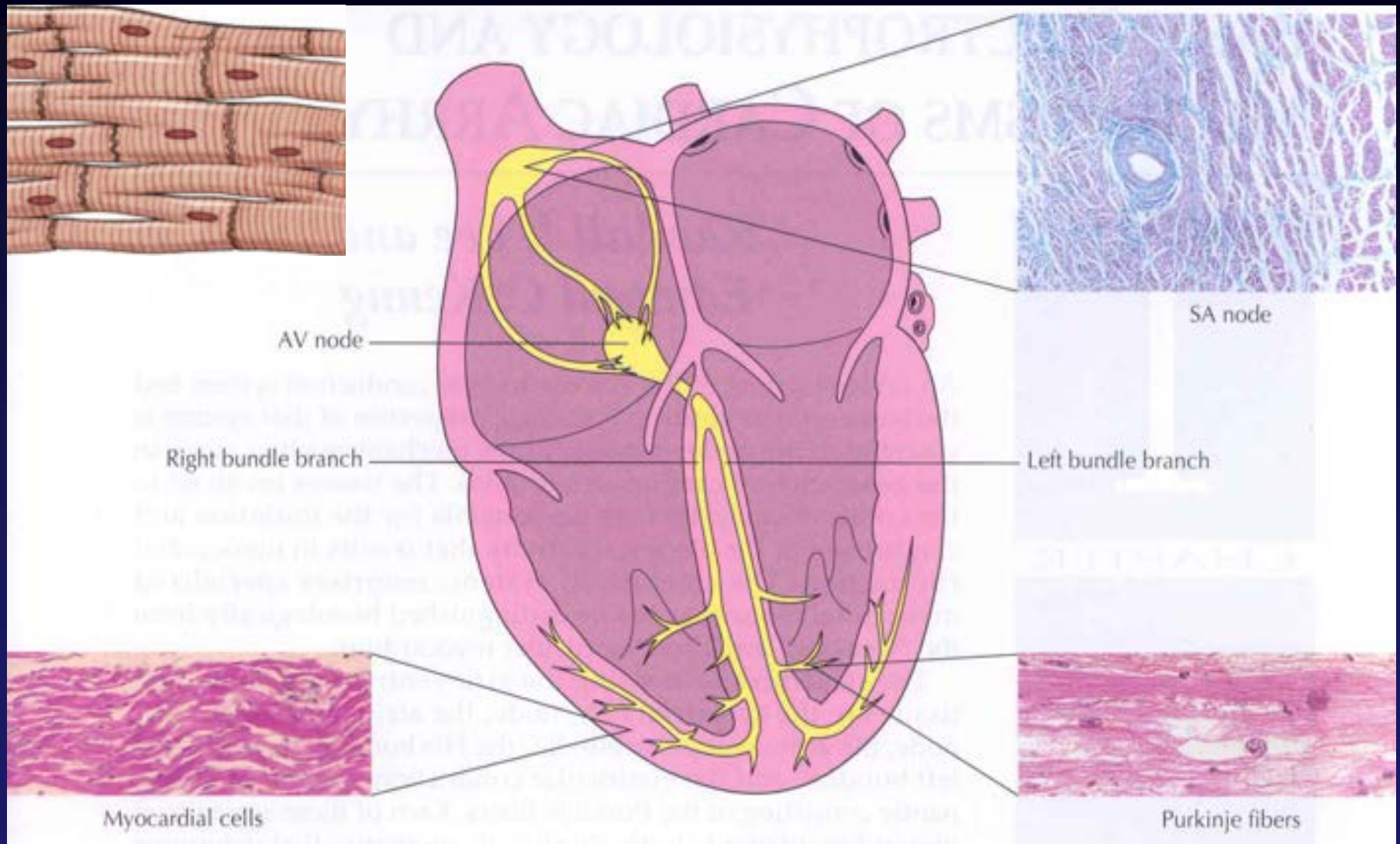
Tehran Arrhythmia Clinic

ECG

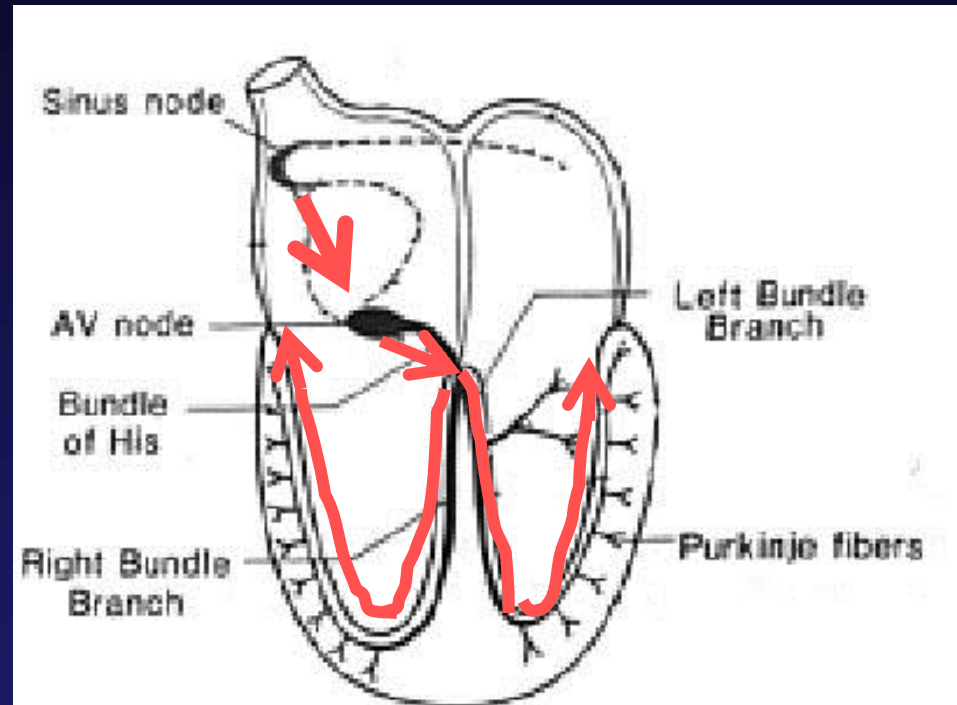
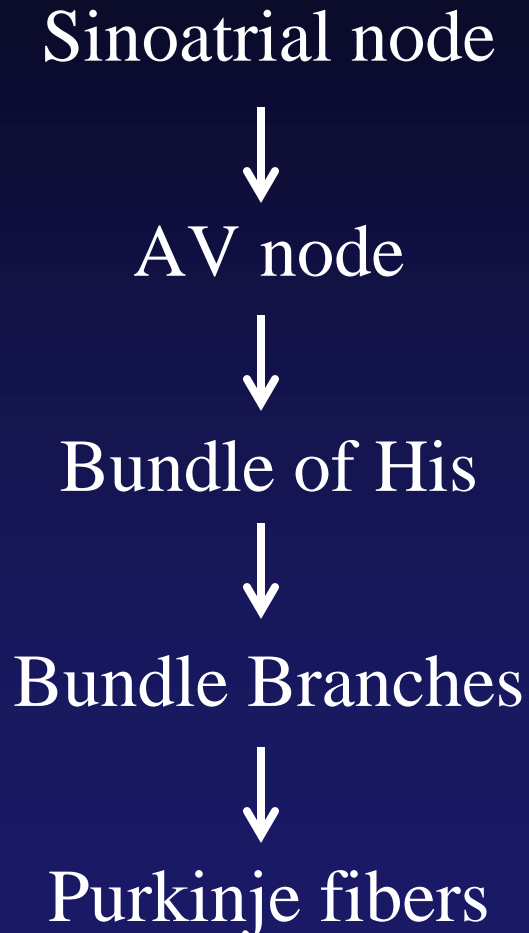
A graphic recording of electrical potentials
generated by the heart

A noninvasive, inexpensive and highly
versatile test

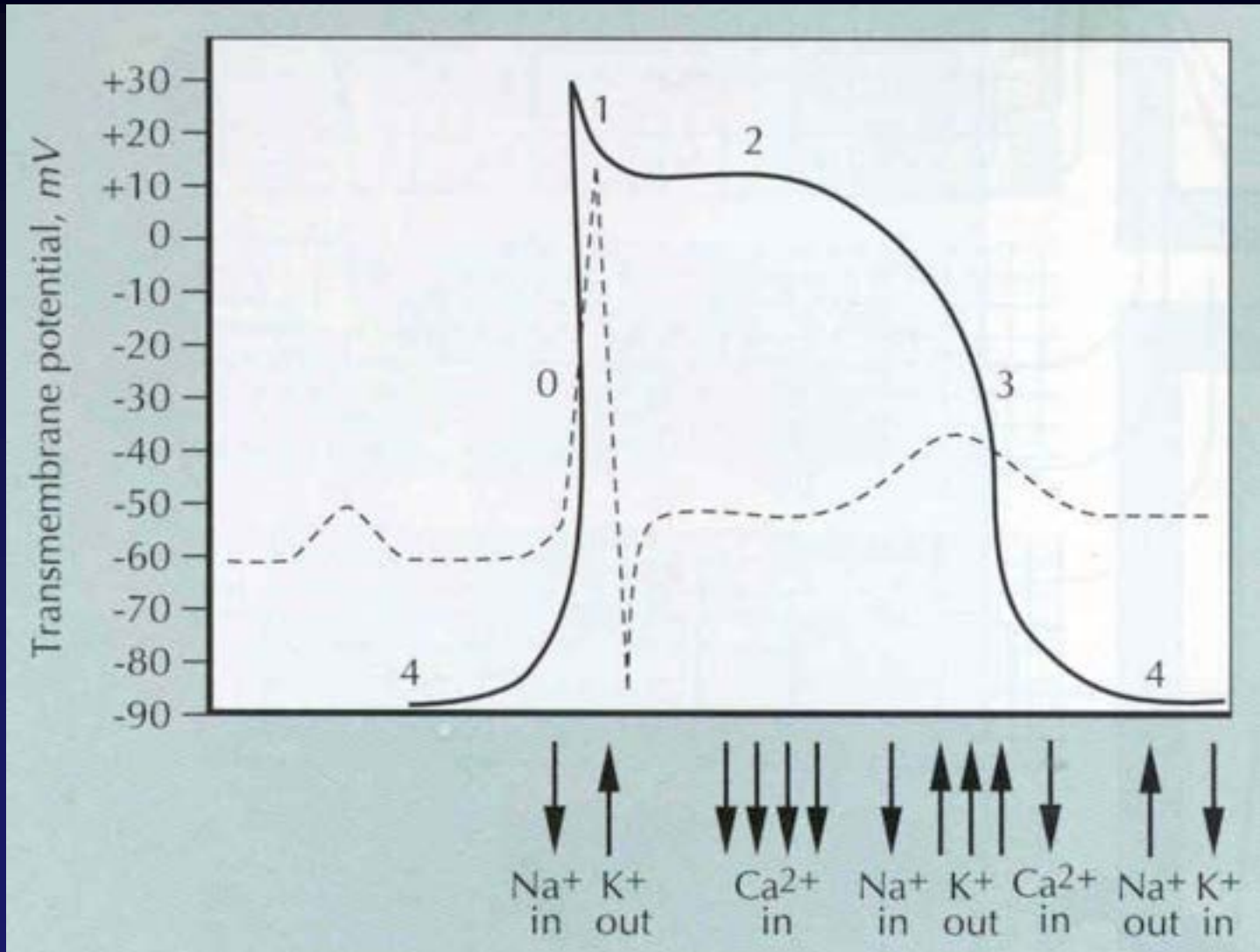
Normal Pathway of Electrical Conduction



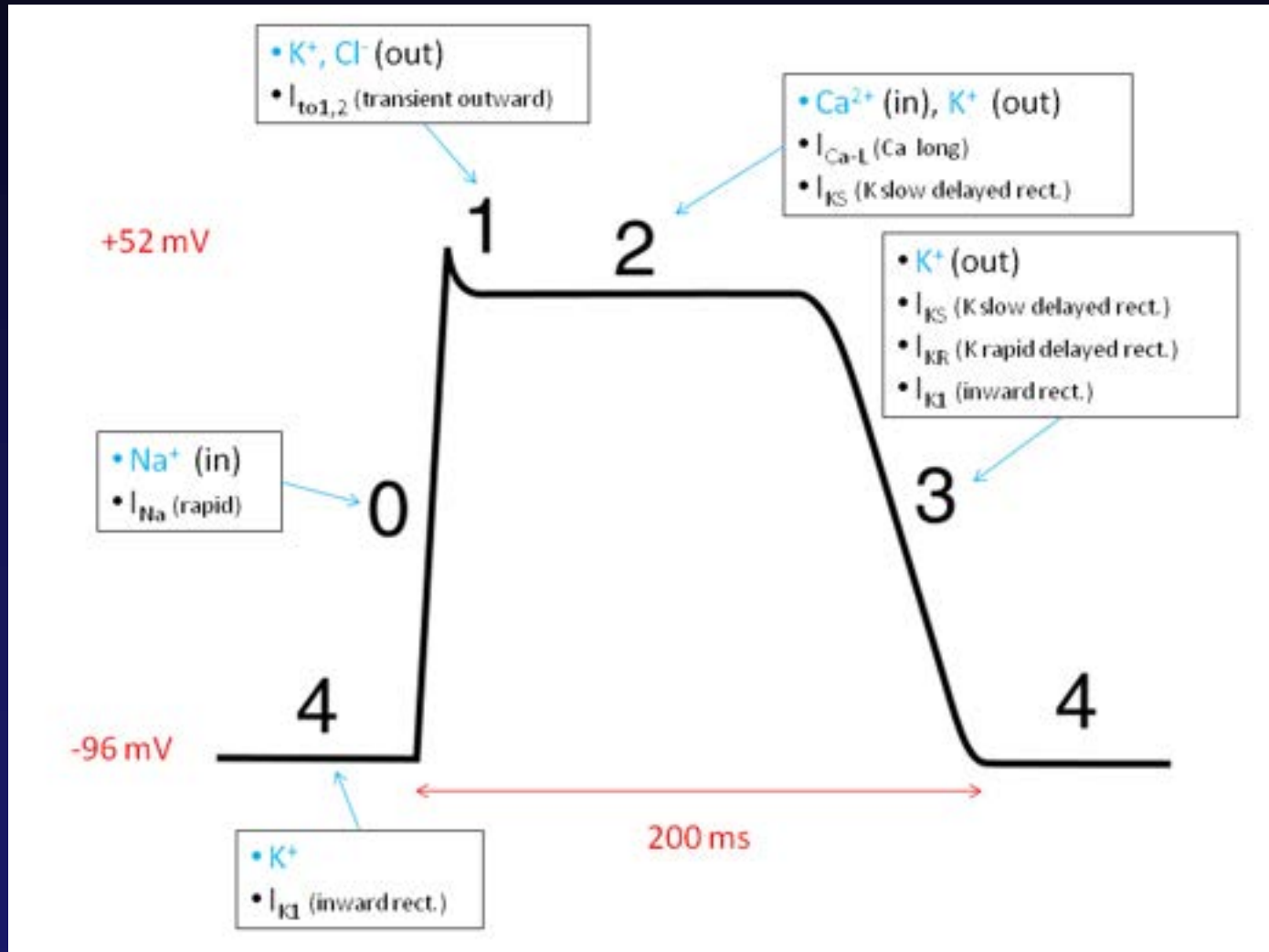
Normal Impulse Conduction



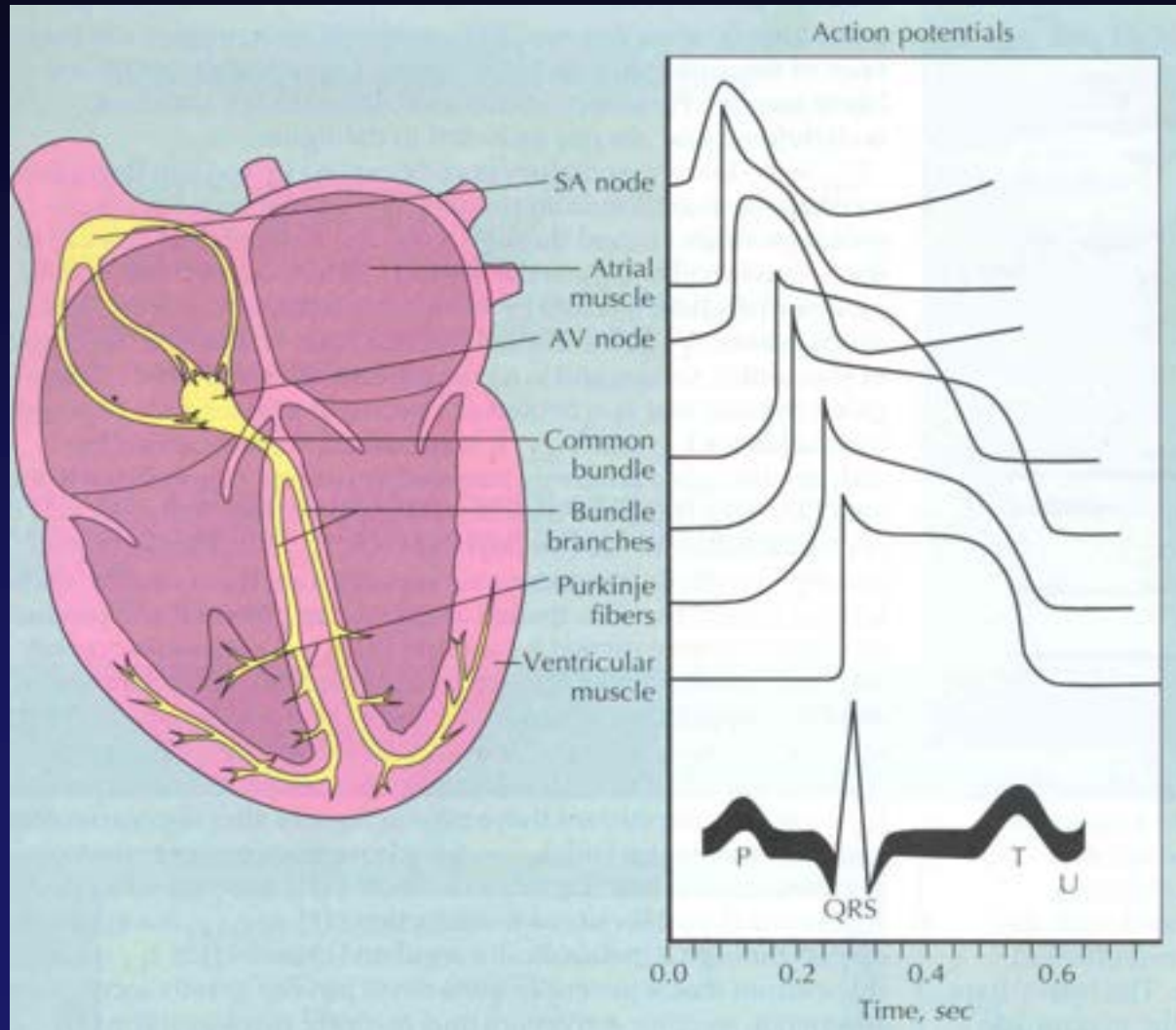
Cardiac Action Potential



Cardiac Action Potential



Cardiac action potentials from different locations have different shapes



Electrophysiology

- Electric currents that spread through the heart are produced by three components
 - Cardiac pacemaker cells
 - Specialized conduction tissue
 - The heart muscle
- ECG only records the depolarization and repolarization potentials generated by atrial and ventricular myocardium.

Electrocardiograph 1903

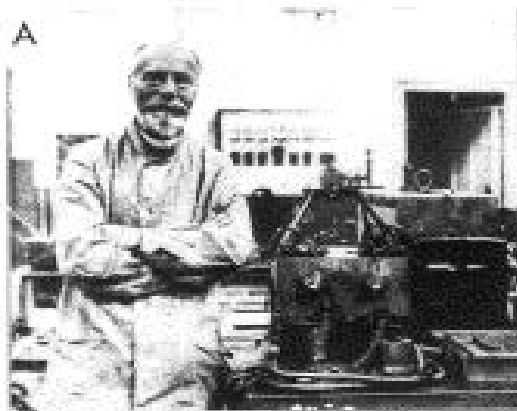
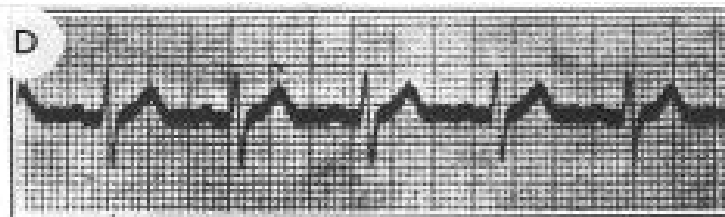
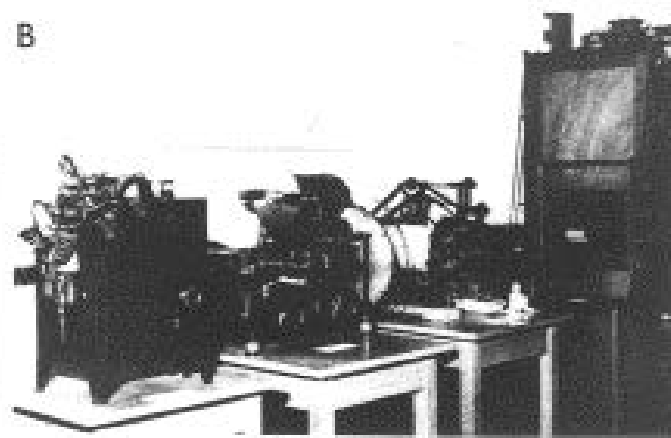
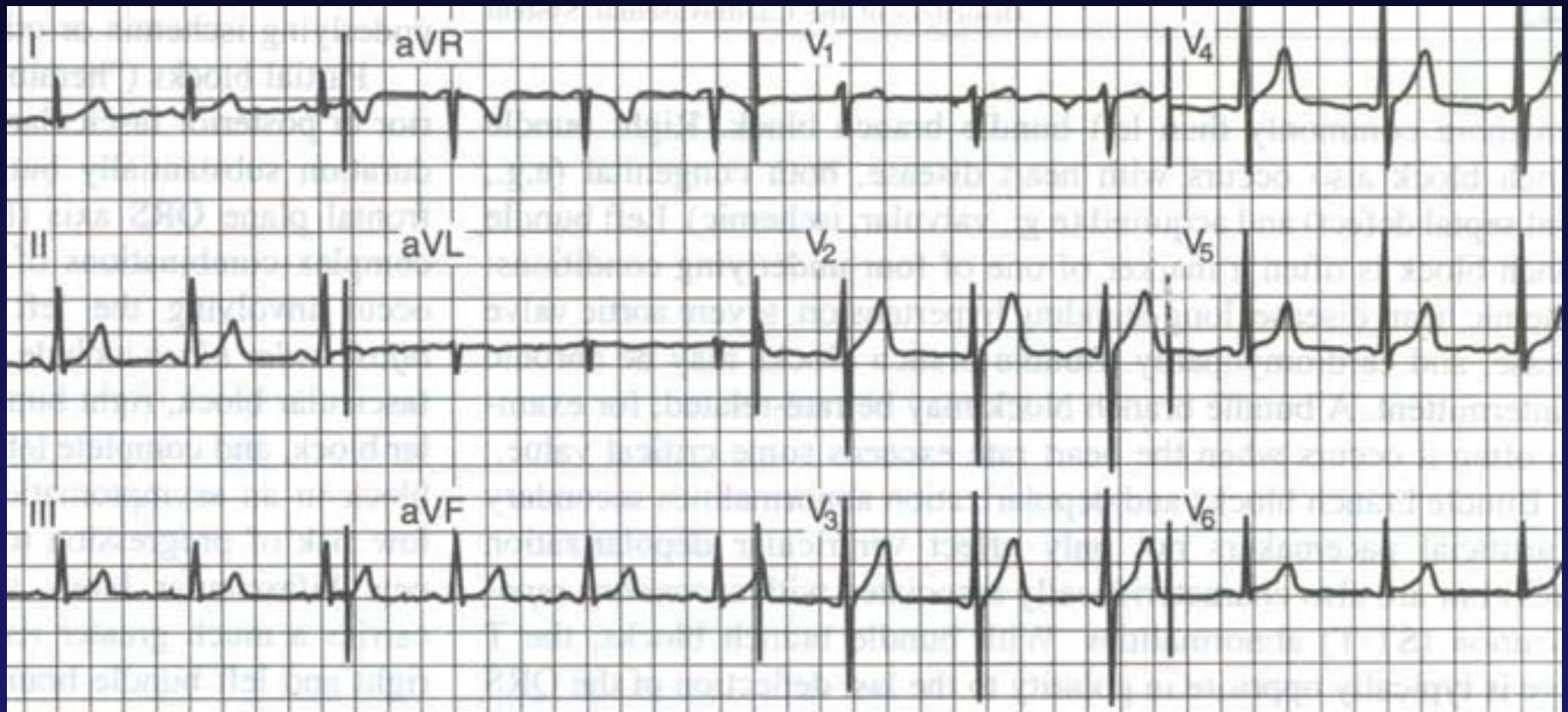


FIG. 1.—Professor W. Einthoven in his laboratory in Leyden with the original "string" galvanometer.

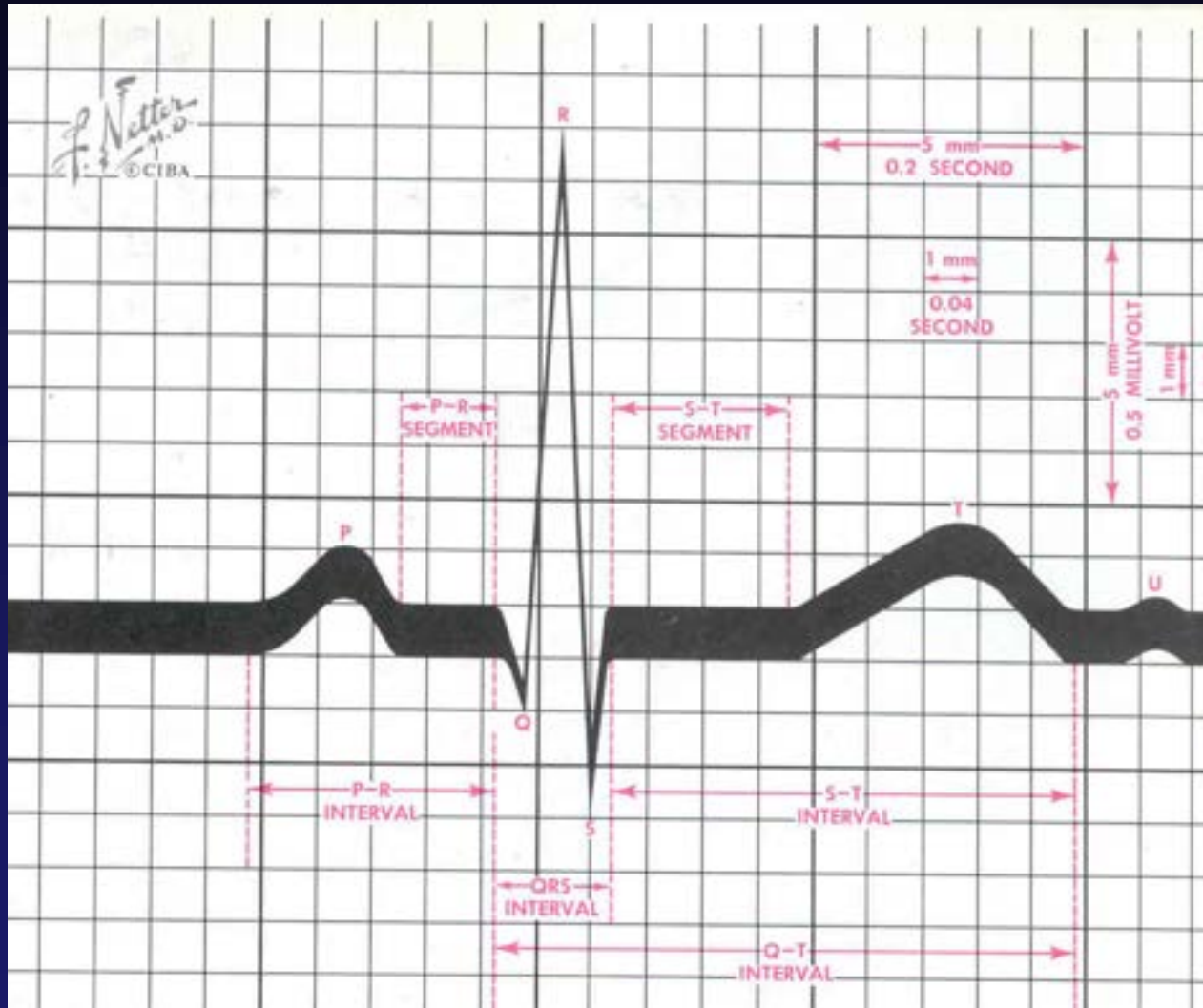


Normal Electrocardiogram

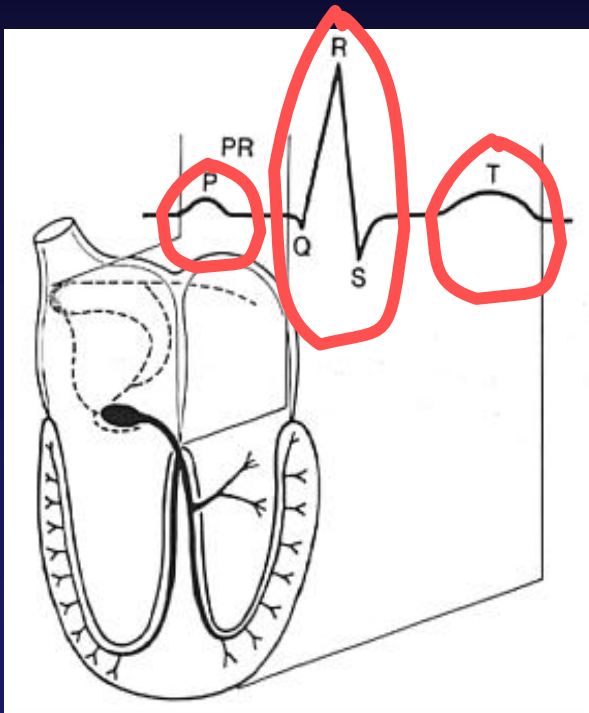


ECG Waveforms

Labeled alphabetically beginning with the P wave

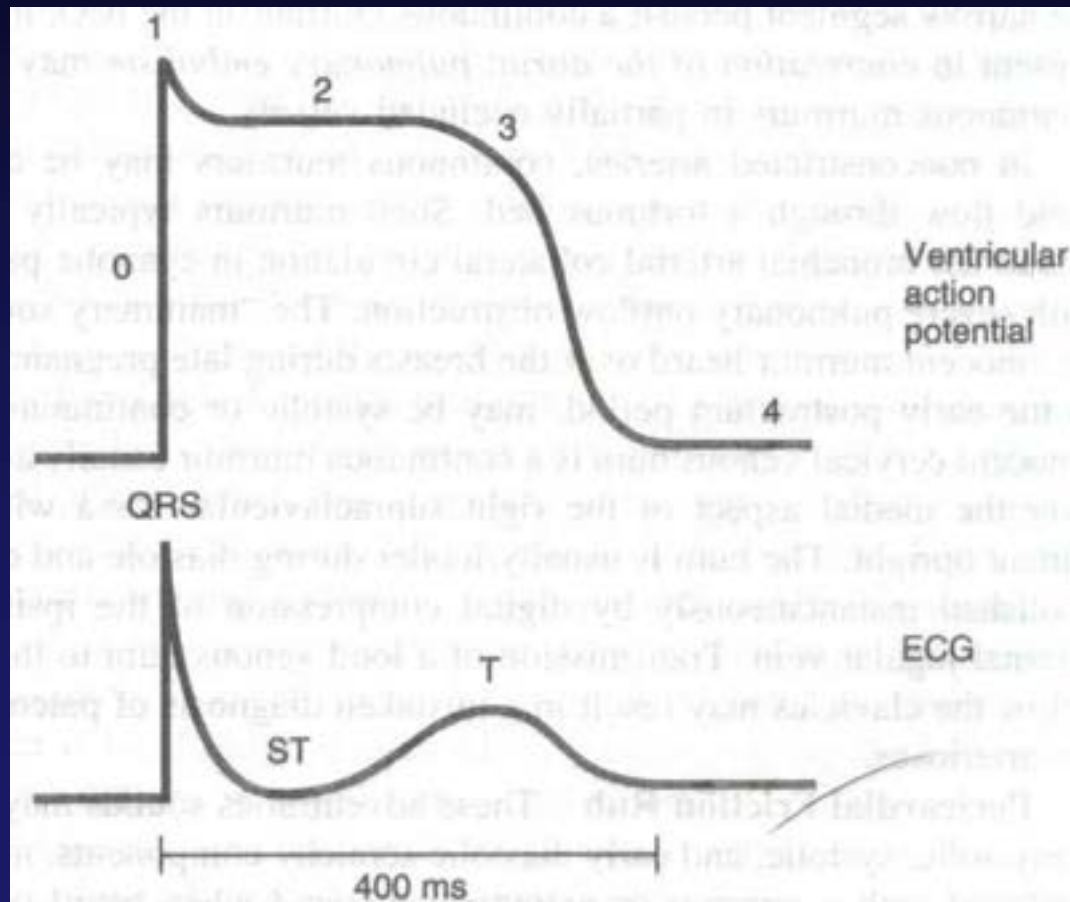


The “PQRST”



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

QRS-T Cycle Corresponds to Different Phases of Ventricular Action Potential



The PR Interval

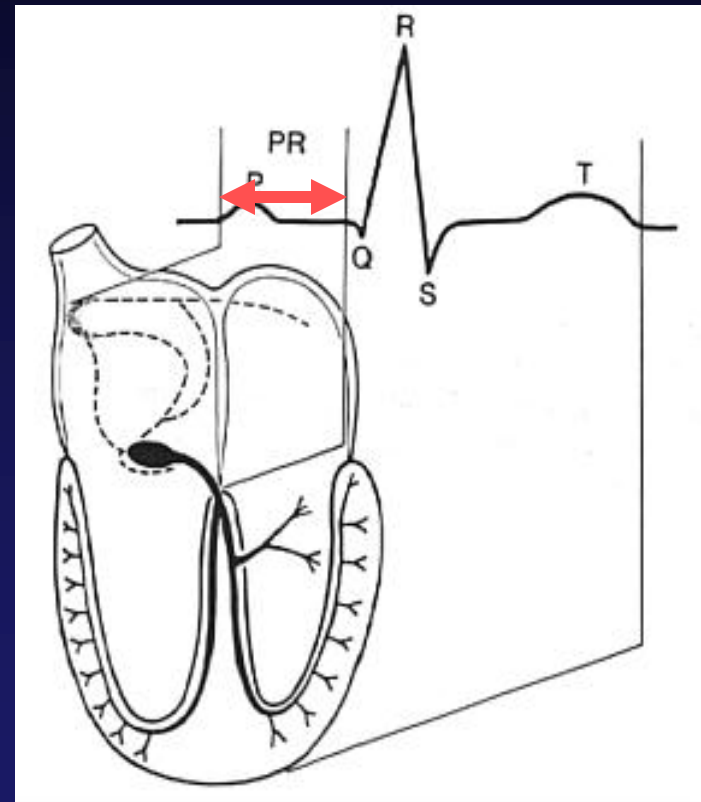
Atrial depolarization

+

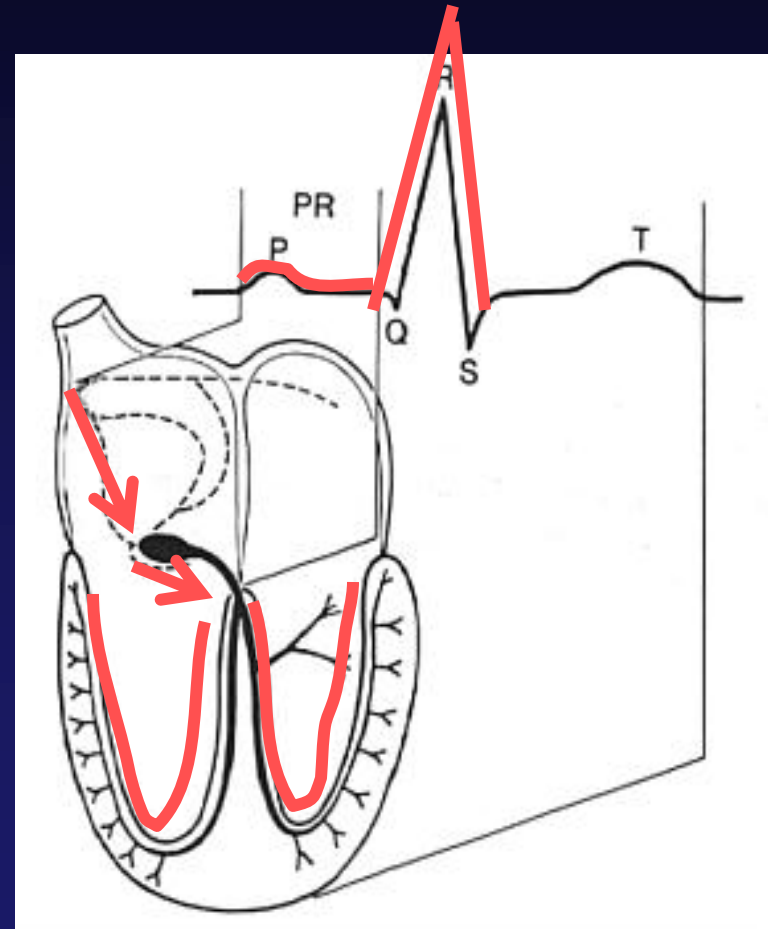
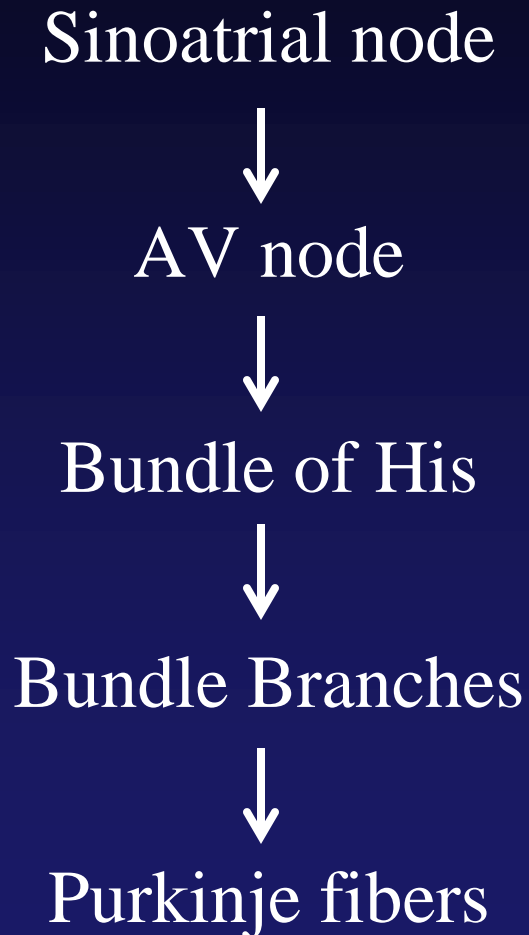
delay in AV junction

(AV node/Bundle of His)

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

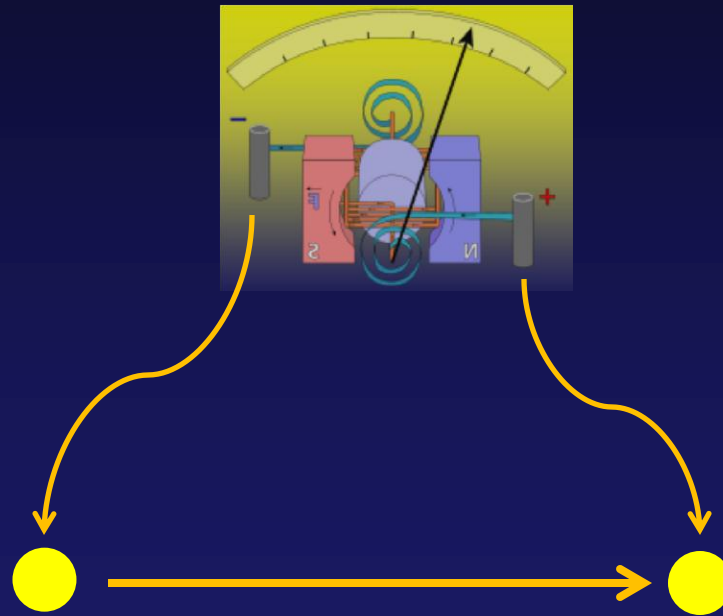


Impulse Conduction & the ECG



ECG Concept

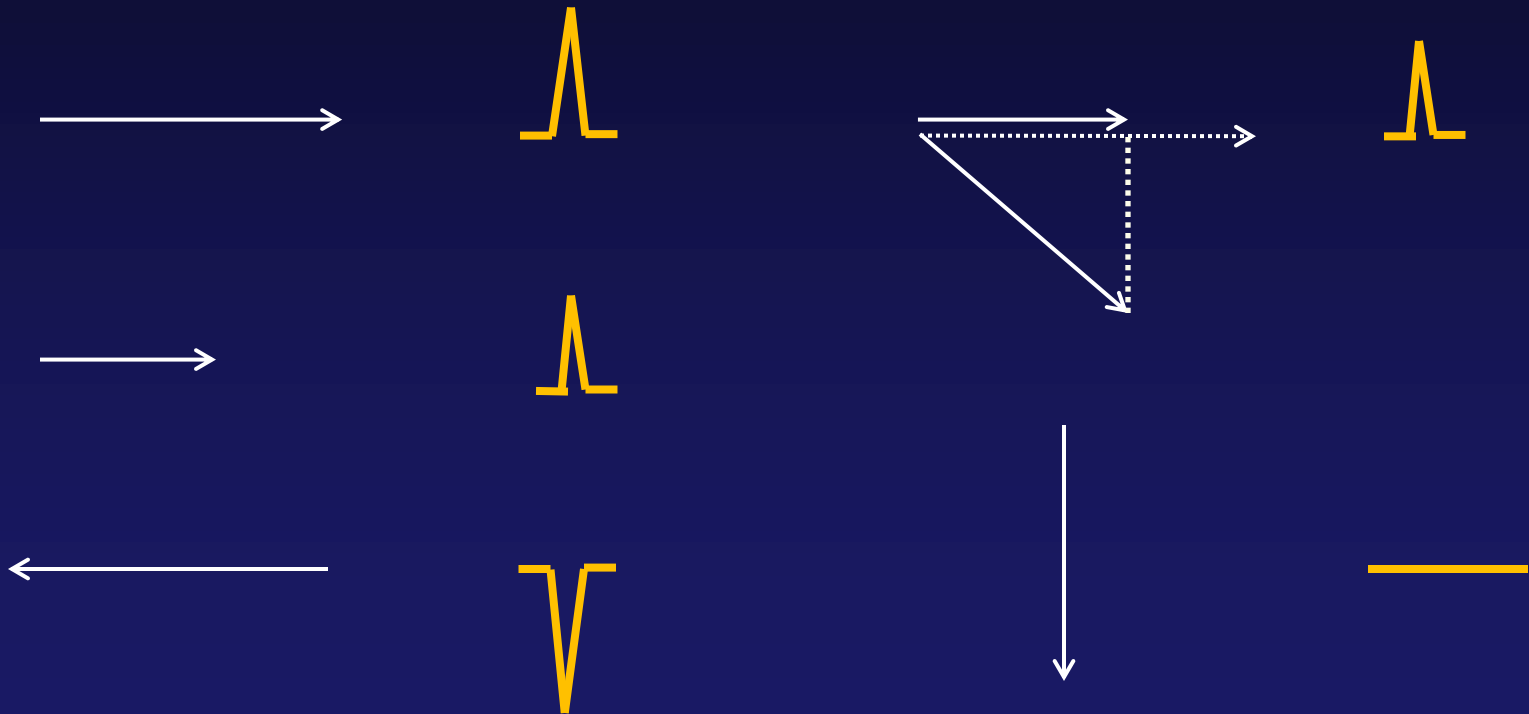
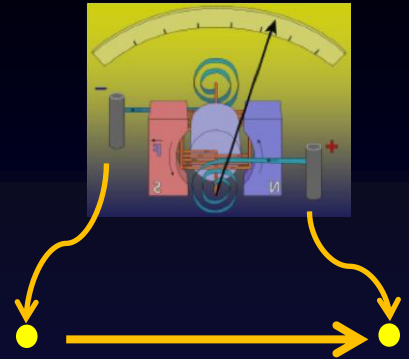
Galvanometer



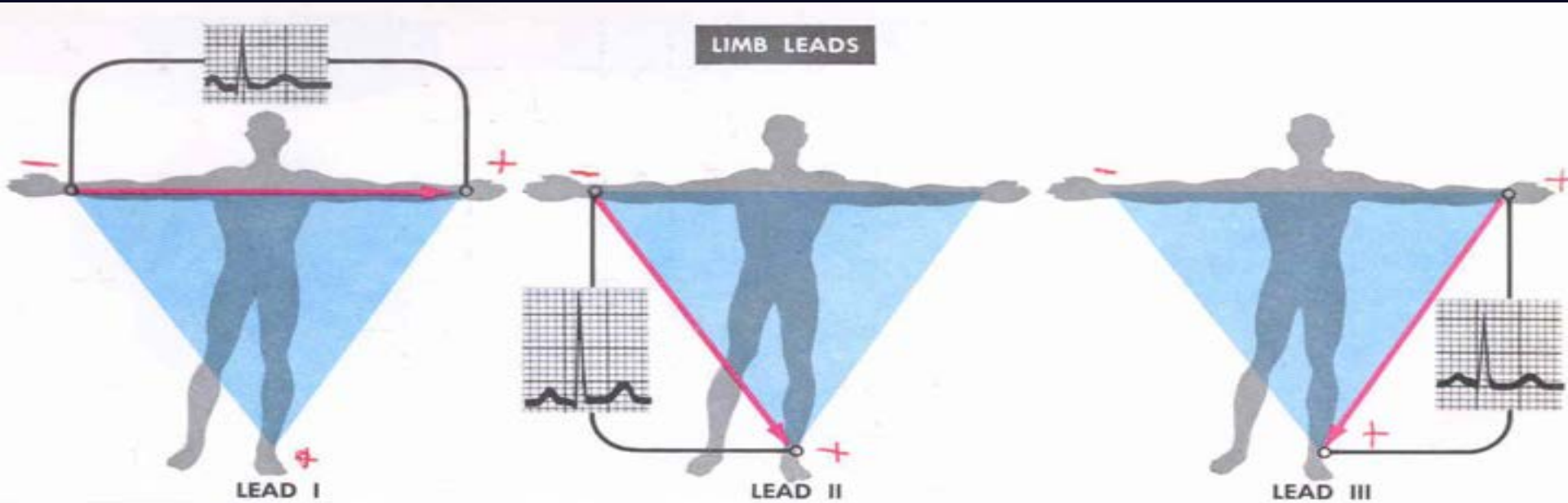
Vector Concept

- Cardiac depolarization and repolarization waves have direction and magnitude.
- They can, therefore, be represented by vectors.
- ECG records the complex spatial and temporal summation of electrical potentials from multiple myocardial fibers conducted to the surface of the body.

