

ECG in cardiology. Part 1.

Distance education course for 5th and 6th year students.

**Holubkina E.O., Ass. Prof., Bogun L.V.,
Assoc. Prof., Polyvana O.S., Ass. Prof.,
Tykhonova T.M., Head of the Department of
Internal Medicine**

**Department of Internal Medicine
V. N. Karazin Kharkiv National University,
Kharkiv, Ukraine**



Introduction



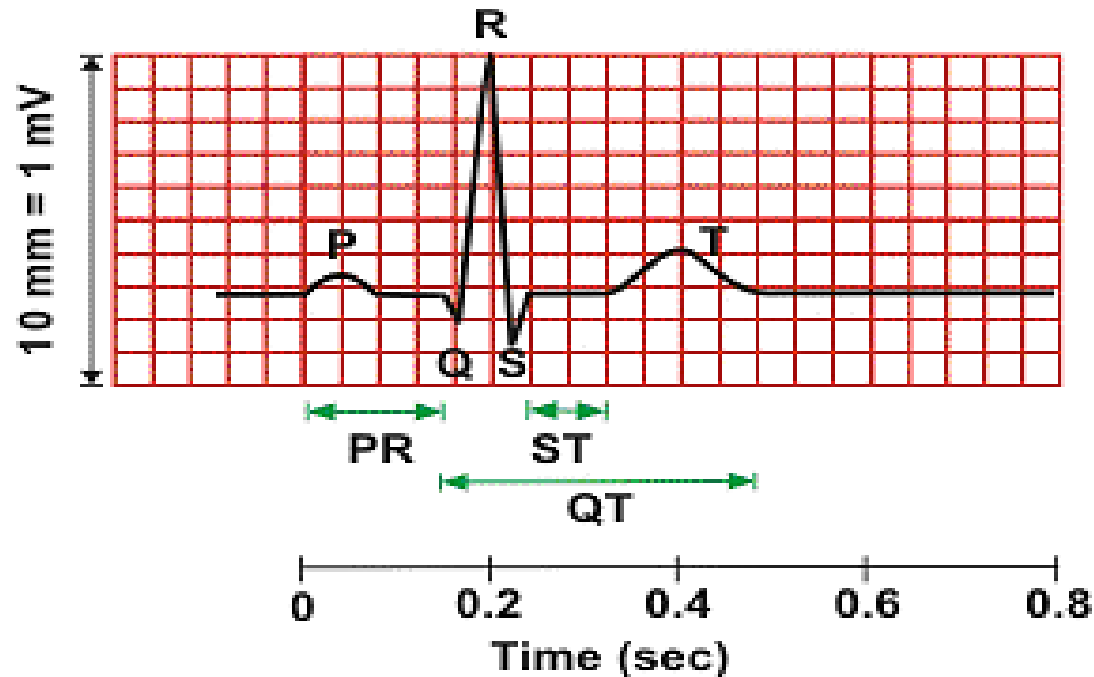
- ECG is highly available, rapid, non-invasive, inexpensive tool for diagnosis of cardiovascular diseases.
- ECG gives direct information only about abnormalities of electrical activity of the heart (rhythm and conduction disorders).
- ECG provides indirect information about other cardiac (hypertrophy of heart chambers, myocardial ischemia/infarction, pericarditis etc.) and non-cardiac conditions (potassium misbalance, drug (digoxin) toxicity, hypotermia etc). In this case other instrumental/laboratory tests are usually required.

ECG basics



- The ECG is a recording of the electrical activity of the heart.
- Each heart beat consists of one complete cycle of cardiac contraction and relaxation that begins when the sinus node (SA node) depolarizes spontaneously.
- Contraction is provided by mechanism of depolarization of cardiac cells, relaxation - by mechanism of repolarization. Thus cardiac cells can be in two states - resting state and depolarized state.
- The main ECG components include: P-wave, PR interval, QRS complex, T wave, ST segment, QT interval.

The main ECG components



P wave (0.08 - 0.10 s)

QRS (0.06 - 0.10 s)

P-R interval (0.12 - 0.20 s)

Q-T_c interval (≤ 0.44 s)*

$$*QT_c = \frac{QT}{\sqrt{RR}}$$

The list of the topics



The main cardiovascular diseases included to the part 1 of this course are:

1. Arterial hypertension
2. Stable forms of ischemic heart disease
3. Acute coronary syndrome

Arterial hypertension



Introduction



According to the WHO arterial hypertension (AH) is a chronic condition characterized by the constant pumping of blood through blood vessels with excessive force.



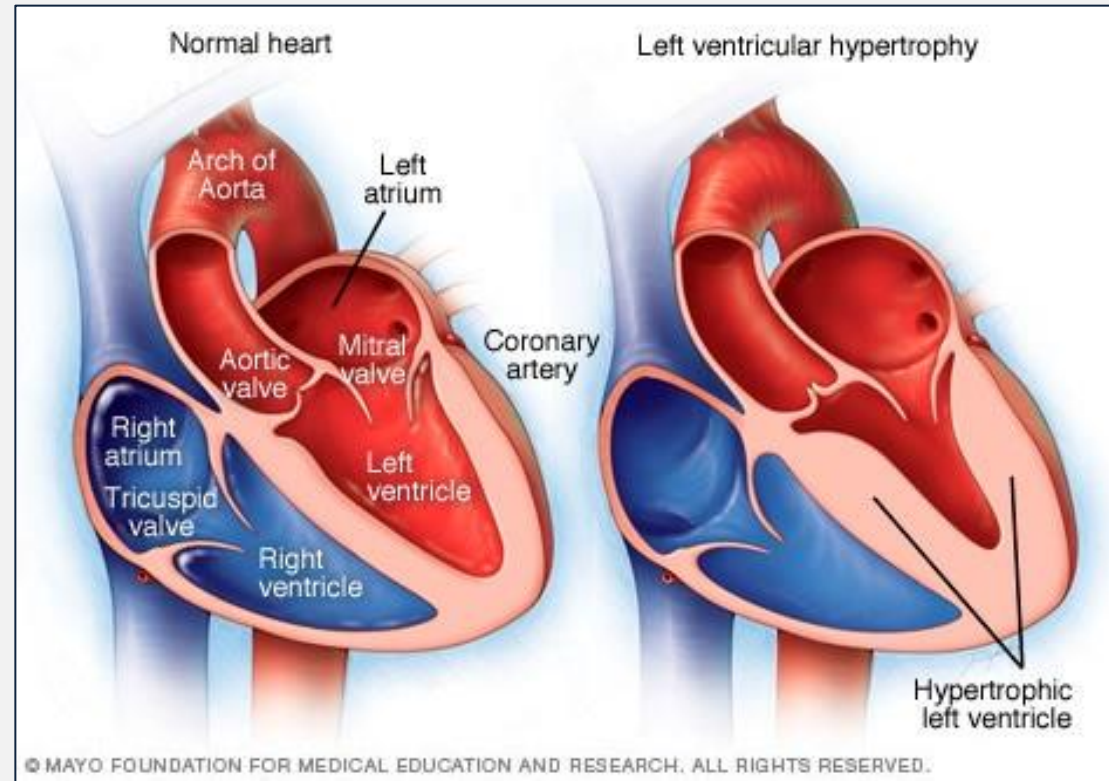
Due to 2018 ESC/ESH Guidelines **AH is defined as:**

- office systolic blood pressure (SBP) values ≥ 140 mmHg and/or
- diastolic blood pressure (DBP) values ≥ 90 mmHg.

Heart remodeling in AH



- High BP places a pressure load on the heart and may lead to left ventricular hypertrophy (LVH);
- Left ventricular hypertrophy (LVH) is an abnormal increase in left ventricular mass.



- LVH is the most common target organ damage in AH, and AH is the commonest cause of LVH.

ECG in AH: key points



- The ECG criteria for diagnosing LVH have low sensitivity - near 50% (which means that ~50% of patients with LVH have absent ECG criteria).
- However, the LVH criteria have high specificity >90% (which means that if the ECG criteria are met, it is very likely that LVH is present).
- The presence of LVH can be definitely confirmed by echocardiography.

ECG signs of LVH

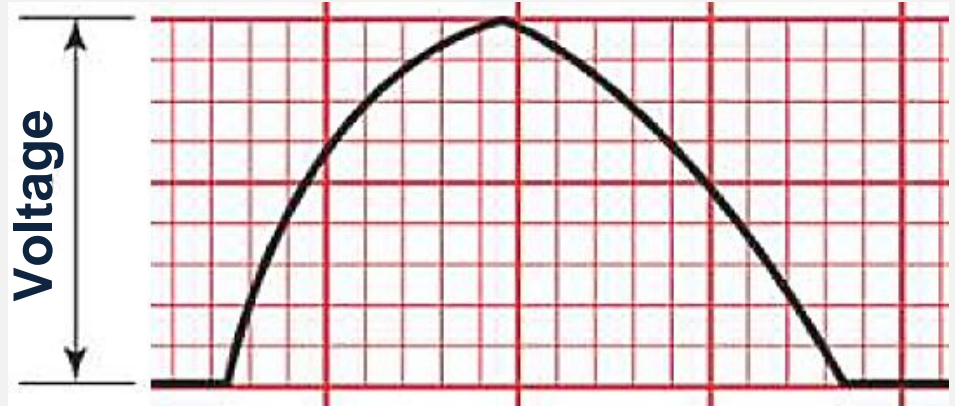


- Left axis deviation (not specific, can be seen in another pathological conditions, the other common cause is left anterior fascicular block);
- Voltage criteria (Sokolow - Lyon's criteria, Cornell voltage criteria, etc. based on the presence of high voltage which is characteristic for LVH)
- Delayed intrinsicoid deflection of the QRS complex (prolongation of initial ventricular depolarization) in lateral leads (V5 and V6).
- Widened QRS/T angle (i.e., left ventricular strain pattern, or ST-T oriented opposite to QRS direction)

High voltage



- Voltage is an amplitude of ECG waves;
- High voltage is the characteristic sign of LVH



Low voltage QRS



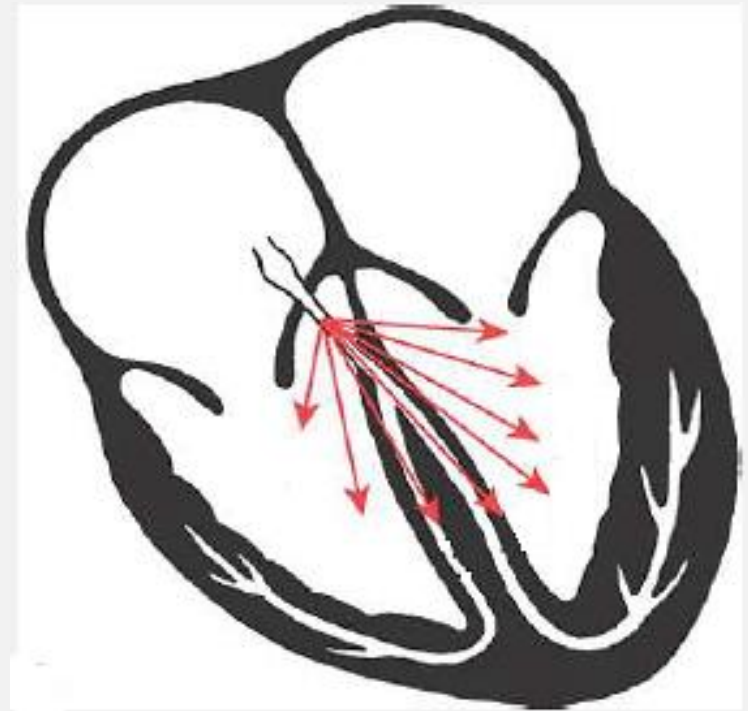
High voltage QRS



Mean electrical vector of the heart



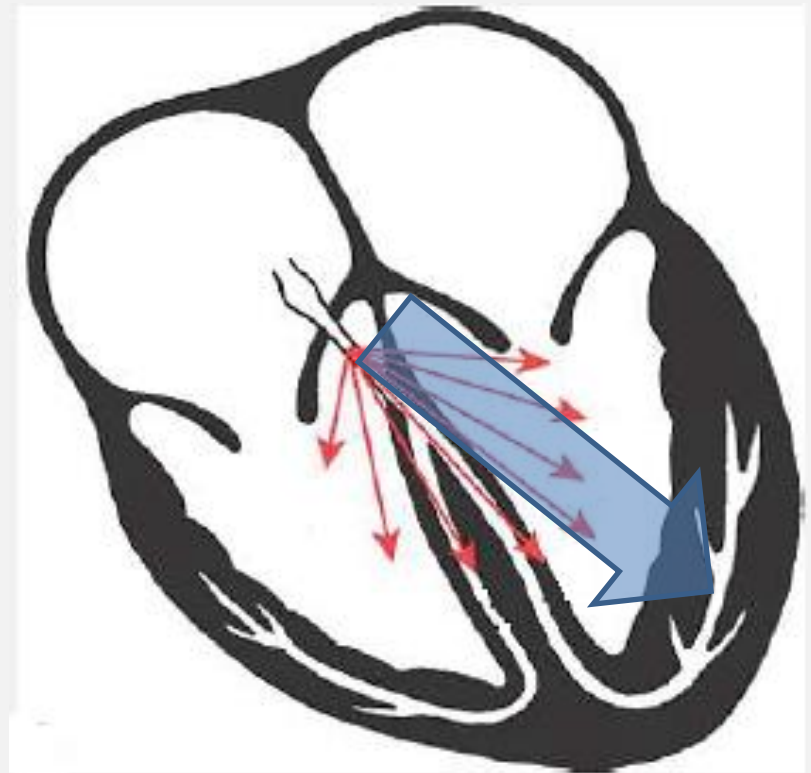
- When the ventricles undergo depolarization, the wave of depolarization that spreads across the muscle mass occurs in many different directions simultaneously (**red arrows**).
- The vectors swing progressively leftward because the electrical activity of the much larger left ventricle increasingly dominates.



