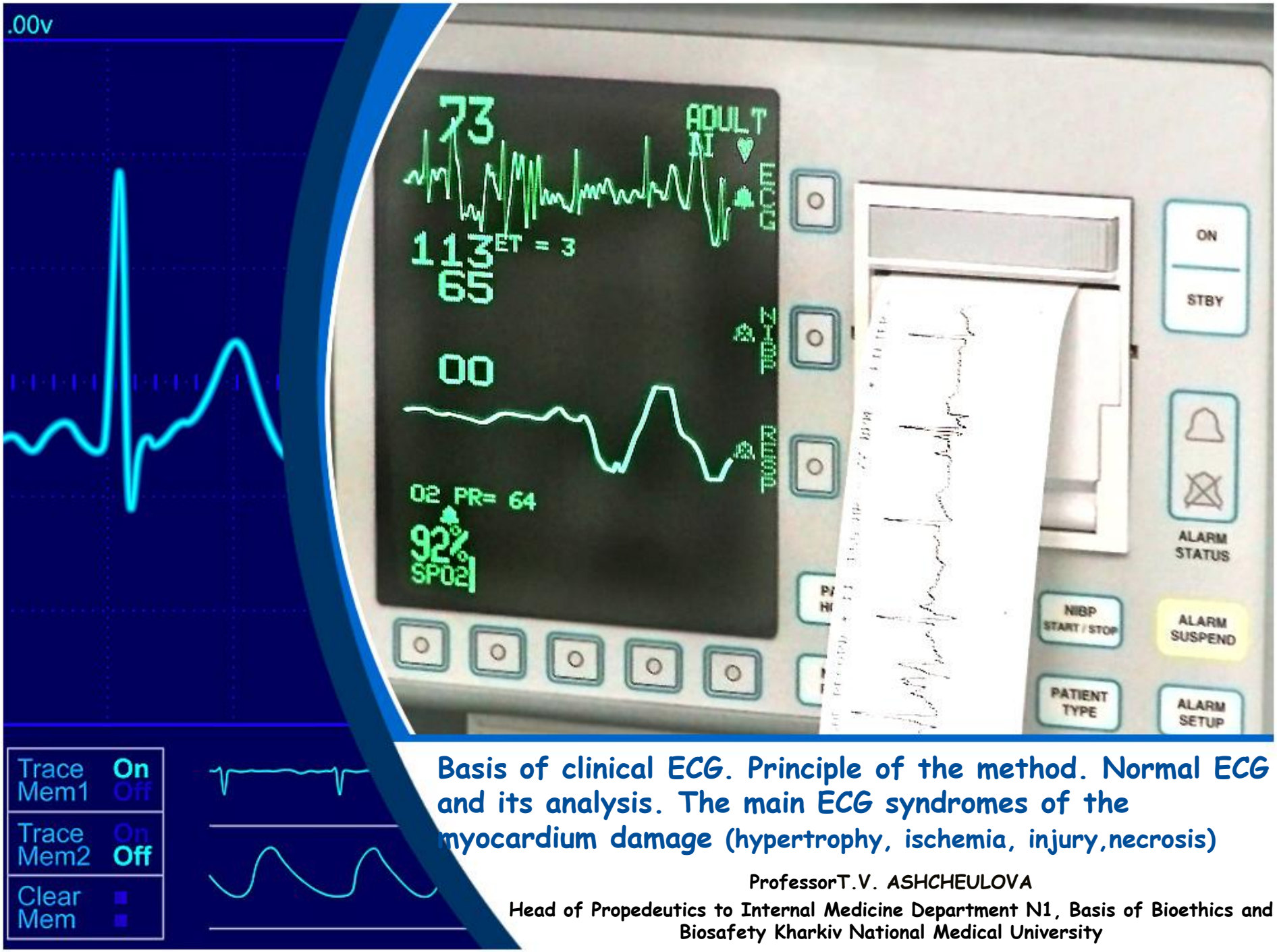


Basis of clinical ECG. Principle of the method. Normal ECG and its analysis. The main ECG syndromes of the myocardium damage (hypertrophy, ischemia, injury,necrosis)

ProfessorT.V. ASHCHEULOVA

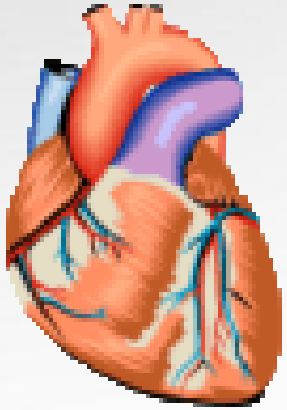
Head of Propedeutics to Internal Medicine Department N1, Basis of Bioethics and Biosafety Kharkiv National Medical University

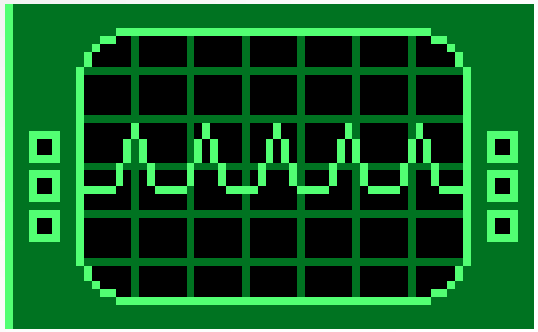

ECG
INTRODUCTION

Electrocardiography is the main instrumental method of heart investigation.

Electrocardiography

Electrocardiography – method of bioelectric potential registration, that appear as a result of heart work.

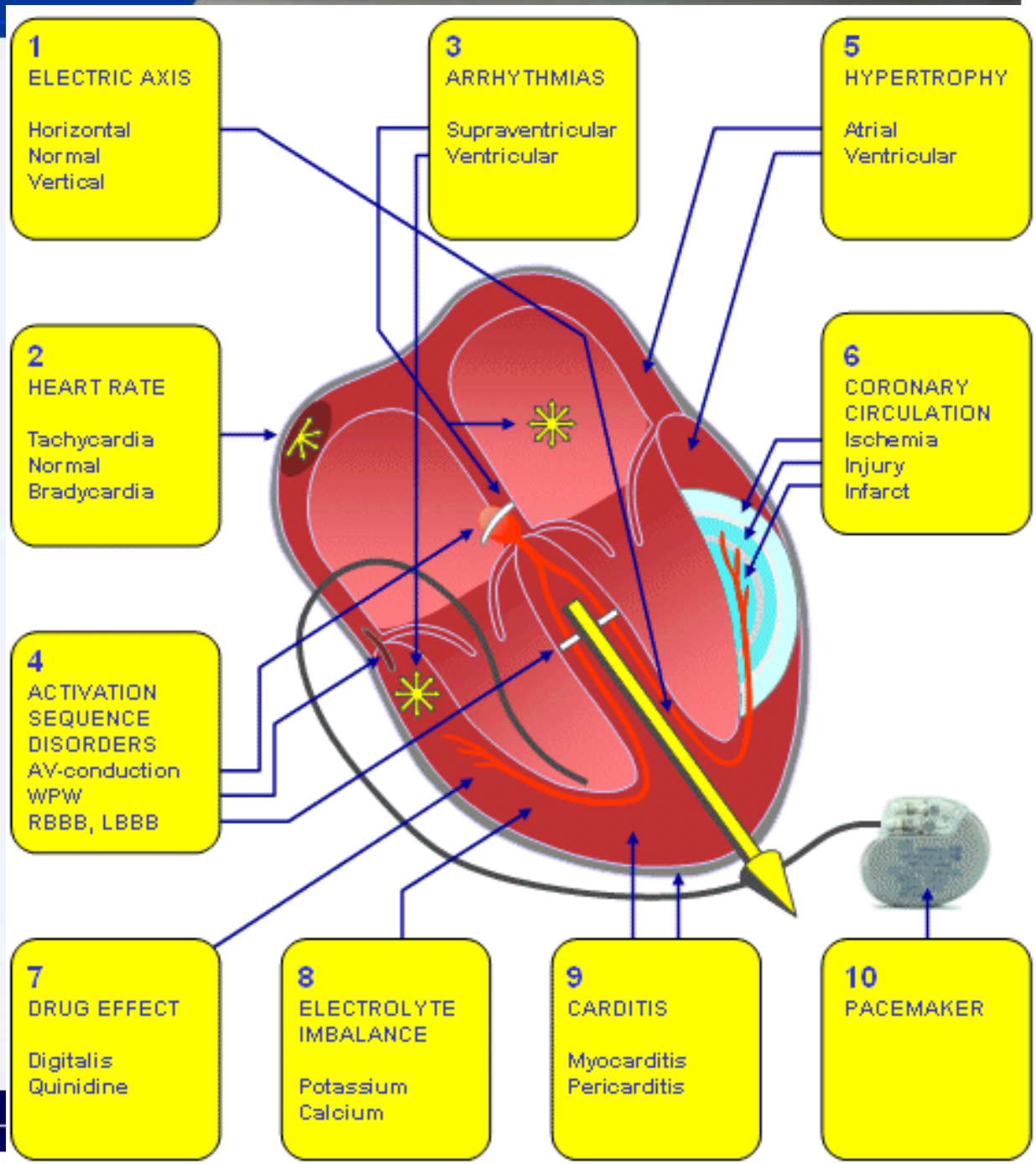




Electrocardiogram

graphic representation of the bioelectrical forces produced by the heart

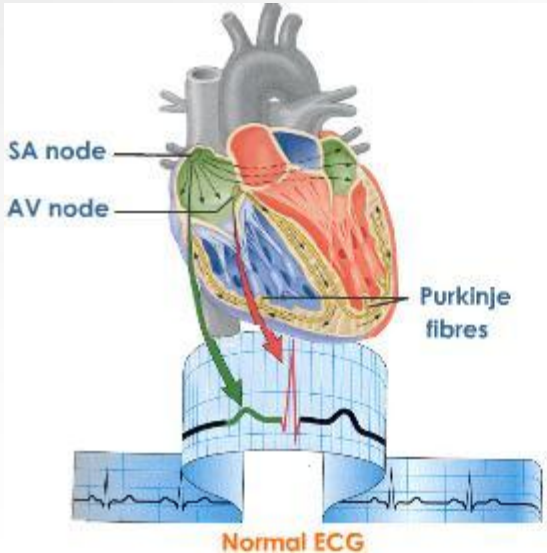
Application areas of ECG diagnosis

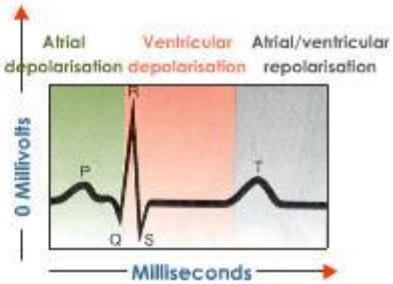



ECG
electrophysiologic
principles

Electrophysiologic principles

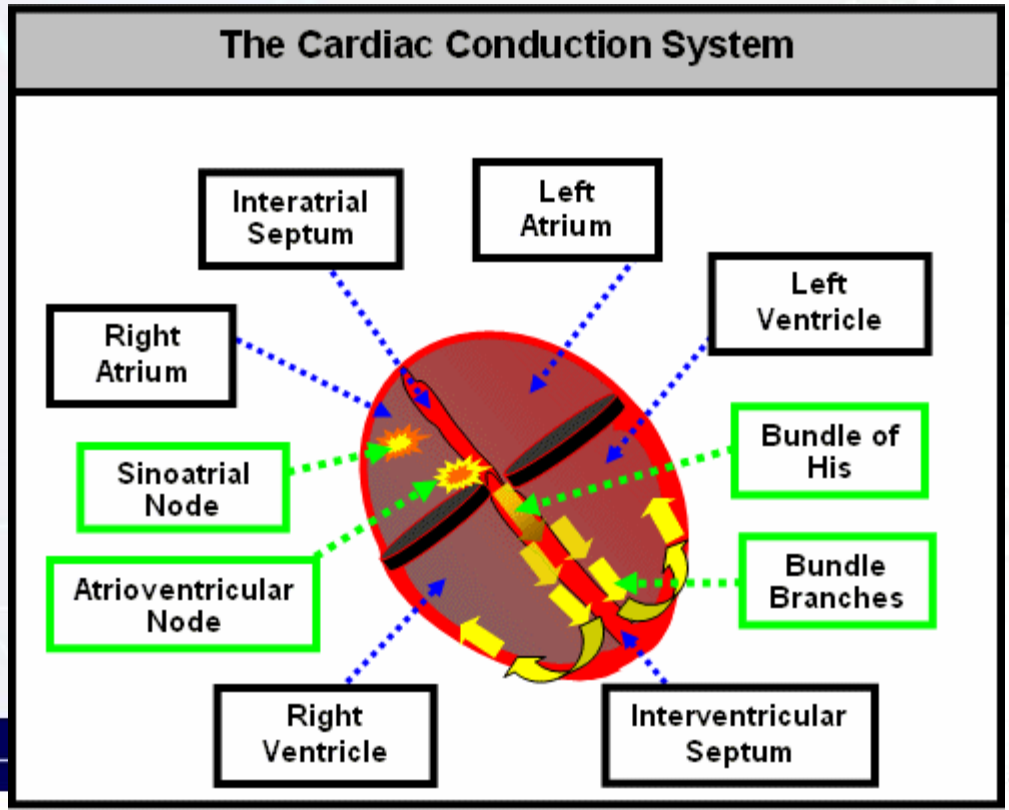
To understand electrocardiography, one must know certain electrophysiologic principles including automaticity, conductivity, depolarization, repolarization, refractoriness, and reentry.





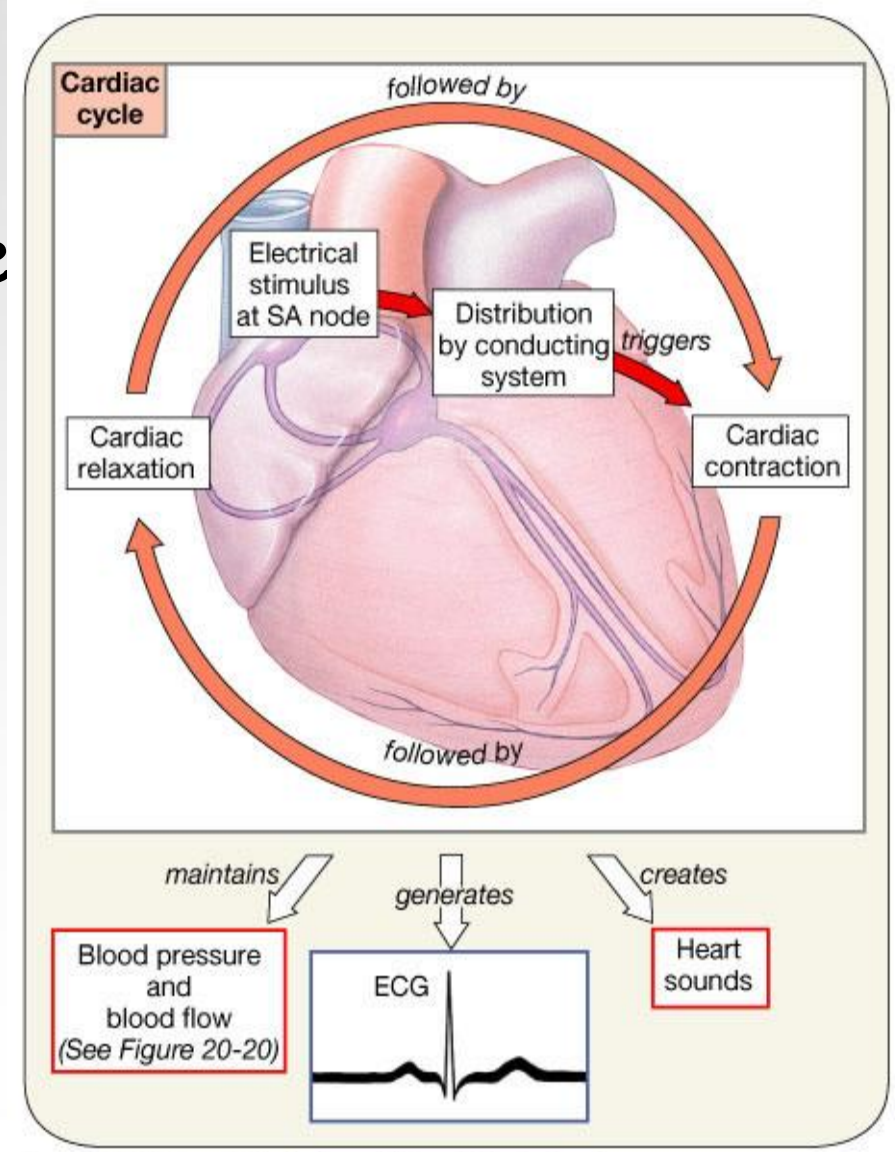
Electrophysiologic principles

The primary function of the heart is to contract so that it can serve its role as a pump. Each contraction is preceded by an electrical stimulus. When a patient has a regular sinus rhythm, the rhythm is initiated by the discharge of the sinus node. This electrical impulse is conducted through the right atrium, into the left atrium and into the ventricles through specialized conduction tissue. These specialized conduction pathways allow the heart to be electrically activated in a predictable manner.



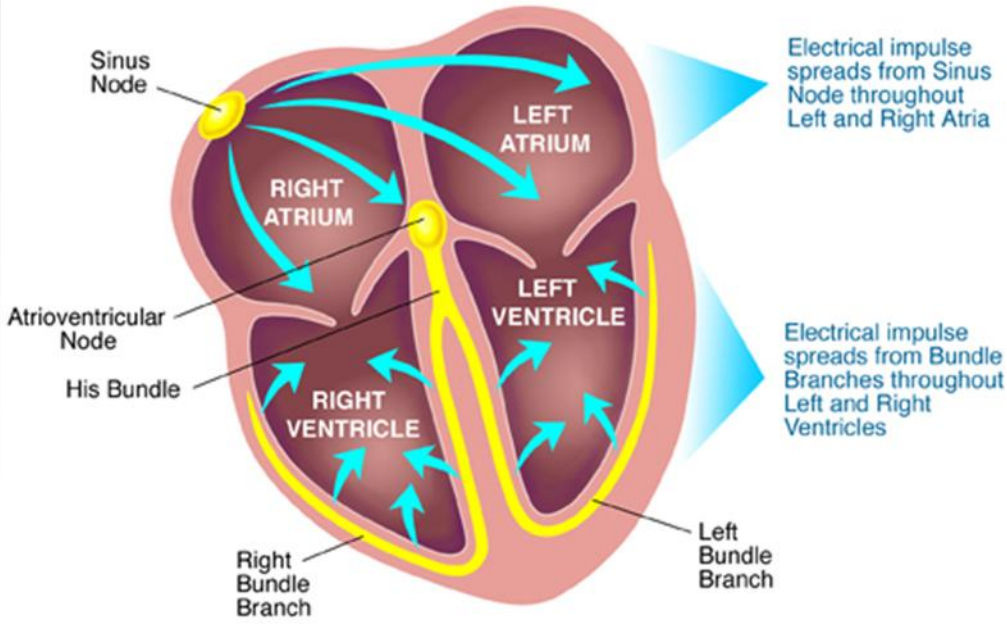
Automaticity

Automaticity is the heart ability to discharge electrical impulse without external stimulus, and is characteristic of the entire conduction system. Contractile myocardium has no automaticity function.



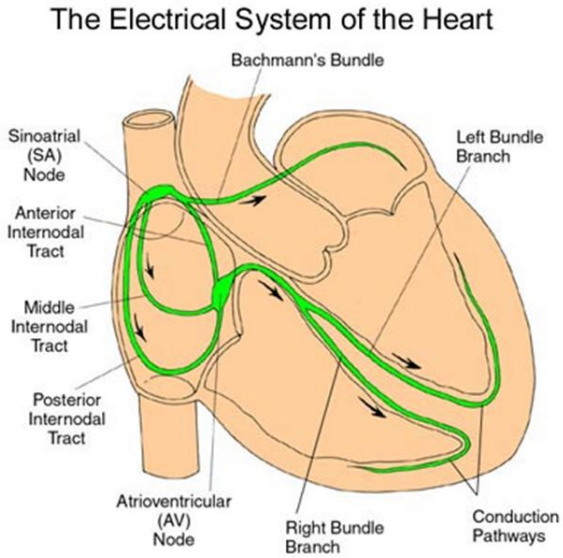
Automaticity

- The sinus node has the highest automaticity and is therefore the pacemaker of the cardiac rhythm. In the adult the usual resting discharge rate of sinus node is 60 to 100 impulses per minutes.



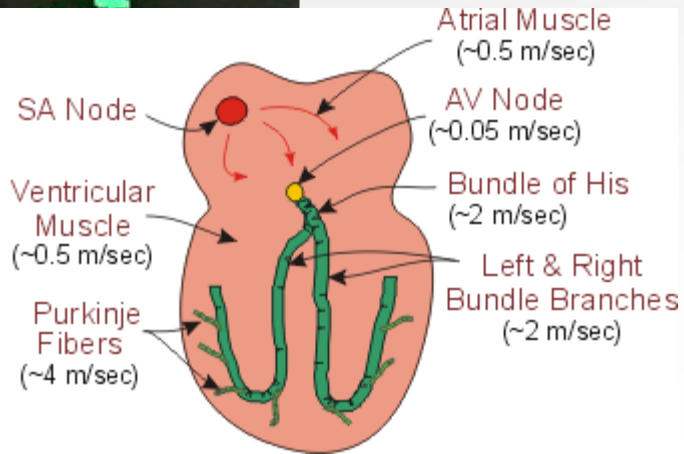
Automaticity

- Automaticity is characteristic of the entire conduction system of the heart, but in normal conditions it is inhibited by the high activity of the SA node, which is the automaticity center of the first order. If SA node is affected, or transmission of excitation to AV node is impaired, the zone of transition of AV node to the common bundle of His becomes the pacemaker (the second-order automaticity center). Impulses are generated here at a rate of 40 to 50 per minute. Lower part of the bundle of His and its branches, and Purkinje fibers are the automaticity center of the third order, but the rate of the cardiac rhythm then slow down to 20-30 per minute.



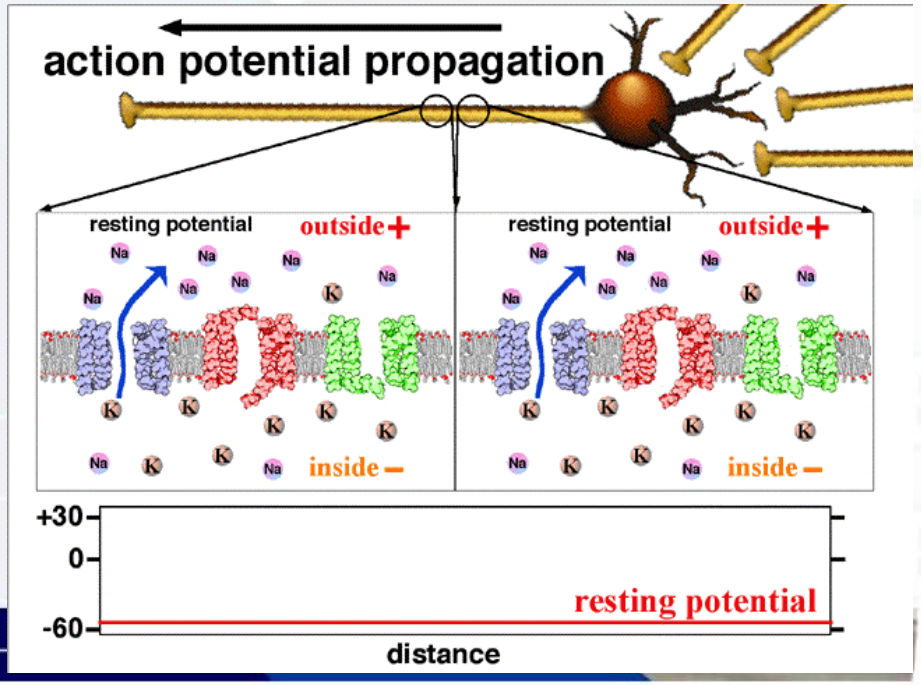
Conductivity

- **Conductivity** is a property of the myocardium that allows the transmission of electrical impulse from one cell to another, and is characteristic and of specialized conduction system, and of contractile myocardium. But by muscle cell-to-cell conduction is significantly slower than by specializes conduction pathways.



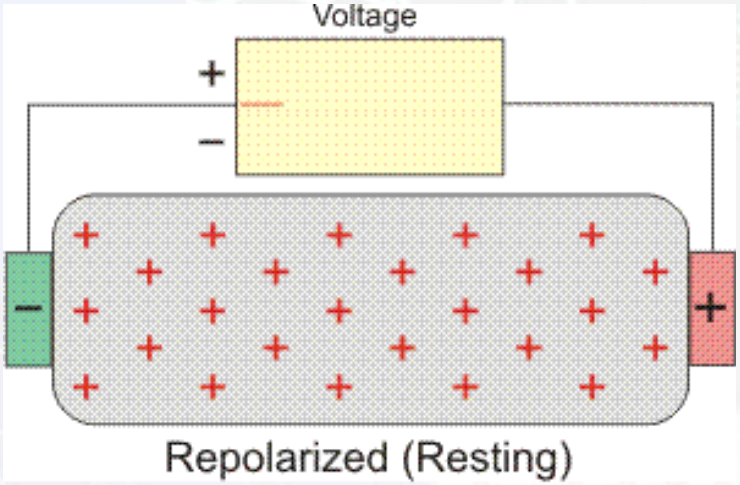
Depolarization. repolarization. Refractoriness

- The transmission of an electric impulse from one muscle cell to another is dependent on depolarization, repolarization, and refractoriness.
- During excitation, physicochemical properties of the cell membranes and ionic composition of the intercellular and intracellular fluid change to cause the electrical current generation.



Depolarization, repolarization, Refractoriness

- As the electrical impulse moves across the cells of the myocardium, the polarity (negative or positive charge) of the cells is changed. The resting cell outside has positive charge. The electrical impulse carries a positive charge into the cell, changing the polarity:
- This is called depolarization: It is followed by a continuing wave of repolarization that restores the cell to its original charge:
- The cell is then ready to receive another stimulus.



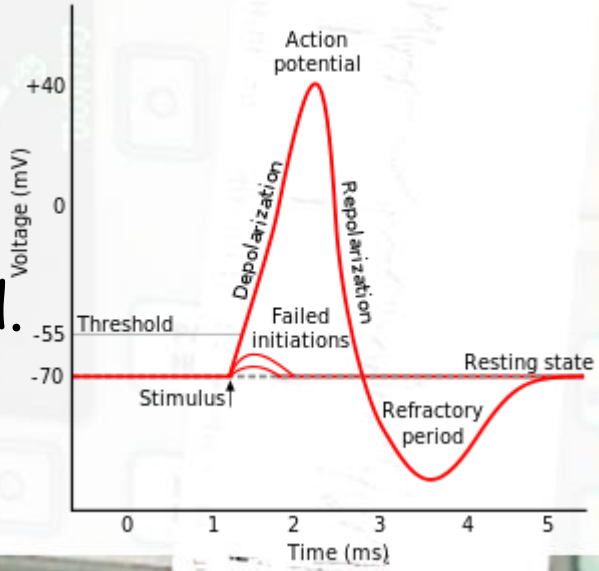
Depolarization. repolarization. Refractoriness

During depolarization and early repolarization, the myocardial cell is completely refractory to further electrical stimulation, and excitability is zero; that is, no stimulus will produce response.

Refractoriness is the property of conductive and heart muscle tissue that prevents it from responding to a stimulus until it has appropriately repolarized. This prevents the muscle from sustaining a titanic contraction.

Refractoriness is rate-dependent.

The slower the rate and the longer the cycle, the longer refractory period.




ECG
recording

Operating principle

- Fluctuations in the potential difference that arise during the excitation of the heart muscle are sensed by the electrodes attached to the patient's body and transmitted to the apparatus.

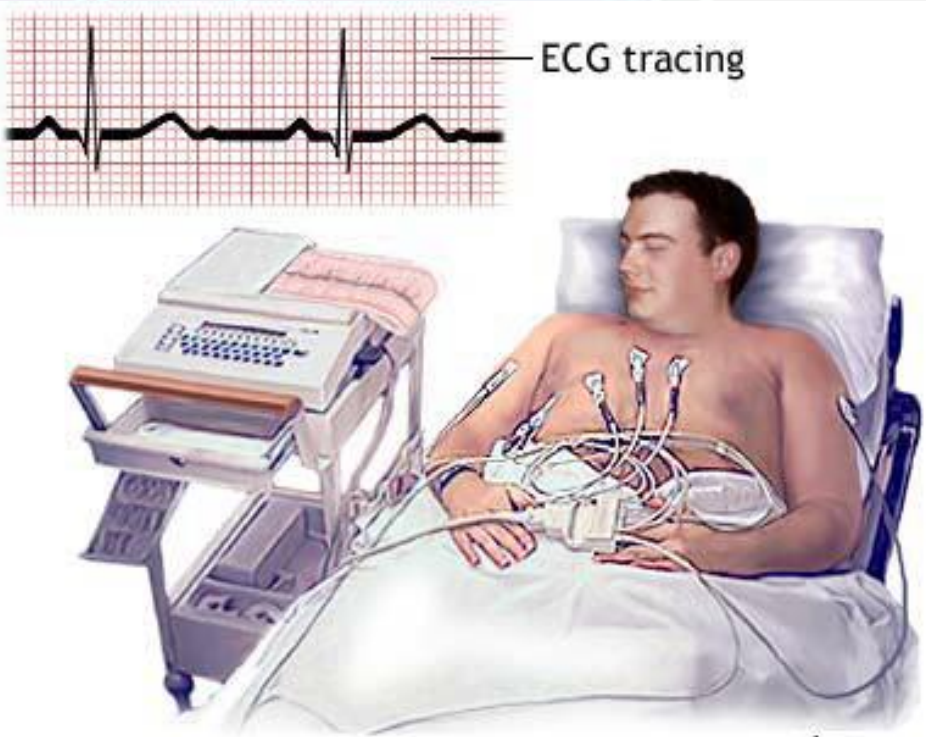
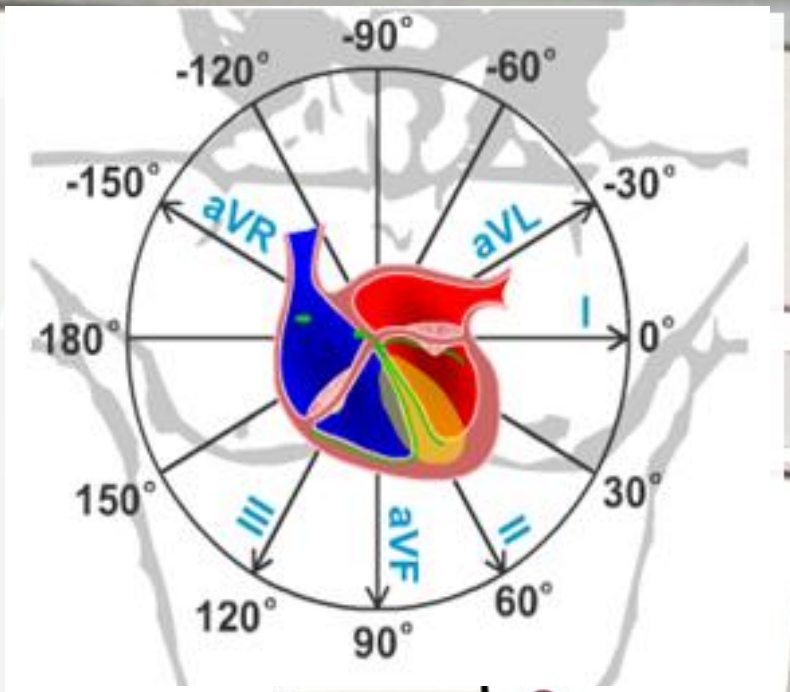


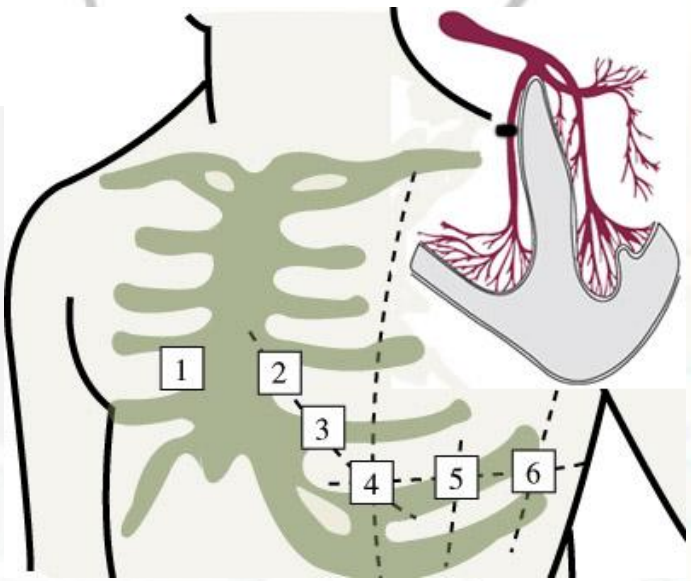
Illustration of a patient getting an ECG. A.D.A.M.

The ECG leads

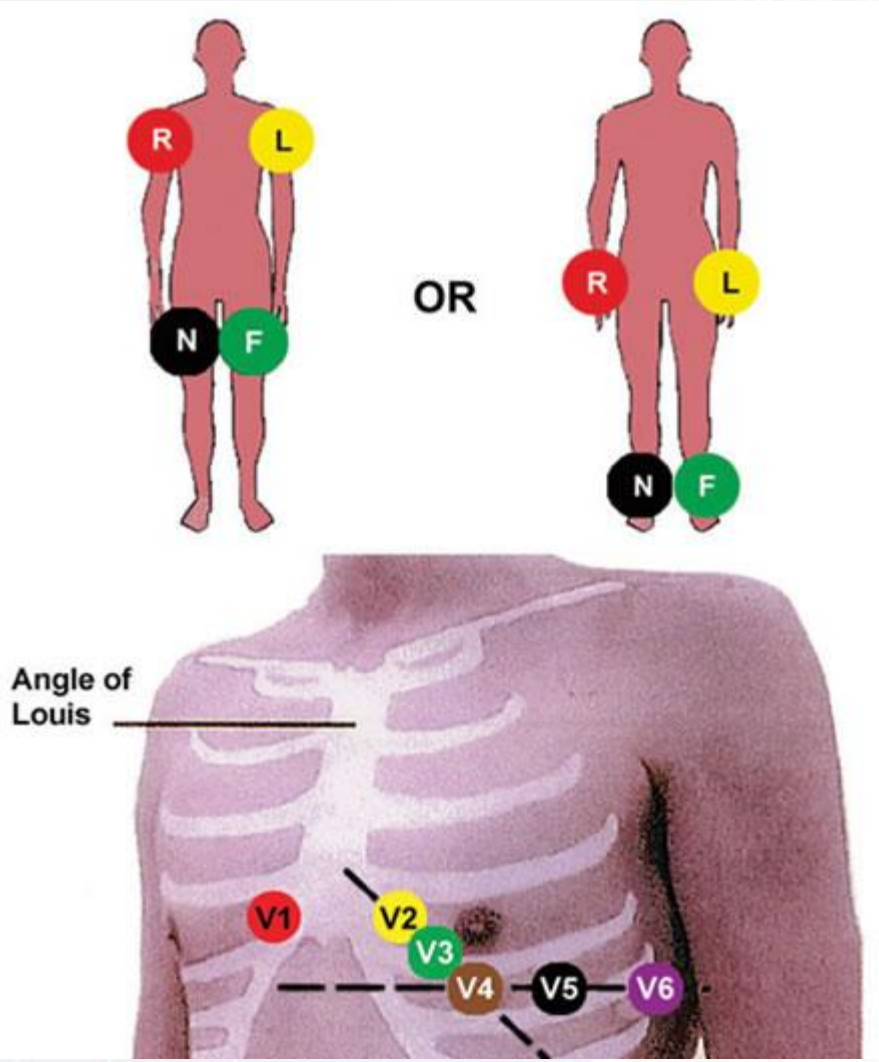
To record a routine ECG, 12 leads are used in clinical practice:

- 3 standard bipolar limb leads,
- 3 augmented unipolar limb leads
- 6 chest leads.



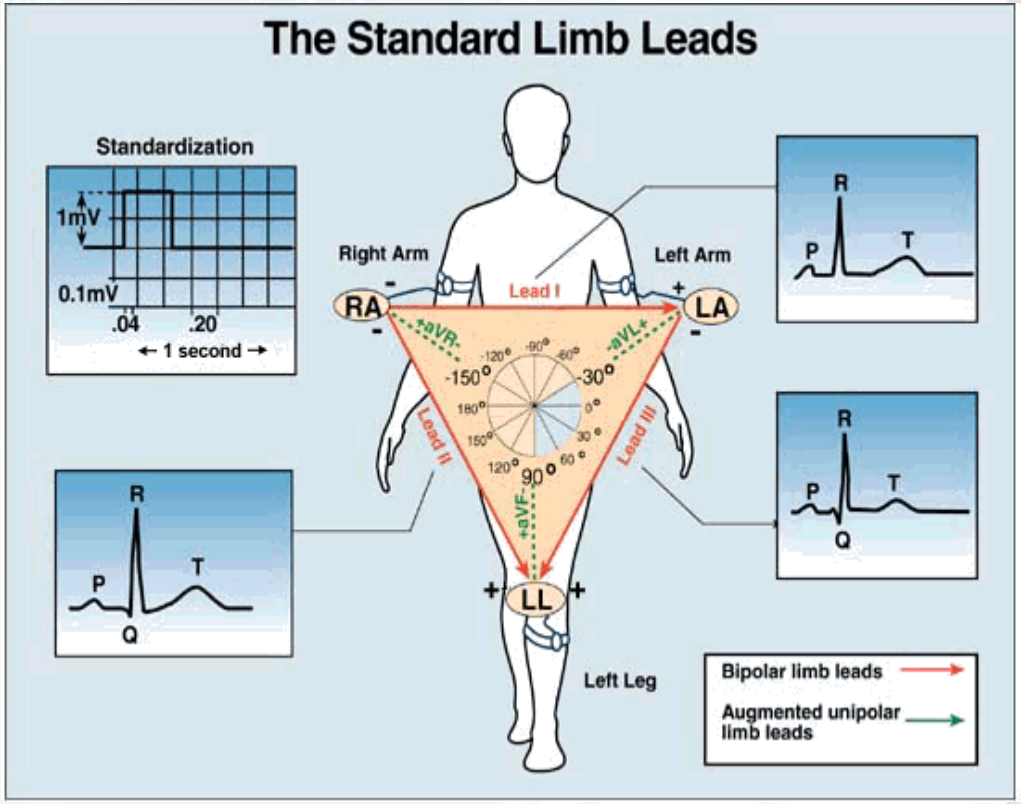


ECG registration. Electrodes placement



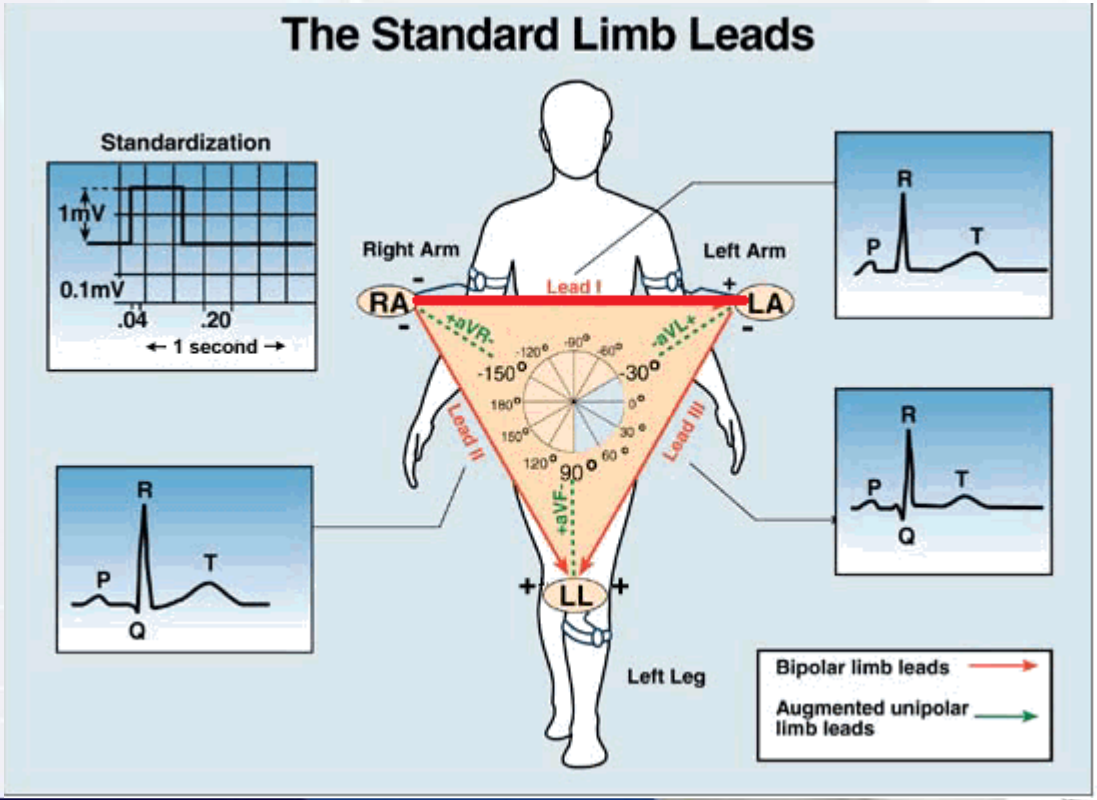
Standard Bipolar Limb Leads

Standard leads proposed by Einthoven in 1913 y., record potential difference between two points of the body. The electrodes are placed on the right arm, the left arm, the left leg, and the fourth electrode on the right leg is connected with the earth wire.



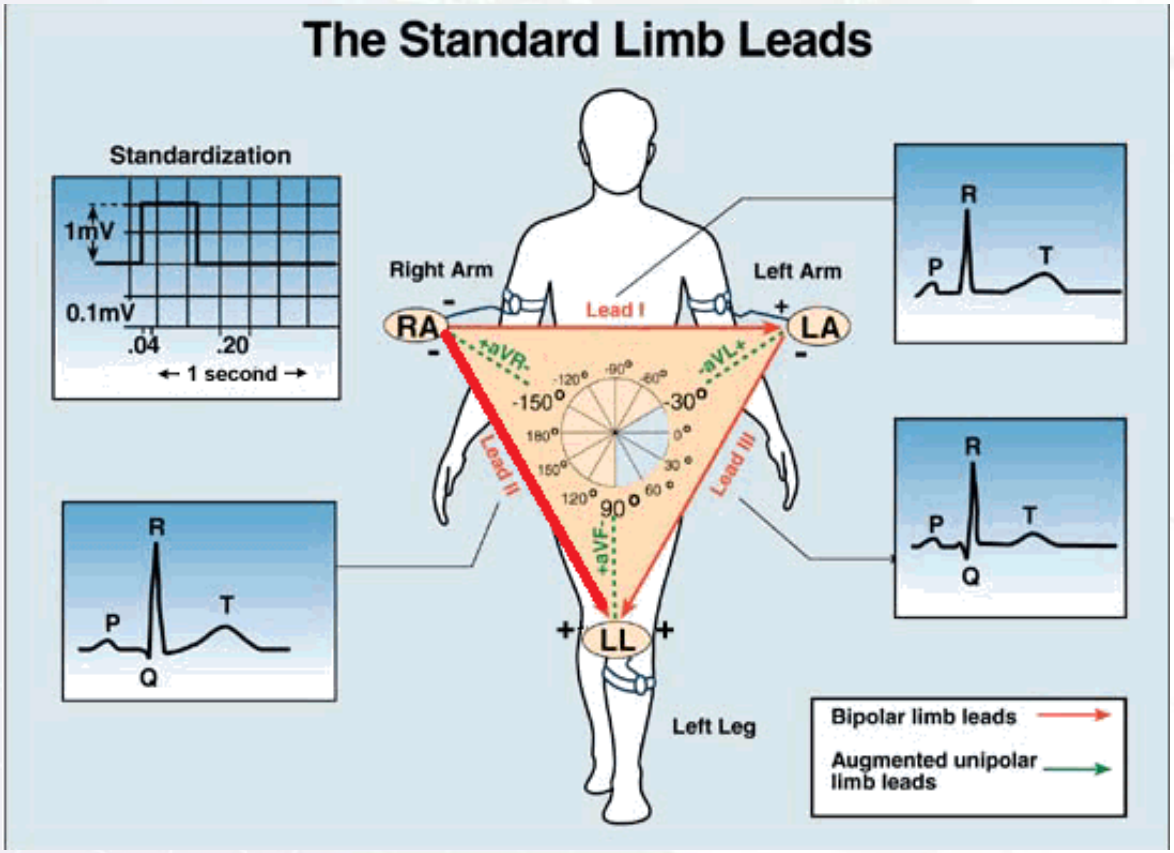
Standard Bipolar Limb Leads

- **Lead I** is obtained by connecting the right arm (RA -) and left arm (LA +) electrodes.