Galvanometer



Bipolar Limb Leads



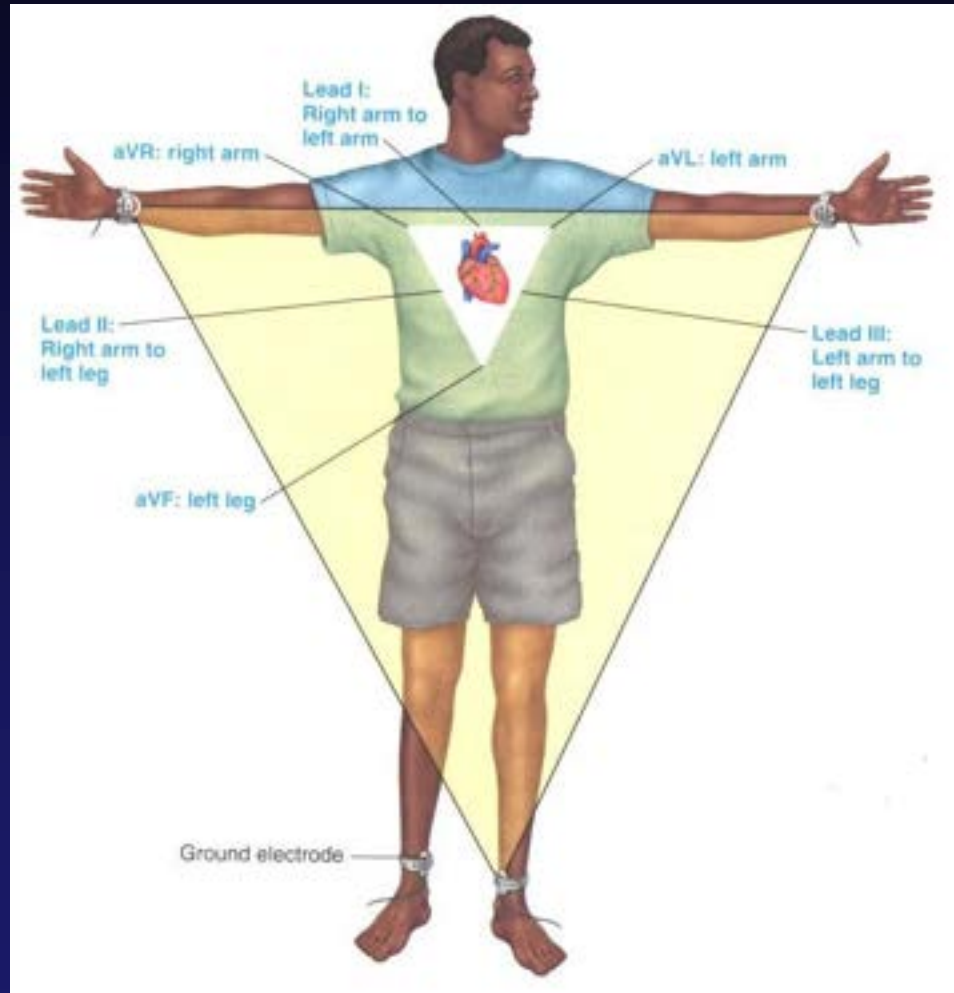
Lead I

Lead II

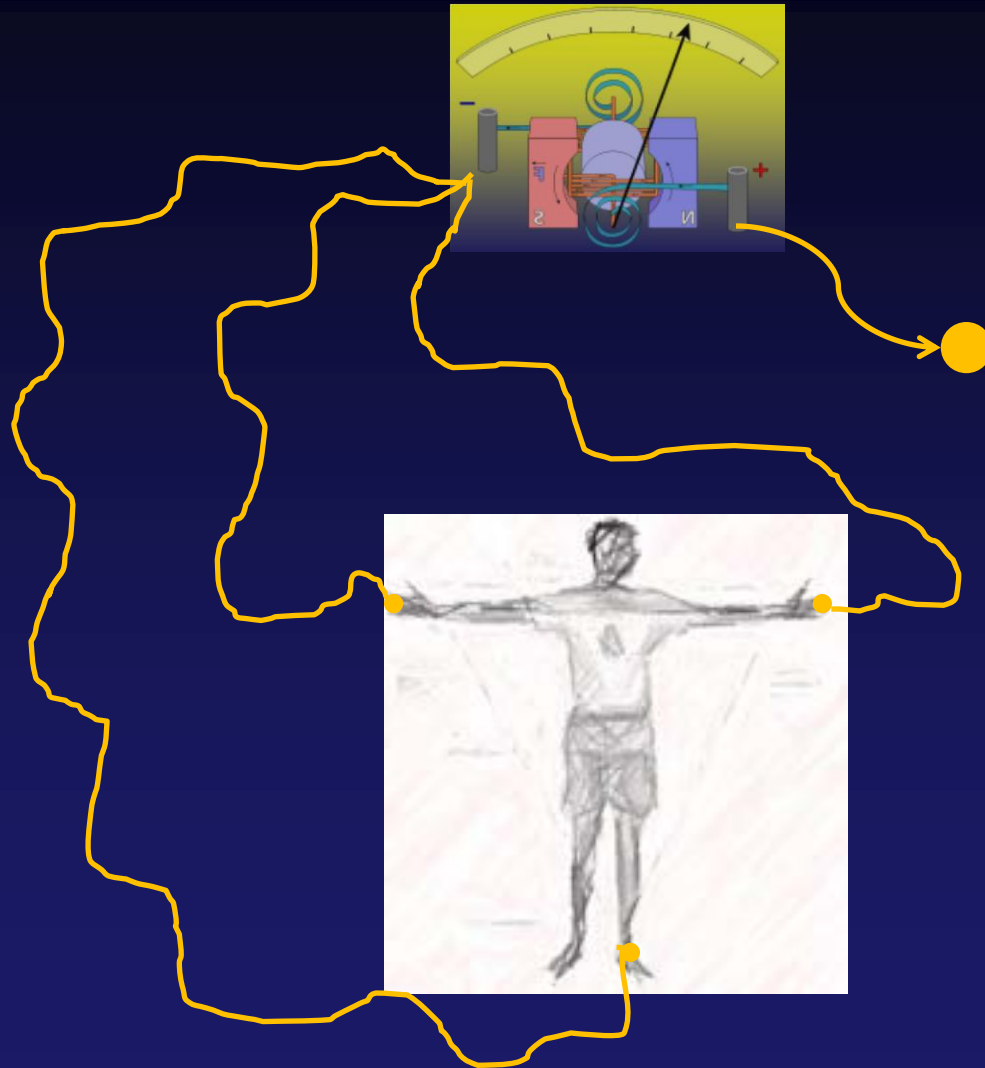
Lead III



Einthoven Triangle

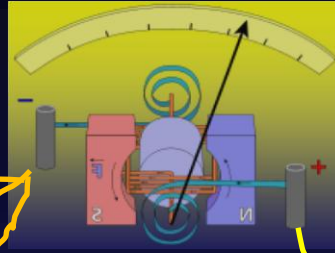


Central Terminal of Wilson



Unipolar Lead

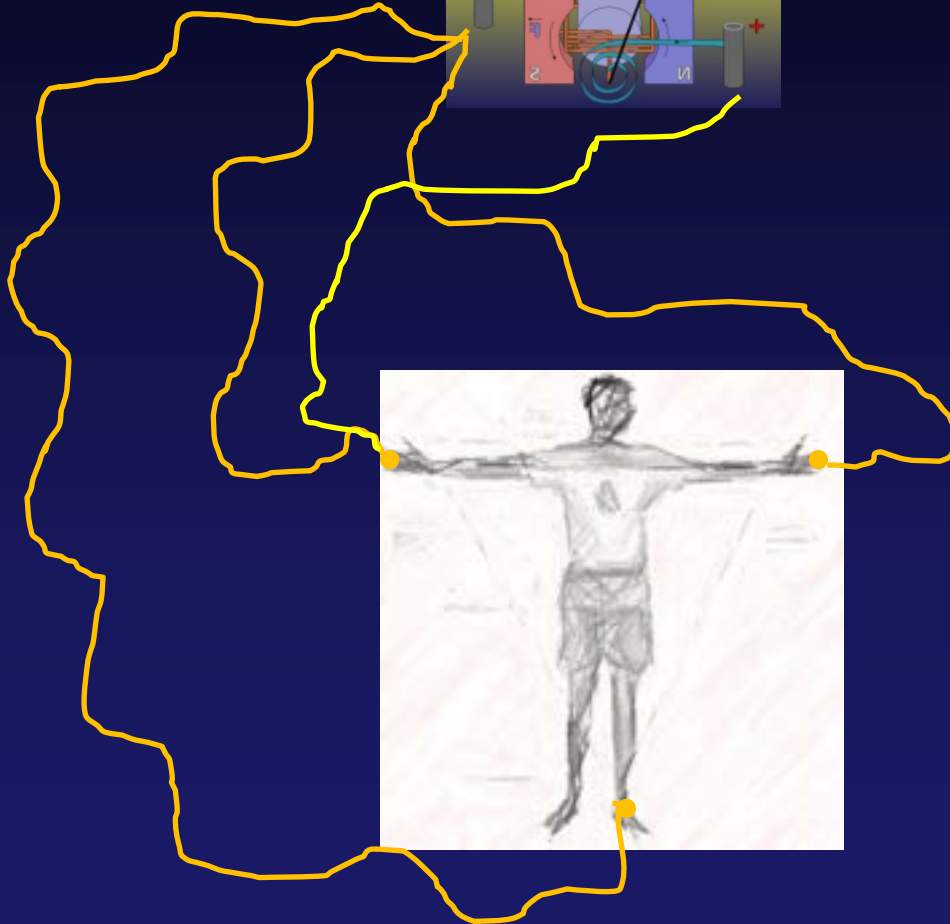
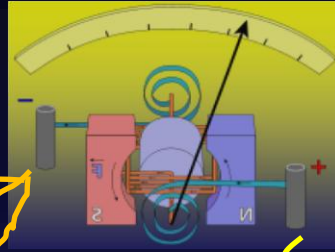
Unipolar Limb Leads



Lead
VL

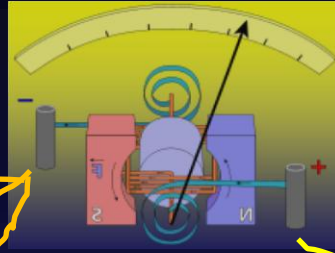


Unipolar Limb Leads



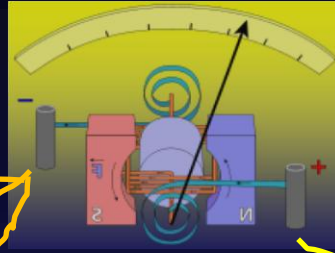
Lead
VR

Unipolar Limb Lead



Lead
VF

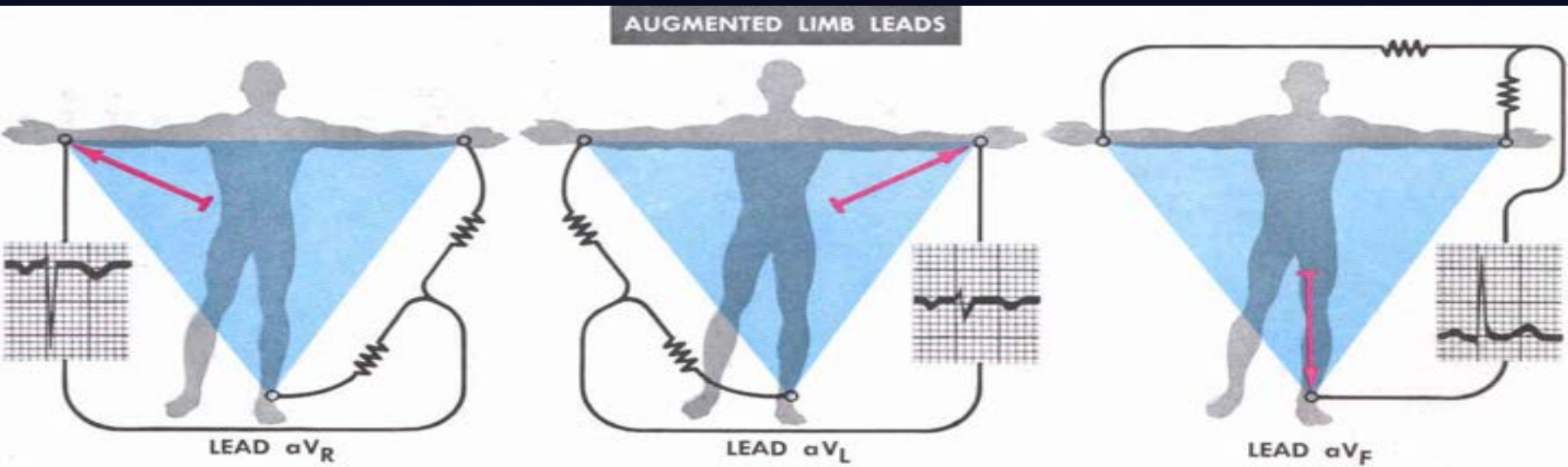
Unipolar Limb Lead



Lead
VF

augmented
VF or
aVF

Unipolar Limb Leads



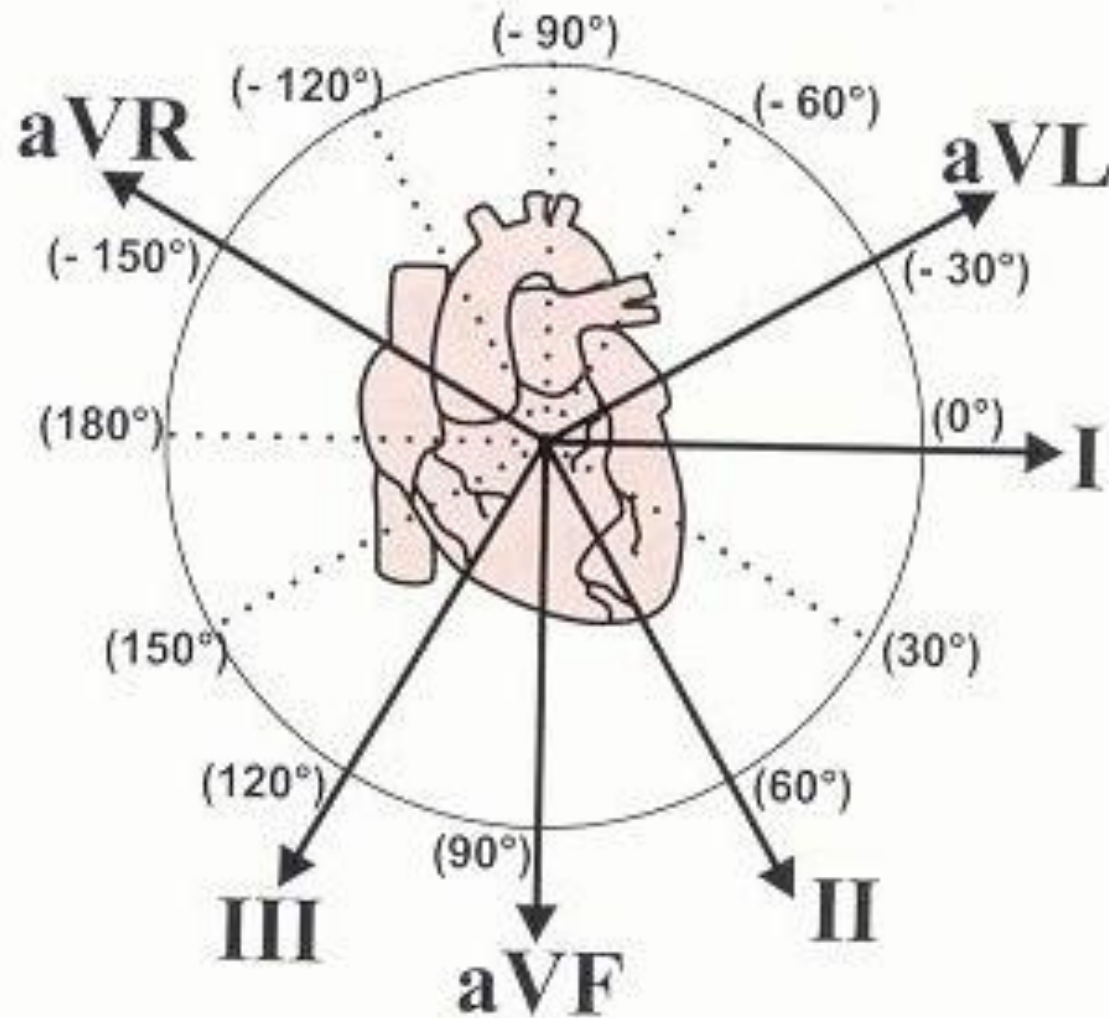
Lead aV_R

Lead aV_L

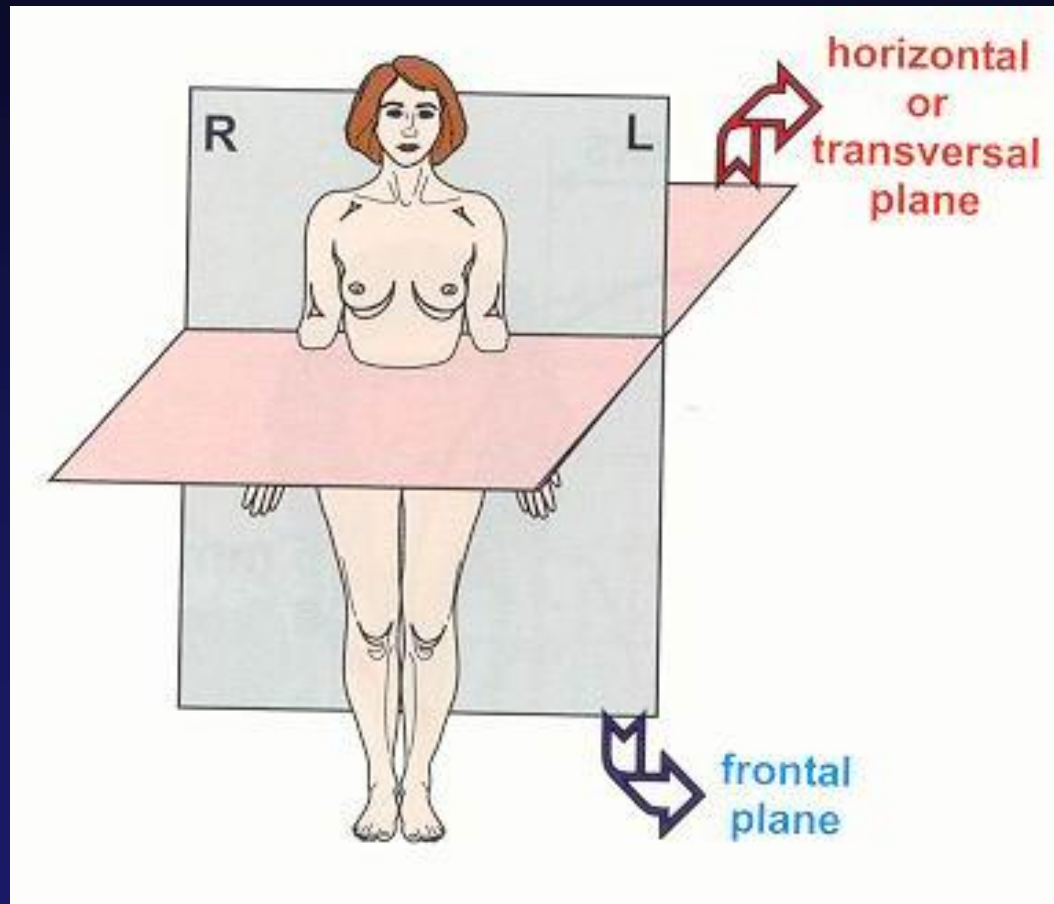
Lead aV_F



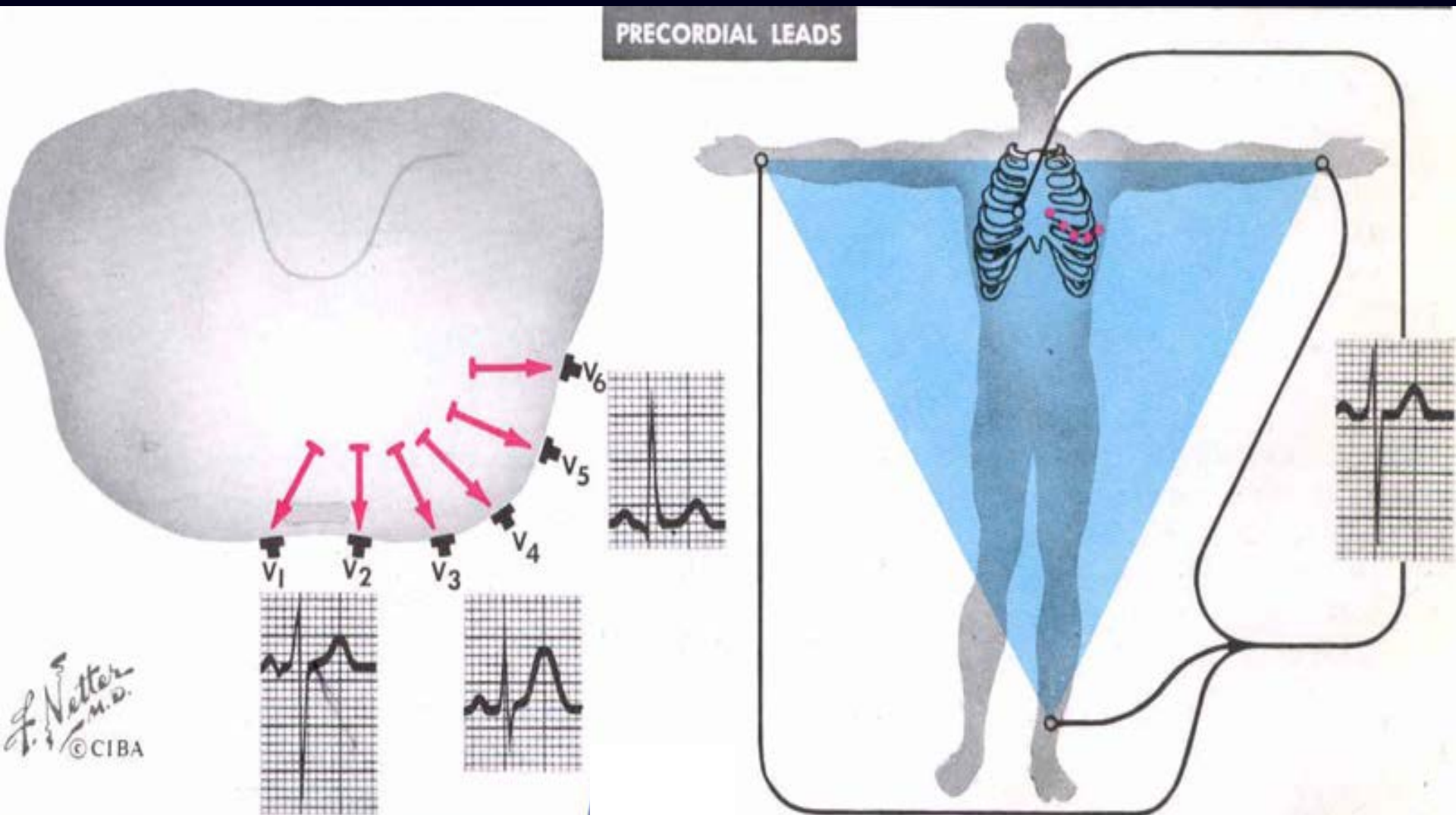
Limb Leads Directions



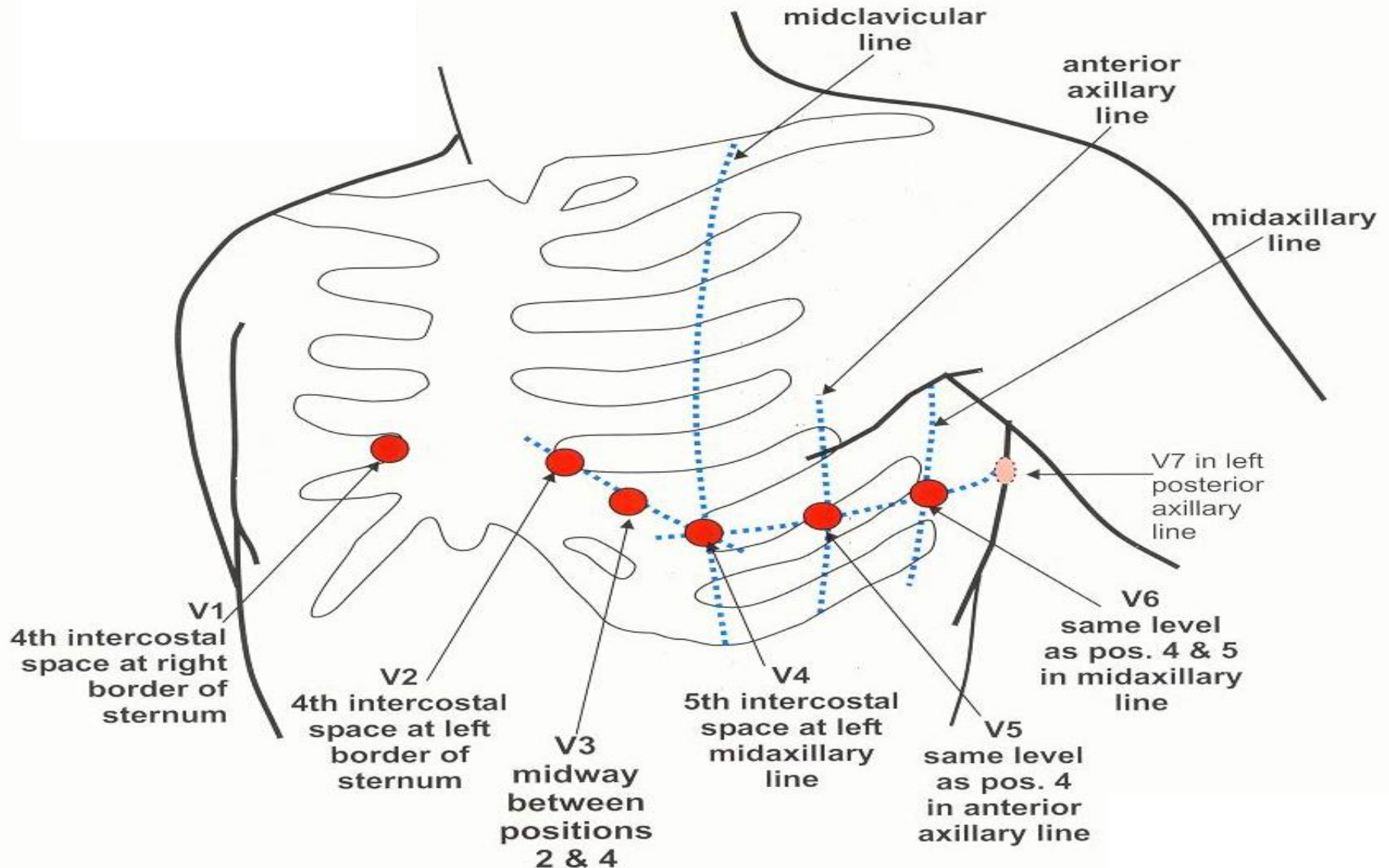
3-D Representation of Cardiac Electrical Activity



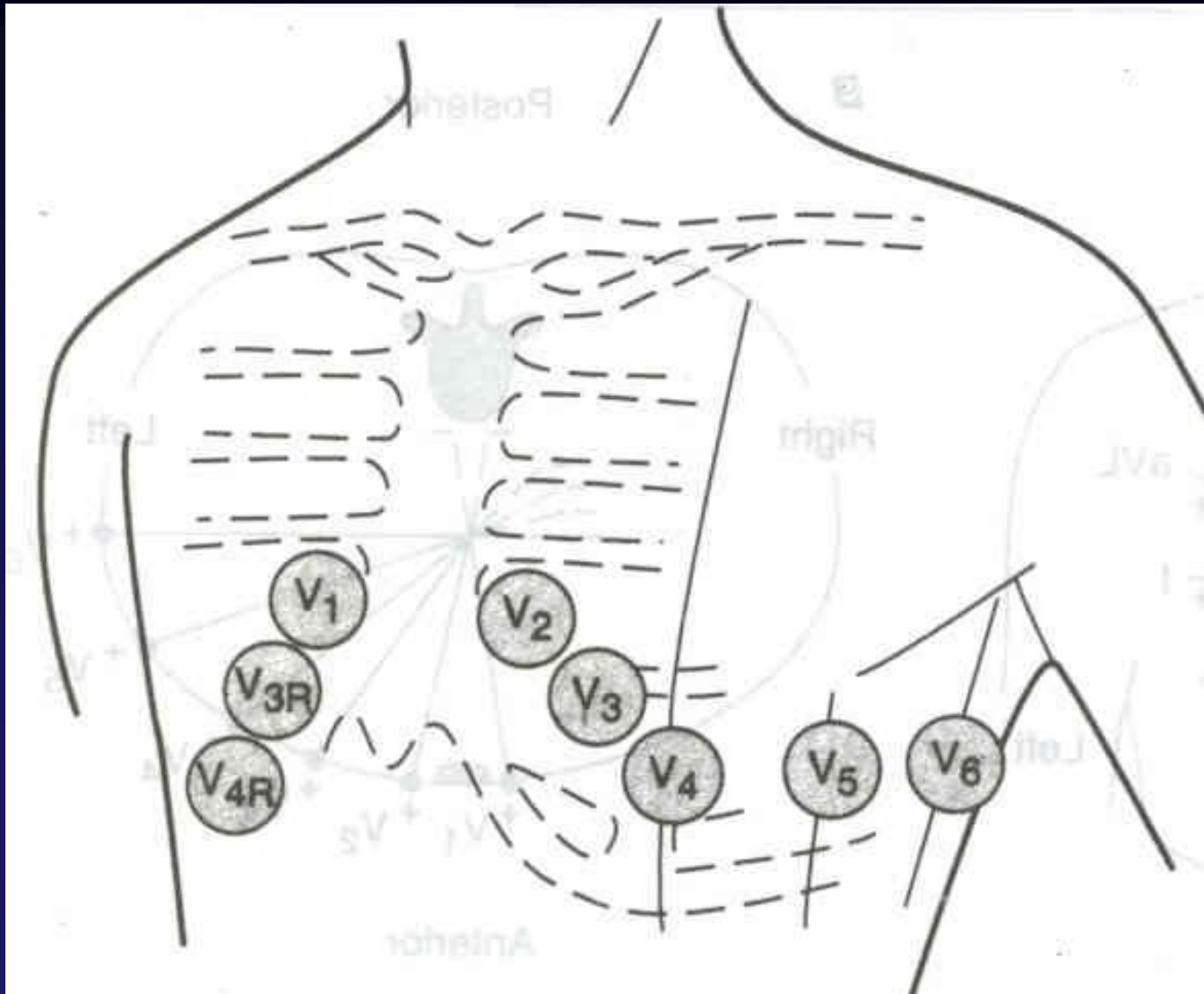
Precordial Leads



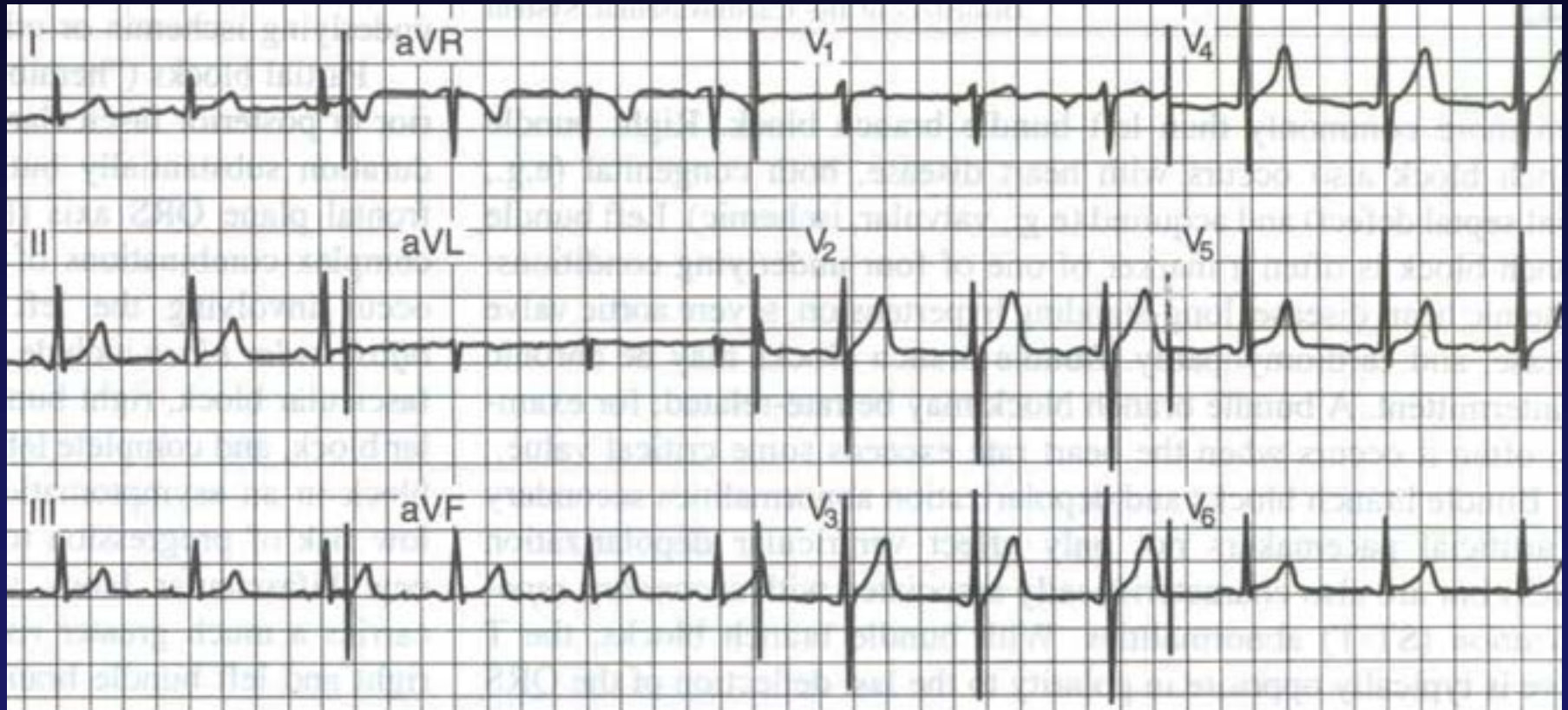
Position of Precordial Electrodes



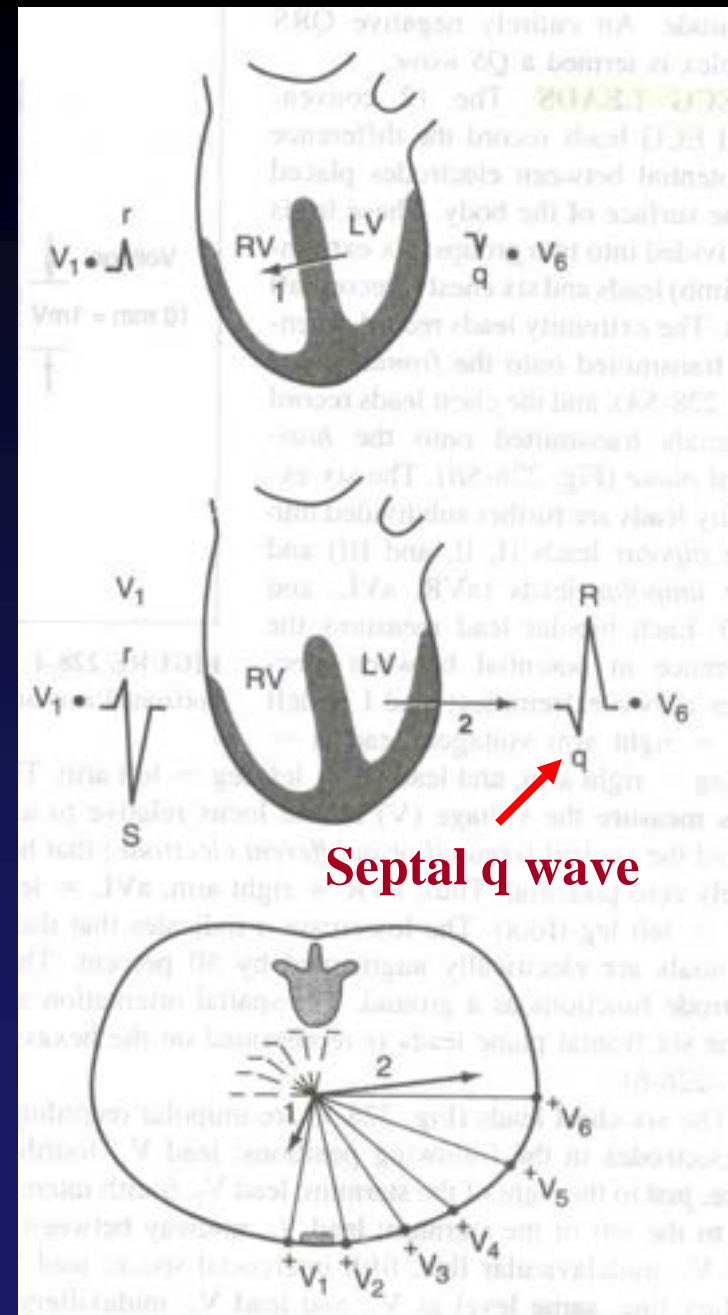
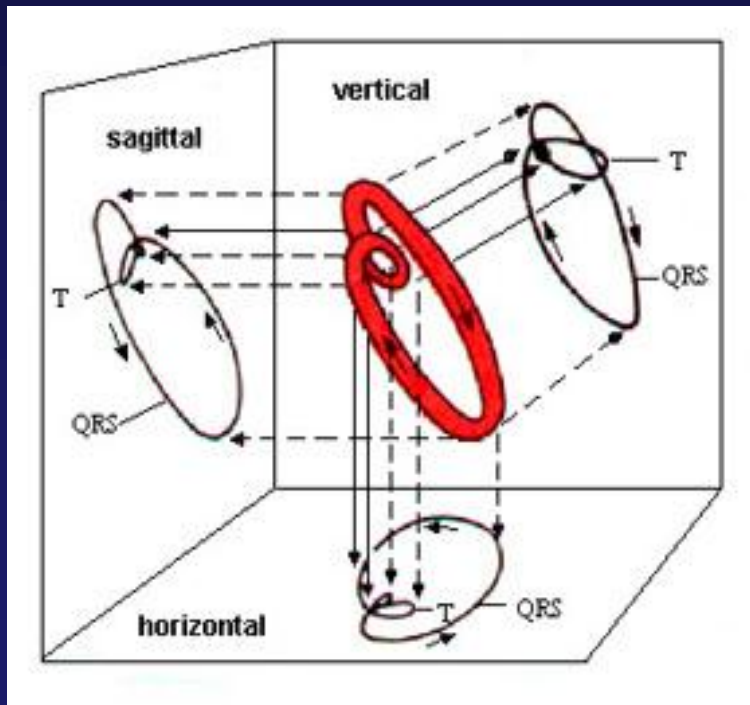
Precordial Leads