Mean electrical vector of the heart



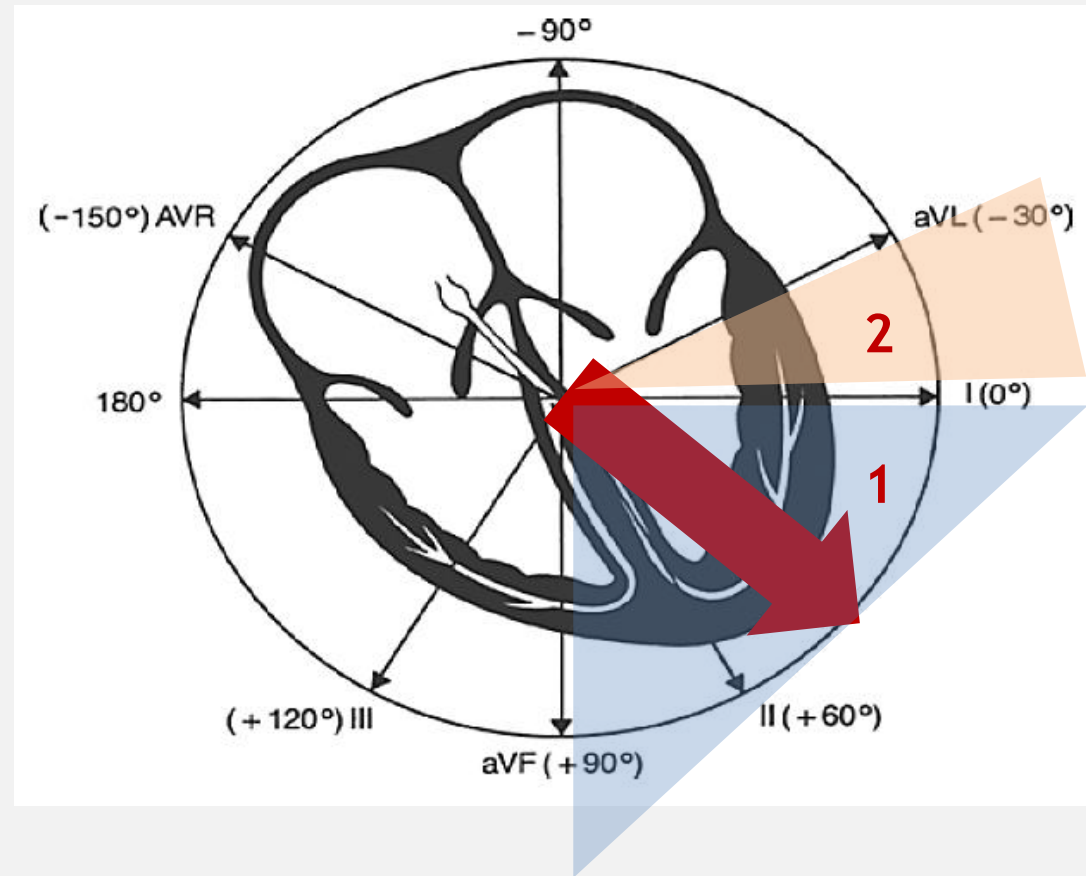
- The average vector of all of the instantaneous vectors is called the mean electrical vector (blue arrow) and normally points leftward and inferiorly.
- The direction of the mean vector is called the electrical axis of the heart or the QRS axis.
- The axis of the P wave and T wave also can be defined.



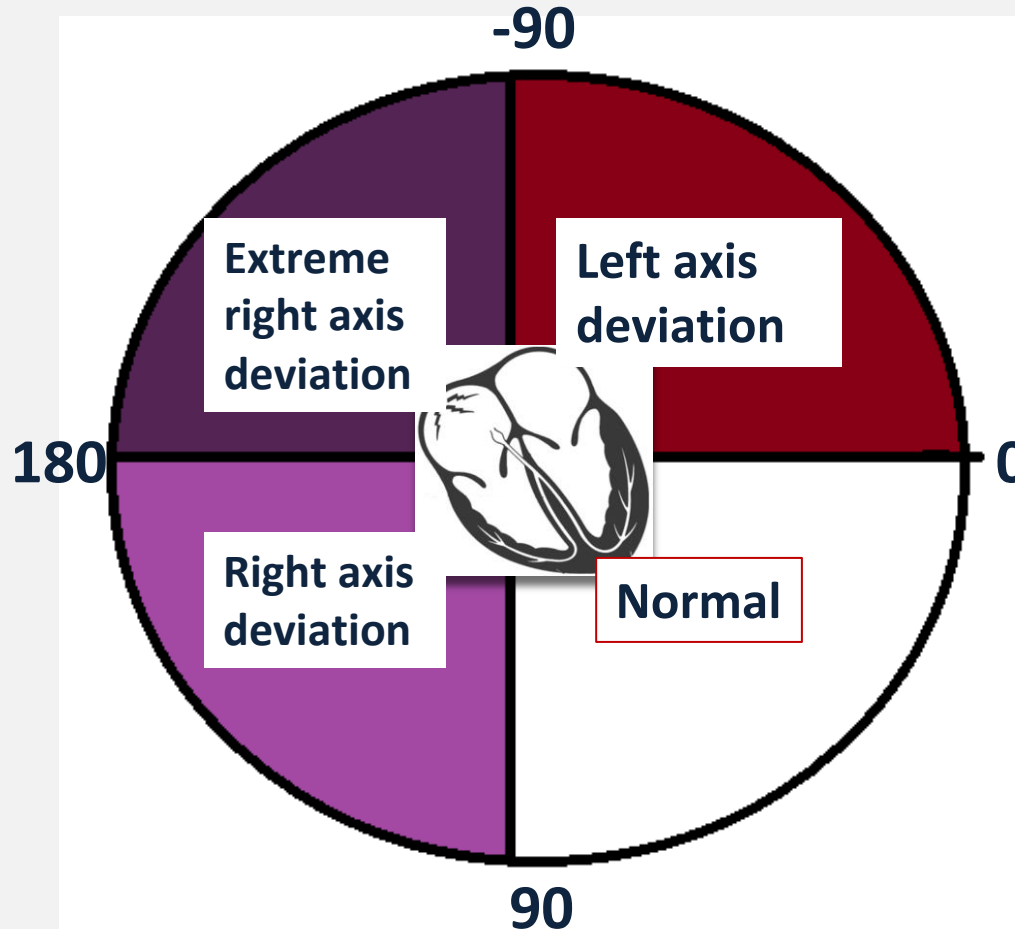
Electrical axis of the heart



- Normally the position of the mean electrical axis lies in the range: 0 - 90 (1).
- Some researchers extend it at the level: -30 - 90 (2).





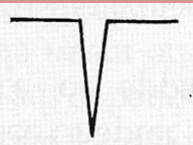
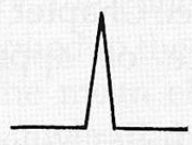
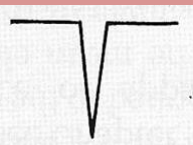
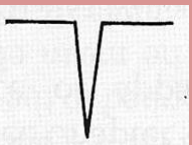


Positions of electric axis of the heart



Electric axis determination (fast method)

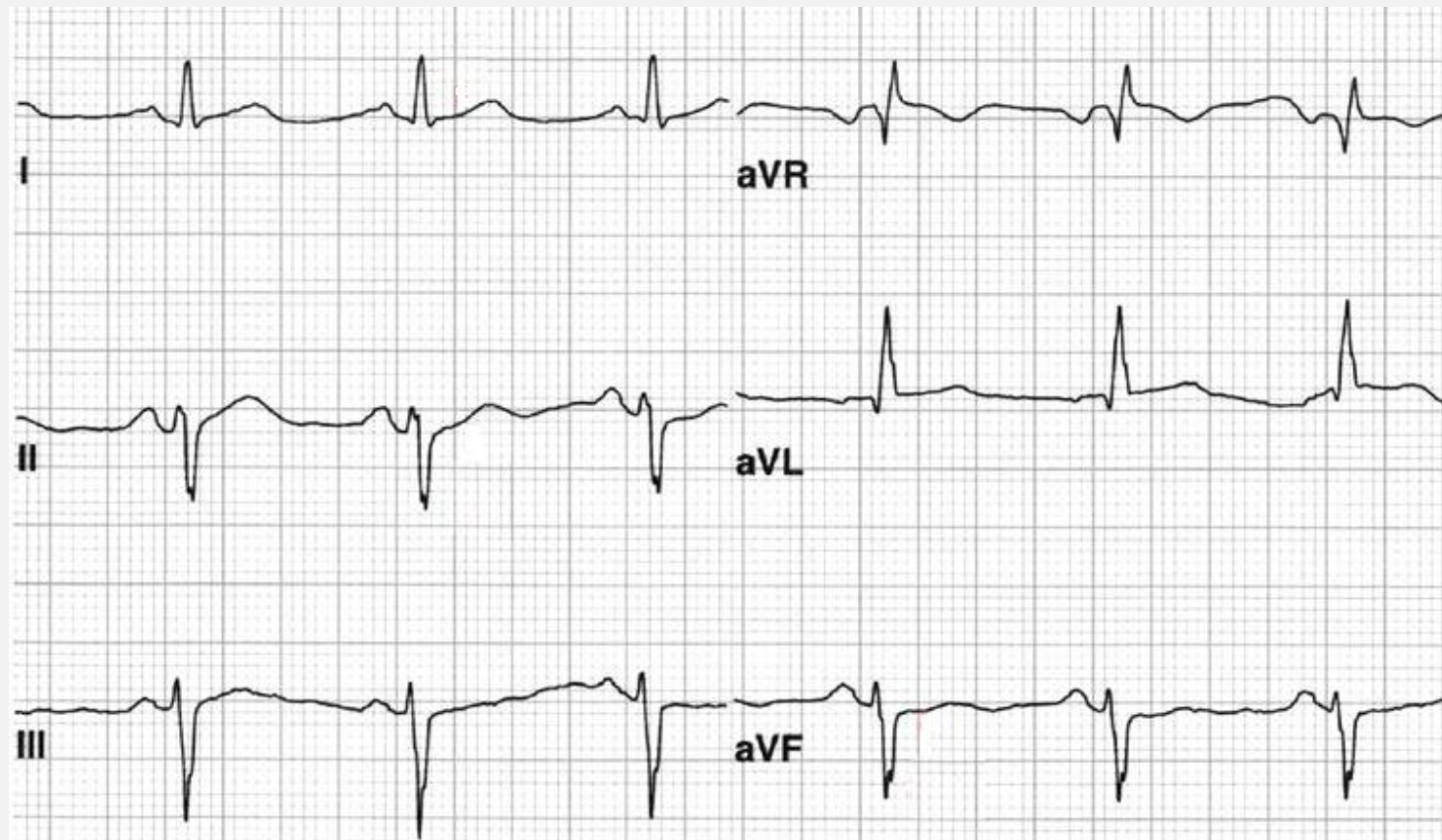


Lead I	Lead aVF	Description	Axis
+ 	+ 	Dominant R wave in lead I, aVF	<u>Normal</u> 0 to 90 degrees
+ 	- 	Dominant R wave in lead I, dominant S wave in aVF	<u>Left axis deviation</u> 0 to (-90) degrees
- 	+ 	Dominant R wave in lead I, dominant S wave in aVF	<u>Right axis deviation</u> 90 to 180 degrees
- 	- 	Dominant S wave in lead I, aVF	<u>Extreme right axis deviation</u> -90 to 180 degrees