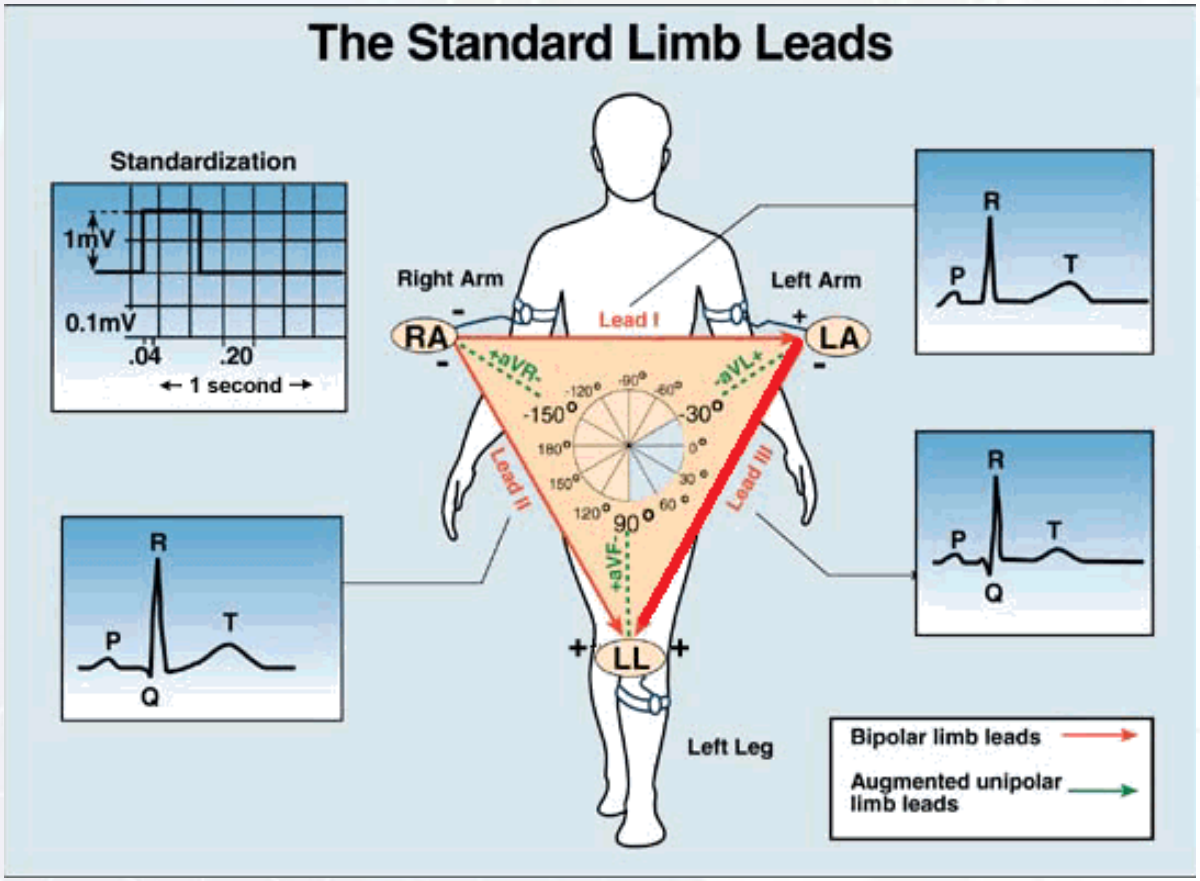
Standard Bipolar Limb Leads

- **Lead II** is obtained by connecting the right arm (RA -) and left leg (LL+) electrodes.



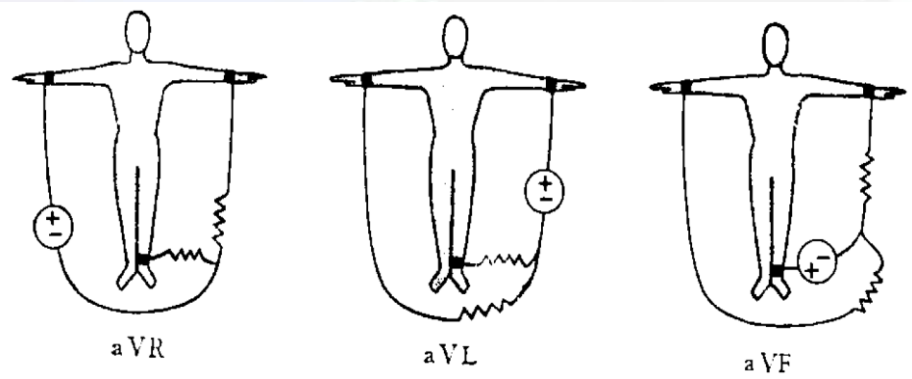
Standard Bipolar Limb Leads

- **Lead III** is obtained by connecting the left arm (LA -) and left leg (LL +) electrodes.



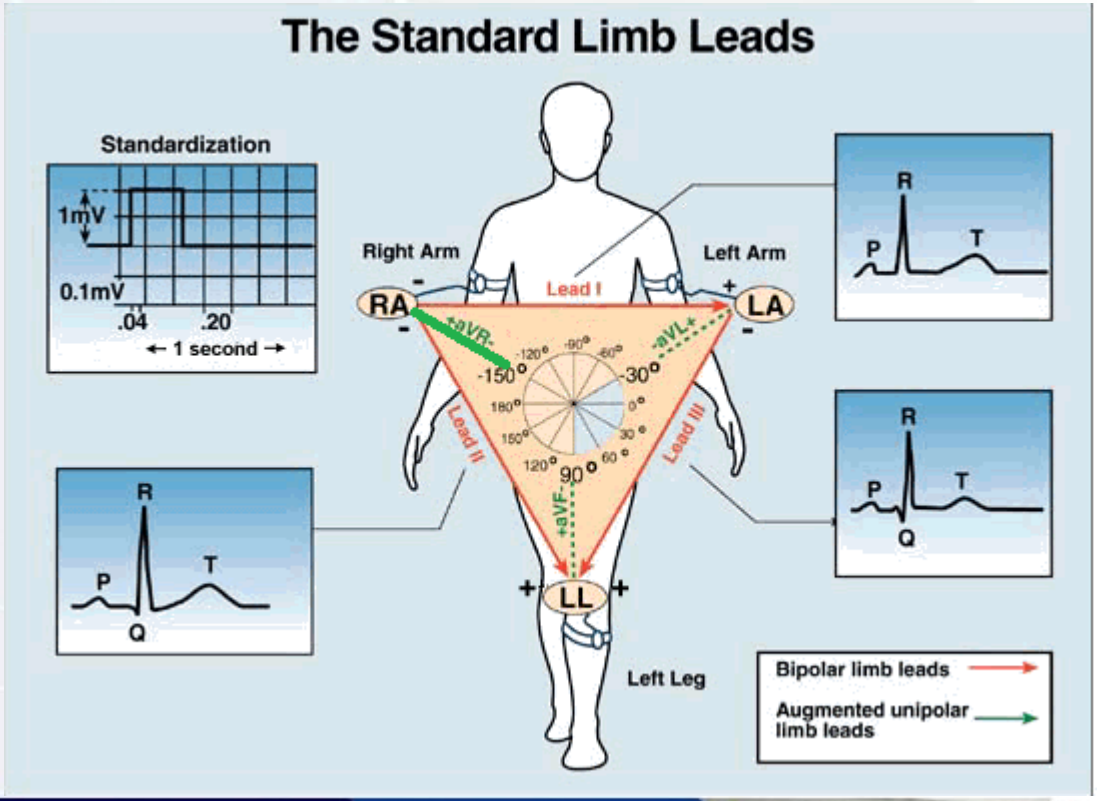
Augmented Unipolar Limb Leads

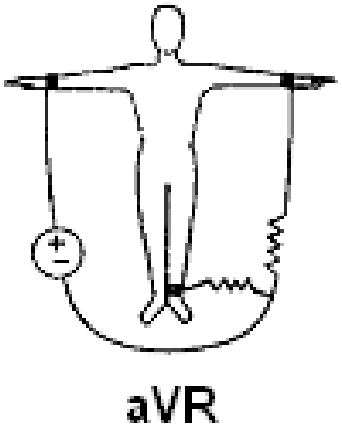
- Goldberger proposed augmented unipolar leads in 1942 year and modified "augmented Wilson's extremity lead".
- Three augmented unipolar leads are distinguished: aVR, aVL, aVF.
- The letter "a" is used to designate the augmented lead.



Augmented Unipolar Limb Leads

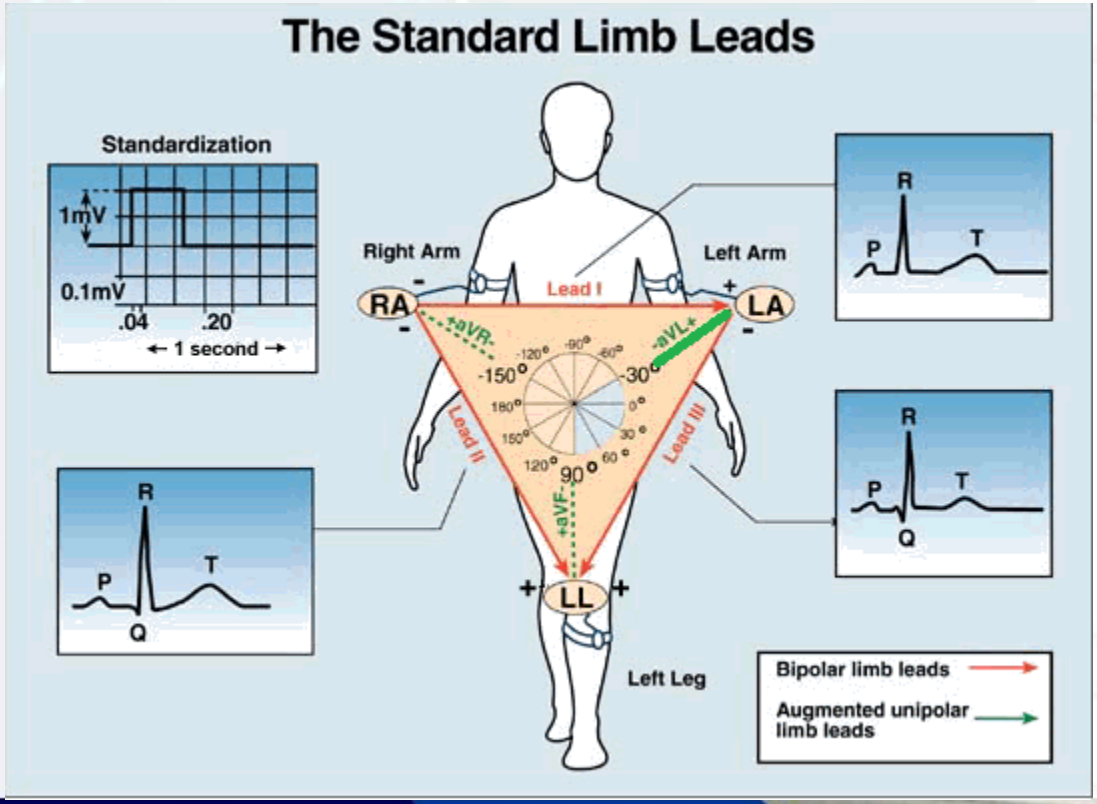
- **aVR:** augmented unipolar right arm (RA +) lead, the central (-) terminal is connection of the left arm (LA) and left leg (LL) electrodes;

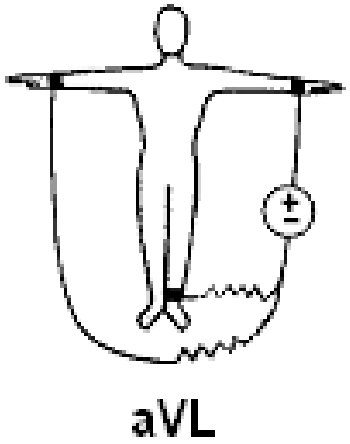




Augmented Unipolar Limb Leads

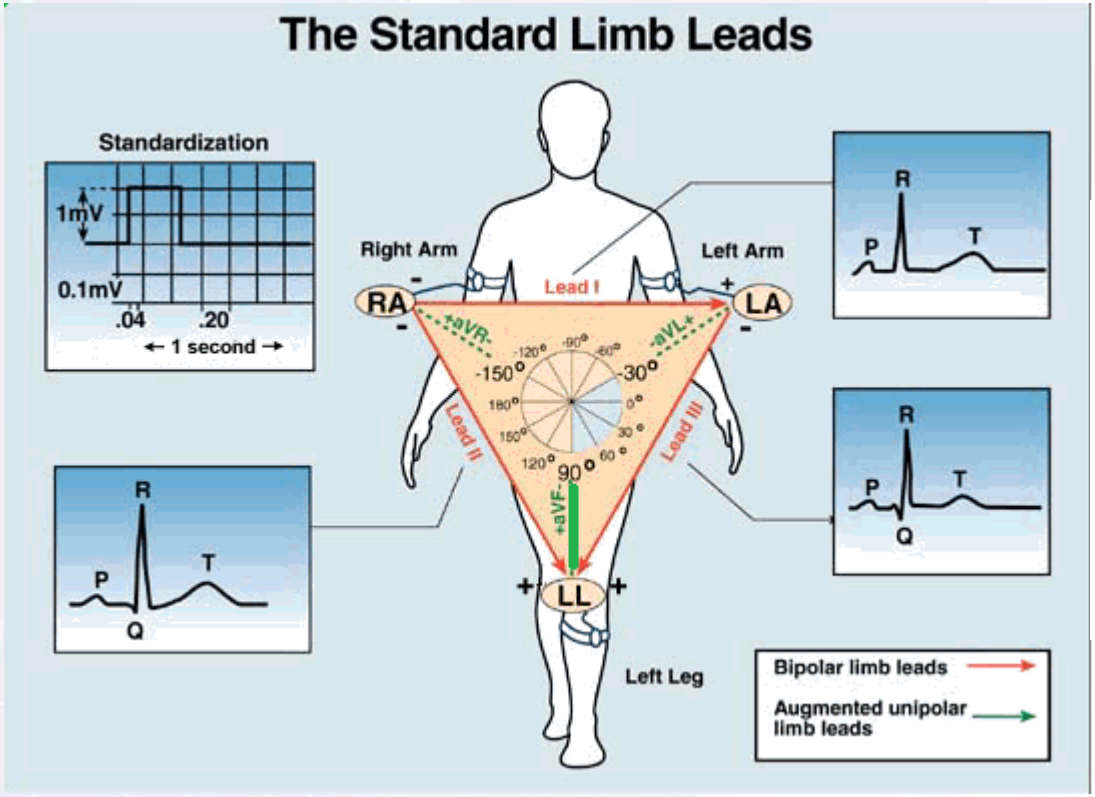
- **aVL**: augmented unipolar left arm (LA +) lead, the central (-) terminal is connection of right arm (RA) and left leg (LL) electrodes;
-

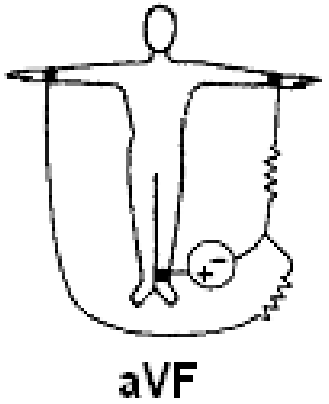




Augmented Unipolar Limb Leads

aVF: augmented unipolar left leg (+) lead, the central terminal is connection of right arm and left arm electrodes.

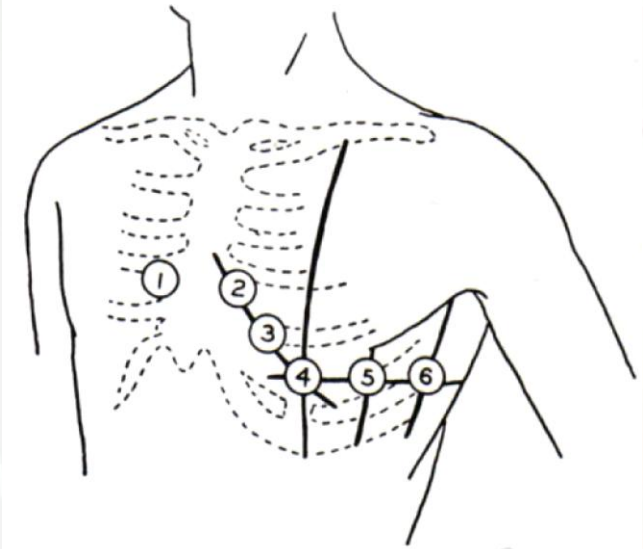




Unipolar Chest Leads

Wilson proposed unipolar chest leads in 1934 year.

In the 1938 the American and British Heart Associations standardize the nomenclature of precordial leads.



Unipolar Chest Leads

V_1: fourth intercostal space, right sternal border

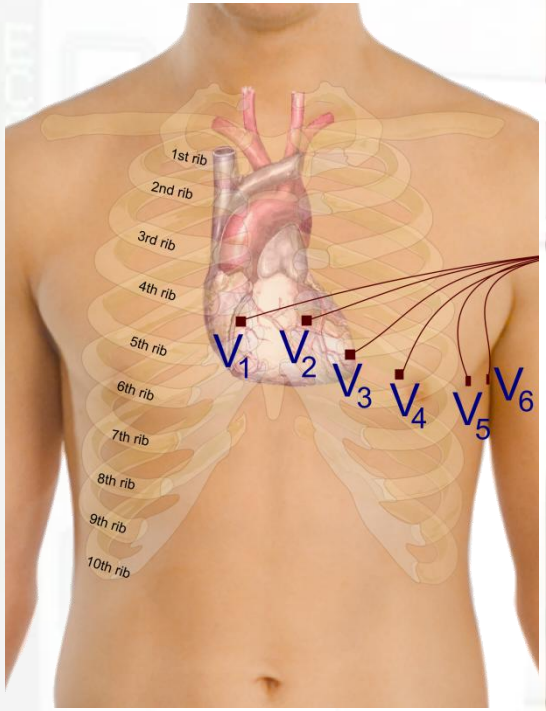
V_2: fourth intercostal space, left sternal border

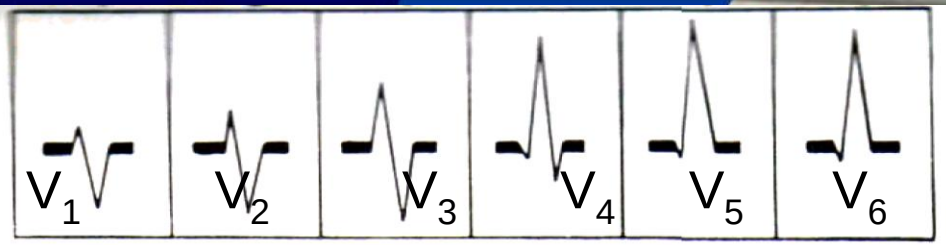
V_3: midway between V_2 and V_4 on a line jointing these two points

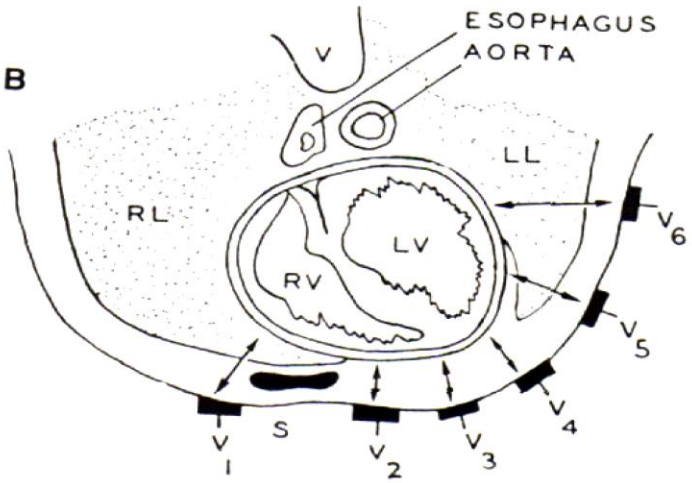
V_4: interspace in which apex is located (fifth or sixth); midclavicular line

V_5: anterior left axillary line; on the same level with V_4

V_6: left midaxillary line; on the same level with V_4 and V_5



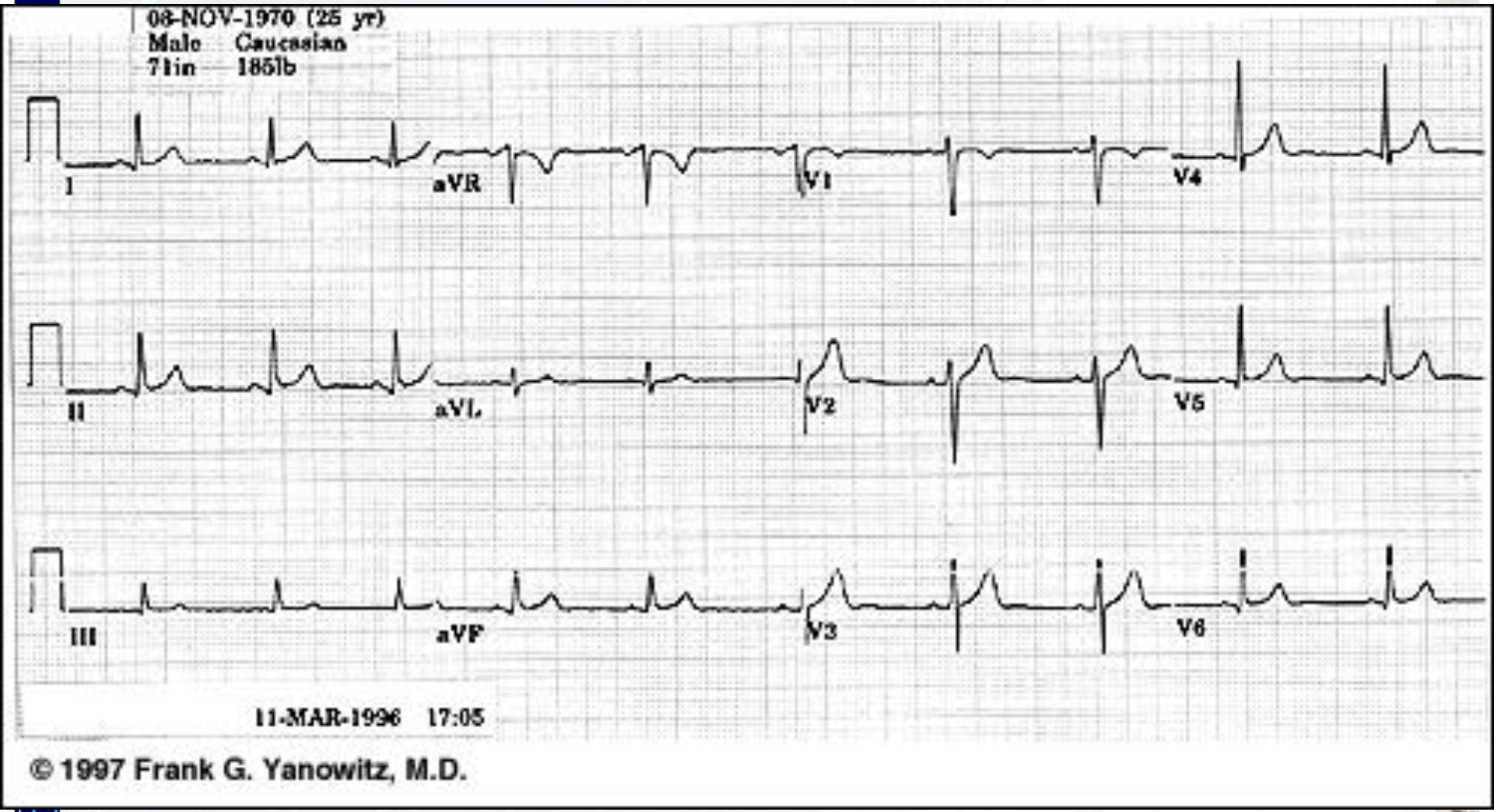




the QRS changes commonly seen in leads V_1 to V_6 in a normal subject.

Cross-section of the heart in the thoracic cage. Note the relation of chest electrodes to the anatomy of the heart.

Normal ECG


08-NOV-1970 (25 yr)
Male Caucasian
71in 185lb
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
11-MAR-1996 17:05
© 1997 Frank G. Yanowitz, M.D.

The most important leads to remember in relation to the anatomy of the heart are:

Right leads

Lead III

Left leads

Lead I

Lead II confirms alterations in lead I or lead III, therefore:

Leads III, II

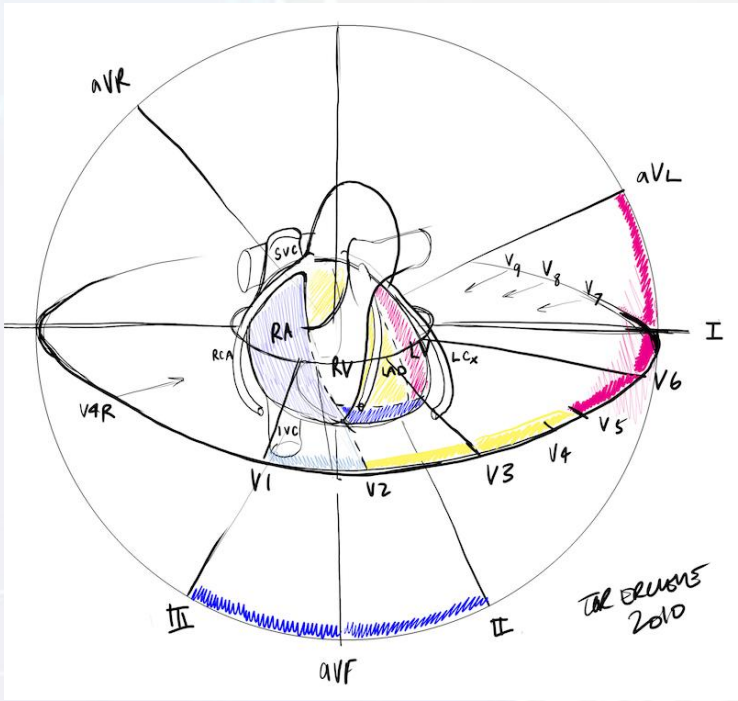
aVF, aVR

V_1, V_2

Leads I, II

aVL

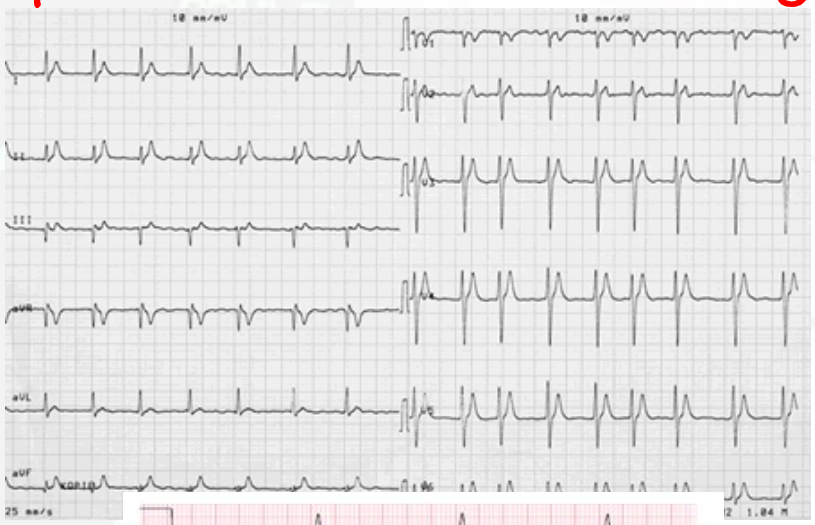
V_4, V_5, V_6

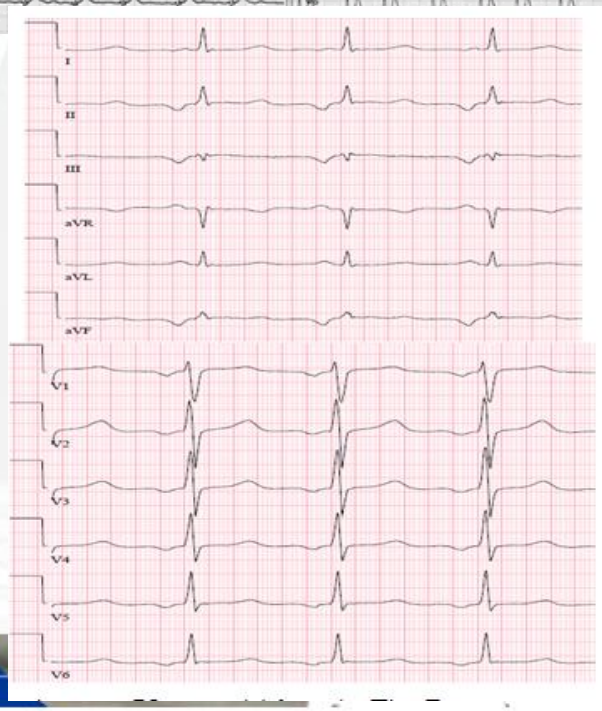


V_3 – transition zone between right and left sides of the heart

Speed of ECG recording

The tape may move at various speeds, from 25 to 100 mm/s, but the speed of 50 mm/s is usually preferred. Once the speed of the tape is known, it is easy to calculate the duration of the separate elements of the ECG. Waves amplitude is measured in mm, duration – in seconds.



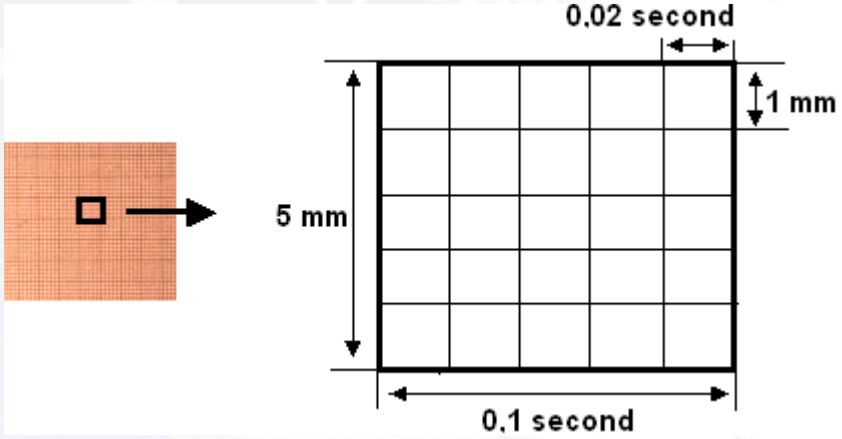


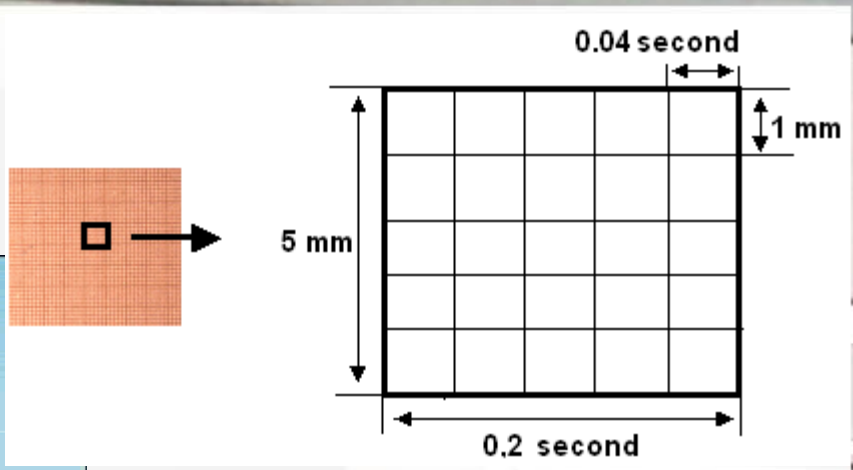
ECG paper

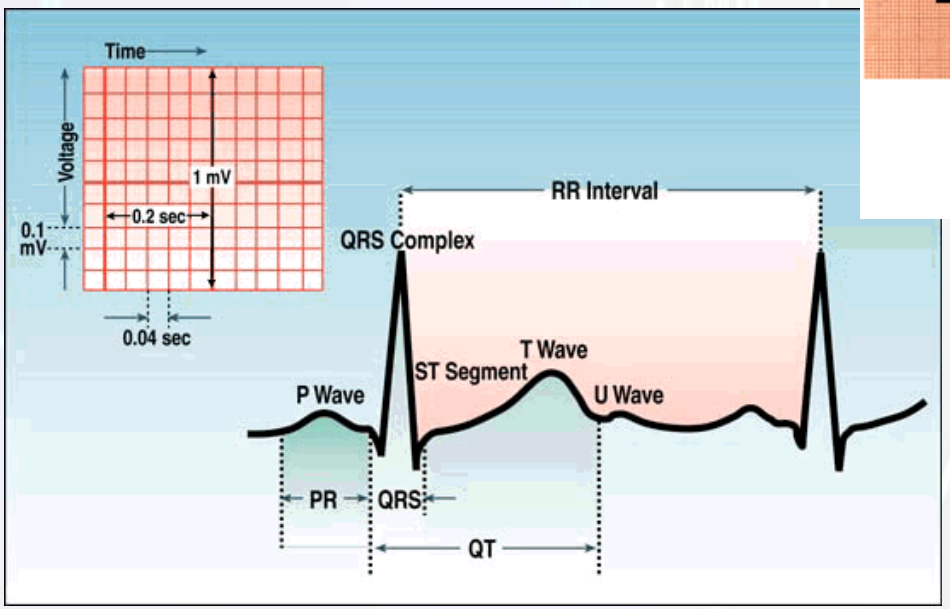
The paper graduated in millimeters is used to record ECG.

If an ECG is recorded at a speed of 50 mm/s, one small block represents 0.02 second on the horizontal line and 1 mm on the vertical line. Since a large block is five small blocks wide and five high, each large block represents

0.1 second (horizontal) and 5 mm (vertical).



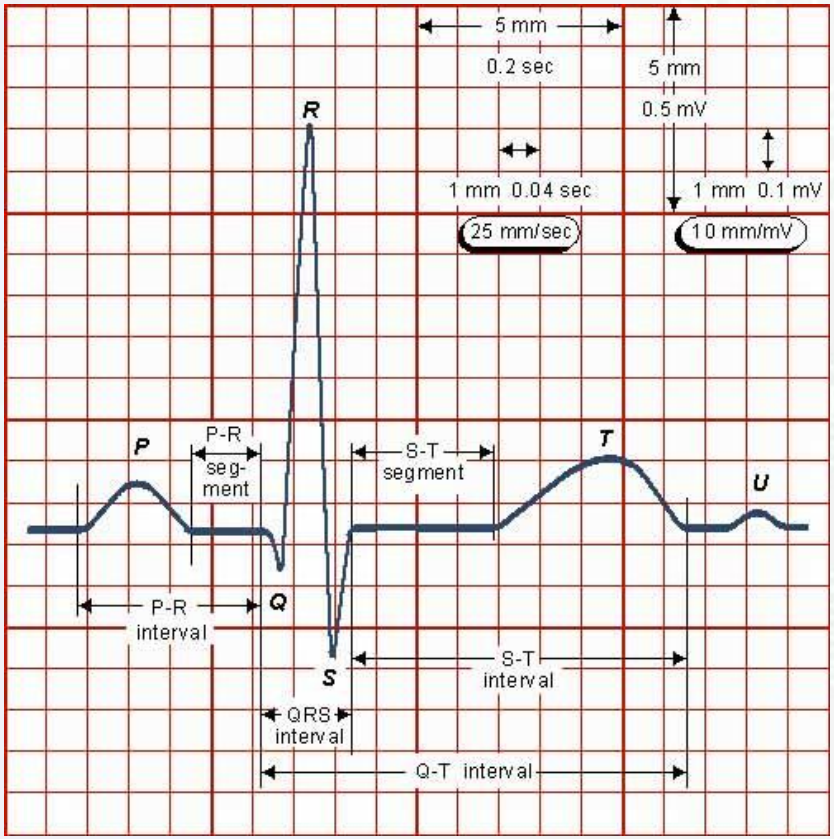




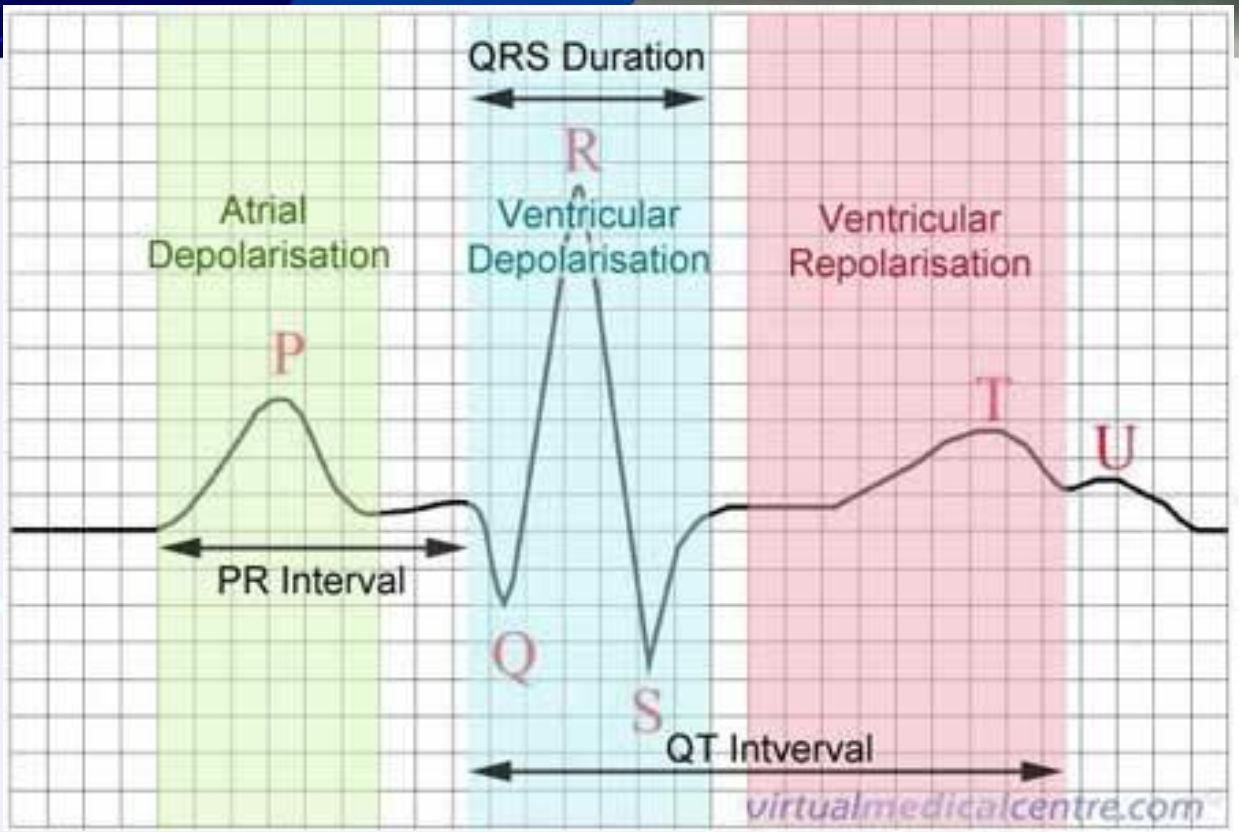
If an ECG is recorded at a speed of 25 mm/s, one small block represents 0.04 second on the horizontal line and 1 mm on the vertical line. A large block represents 0.2 second (horizontal) and 5 mm (vertical).


ECG
reading

ELECTROCARDIOGRAM



The electrocardiogram is simply a graphic representation of the electrical forces produced by the heart .



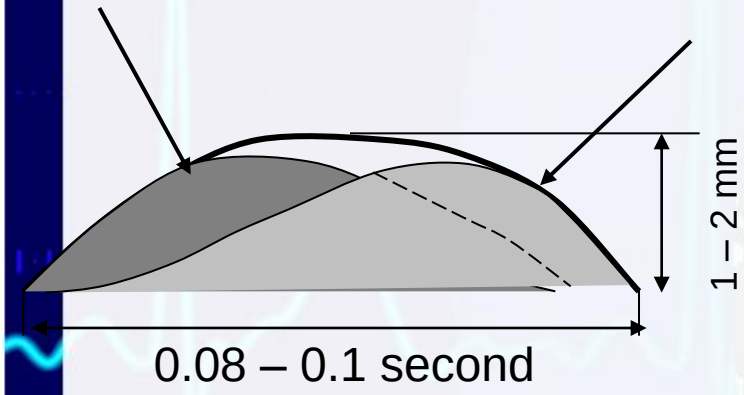
In each cardiac cycle there are two electrical processes: **depolarization and repolarization.**

Depolarization is an electrochemical phenomenon that occurs rapidly.

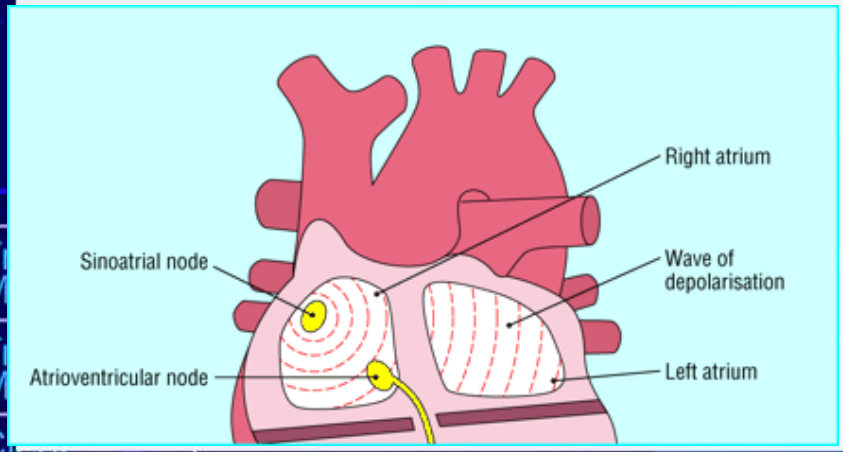
Repolarization is an electrochemical phenomenon of energy restoration that occurs more slowly.

P wave

Right atrium depolarization Left atrium depolarization



The normal P wave formation.

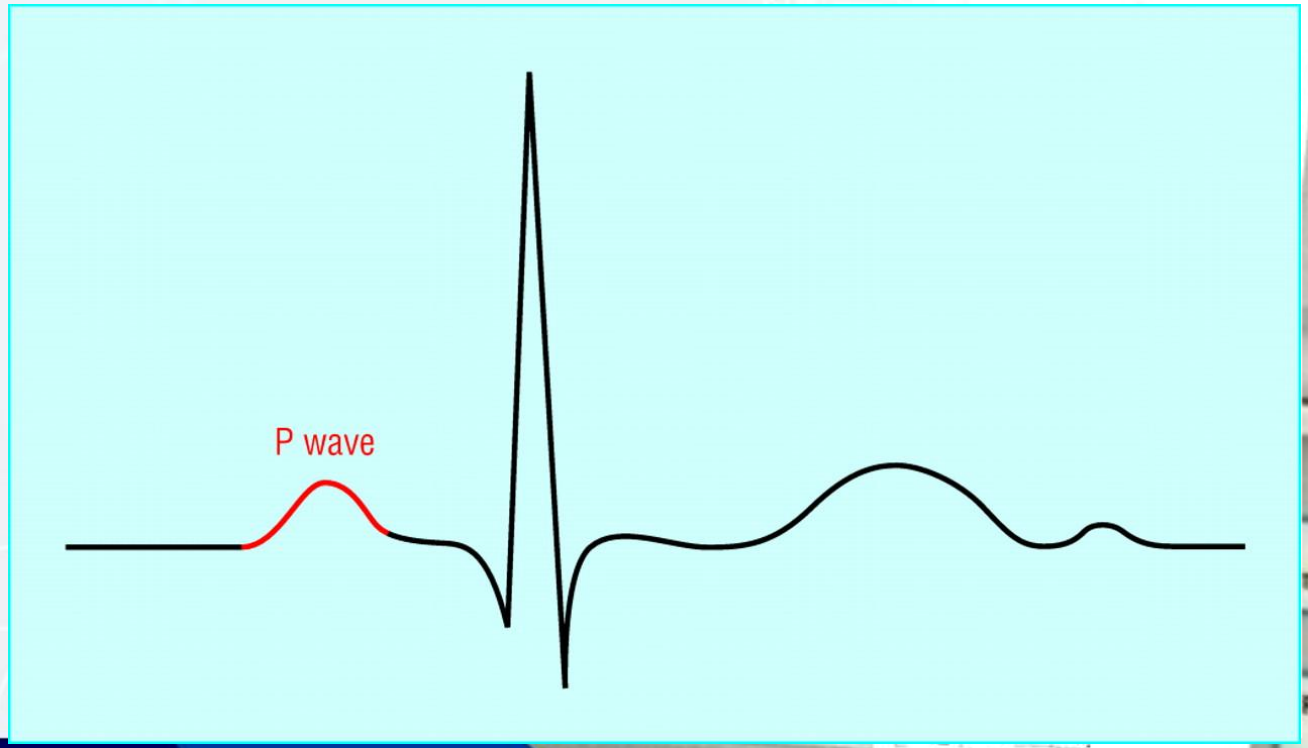


The P wave is the first upward deflection and is the graphic representation of the electrical activation of the atria.