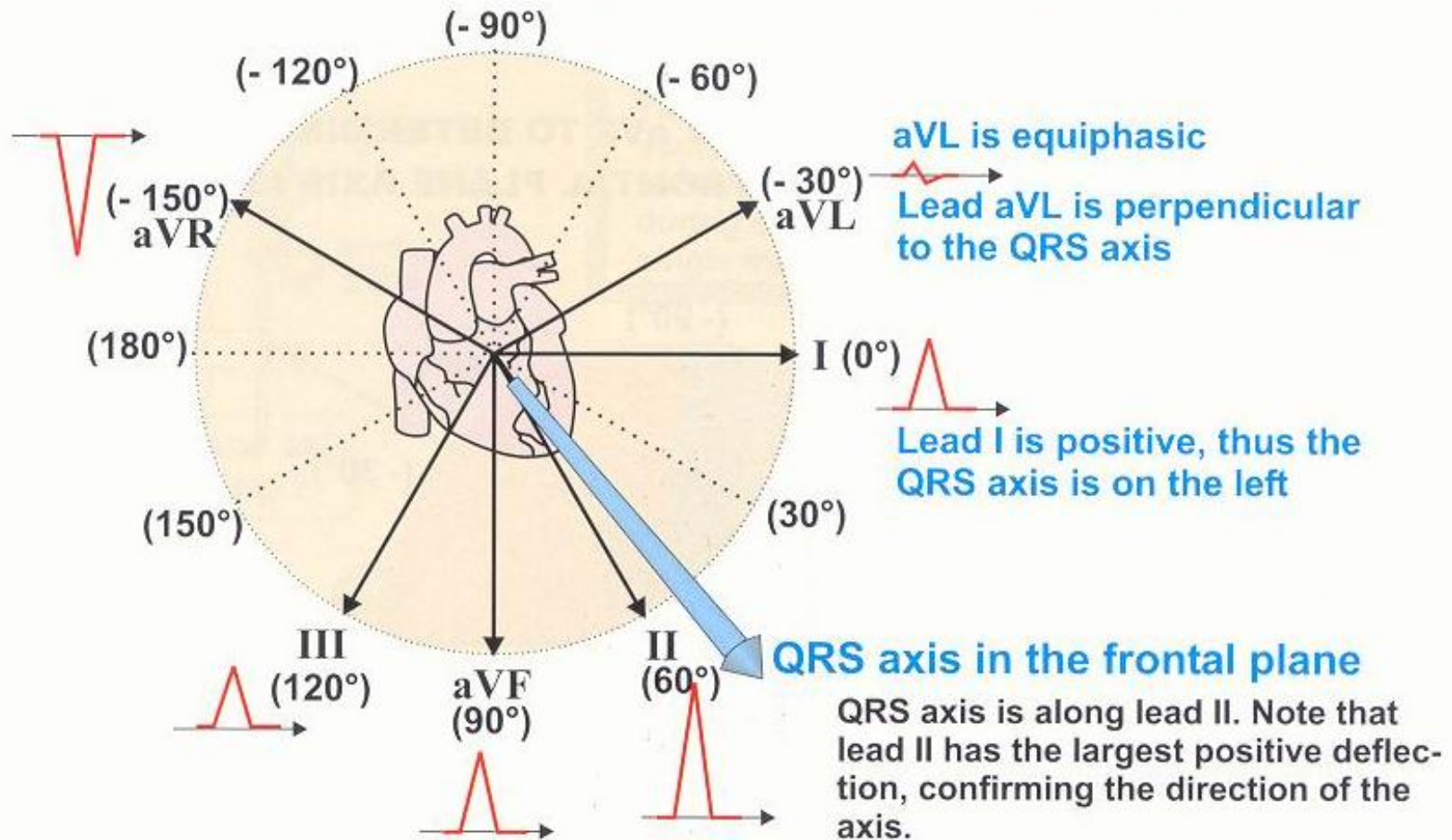
Normal Electrocardiogram



Ventricular Depolarization Axis



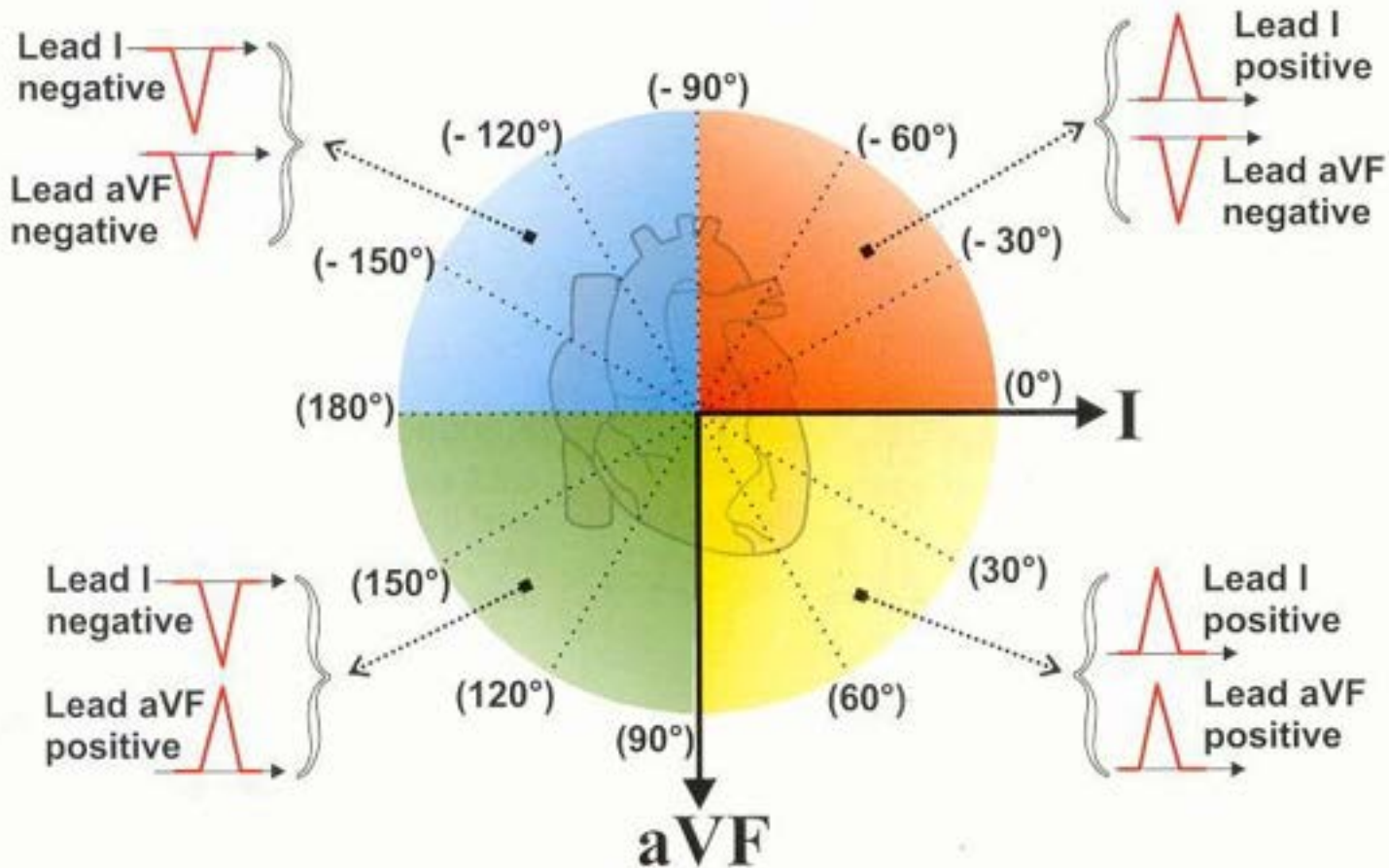
Mean Activation Vector



Determination of QRS Axis

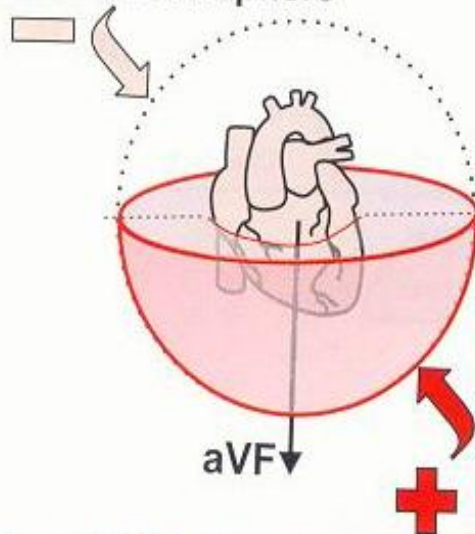


STEP 1 : LOOK AT LEADS I & aVF TO DETERMINE IN WHICH QUADRANT THE FRONTAL PLANE AXIS IS SITUATED

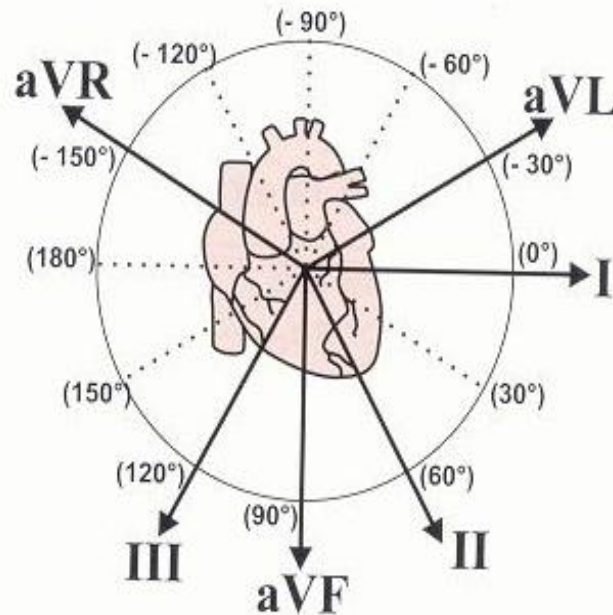


Direction of Propagation

Lead aVF will be negative if the mean QRS vector is in this hemisphere

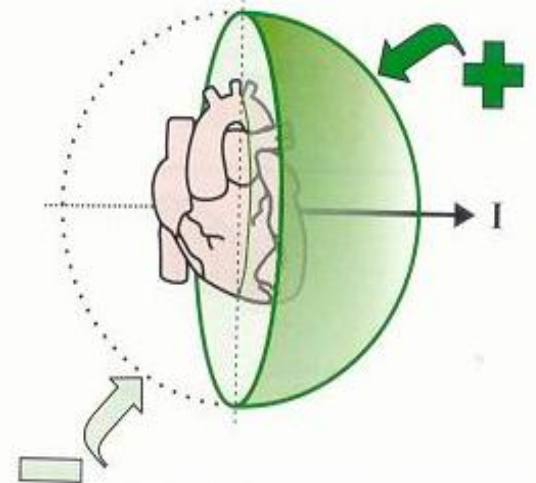


Lead aVF will be positive if the mean QRS vector is situated in this hemisphere



A. F. Finnaeve

Lead I will be positive if the mean QRS vector is situated in this hemisphere

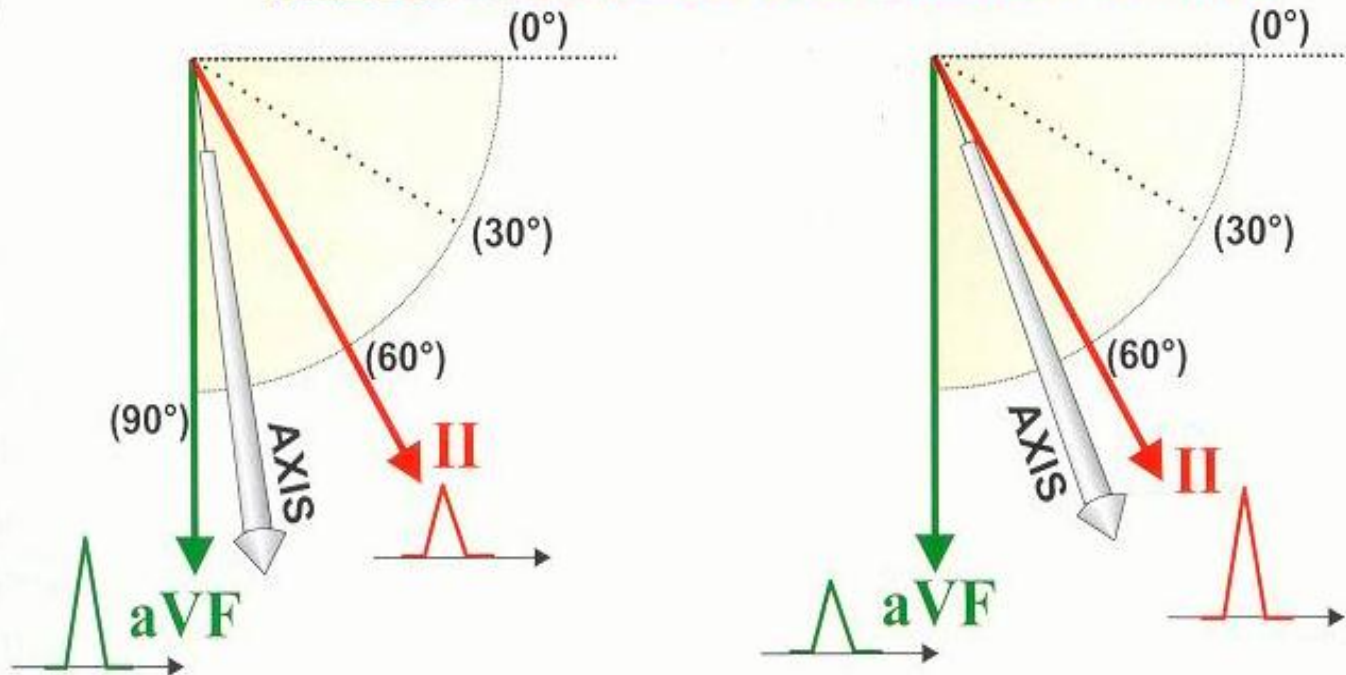


Lead I will be negative if the mean QRS vector is in this hemisphere

Determination of QRS Axis



STEP 2 : LOOK IN THE APPROPRIATE QUADRANT FOR THE TALLEST R WAVE OR THE DEEPEST S WAVE



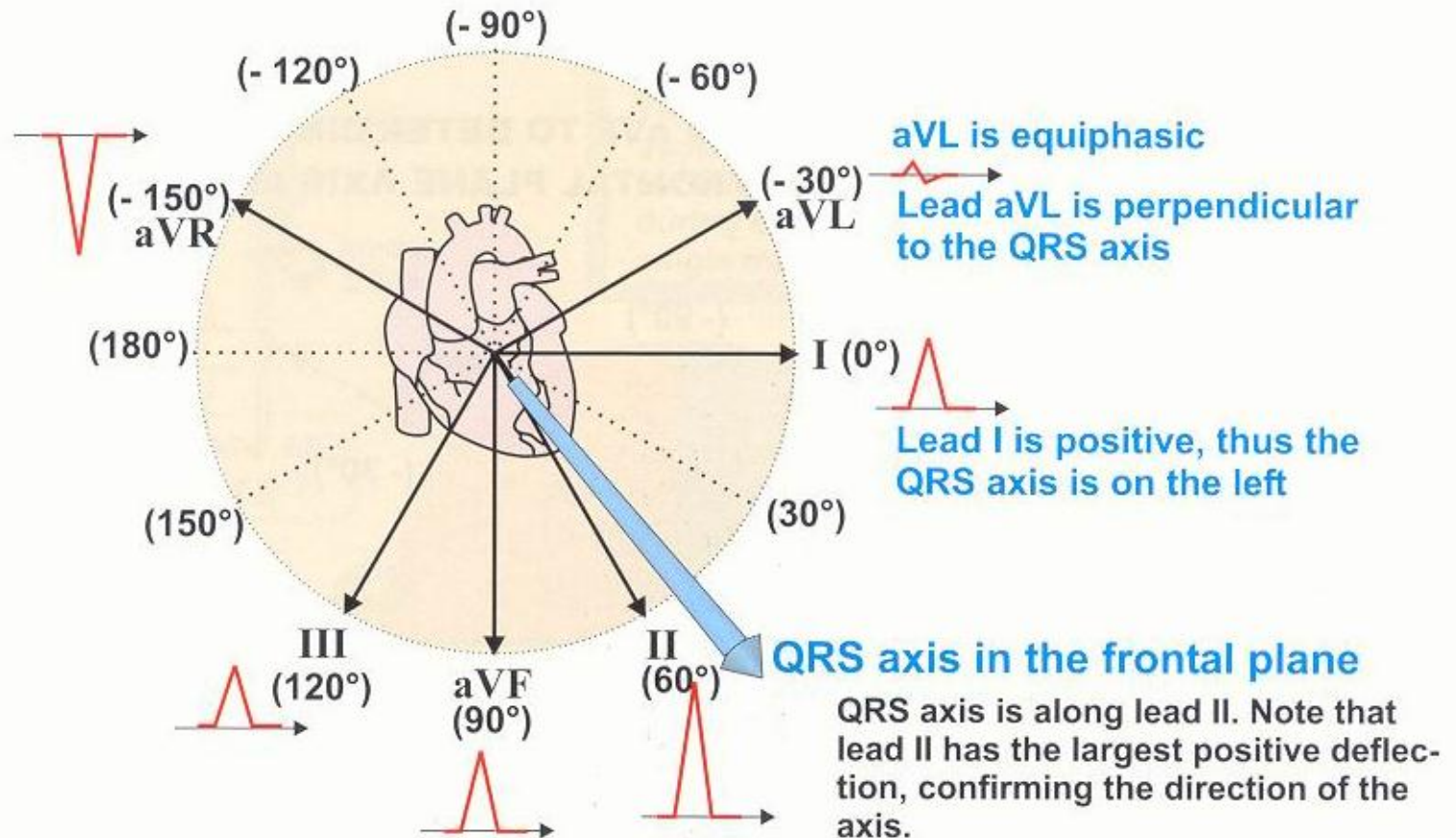
A. F. Pinnaere

The lead nearest to (or parallel along) the QRS axis has the largest positive deflection. If two leads have equal positive deflections, the axis is exactly in the middle between these two leads.

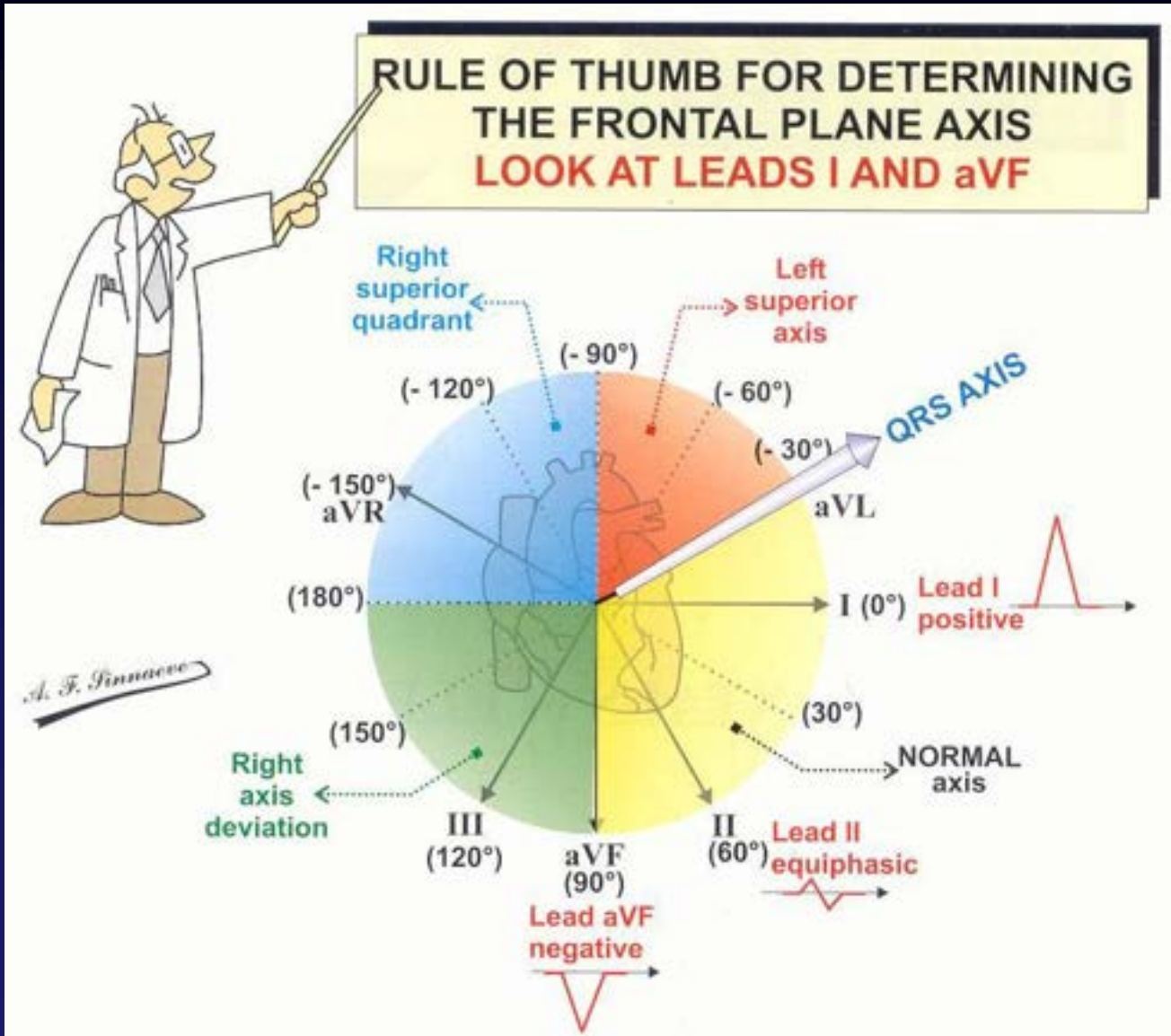
Determination of QRS Axis



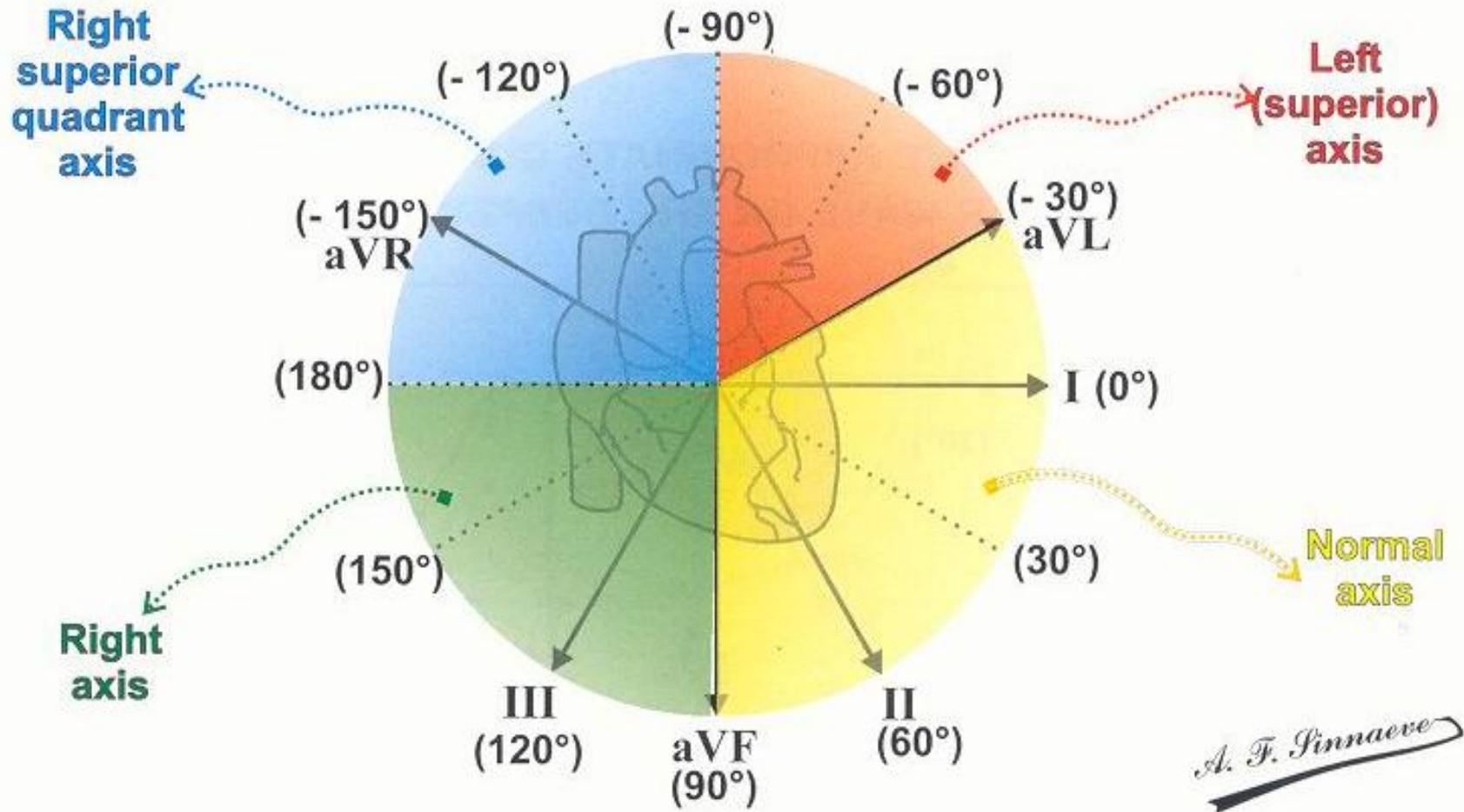
STEP 3 : LOOK FOR THE MOST EQUIPHASIC LEAD (where the positive minus the negative deflection is closest to zero)
THIS LEAD IS PERPENDICULAR TO THE QRS AXIS



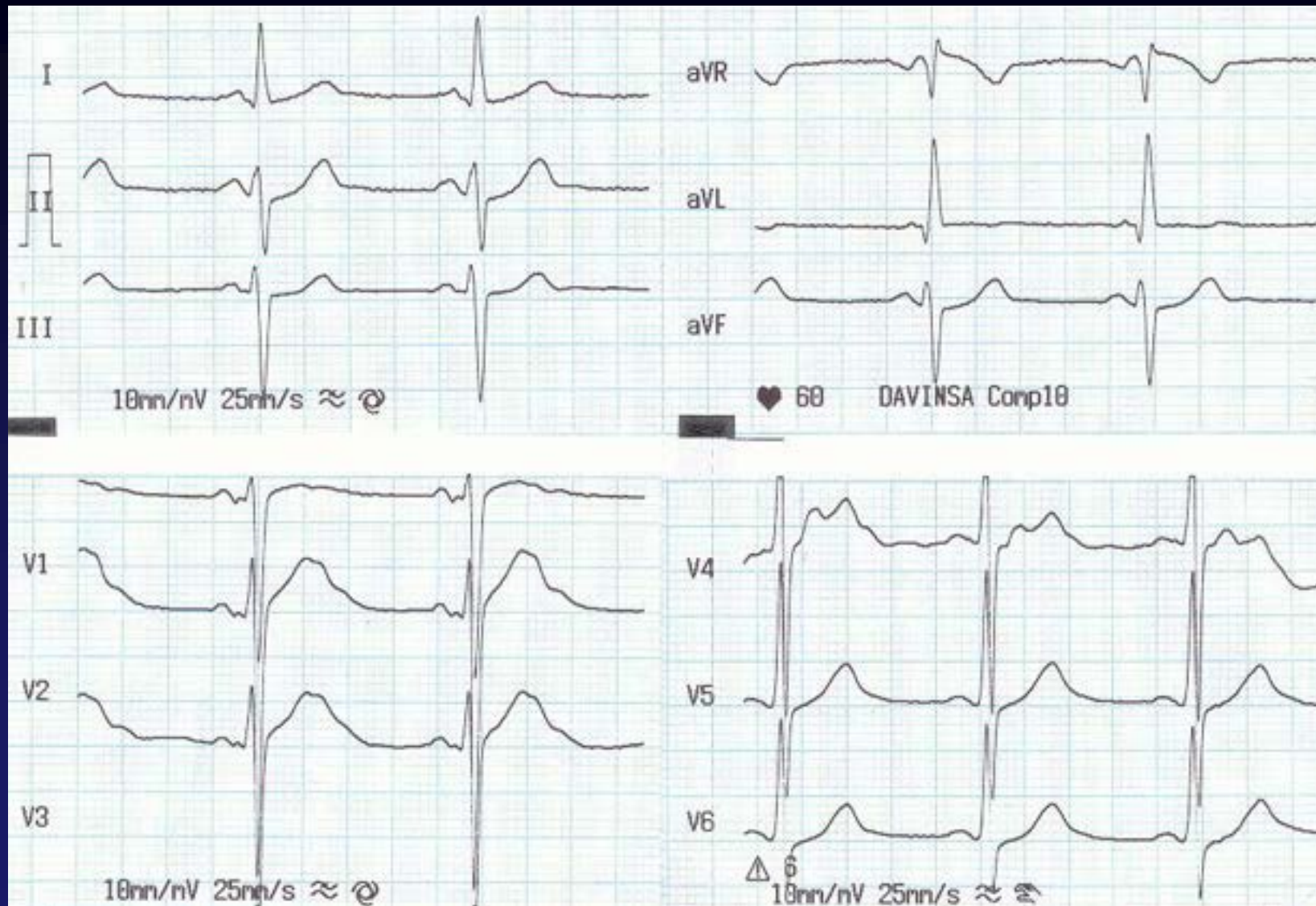
QRS Axis



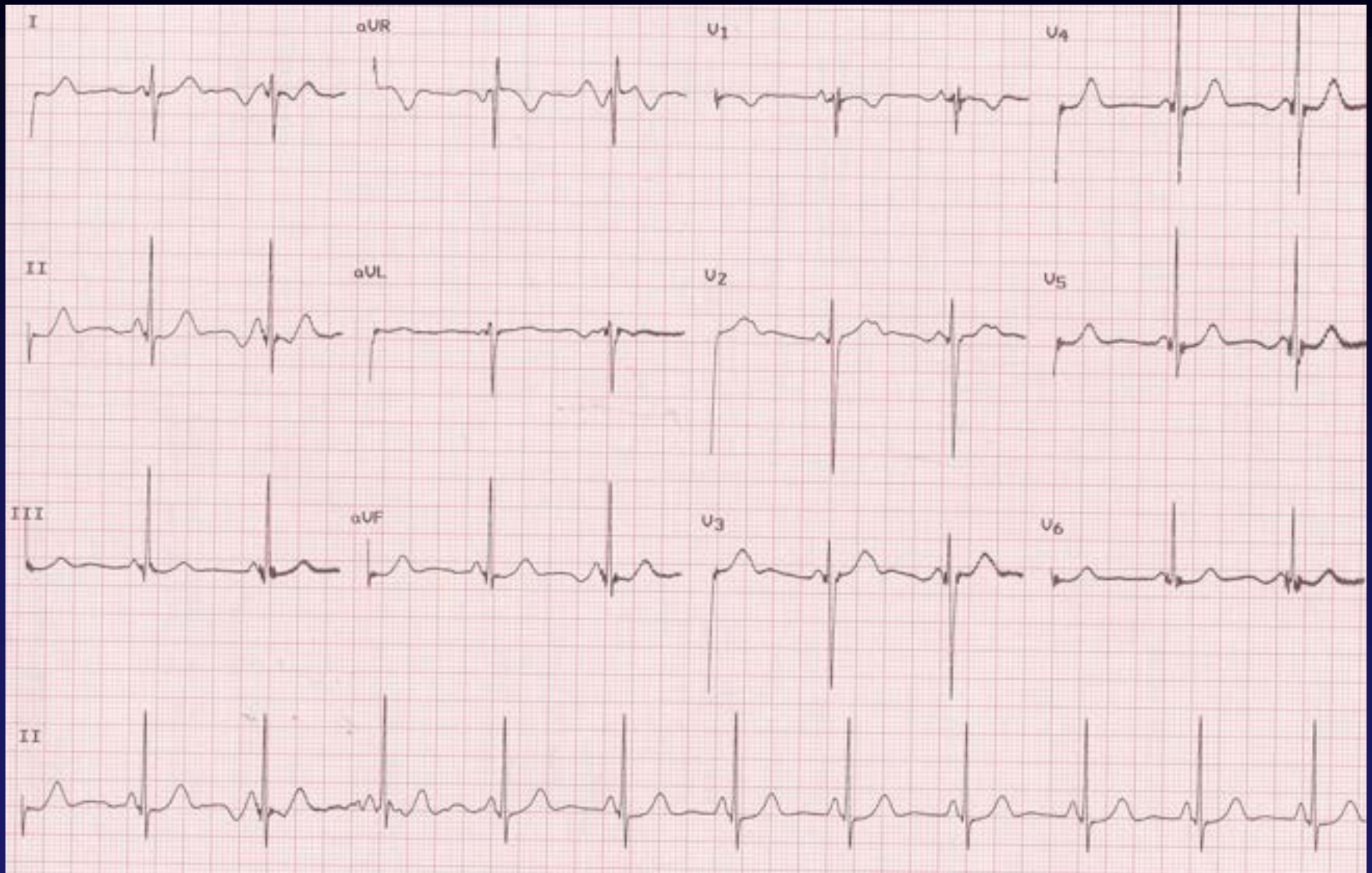
Normal QRS Axis



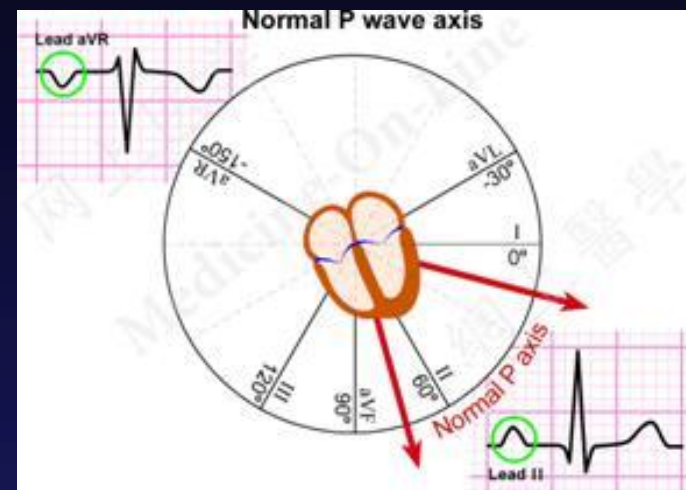
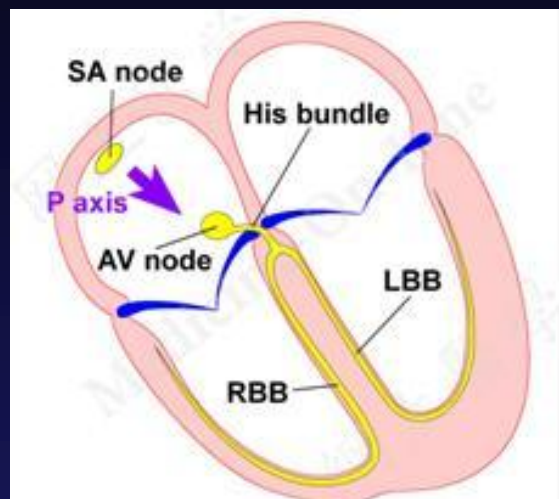
Left Axis Deviation



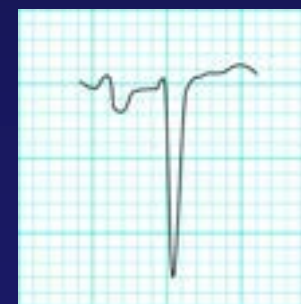
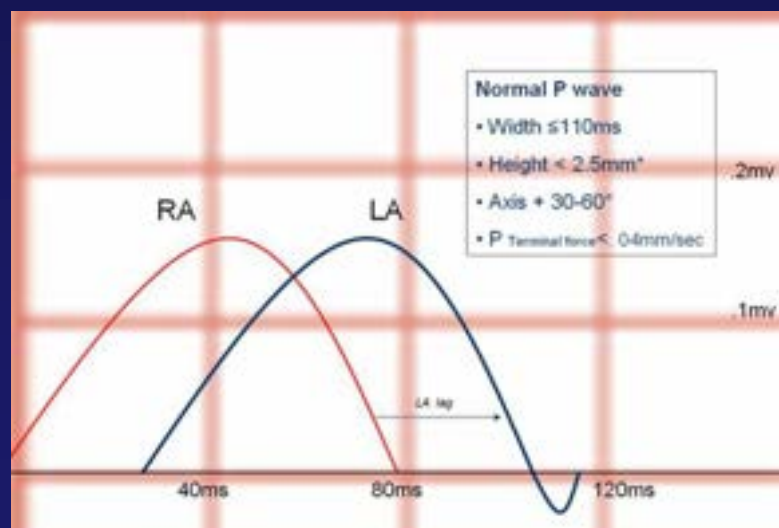
Right Axis Deviation



Sinus P Wave



II

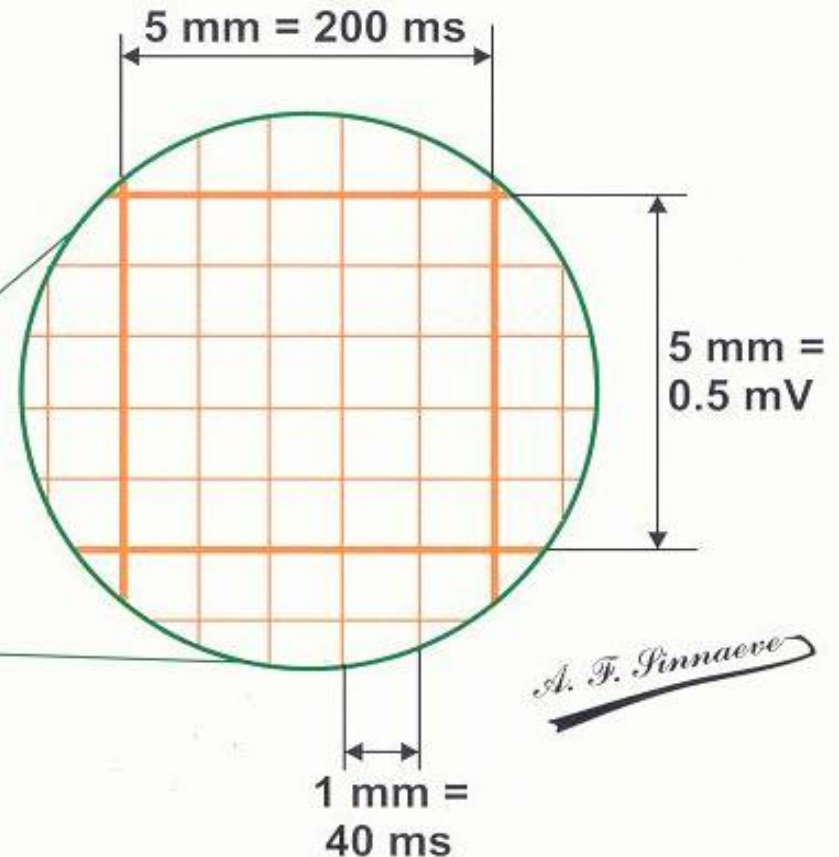
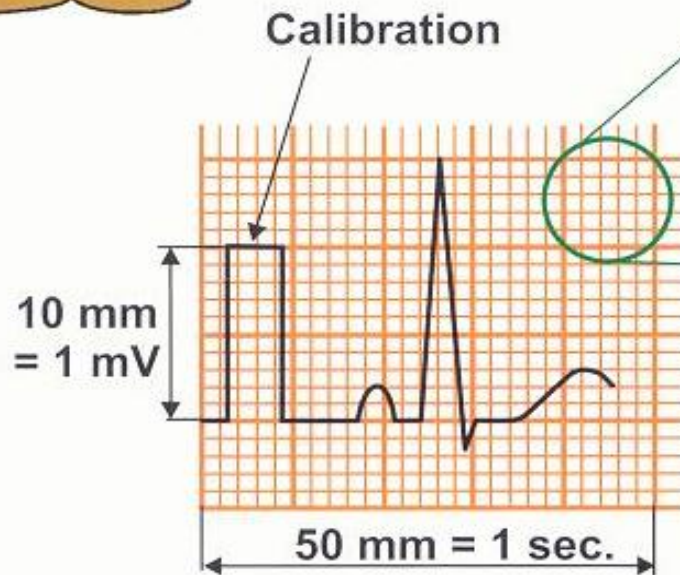


V1

TIMING INTERVALS VERSUS RATE



This is elementary !
Everybody should
know that !!!