Practical part



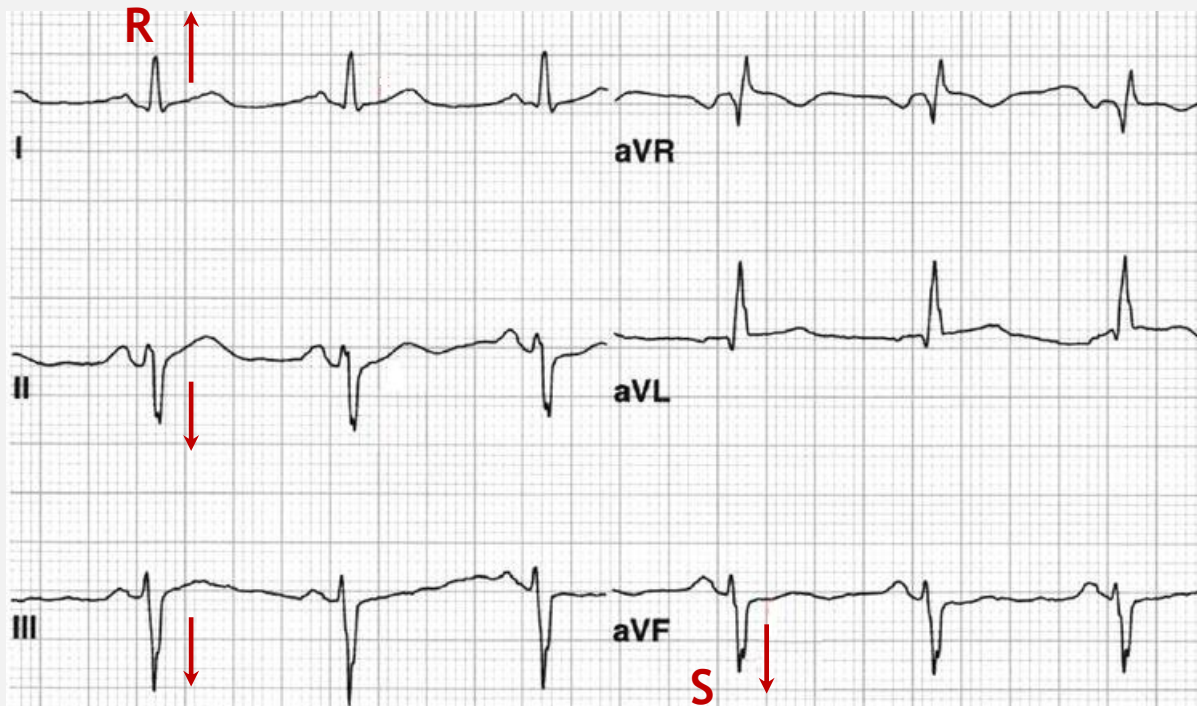
- What is position of the electrical axis of the heart on this ECG?



Practical part



- Lead I: R wave is dominant (so called positive QRS);
- Lead III: S wave is dominant (negative QRS);
- Also negative QRS in leads II, avF;
- It's left axis deviation



https://www.researchgate.net/profile/Sanjay_Sharma32/publication/234099418/figure/fig5/AS:300132902948868@1448568668528/ECG-demonstrates-abnormal-left-axis-deviation-defined-as-frontal-plane-QRS-axis-of-less_W640.jpg

Voltage Criteria for LVH -1



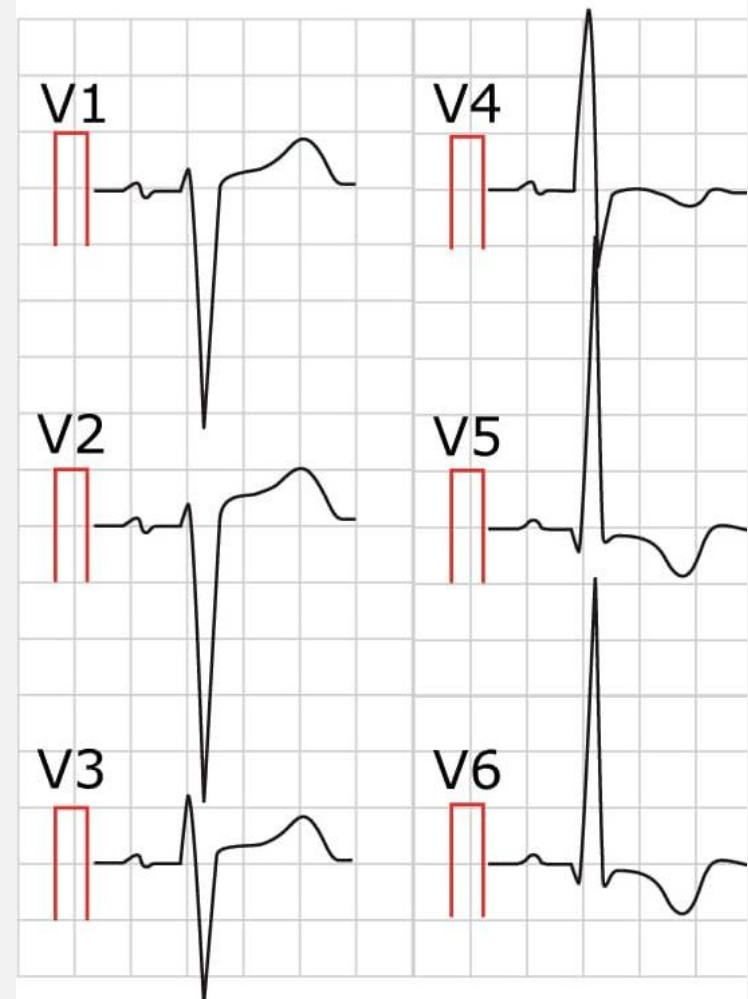
Sokolow - Lyon's criteria are the most commonly used criteria for LVH:

- R in $V5$ or $V6$ + S in $V1$ or $V2$ > 35 mm;

NB! Choose the deepest S wave in $V1$ or $V2$ (not both!), tallest R wave in $V5$ or $V6$ (not both!);

- R in aVL > 11mm;
- R in $V5$ or $V6$ > 26 mm.

A) Left ventricular hypertrophy (LVH)



Voltage Criteria for LVH -2



Cornell Voltage Criteria

- S in $V3 + R$ in $aVL > 24$ mm (men)
- S in $V3 + R$ in $aVL > 20$ mm (women)

Other Voltage Criteria for LVH:

Limb-lead voltage criteria:

- R in aVL 11 mm or, if left axis deviation, R in aVL 13 mm plus S in III 15 mm
- R in $I + S$ in $III > 25$ mm

Chest-lead voltage criteria:

- S in $V1 + R$ in $V5$ or $V6$ 35 mm

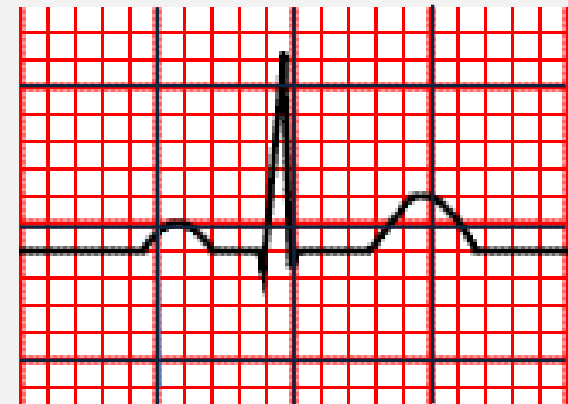
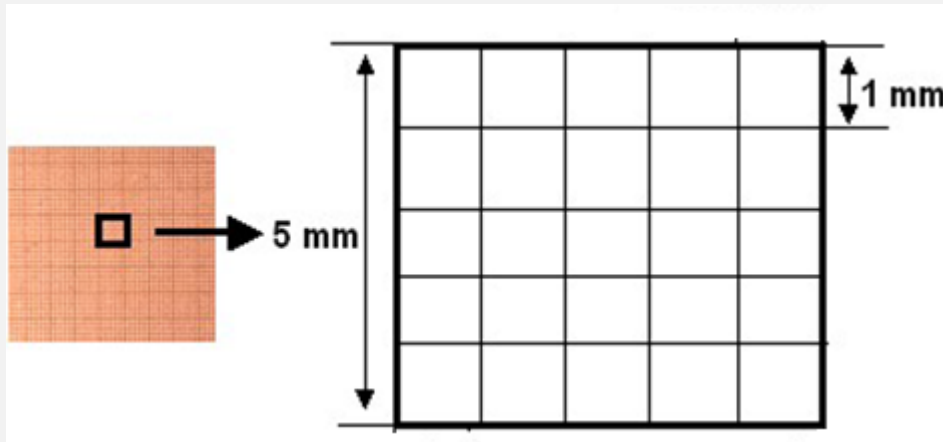
New (Peguero-Lo Presti criteria):

- Deepest S wave in any lead (SD) + $SV4 > 22$ mm (women) or > 27 (men)

How to calculate -1



- The amplitude of waves can be calculated in mm. Each small box on ecg paper is estimated as 1mm. Each big box is estimated as 5 mm (it contains 5 small boxes).



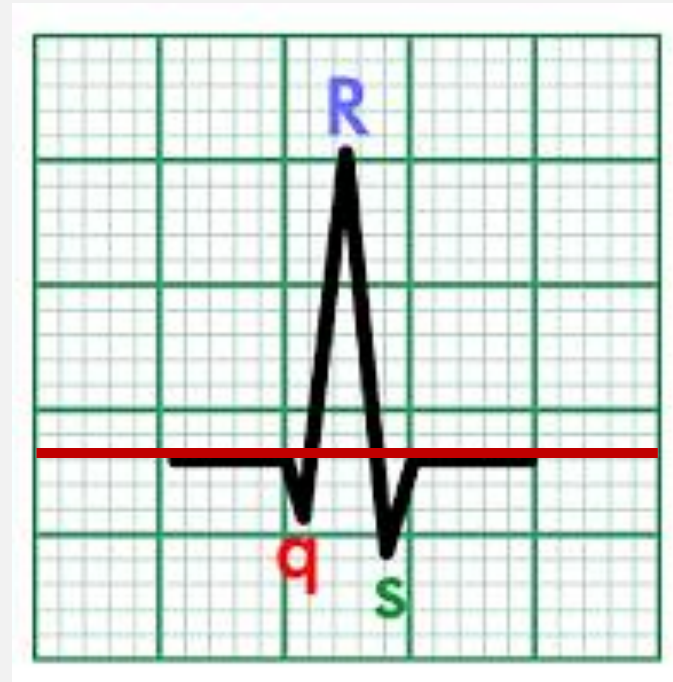
How to calculate -2



Amplitude should be counted from baseline (red color line).