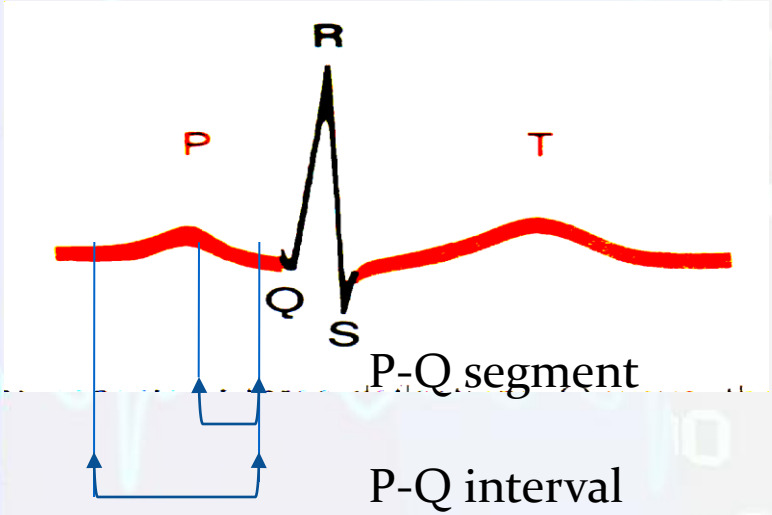
As impulse at first cause excitation of the right atrium and than the left atrium, the ascending portion of P wave reflects depolarization of the right atrium, and descending portion reflects depolarization of the left atrium.

P wave

1. The normal P wave duration is 0.06 – 0.10 second, amplitude - 1-2 mm.
2. The normal P wave is upright in the I, II, aVF, V_2 – V_6 leads.
3. P wave may be upright, two-phased in III, aVL, V_1 leads, and sometimes even inverted in the III and aVL leads.
4. P wave is always inverted in the aVR lead.



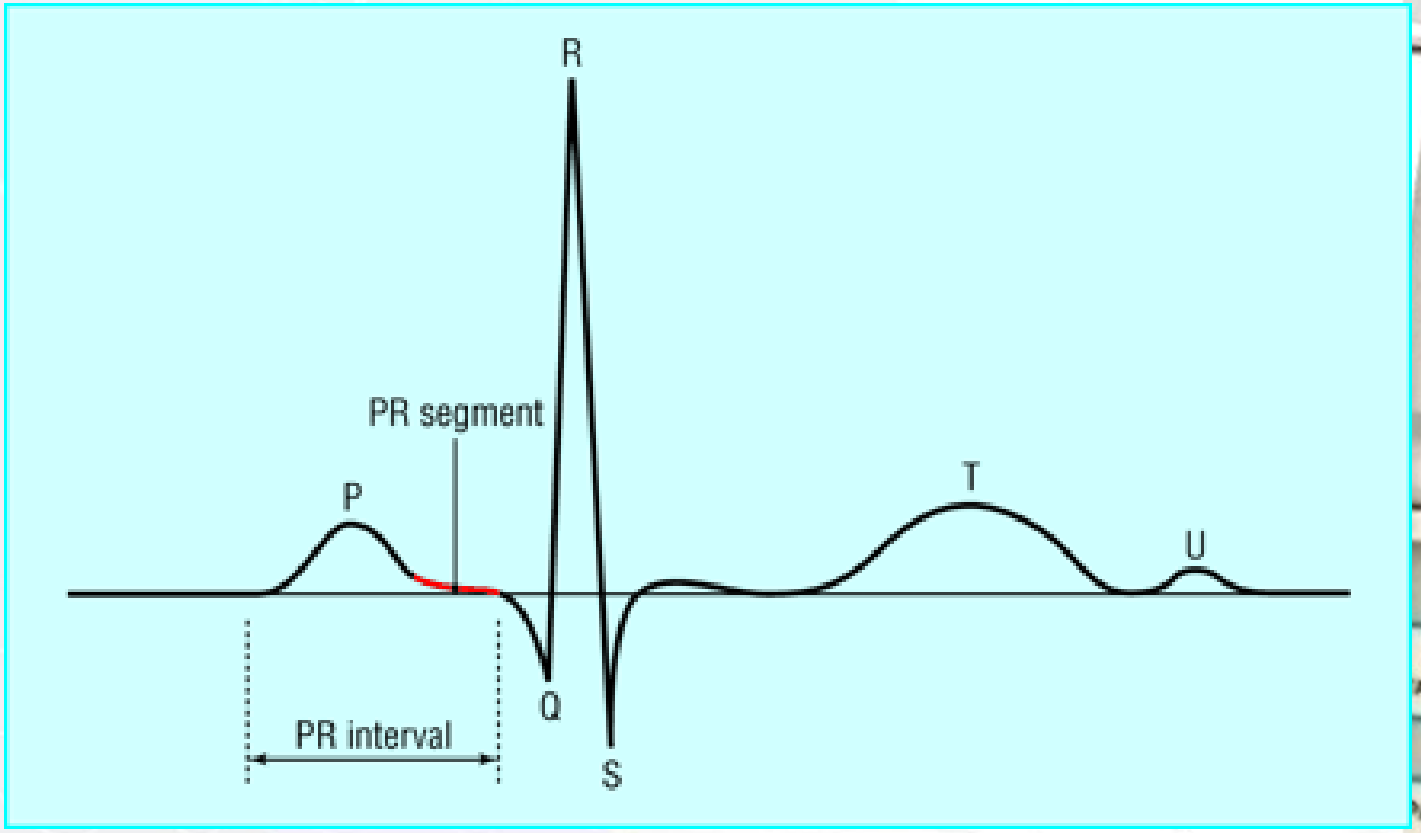
P – Q interval



The P–Q interval measured from the beginning of the P wave to the onset of the Q (or R) wave. Includes activation in atria, the AV node,

The normal P – Q duration is 0.12 – 0.2 second.

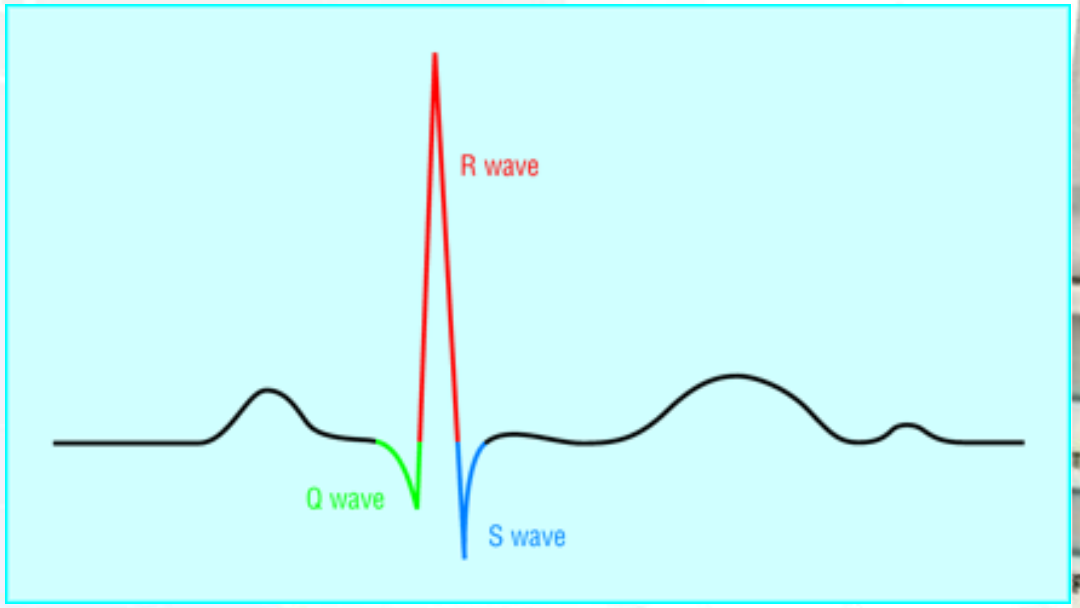
The P – Q segment, measured from the end of the P wave to the onset of the Q wave, represents mostly the delay in activation as the impulse passes through the AV node.



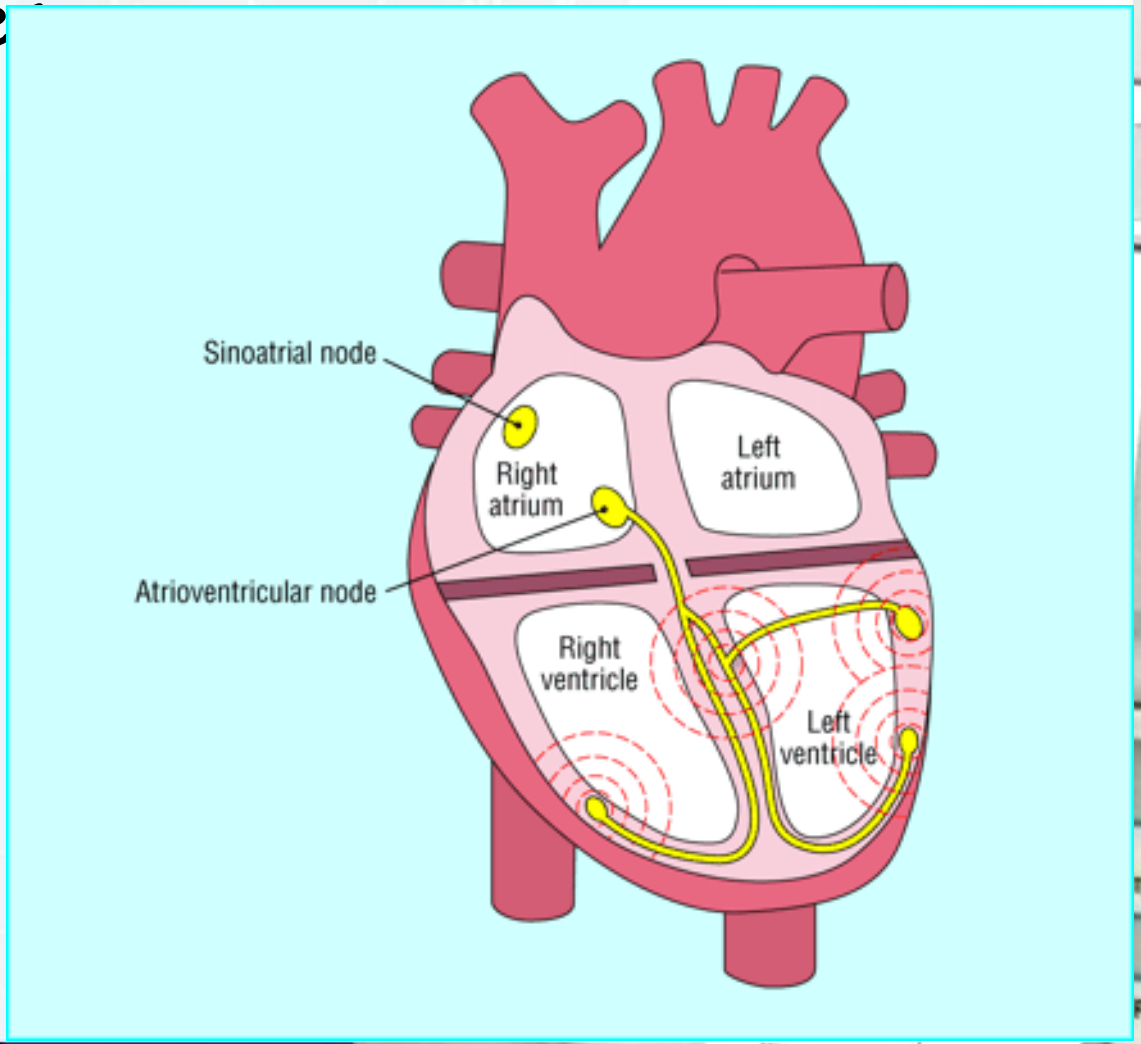
Ventricular complex QRS

Q wave represents depolarization of the interventricular septum.

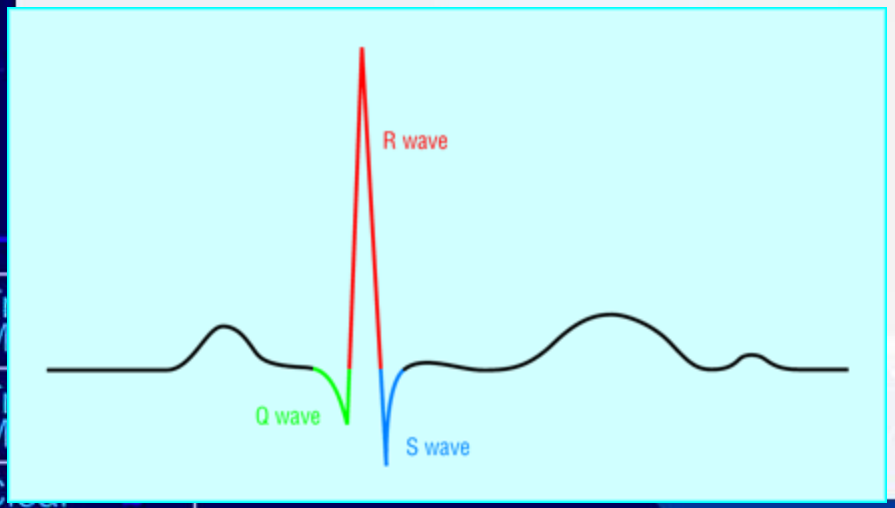
- The normal Q wave amplitude in all leads except aVR is not more than $\frac{1}{4}$ of the R wave amplitude, and its duration is 0.03 second.
- The normal Q wave is inverted in I, II, III, aVL, aVF, V_4 – V_6 leads.
- Q wave may be deep and wide in the aVR lead, or even QS complex may register.



R wave represents the process of depolarization of the ventricle



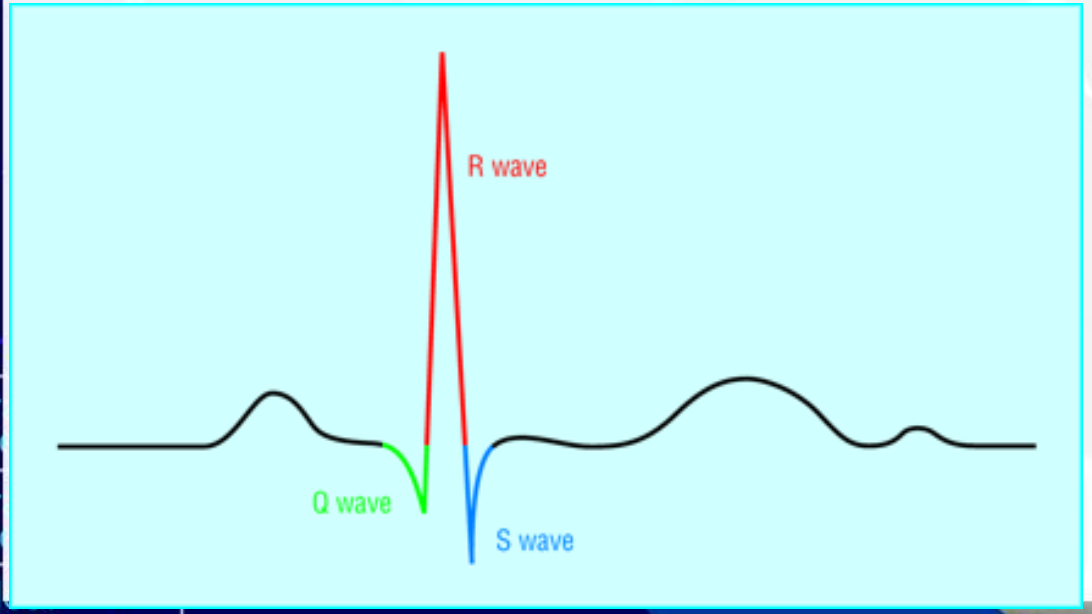
R wave



1. The normal R wave amplitude is 5-15 mm
2. The normal R wave is recorded in all standard and augmented limb leads. In the aVR lead R wave may be low or even absent.
3. In the chest leads R wave amplitude increases from V_1 to V_4, and than slightly decreases in V_5 and V_6 leads.
4. $R_{V1,V2}$ reflects activation of the interventricular septum, and R wave in V_4, V_5, V_6 leads activation of the right and left ventricles.

S wave

S wave formation on the ECG reflects depolarization of the basal parts of interventricular septum of right and left ventricles.



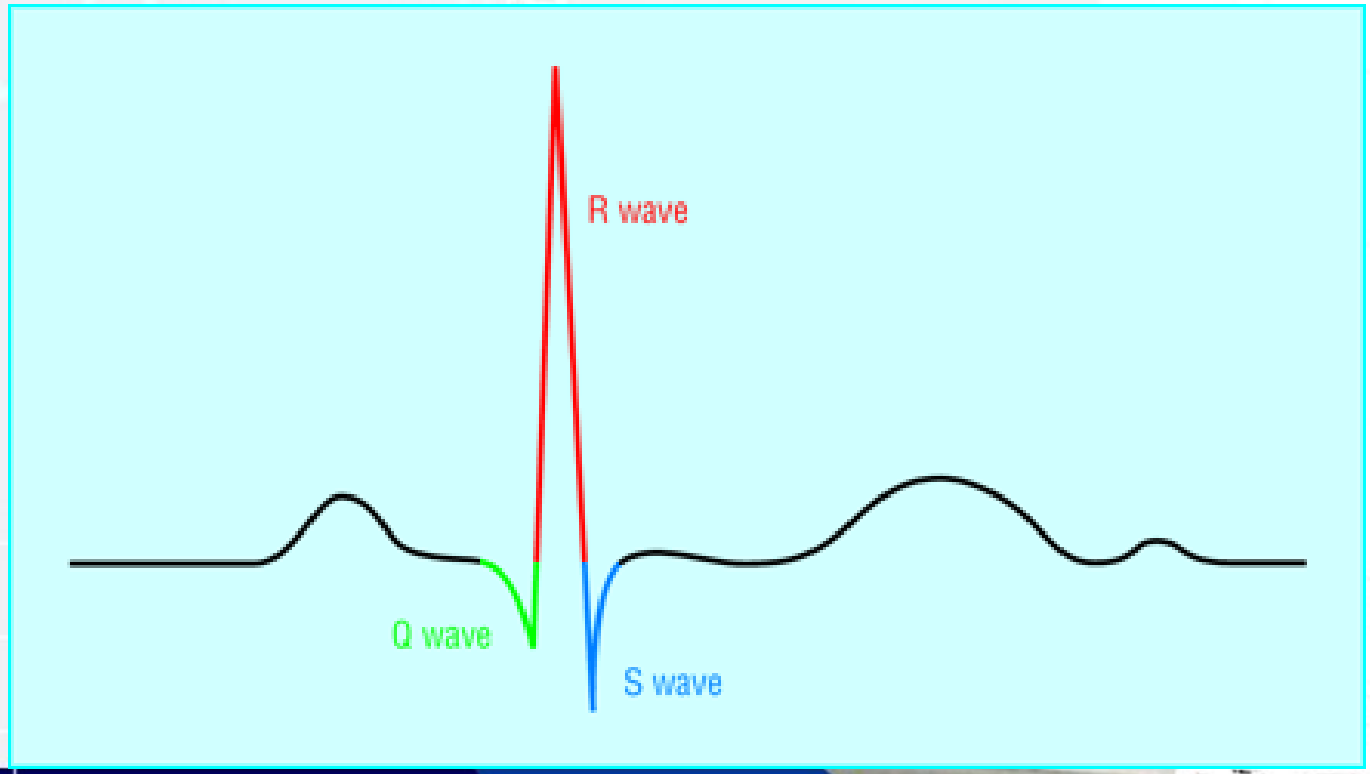
S wave

1. The normal S wave is inverted, its amplitude in the different electrocardiographic lead is within the large ranges, not exceed 20 mm (2.5 mm on the average).

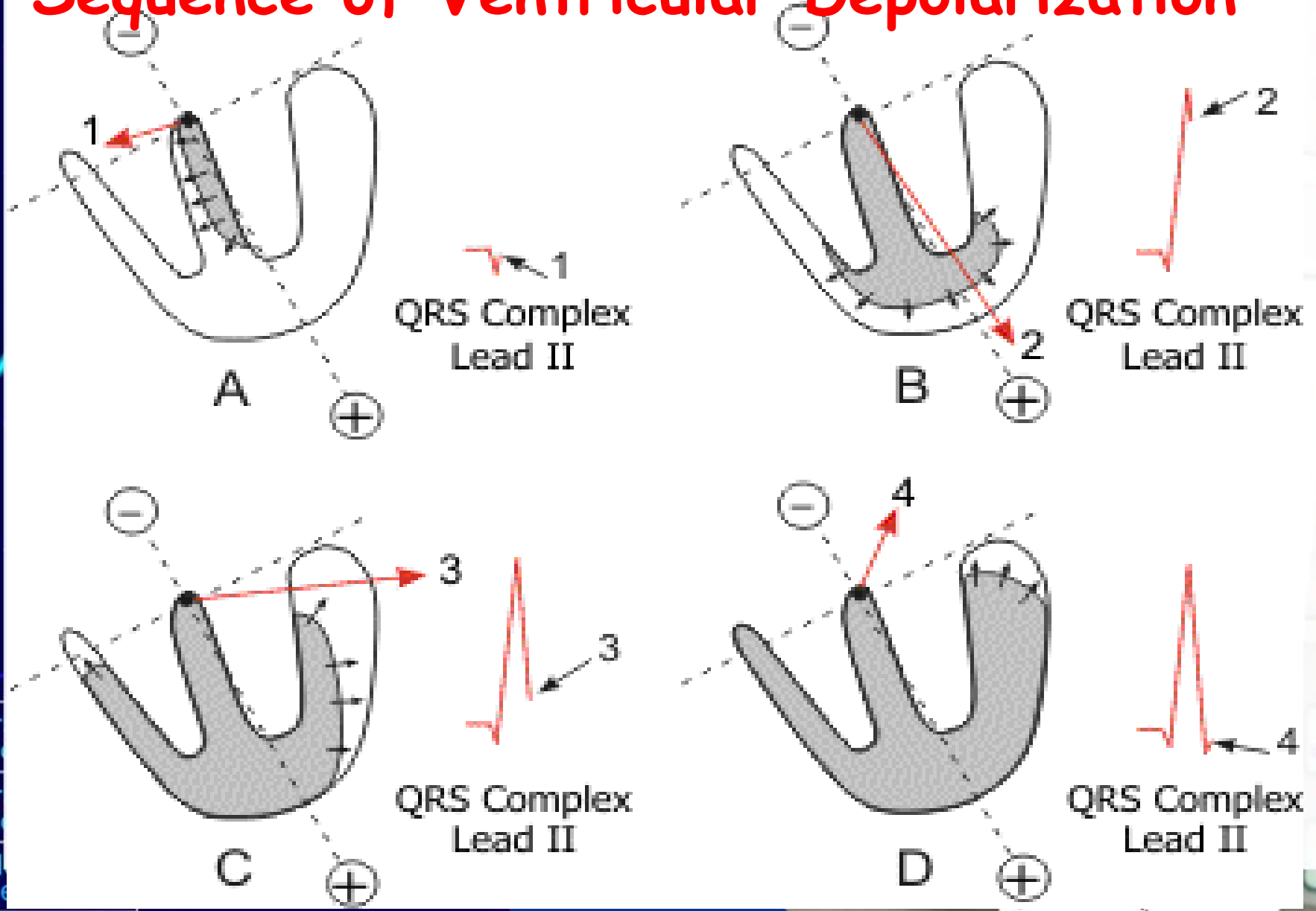
2. In the limb lids S wave amplitude is low, except aVR lead, in the normal position of the heart in the chest.

3. In the chest leads S wave amplitude decreases from V_1, V_2 to V_4, and in V_5, V_6 leads S wave amplitude is very low or S wave may be even absent.
4. Equal S wave and R wave amplitude in the chest leads are usually in the V_3 ("transition zone") or (rarer) between V_2 and V_3 or V_3 and V_4.

The QRS interval, measured from the beginning of the Q wave to the end of the S wave, represents the process of depolarization of the ventricles. The normal duration of the QRS interval is 0.06 – 0.1 sec., this time corresponds to the intraventricular conduction.

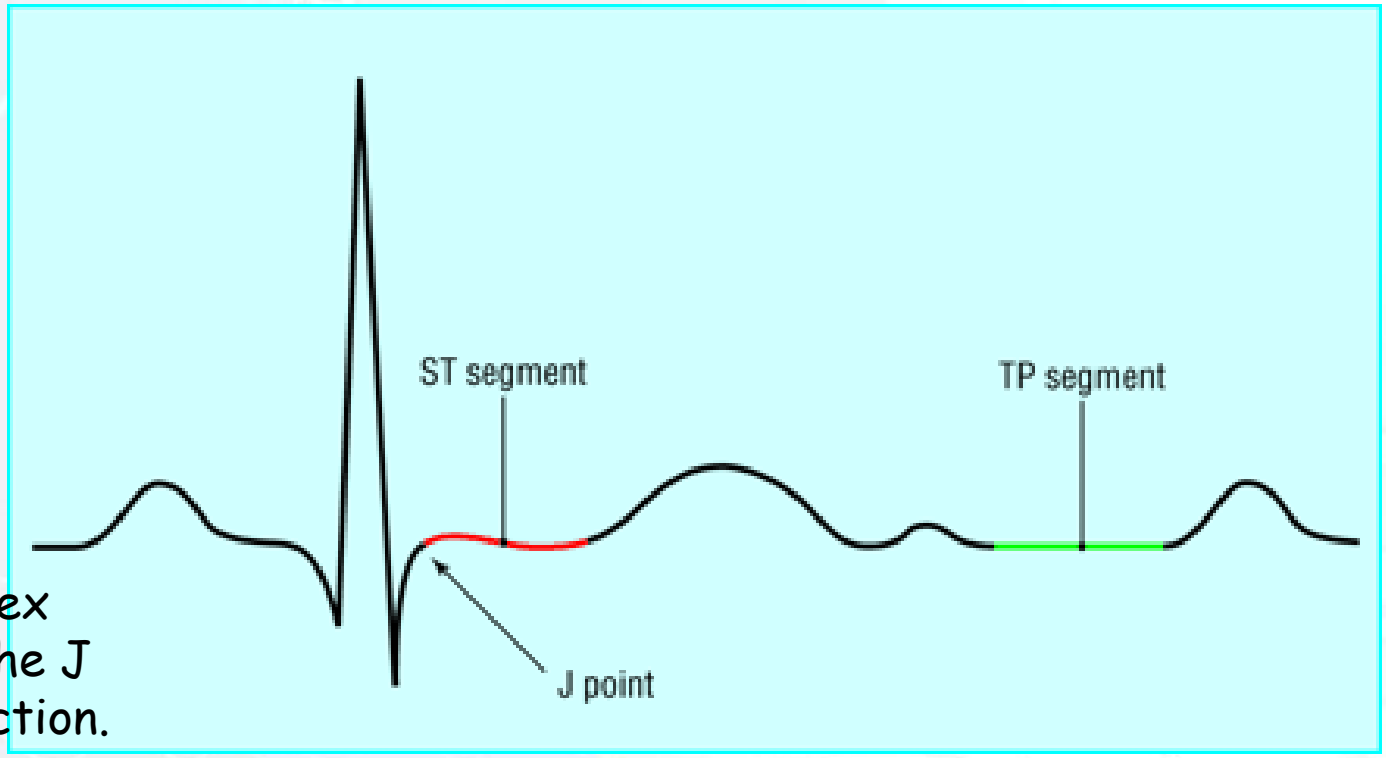


Sequence of Ventricular Depolarization



The ST segment

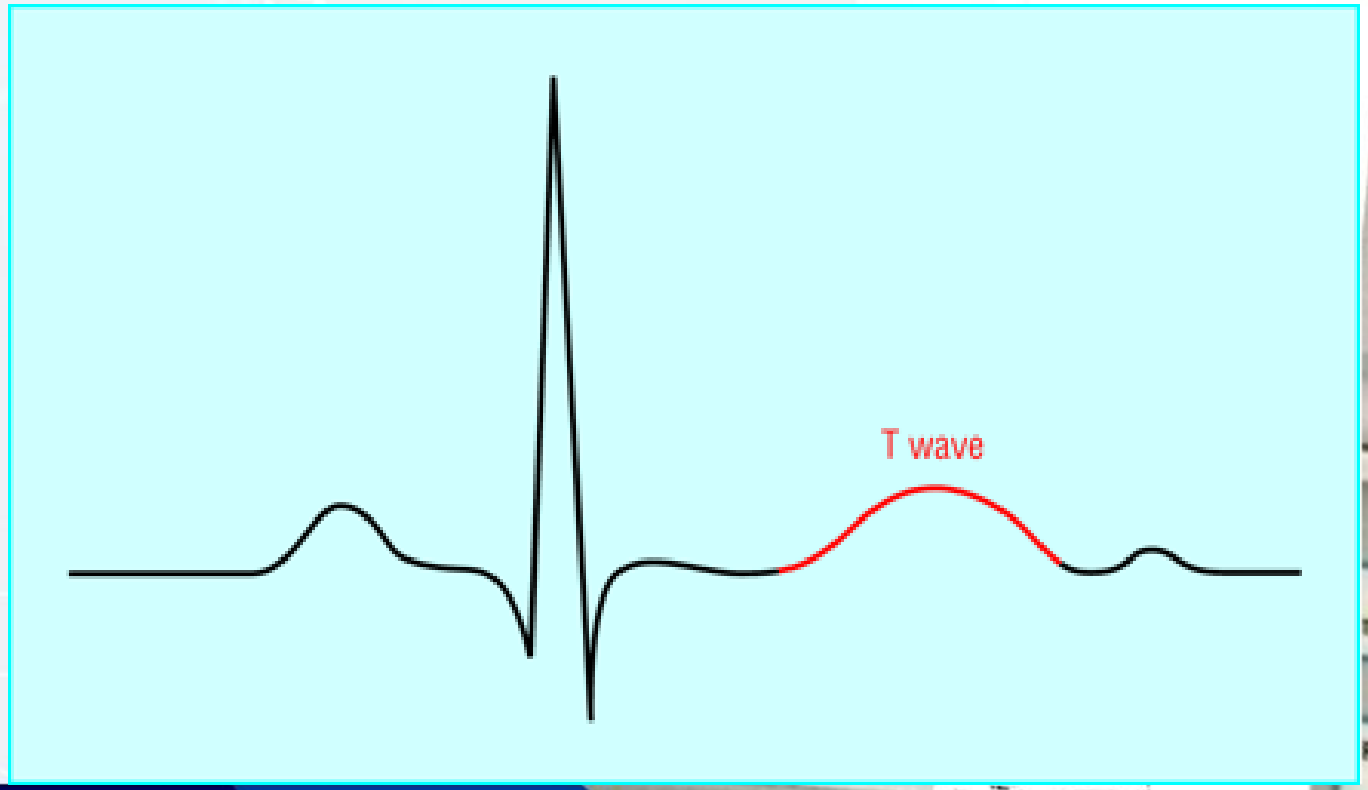
- represents the period when all parts of the ventricles are in the depolarized state. The ST segment duration depends on the heart rate.



The QRS complex terminates at the J point or ST junction.

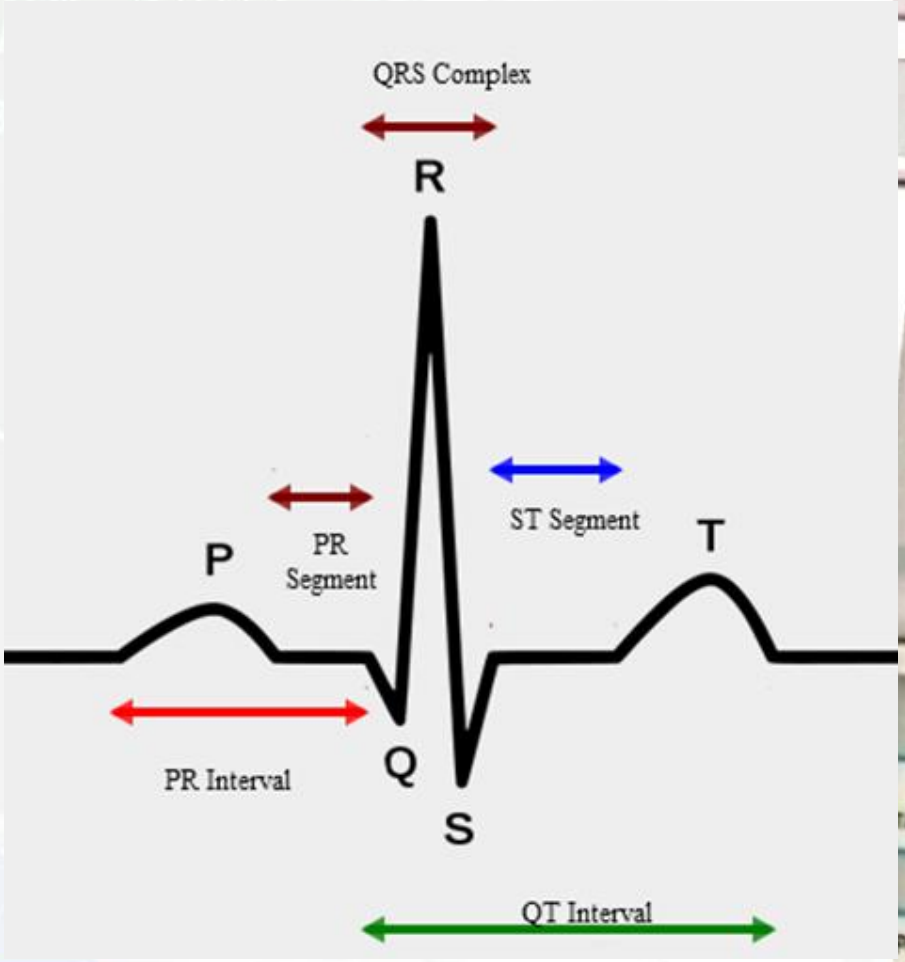
T wave represents repolarization of both ventricles.

The normal T wave is asymmetric: the gradual ascent converts into a rounded summit, which is followed by abrupt descent.



T wave

1. The normal T wave duration is 0.12 – 0.16 second, amplitude – 2.5 – 6 mm.
2. The normal T wave is always upright in I, II, aVF, V_2 – V_6, $T_I > T_{III}$, and $T_{V6} > T_{V1}$ leads.
3. T wave may be upright, two-phased or inverted in III, aVL and V_1 leads.
4. The normal T wave is always inverted in aVR lead.



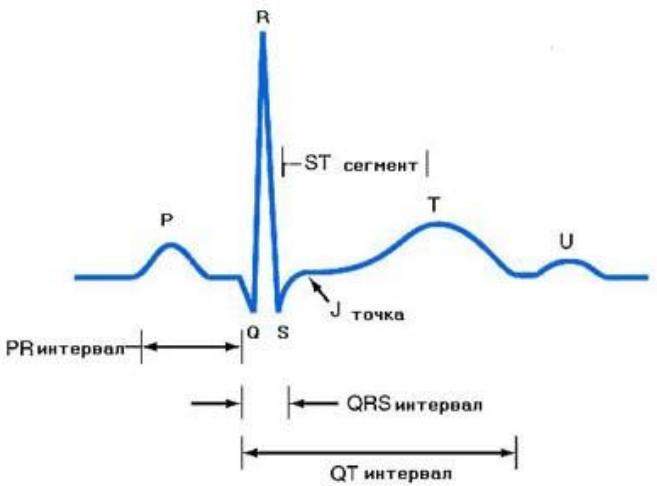
Q – T interval **(QRST complex)**, measured from the beginning of the Q (or R) wave to the end of the T wave, represents electrical ventricular **systole**.

Its duration depends on the cardiac rate.

The Q – T interval in women is longer than in man.

For example, at the rate of 60 – 80 beats per minute, Q – T duration is 0.32 – 0.37 second in men,

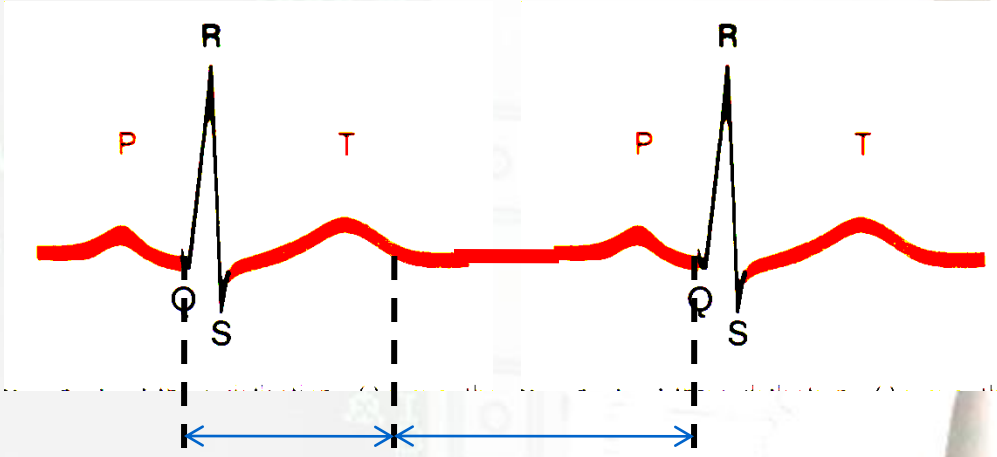
and 0.35 – 0.4 in women.



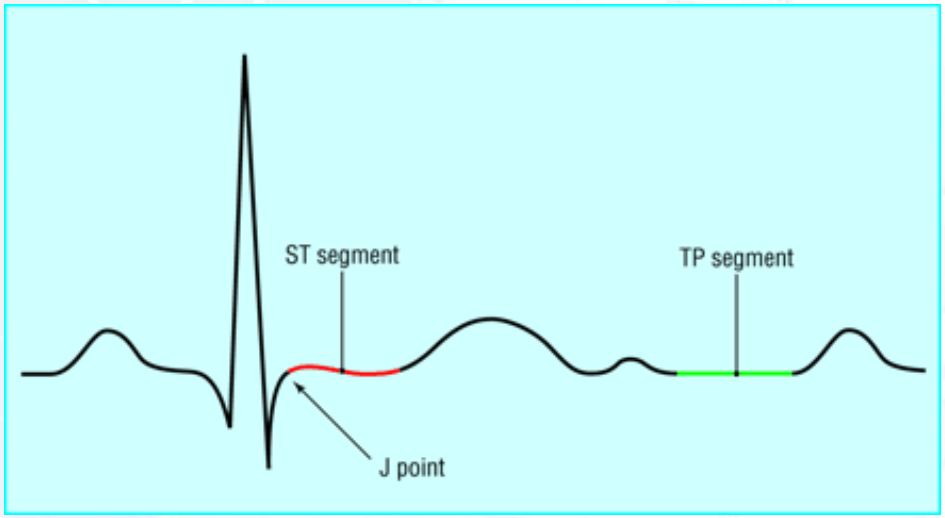
T – P interval,

measured from the end of the T wave to the beginning of the P wave, represents electrical **diastole** of the heart.

Its duration depends on the heart rate .



Electrical systole (QRST) Electrical diastole (T – P)



Interpretation of the ECG

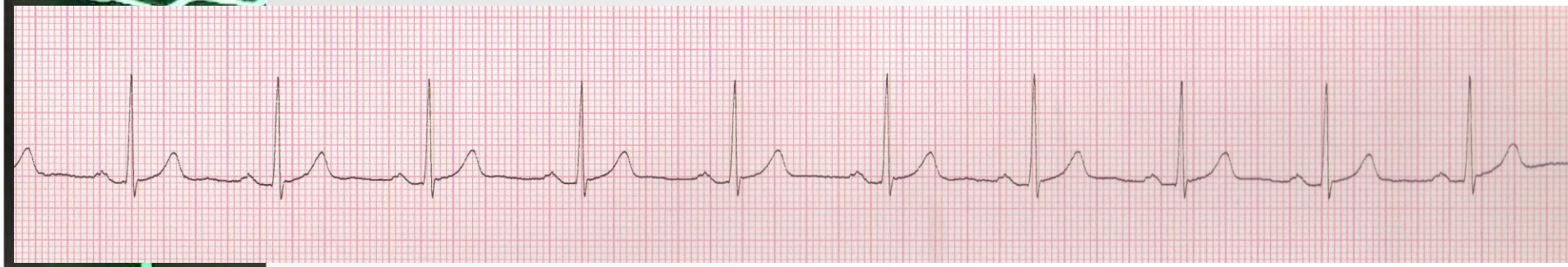
1. Determination of the Cardiac Rhythm Regularity (regular, irregular).
2. Determination of the Cardiac Rhythm Pacemaker Site (sinus, nonsinus).
3. Measurement of the ECG Amplitude.
4. Calculation of the Heart Rate.
5. Determination of the Electrical Axis of the Heart.
6. Measurement of the duration and amplitude of the ECG waves and intervals.
7. ECG conclusion

Interpretation of the ECG

1. **Determination of the Cardiac Rhythm Regularity.** The R-R intervals in regular rhythm should be equal. Its fluctuation normally does not exceed 0.1 second (±10%). Greater variations in the R-R intervals duration indicate irregular cardiac rhythm.

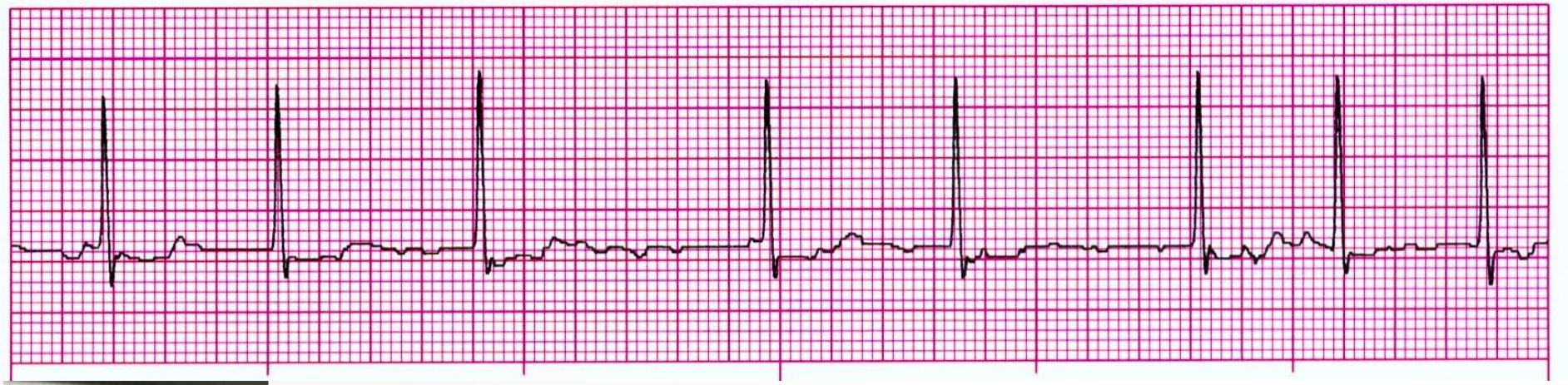
Regular cardiac rhythm

1. The R-R intervals are equal;
2. Its fluctuation does not exceed 0.1 second (±10%).



Irregular cardiac rhythm

1. The R-R intervals are different;
2. Its fluctuation exceed 0.1 second (±10%).



Determination of the Cardiac Rhythm Regularity

- Compare regular and irregular cardiac rhythm

ECG tracing of a normal heart rhythm.

In atrial fibrillation, the tracing shows tiny, irregular "fibrillation" waves between heartbeats. The rhythm is irregular and erratic.