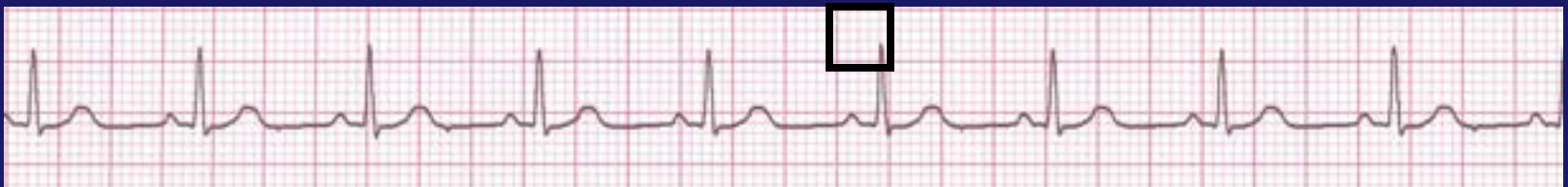
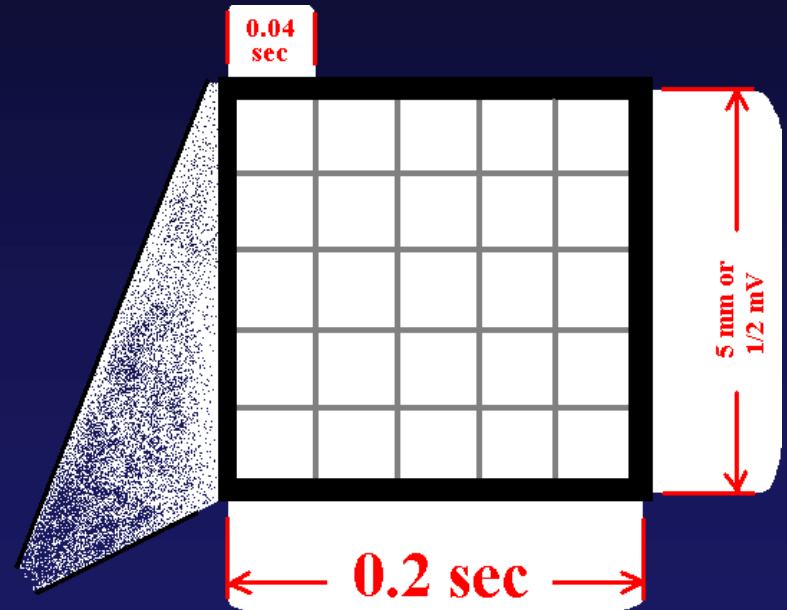


A. F. Pinnaeve

The paper speed is normally 25 mm/s,
thus 1 mm on the paper corresponds
with $1/25 \text{ s} = 0.04 \text{ s} = 40 \text{ ms}$

The ECG Paper

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



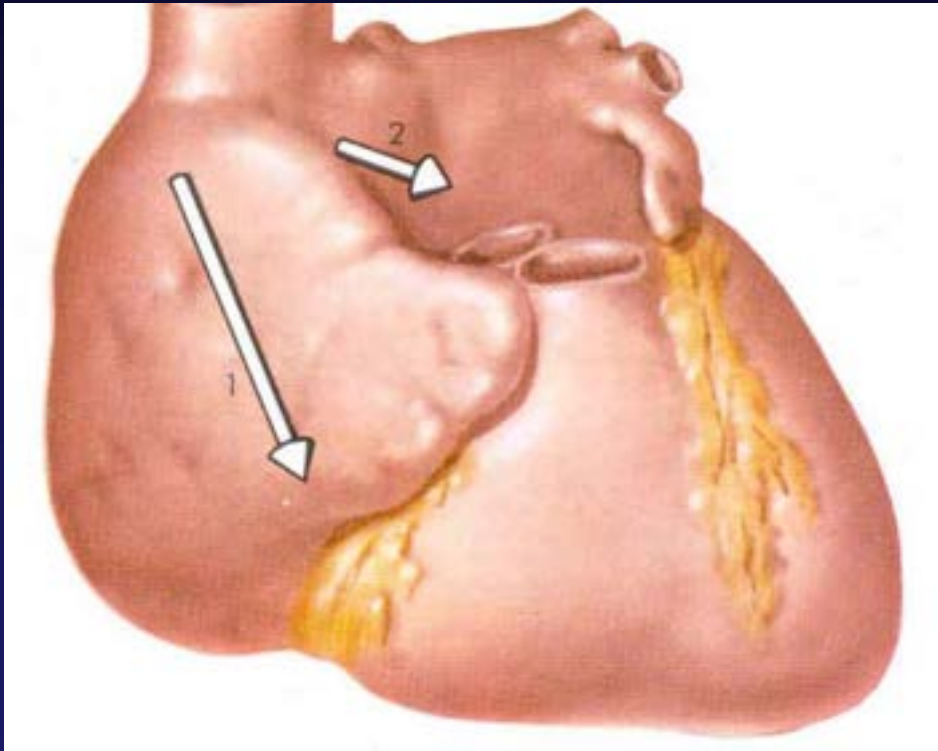
Timing in the ECG Paper



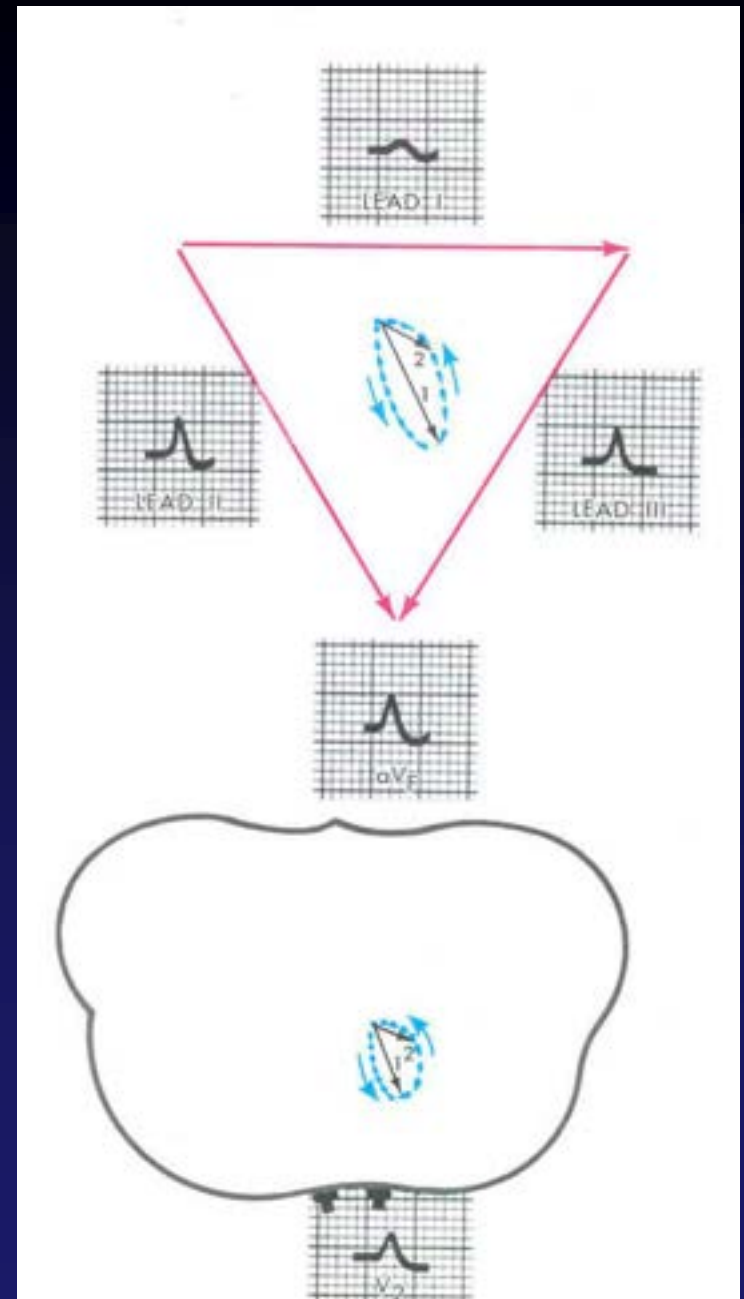
- Every 3 seconds (15 large boxes) is marked by a vertical line.
- This helps when calculating the heart rate.

Major ECG Abnormalities

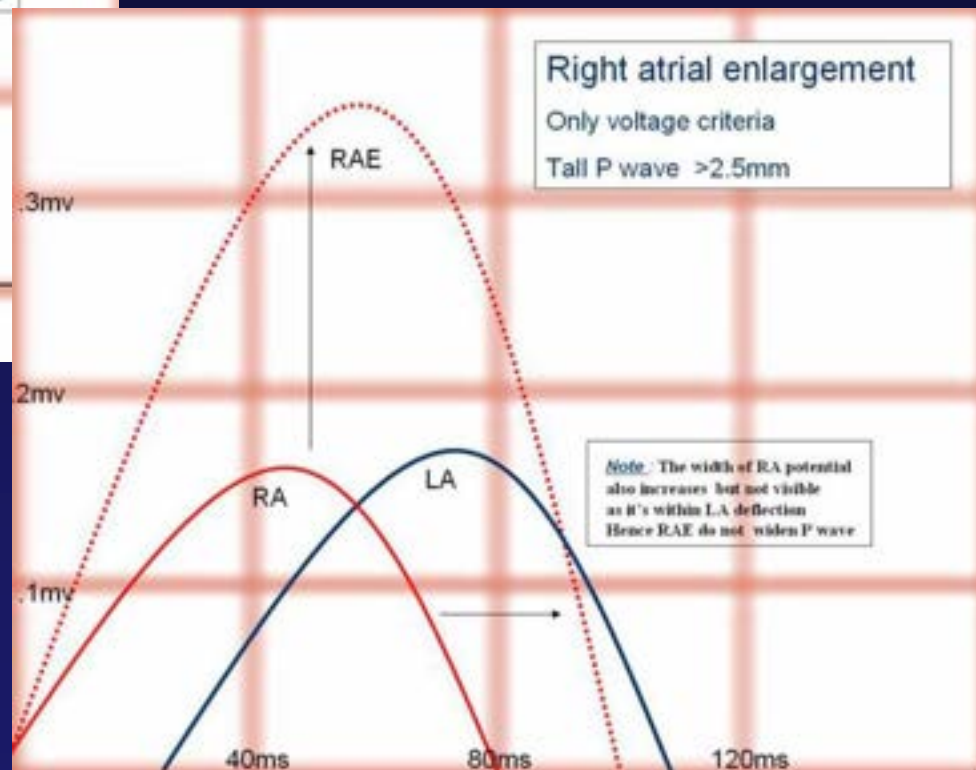
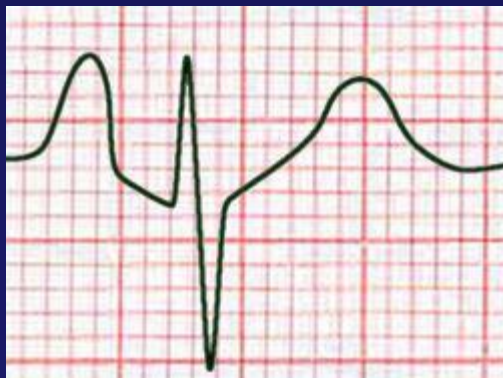
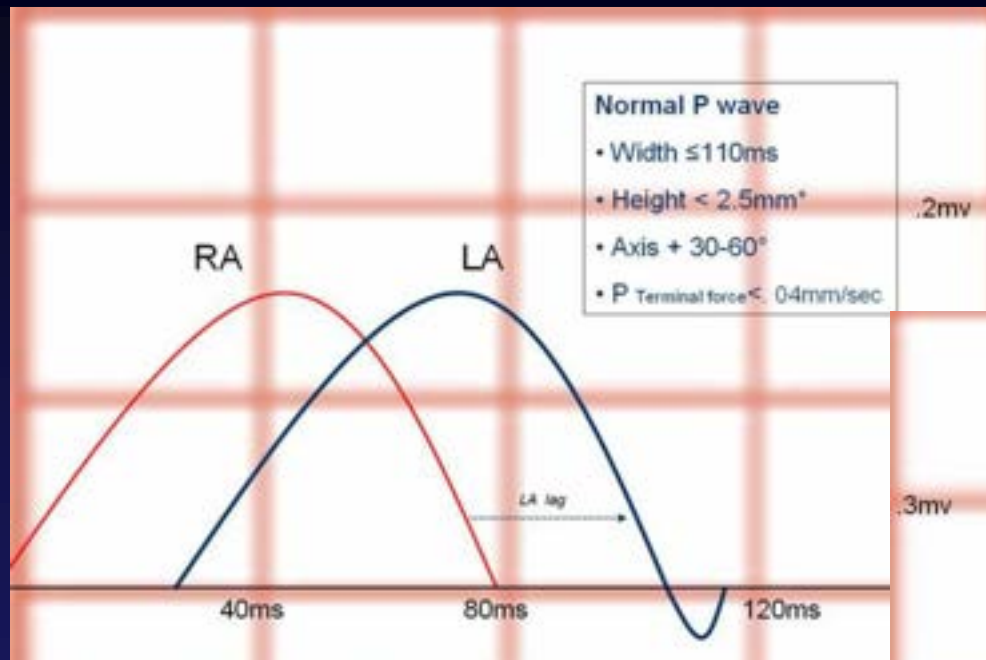
Right Atrial Enlargement



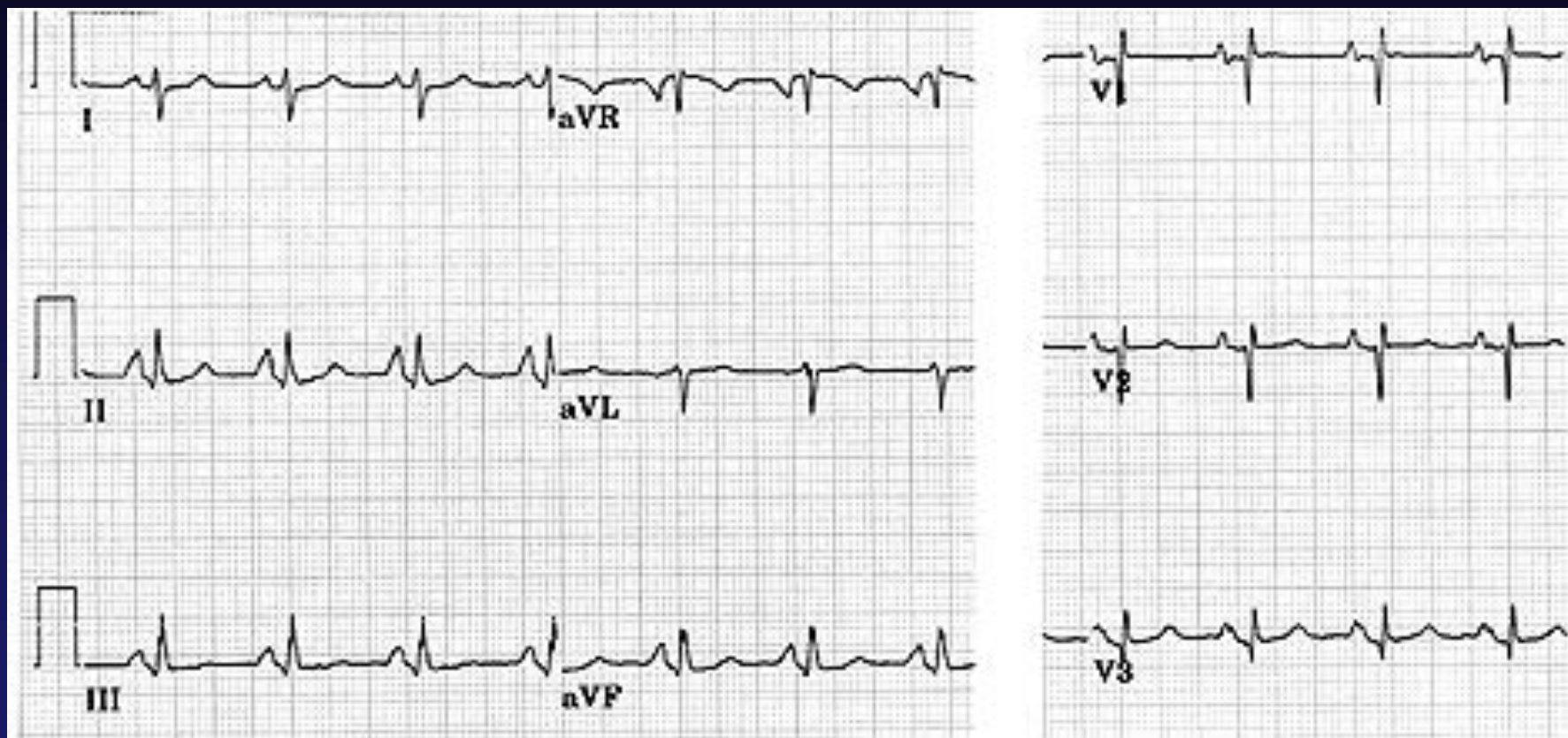
P Pulmonale, Amplitude ≥ 2.5 mm



Atrial Activation



Right Atrial Enlargement

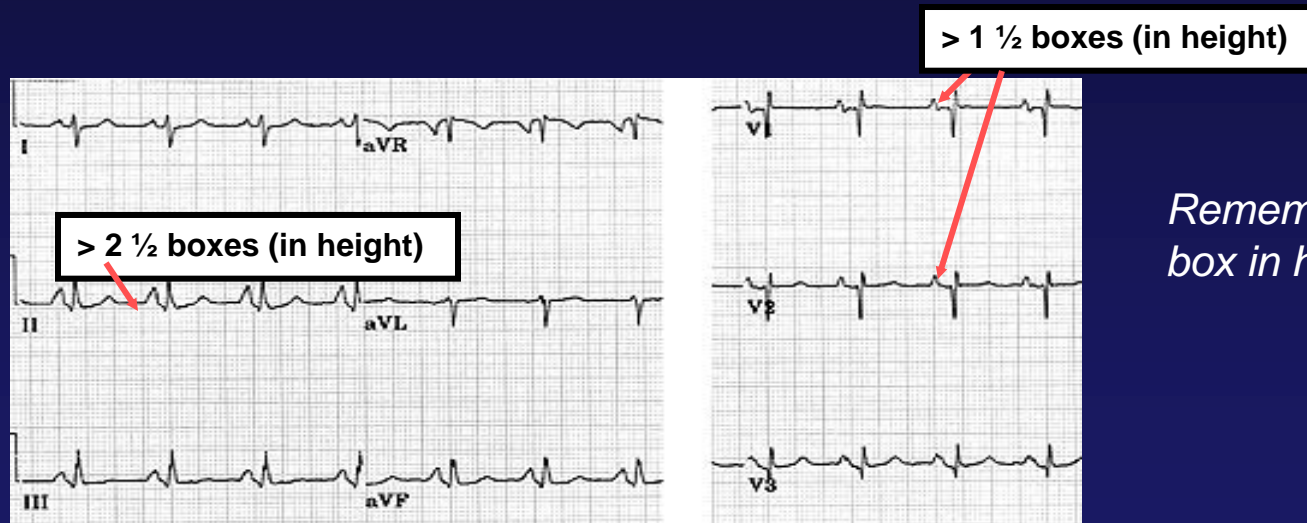


The P waves are tall, especially in leads II, III and aVF.

Right Atrial Enlargement

– To diagnose RAE you can use the following criteria:

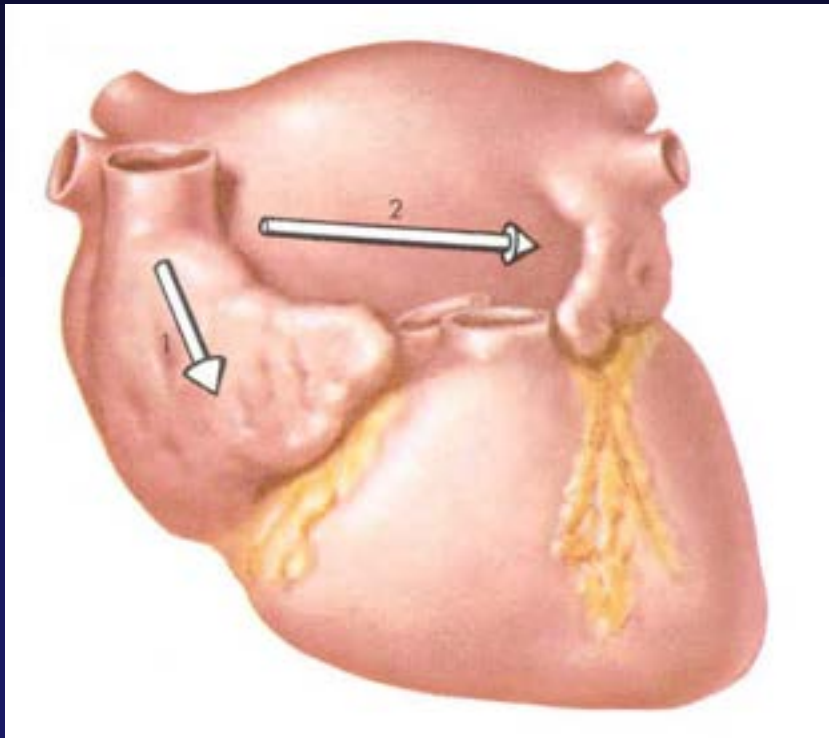
- II $P > 2.5 \text{ mm}$, or
- V1 or V2 $P > 1.5 \text{ mm}$



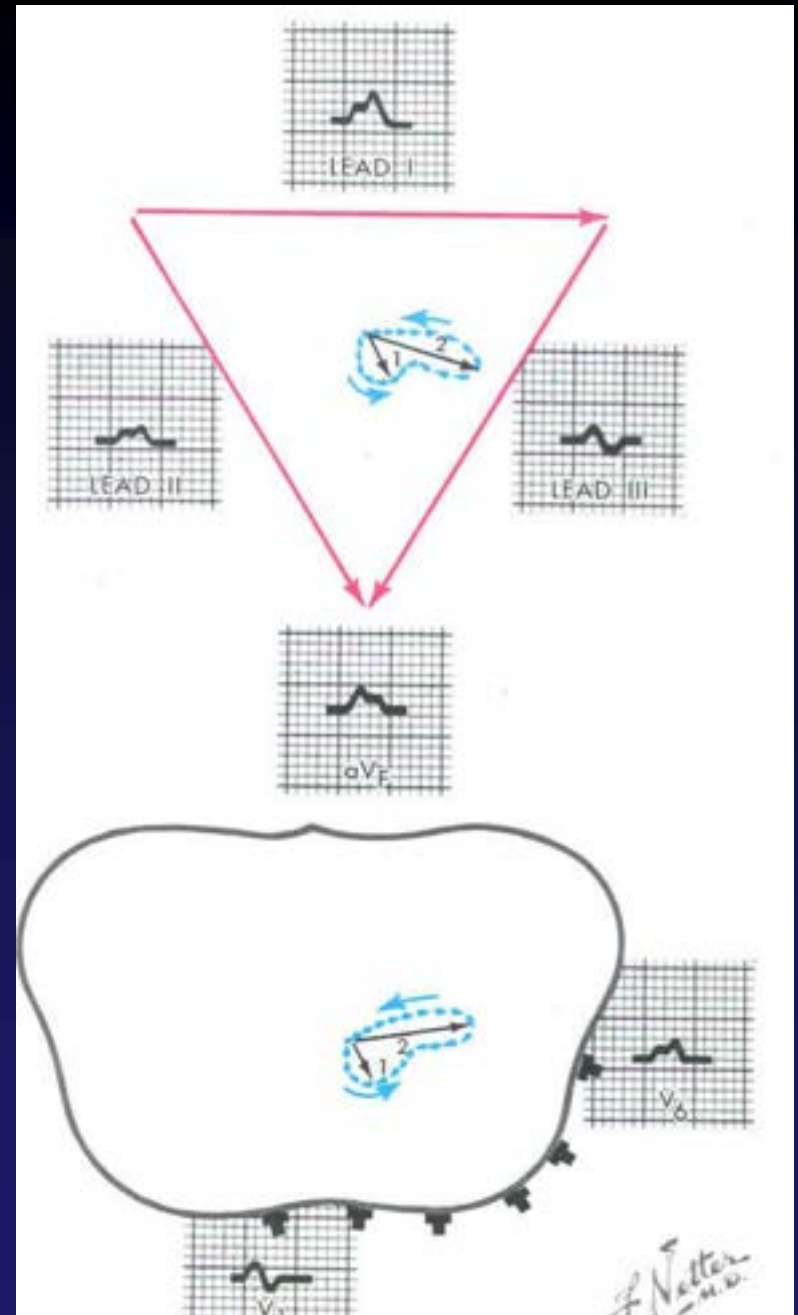
Remember 1 small box in height = 1 mm

A cause of RAE is RVH from pulmonary hypertension, hence P Pulmonale.

Left Atrial Enlargement



P Mitrale, Duration ≥ 120 ms



Atrial Activation

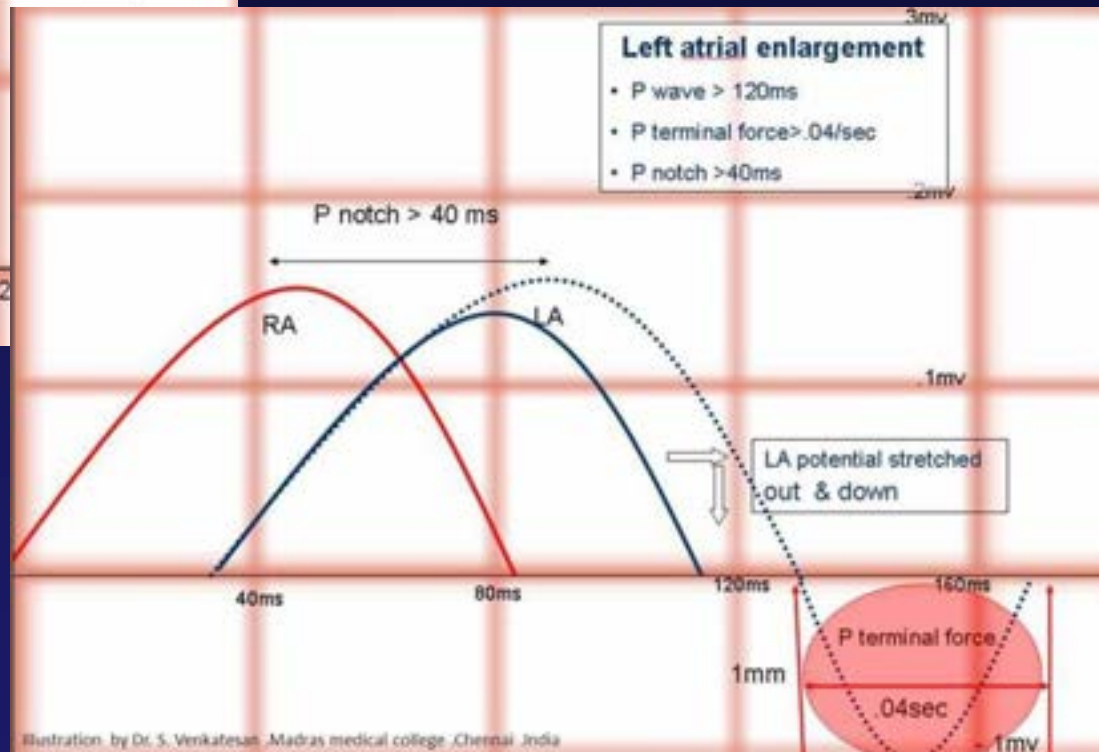
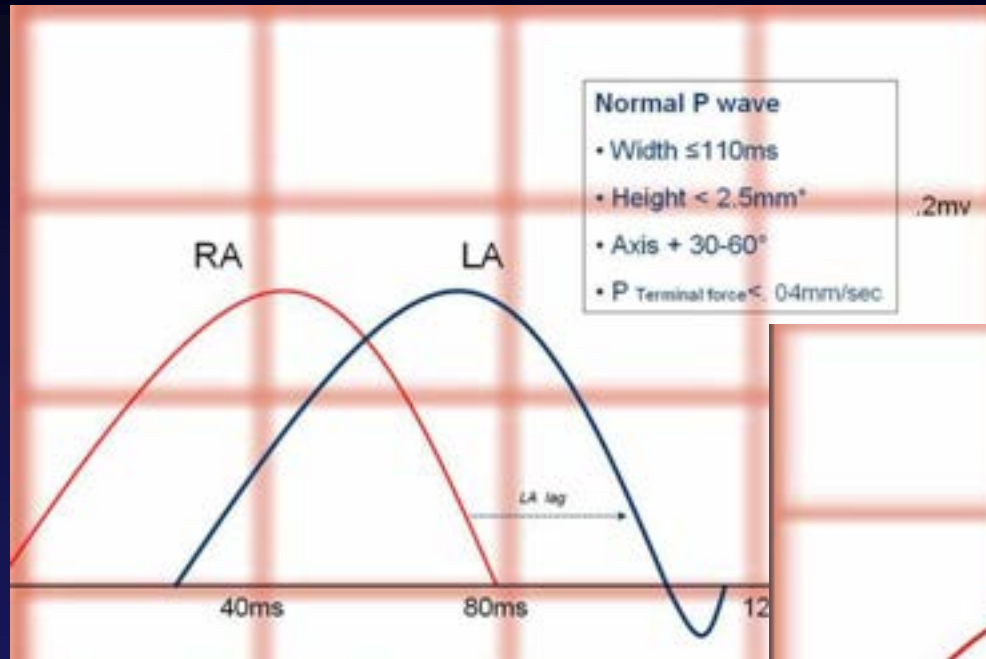
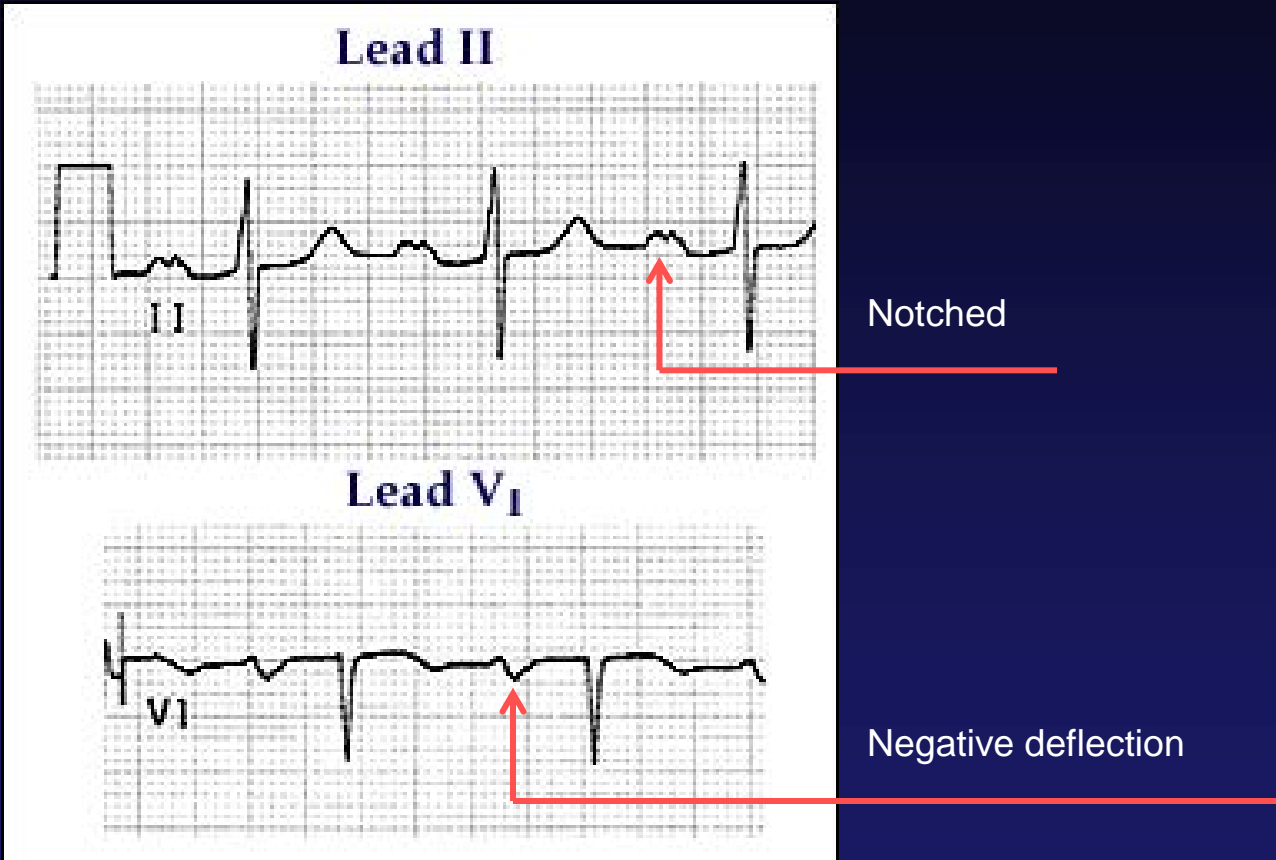


Illustration by Dr. S. Venkatesan, Madras medical college, Chennai, India

Left Atrial Enlargement



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

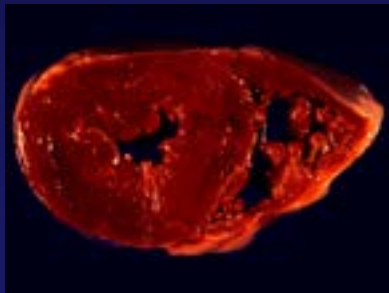
LAE

A common cause of LAE has been Mitral Stenosis, hence P Mitrale.

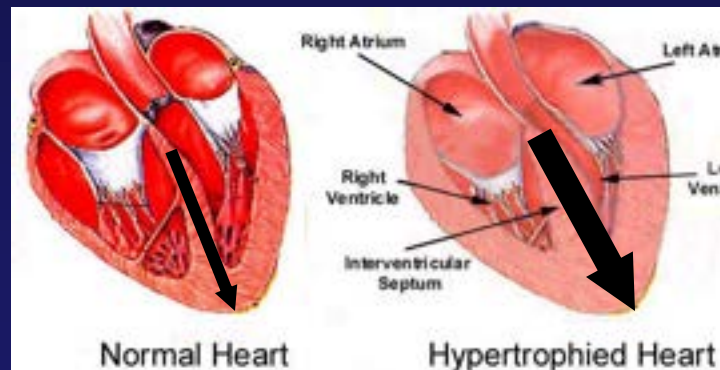
Left Ventricular Hypertrophy

Why is left ventricular hypertrophy characterized by tall QRS complexes?

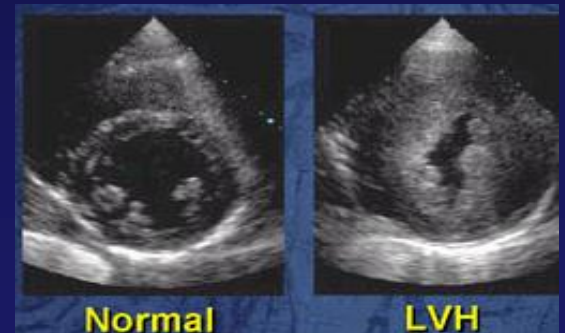
As the heart muscle wall thickens there is an increase in electrical forces moving through the myocardium resulting in increased QRS voltage.



LVH

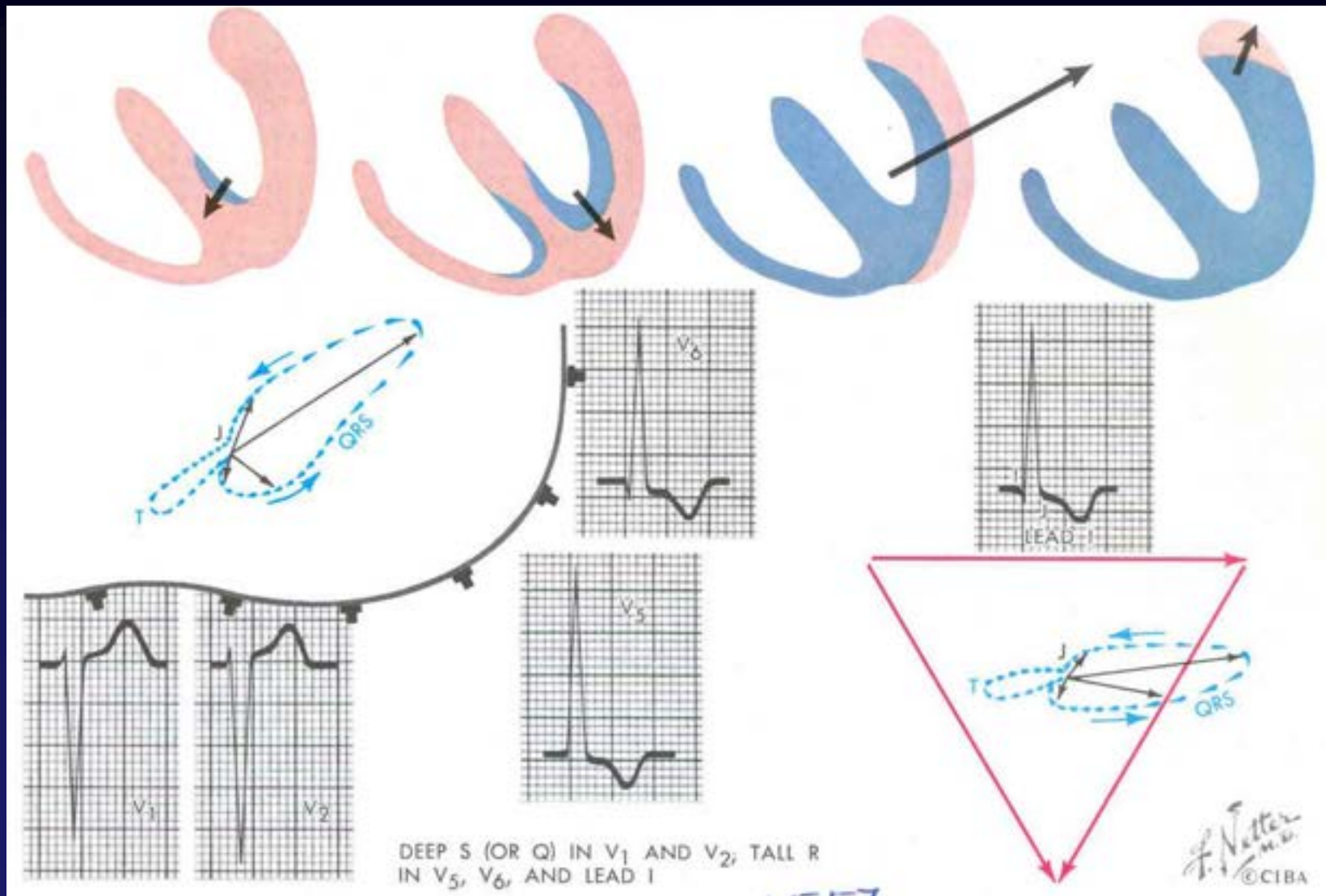


Increased QRS voltage



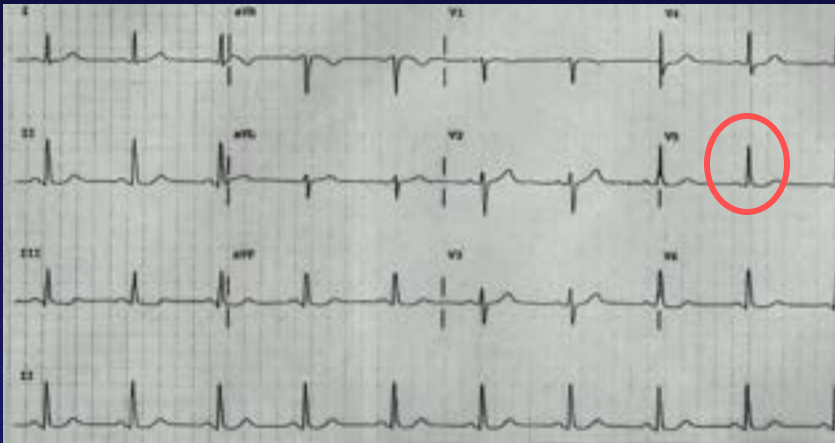
Echocardiogram

Left Ventricular Hypertrophy



Left Ventricular Hypertrophy

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



Normal

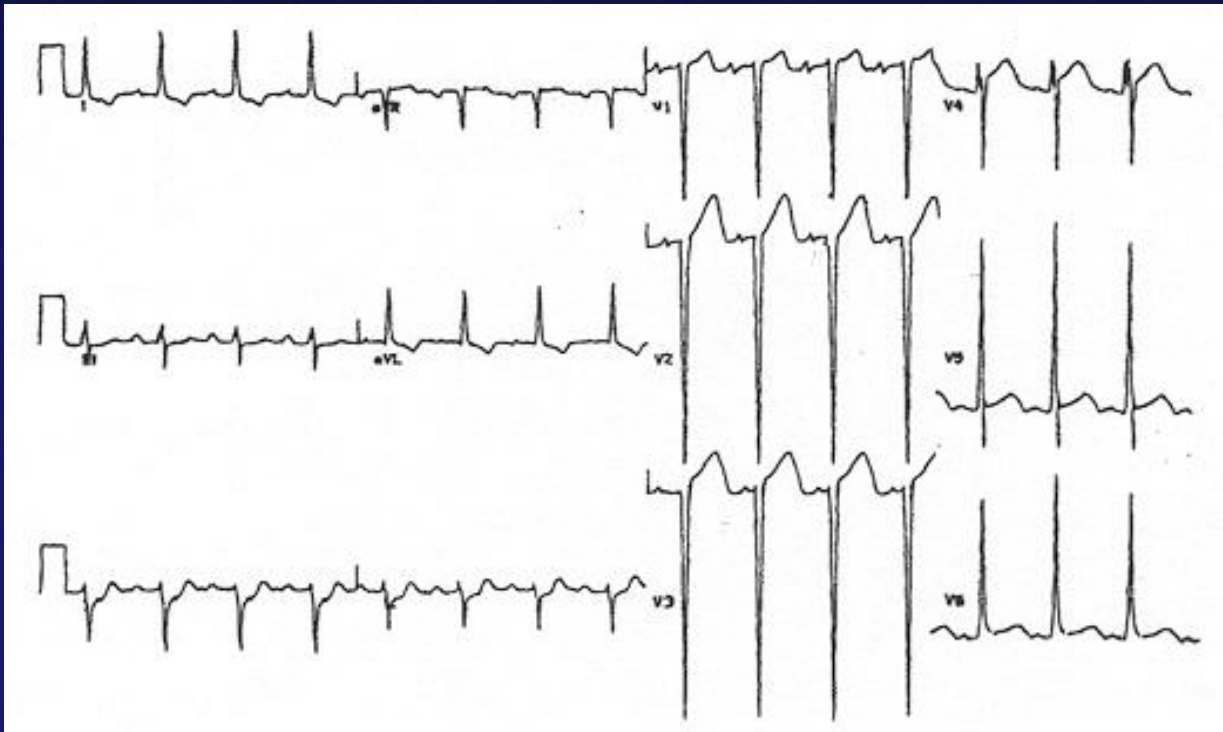


Left Ventricular Hypertrophy

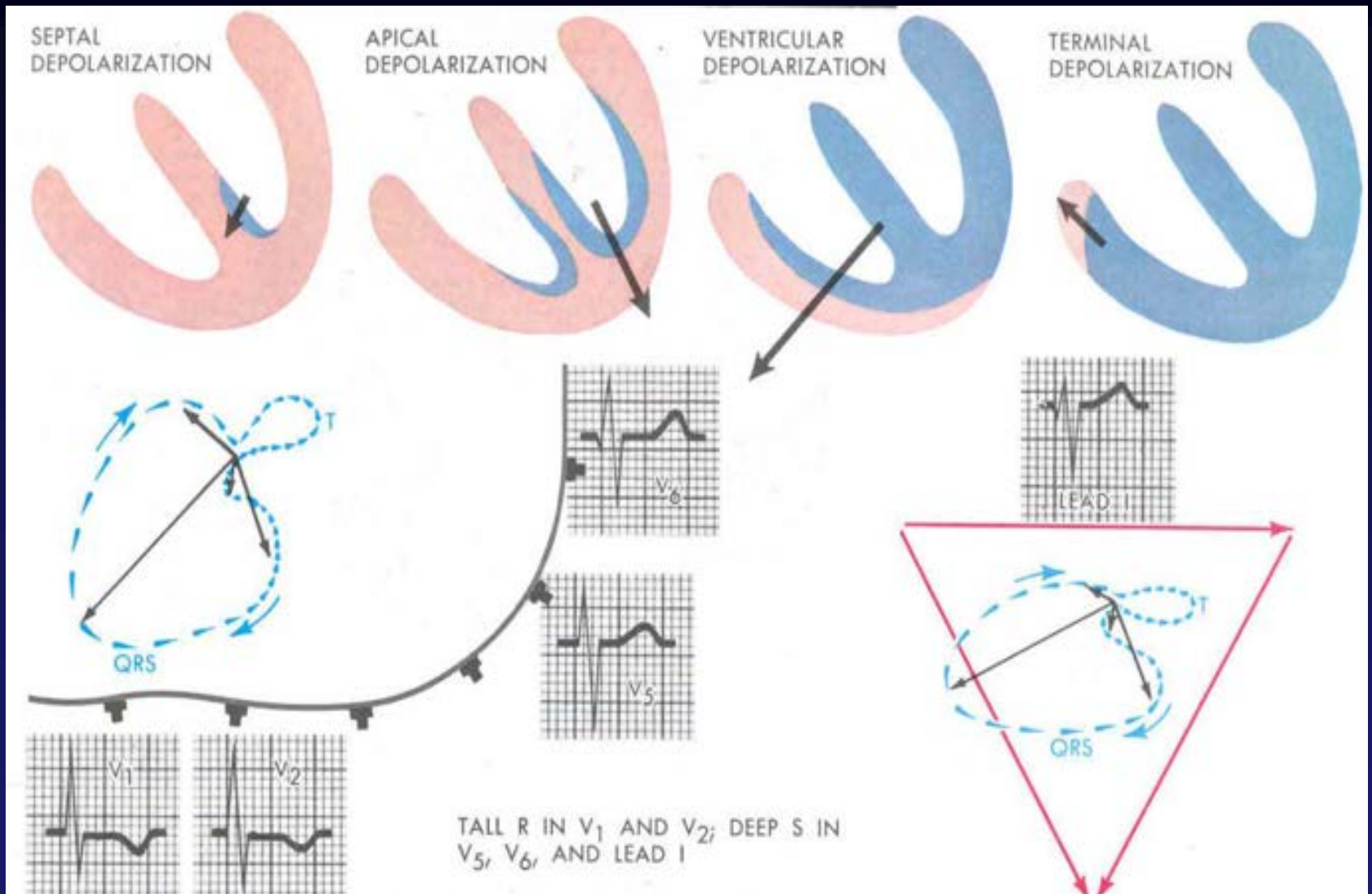
Answer: The QRS complexes are very tall
(increased voltage)

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.