In this picture:

- R wave amplitude is:
13 small boxes = 13 mm
- S wave amplitude is:
4 small boxes = 4 mm



Practical part

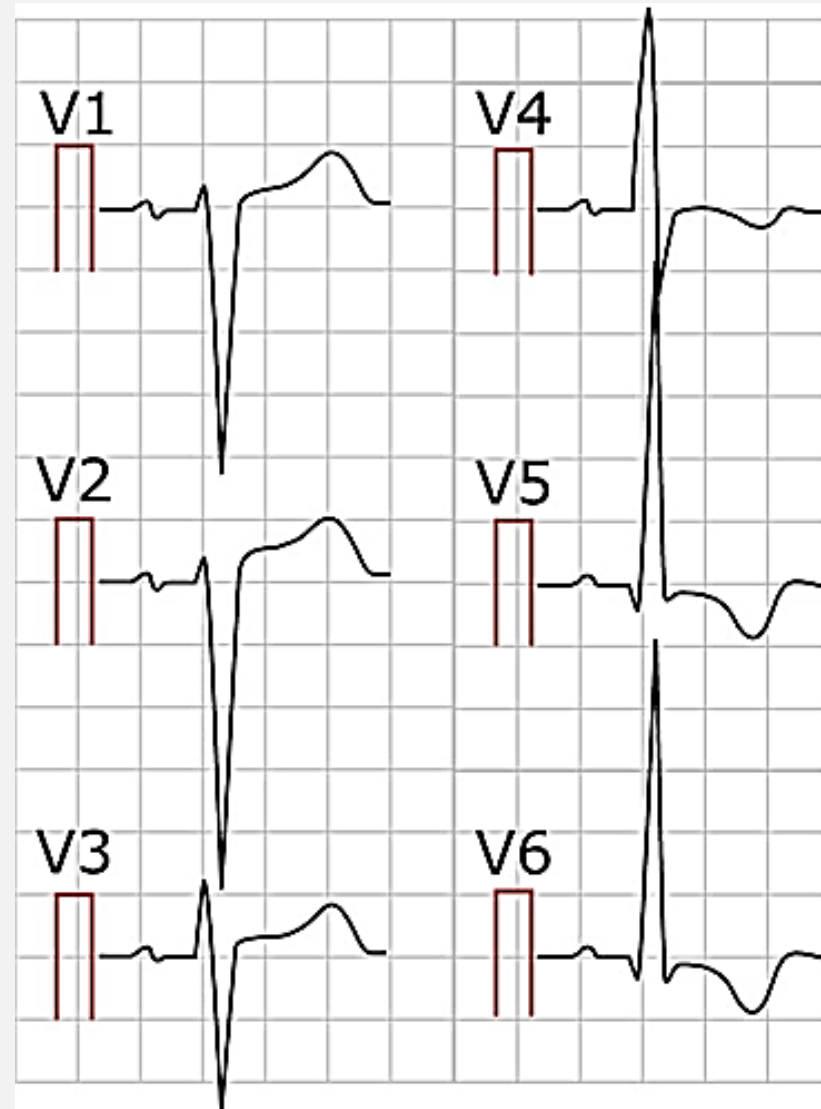


Try to evaluate this ECG due to Sokolow - Lyon's criteria:

- S in V1 or V2 + R in V5 or V6

If you the result of your calculations will be equal or more than 35 mm - it should be considered as a sign of LVH.

NB! Note that only big boxes are visible on this ECG

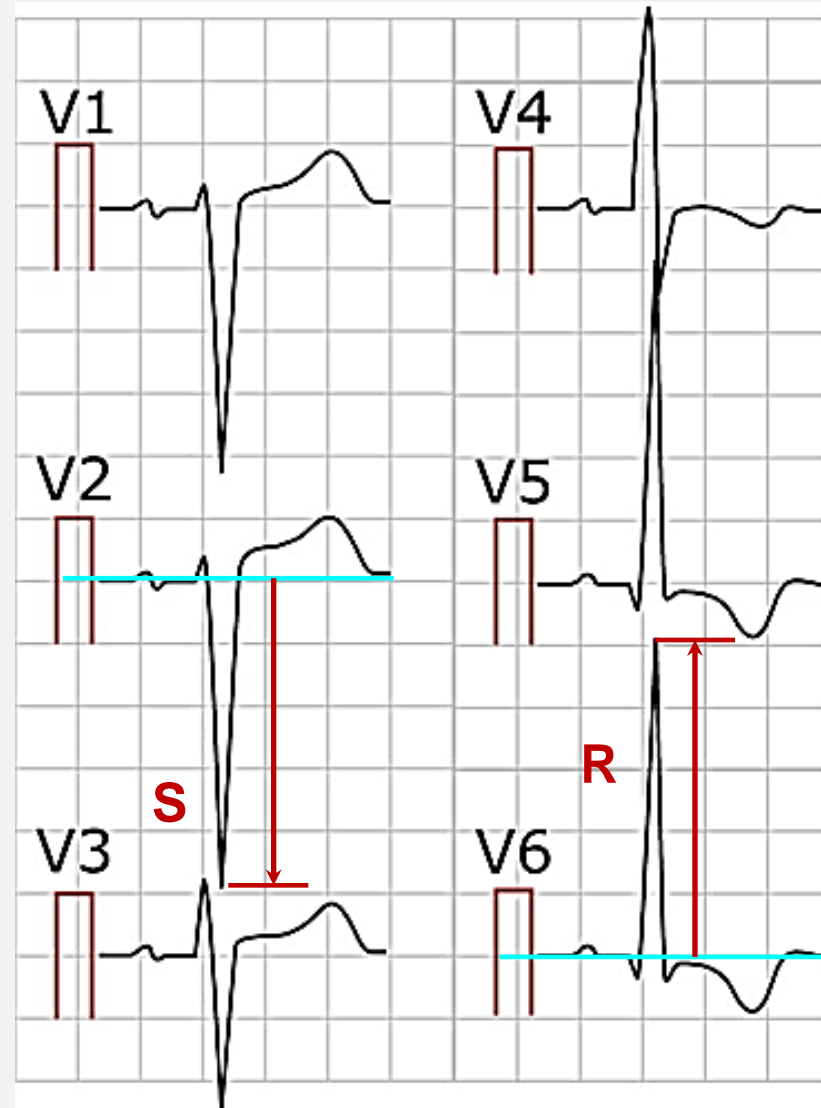


Practical part



Sokolow - Lyon's criteria:

- S in V_1 or V_2 + R in V_5 or V_6 > 35 mm;
- 1. A baseline is indicated (blue line) in leads V_2 and V_6 (leads with the largest waves);
- 2. S wave in V_2 is:
5 big boxes = 25 small boxes = 25 mm.
- 3. R wave in V_6 is:
5 big boxes = 25 small boxes = 25 mm.
- 4. R in V_6 + S in V_2 = 50 mm. It is left ventricular hypertrophy

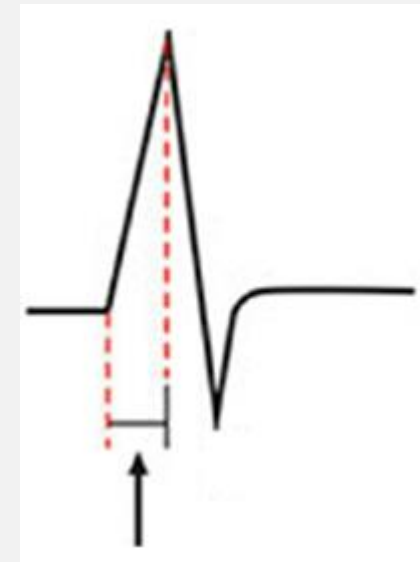


Delayed intrinsicoid deflexion of the QRS



- Intrinsicoid deflection, or R-wave peak time, represents the early phase of ventricular depolarization, and is defined as the time period from the onset of the QRS complex to the peak of the R wave.
- Delayed intrinsicoid deflection ≥ 0.05 sec (i.e., time from QRS onset to peak R is 0.05sec) in lateral precordial leads V_5 and V_6 has been associated with LVH.
- It also can be seen in bundle branch blocks.

R wave



R-wave peak time

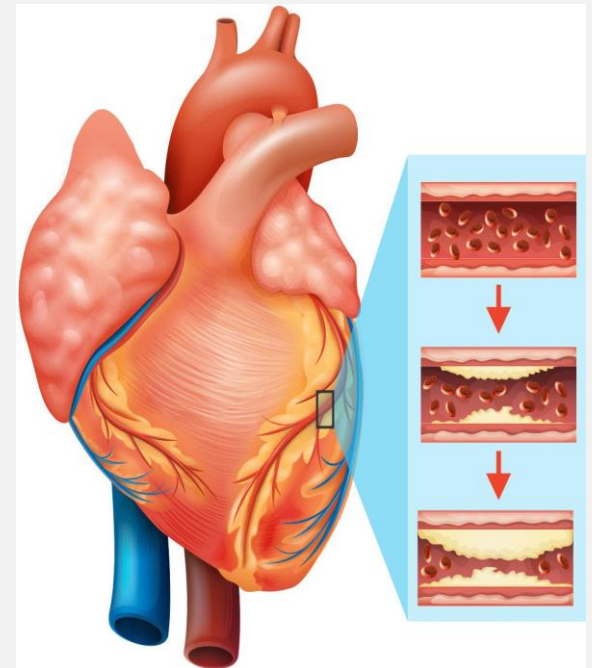
Stable forms of ischemic heart disease



Definition



- **Stable forms of ischemic heart disease** (IHD) encompass a variety of conditions associated with episodes of reversible myocardial ischemia and are characterized by stable pattern of angina. Myocardial ischemia occurs as a result of an imbalance between the supply (perfusion) and demand of the heart for oxygenated blood.



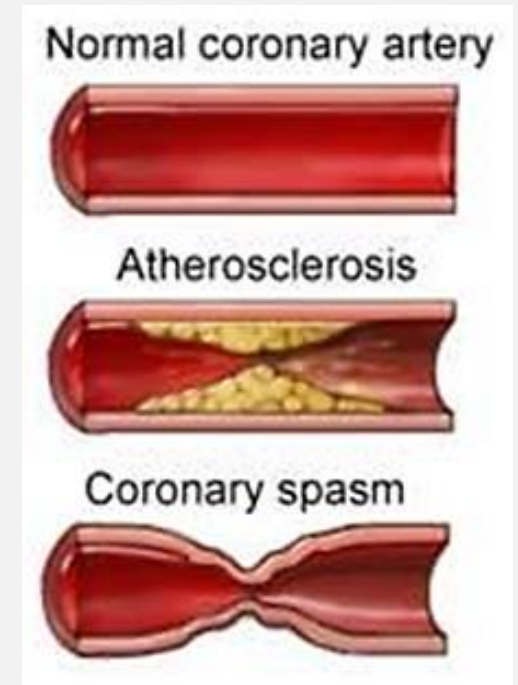
https://www.health.harvard.edu/media/content/images/CAC_Heart_N1803_gi473342614.jpg

<https://www.ncbi.nlm.nih.gov/books/NBK507703/>

Causes of IHD



- The most common cause for IHD is atherosclerotic plaque deposition within the coronary arteries resulting in coronary stenosis and myocardial ischemia.
- Another mechanisms of myocardial ischemia include: decreased oxygen supply (coronary artery vasospasm, microcirculation dysfunction, congenital anomalies of coronary arteries etc.), increased oxygen demand (LVH, LV contractility, elevated BP) or their combination (the most common)



https://www.dicardiology.com/sites/default/files/styles/content_feed_large_new/public/ans7_cardiacischemia.jpg?itok=eSTNhrZA

<https://www.ncbi.nlm.nih.gov/books/NBK507703/>

Stable forms of IHD



- Stable effort angina
- Prinzmetal's or vasospastic angina
- Microvascular angina or cardiac syndrome X
- Silent myocardial ischemia
- Cardiosclerosis: large-focal (postinfarctional) and small-focal (diffuse) cardiosclerosis
- Postinfarctional heart aneurism

ECG in stable forms of IHD



Ischemia affects process of repolarization and is characterized by non-specific changes in ST segment and T wave (ST-T changes):

- ST segment depression (in transient subendocardial ischemia)
- T wave inversion
- Flattening of T waves
- ST segment elevation (in transient transmural ischemia)

ECG in IHD: key points

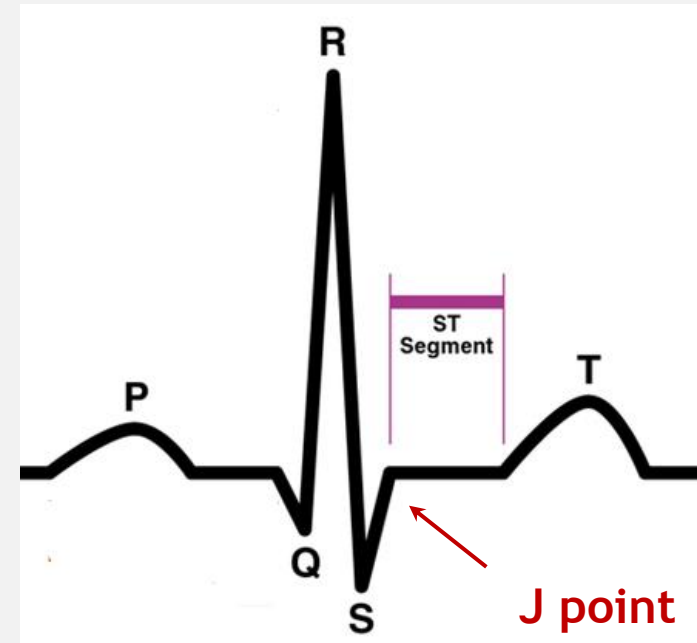


- ST-T changes are non-specific but in the setting of the ischemic chest pain these changes strongly suggest myocardial ischemia.
- Resting ECG between attacks of chest pain can be normal. In this case exercise stress ECG testing is used to provoke ischemia and reveal ST-T changes during physical load.
- Prinzmetal's angina is characterized by transient ST elevation.
- Presence of pathological Q wave (which is deep and wide) suggests previous myocardial infarction (postinfarctional cardiosclerosis).