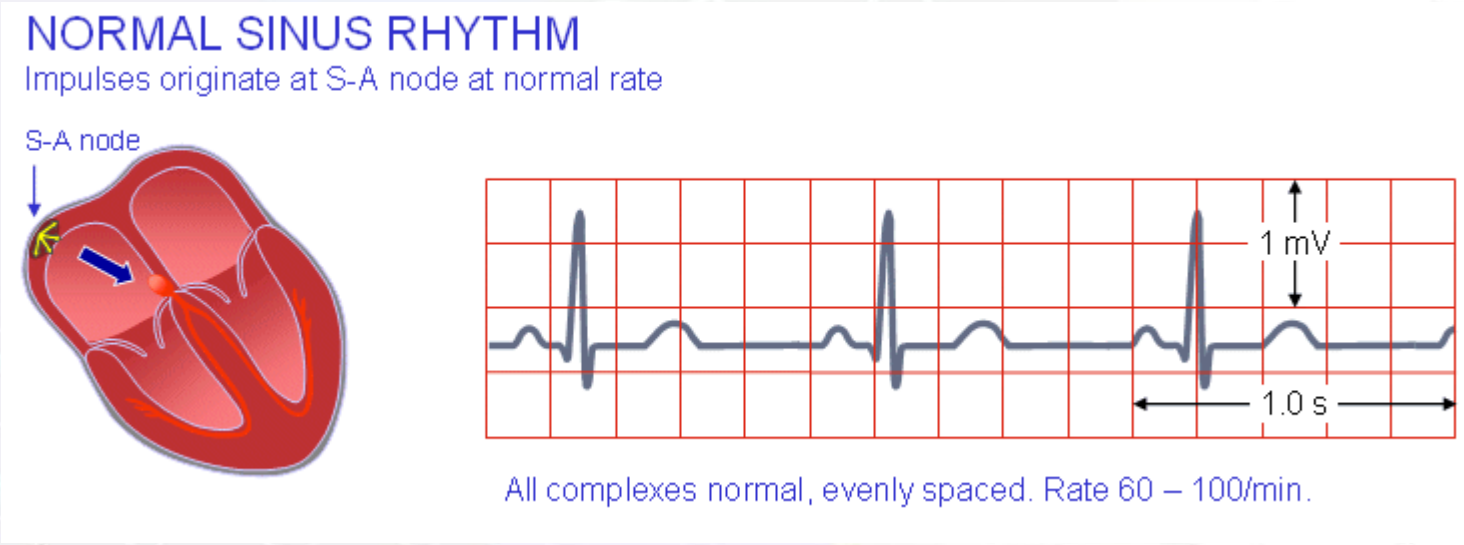
2. Determination of the Cardiac Rhythm Pacemaker Site

Sinus rhythm criteria

1.The normal sinus rhythm is characterized by upright P wave in lead II following by QRS complex;

2. The P waves configuration is equal in each lead;

3. Heart rate is 60-80 beats per min.



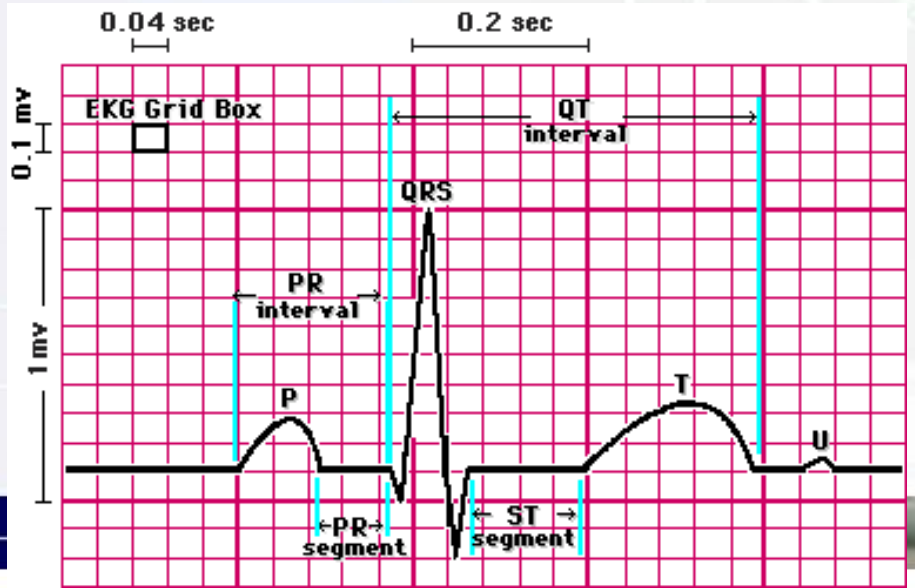
2. Determination of the Cardiac Rhythm Pacemaker Site

Nonsinus rhythm

1. *Atrial rhythm;*
2. *Junctional rhythm;*
3. *Ventricular rhythm;*
4. *Wandering pacemaker;*
5. *Pacer rhythm*

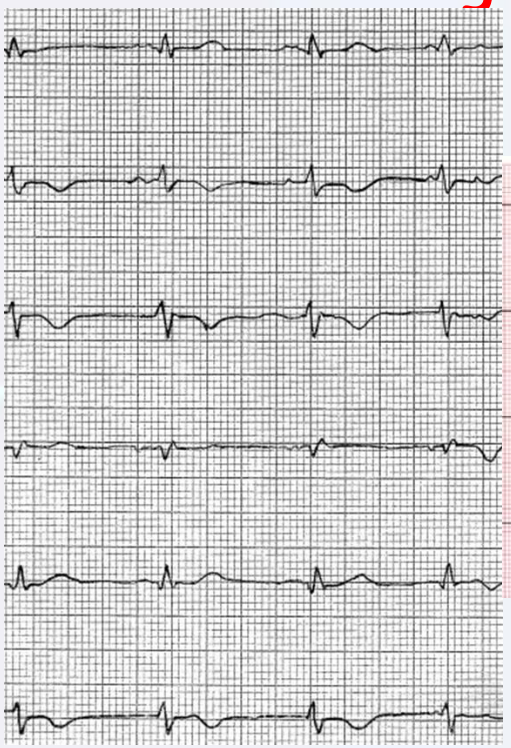
3. Measurement of the ECG Amplitude (voltage).

The R waves amplitude are measured in standard leads. If the amplitude of the highest R wave does not exceed 5 mm, or the sum of the R waves amplitude in the three standard leads is less than 15 mm the ECG voltage is considered decreased.



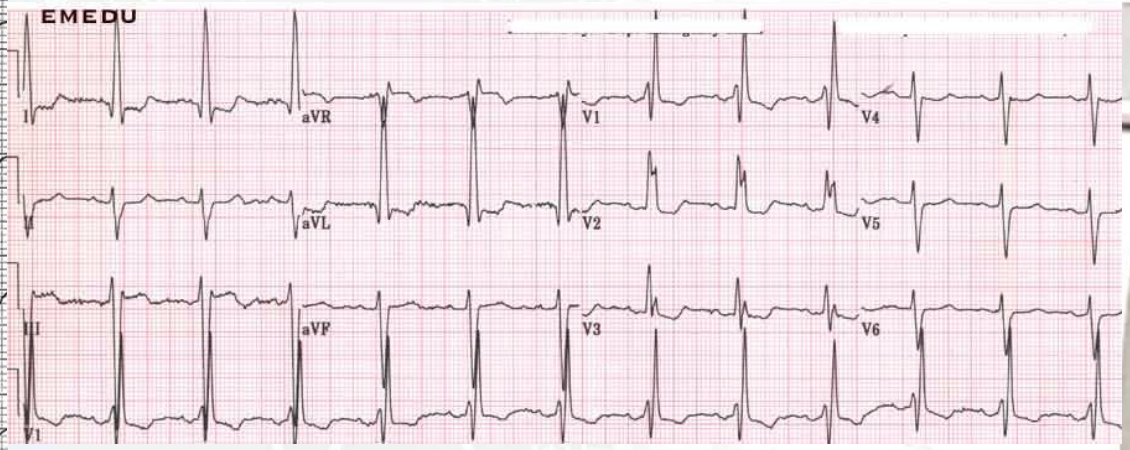
3. Measurement of the ECG Amplitude (voltage)

Low ECG voltage

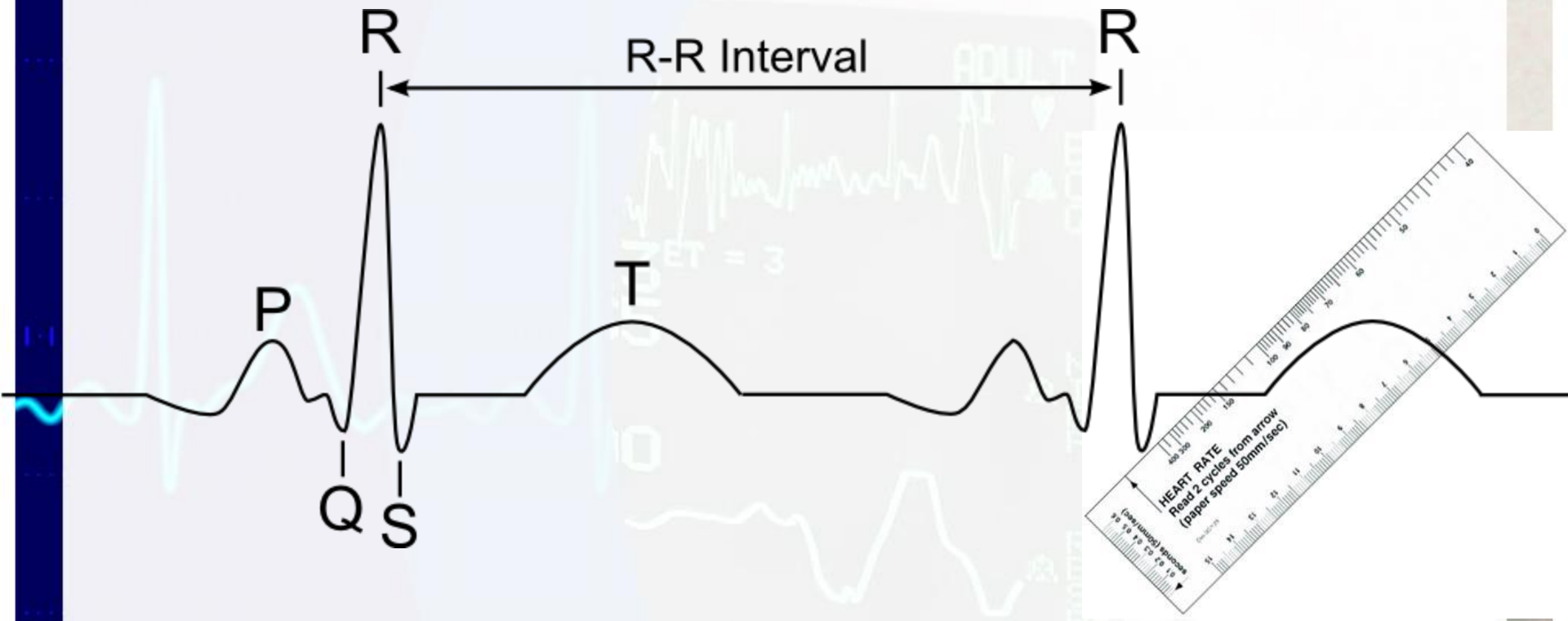


- MI
- Myocarditis
- Pericarditis
- Cardiosclerosis

High ECG voltage



- Ventricular hypertrophy
- His bundle branch block



4. Calculation of the Heart Rate. In regular cardiac rhythm the heart rate is determined by formula:

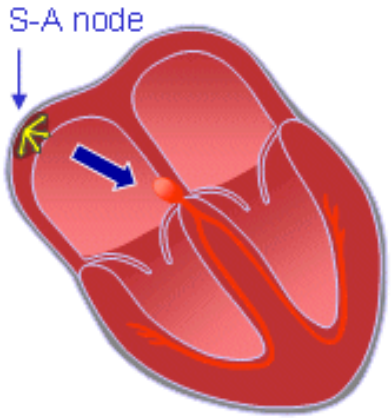
$$HR = \frac{60}{R-R}$$; where 60 is a number of seconds in minute,

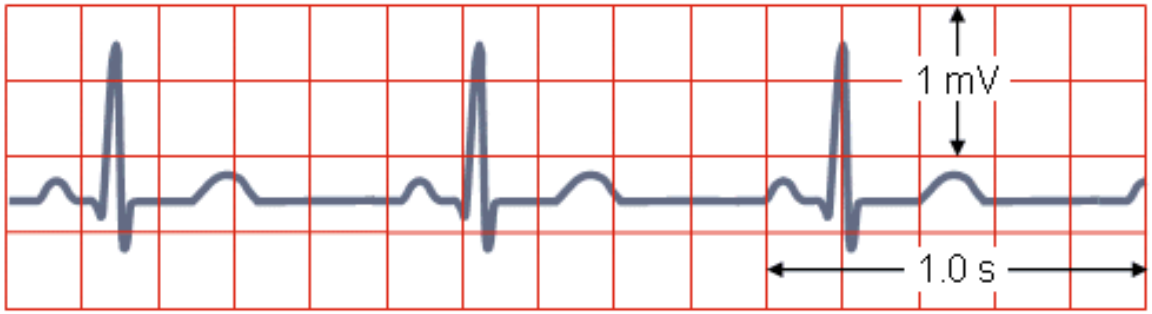
R-R – duration of the R-R intervals in seconds.

Normal heart rate

NORMAL SINUS RHYTHM

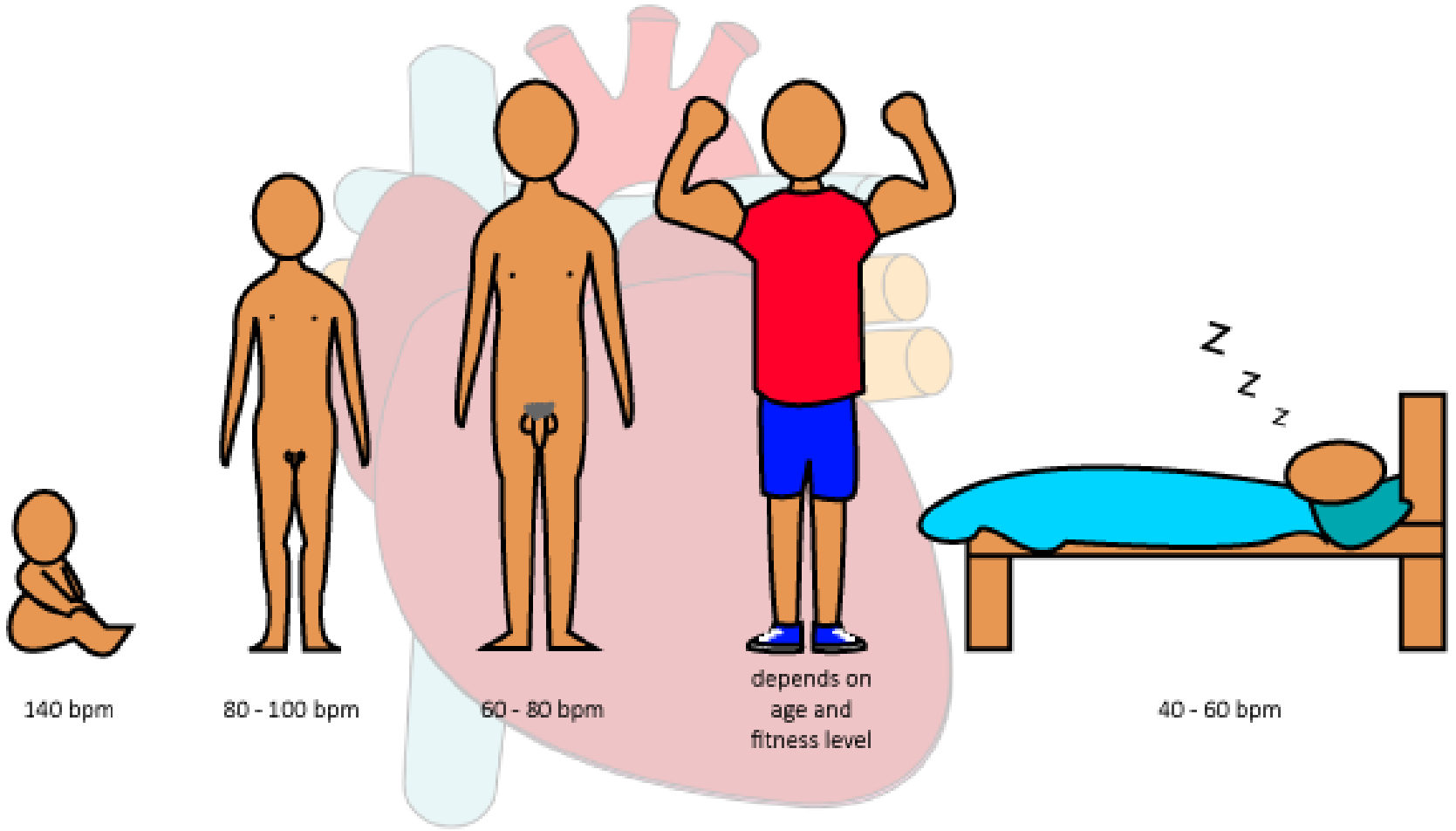
Impulses originate at S-A node at normal rate





All complexes normal, evenly spaced. Rate 60 – 100/min.

Beat per minute (bpm)

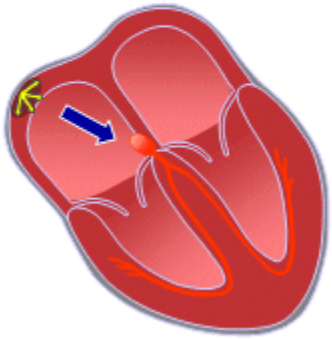


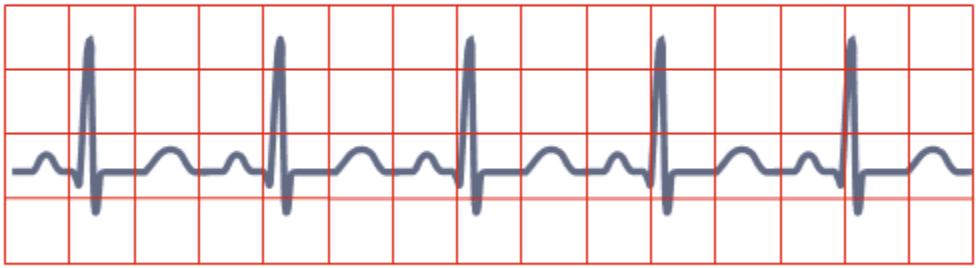
Sinus tachycardia

A sinus rhythm of higher than 90/min is called sinus tachycardia. It occurs as a physiological response to physical exercise or psychical stress, but may also result from heart failure.

SINUS TACHYCARDIA

Impulses originate at S-A node at rapid rate





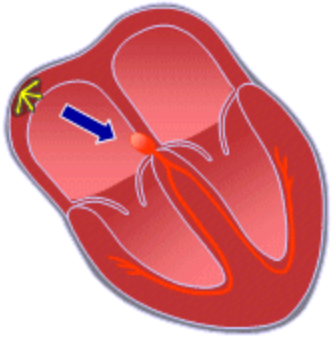
All complexes normal, evenly spaced. Rate >100/min.

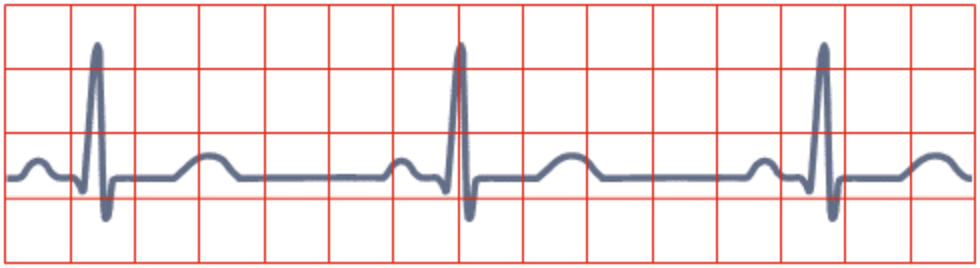
Bradycardia

- A sinus rhythm of less than 60/min is called sinus bradycardia. This may be a consequence of increased vagal or parasympathetic tone.

SINUS BRADYCARDIA

Impulses originate at S-A node at slow rate





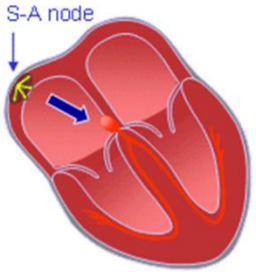
All complexes normal, evenly spaced. Rate < 60/min.

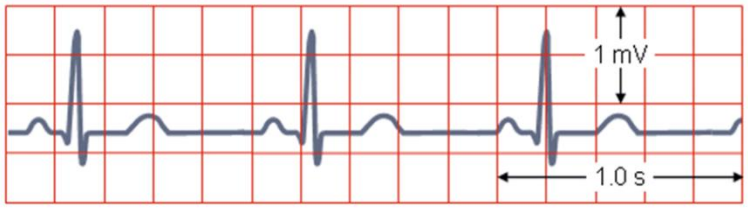
Calculation of the Heart Rate

Compare R-R intervals

NORMAL SINUS RHYTHM

Impulses originate at S-A node at normal rate

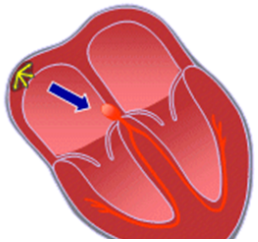


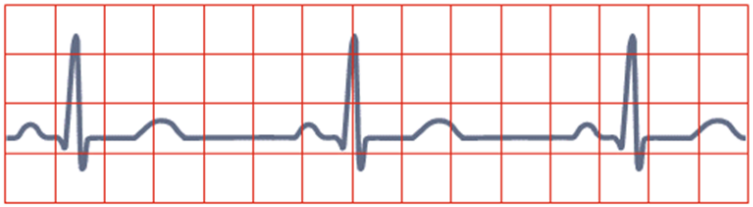


All complexes normal, evenly spaced. Rate 60 – 100/min.

SINUS BRADYCARDIA

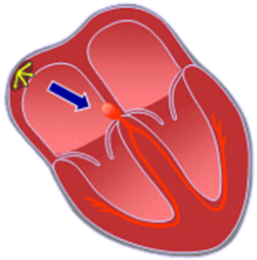
Impulses originate at S-A node at slow rate

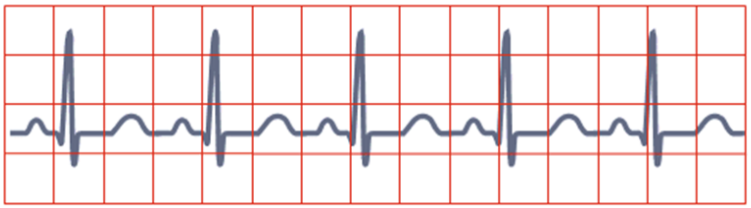




SINUS TACHYCARDIA

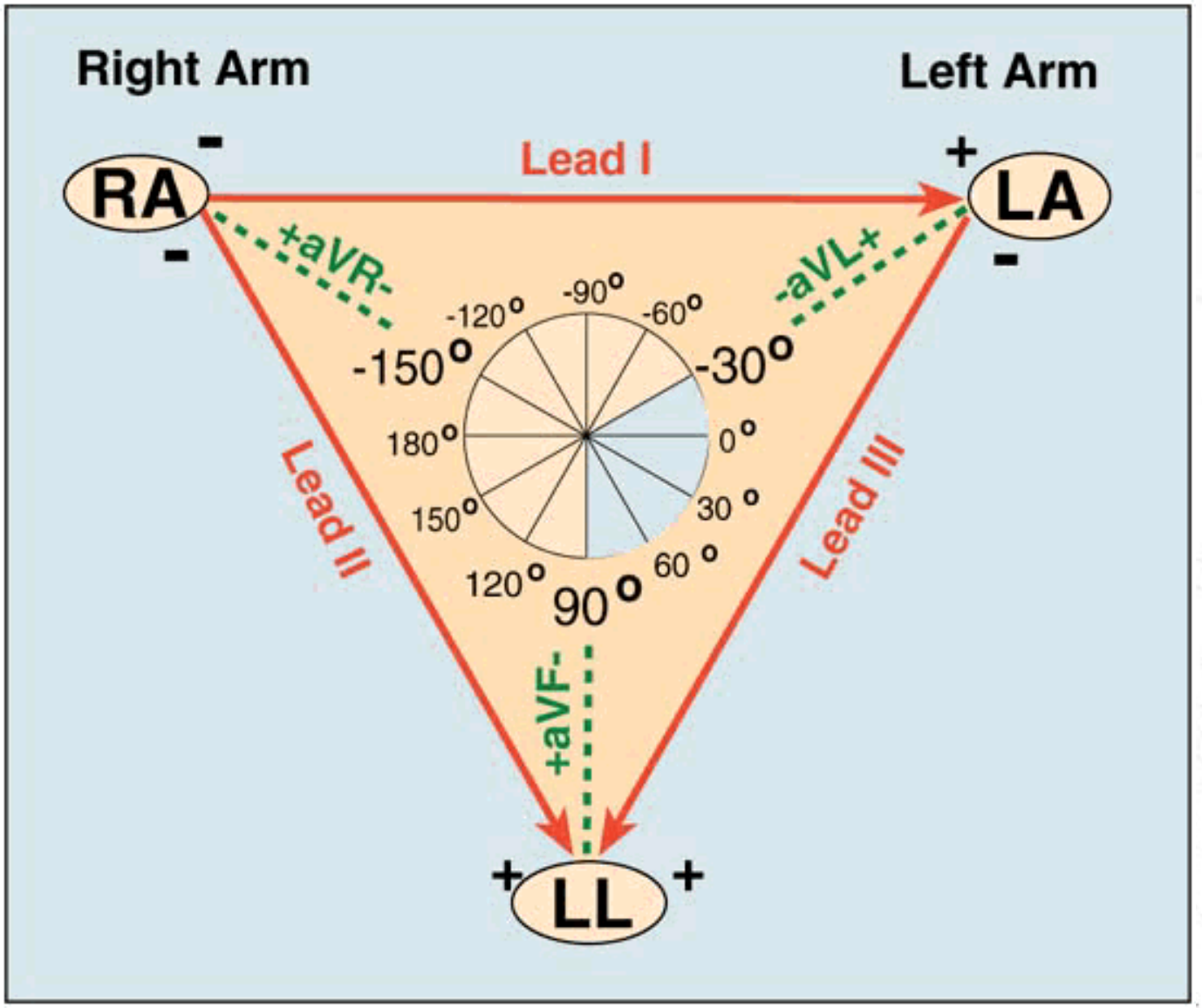
Impulses originate at S-A node at rapid rate





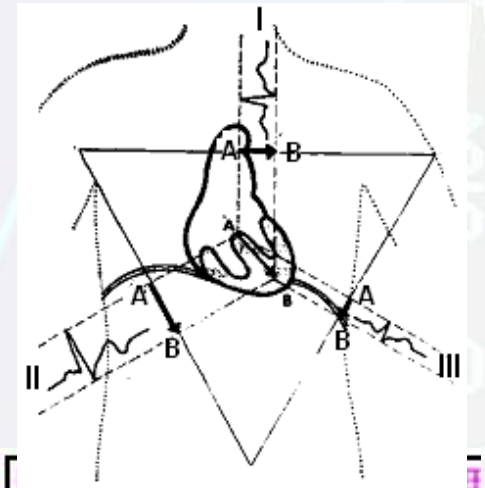
All complexes normal, evenly spaced. Rate >100/min.

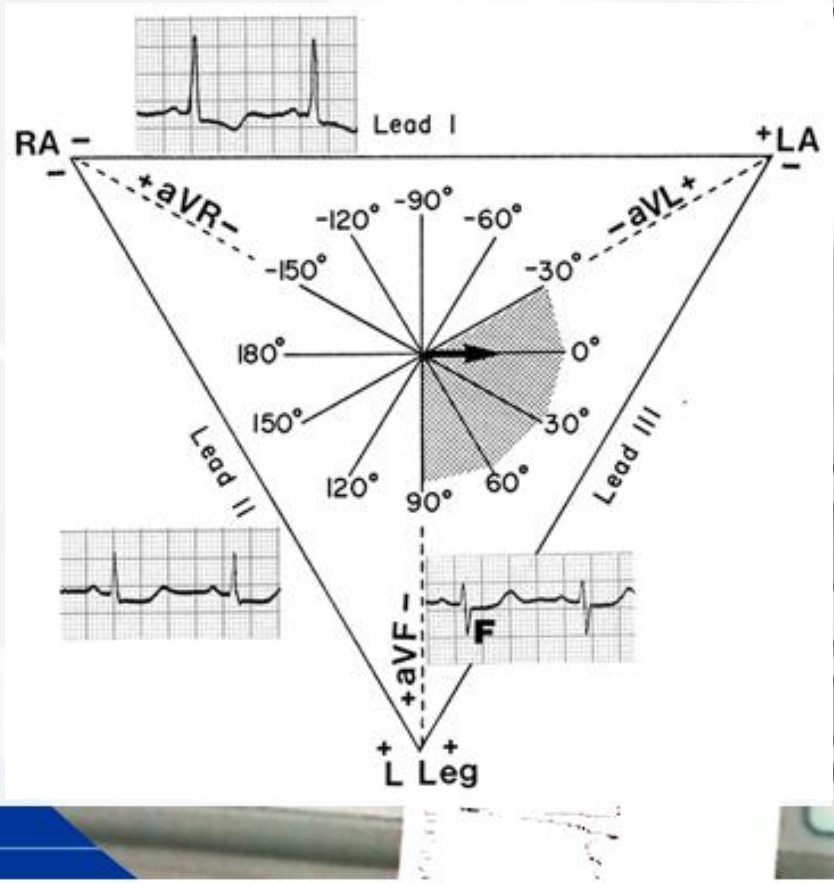
5. Determination of the Electrical Axis of the Heart



5. Determination of the Electrical Axis of the Heart

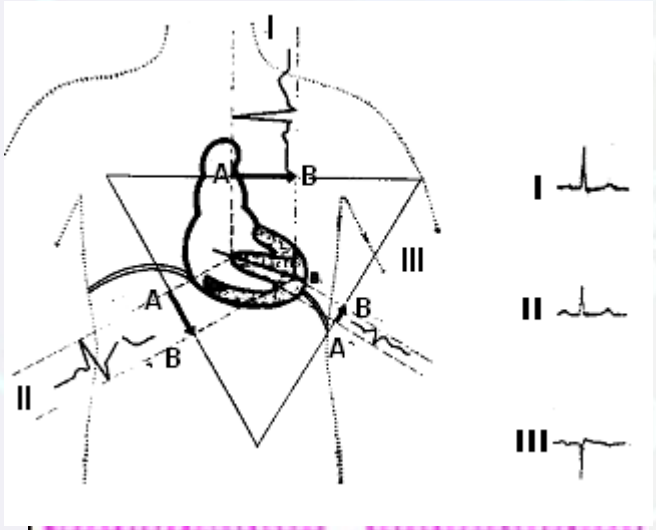
Axis in the normal range

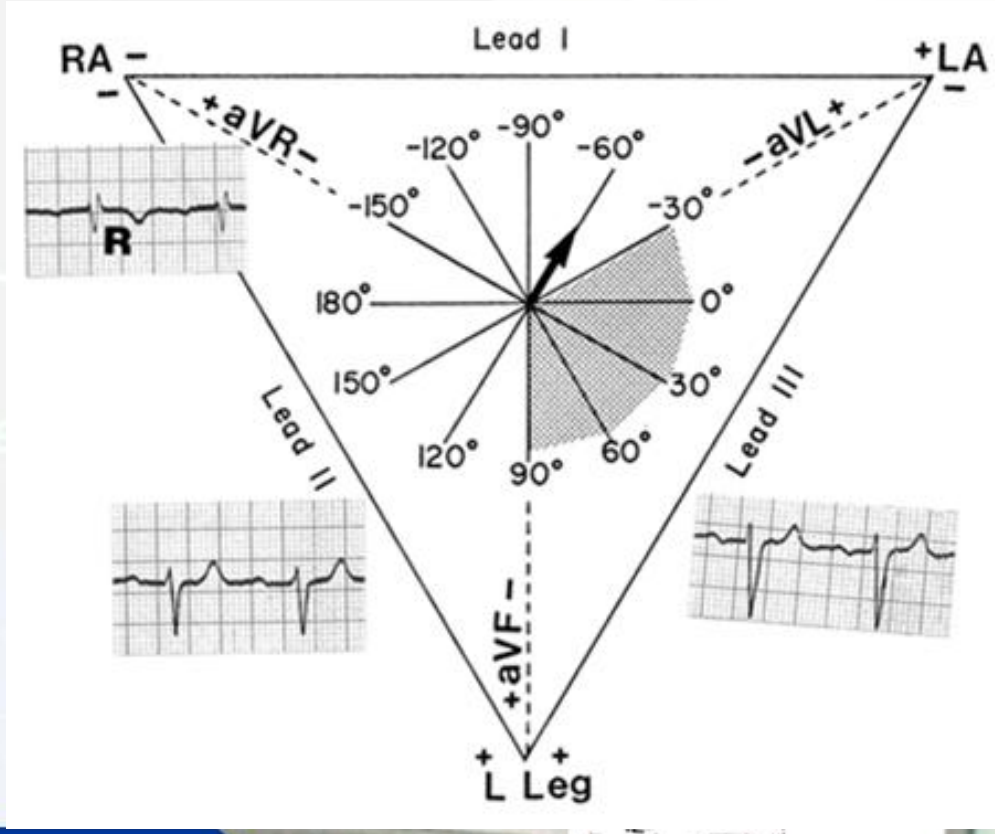




5. Determination of the Electrical Axis of the Heart

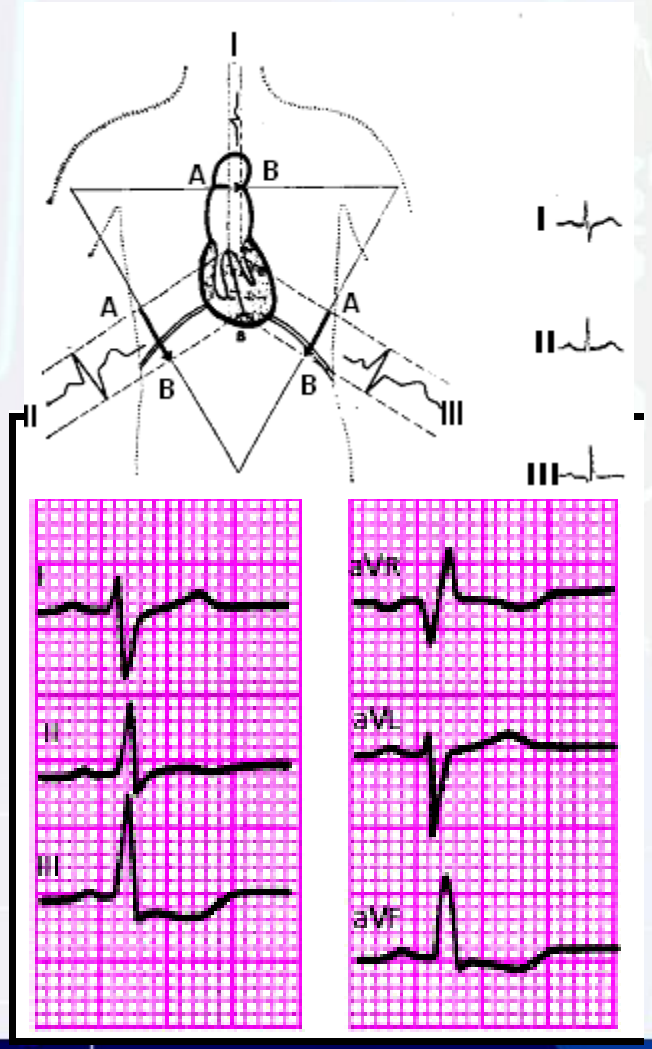
- Left axis deviation (LAD) range

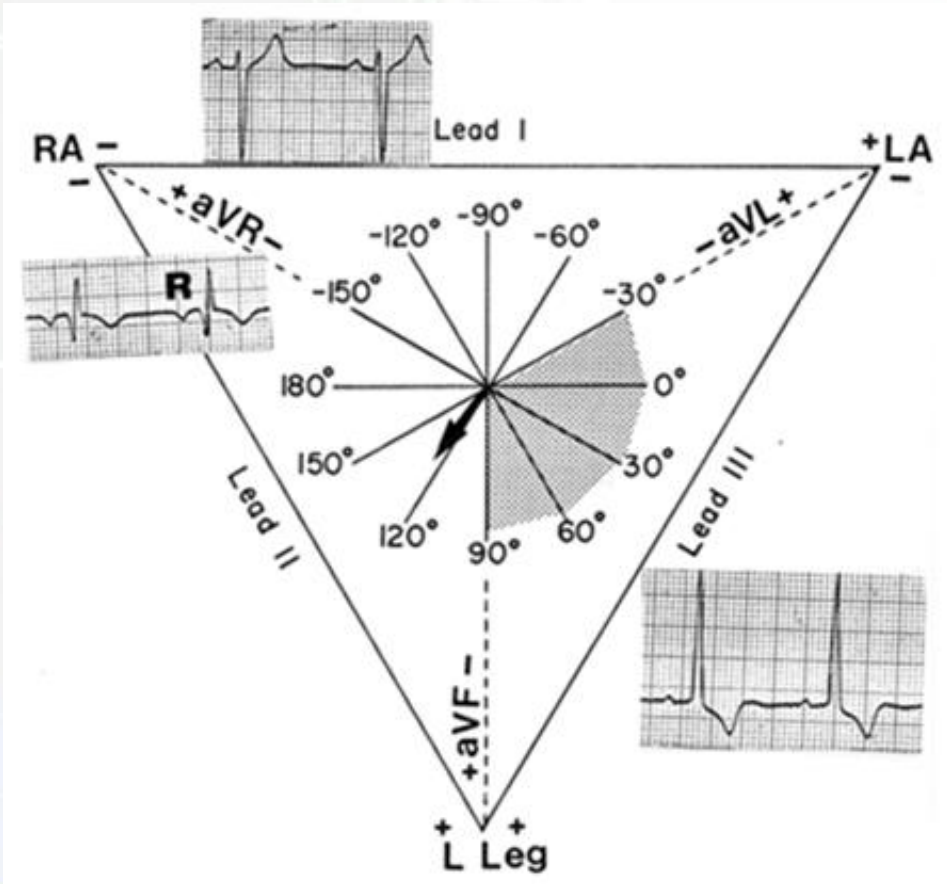




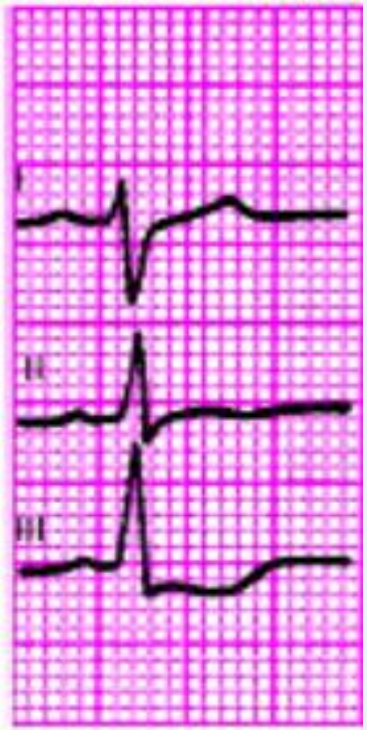
5. Determination of the Electrical Axis of the Heart

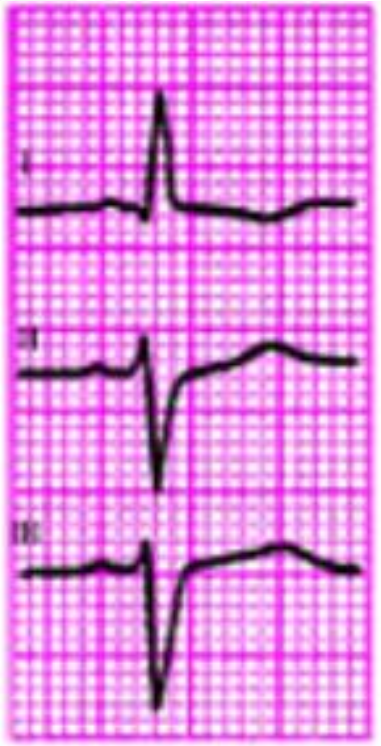
Right axis deviation (RAD) range





Visual method of electrical axis of the heart determination



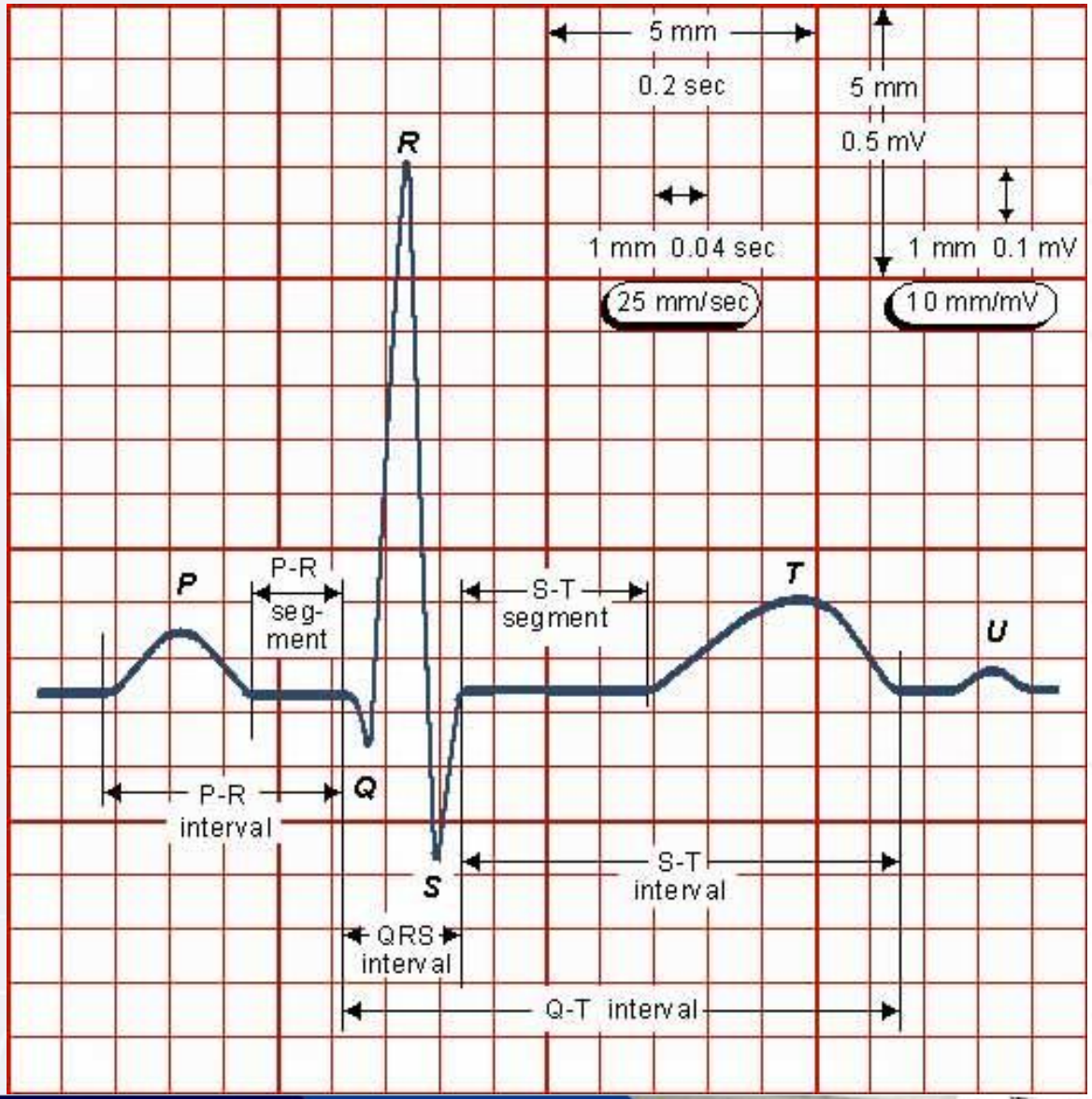


RAD

Normal

LAD

6. Measurement of the duration and amplitude of the ECG waves and intervals.



7. ECG conclusion. In the ECG conclusion it is necessary to note following:

1.Regularity of the cardiac rhythm (regular or irregular);

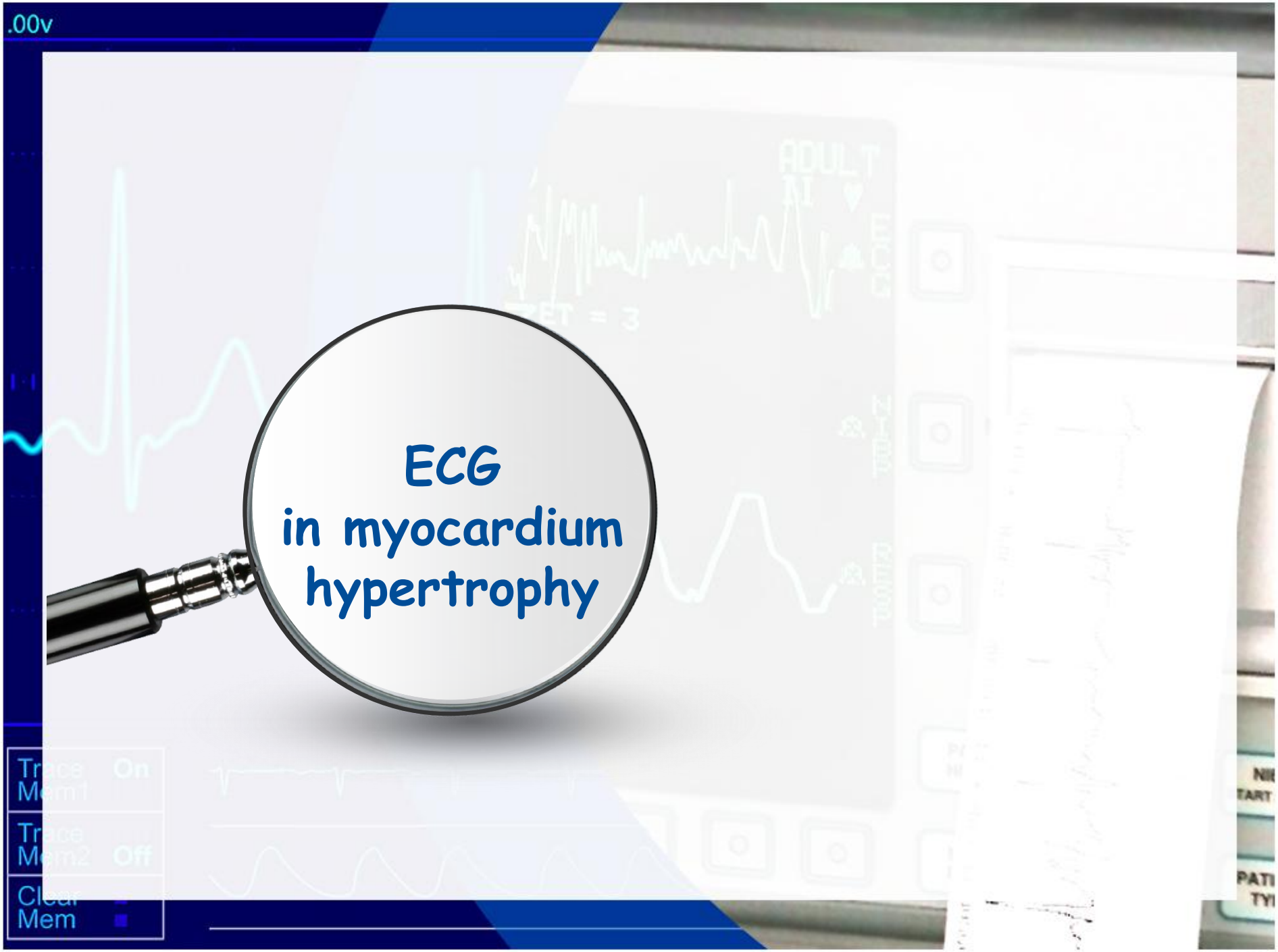
2.The cardiac rhythm pacemaker (sinus or nonsinus rhythm);

3.The heart rate;

4.Position of the electrical axis of the heart;

5.Presence of the four ECG syndromes:

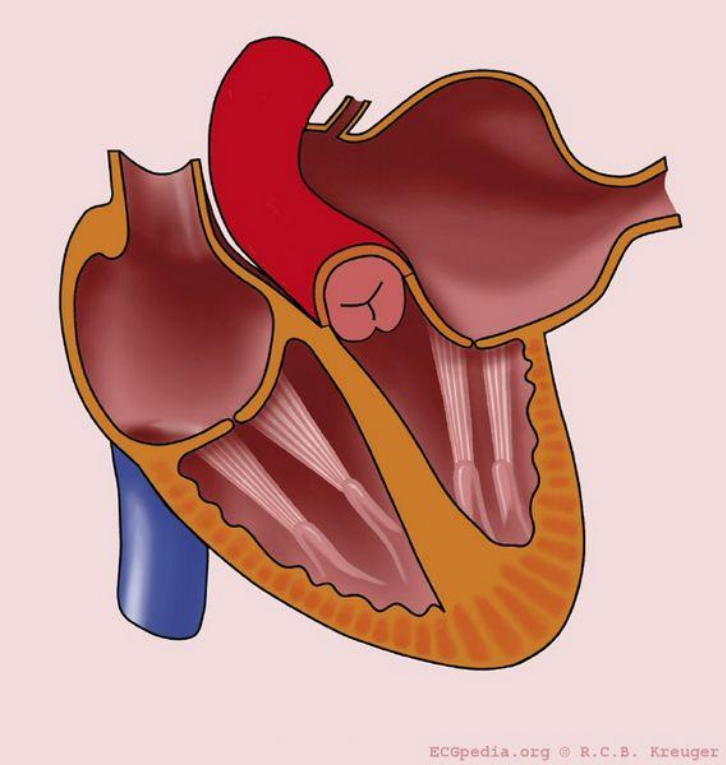
- arrhythmias,
- abnormalities of conductivity,
- atrial and ventricular hypertrophy,
- myocardial damage (ischemia, injury, necrosis, scar).