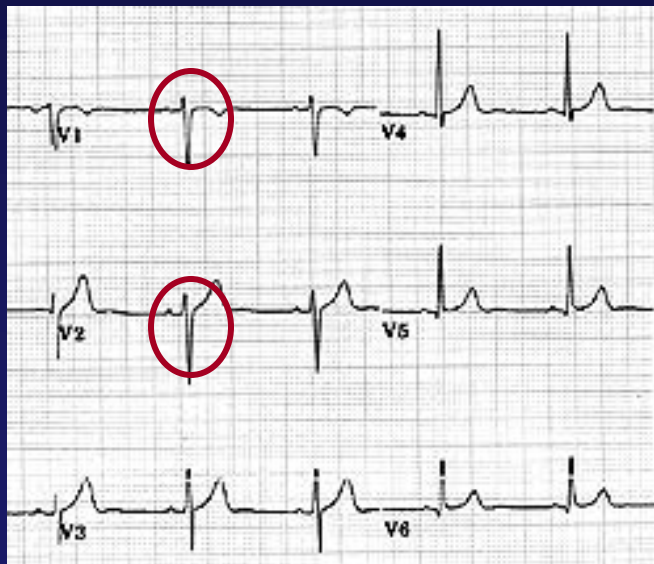


Right Ventricular Hypertrophy

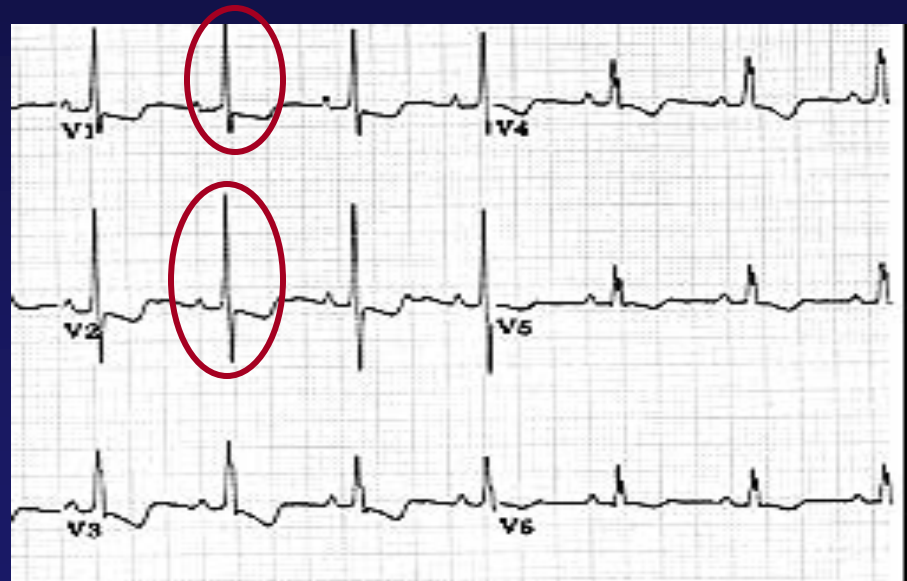


Right Ventricular Hypertrophy

- 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

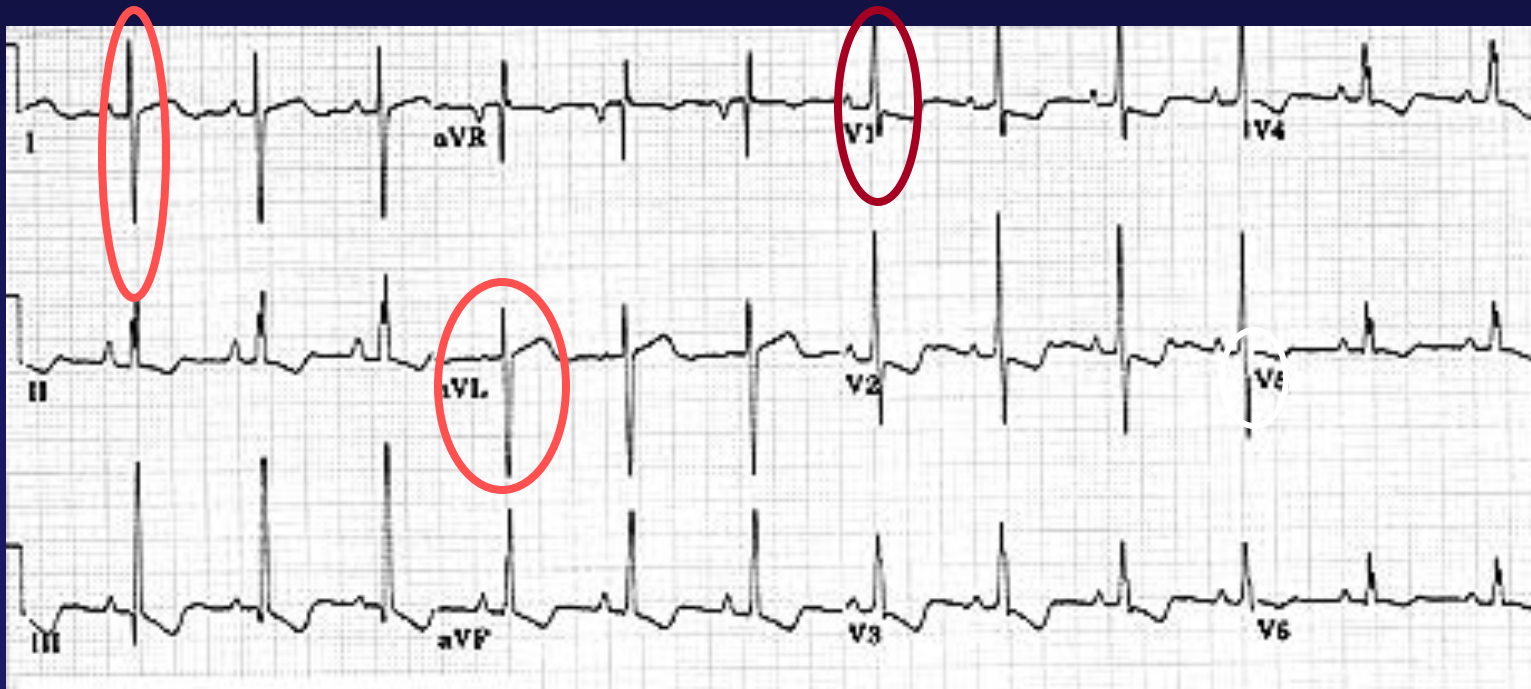


RVH

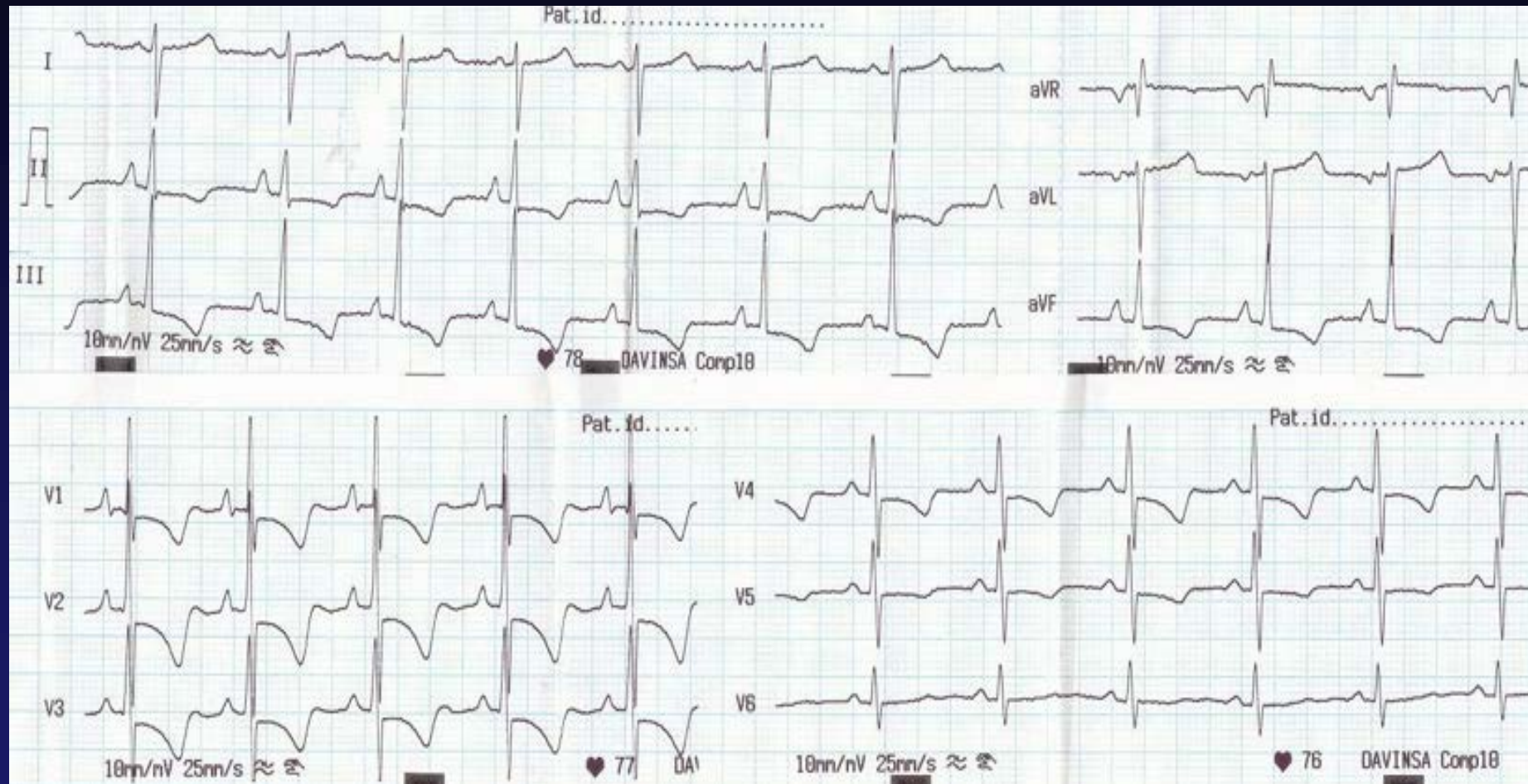
Right Ventricular Hypertrophy

To diagnose RVH you can use the following criteria:

- **Right axis deviation**, and
- **V1 R wave > 7mm tall**



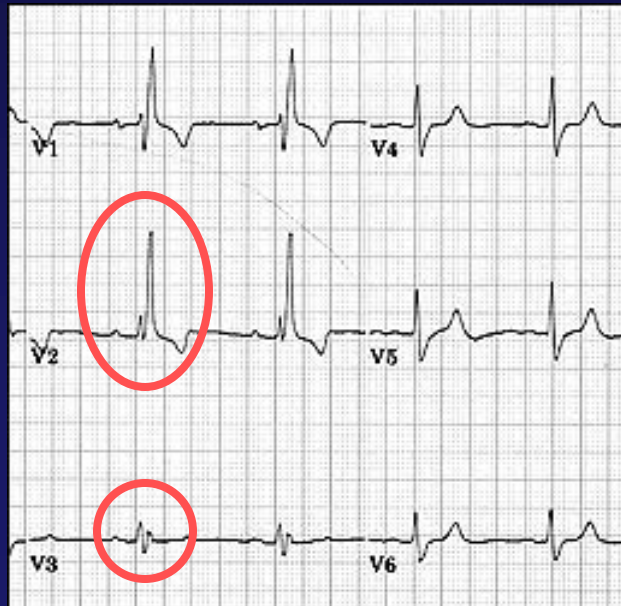
RVH, RA enlargement



Bundle Branch Blocks

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

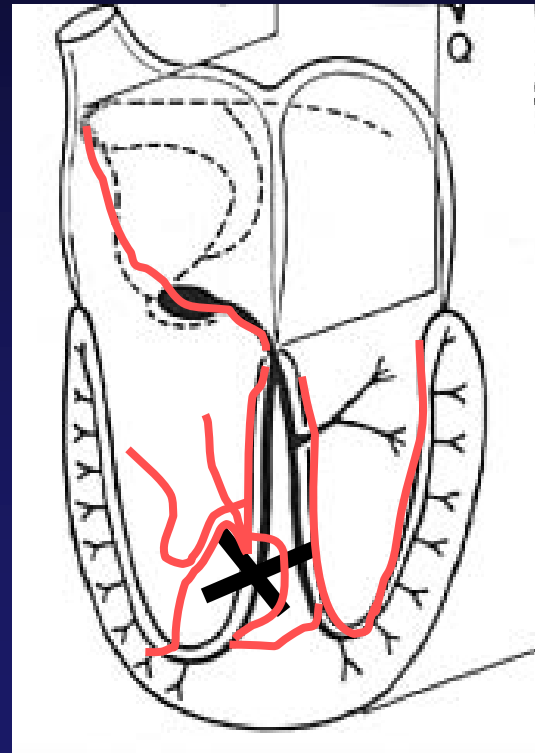
1. **QRS complex widens** (> 0.12 sec).
2. **QRS morphology changes** (varies depending on ECG lead, and if it is a right vs. left bundle branch block).



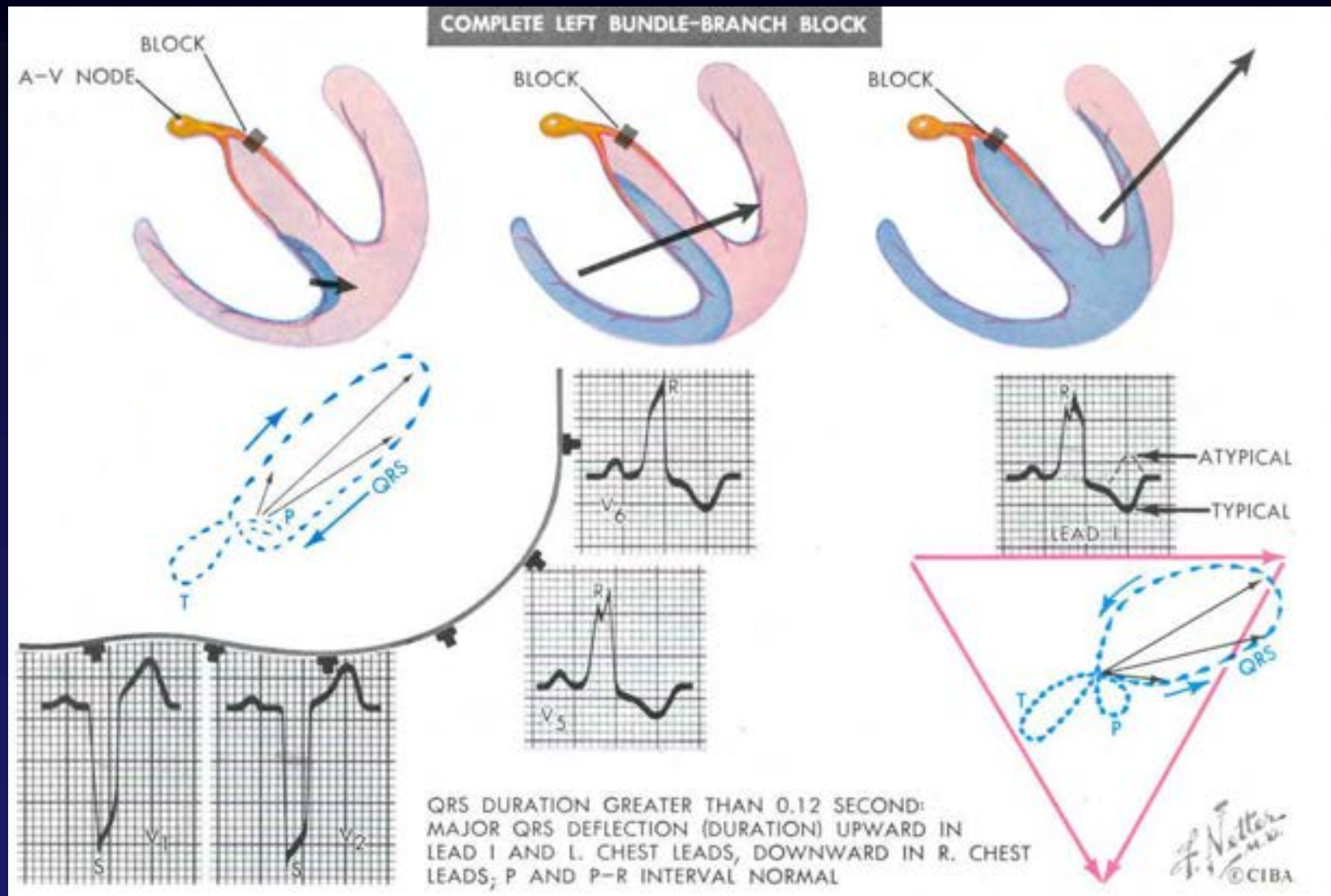
Bundle Branch Blocks

Why does the QRS complex widen?

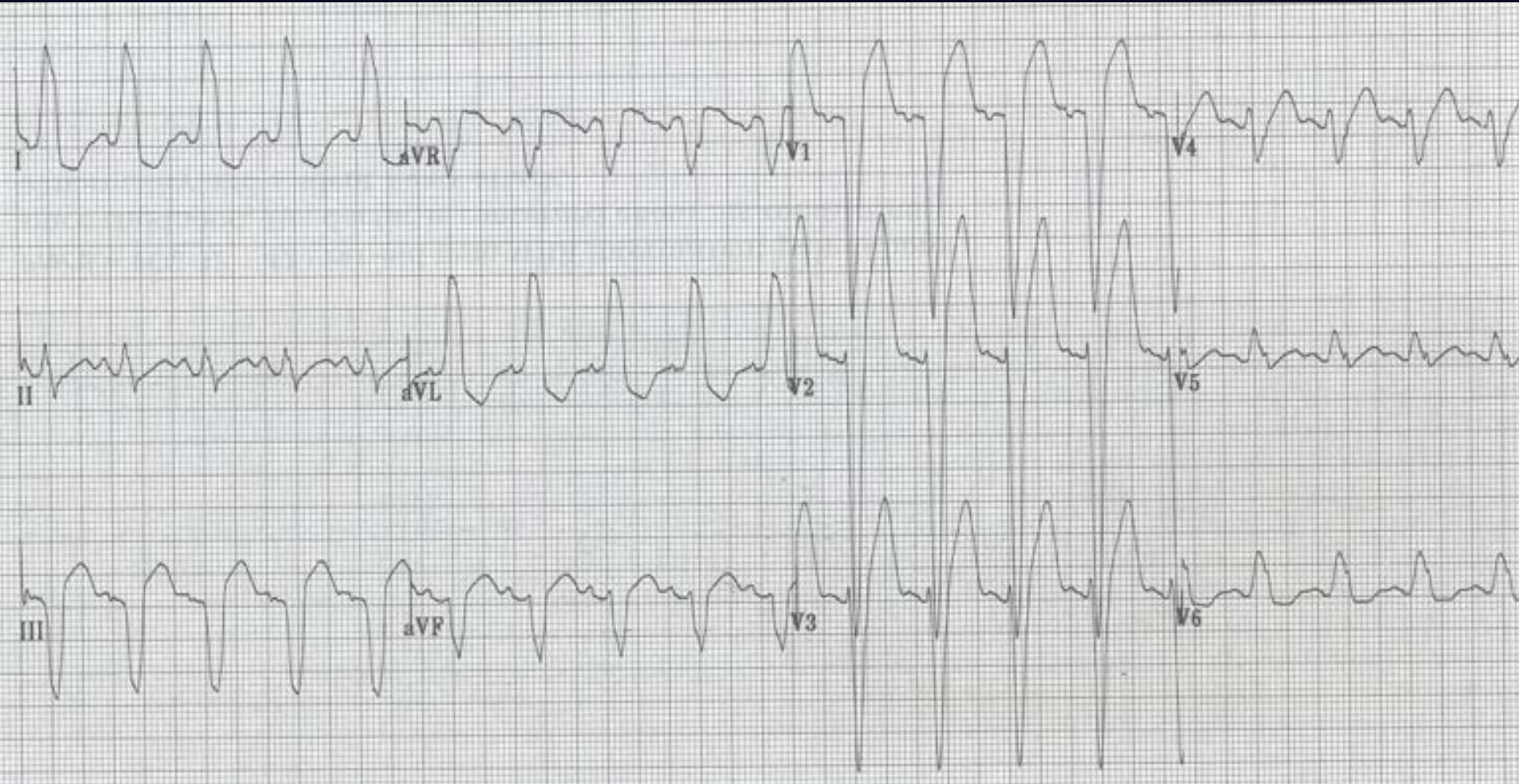
When the conduction pathway is blocked it will take longer for the electrical signal to pass throughout the ventricles.



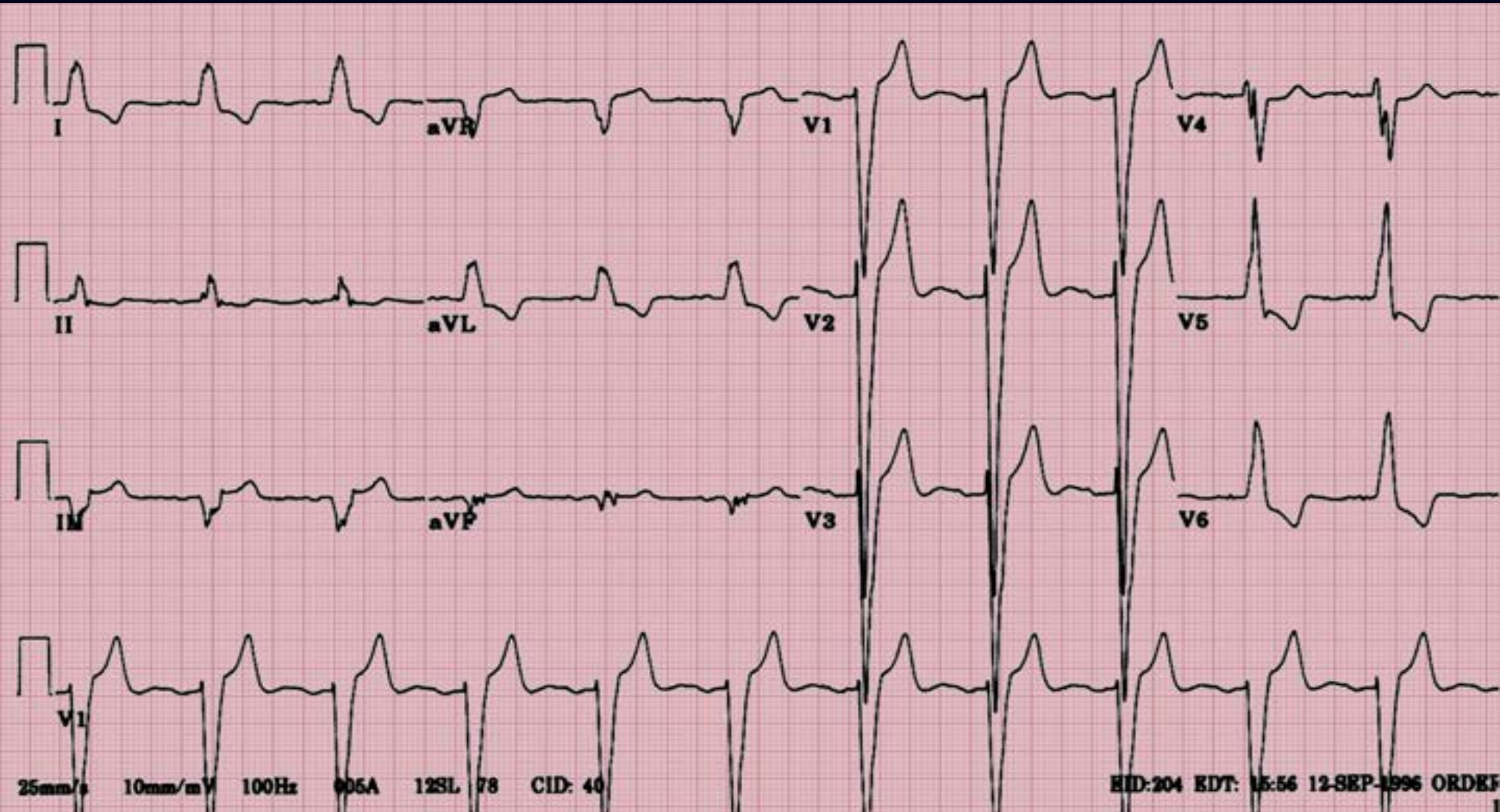
Left Bundle Branch Block



Left Bundle Branch Block

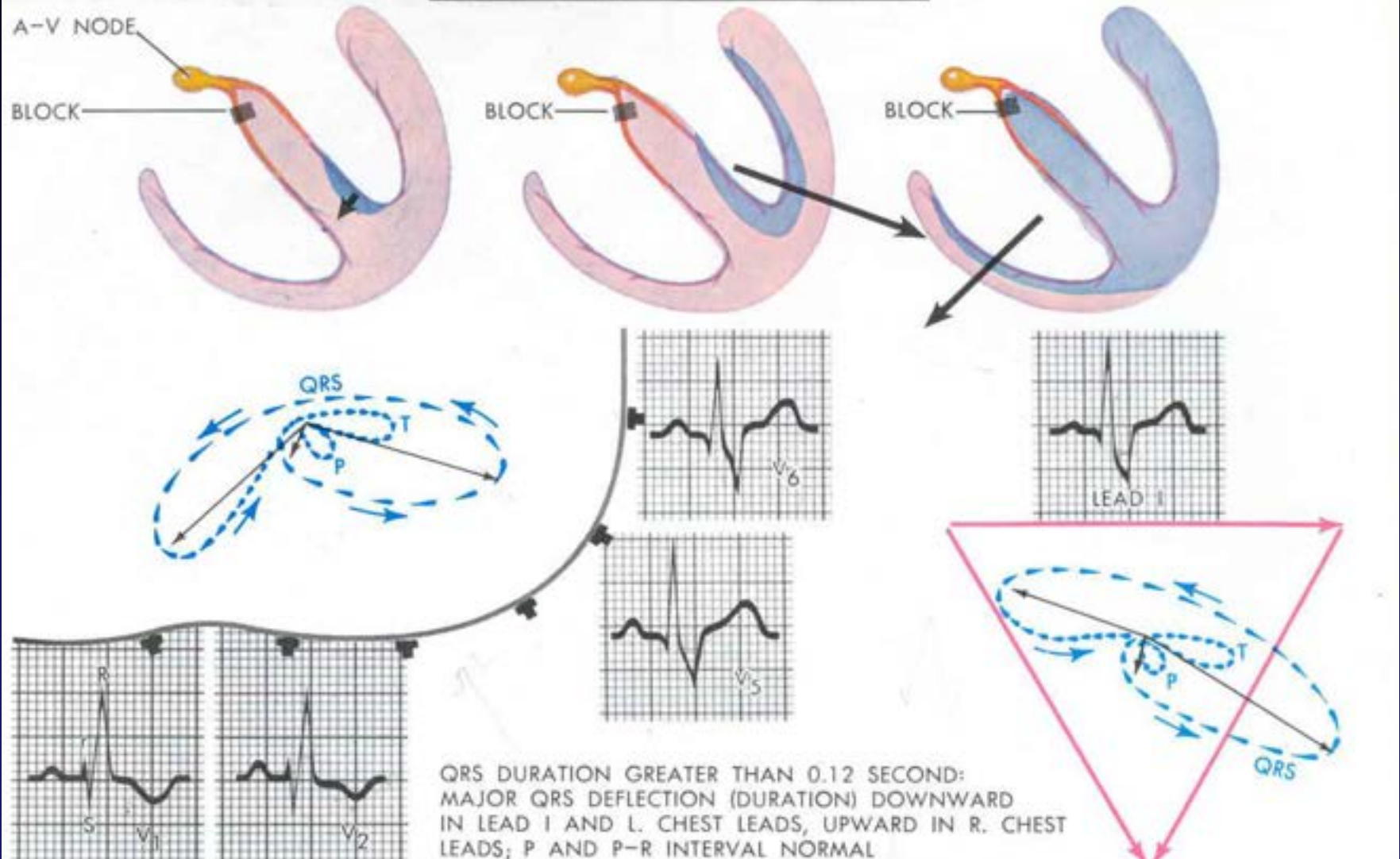


Left Bundle Branch Block



Right Bundle Branch Block

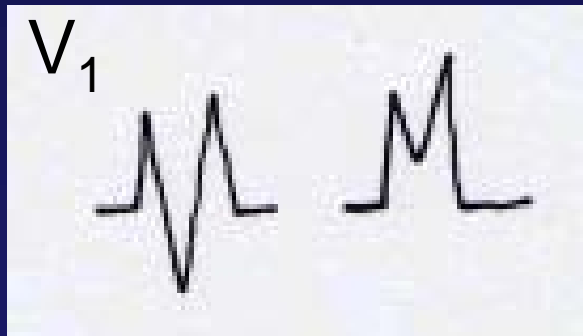
COMPLETE RIGHT BUNDLE-BRANCH BLOCK



Right Bundle Branch Blocks

What QRS morphology is characteristic?

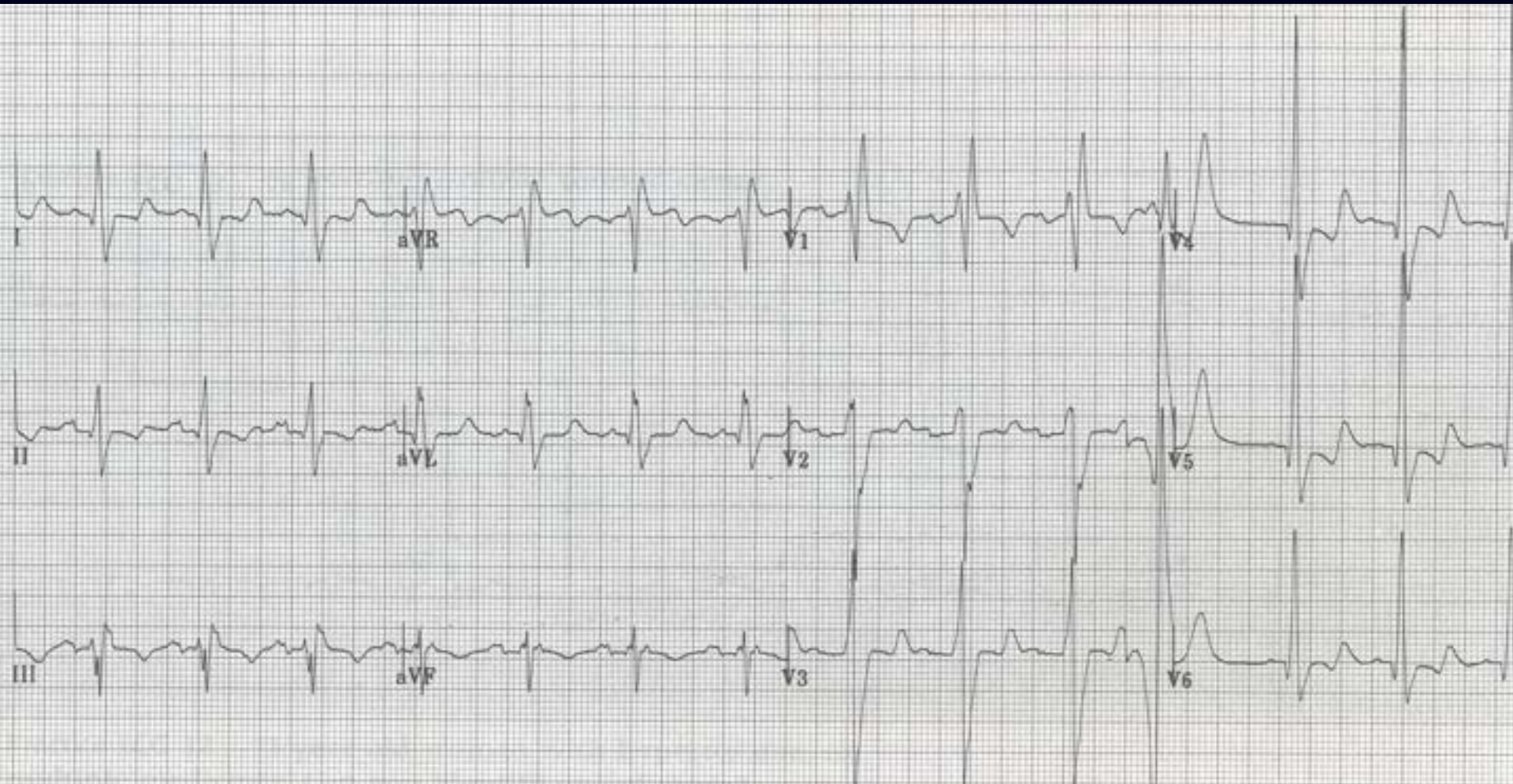
For RBBB the wide QRS complex assumes a unique, virtually diagnostic shape in those leads overlying the right ventricle (V_1 and V_2).



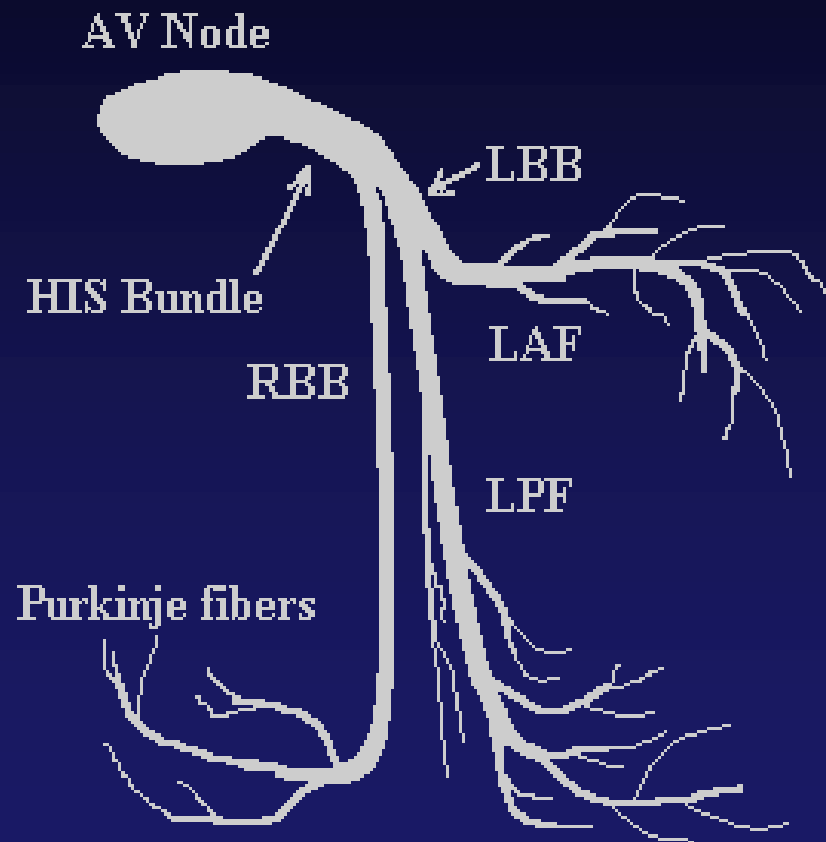
“Rabbit Ears”



RBBB

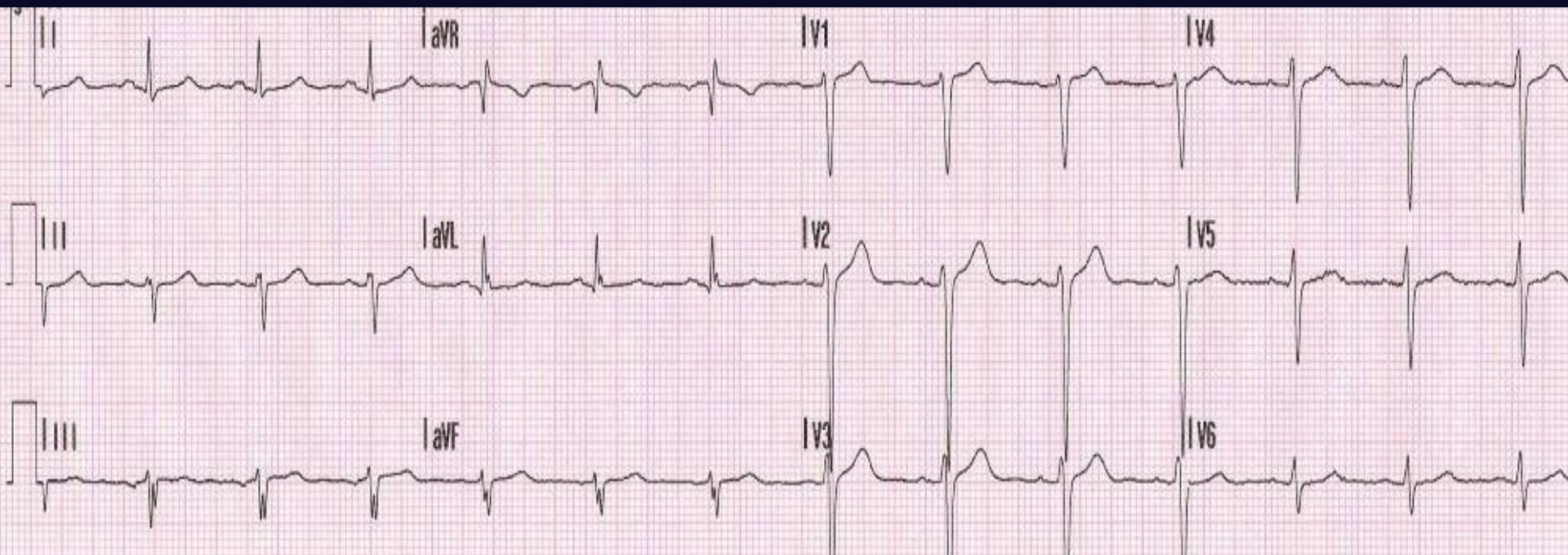


Left Bundle Branch Fascicles



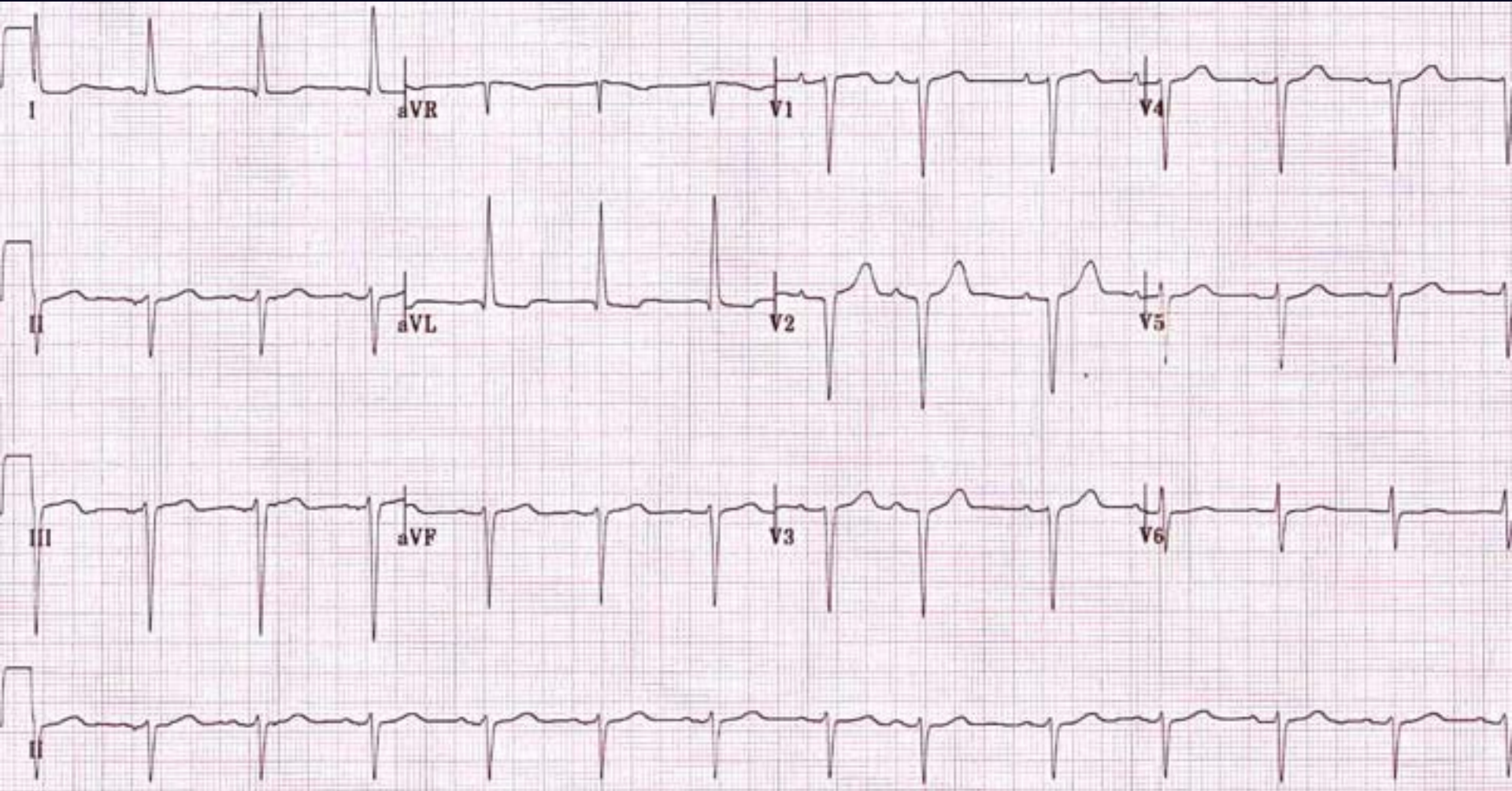
Left Anterior Fascicular Block

Left Anterior Hemiblock



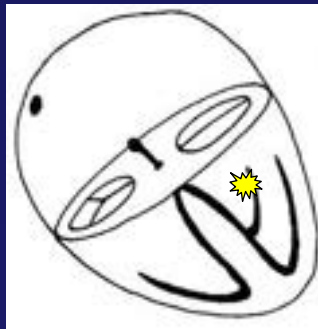
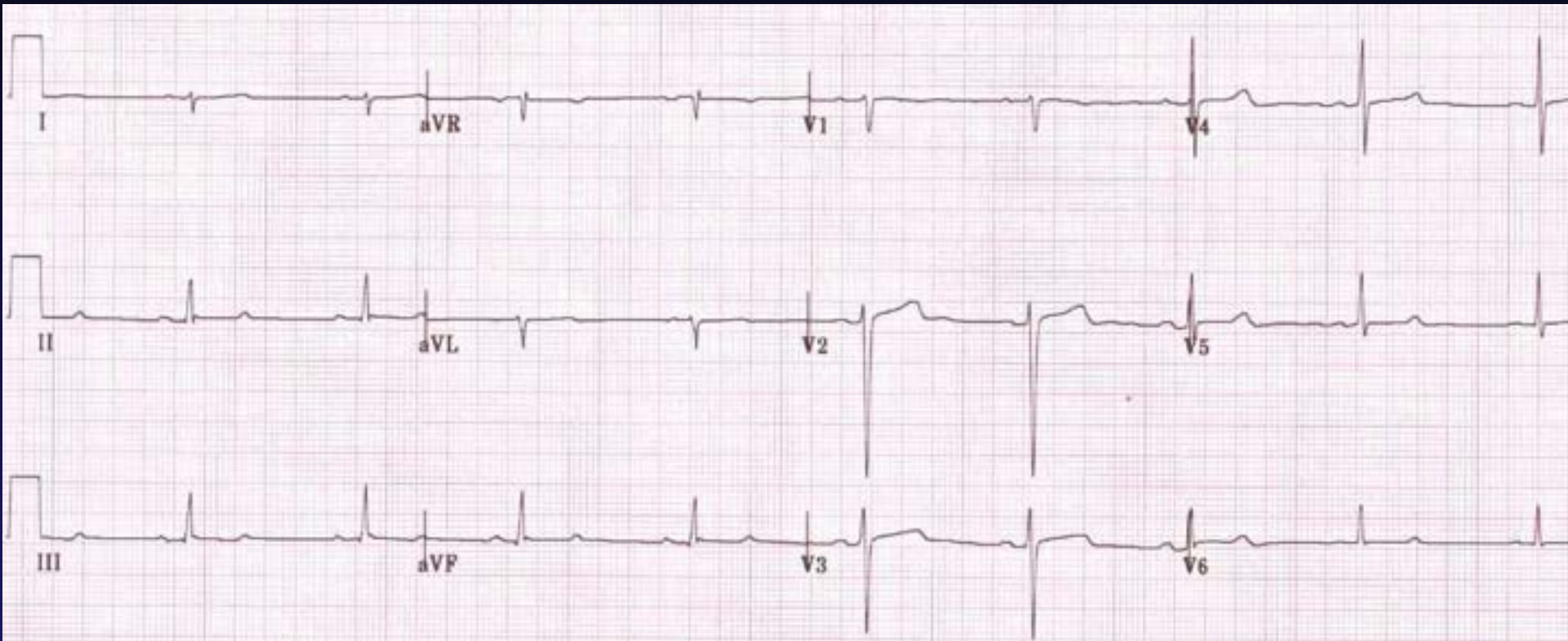
Left Anterior Fascicular Block