ST segment & T wave features

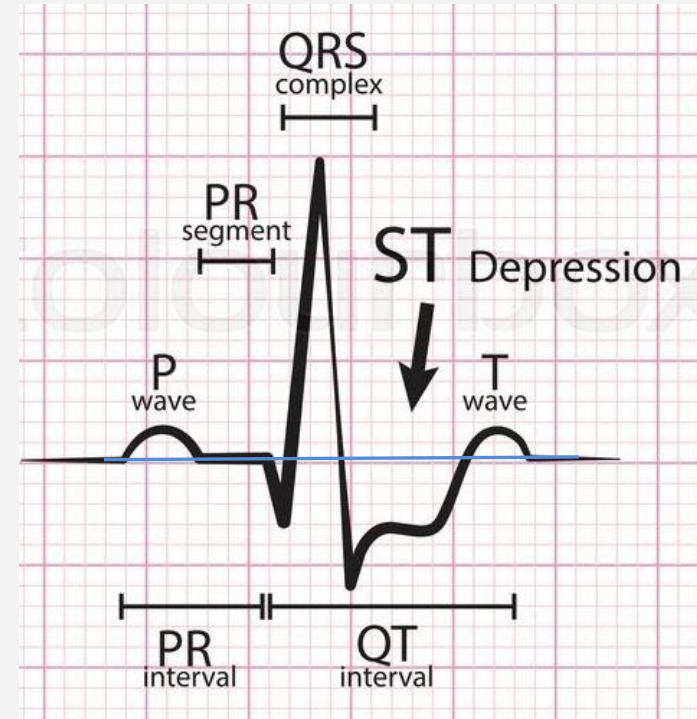
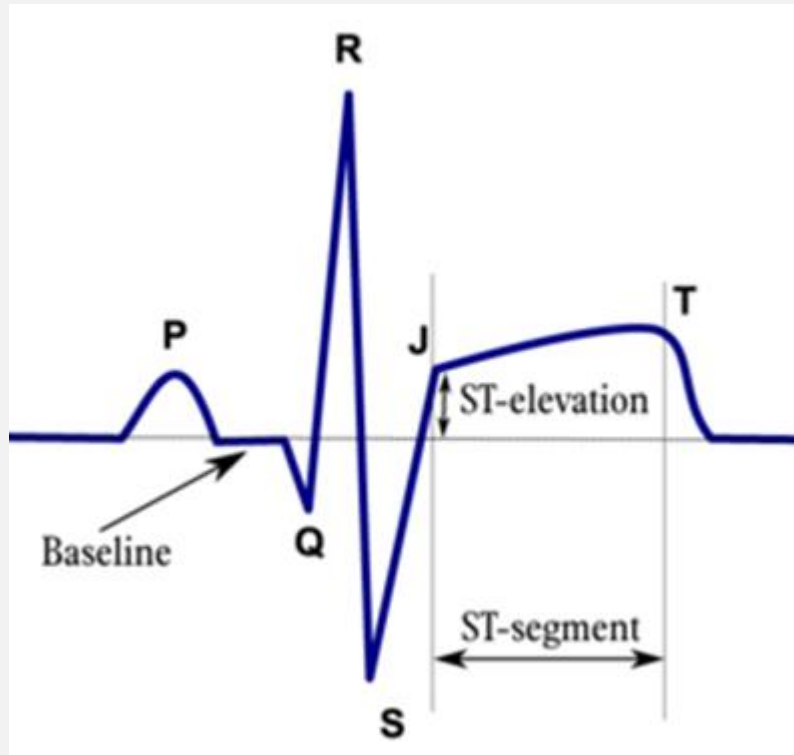


- The ST segment is the flat, isoelectric section between the end of the S wave (**the J point**) and the beginning of the T wave.
- The T wave is normally concordant with the QRS complex, which means that a positive QRS complex should be followed by a positive T wave and vice versa.
- The T wave is always upright in I, II, V4-V6 and inverted in aVR; it may be inverted in V1 too;
- Deviation of J point from a baseline defines changes of ST segment (elevation or depression).



http://uziwiki.ru/img/224_img3.png

ST elevation vs ST depression



- ST elevation: the J point is displaced above baseline.
- ST depression: the J point is displaced below baseline.

NB! ST segment deviations are measured in mm.

Causes for ST segment abnormalities



- ST segment elevation: acute myocardial infarction (STEMI), Prinzmetal's angina, pericarditis, early benign repolarization, left bundle brunch block (LBBB), non-specific bundle brunch block, left ventricular hypertrophy, ventricular extrasystoles, Wolff-Parkinson-White syndrome, hyperkalemia, etc.
- ST segment depression: myocardial ischaemia, myocardial infarction (NSTEMI), supraventricular tachycardia, heart failure, ventricular hypertrophy, LBBB, digoxin effect, pulmonary embolism, etc.

ST elevation in myocardial ischemia



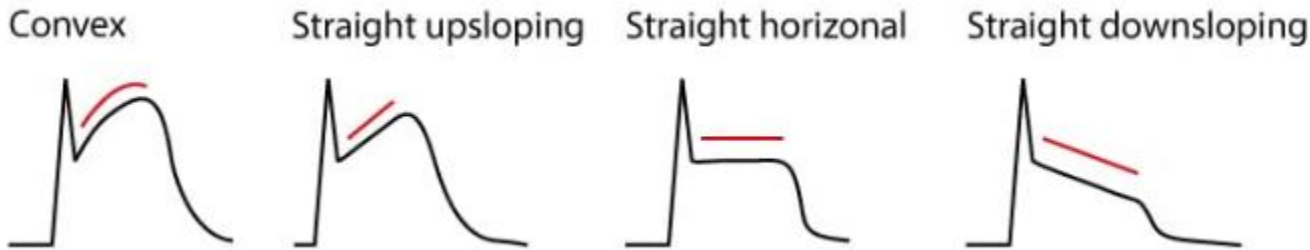
ST segment elevations in at least 2 contiguous leads (in the absence of left ventricular hypertrophy or left bundle branch block):

- Men age ≥ 40 yrs: ≥ 2 mm in V2-V3 and ≥ 1 mm in all other leads;
- Men age < 40 yrs: $\geq 2,5$ mm in V2-V3 and ≥ 1 mm in all other leads;
- Women (any age): $\geq 1,5$ mm in V2-V3 and ≥ 1 mm in all other leads;

ST elevation characteristics

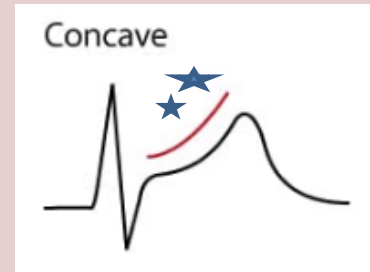


Ischemic ST elevation types



- Usually are present convex, straight ST segment elevations, straight downsloping ST elevation is unusual;
- ST segment tends to merge imperceptibly with the T wave
- There is usually reciprocal ST depression in the electrically opposite leads.

Non-ischemic ST elevation type



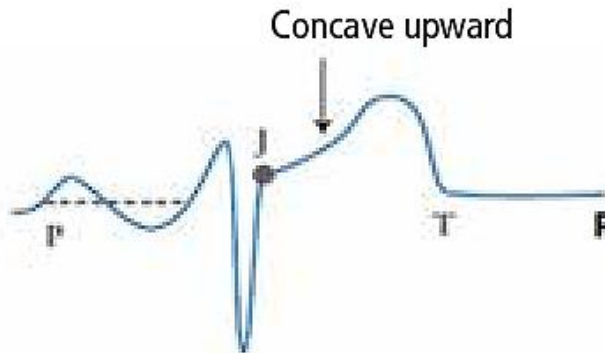
- Concave ST segment,
- not merged with T wave,
- T wave maintain its independent waveform

ST elevation morphology in different non-ischemic conditions



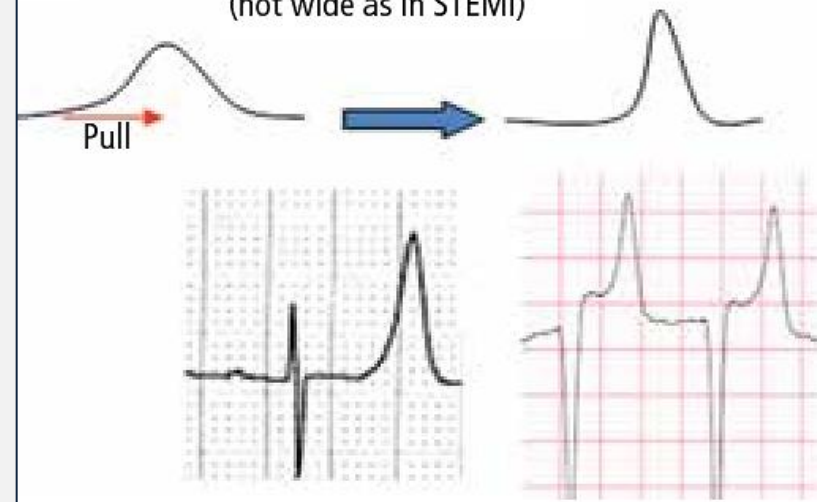
Pericarditis

Concave ST elevation
PR depression
ST/T ratio > 25%



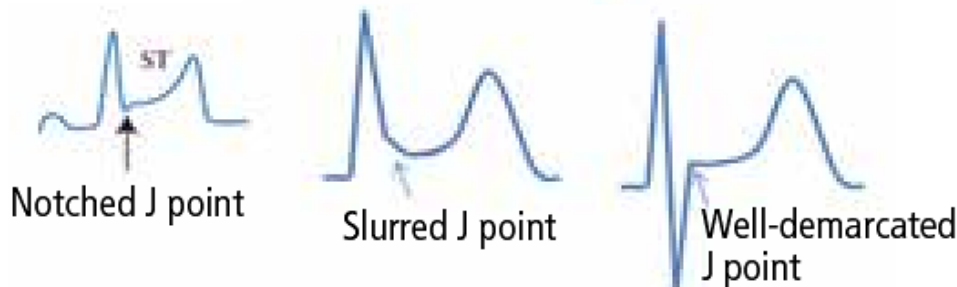
Hyperkalemia

ST segment pulled by T wave
T tall and *narrow*
(not wide as in STEMI)