ECG
in myocardium
hypertrophy

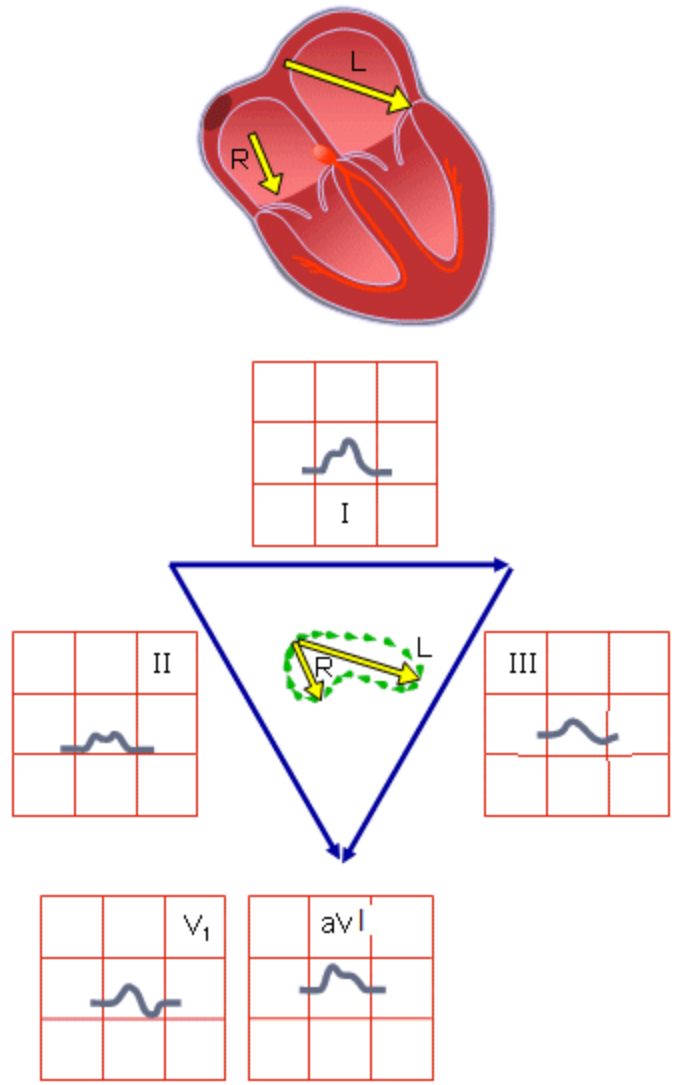

ECG
in left atrial
hypertrophy

P – mitrale

- The left atrial enlargement occurred most commonly in the patients with mitral valve defects.
- For this reason, this type of the P wave morphology if often termed **P – mitrale**



LEFT ATRIAL HYPERTROPHY

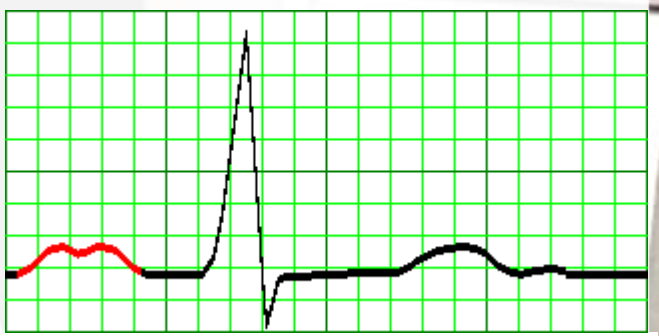


With the left atrium enlargement, the frontal plane P vector is oriented more horizontally and is of longer duration because the left atrial activation is prolonged.

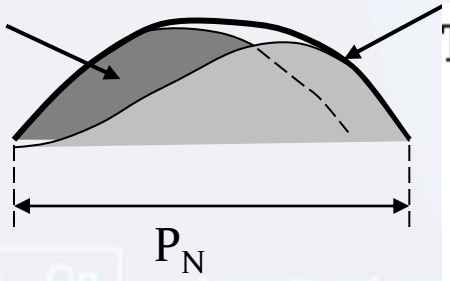
Left Atrium Enlargement

P - mitrale

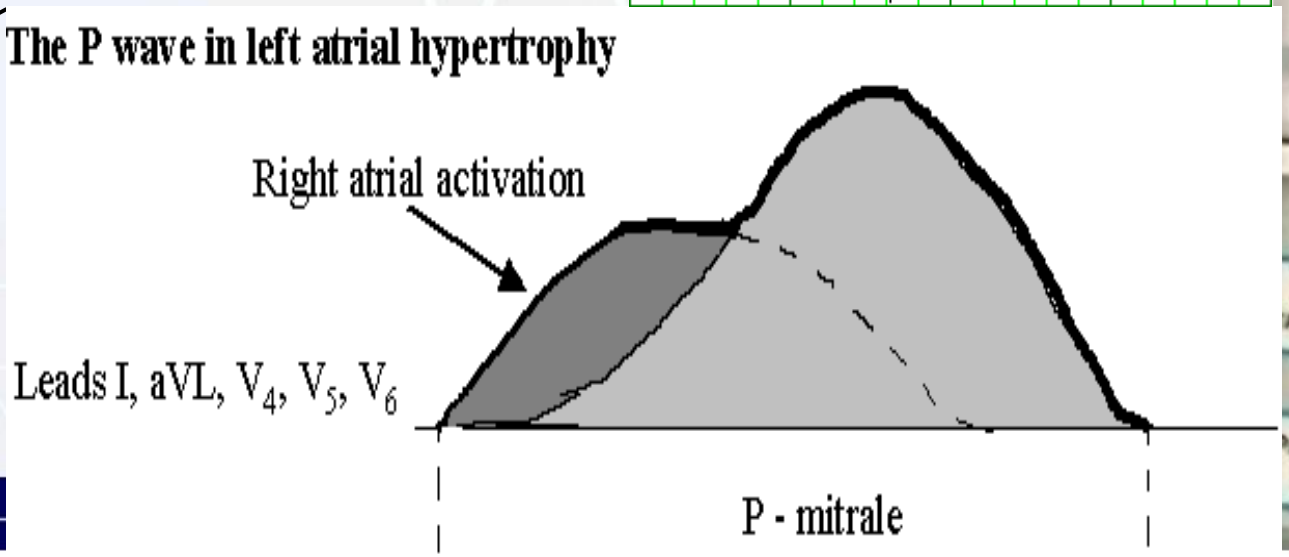
Therefore, P wave duration is greater than 0.1 second, and amplitude of the left atrial phase of P wave increases that can cause splitting of this wave .



Right atrial activation Left atrial activation

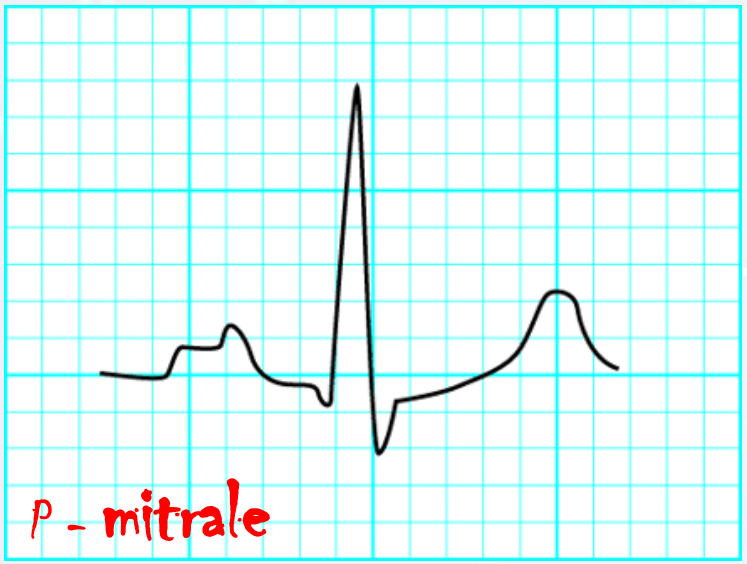


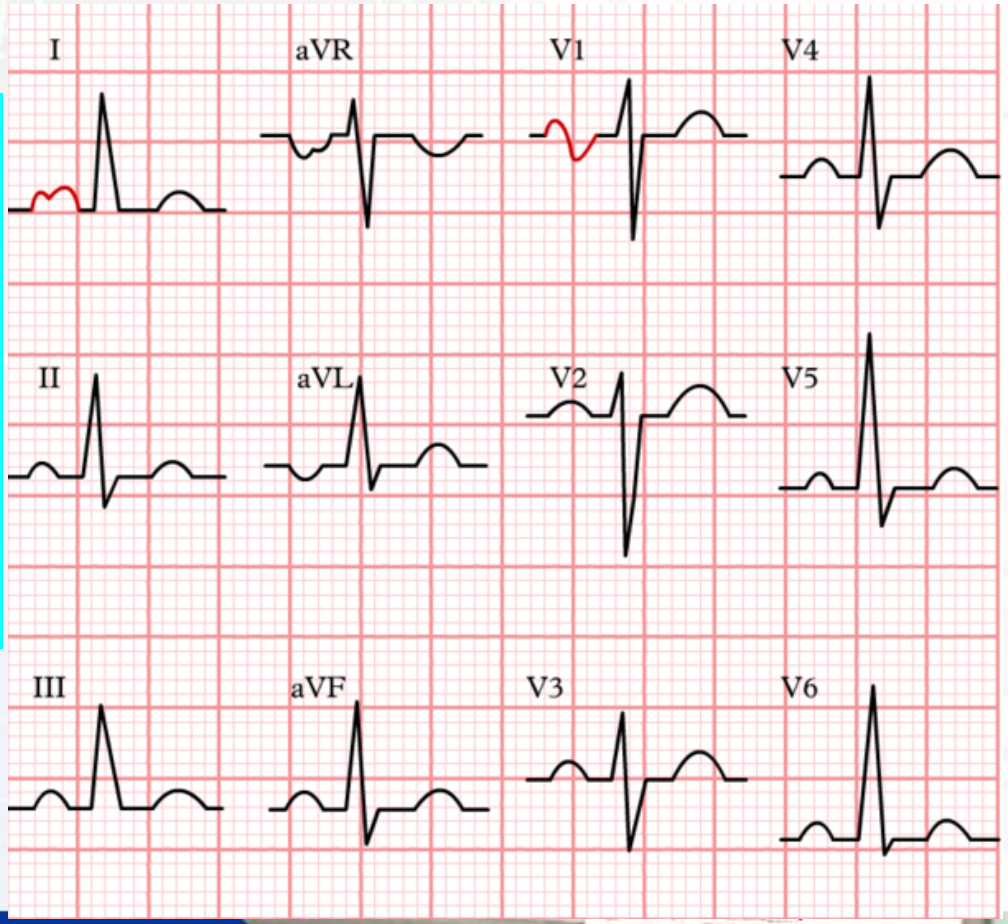
Leads I, aVL, V_4, V_5, V_6



Diagnostic ECG Signs of Left Atrial Enlargement

- 1. High-amplitude and two-peaked P wave in leads I, II, aVL, V_4, V_5, V_6.

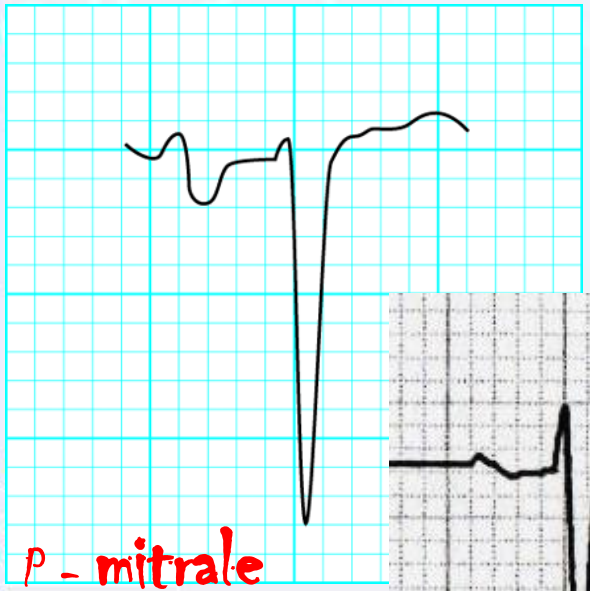


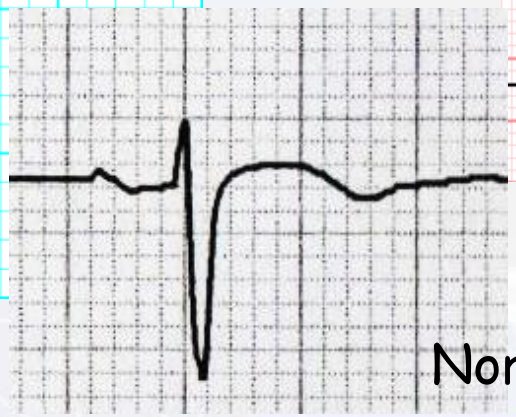


Normal monophasic P wave in lead II

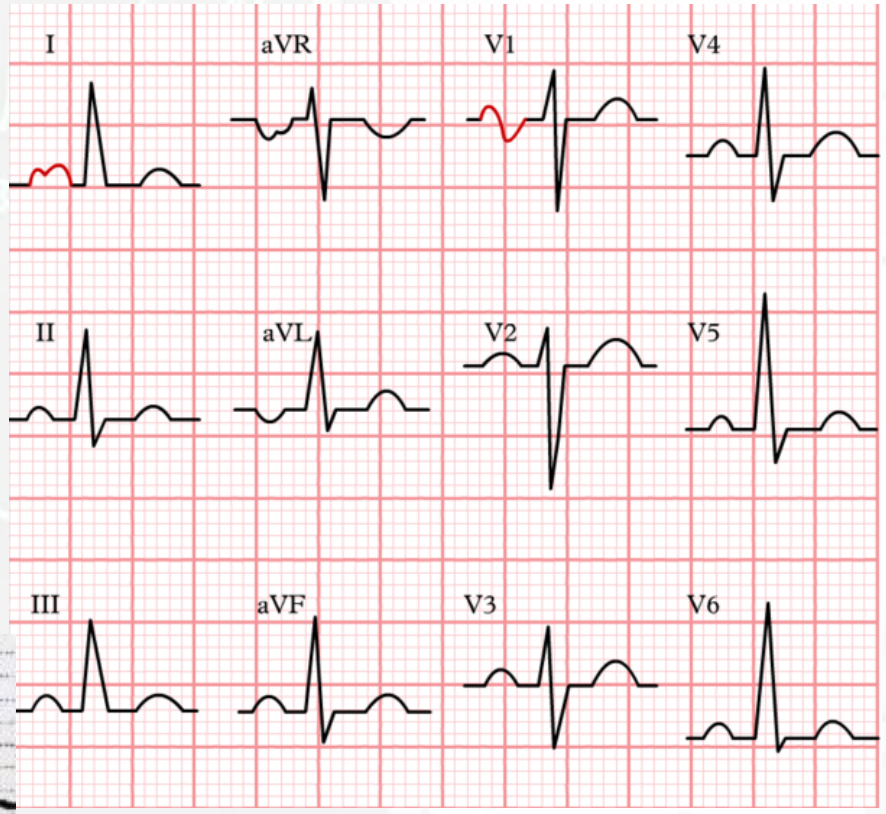
Diagnostic ECG Signs of Left Atrial Enlargement

- 2. In lead V_1 (rarer in V_2) P wave is initially positive and terminally negative or negative P wave in V_1 is formatted.

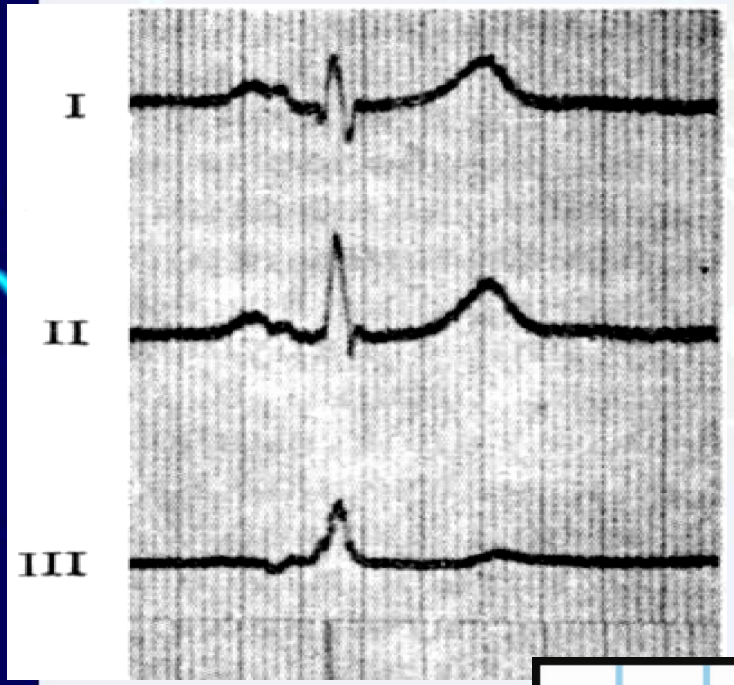




Normal biphasic P wave in V1



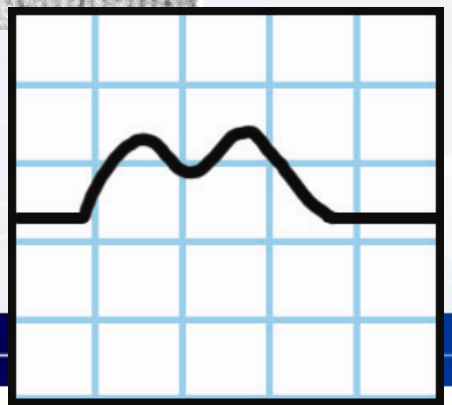
Diagnostic Electrocardiographic Signs of Left Atrial Enlargement

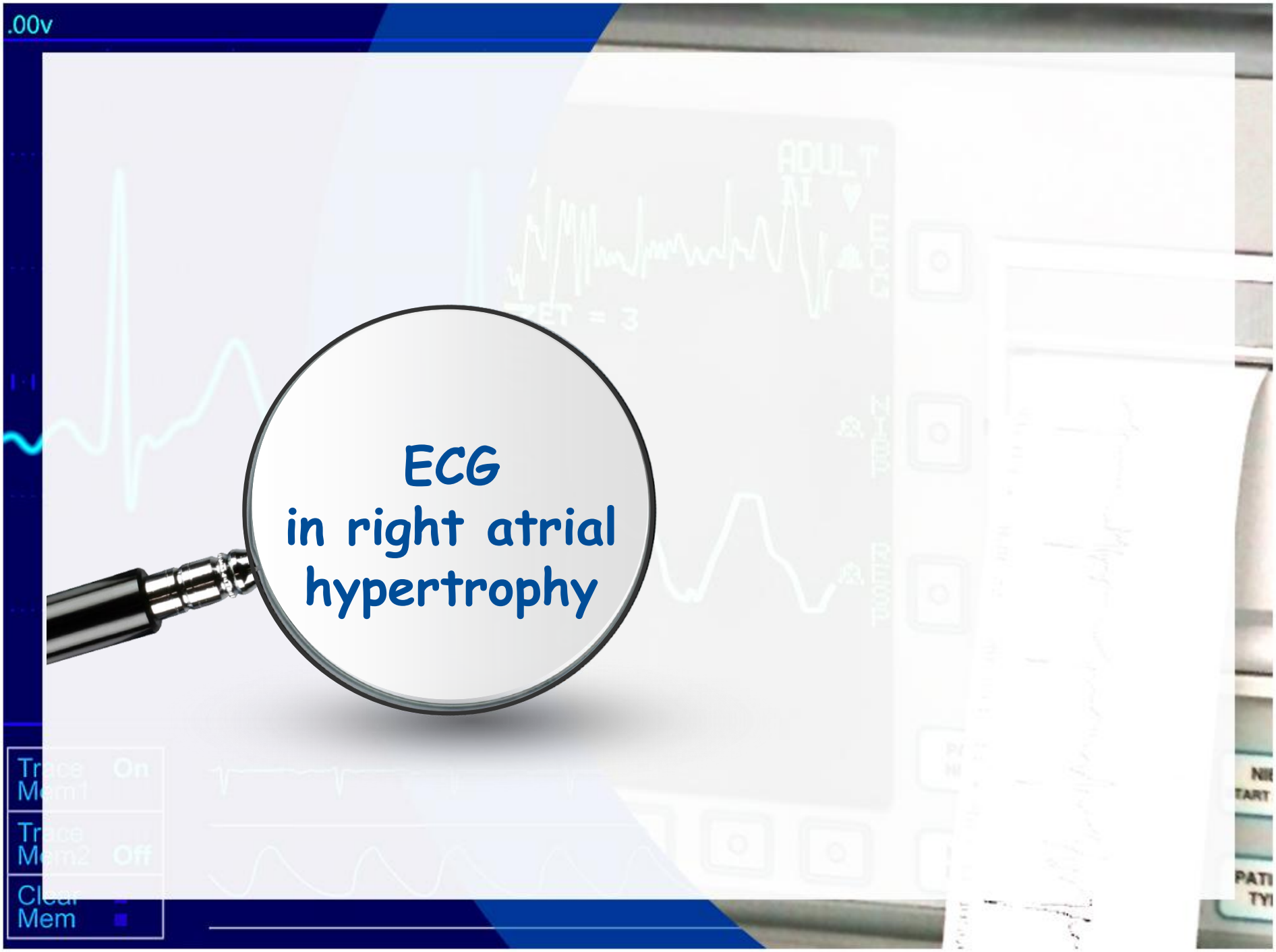


- 3. In lead III negative or two-phased (+ -) P wave (inconstant sign).

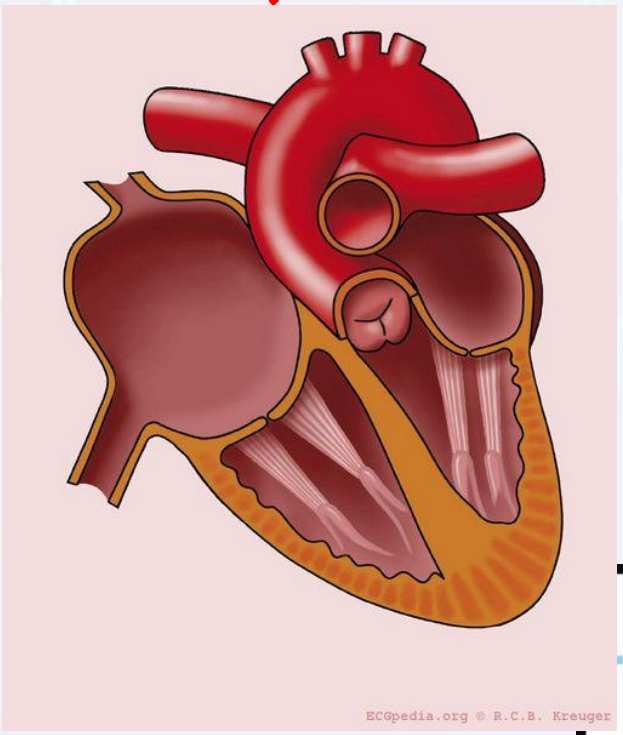
- 4. The P wave duration is more than 0.1 second.

- **P-mitrale**

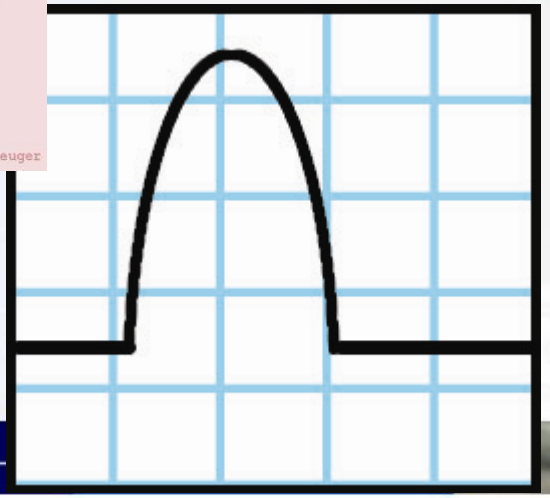



ECG
in right atrial
hypertrophy

P – pulmonale

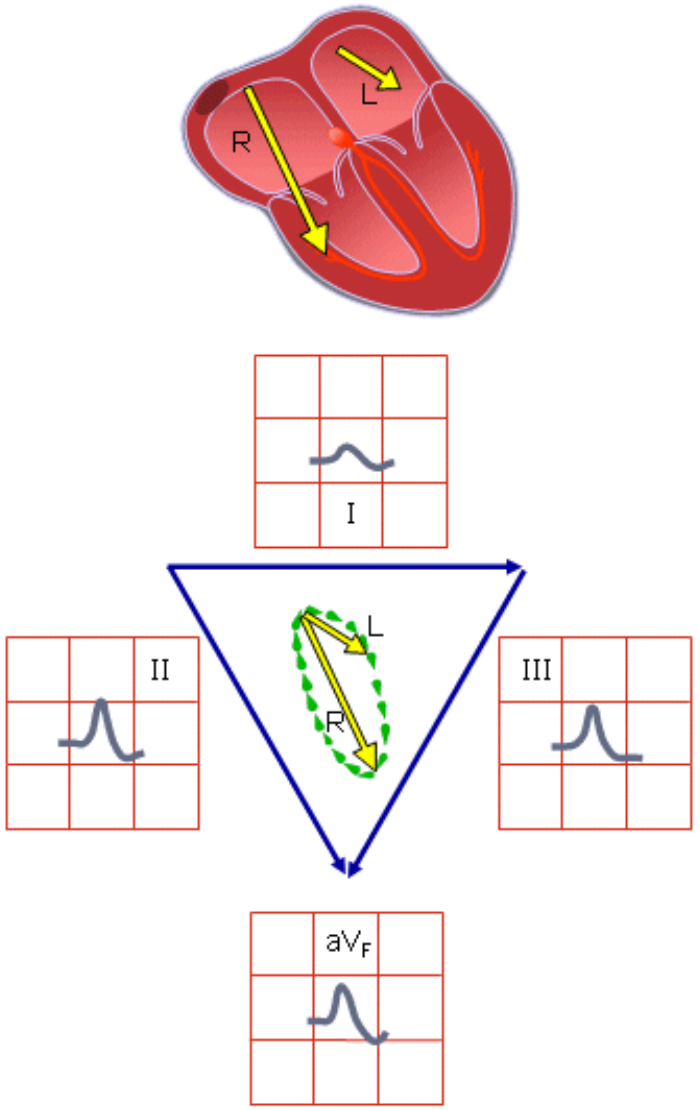


- Since right atrial enlargement is often due to pulmonary diseases, the terms
- **P – pulmonale** is used to describe this P wave morphology



RIGHT ATRIAL HYPERTROPHY

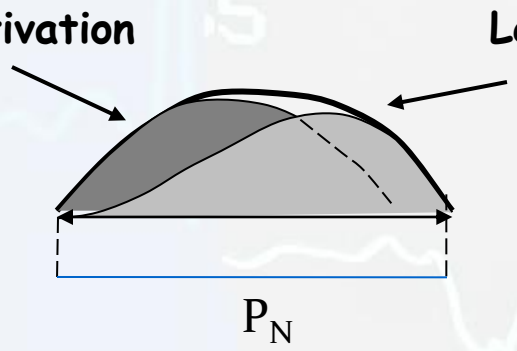
- Right atrial enlargement causes the frontal plane P wave axis to shift vertically.

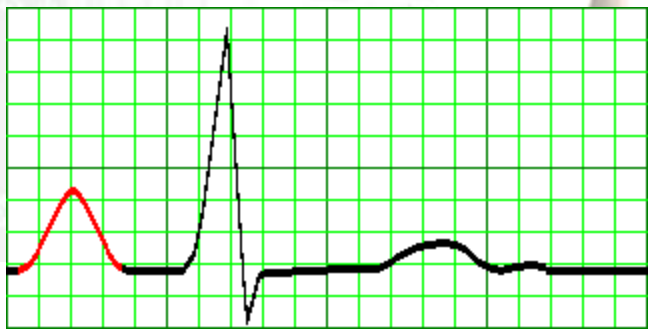


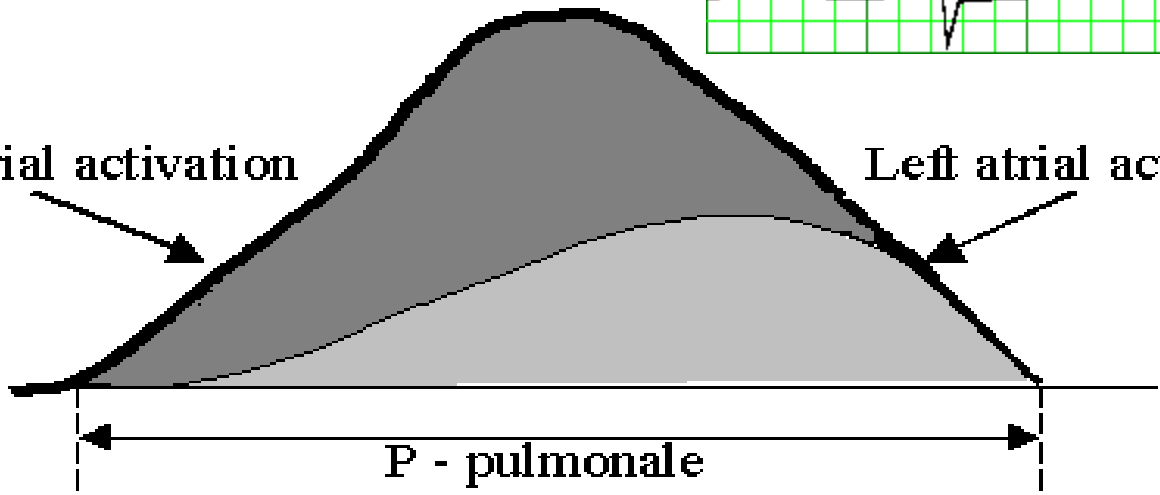
Right Atrium Enlargement

Since the right atrium if the first to be activated, prolongation of its activation time as a result of enlargement does not cause widening of the P wave, but only increased amplitude.

The normal P wave

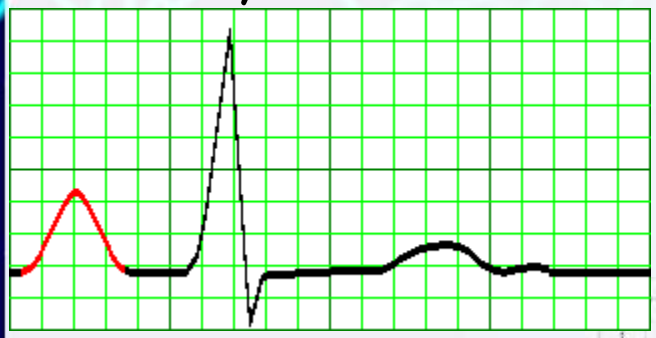




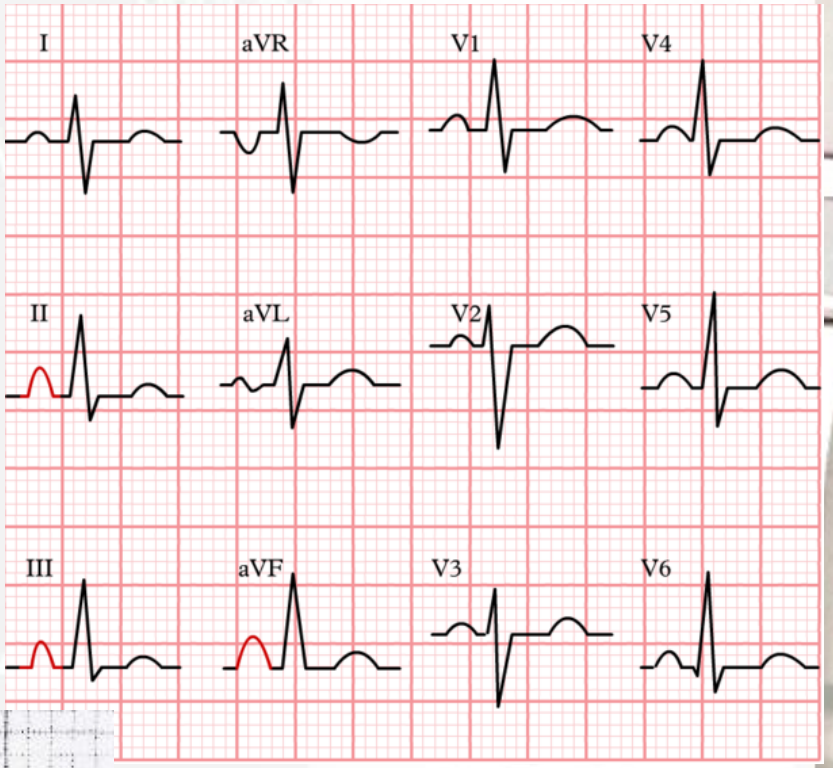


Diagnostic Electrocardiographic Signs of Right Atrial Enlargement

1. High-amplitude, peaked P wave more than 2 mm, higher than $\frac{1}{4}$ of the R wave amplitude in leads II, III, aVF.



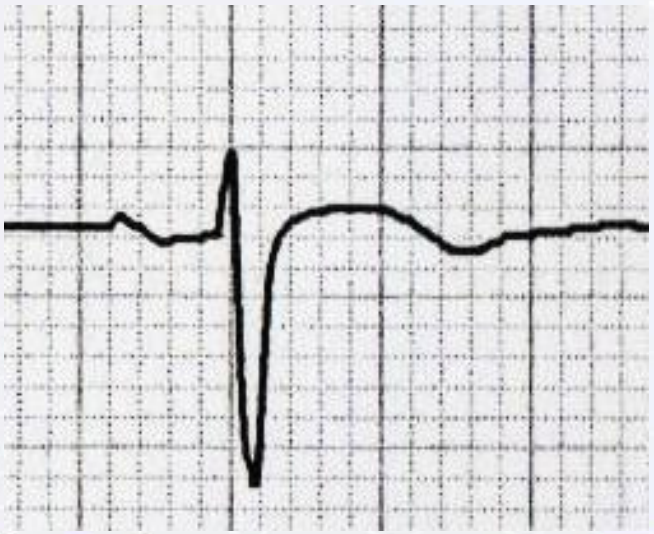
P - pulmonale

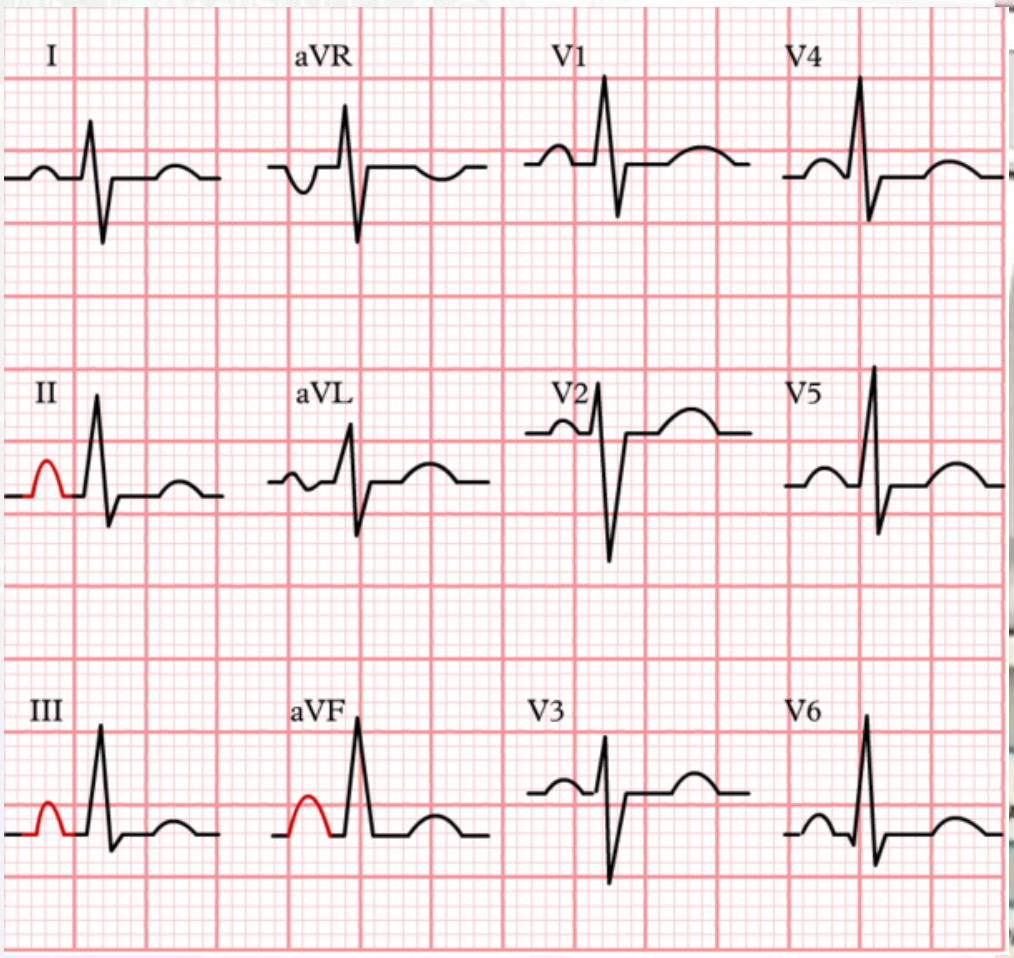


Normal monophasic P wave in lead II

Diagnostic Electrocardiographic Signs of Right Atrial Enlargement

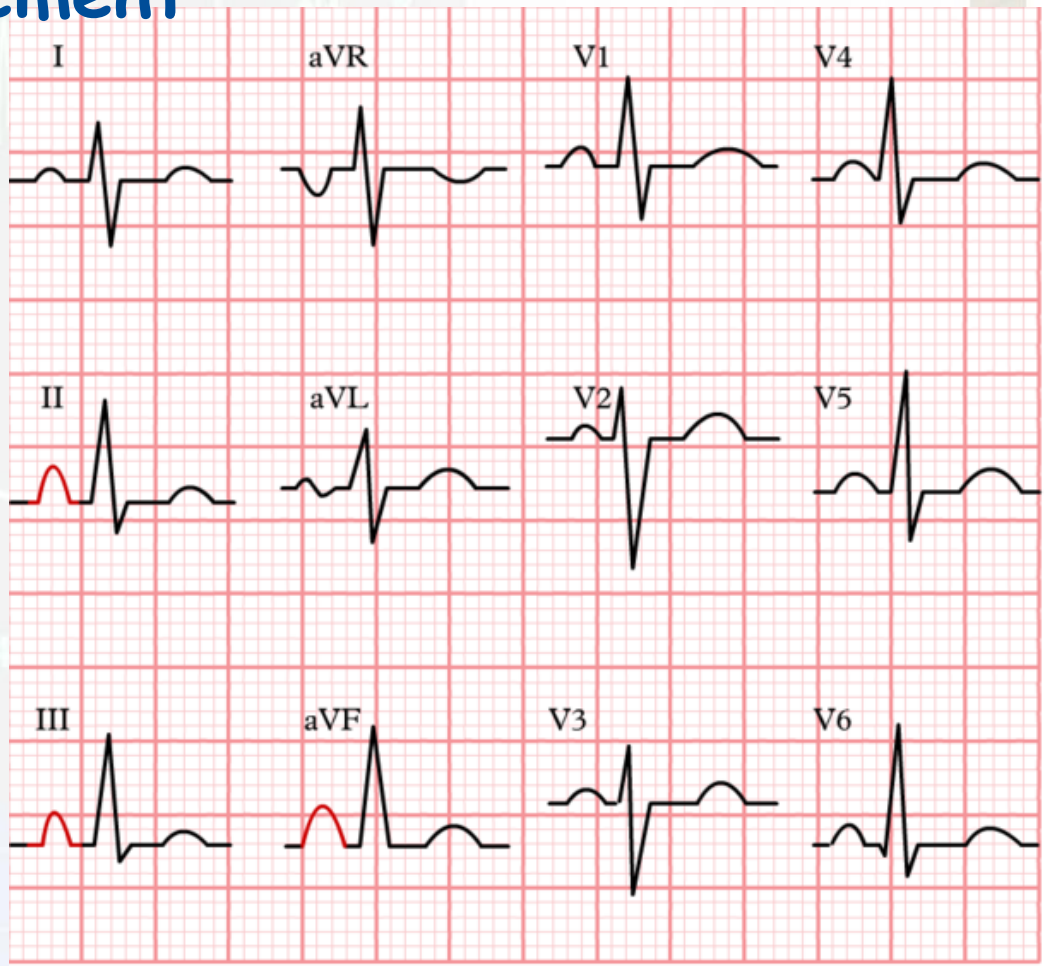
2. Positive, peaked P wave (or its initial right atrial phase) in leads V_1, V_2.