Left Anterior Hemiblock



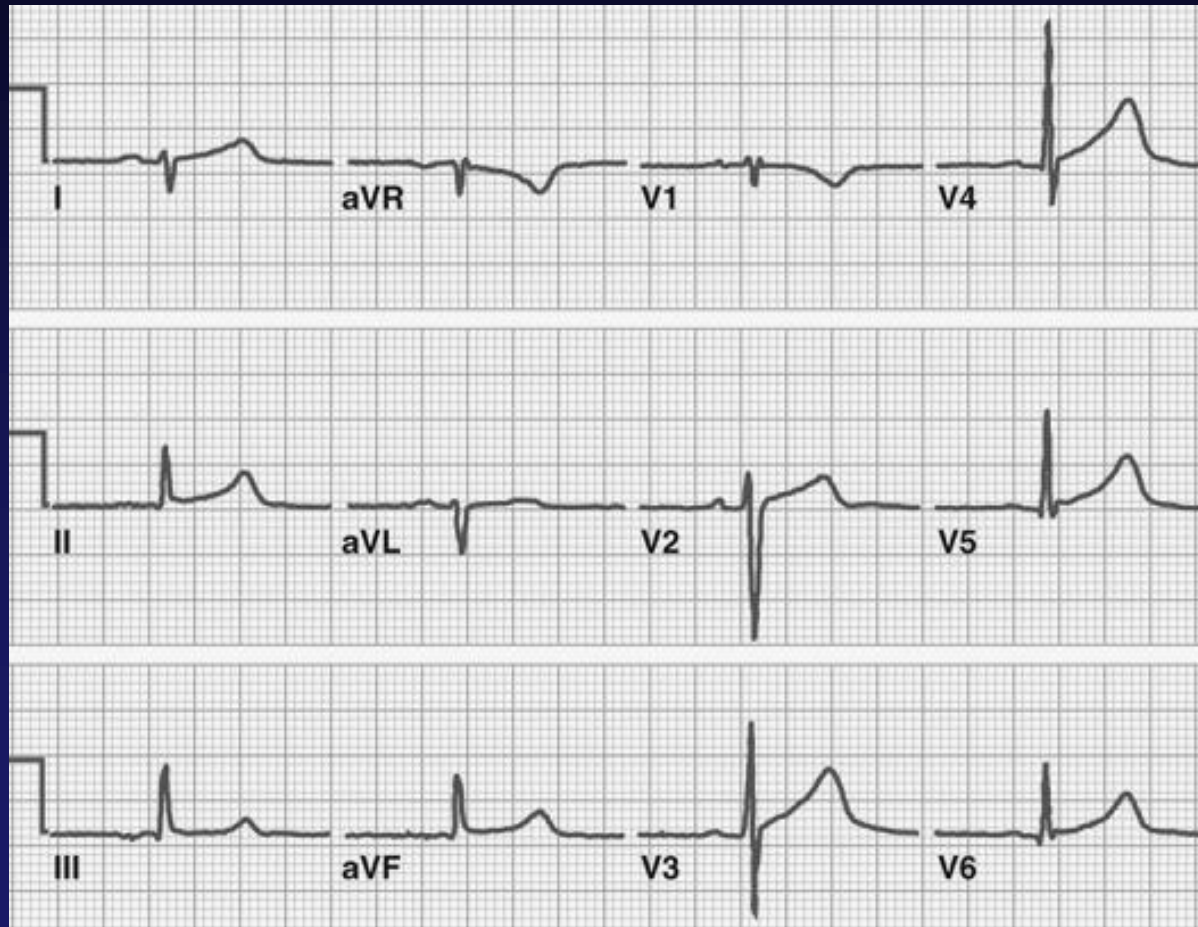
Left Posterior Fascicular Block

Left Posterior Hemiblock

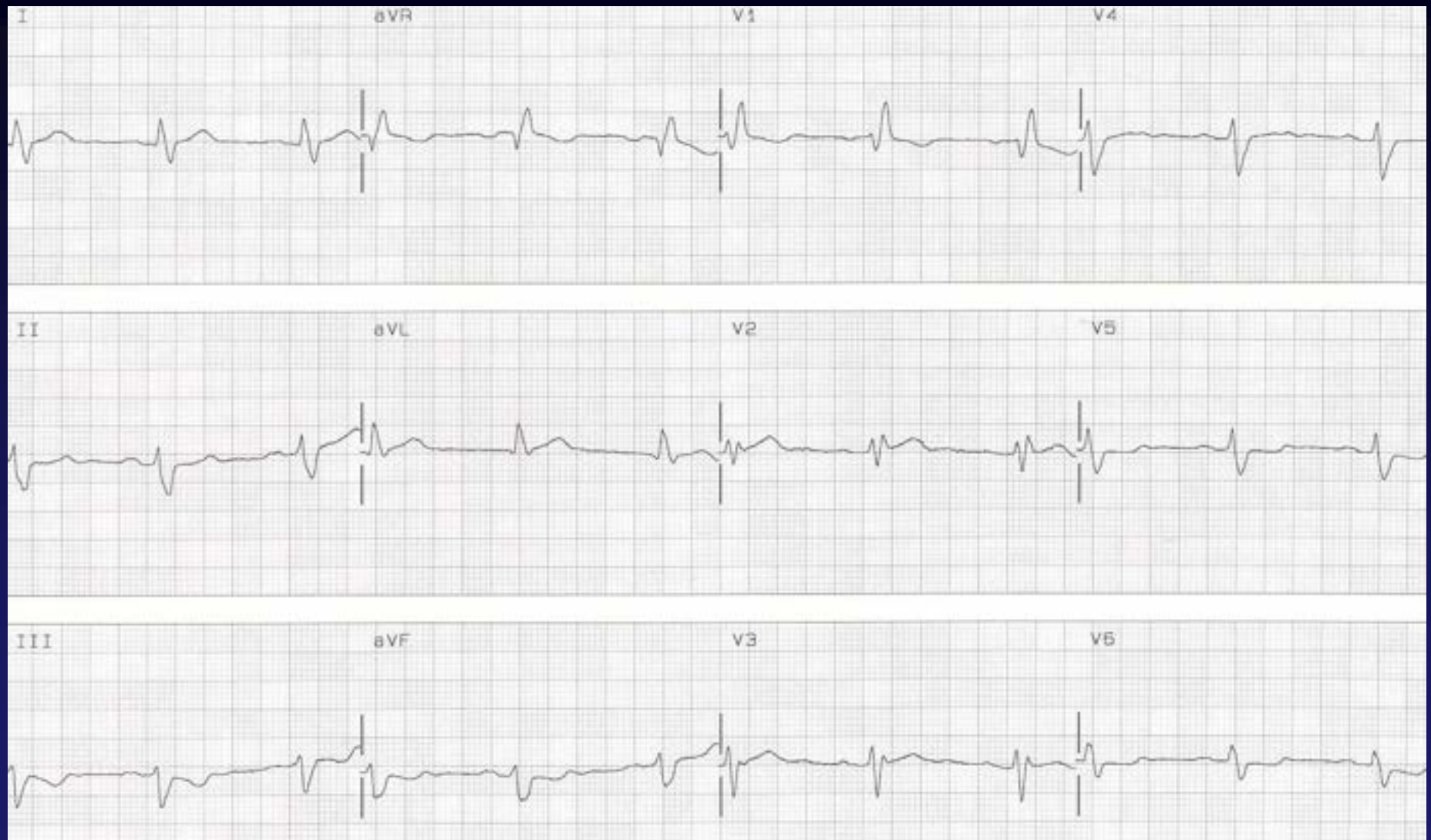


Left Posterior Fascicular Block

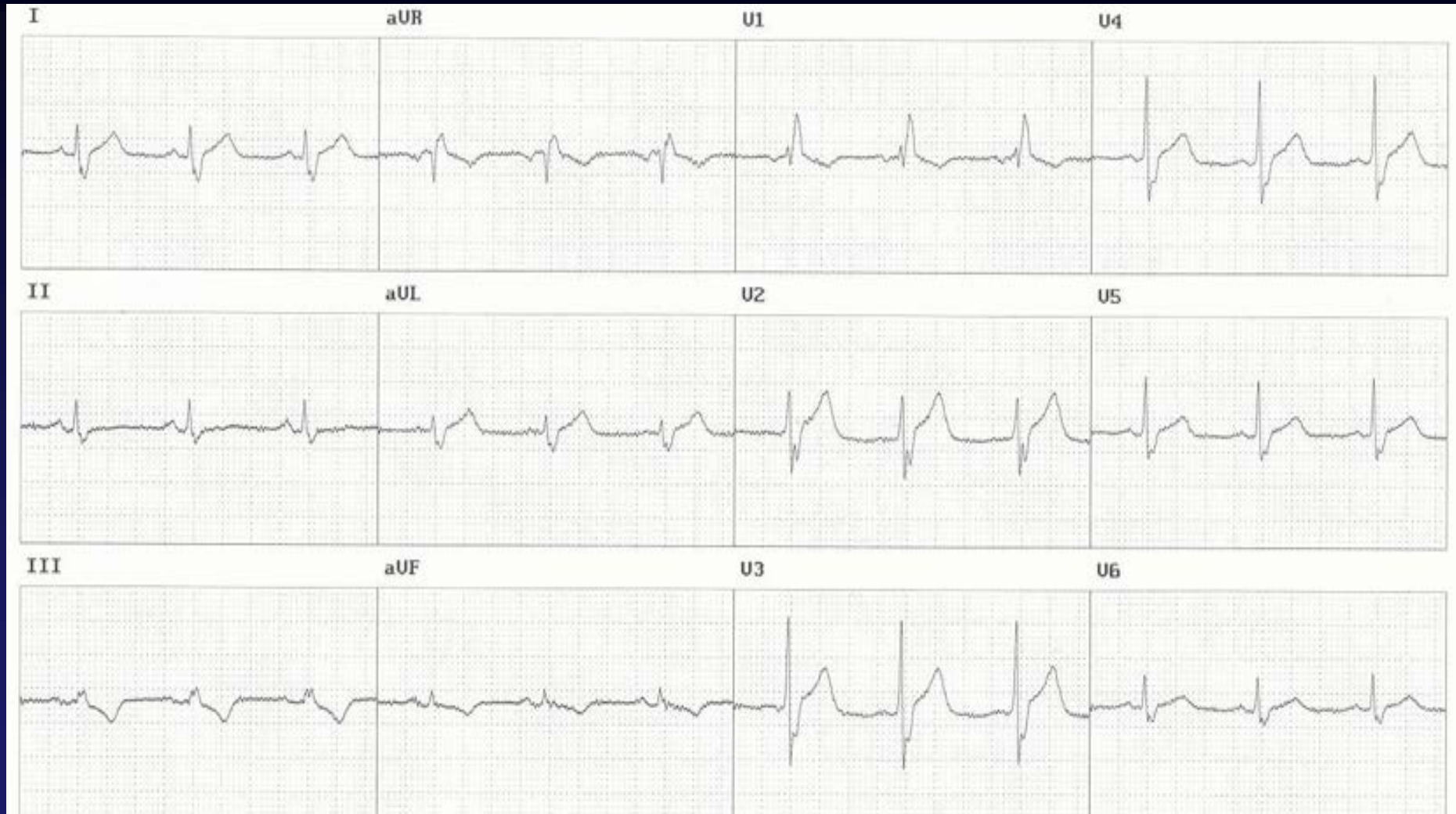
Left Posterior Hemiblock



RBBB, LAH (Bifascicular Block)



RBBB, LPH (Bifascicular Block)





Myocardial Ischemia

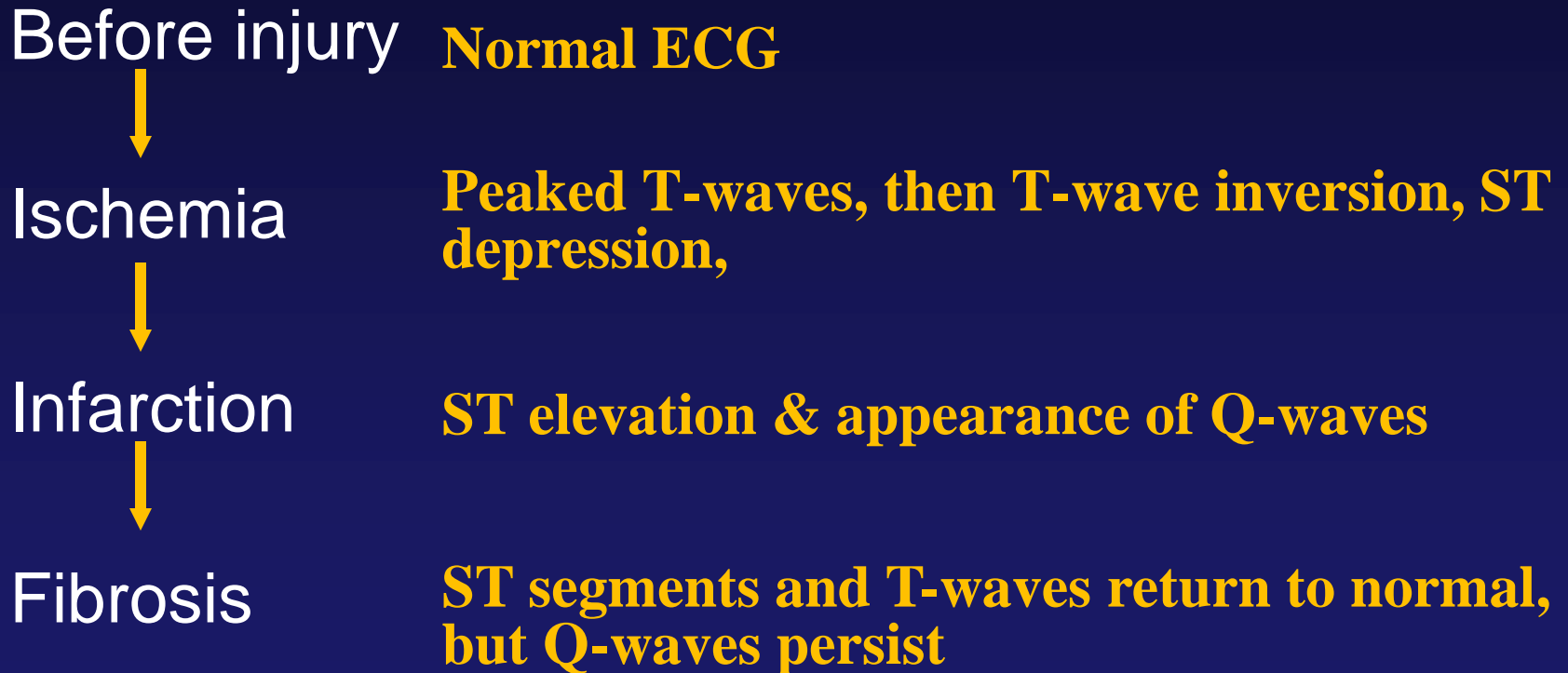
- ECG is the cornerstone in the diagnosis of myocardial ischemia
- Findings depend on several factors:
 - Nature of the process, reversible vs. irreversible
 - Duration, acute vs. chronic
 - Extent, transmural vs. subendocardial
 - Localization, anterior vs. inferoposterior
 - Other underlying abnormalities

Evolution of a Myocardial Infarction

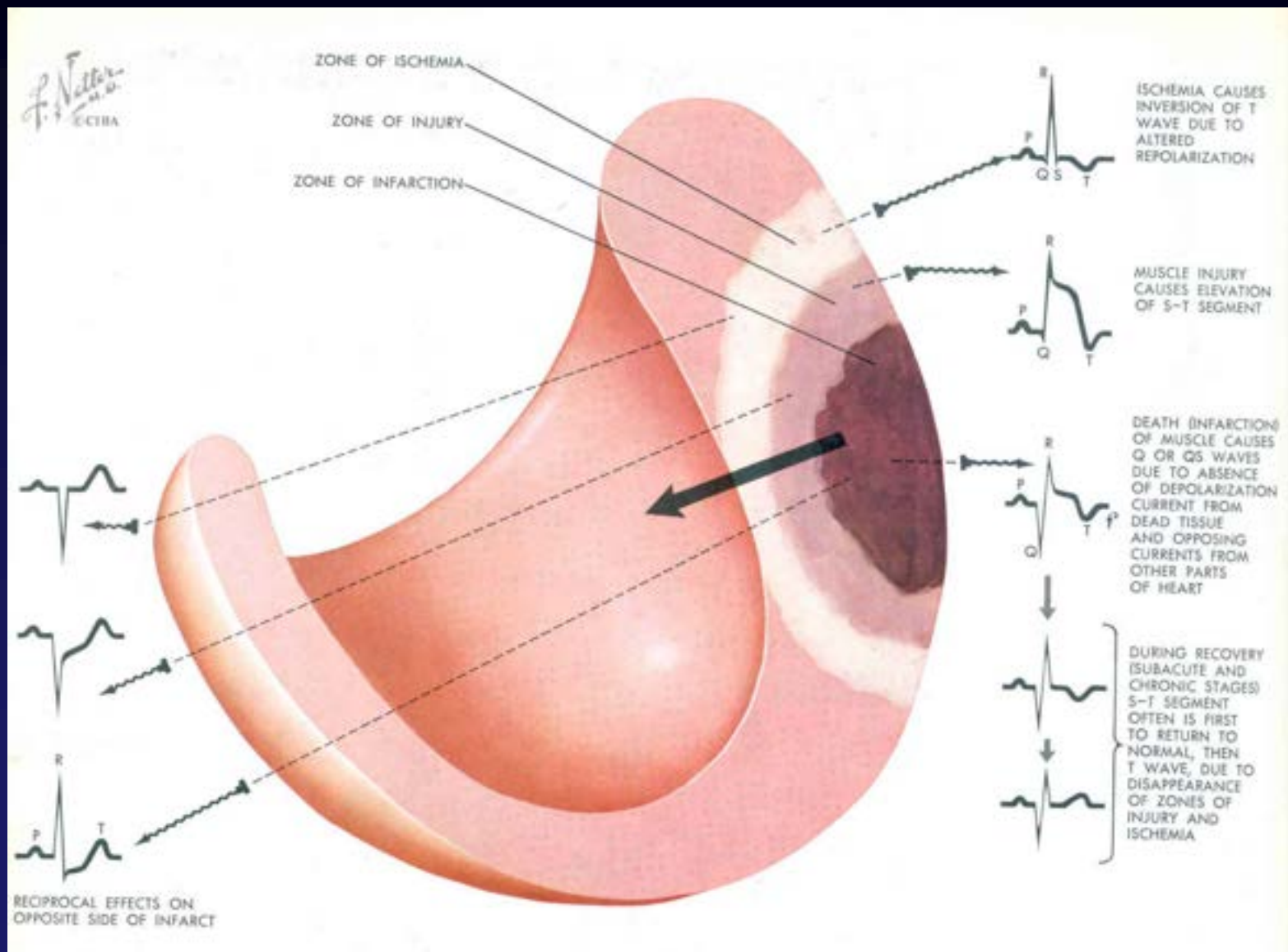
- When myocardial blood supply is abruptly reduced or cut off to a region of the heart, a sequence of injurious events occur beginning with **ischemia** (inadequate tissue perfusion), followed by **necrosis** (infarction), and eventual **fibrosis** (scarring) if the blood supply isn't restored in an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

ST Elevation Infarction

The ECG changes seen with a ST elevation infarction are:

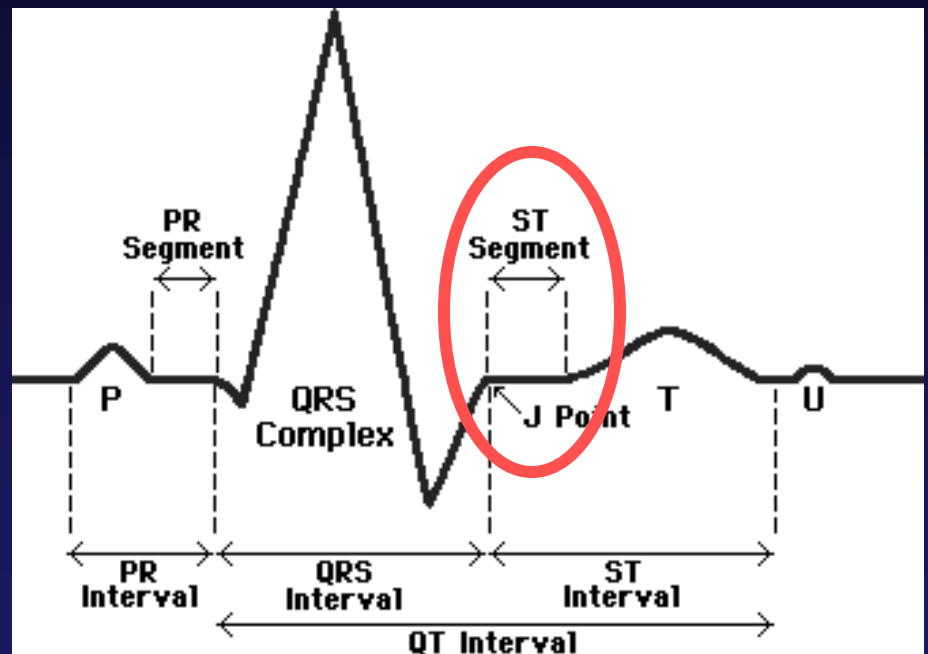


Acute Ischemia



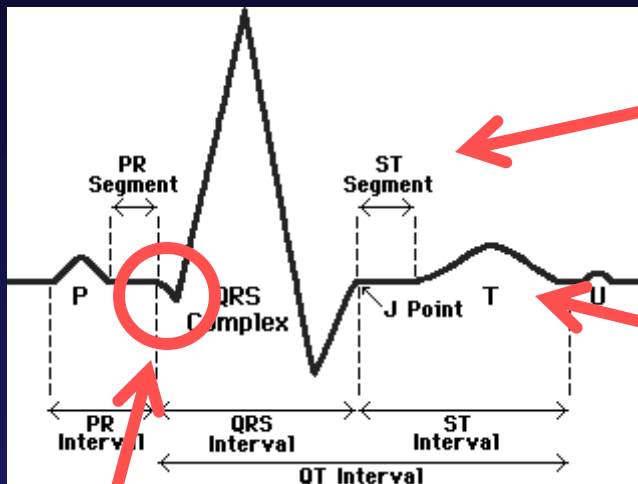
ST Elevation

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

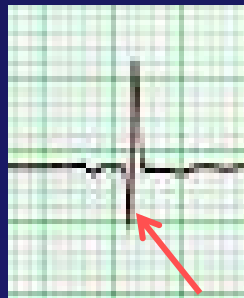


ECG Changes

Ways the ECG can change include:



Appearance of pathologic Q-waves

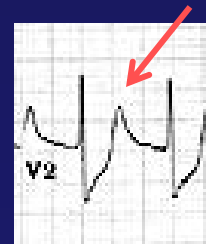


ST elevation & depression

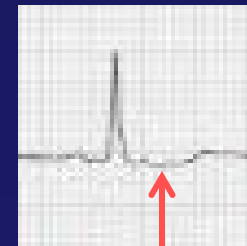


T-waves

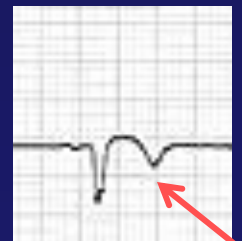
peaked



flattened

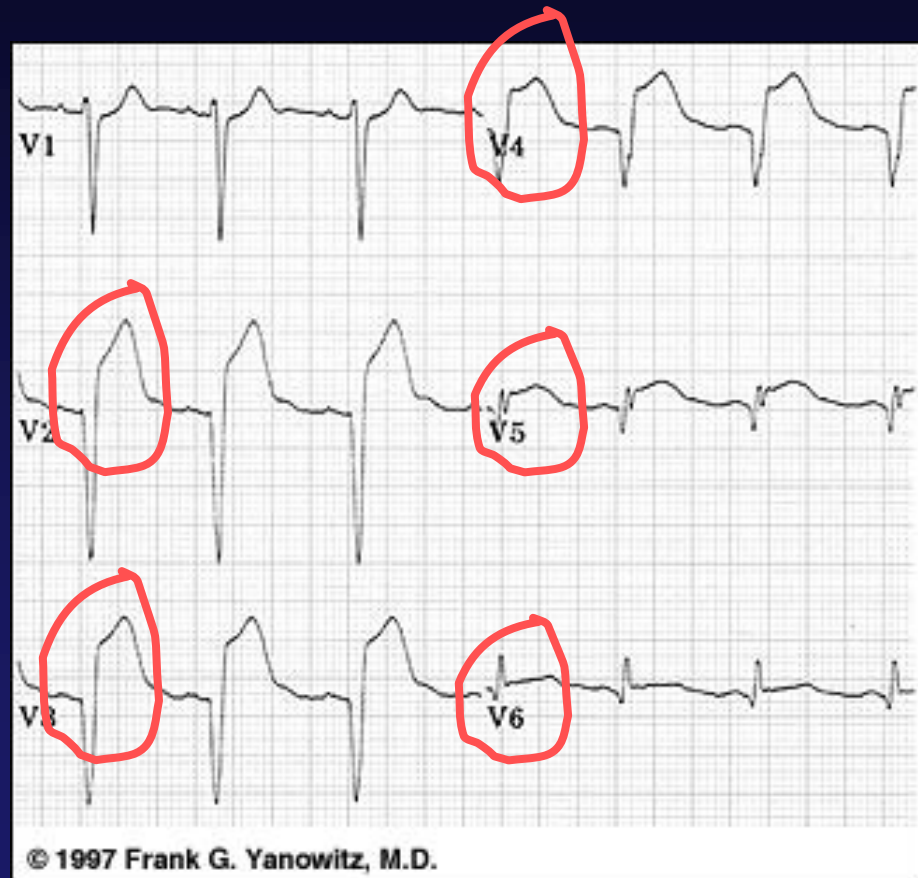


inverted



ST Elevation

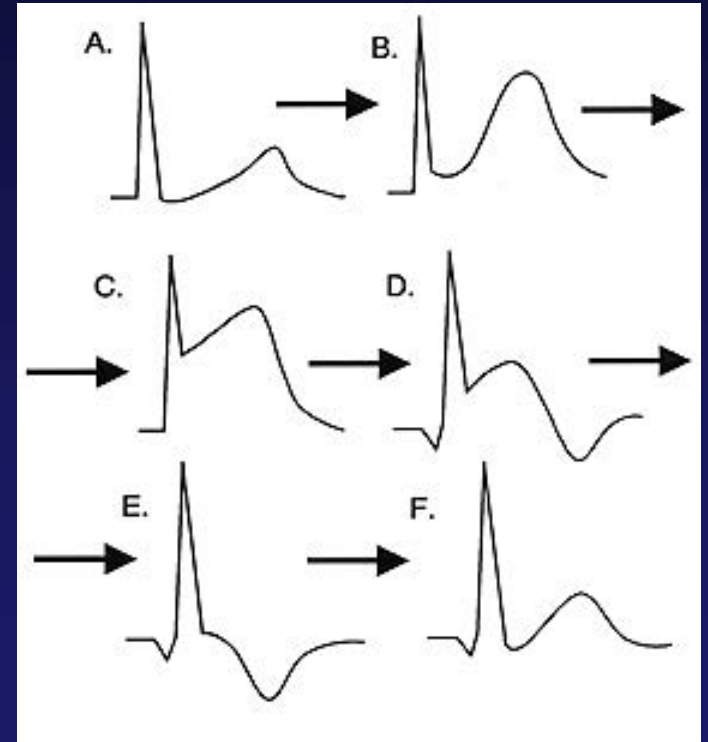
Elevation of the ST segment (greater than 1 small box) in 2 leads is consistent with a myocardial infarction.



ST Elevation Infarction

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



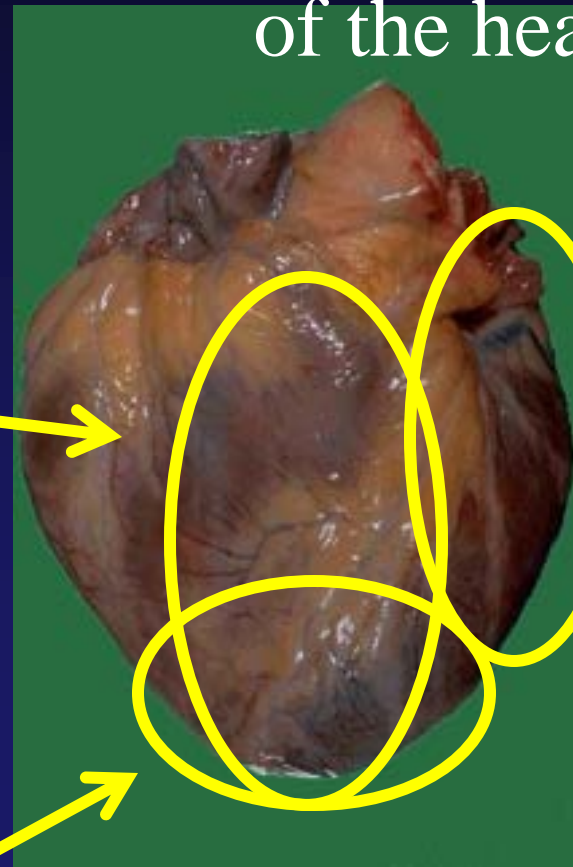
Views of the Heart

Some leads get a good view of the:

Anterior portion
of the heart

Inferior portion
of the heart

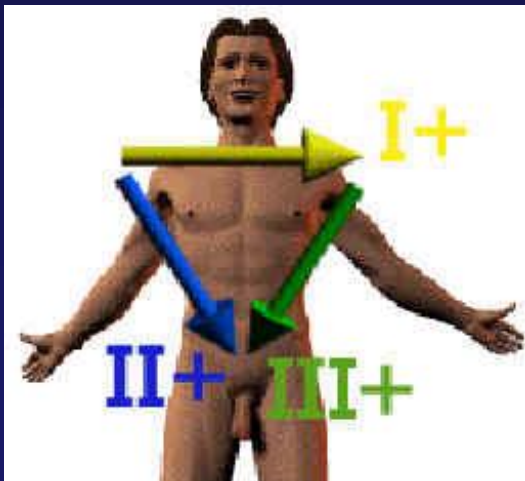
Lateral portion
of the heart



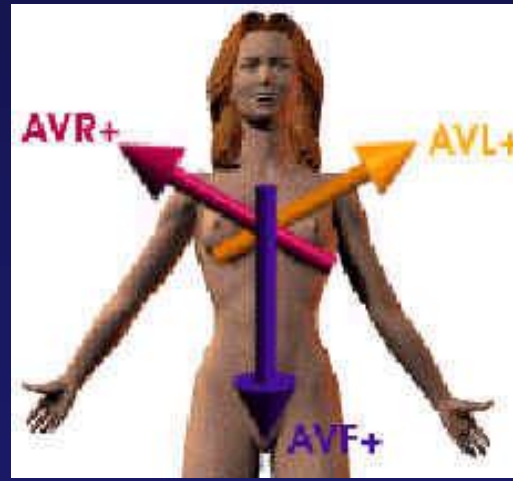
Anterior MI

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

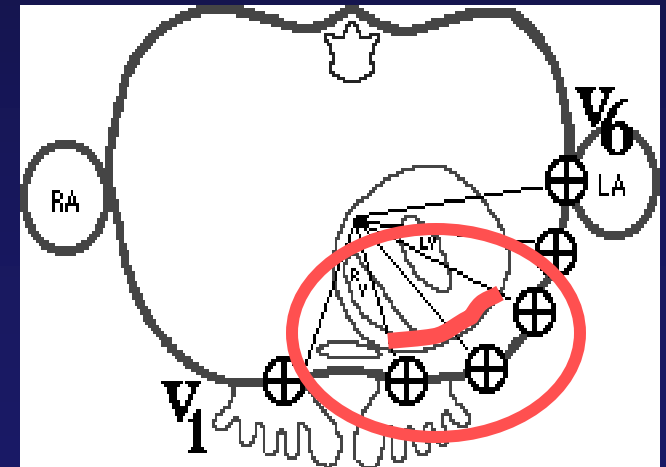
Limb Leads



Augmented Leads



Precordial Leads

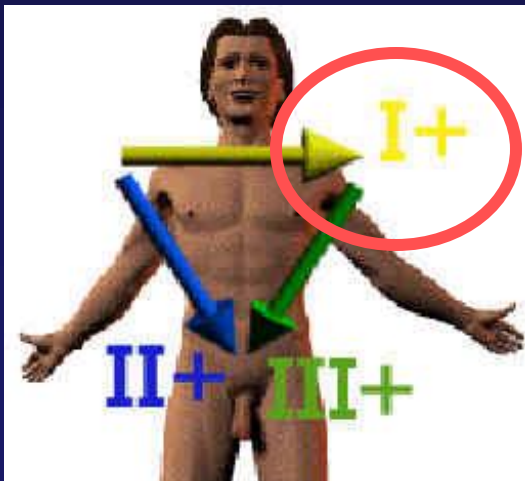


Lateral MI

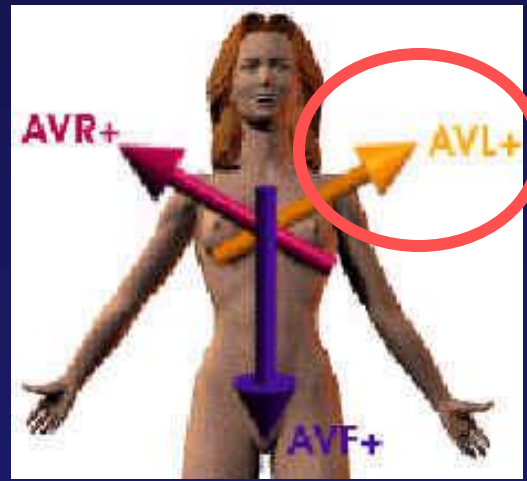
The lateral portion of the heart is best viewed by:

Leads I, aVL, and V₅-V₆

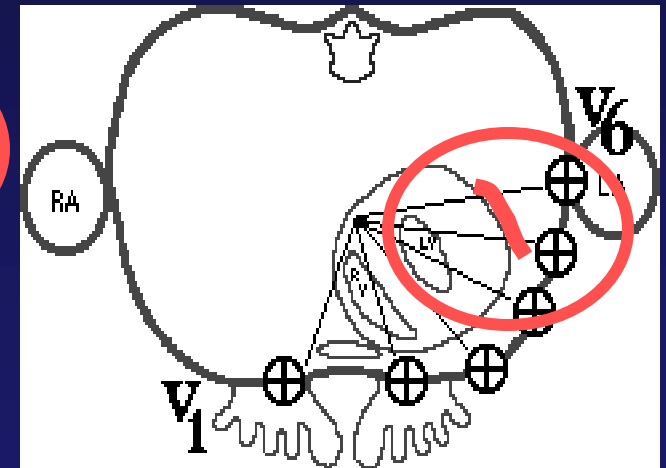
Limb Leads



Augmented Leads



Precordial Leads

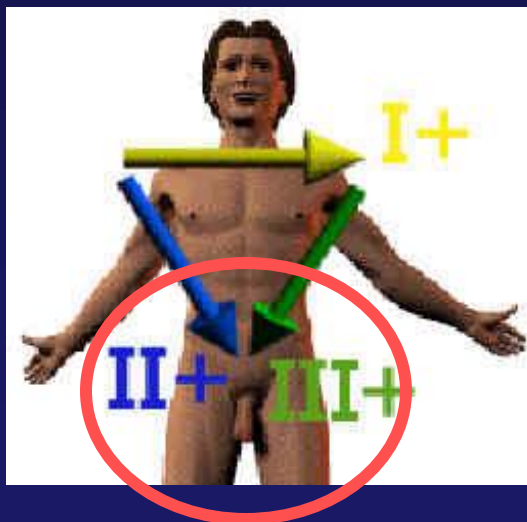


Inferior MI

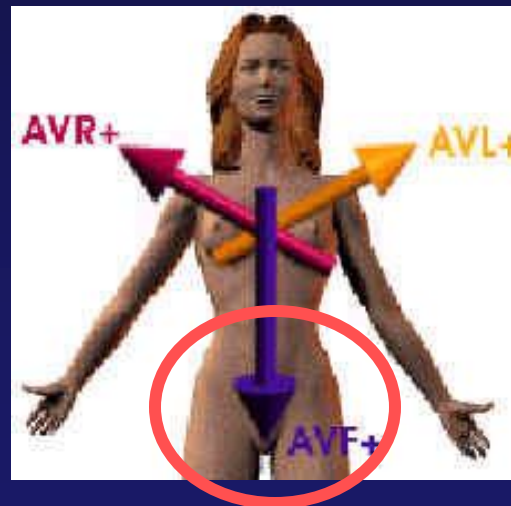
The inferior portion of the heart by:

Leads II, III and aVF

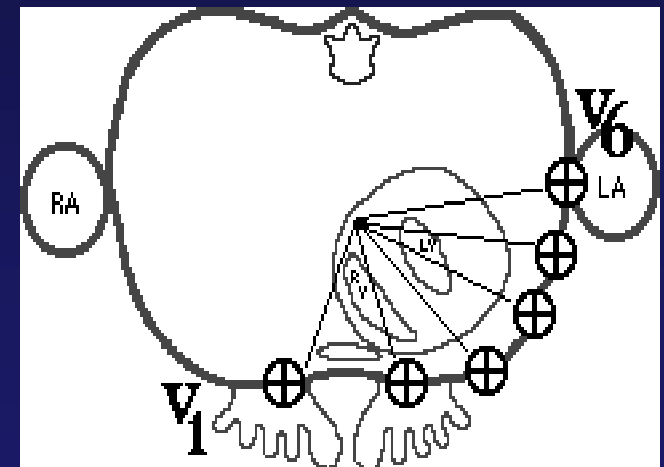
Limb Leads



Augmented Leads

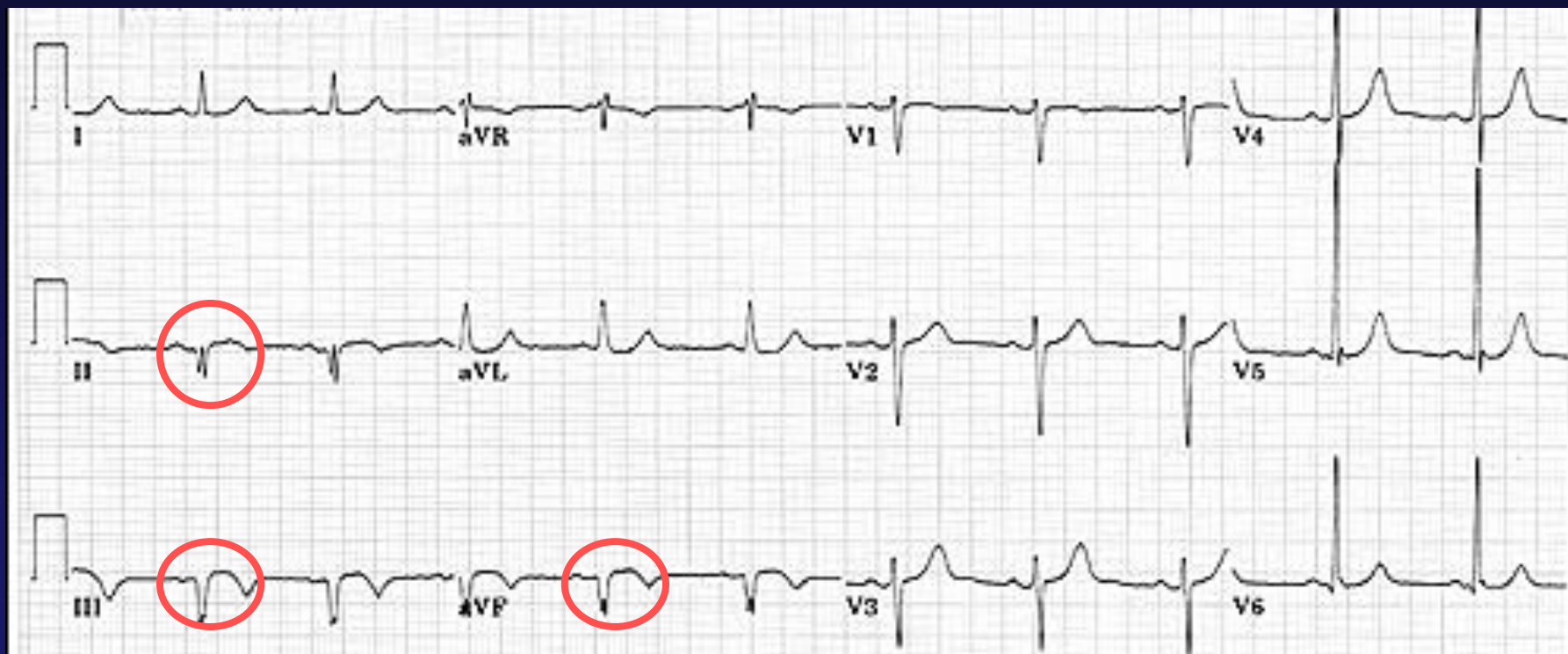


Precordial Leads



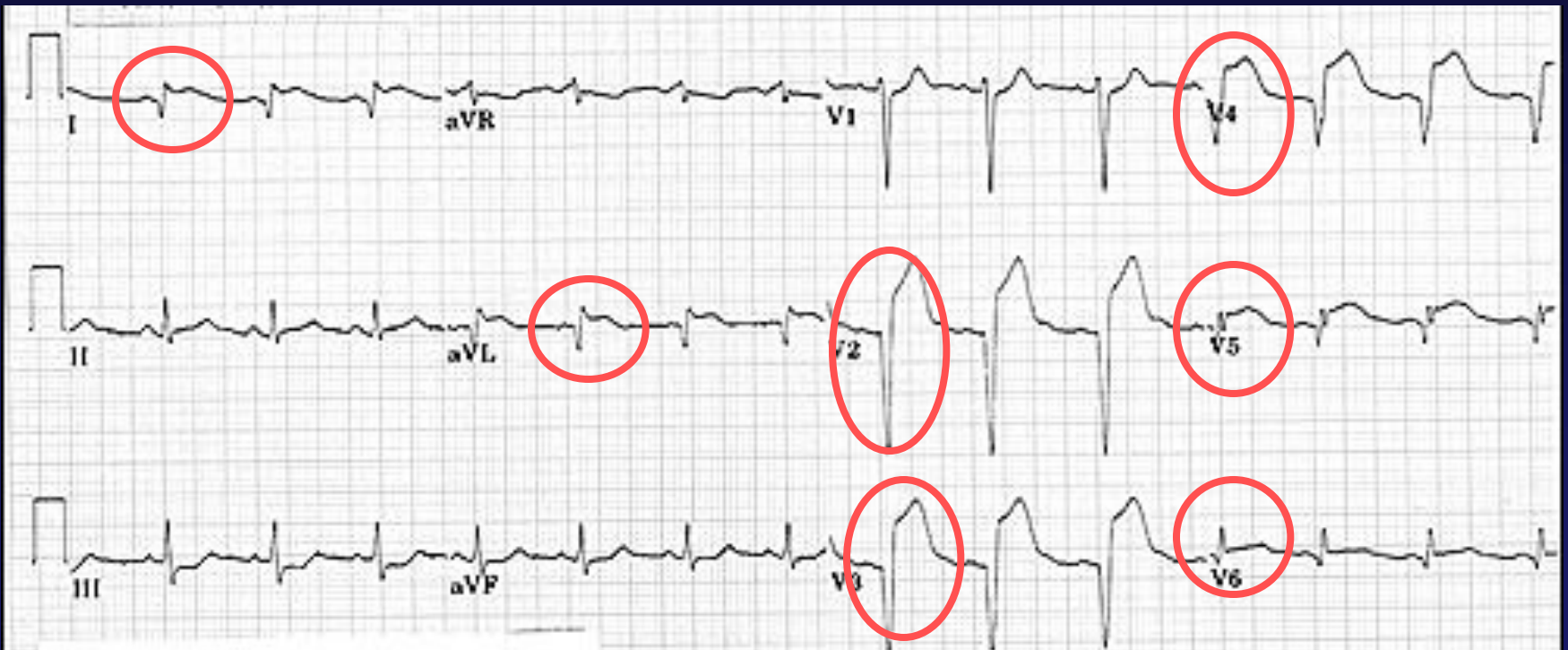
Inferior Wall MI

Note the ST elevation in leads II, III and aVF.