Early repolarization

Concave ST elevation
Notched, slurred, or otherwise well-demarcated J point

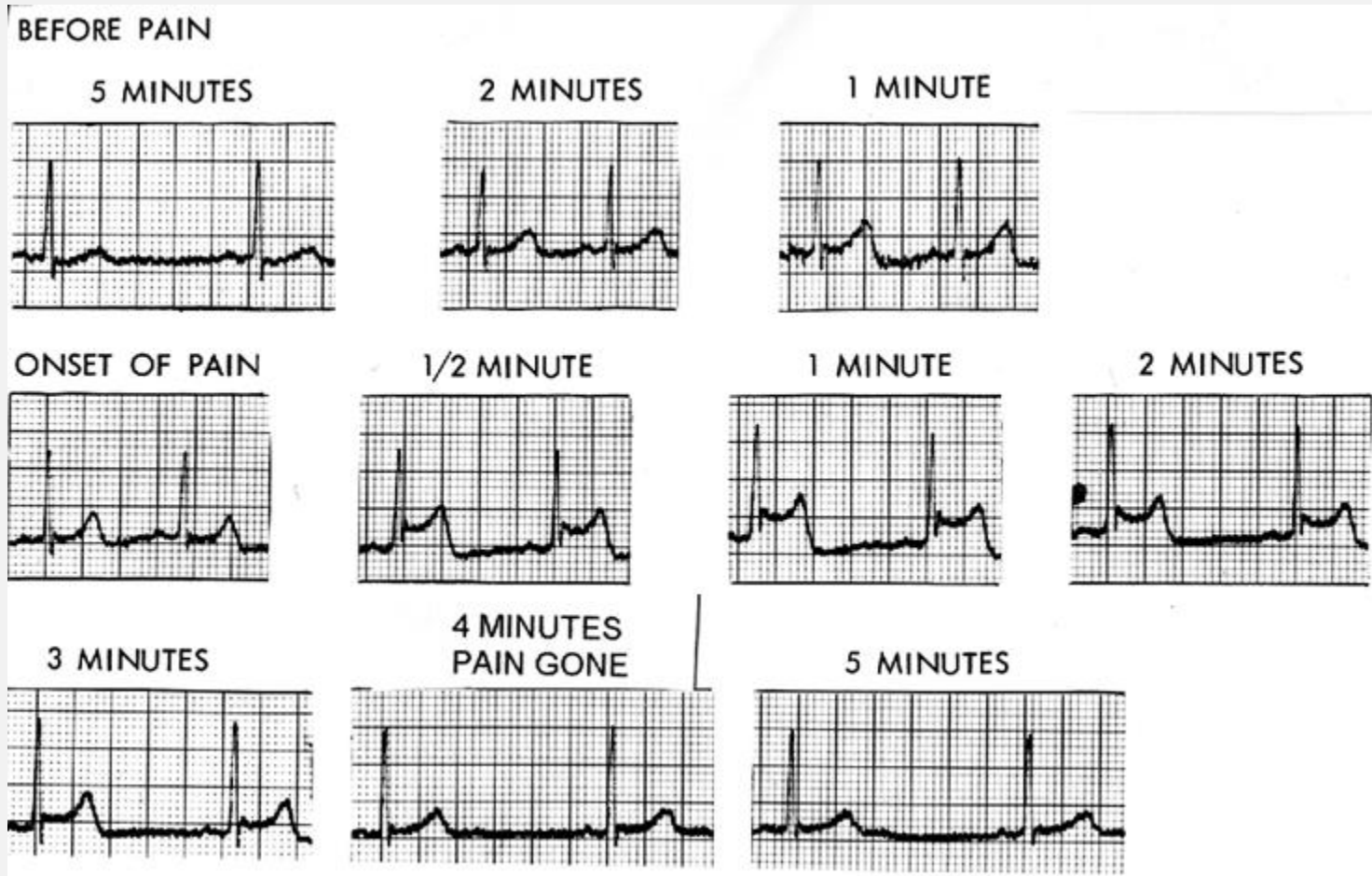


Prinzmetal's angina

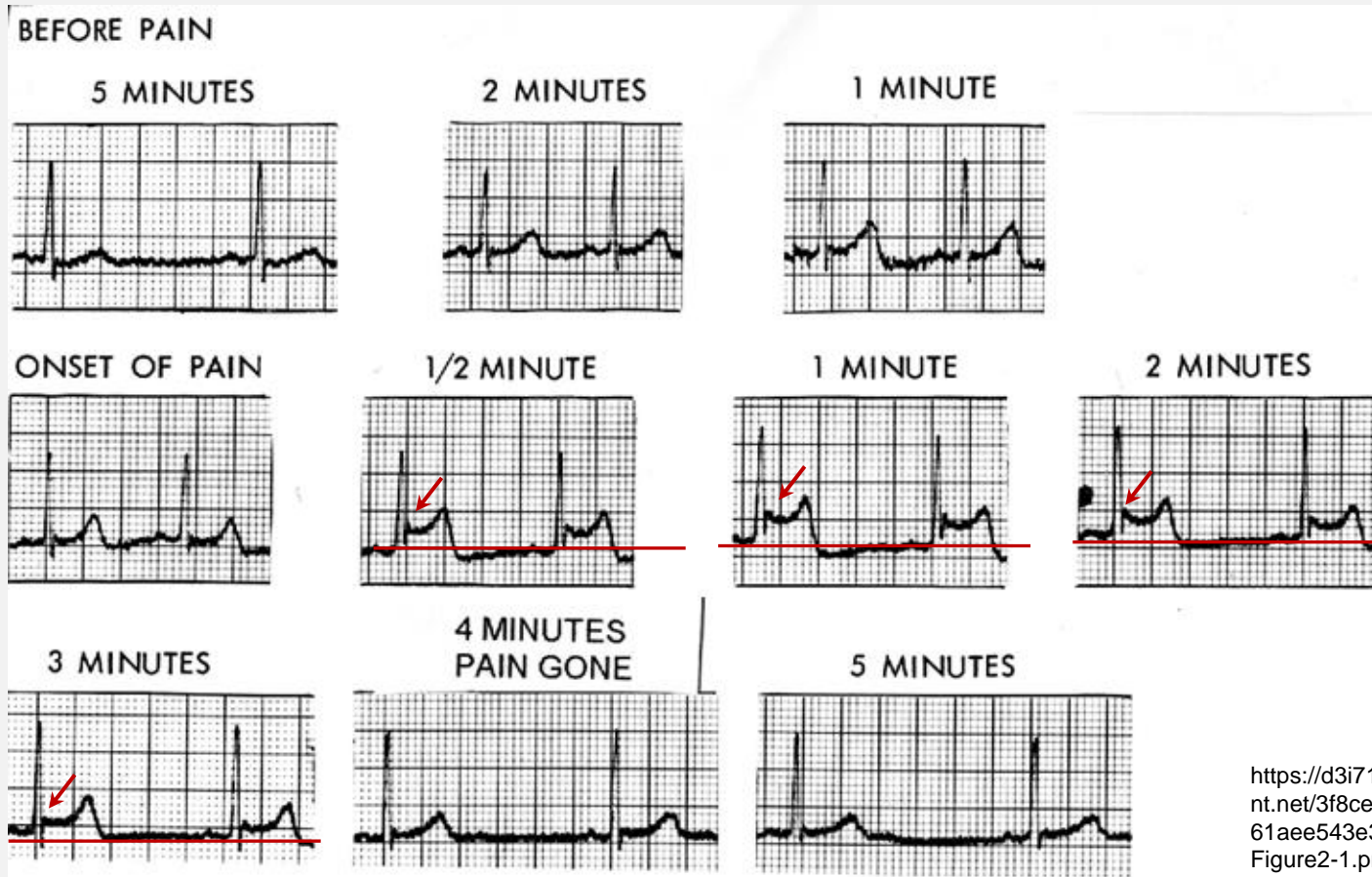


- When an epicardial artery is completely “pinched off” as a result of spasm, the ST segment becomes elevated in the leads facing the affected area, reflecting transmural ischemia.
- In this condition, called Prinzmetal's angina, the spasm is usually brief and the ST segment returns to normal, with no resultant myocardial damage.
- The ST-segment elevations in Prinzmetal's angina and in acute myocardial infarction (MI) are indistinguishable, since they reflect the same pathophysiological process: transmural ischemia from occlusion of an epicardial artery by transient spasm in Prinzmetal's angina and by persistent thrombus in acute infarction.
- If the spasm lasts long enough, MI occurs.

What is on ECG?



What is on ECG?



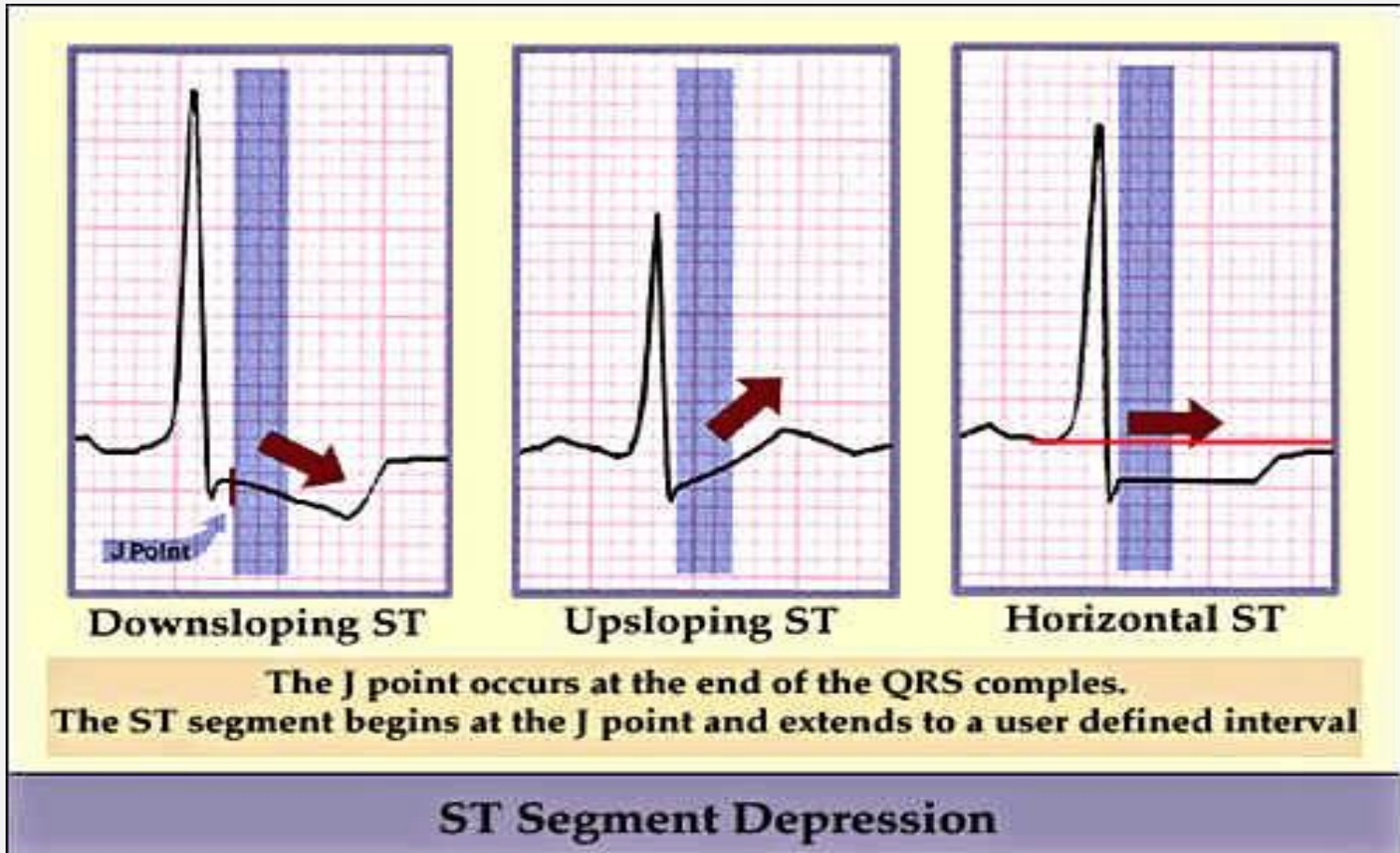
- There is ST segment elevation during chest pain attack (J point above baseline - red arrows). After the pain is gone ST segment returns back to normal. It is an **episode of Prinzmetal's angina**.

Morphology of ST Depression



- ST depression less than 0,5 mm is accepted as normal in all leads;
- Horizontal or downsloping ST depression ≥ 0.5 mm at the J-point in ≥ 2 contiguous leads indicates myocardial ischaemia
- ST depression ≥ 1 mm is more specific and conveys a worse prognosis.
- Upsloping ST depression is non-specific for myocardial ischaemia

Morphology of ST Depression



Distribution of ST segment depression



- ST depression due to subendocardial ischaemia is usually widespread – typically present in leads I, II, V4-6 and a variable number of additional leads.
- A pattern of widespread ST depression plus ST elevation in aVR > 1 mm is suggestive of left main coronary artery occlusion.
- ST depression localized to a particular territory (esp. inferior or high lateral leads only) is more likely to represent reciprocal change due to STEMI. The corresponding ST elevation may be subtle and difficult to see, but should be sought.

T wave inversion



T wave inversion may be considered to be evidence of myocardial ischaemia if:

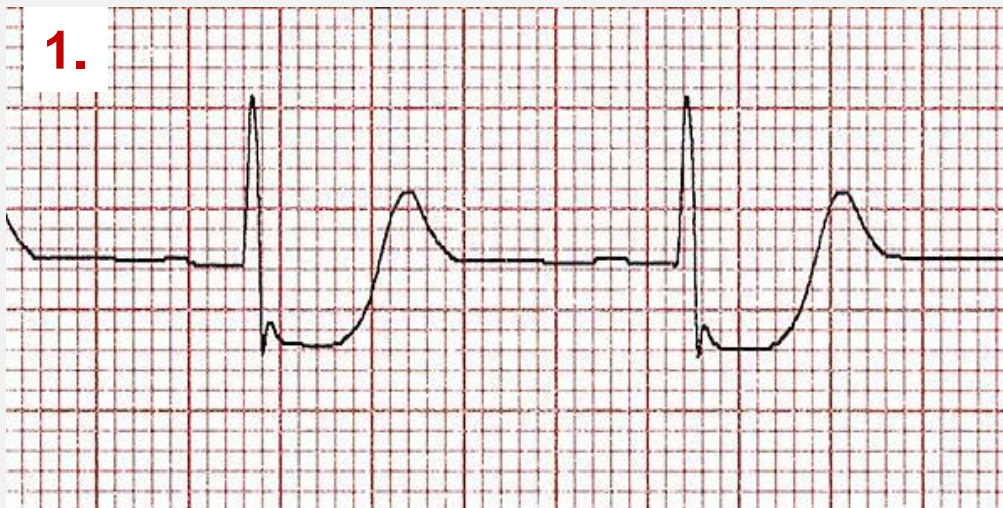
- The depth of T inversion is at least 1 mm;
- It is present in ≥ 2 continuous leads that have dominant R waves (R/S ratio > 1);
- Dynamic pattern (appearance of “new” T wave inversion i.e. it is absent in old ECGs; T wave inversion is changing over time.