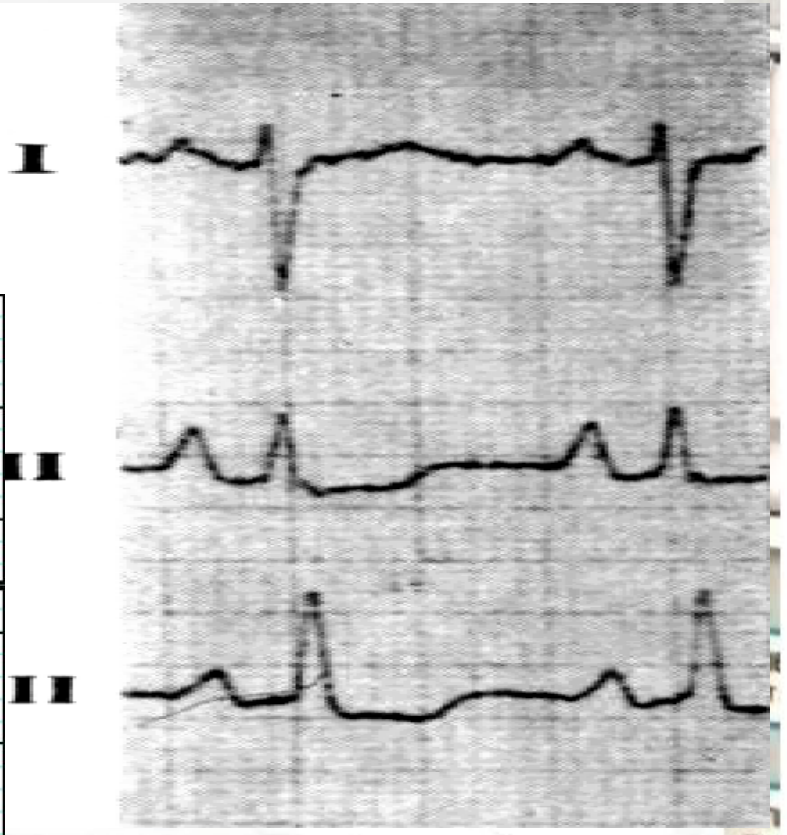
Diagnostic Electrocardiographic Signs of Right Atrial Enlargement

3. Low-amplitude P wave in leads I, aVL, V_4, V_5, V_6, in lead aVL it may be negative (inconstant sign).

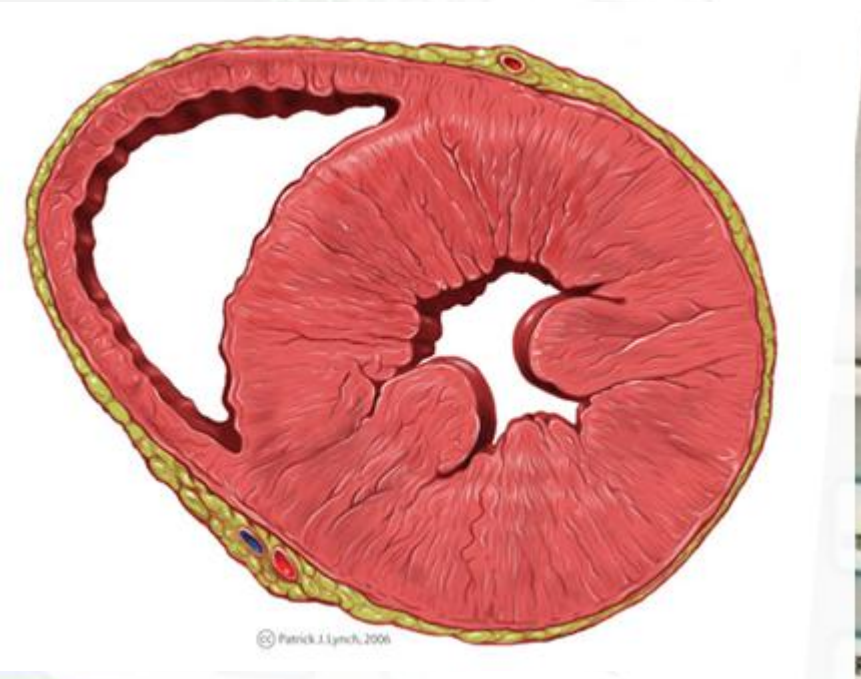


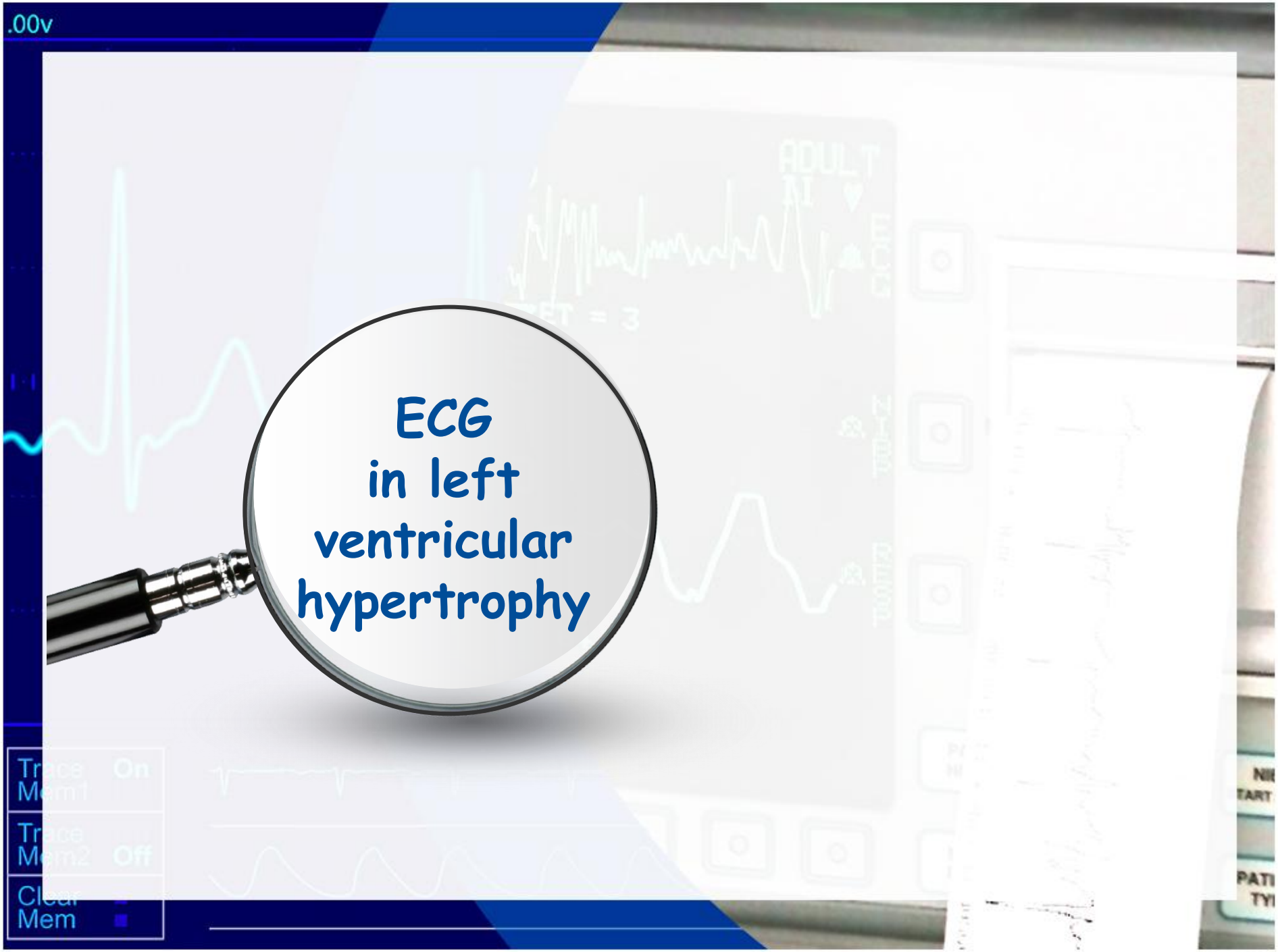
Diagnostic Electrocardiographic Signs of Right Atrial Enlargement

4.The P wave duration is not more than 0.1 second.

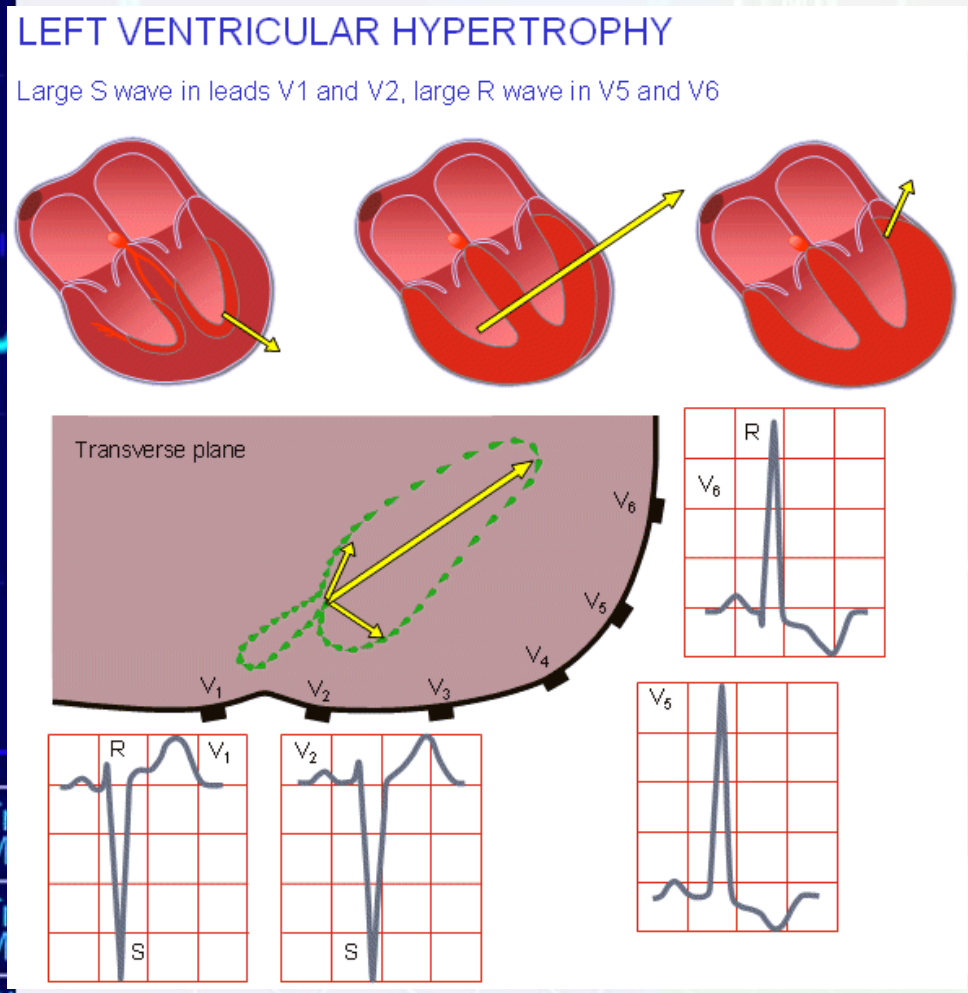


DIAGNOSTIC ELECTROCARDIOGRAPHIC SIGNS OF THE VENTRICULAR HYPERTROPHY




ECG
in left
ventricular
hypertrophy

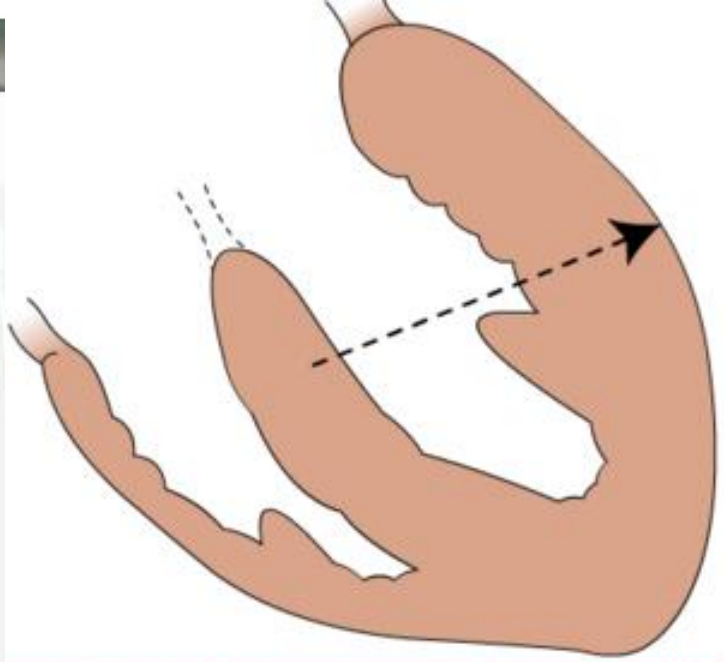
Diagnostic ECG Signs of Left Ventricular Hypertrophy

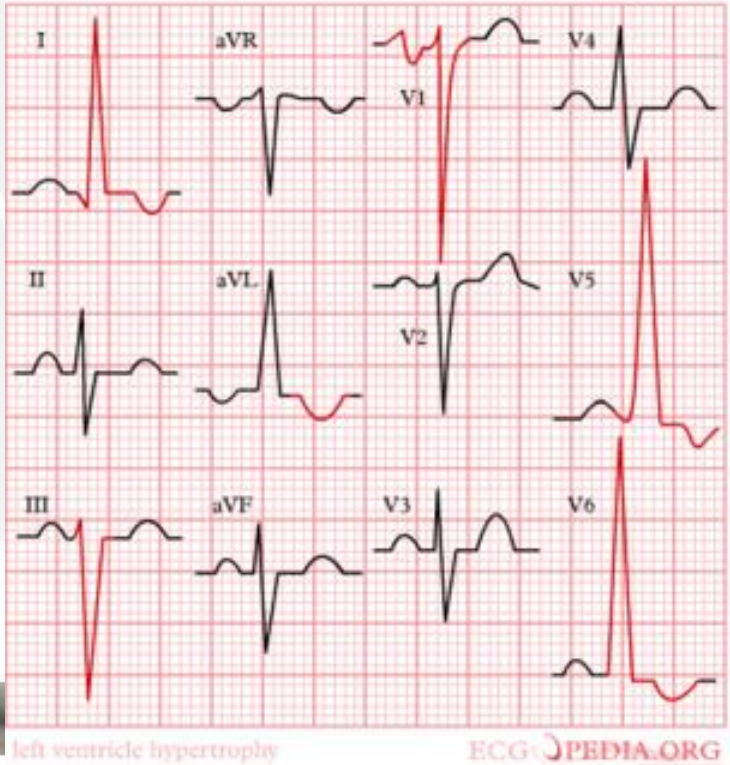


- 1. **Increased voltage of QRS deflection.**
- In the presence of ventricular hypertrophy the increased magnitude of the left ventricular forces from the hypertrophied left wall results in a increased magnitude of the main QRS vector.

Diagnostic ECG Signs of Left Ventricular Hypertrophy

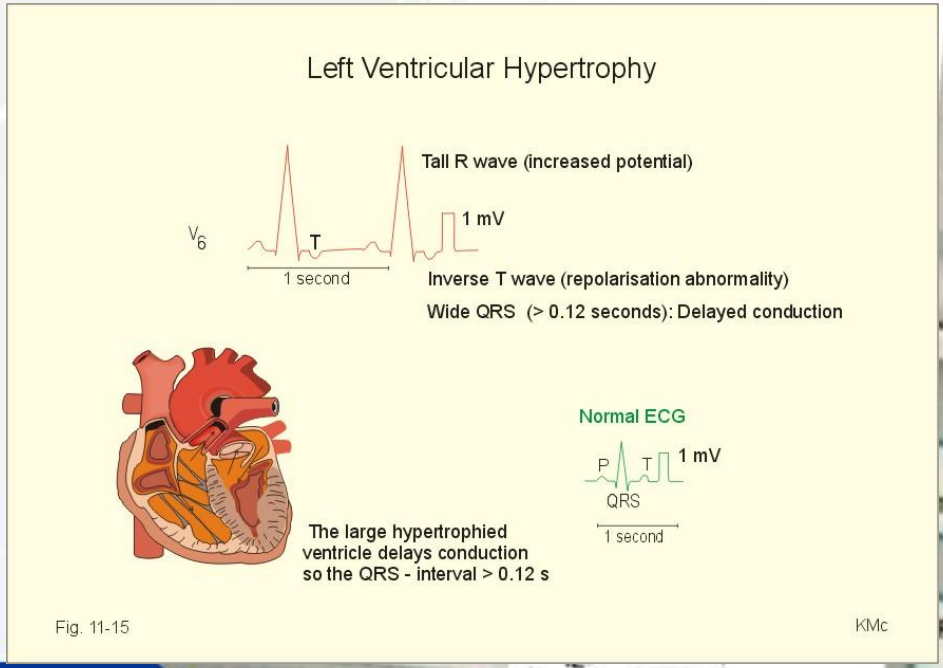
The left leads **I, aVL, V_4, V_5, V_6** show **high-amplitude R waves** and the right leads **III, aVF, V_1, V_2, V_3** show a **deep S wave**.



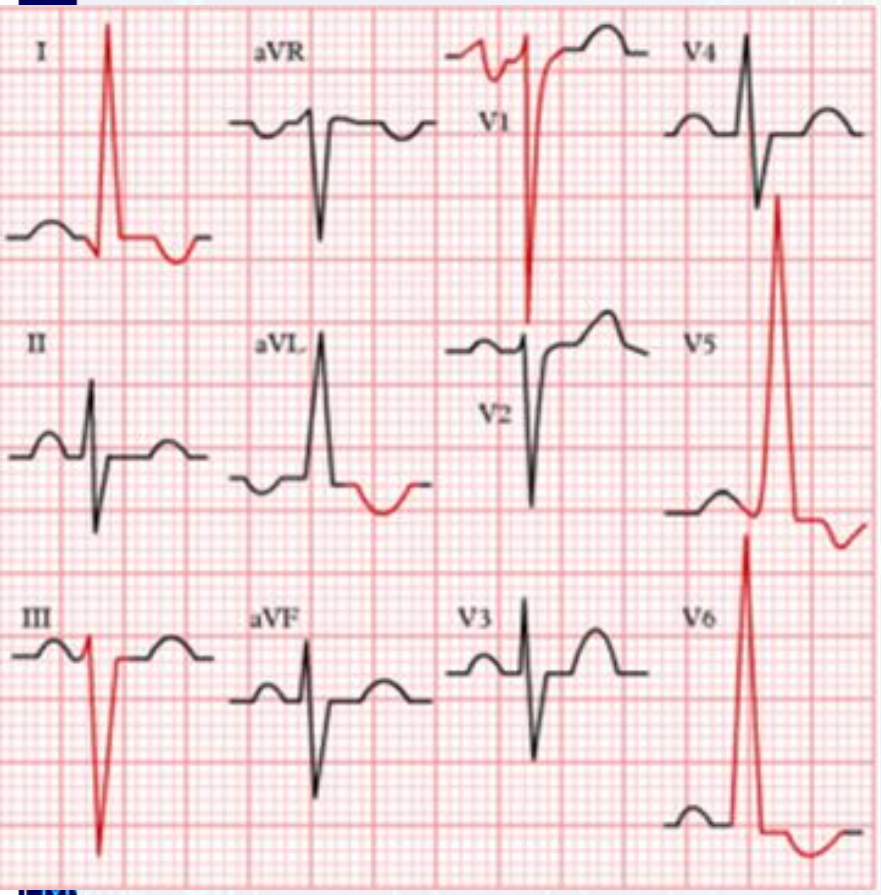


Diagnostic ECG Signs of Left Ventricular Hypertrophy

- 2. Increased duration of the QRS complex as a consequence of the increased muscle mass, and the **QRS complex is widened to 0.12 second.**

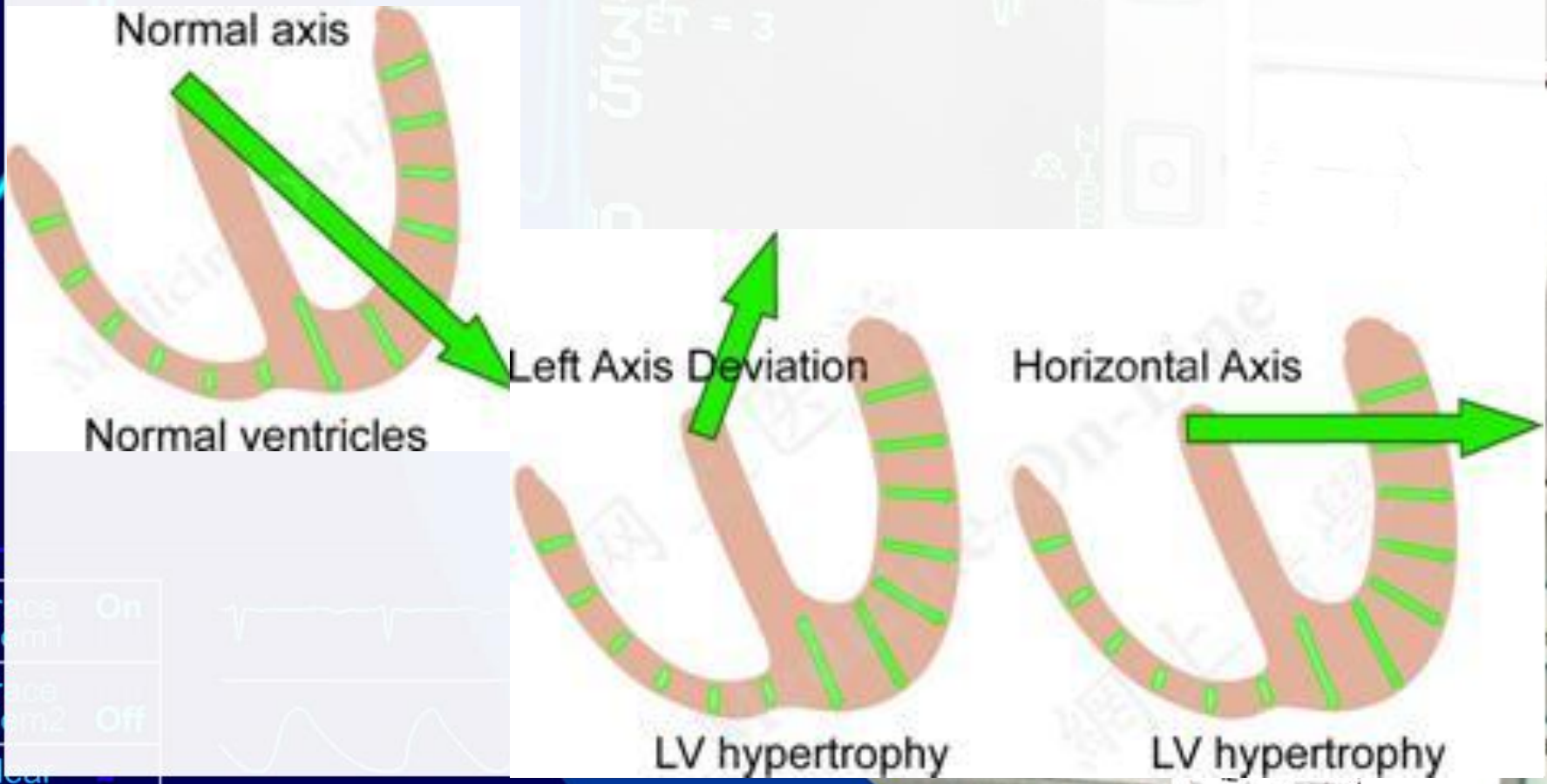


Diagnostic ECG Signs of Left Ventricular Hypertrophy

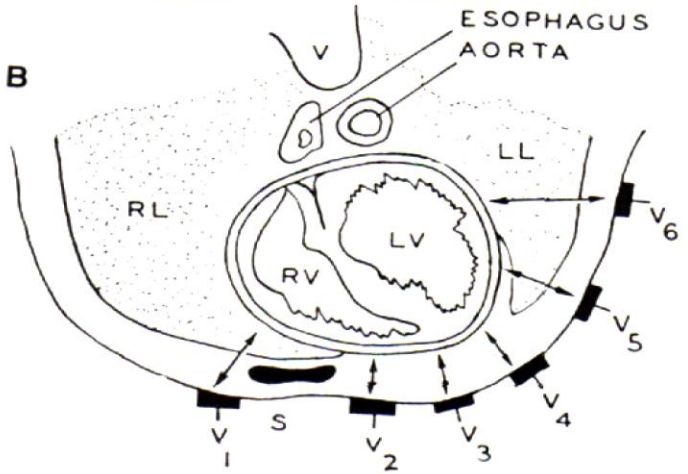


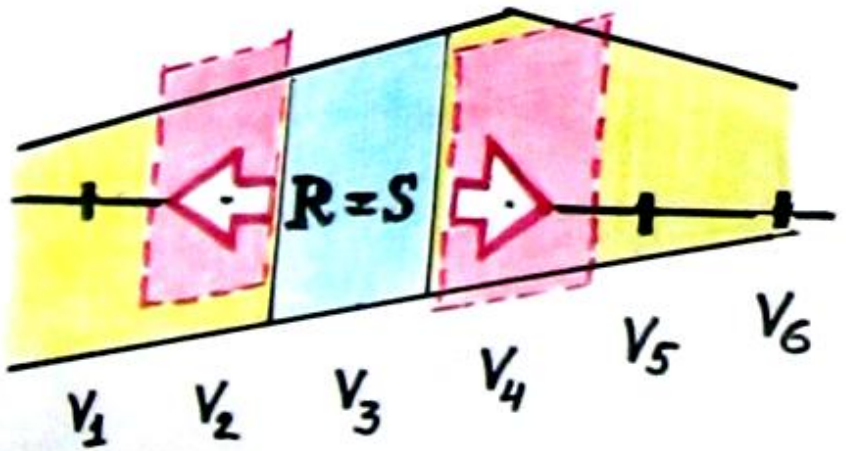
- 3. **The <u>ST segment is depressed and the T wave is inverted</u> in the leads I, II, aVL, V_4, V_5, V_6.** In the right precordial leads (V_1, V_2) the T wave is upright and the ST segment may be slightly elevated.
- ST segment and T wave changes are result from the altered ventricular depolarization and repolarization processes in the presence of left ventricular hypertrophy.

4. **Left axis deviation.** In the left ventricular hypertrophy electrical axis of the heart have a leftward orientation because the left ventricular mass is greatly increased and there may be an anatomical change in the position of the heart. **$R_I \geq 15mm$, $R_{aVL} \geq 11mm$, or $R_I + S_{III} > 25mm$.**



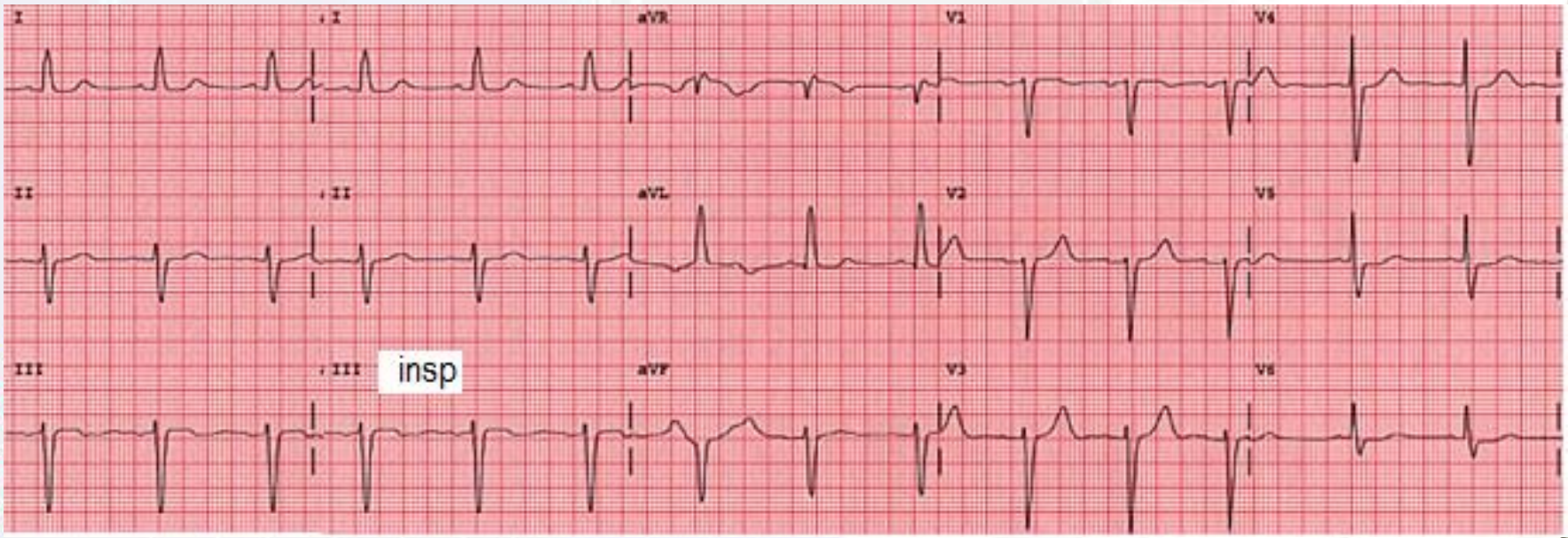
- 5. **The transition zone is displaced to the right (V_2).** In marked hypertrophy, the enlarged left ventricle as moves a little the right ventricle to the right, and heart turns around its vertical axis against clock hand.





Diagnostic ECG Signs of Left Ventricular Hypertrophy

6. **The deep S wave is preserved in lead III during deep inspiration.** During deep inspiration the diaphragm lowers, and if axis deviation is positional, deep S wave disappears because the heart assumes normal position. And, if axis deviation is caused by enlarged left ventricle, deep S wave preserves in lead III during deep inspiration.



ECG criteria for LVH

- There are several sets of criteria used to diagnose LVH via electrocardiography
- None of them is perfect, though by using multiple criteria sets, the sensitivity and specificity are increased.

- The **Sokolow-Lyon index**:
  - S wave in V_1 + R wave in V_5 or V_6 (whichever is larger) ≥ 35 mm (≥ 7 large squares)
  - R in aVL ≥ 11 mm

ECG criteria for LVH

- The **Cornell voltage criteria** for the ECG diagnosis of LVH involve measurement of the sum of the R wave in lead aVL and the S wave in lead V_3.

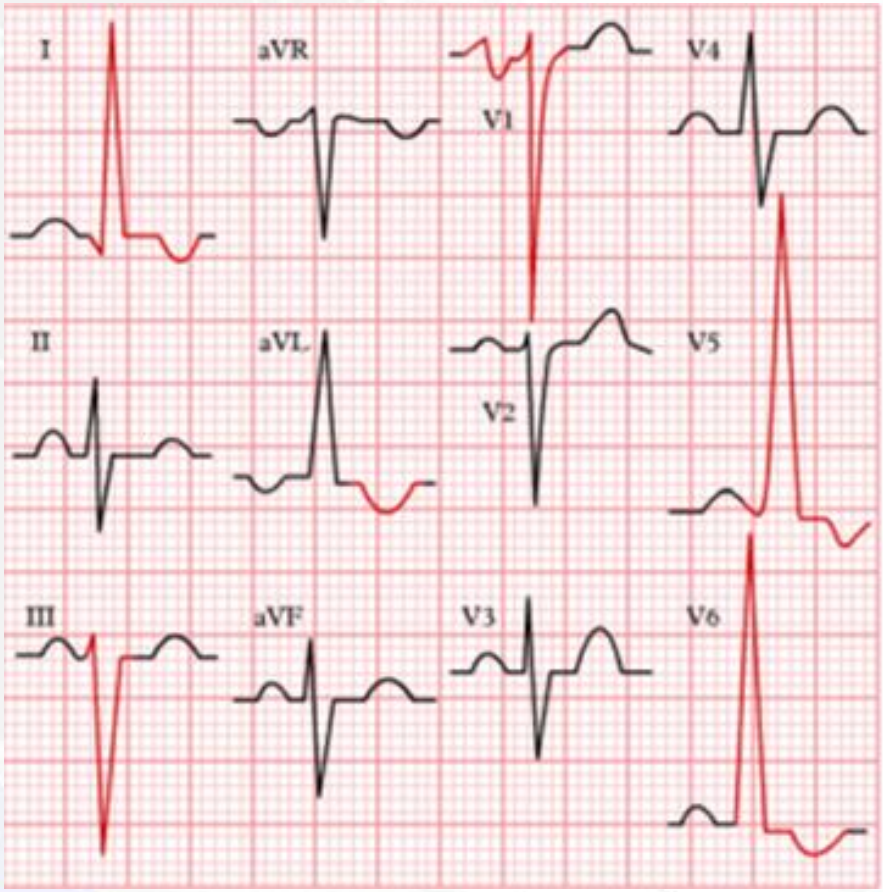
- The Cornell criteria for LVH are:
- S wave in V_3 + R wave in aVL > 28 mm (men)
- S wave in V_3 + R wave in aVL > 20 mm (women)

The Romhilt-Estes point score system ("diagnostic" >5 points; "probable" 4 points):

ECG Criteria	Points
Voltage Criteria (any of): R or S in limb leads ≥20 mm S in V_1 or V_2 ≥30 mm R in V_5 or V_6 ≥30 mm	3
ST-T Abnormalities:	
ST-T vector opposite to QRS without digitalis	3
ST-T vector opposite to QRS with digitalis	1
Negative terminal P mode in V_1 1 mm in depth and 0.04 sec in duration (indicates left atrial enlargement)	3
Left axis deviation (QRS of -30° or more)	2
QRS duration ≥0.09 sec	1

ECG criteria for LVH

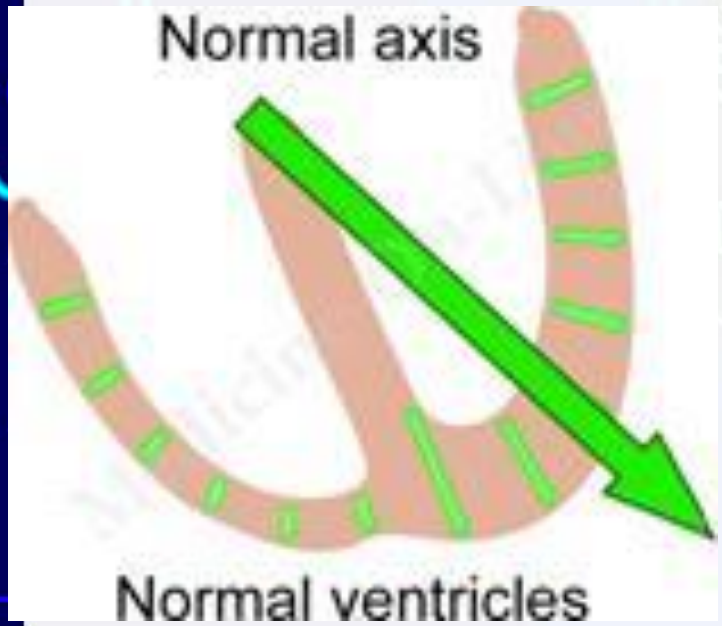
- Other voltage-based criteria for LVH include:
- Lead I: R wave > 14 mm
- Lead aVR: S wave > 15 mm
- Lead aVL: R wave > 12 mm
- Lead aVF: R wave > 21 mm
- Lead V_5: R wave > 26 mm
- Lead V_6: R wave > 20 mm

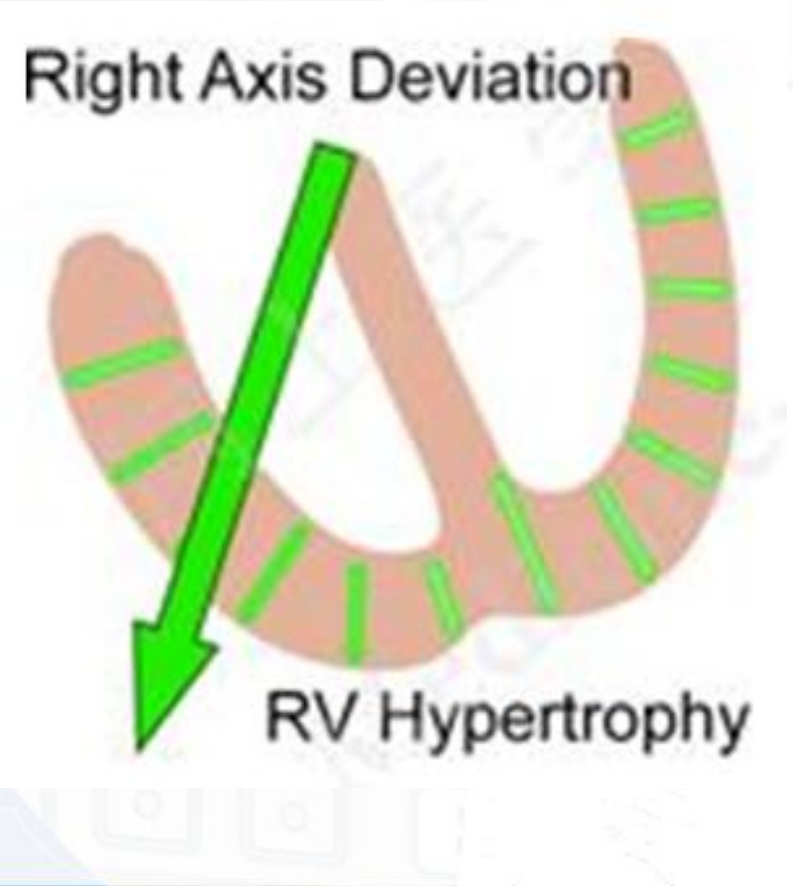


ECG in right ventricular hypertrophy

Diagnostic ECG Signs of Right Ventricular Hypertrophy

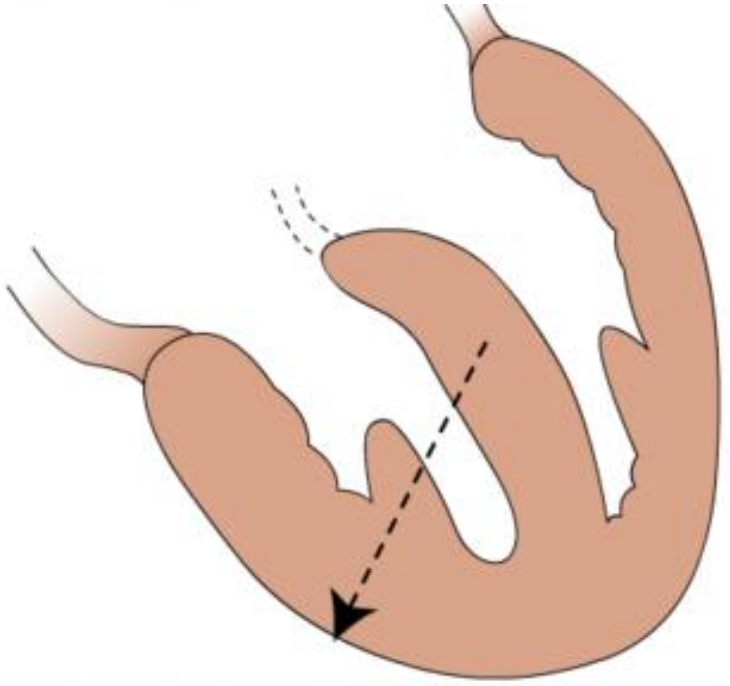
1. **Right axis deviation.**

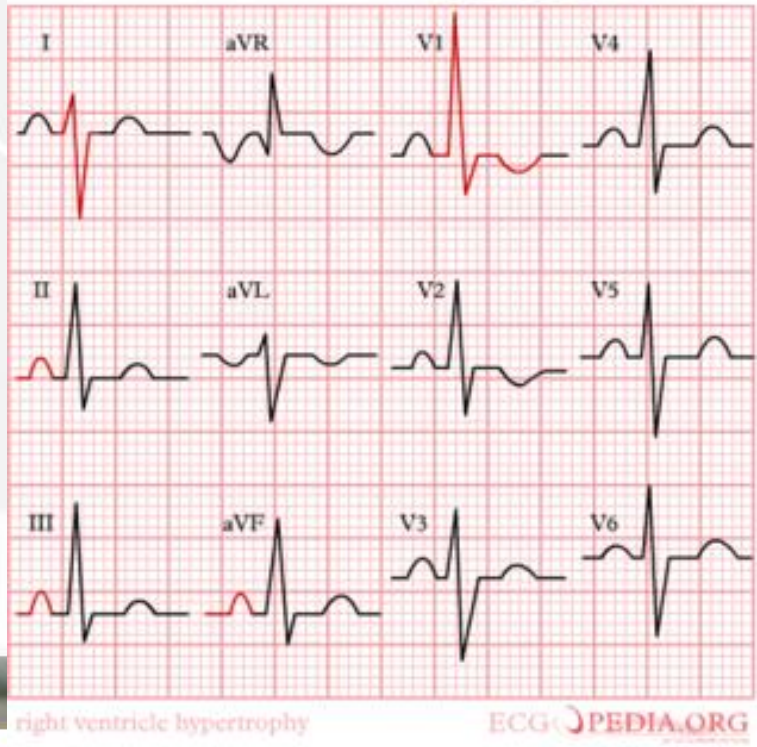




Diagnostic ECG Signs of Right Ventricular Hypertroph

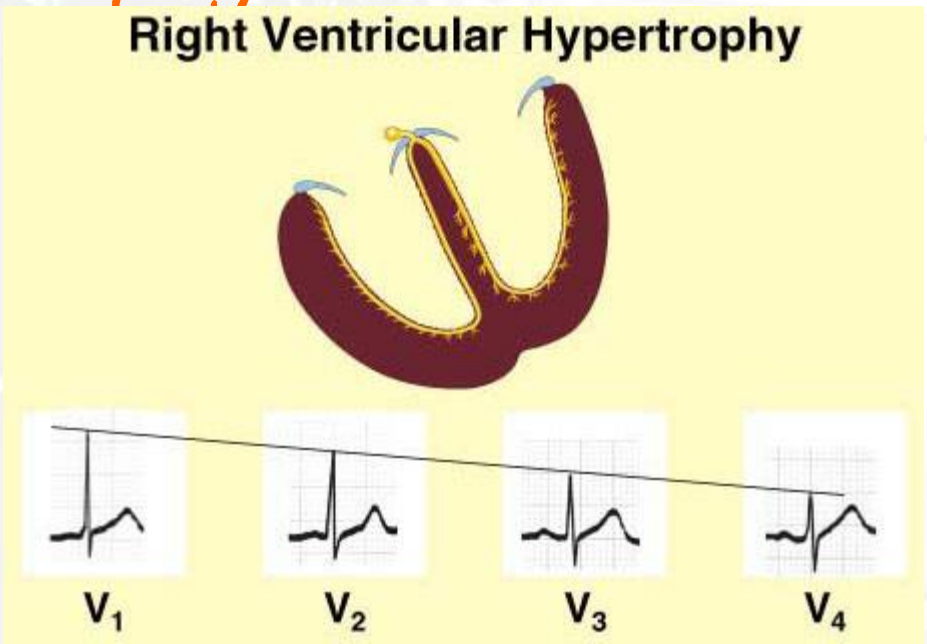
- 2. The right leads III, aVF, V_1, V_2, V_3 show **high-amplitude R waves** and the left leads I, aVL, V_4, V_5, V_6 show a **deep S wave**. $R_{V1} \geq 7$ mm or $R_{V1} + S_{V5,6} \geq 10,5$ mm.