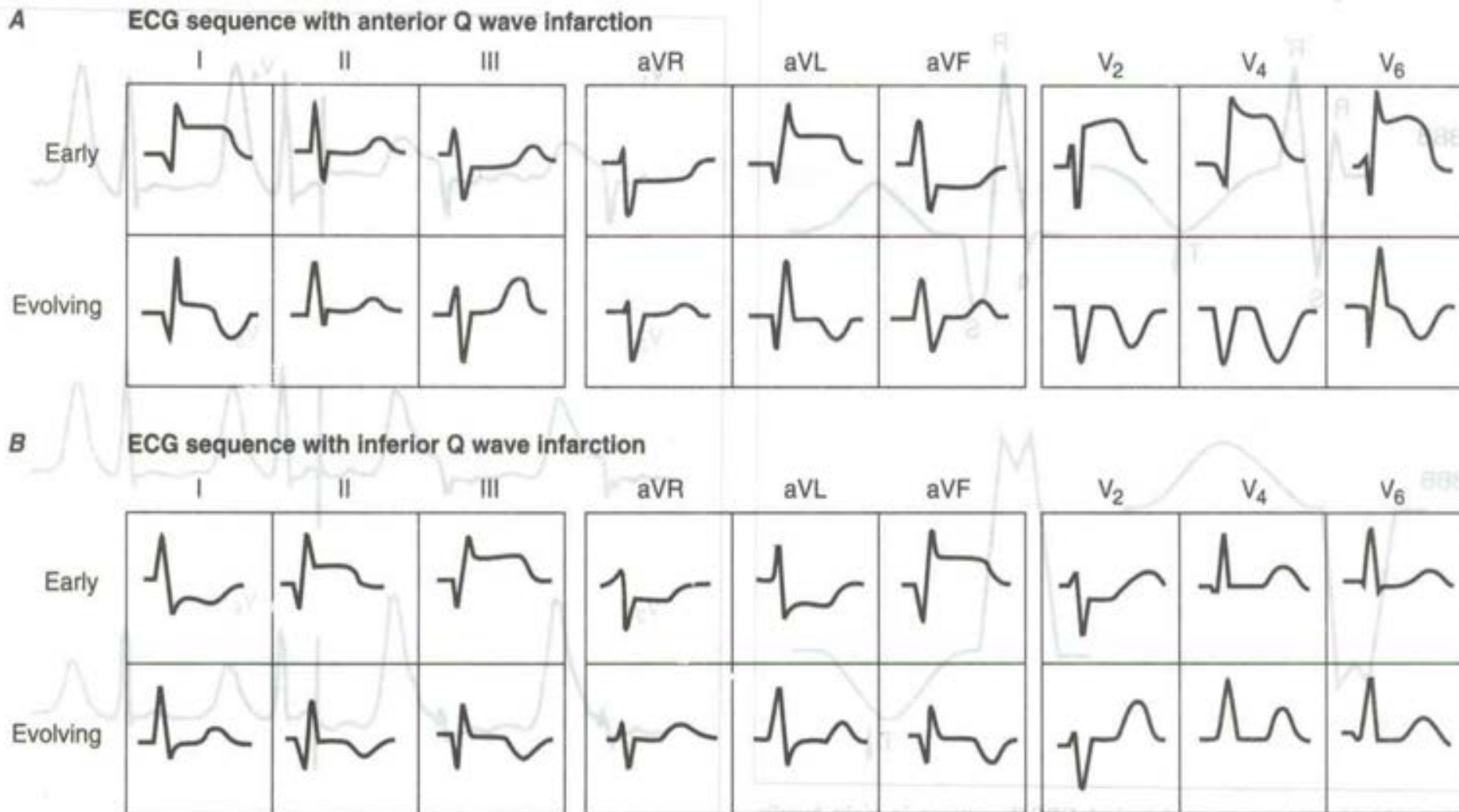


Anterolateral MI

This person's MI involves **both** the anterior wall (V₂-V₄) and the lateral wall (V₅-V₆, I, and aVL)!

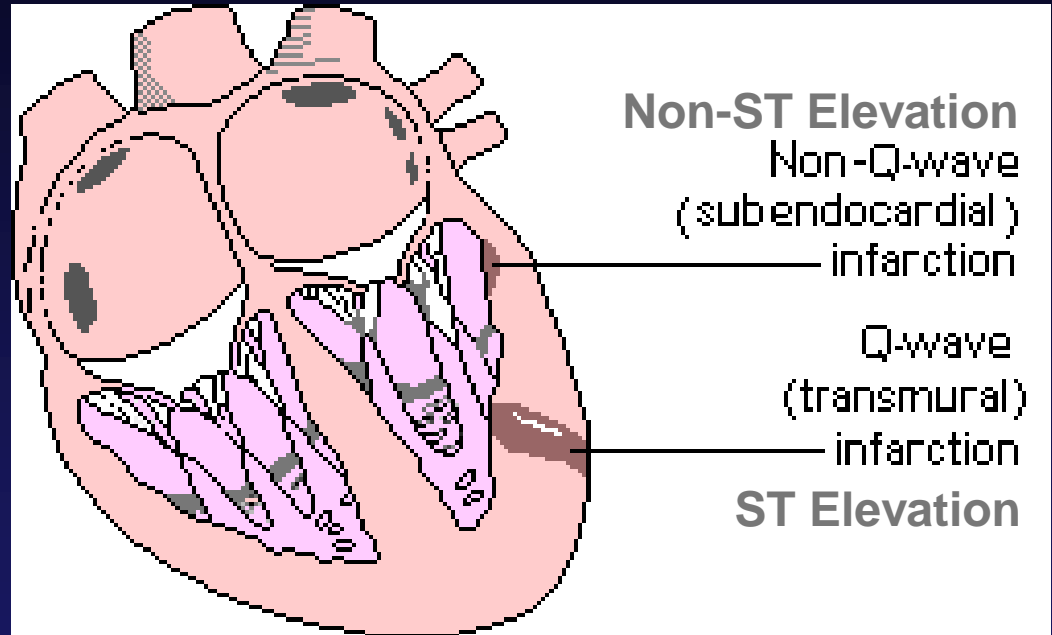


Myocardial Infarction



Non-ST Elevation 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)

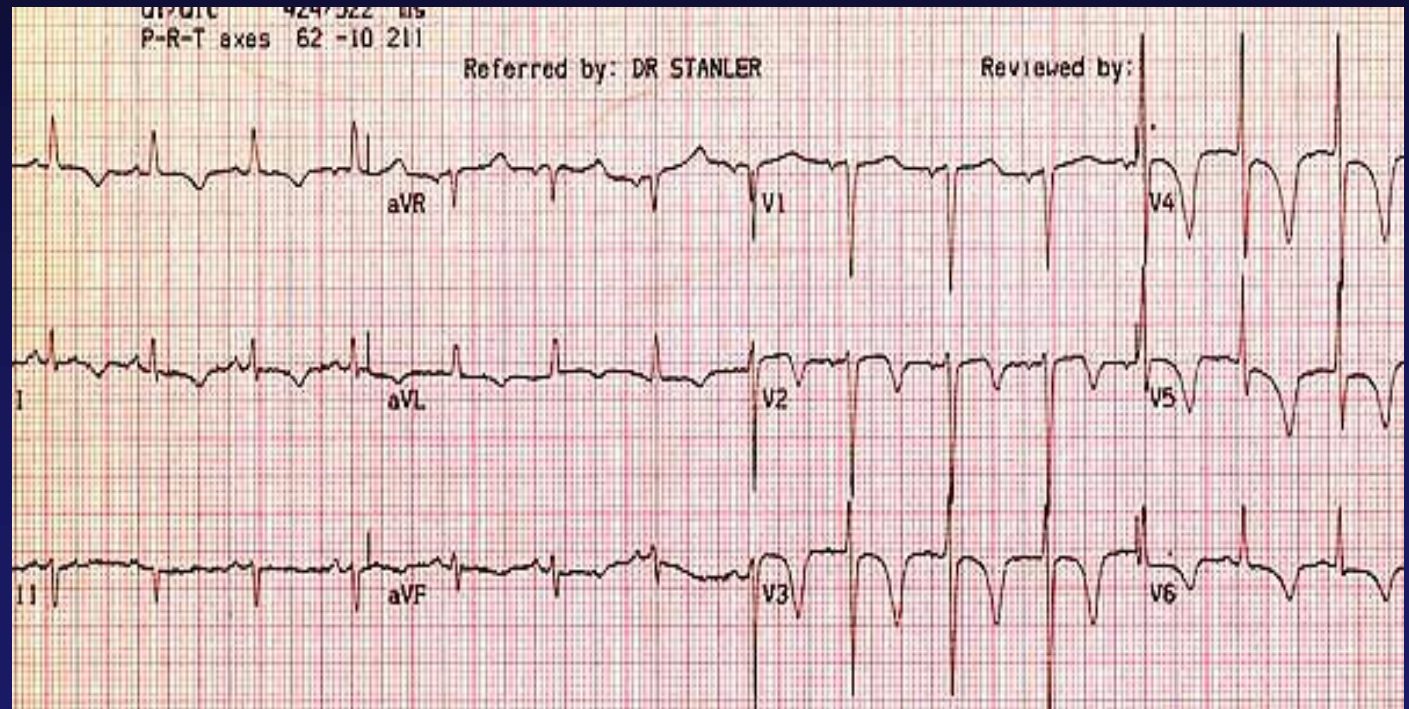
Non-ST Elevation Infarction

ECG of an evolving non-ST elevation MI:

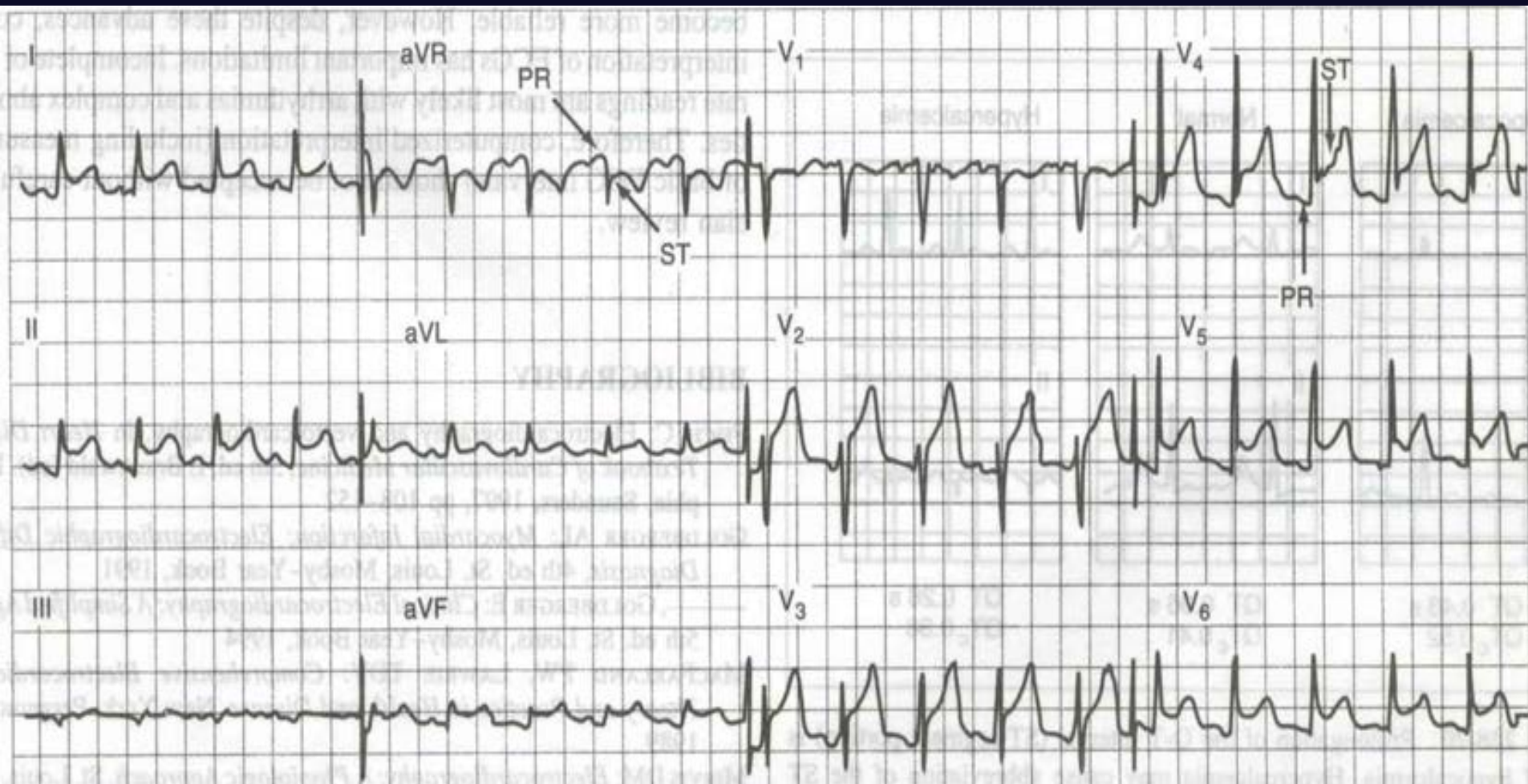
Note the ST depression and T-wave inversion in leads V_2 - V_6 .

Question:
What area of the heart is infarcting?

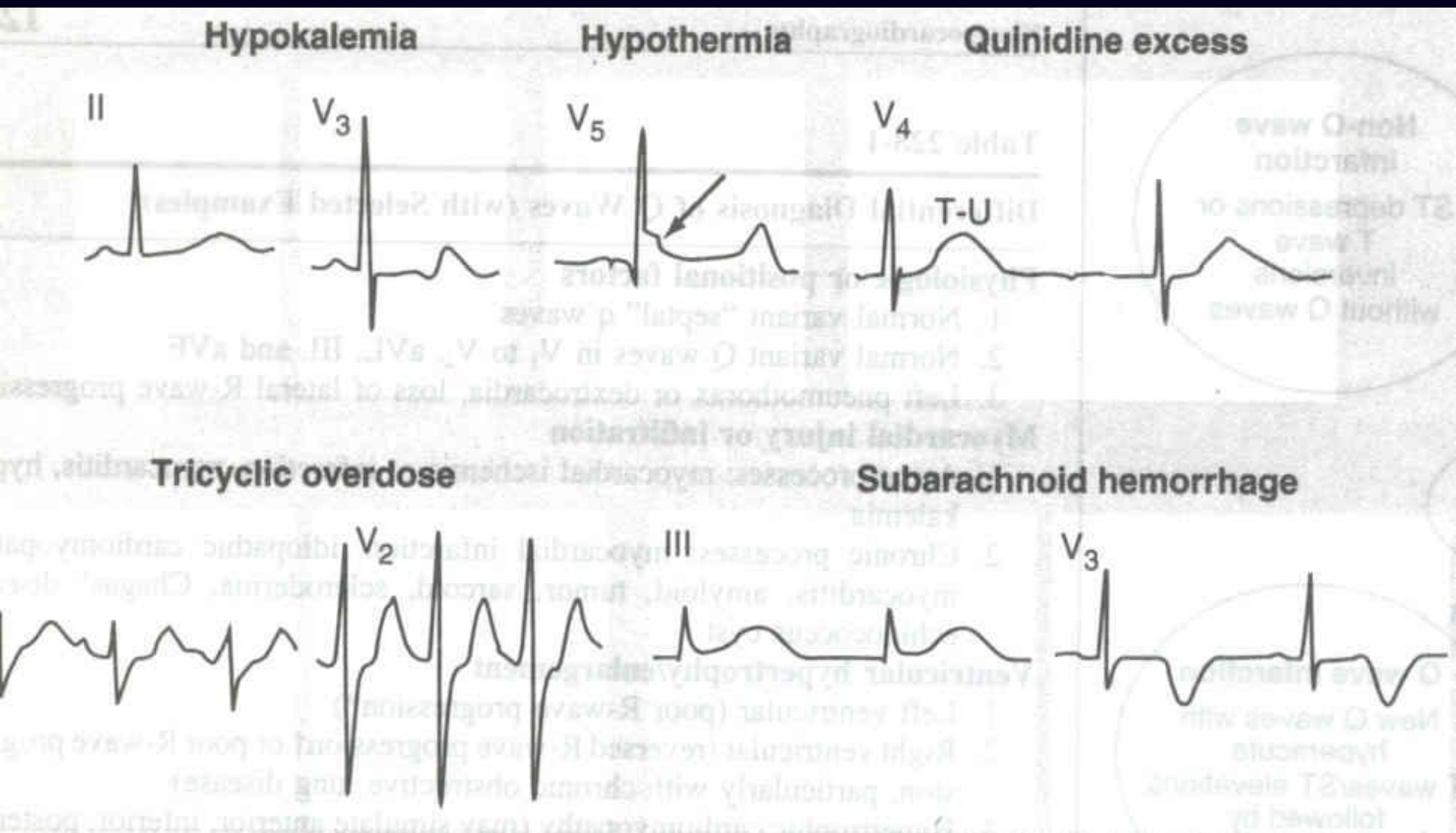
Cannot say!

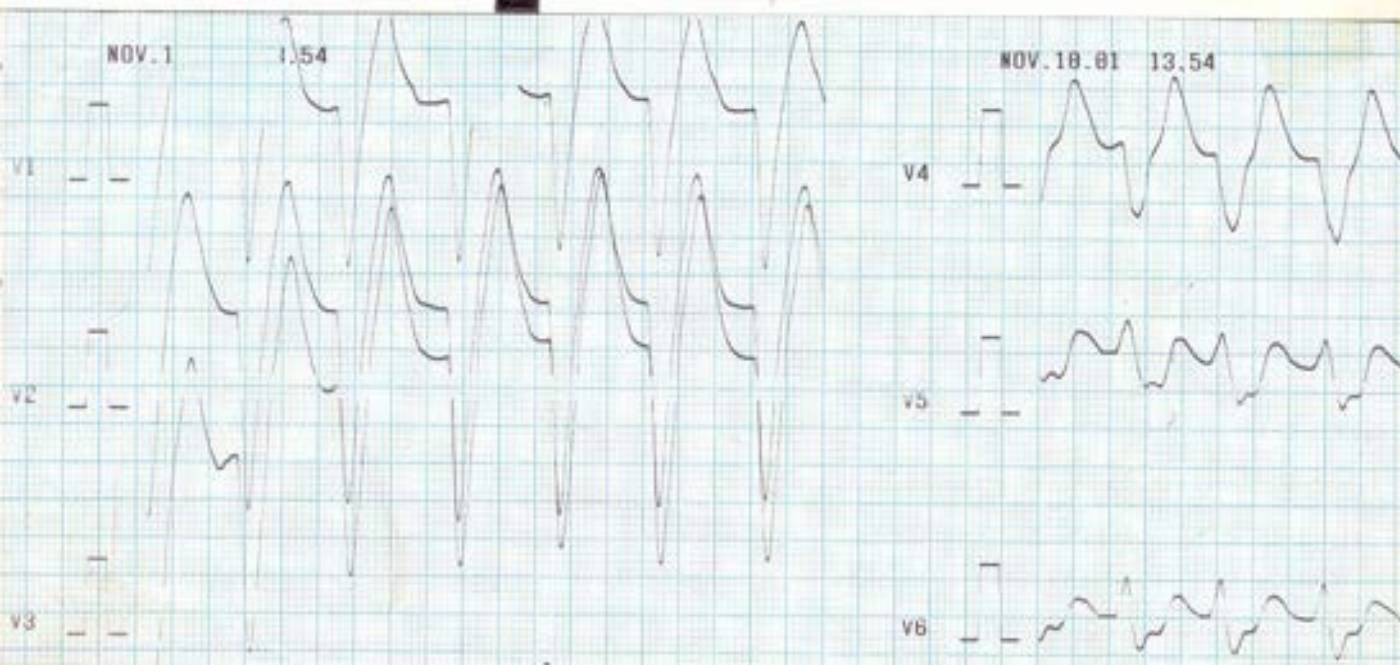
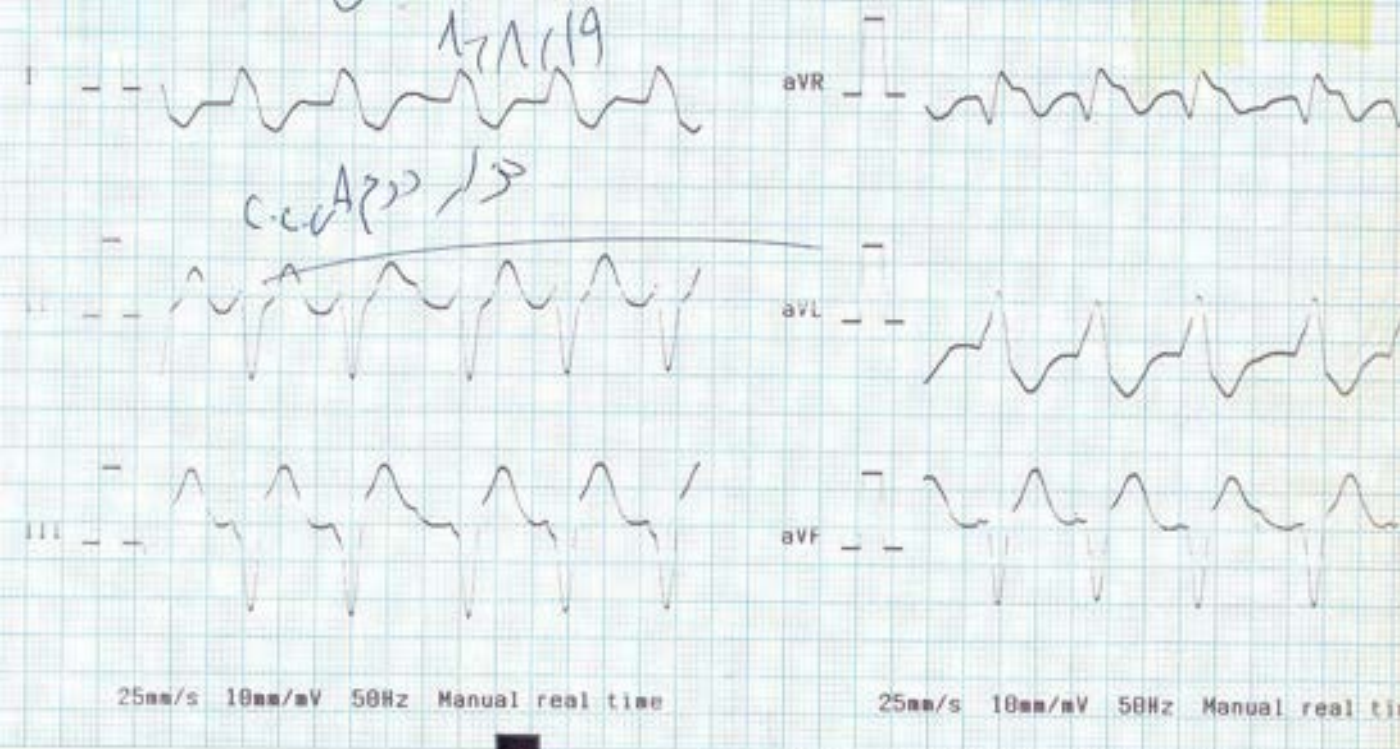