NB! T wave inversion is only significant if seen in leads with upright QRS complexes (dominant R waves)

Practical part



- Try to decode ECGs. Reveal signs characteristic for repolarization abnormalities on ECG

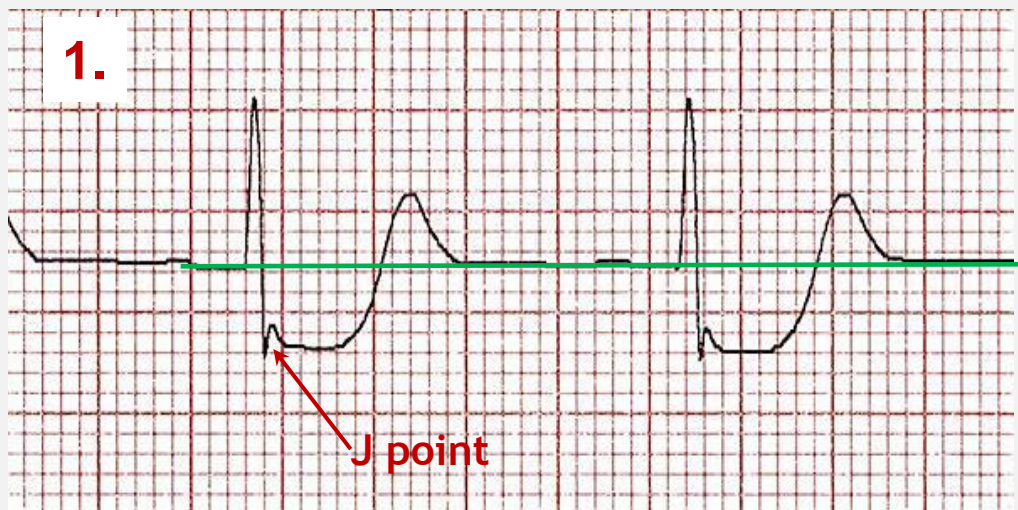


Practical part

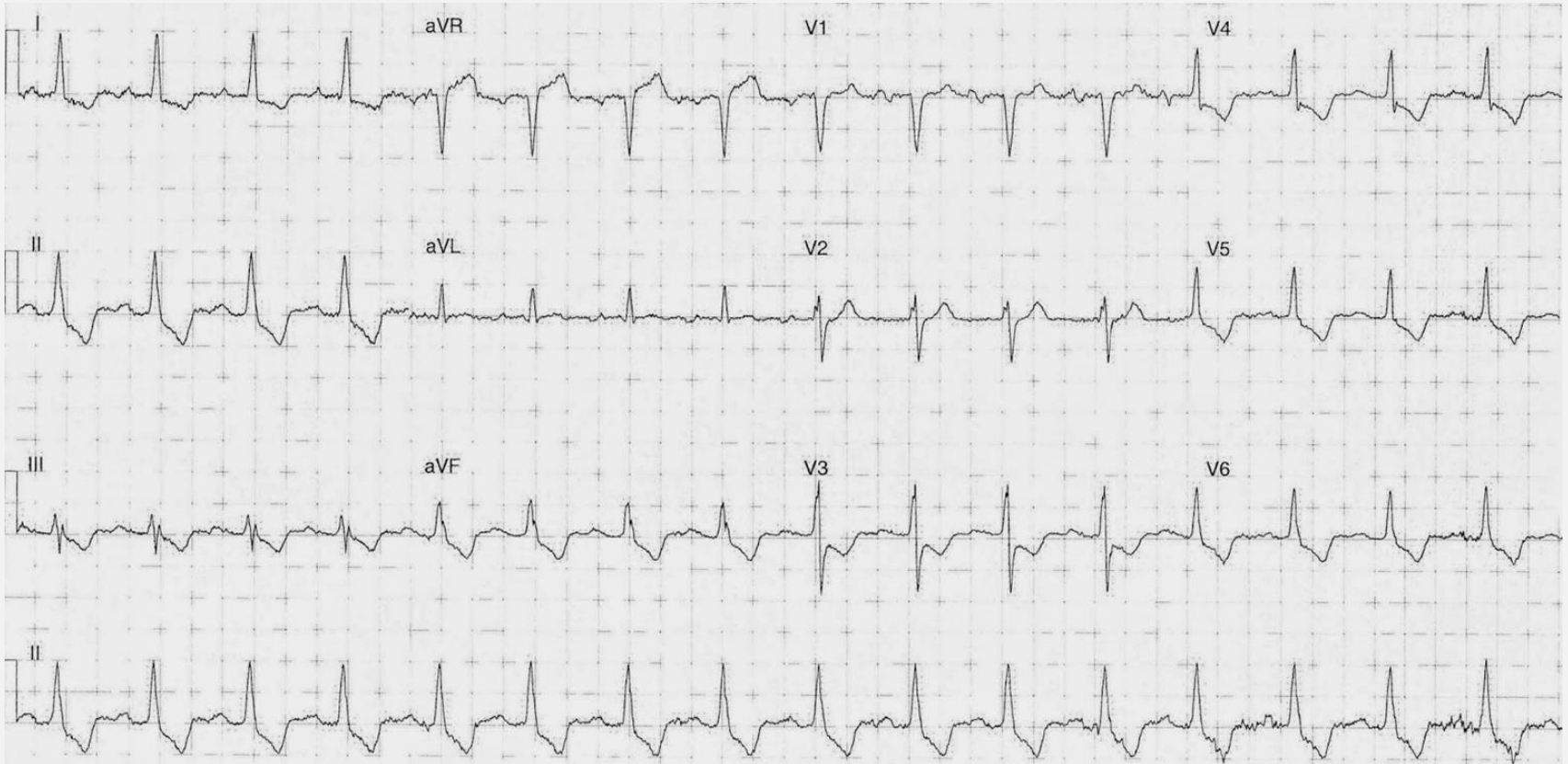


- **Picture 1.** There is 3,5 mm horizontal ST depression (J point is below an baseline). Note that 1 small box is equal to 1 mm.
- **Picture 2.** There is no ST segment deviation (J point is on isoline). There is T wave inversion.

NB! Such changes can be seen in effort angina during an episode of chest pain.

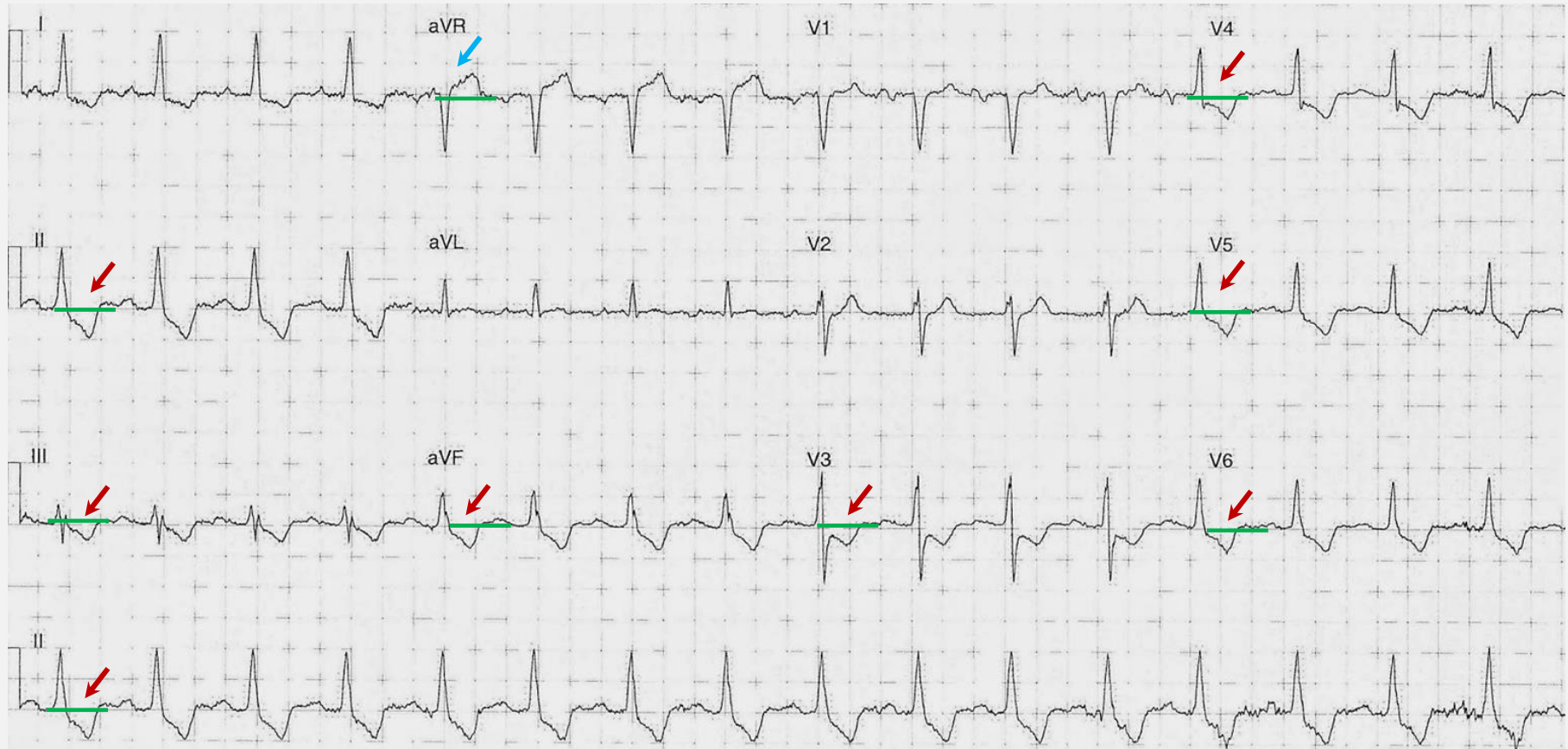


Practical part



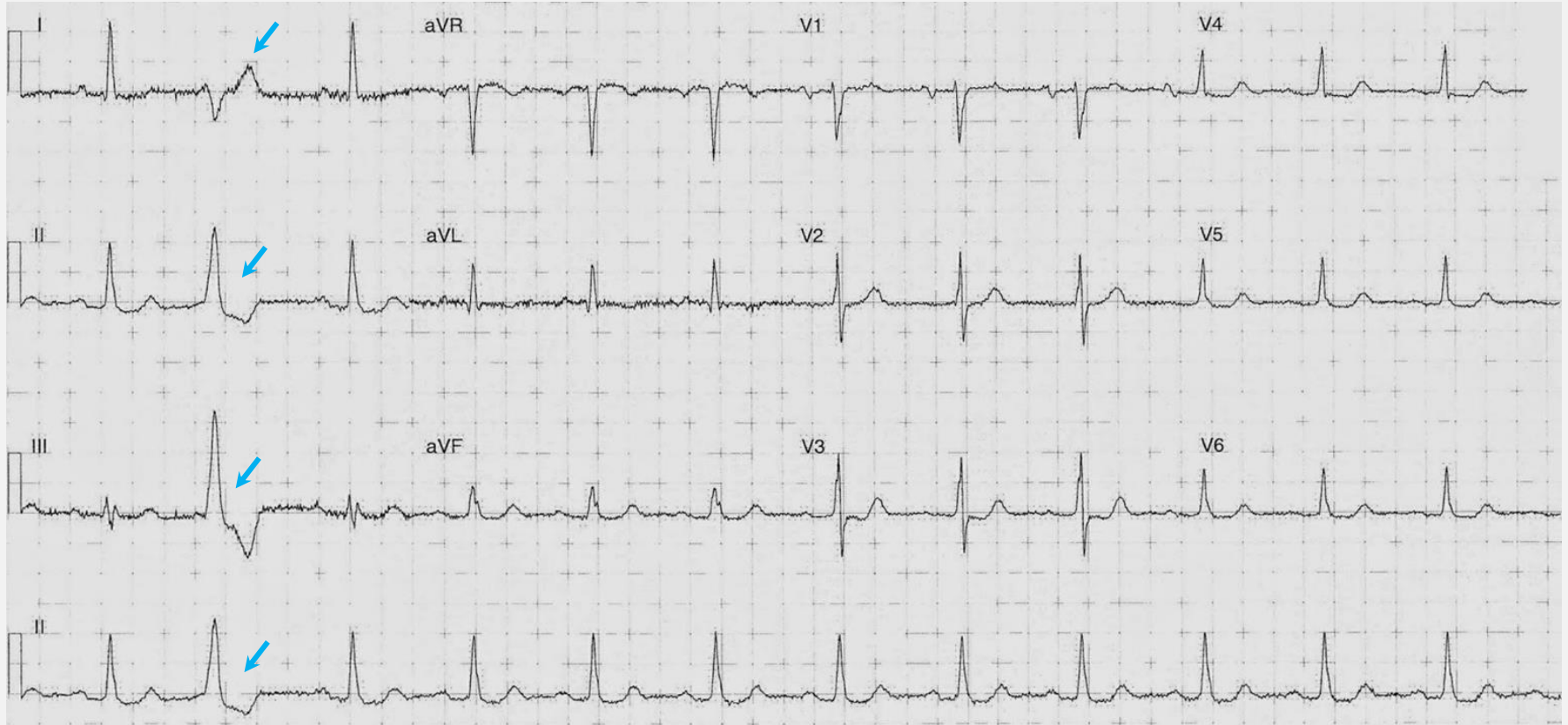
Try to decode ECG of patient with an episode of substernal pain.