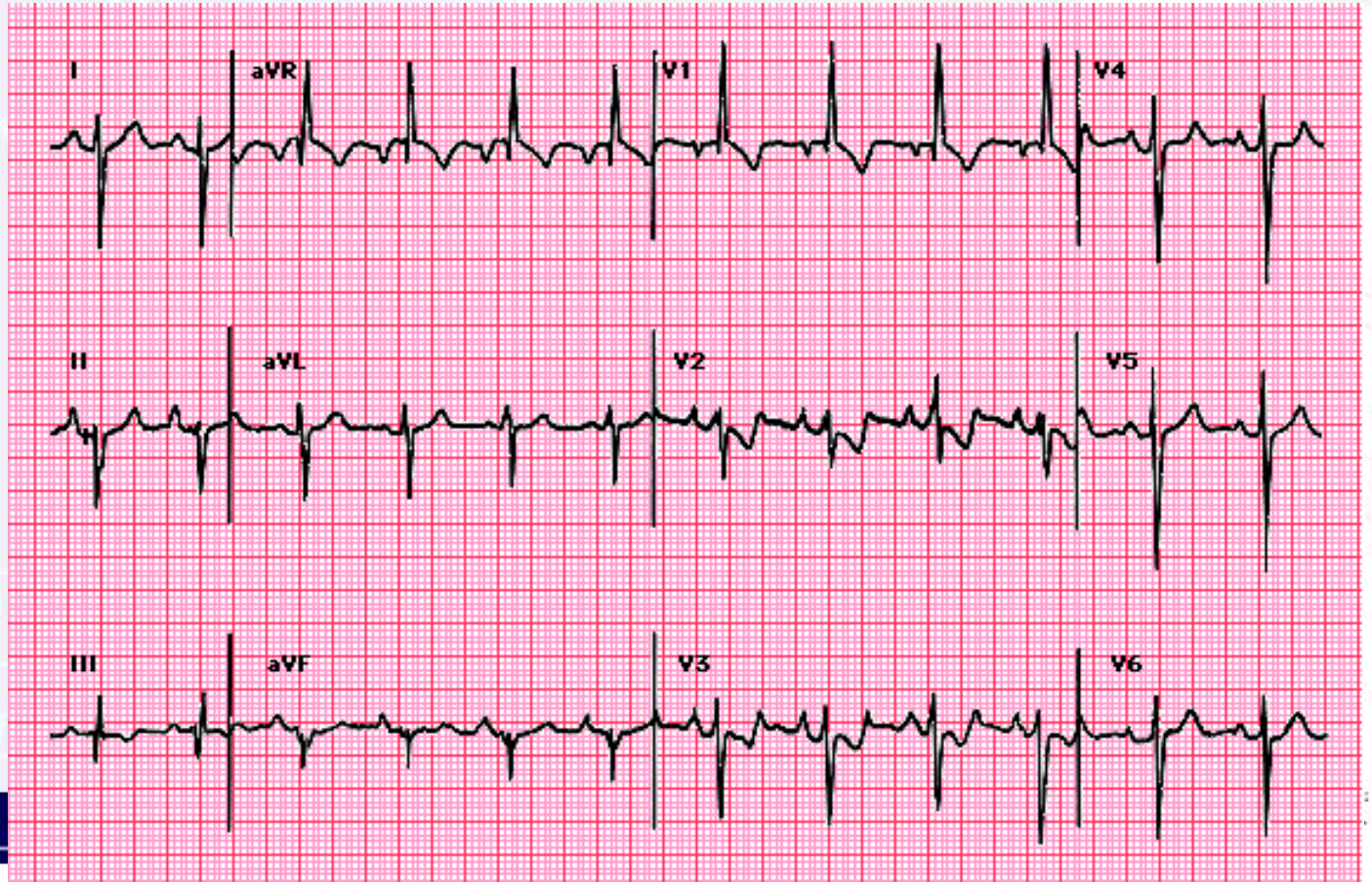
Diagnostic ECG Signs of Right Ventricular Hypertrophy

- 3. Duration of the QRS complex. In contrast to the pattern in left ventricular hypertrophy, the **QRS duration seldom is prolonged (to 0.12 second),** because even with hypertrophy, the thickness of the right ventricle does not exceed that of the left.

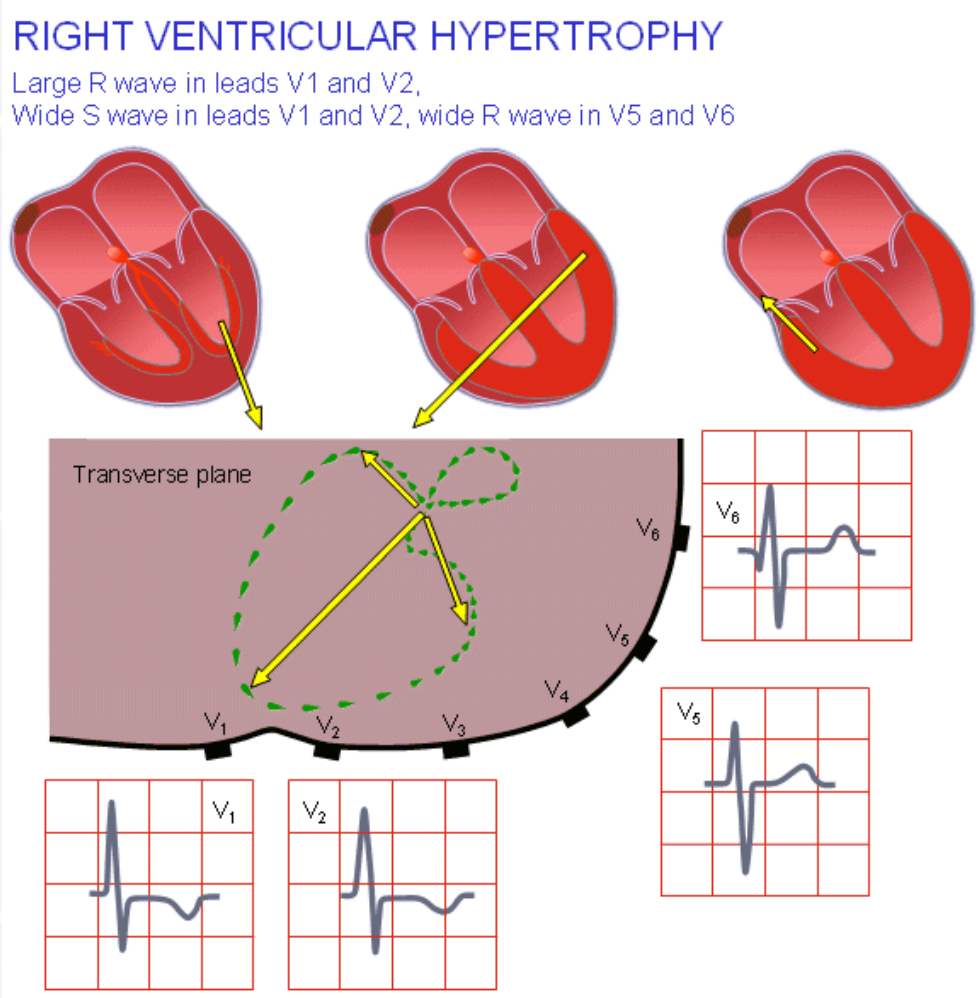


Diagnostic ECG Signs of Right Ventricular Hypertrophy

4. In leads III, aVF, V_1, V_2, V_3 the **ST segment** may be **depressed** and the **T wave inverted** over the hypertrophied right ventricle. When ST – T wave changes are present, it is often indicate of more severe right ventricular hypertrophy.

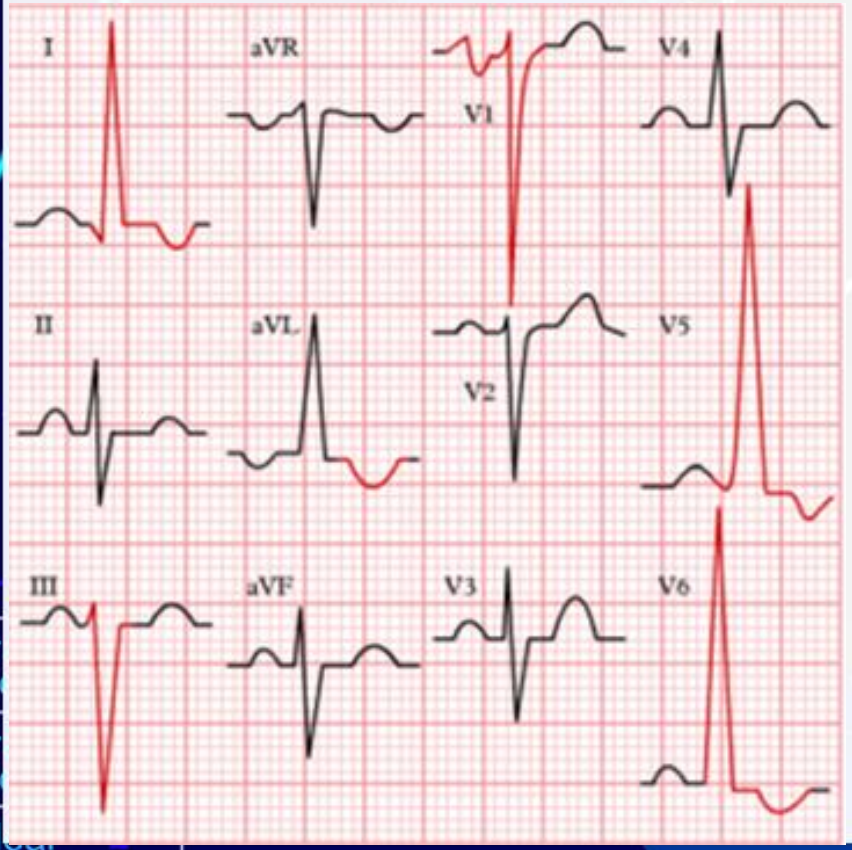


5. **The transition zone is displaced to the left (V_5 – V_6).** In the right ventricular the muscle mass is increased, and the heart tends to rotate on its longitudinal axis in a clockwise manner, so that the right ventricle becomes more anterior and the left ventricle rotates posteriorly. The septum rotates similarly, becoming more parallel to the frontal plane of the body.

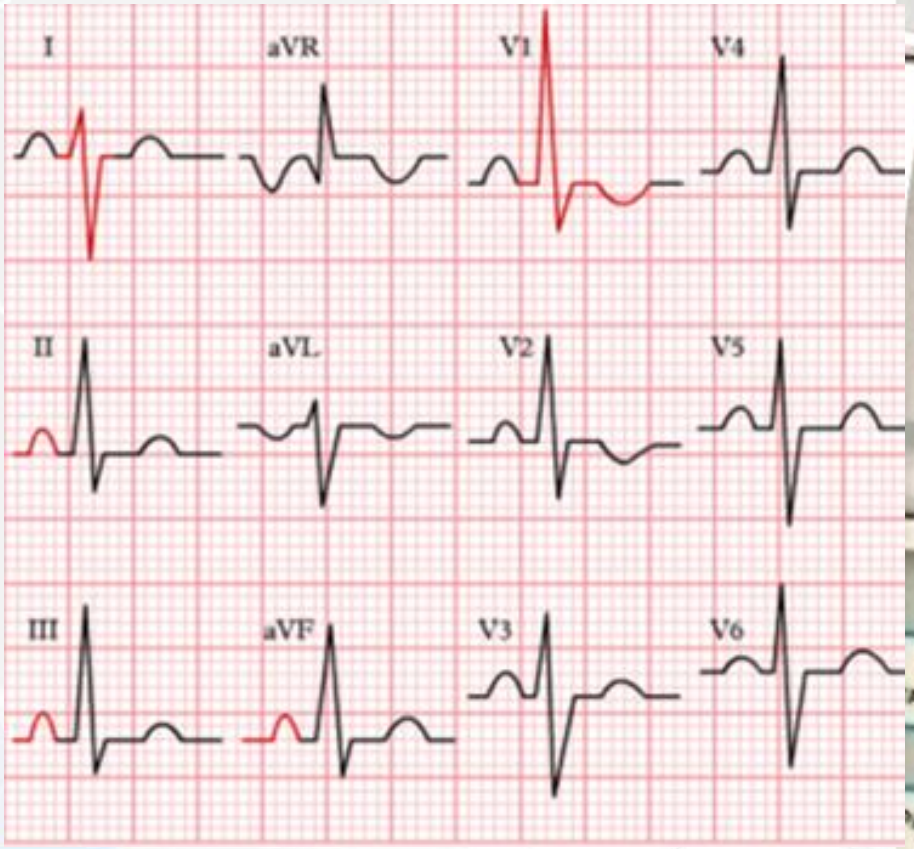


ECG criteria for

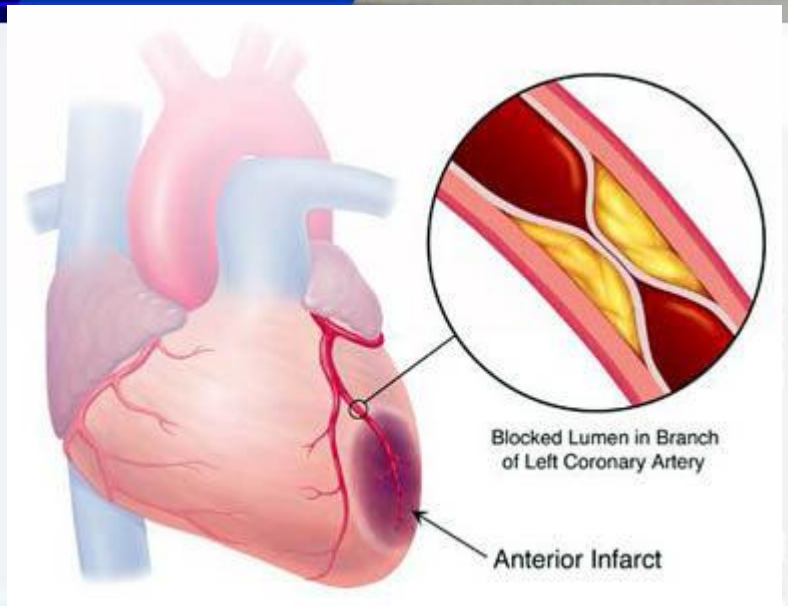
LVH



RVH




ECG
in CHD

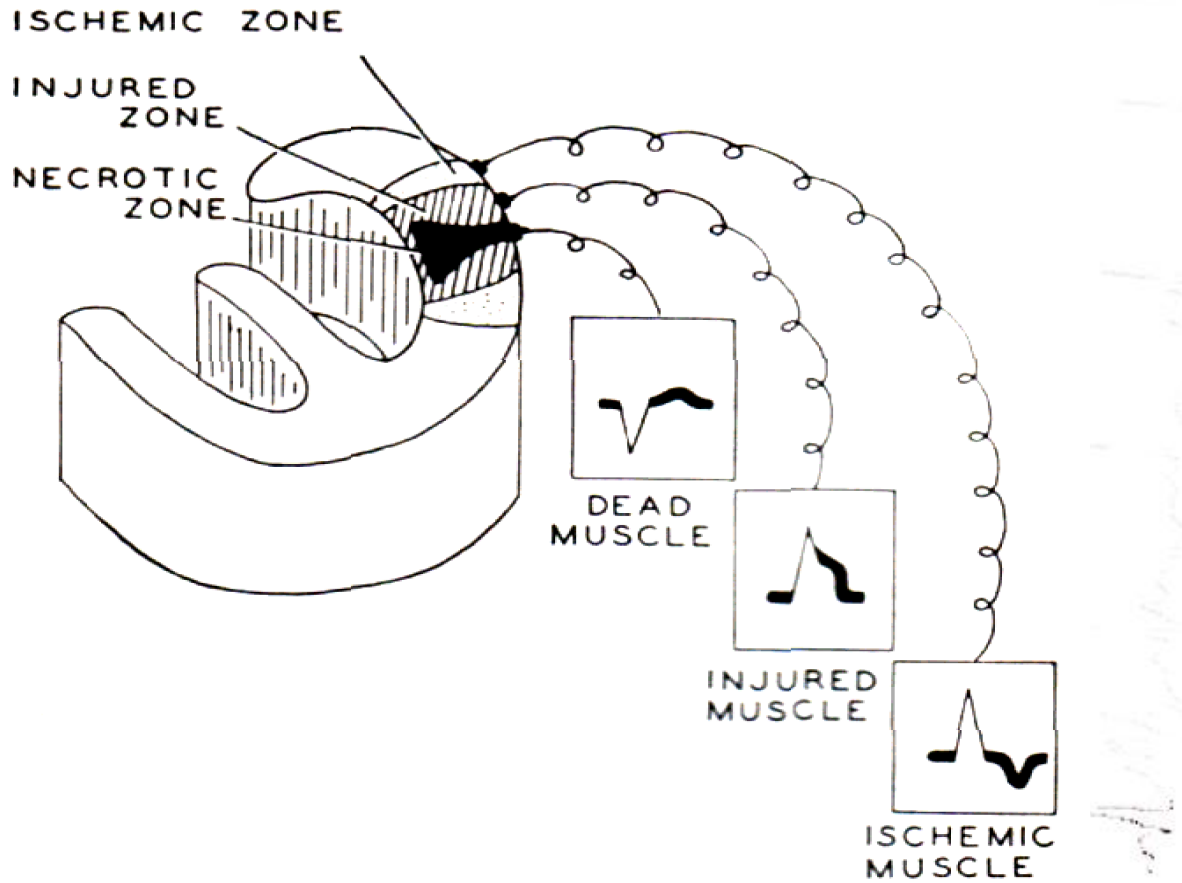


CORONARY (ISCHEMIC) HEART DISEASE

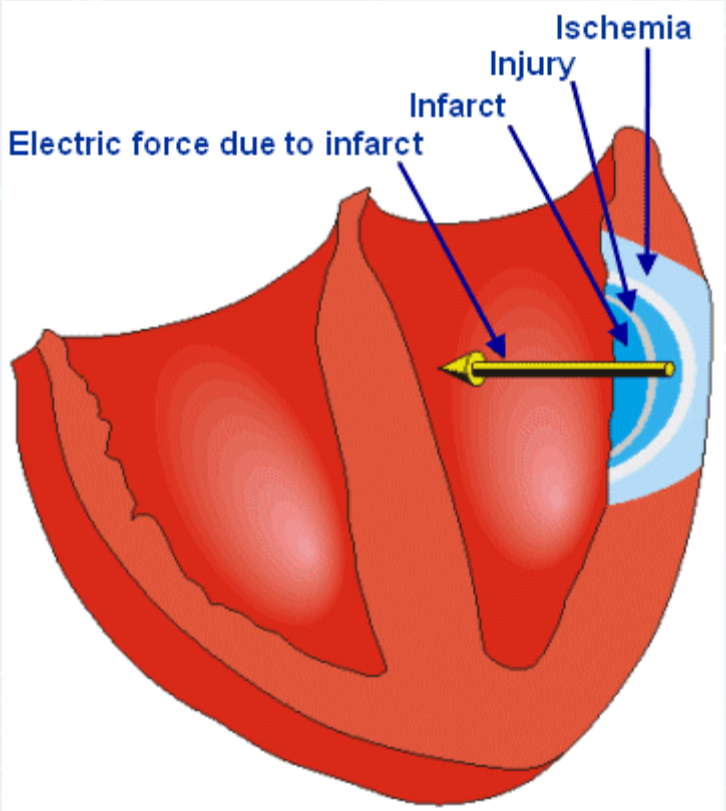
The term coronary heart disease includes such diseases as angina pectoris, myocardial infarction, and coronary cardiosclerosis.

ECG signs of the coronary heart disease

If the coronary artery is occluded the involved heart muscle progresses in sequence through three stages of damage toward infarction. Each stage is associated with electrical changes.



Ischemia



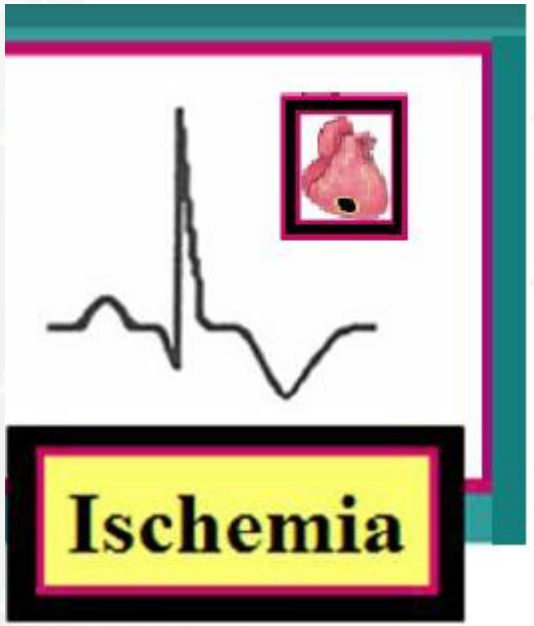
- *Ischemia* develops in conditions when the insufficient amount of blood is delivered to the heart muscle through the coronary arteries, and themyocardium does not receive the necessary amount of oxygen. Ischemic damage to the myocardium alters the sequence of ventricular activation and affects the repolarization or the recovery process of the heart.

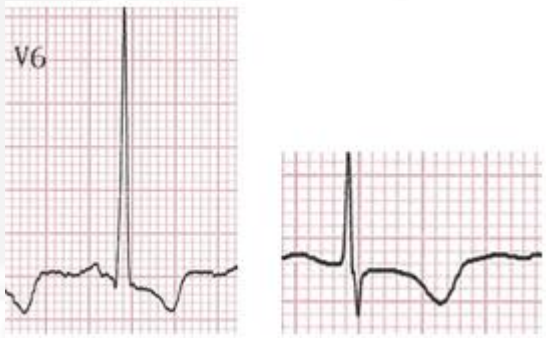
Ischemia

These changes alter the electrical field of the heart, resulting in T wave modifications.

Ischemia is recognized by symmetrically inverted T wave.

Ischemia is a reversible process unassociated with histological changes.



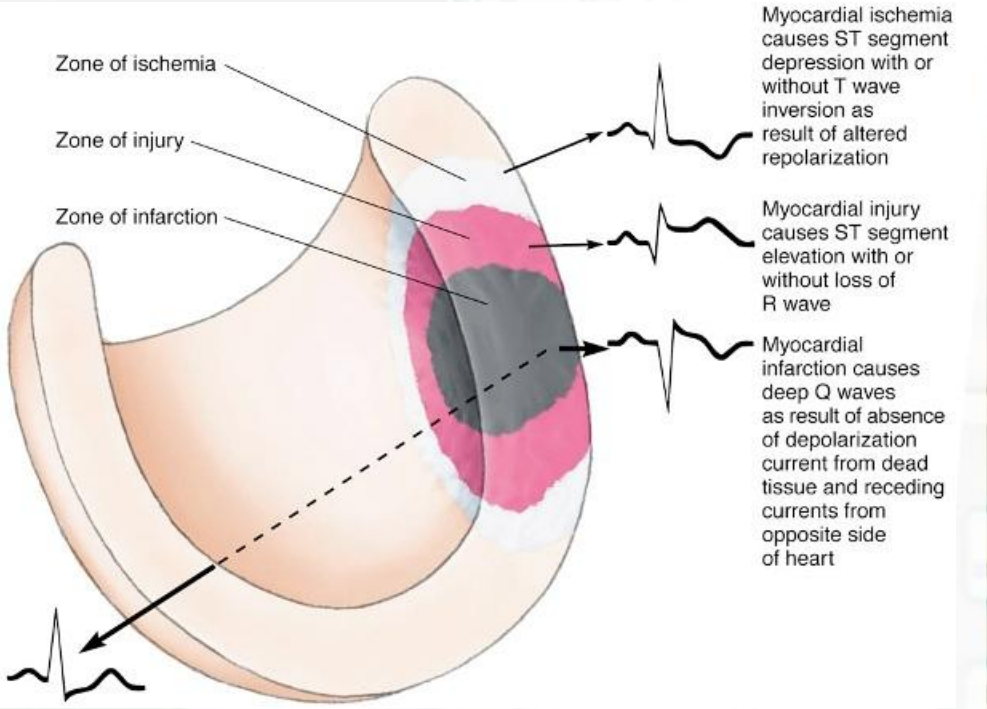


Injury

is recognized by ST segment elevation.

These changes are caused by persisted ischemia, which lead to more significant alteration of the repolarization processes.

The injury patterns are also reversible.
If a deficient blood supply persists injured muscle progresses to necrosis.



Necrosis or infarction

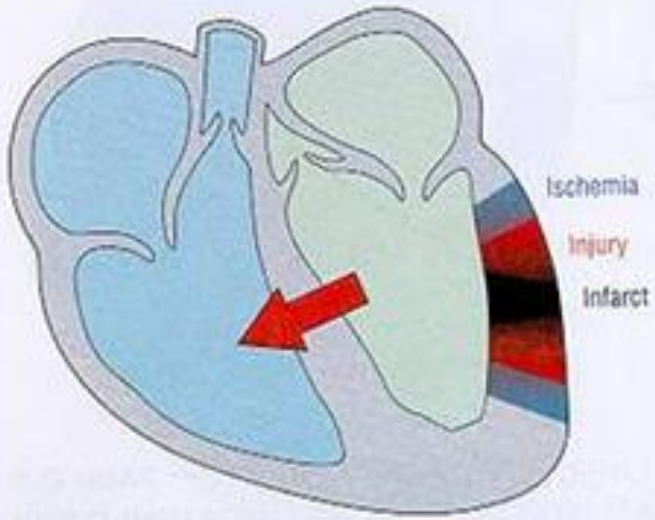
With infarction, "a dead zone" appears and become electrically inactive.

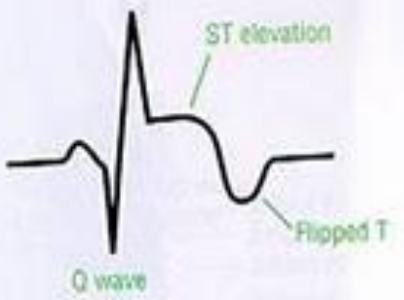
Dead or necrotic muscle tissue is inexcitable and incapable of producing an action potential.

Such tissue acts as a passive conductor of the potential forces generated in viable areas of the myocardium.

In the event of myocardial death, a Q wave appears and the R wave deflection decreases in amplitude or disappears.

The depth of the Q wave is directly proportional to the relative thickness of the dead zone, and the height of the R wave is directly proportional to the amount of living tissue that escapes death.





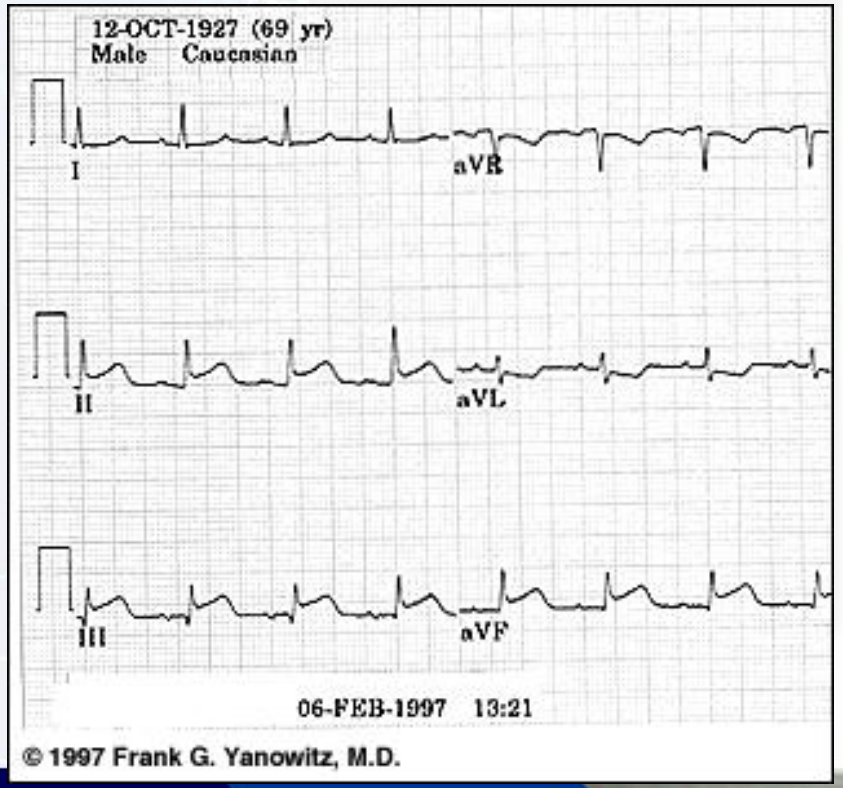
WHAT

WHEN

WHERE

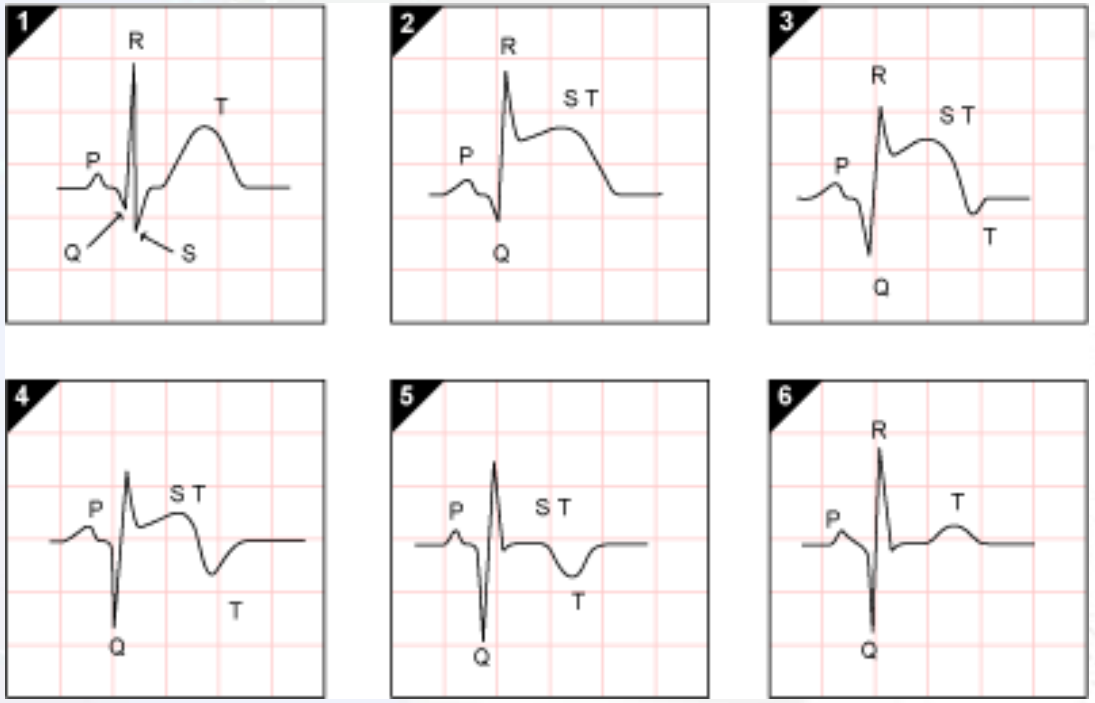
Diagnostic ECG signs of myocardial infarction

- Decreased R wave amplitude or its absence in the leads facing the necrotic myocardium. ($R_I + R_{II} + R_{III}$) : 3 = less than 5 mm.



Diagnostic ECG signs of myocardial infarction

- Deep and wide Q wave, deeper than $\frac{1}{4}$ of the R wave amplitude, and wider than 0.03 second.

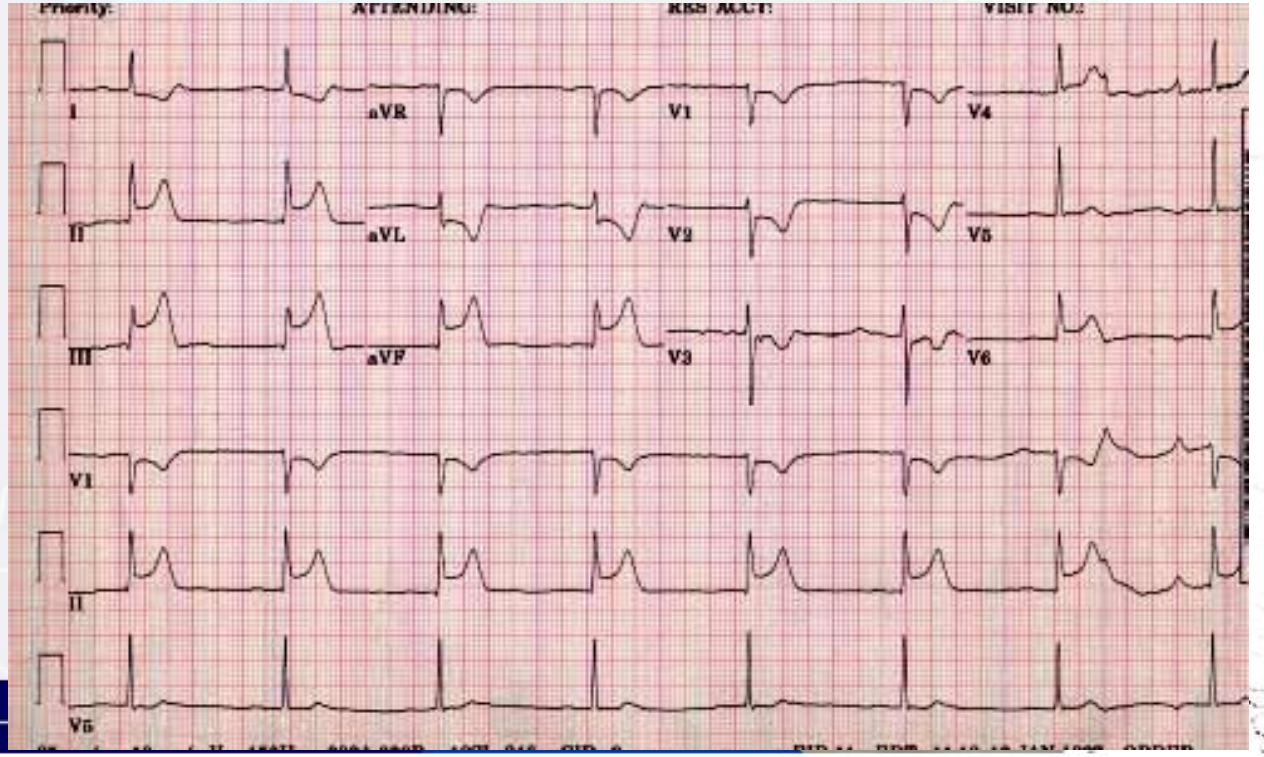


WHAT

Diagnostic ECG signs of myocardial infarction

- Pathological T wave – high ischemic or inverted (++, +-, --, -+).
- ST segment changes.
- Discordance of ST segment and T wave in opposite leads.

That is, ST elevation in the lead I and depression in the lead III; T wave upright in the lead I and inverted in the lead III. Concordance of ST segment and T wave in opposite leads is an ECG sign of the angina pectoris.

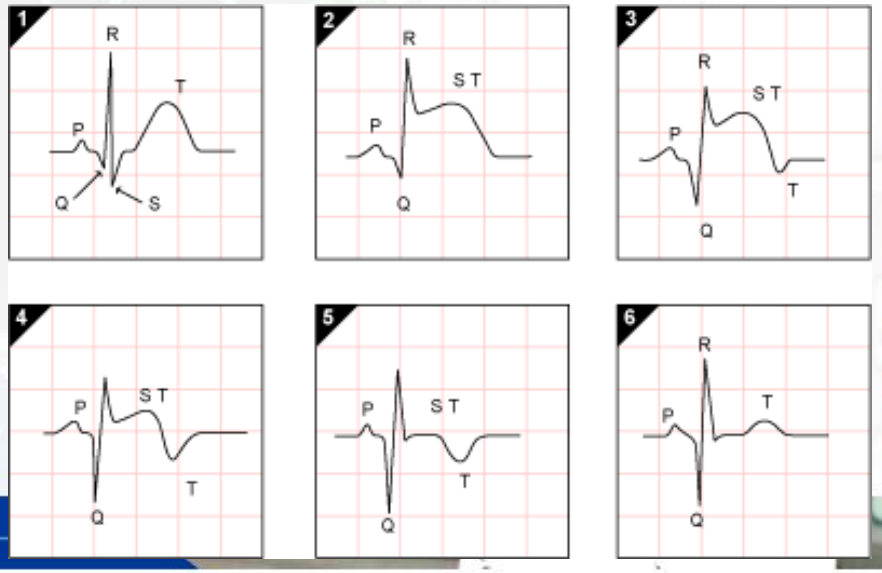


Evolution of myocardial infarction

The clinical myocardial injury pattern does not remain stationary, but changes.

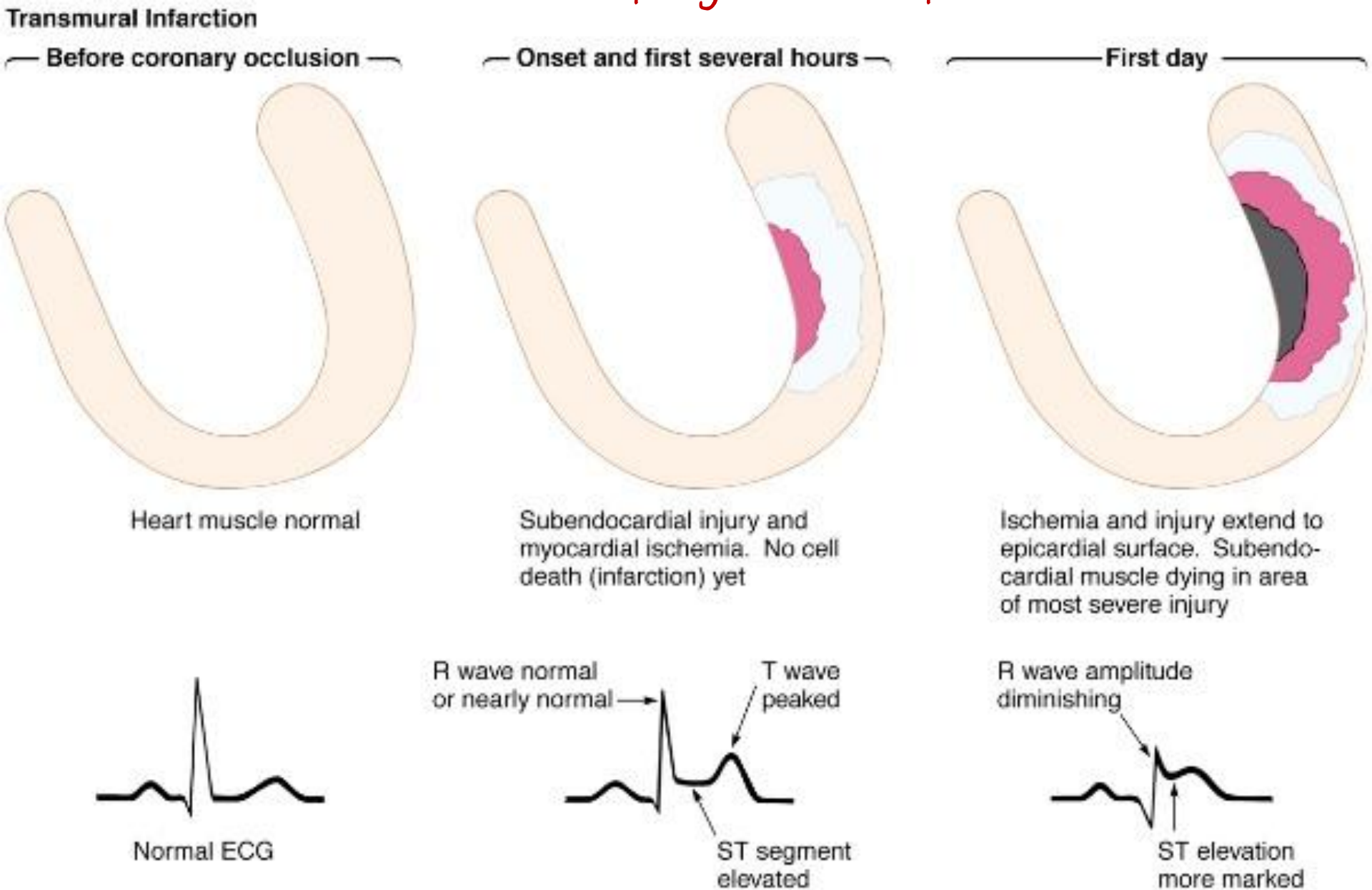
Evolutionary ECG alterations take place over a period of hours, days, weeks, month, or even years.

The series of changes has been arbitrarily separated into stages .