Acute Pericarditis



Metabolic Abnormalities

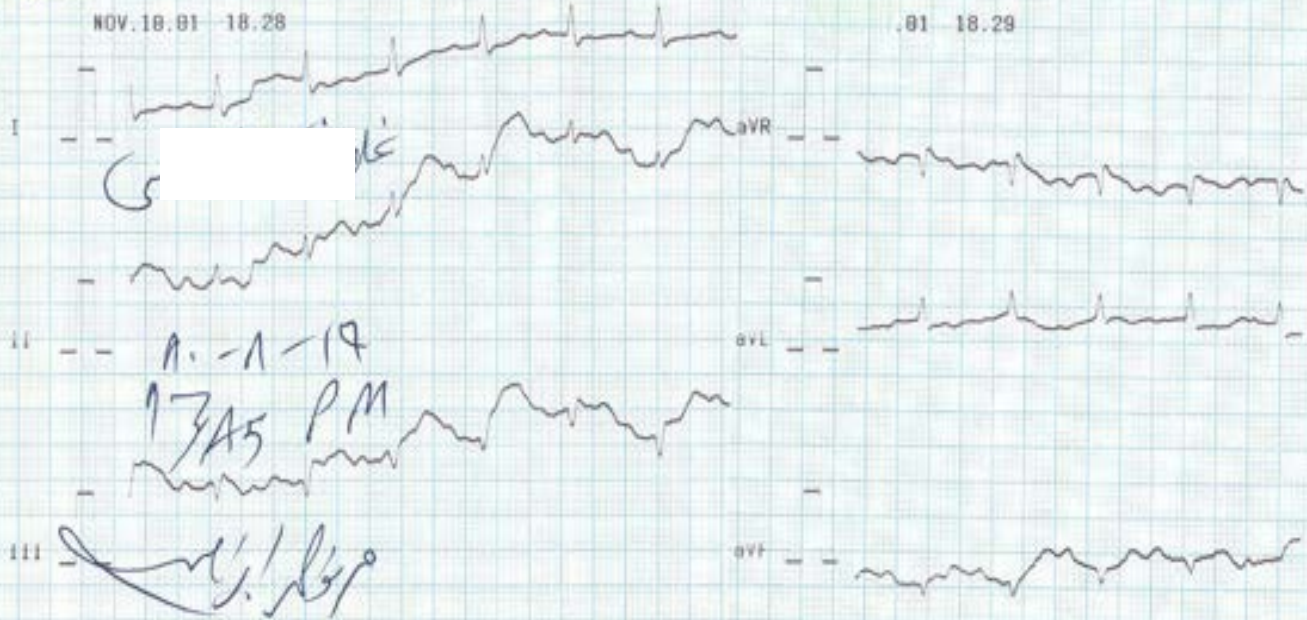




Hyper-
kalemia
K 6.9

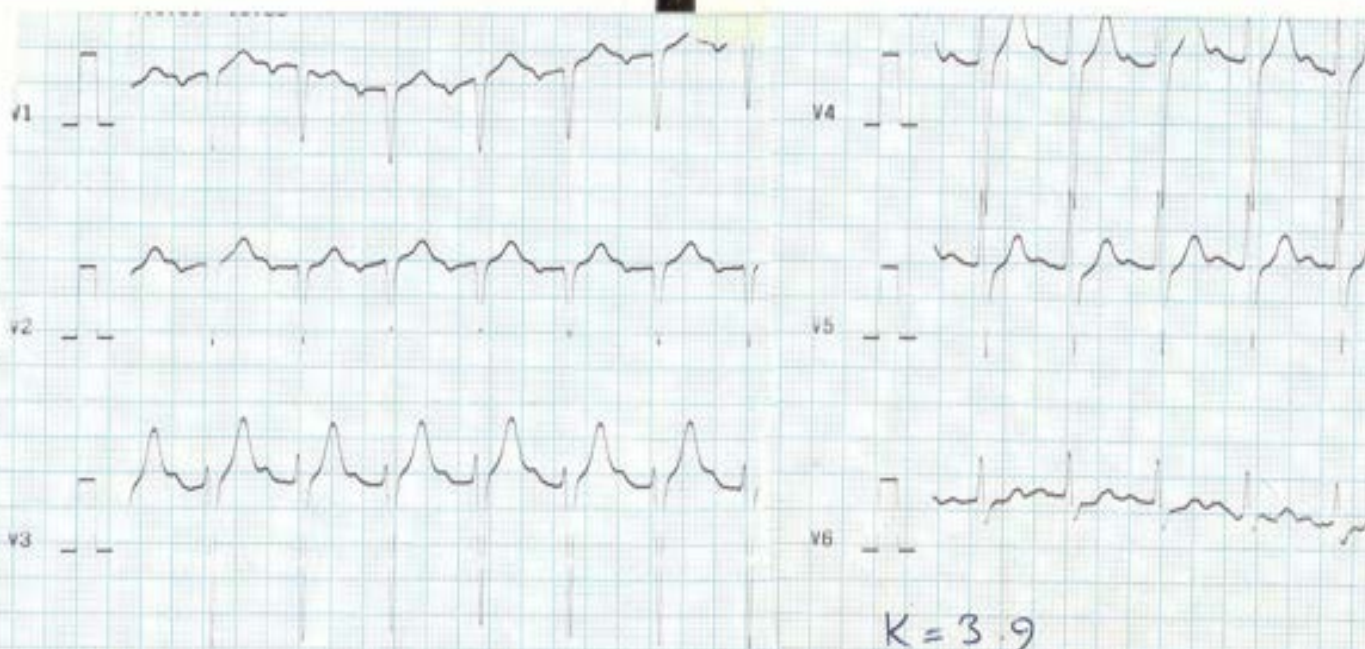
NOV. 18. 01 18.28

.01 18.29



25mm/s 10mm/mV 50Hz Manual real time

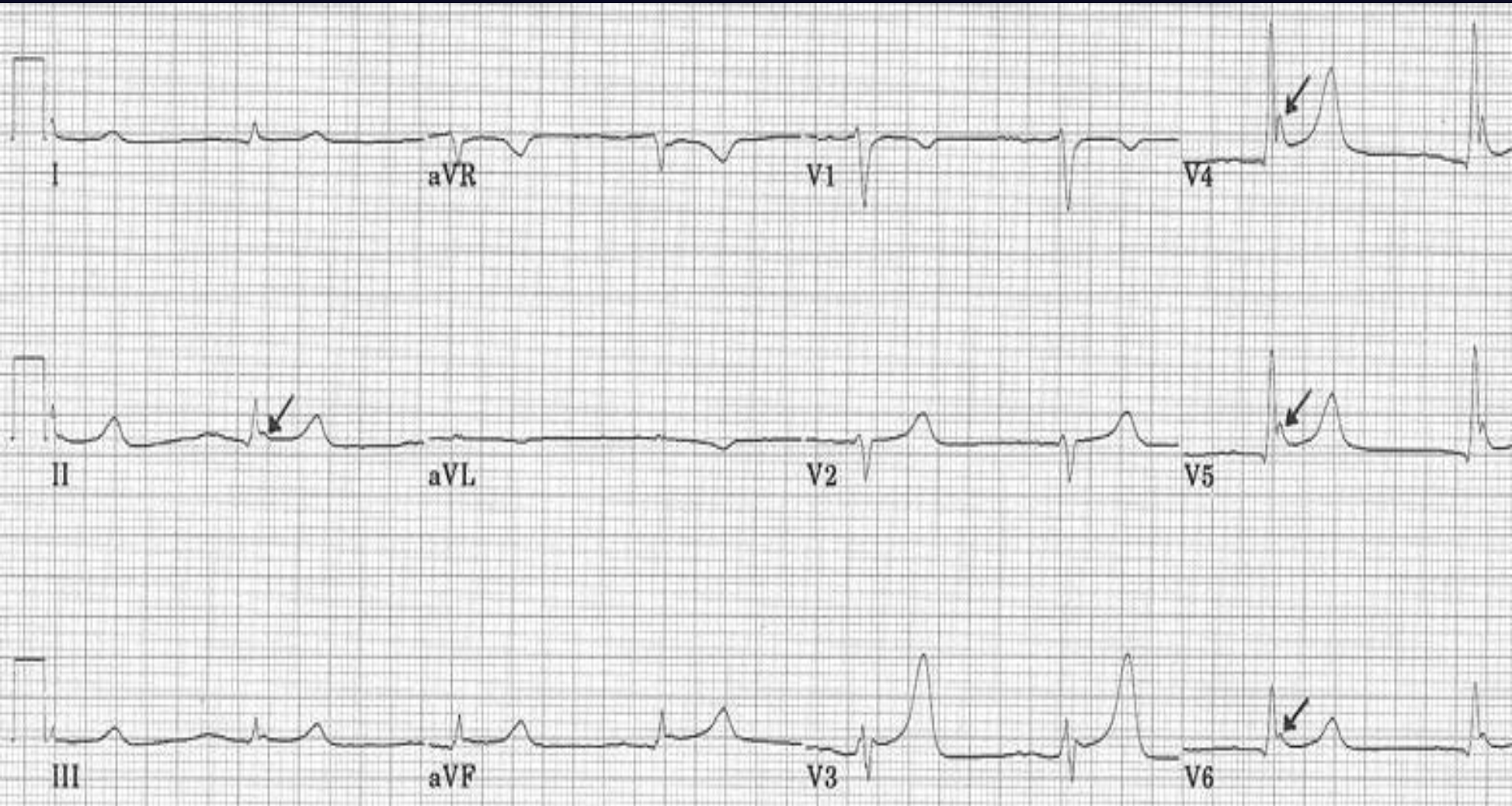
25mm/s 10mm/mV 50Hz Manual real time



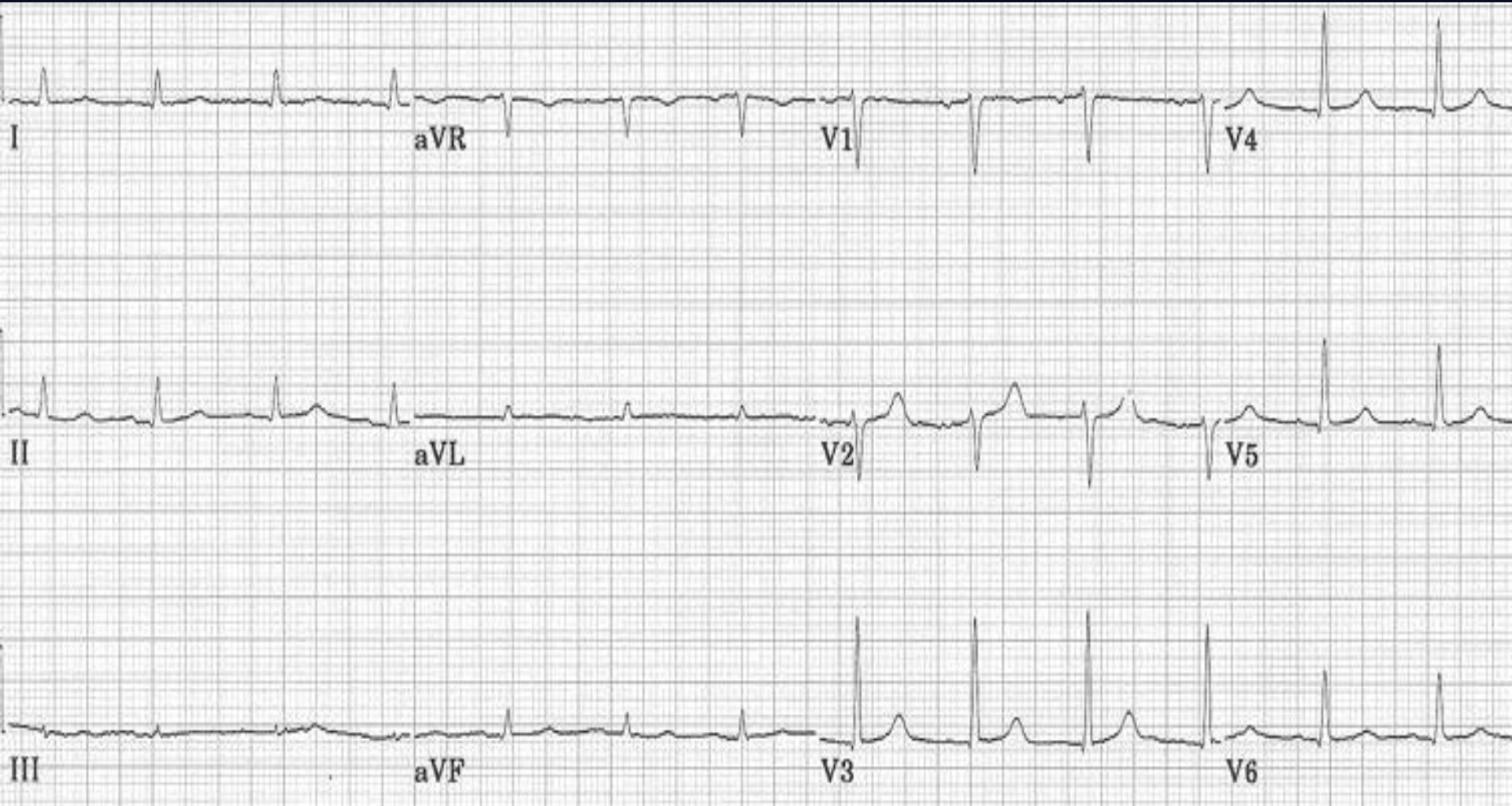
Same
patient

K 3.9

Hypothermia, Osborn Wave

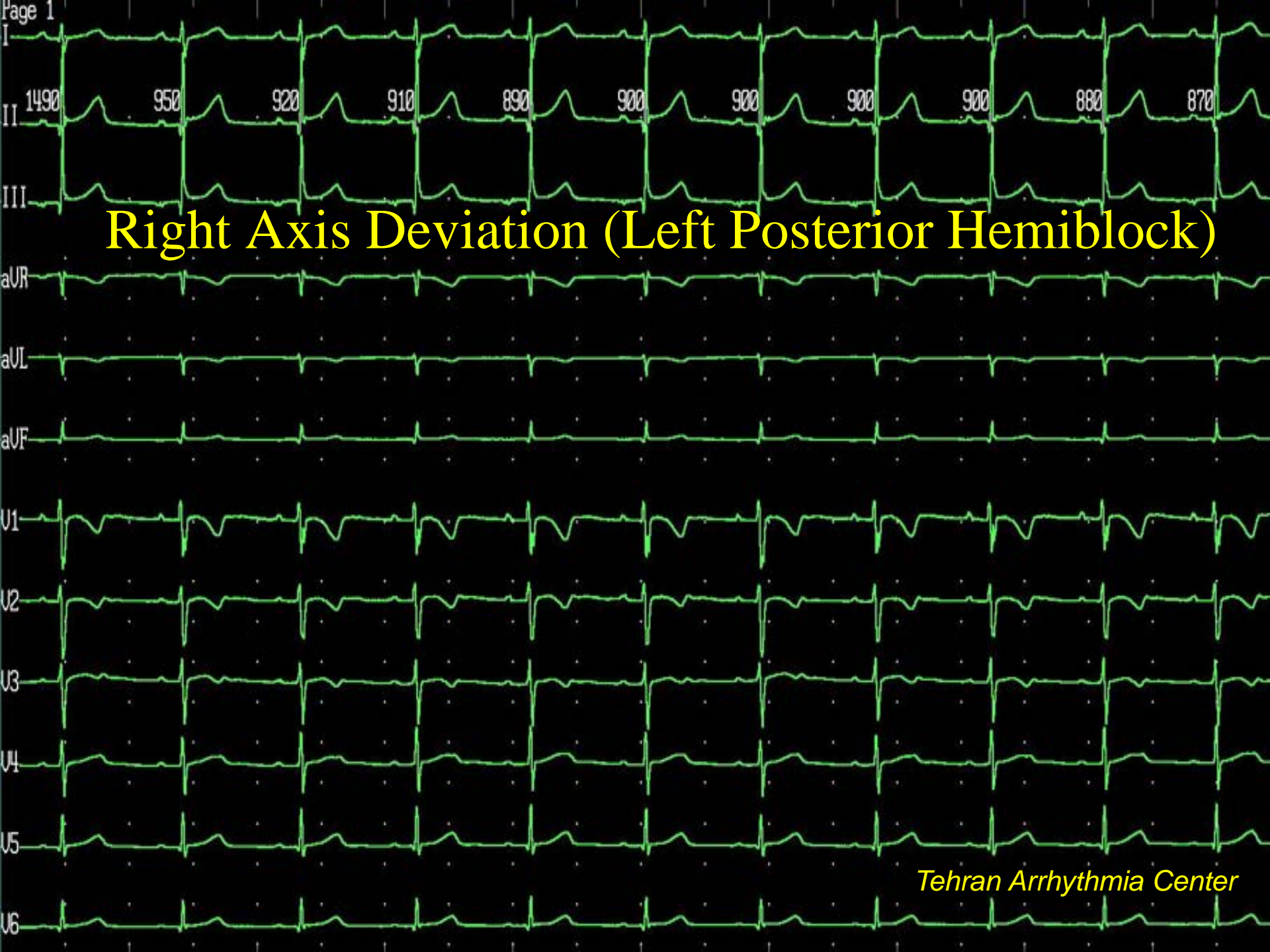


Hypothermia, Corrected



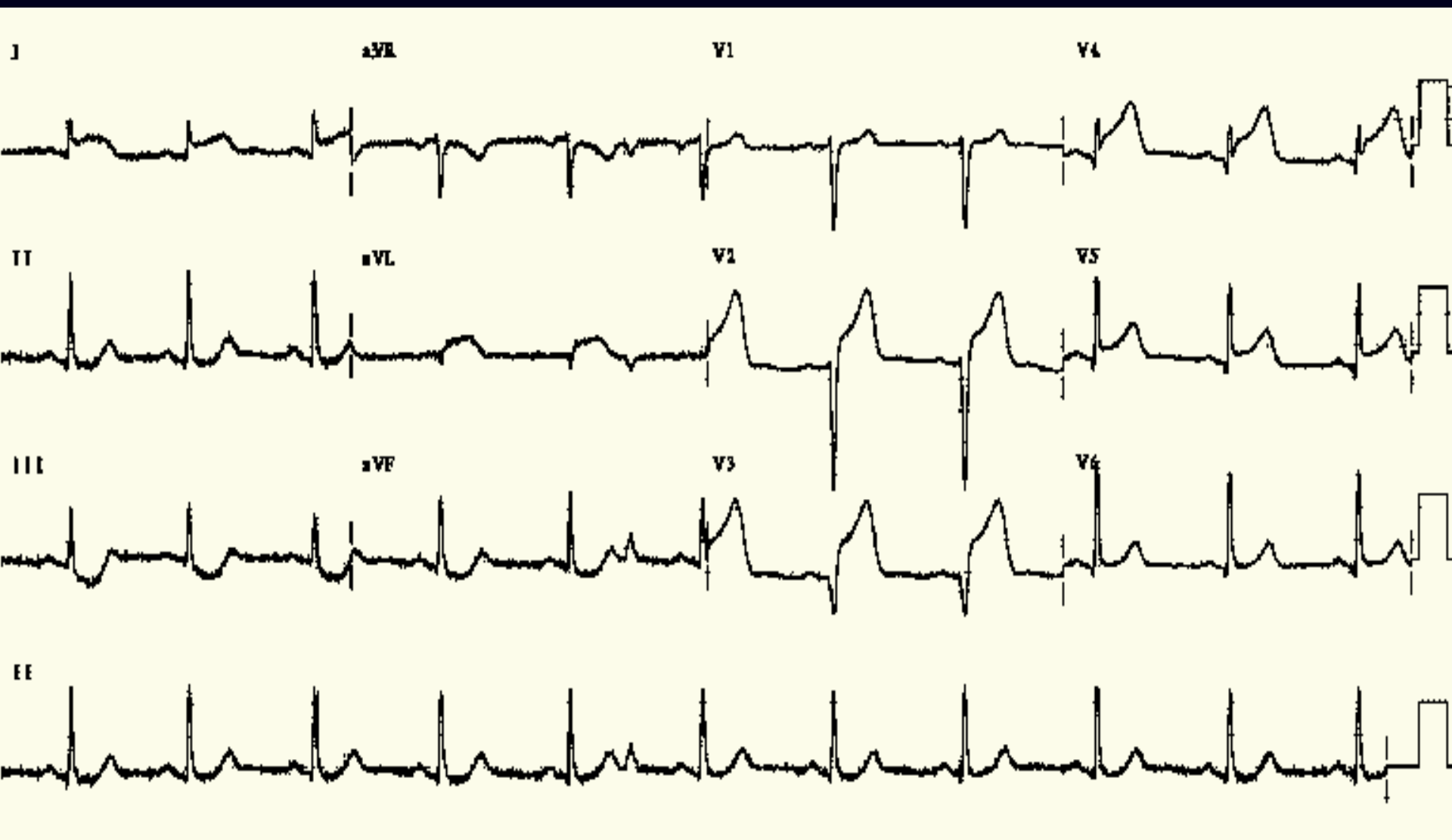


2003 5 29

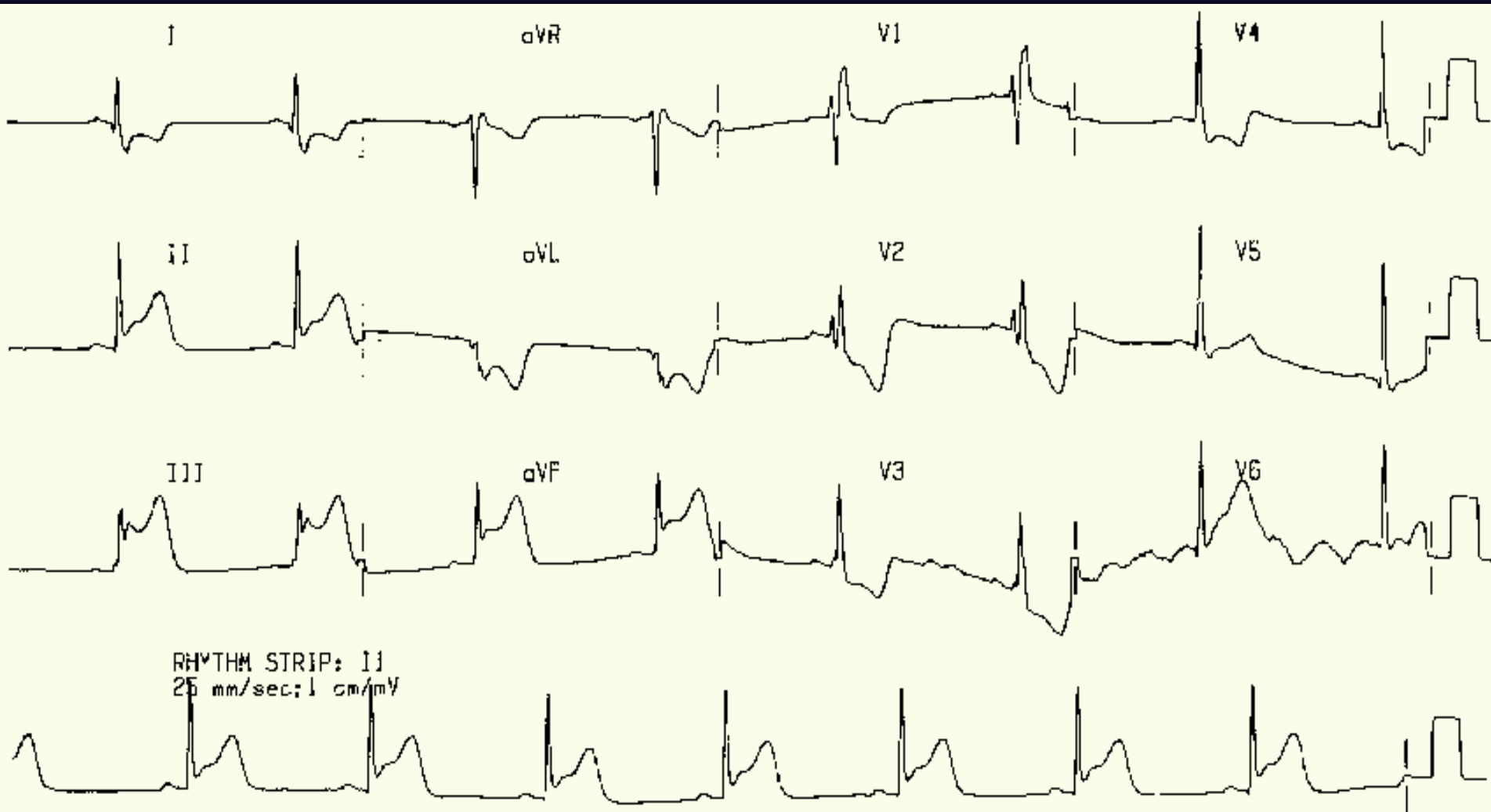


Right Axis Deviation (Left Posterior Hemiblock)

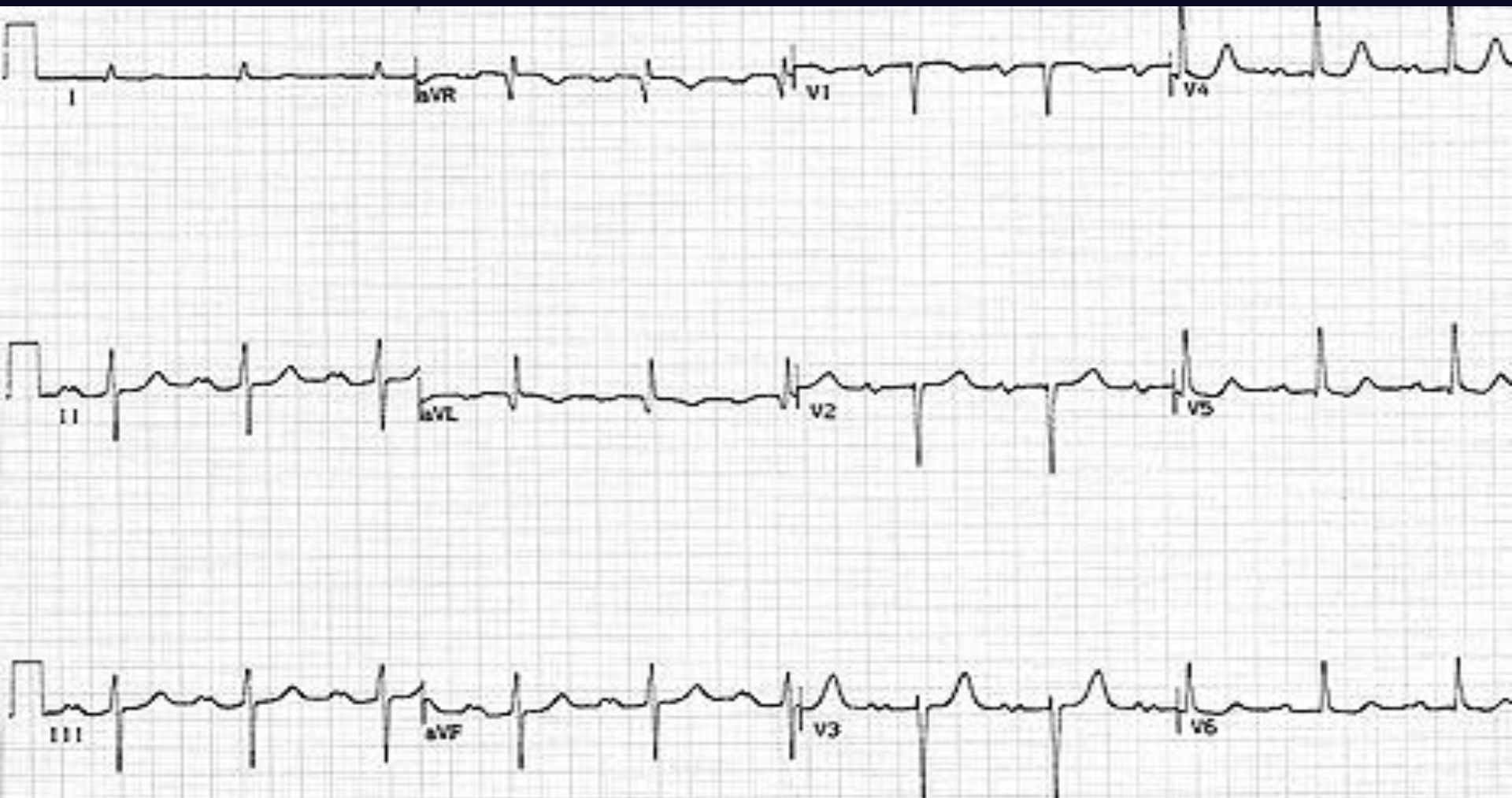
Anterior MI



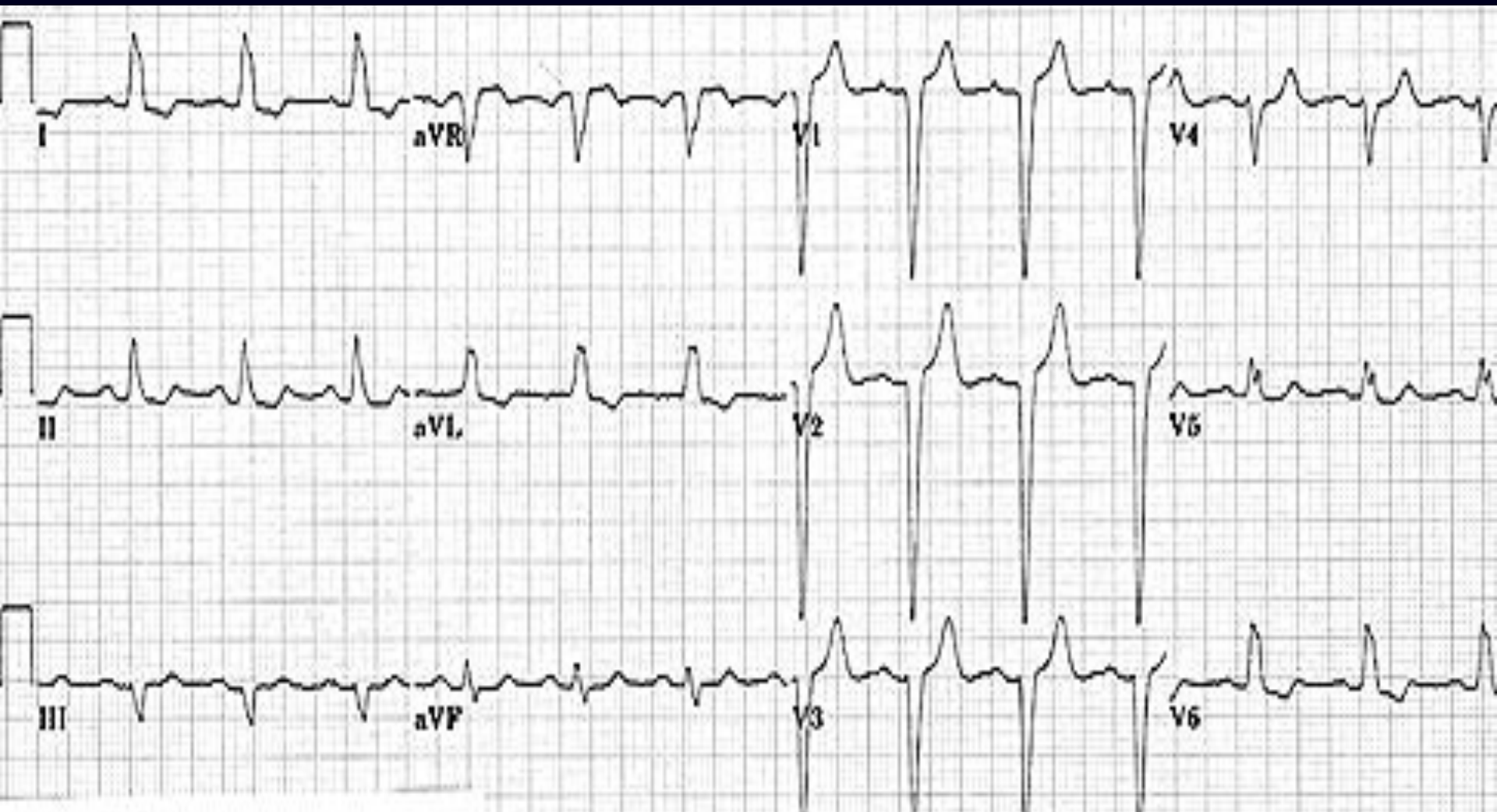
RBBB and Inferior MI



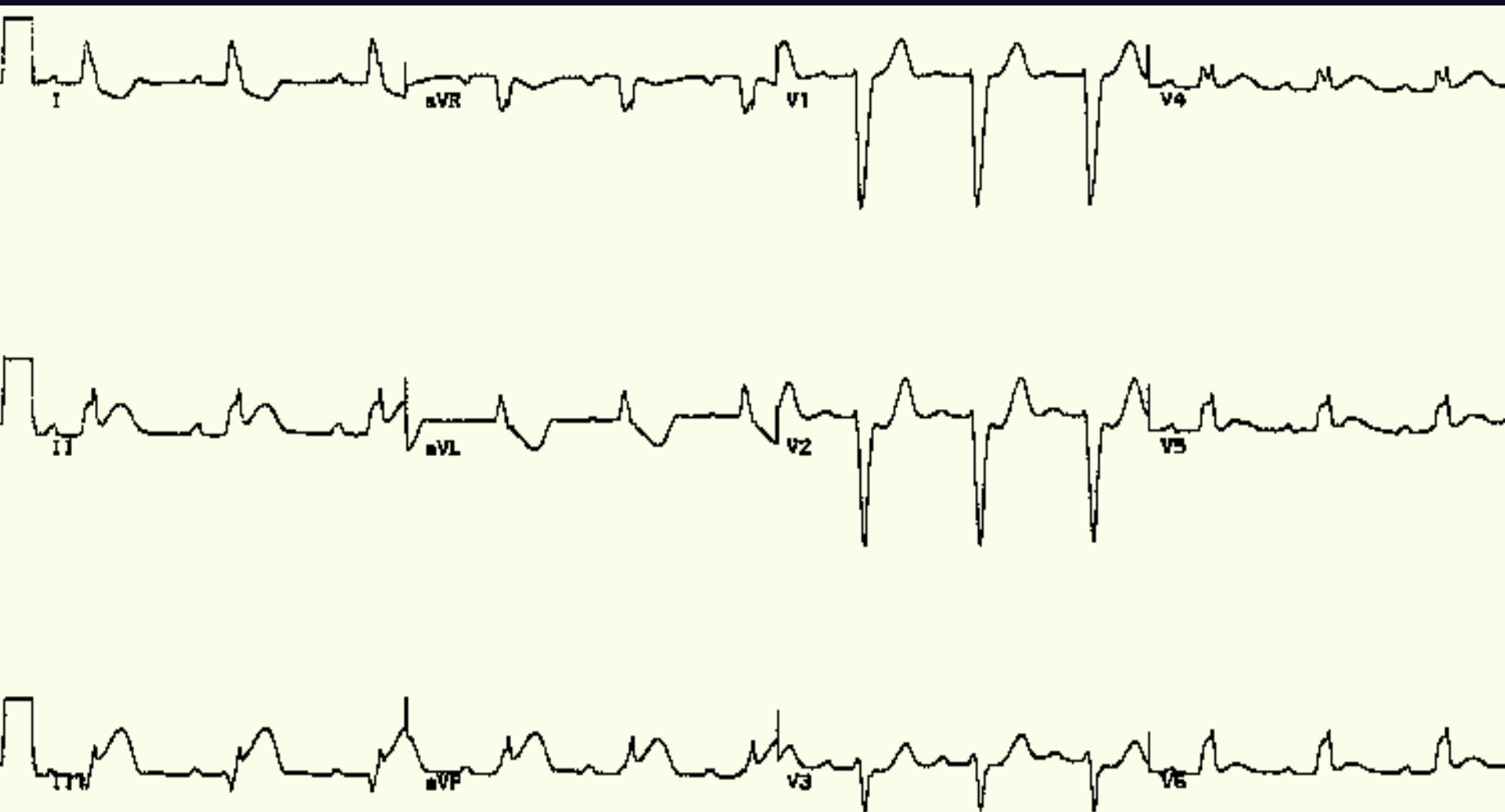
LA Enlargement and Prolonged PR Interval



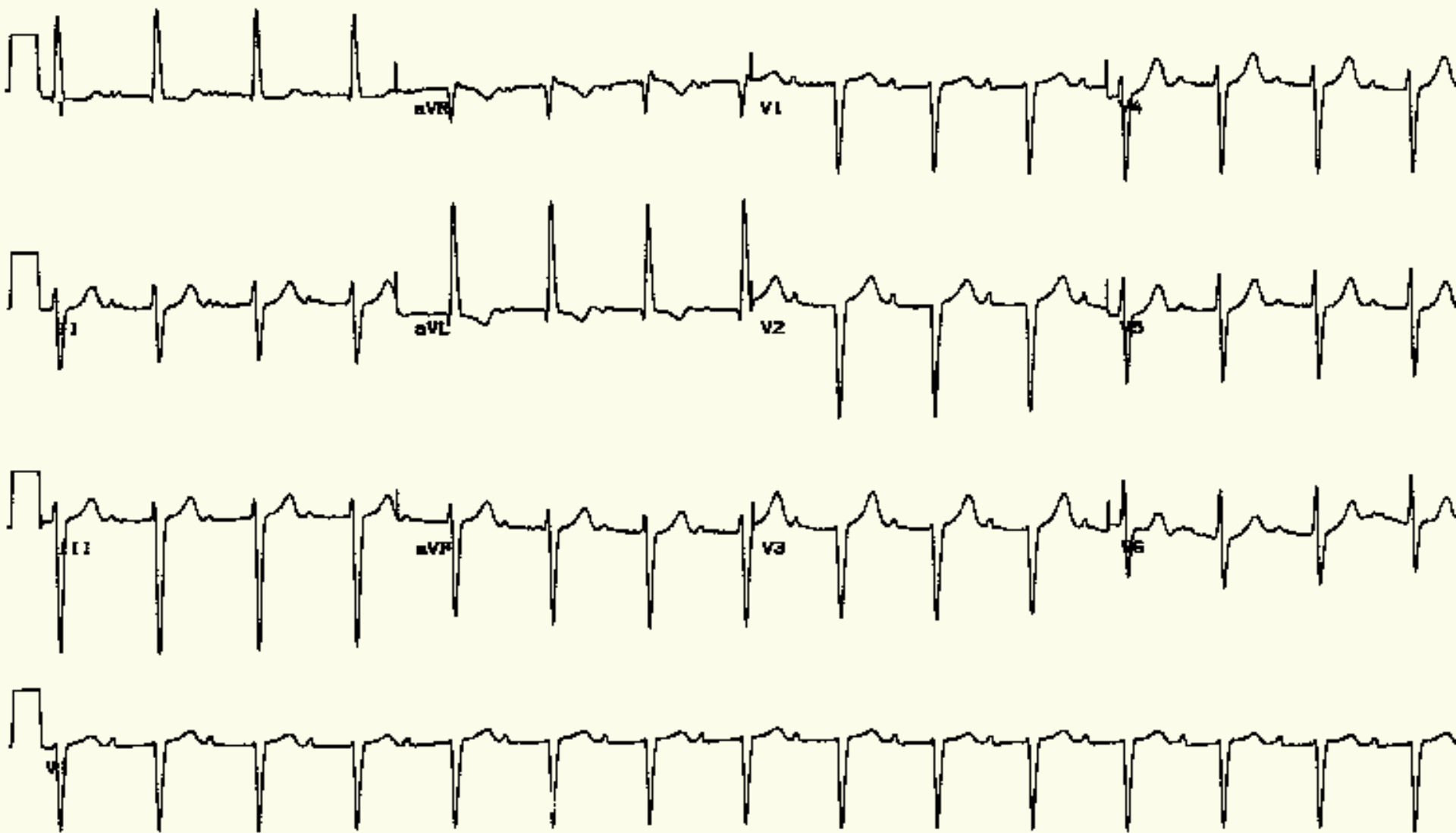
LBBB



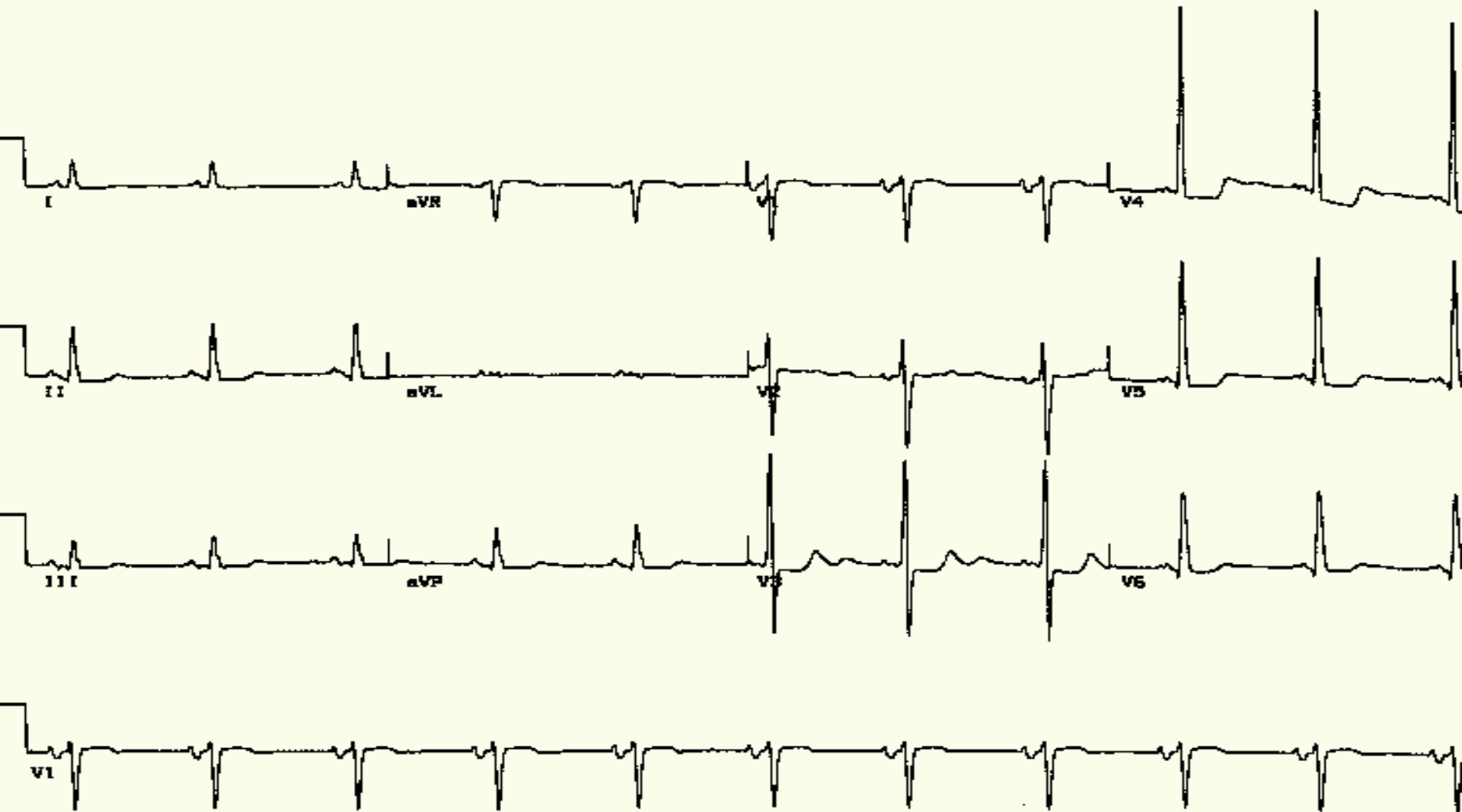
Acute Inferior MI



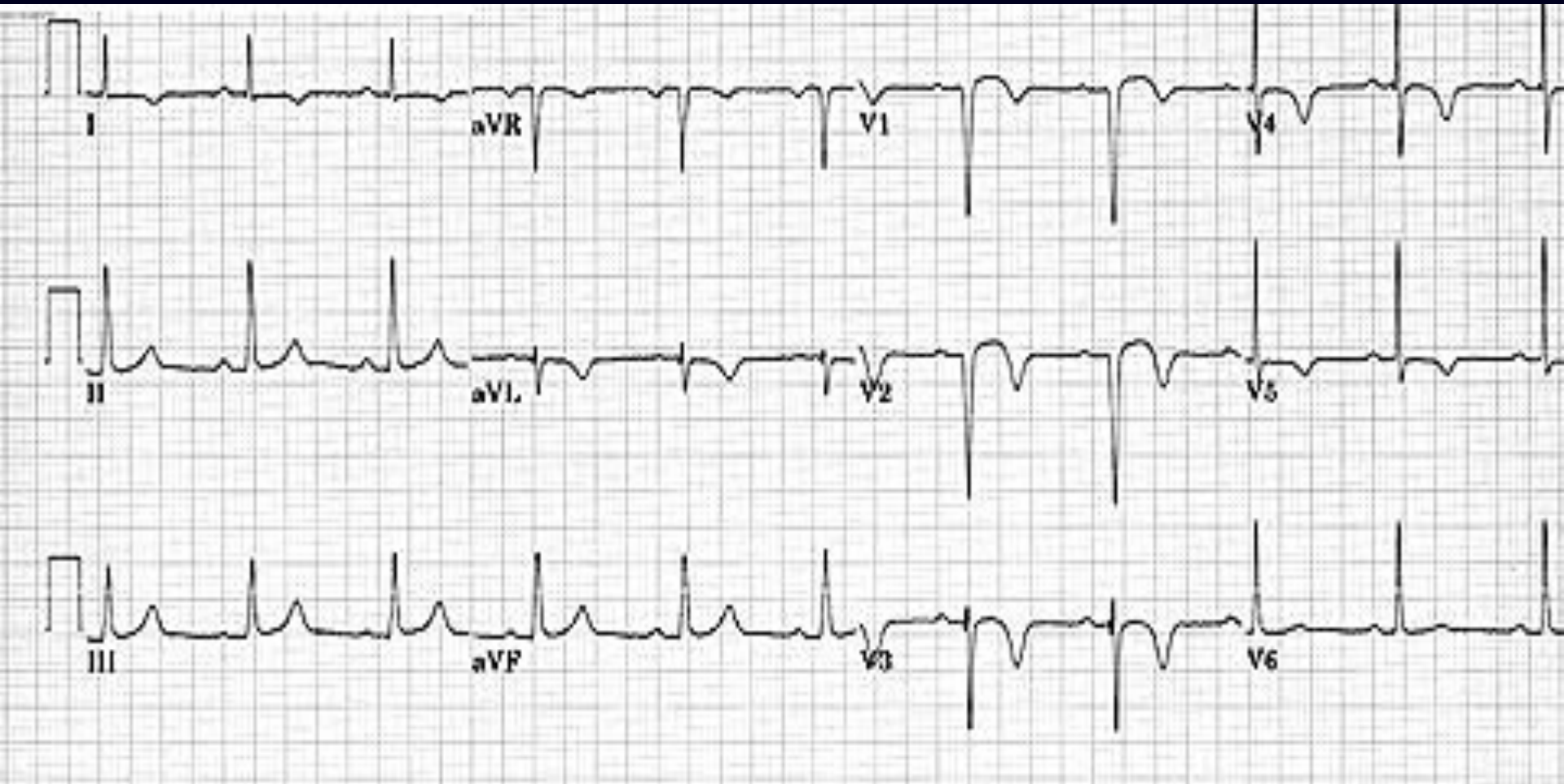
Left Anterior Hemiblock, Prolonged PR interval



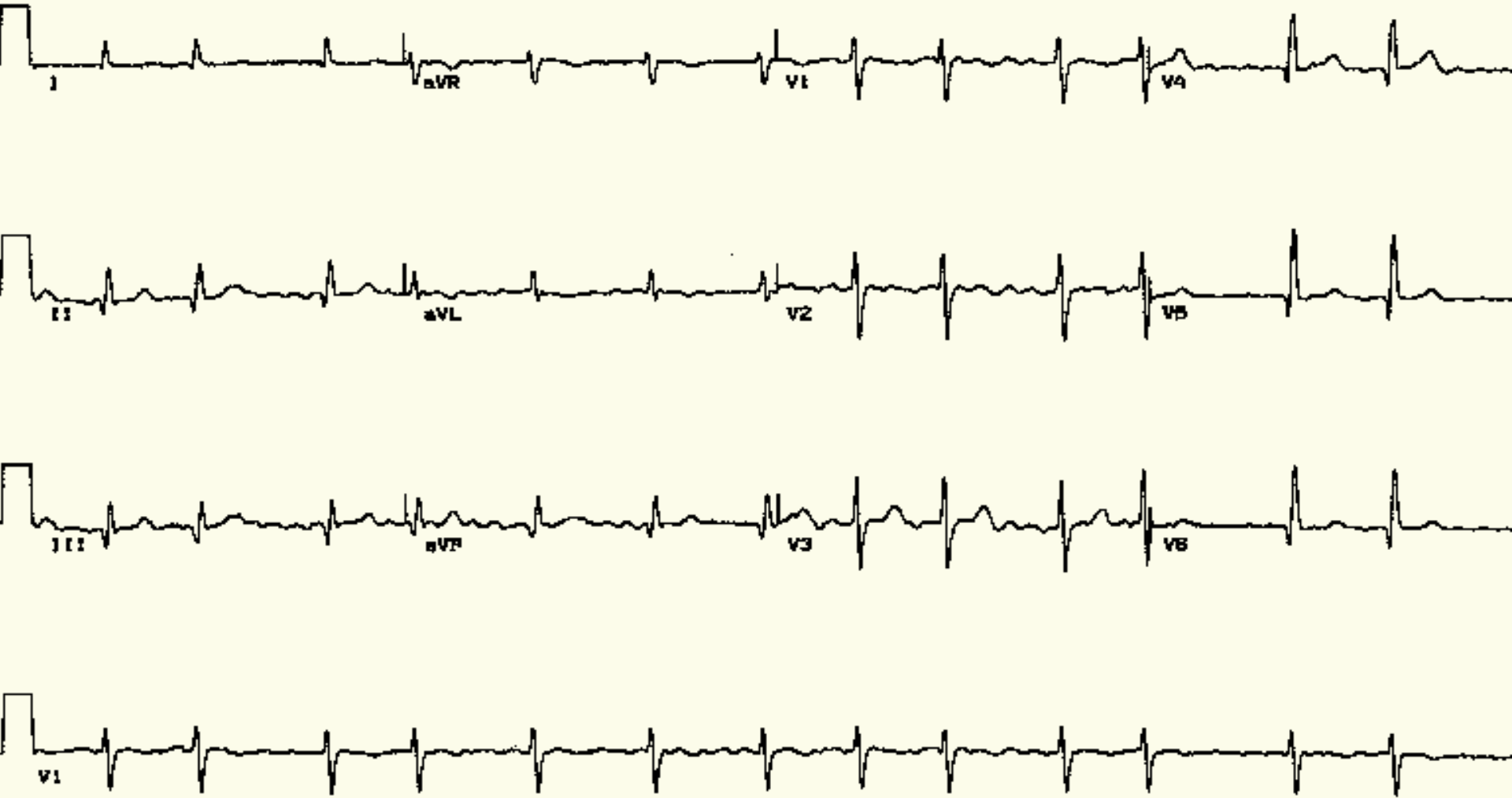
LVH and LA Enlargement



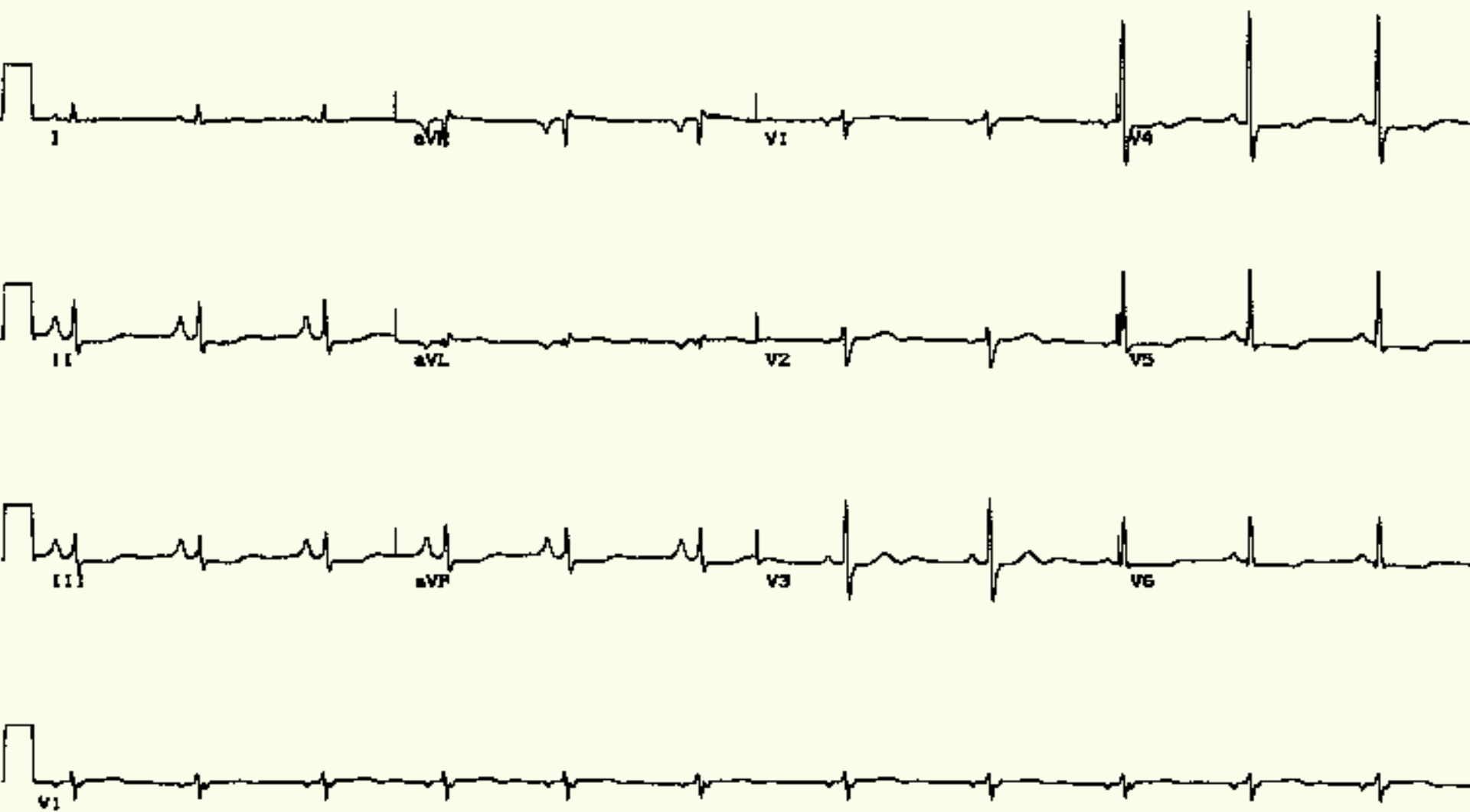
Anterior MI



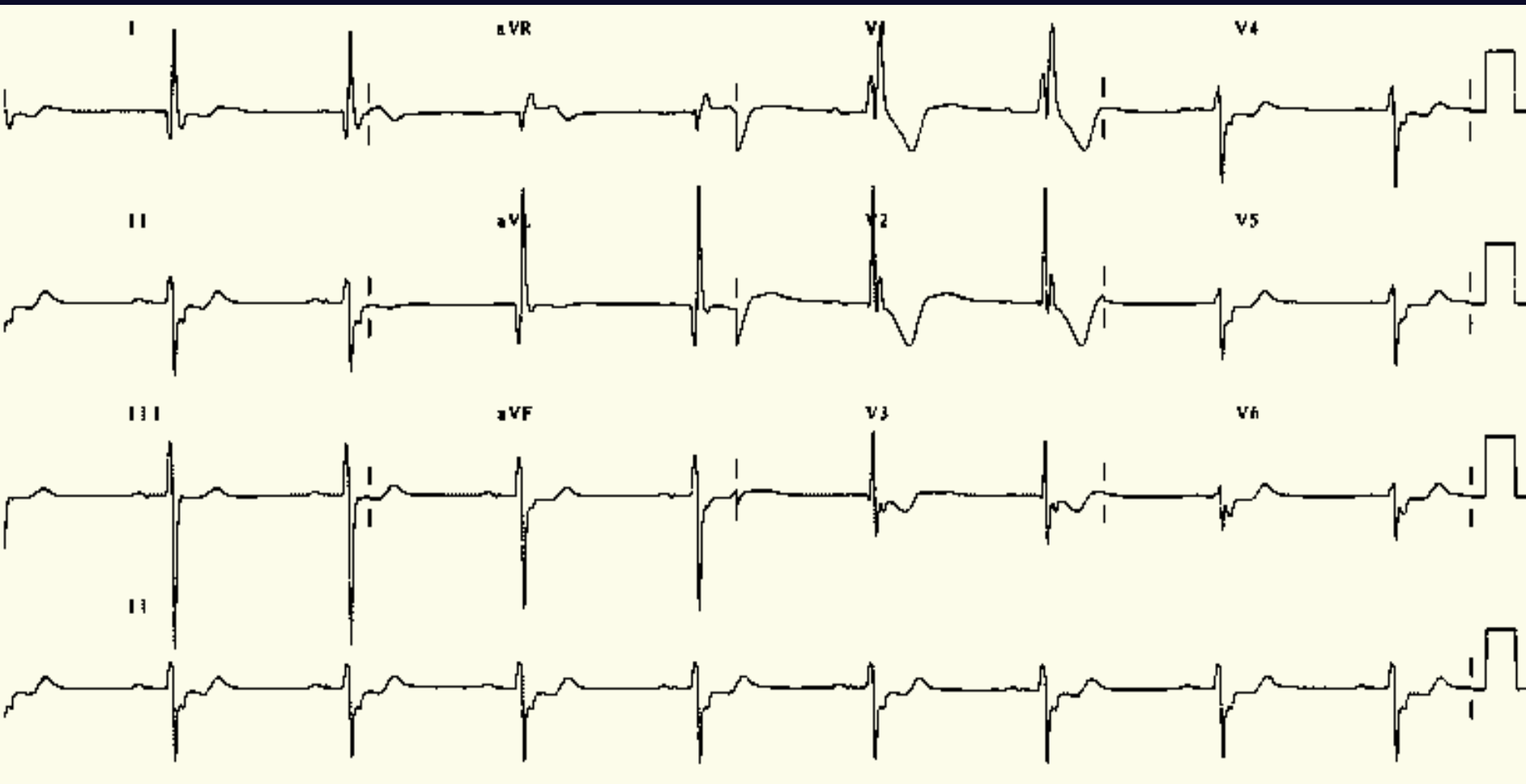
Old Inferior MI and Atrial Fibrillation



RA Enlargement



RBBB, LAH, Prolonged PR (Trifascicular Block)



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