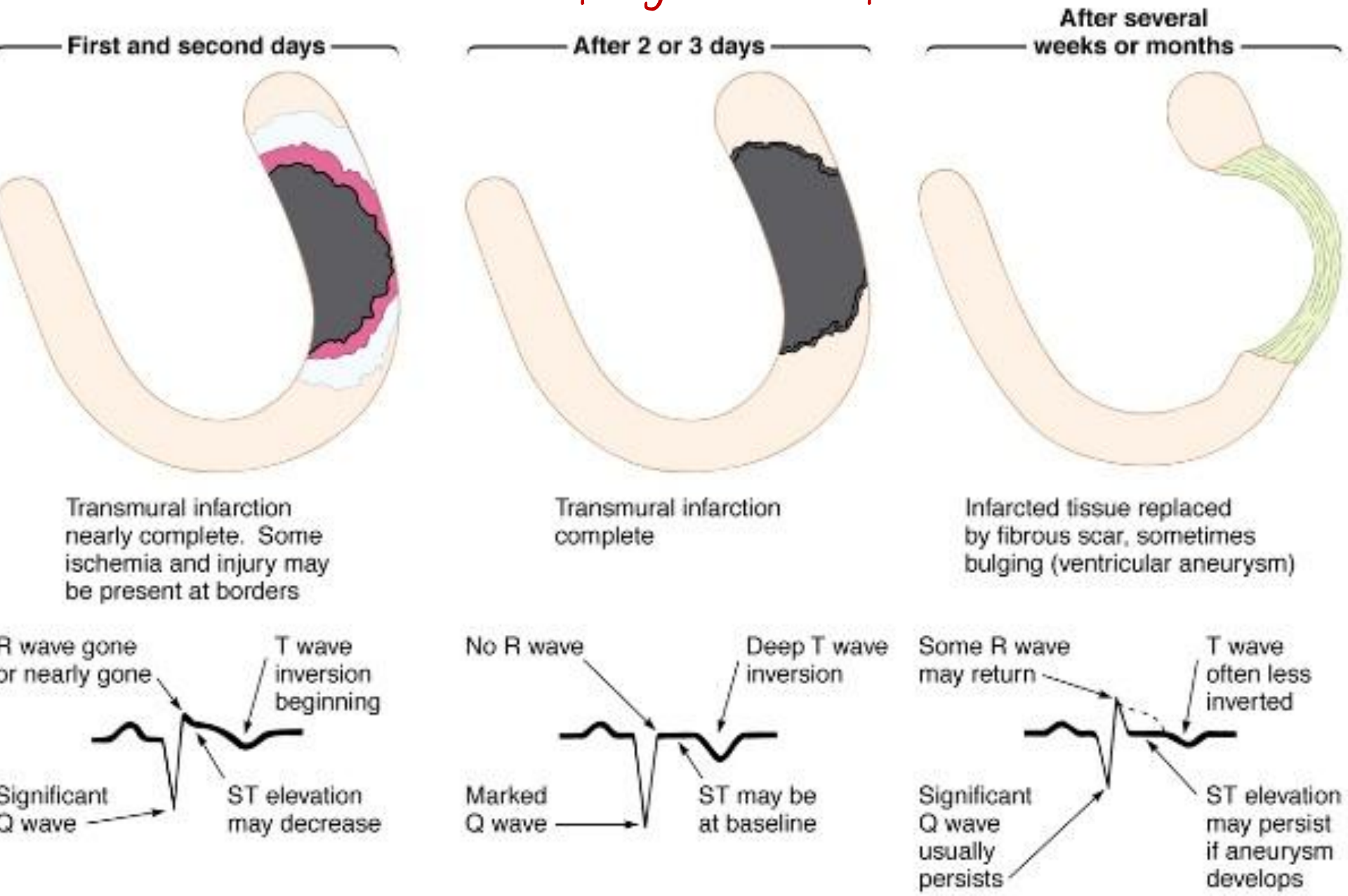


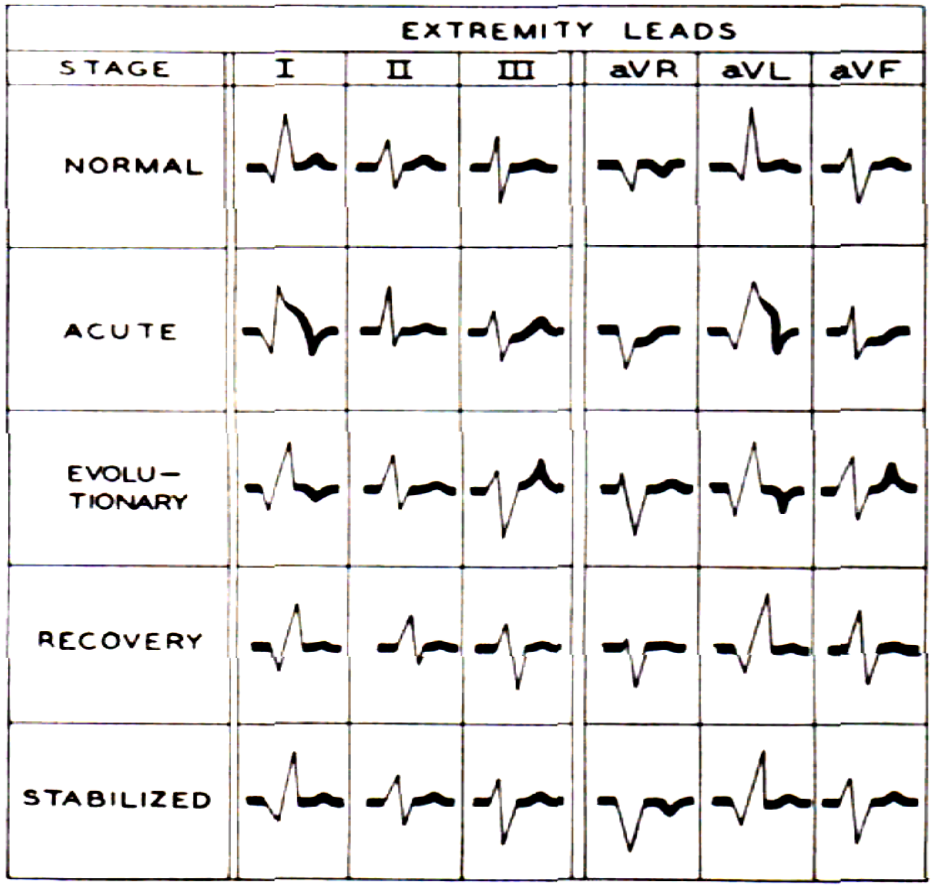
WHEN

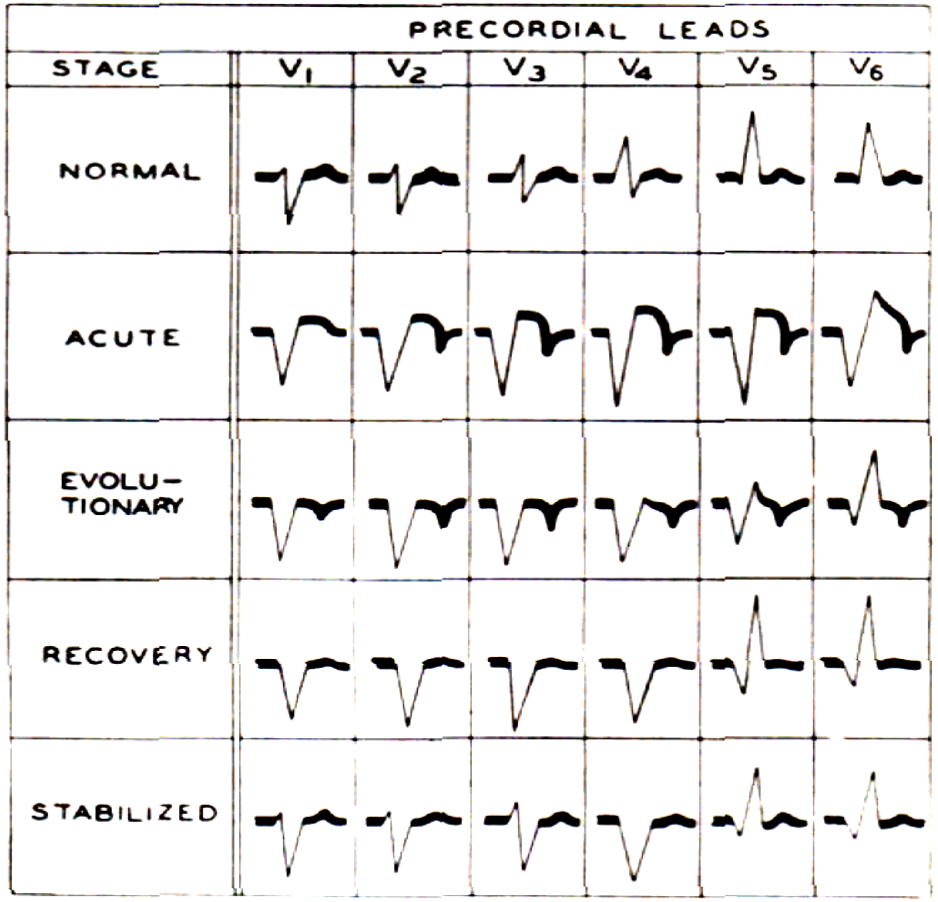
Evolution of myocardial infarction



Evolution of myocardial infarction

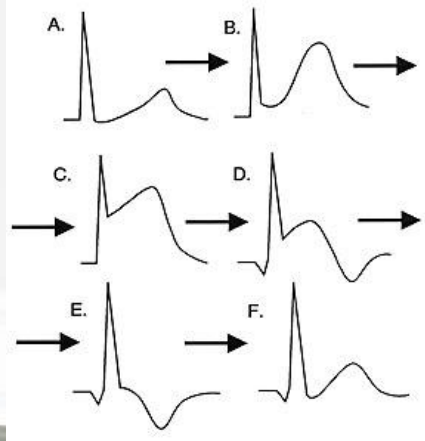






Stages of the myocardial infarction

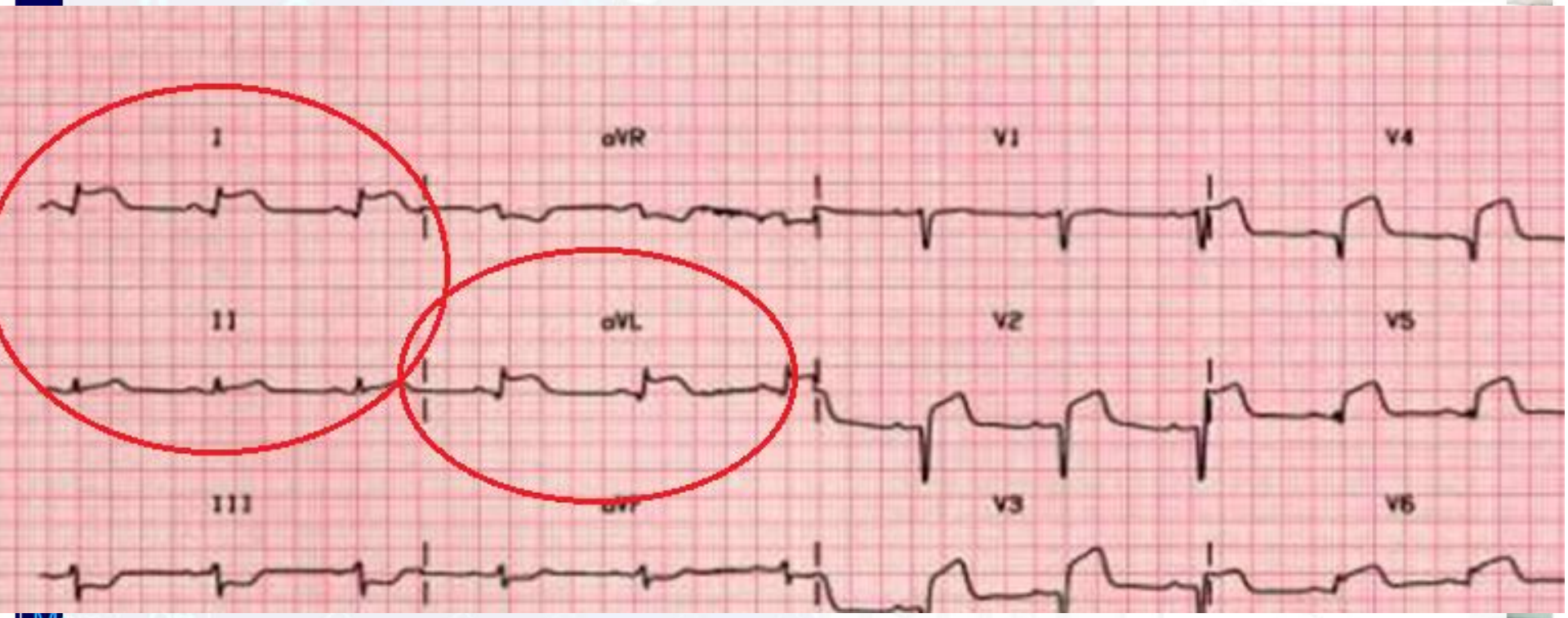
Stage 1 – acute: abnormal Q wave, elevated ST segment, and inverted T wave.
Stage 2 – evolutionary: deep Q wave, ST segment isoelectric, and inverted symmetrical T wave.
Stage 3 – recovery: deep Q wave, ST isoelectric, and T wave returning to normal.
Stage 4 – stabilized: T waves normal, only evidence of old infarction is the deep Q wave.



Evolution of Acute MI

Location of the myocardial infarction

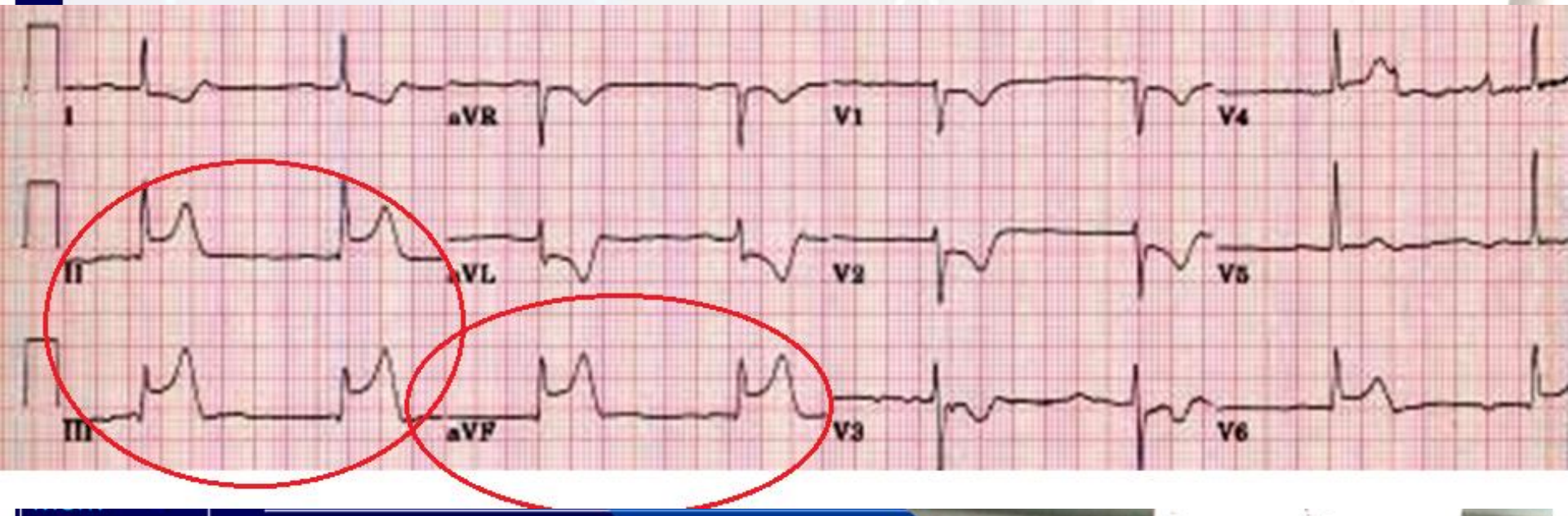
- Leads I, II, aVL – anterior infarction.



WHERE

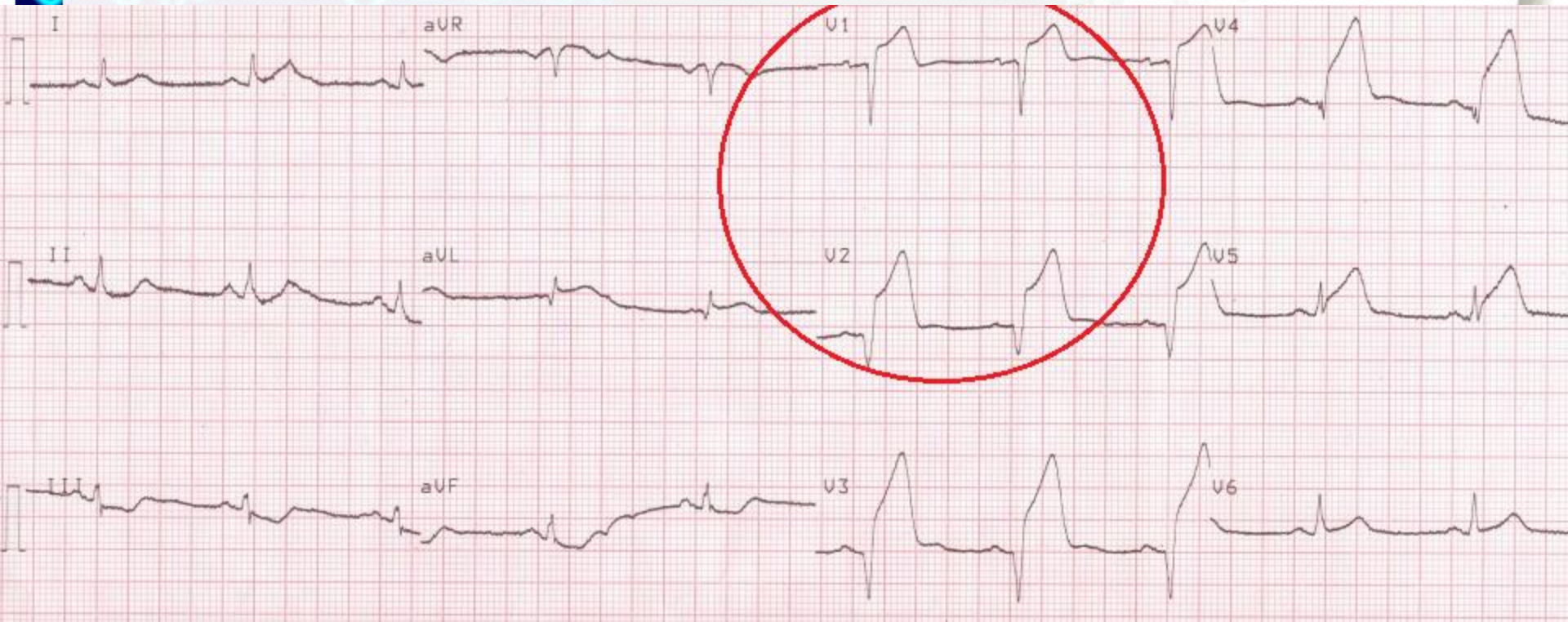
Location of the myocardial infarction

- Leads III, II, aVF – posterior infarction.



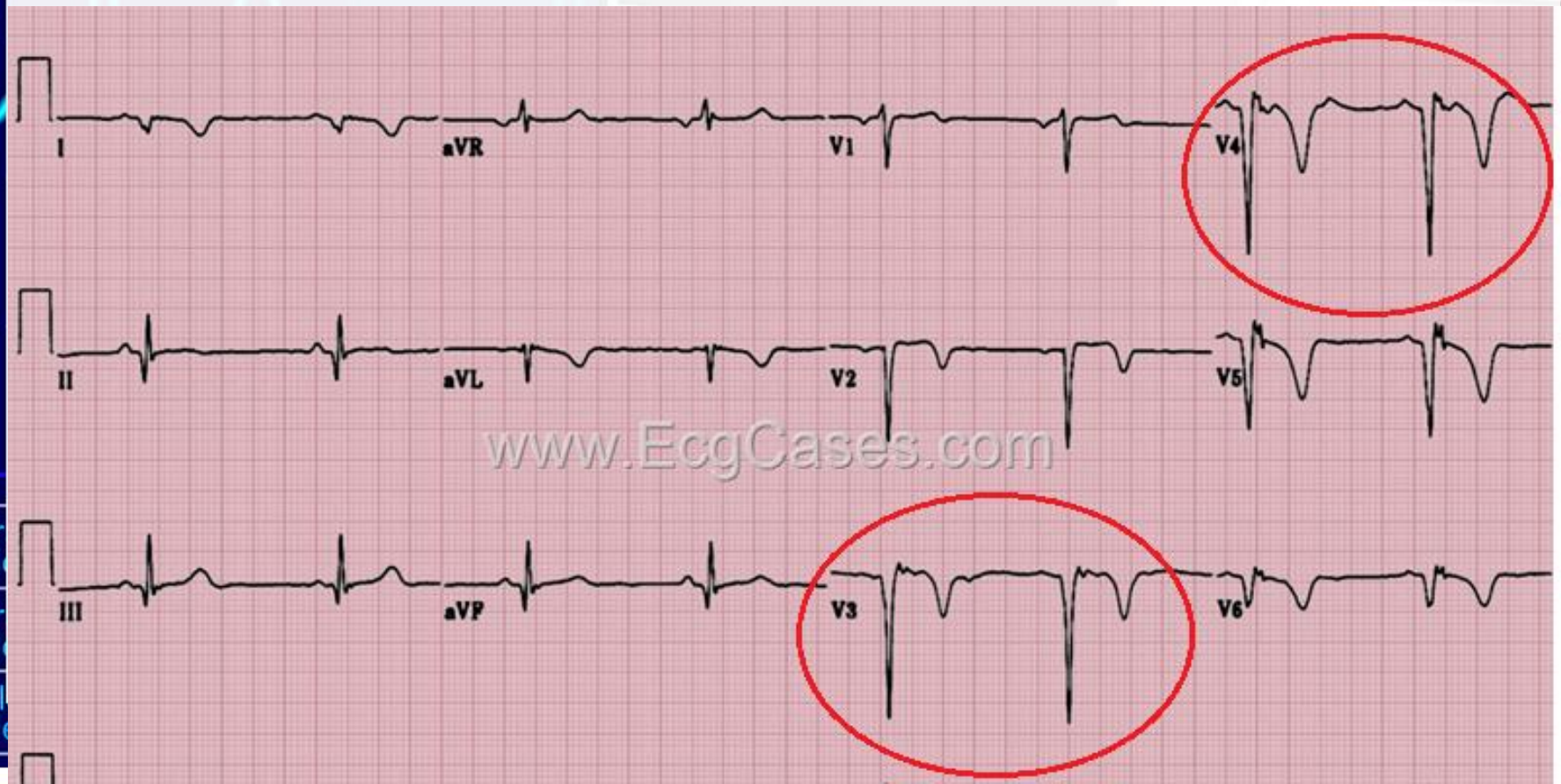
Location of the myocardial infarction

- V_1, V_2 – septal infarction.



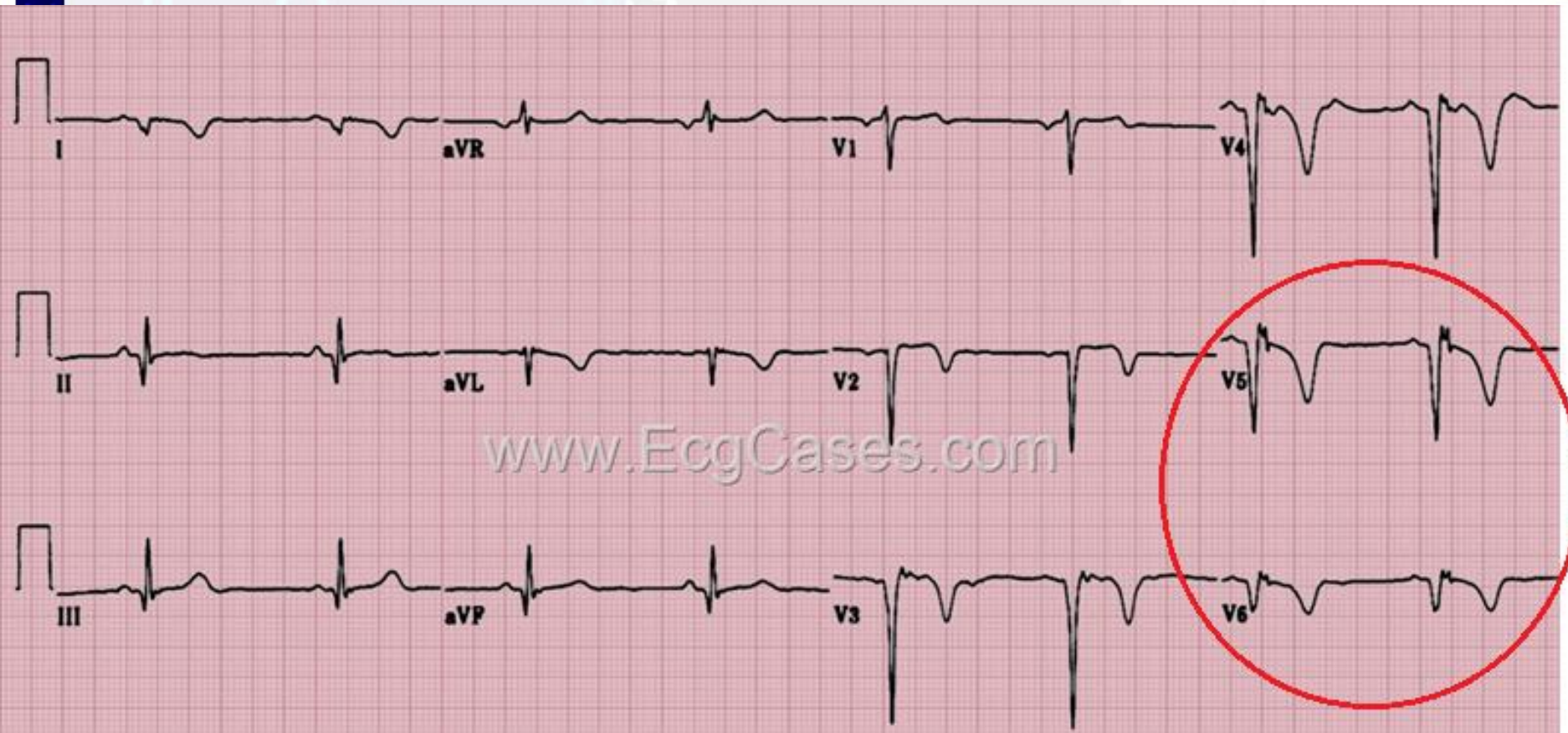
Location of the myocardial infarction

- V_3, V_4 – apical infarction.



Location of the myocardial infarction

- V_5, V_6 - lateral infarction.

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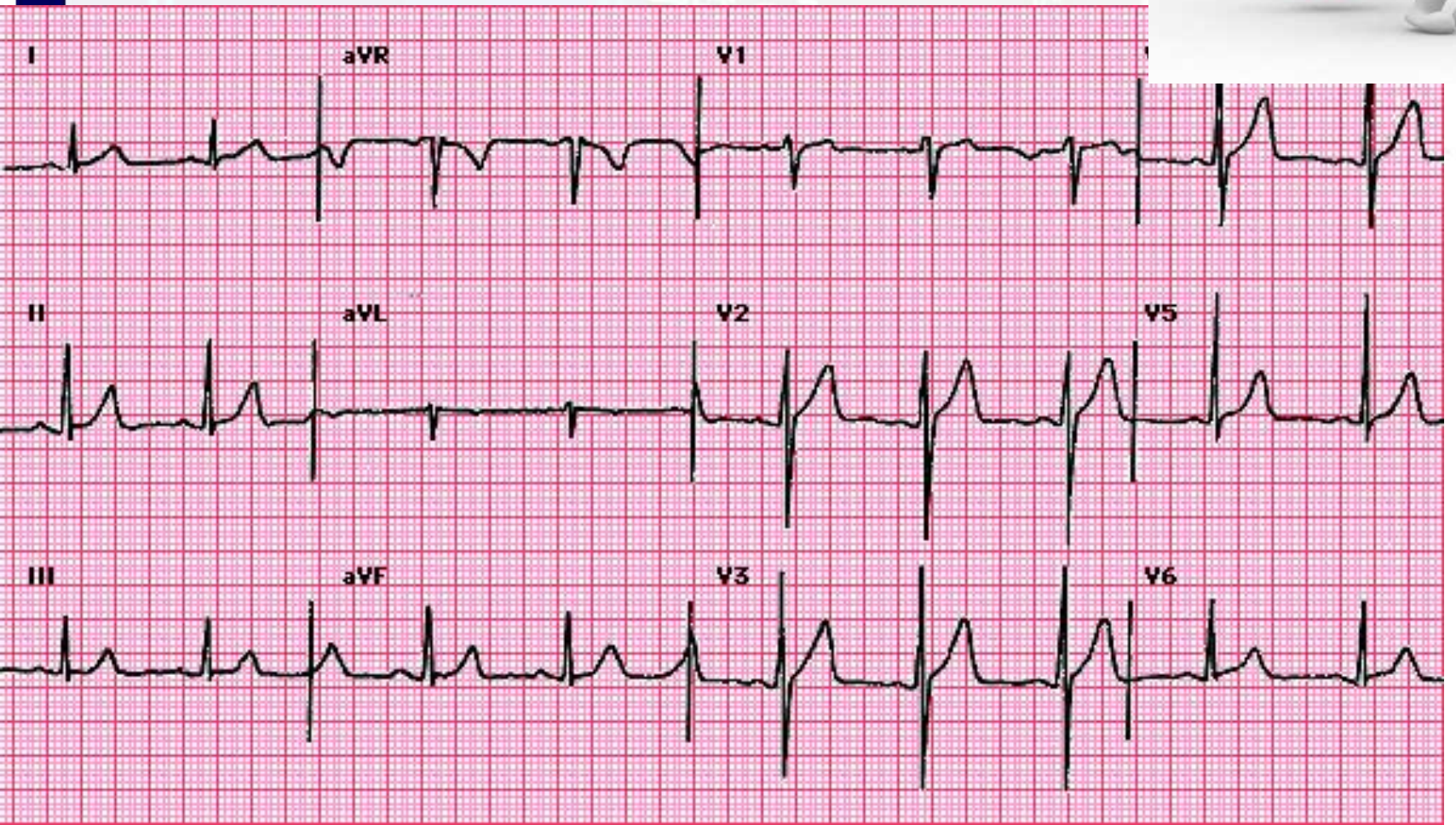
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V3
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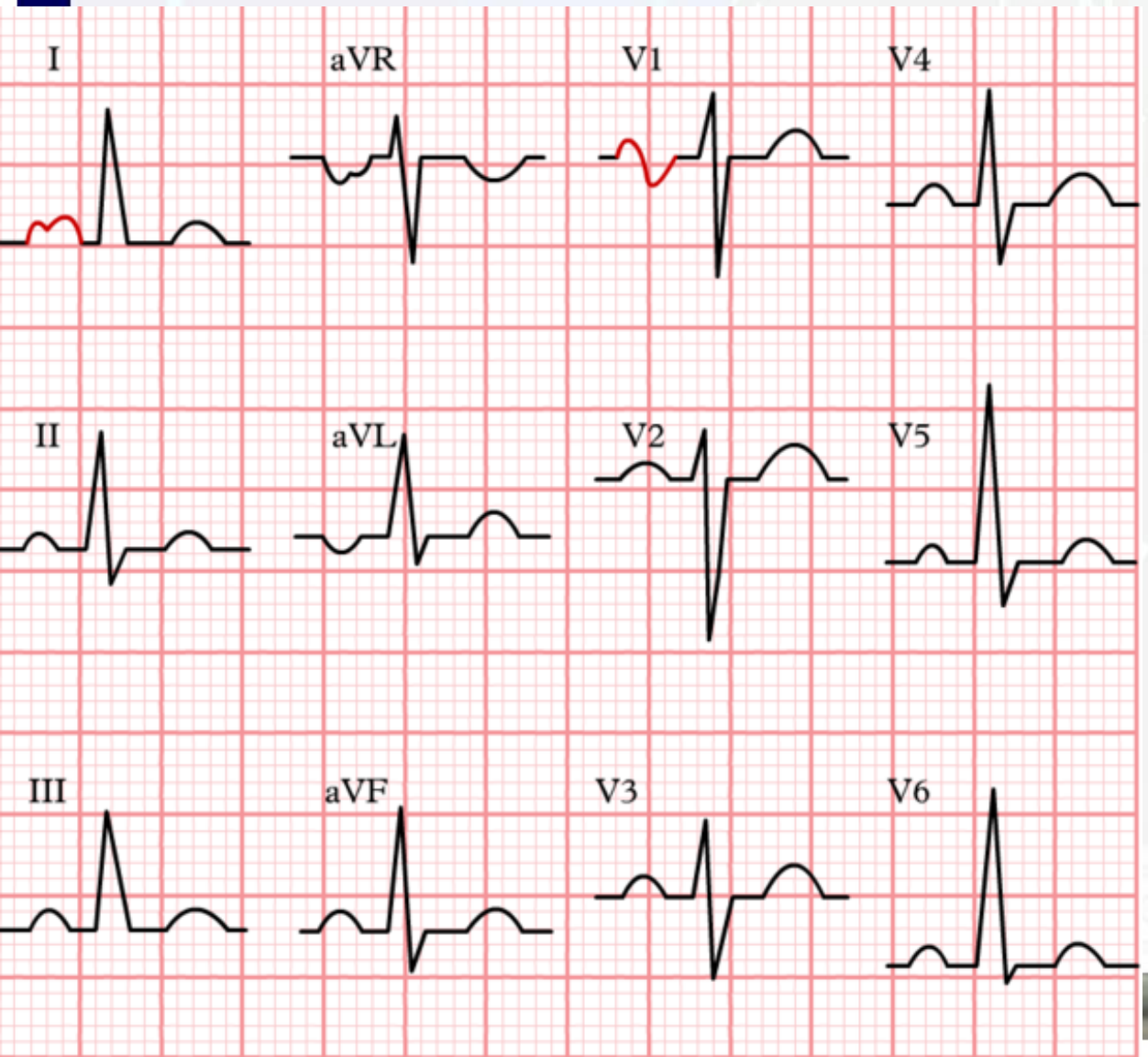
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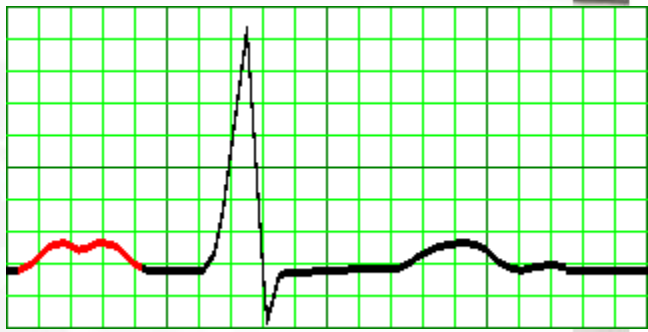

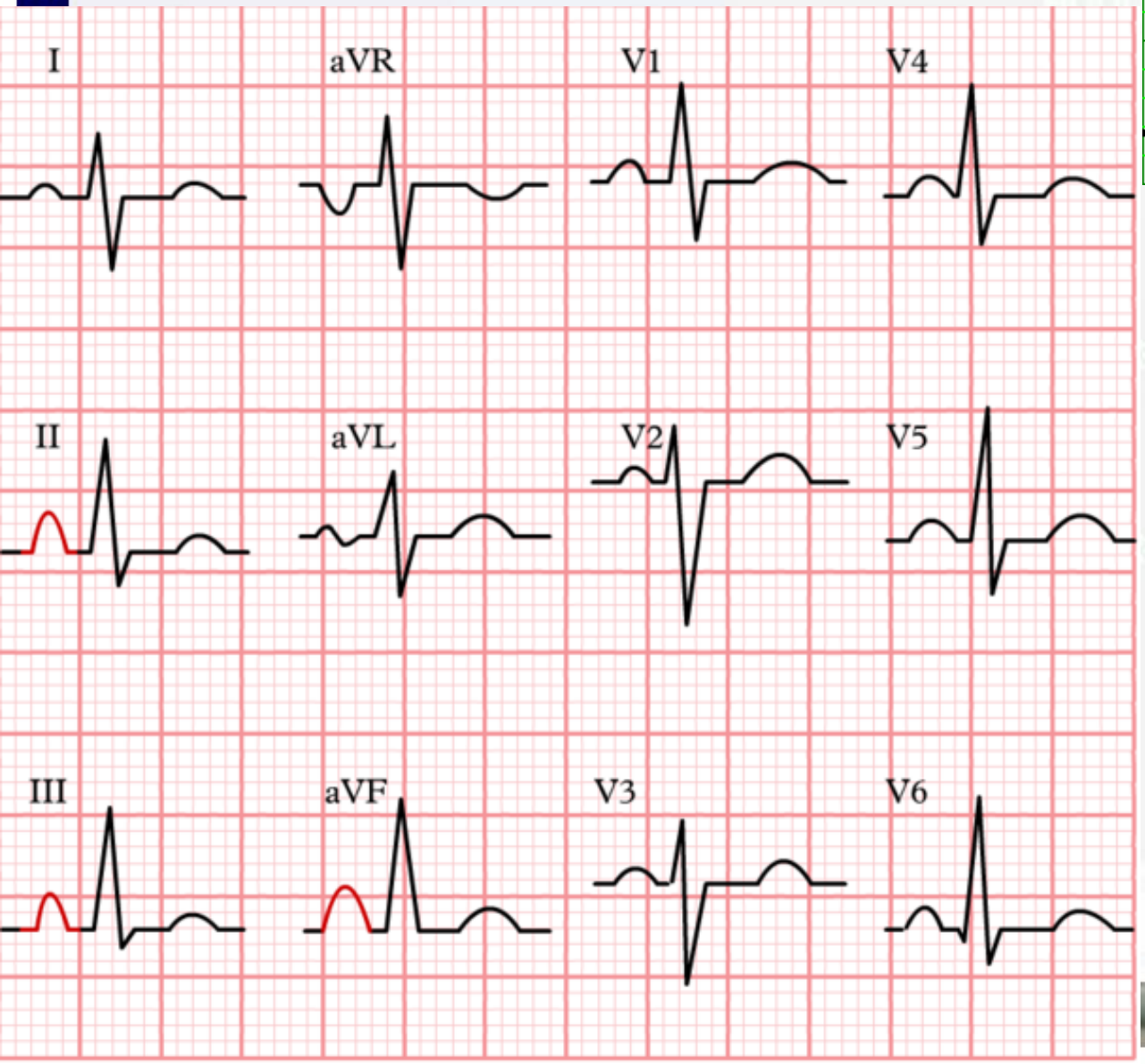
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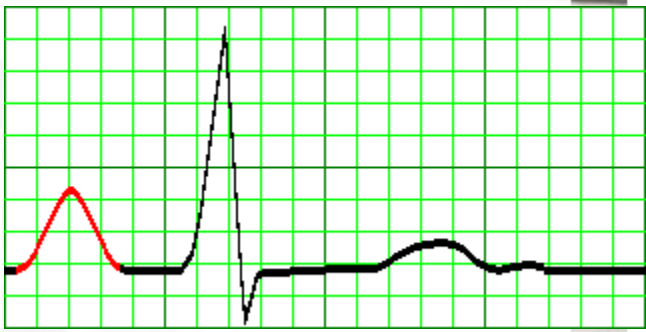

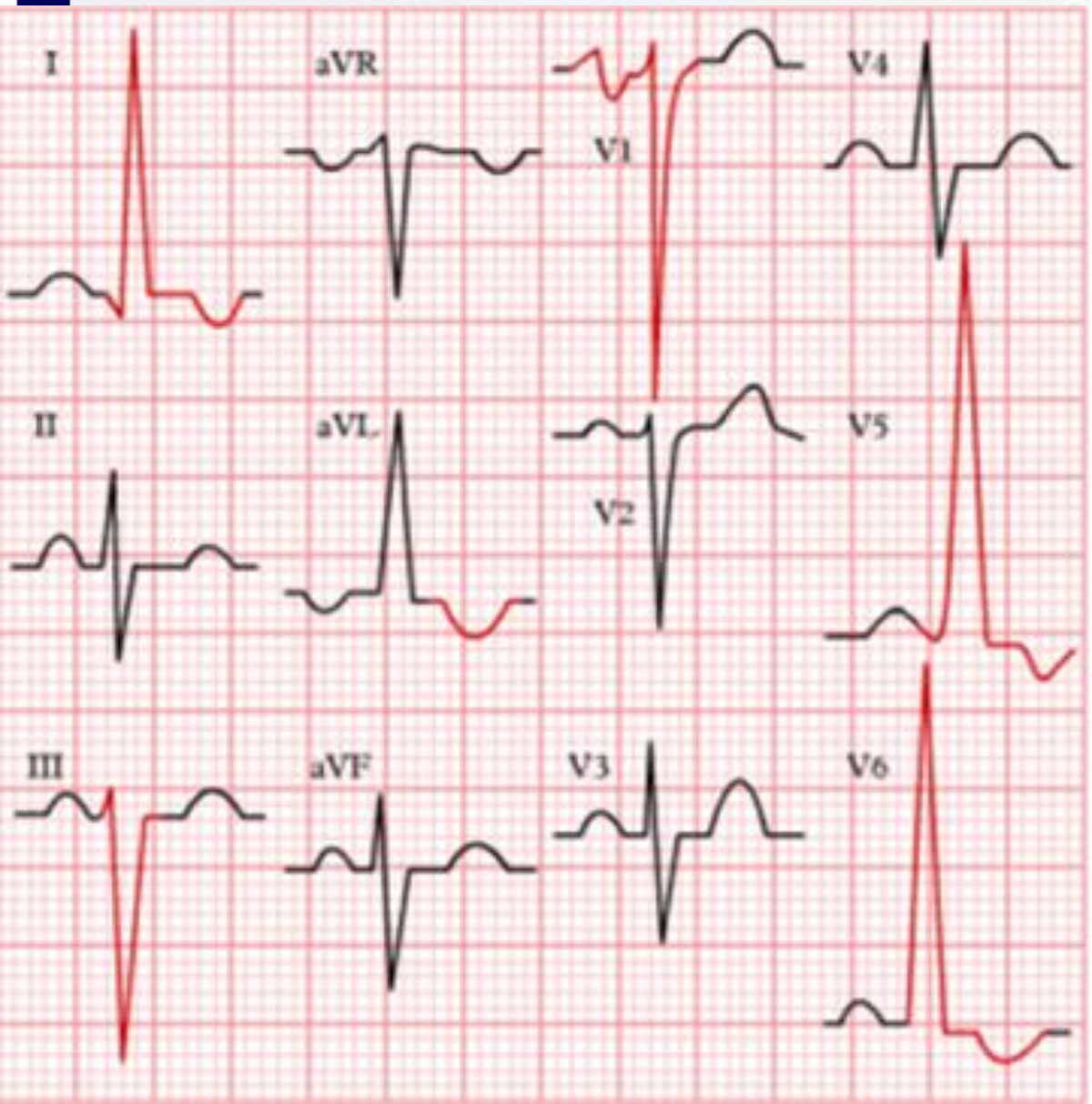
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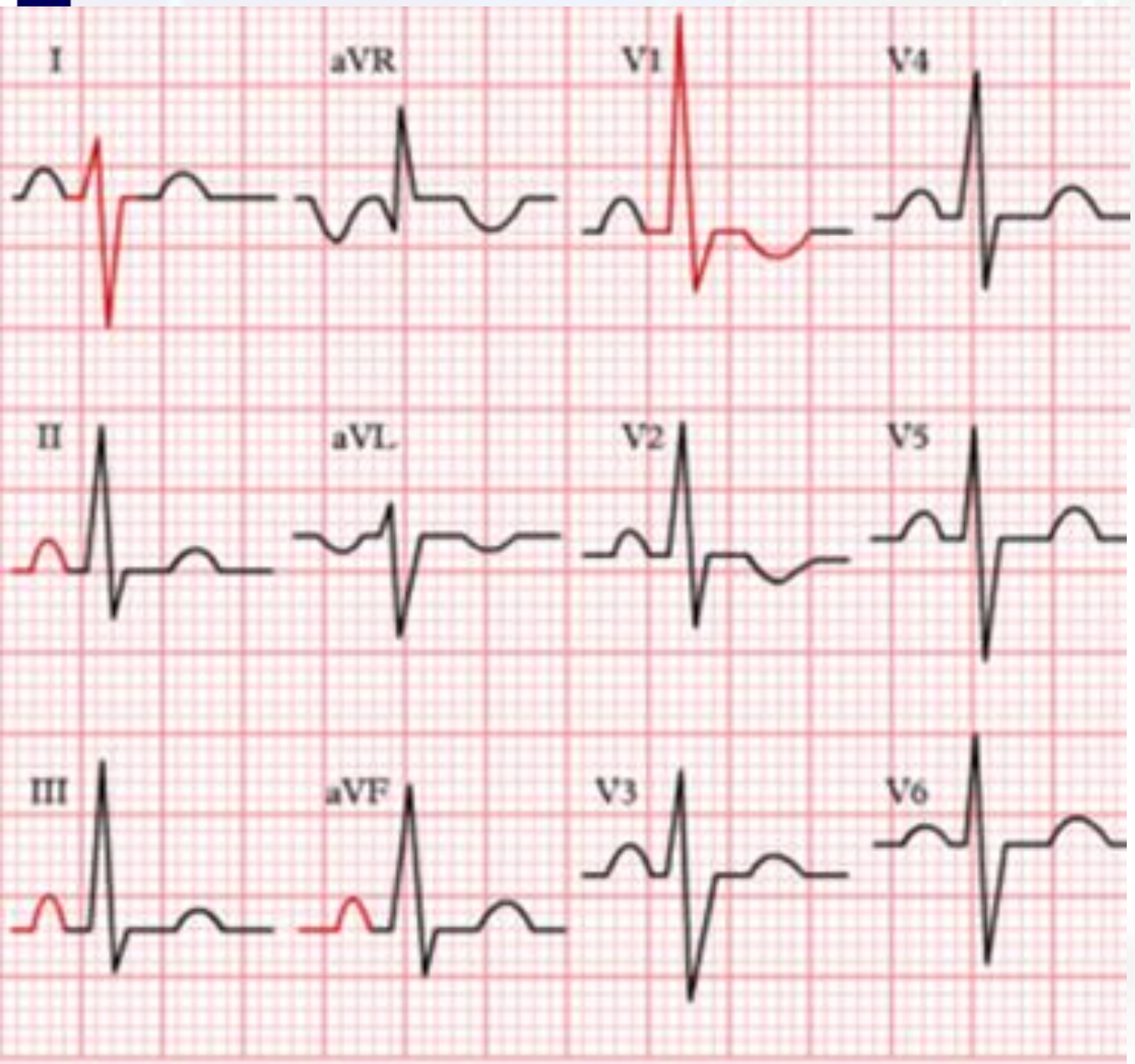
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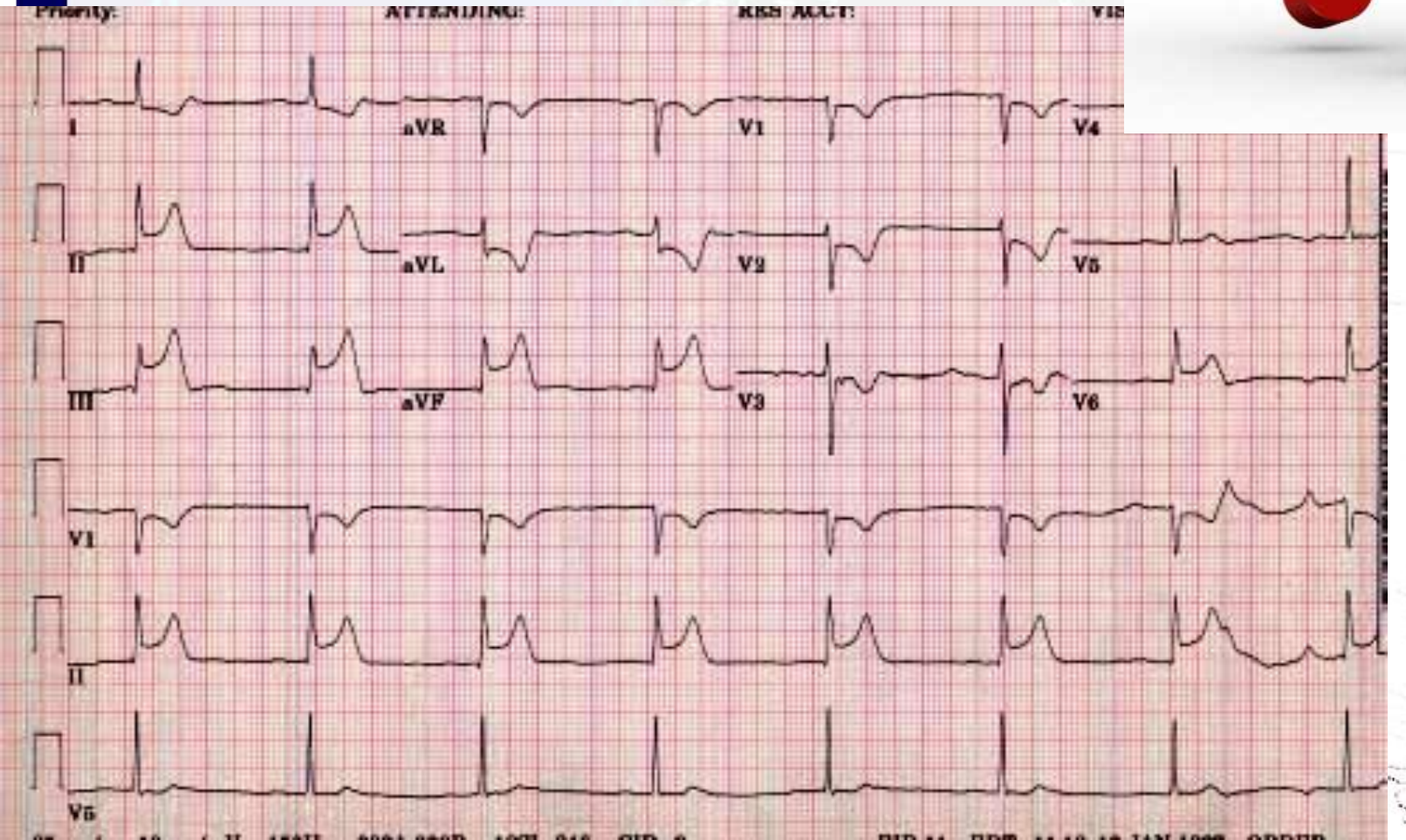
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