ECG in stable effort angina



The ECG shows ST depression in leads I, II, aVF, V3-V6 (red arrows), as well as reciprocal ST elevation in aVR (blue arrow). Thus, the patient has an attack of angina pectoris

ECG in stable effort angina



ECG of the same patient recorded after the cessation of pain. ST elevation in lead aVR and ST depression in leads I, II, aVF, V3-6 - subtle and significantly less than on previous ECG. Note presence of premature ventricular contraction PVC (blue arrow).

Non-specific ST segment and T wave changes



The following changes may occur with myocardial ischaemia but are relatively non-specific:

- ST depression < 0.5 mm
- T wave inversion < 1 mm
- T wave flattening
- Upsloping ST depression with one notable exception (de Winter's ECG)

de Winter's ECG



- In patients presenting with chest pain, ST-segment depression at the J-point with upsloping ST-segments and tall, symmetrical T-waves in the precordial leads of the 12-lead ECG signifies proximal left anterior descending artery occlusion.

Exercise ECG tests: indications



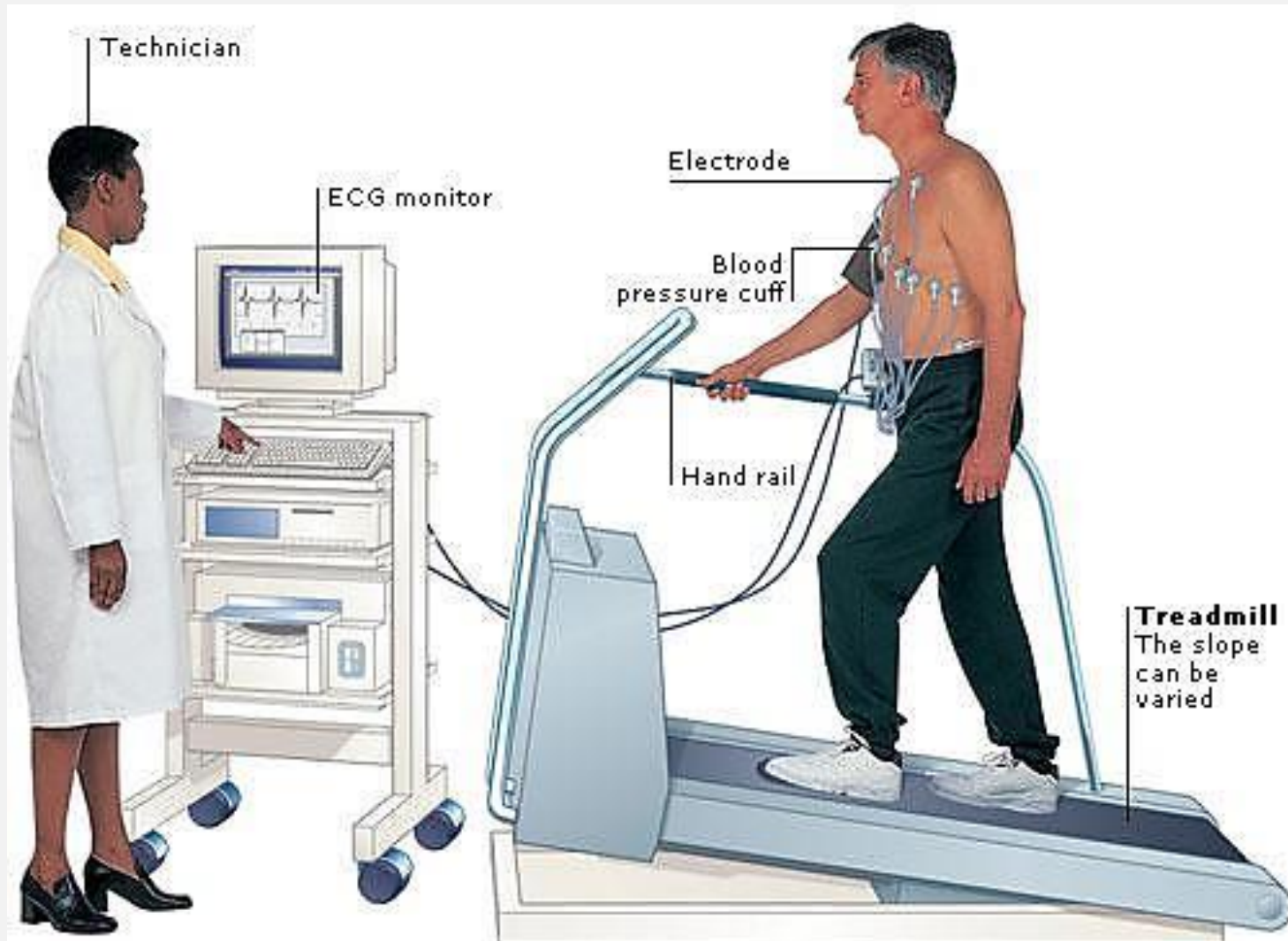
- Confirmation of a suspected diagnosis of angina, painless myocardial ischemia (because ECG abnormalities appear in patient with stable angina during physical activity we provoke myocardial ischemia by exercise and confirm ECG ischemic changes during stress test). In such case ECG stress test is usually made against the background of temporary cancellation of anti-ischemic drugs.
- Assessment of cardiac function and exercise tolerance.
- Prognosis following MI.

Exercise ECG tests: indications-cont.

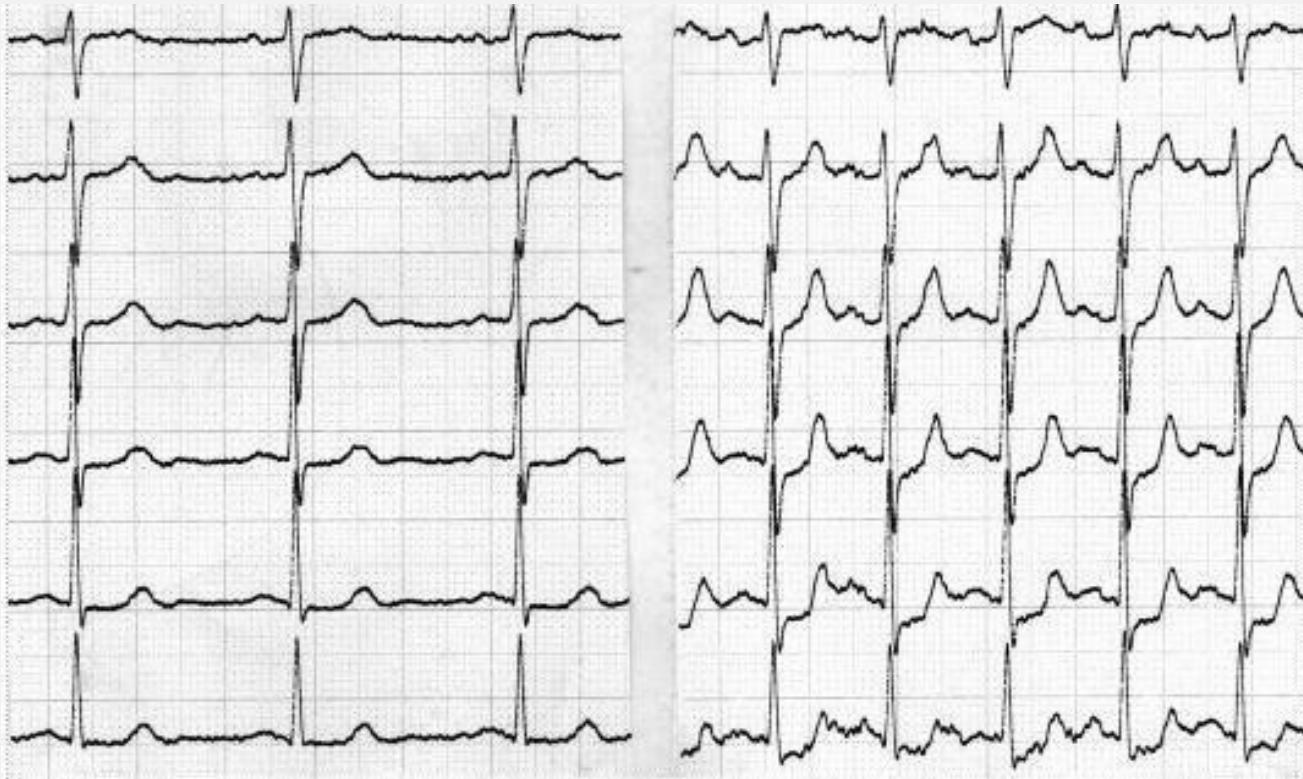


- Evaluation of response to treatment (drugs, angioplasty, coronary artery bypass grafting). In such case paired tests are used (before and against the background of anti-ischemic drugs, or after the surgery). Increased tolerance to physical exertion indicates efficacy of treatment.
- Assessment of exercise-induced arrhythmias.

Treadmill exercise test



ECG exercise test in stable exertional angina



ECG in chest leads at rest (left) and at threshold load (right) in a patient with IHD.

Simplified approach to some stable forms of IHD-1



Type of angina	The leading pathophysiological mechanism	Main clinical features
Stable effort angina	Fixed stenosis of coronary artery with atherosclerotic plaque	<ul style="list-style-type: none">• Pain is provoked by physical exertion.• Patient usually has risk factors for IHD (aged males)
Microvascular angina	Affection of heart's smallest coronary arteries	
Prinzmetal's angina	Spasm in the coronary arteries	<ul style="list-style-type: none">• Pain occurs at rest (usually - early morning hours).• It is more common in young patients and females.• Pt has perfect tolerance to physical exertion due to absence of fixed stenosis of coronary artery.

Simplified approach to some stable forms of IHD-2



Type of angina	ECG during angina attack	Test for confirmation	Coronaroangiography
Stable effort angina	ST depression	Exercise stress test is positive	Detection of the narrowing of coronary artery by plaque
Microvascular angina			Normal
Prinzmetal's angina	ST elevation	Exercise stress test is negative Drug stress test (with ergonovine) is positive	Usually normal, during episode of angina spasm of coronary artery can be detected

Silent myocardial ischemia



- Silent myocardial ischemia is defined as the presence of objective evidence of myocardial ischemia in the absence of chest discomfort or another anginal equivalent symptom (eg, dyspnea, nausea, diaphoresis, etc).
- Objective evidence of silent myocardial ischemia may be obtained in the same ways as painful episodes (angina)
- It is the most common manifestation of IHD, accounting for more than 75 percent of ischemic episodes during daily life

Cardiosclerosis



- Large-focal cardiosclerosis develops as a result of past myocardial infarction. Pathological Q-wave is the most specific ECG finding.
- Diffuse small-focal cardiosclerosis develops as a result of relative coronary insufficiency with the development of small foci of ischemia. Clinical manifestations include angina attacks, arrhythmias and heart failure.
- Cardiosclerosis will be discussed in the next slides (ACS topic).

Acute coronary syndrome



Definition



Acute coronary syndrome (ACS) refers to a spectrum of clinical presentations ranging from those for ST-segment elevation myocardial infarction (STEMI) to presentations found in non-ST-segment elevation myocardial infarction (NSTEMI) or in unstable angina (UA).

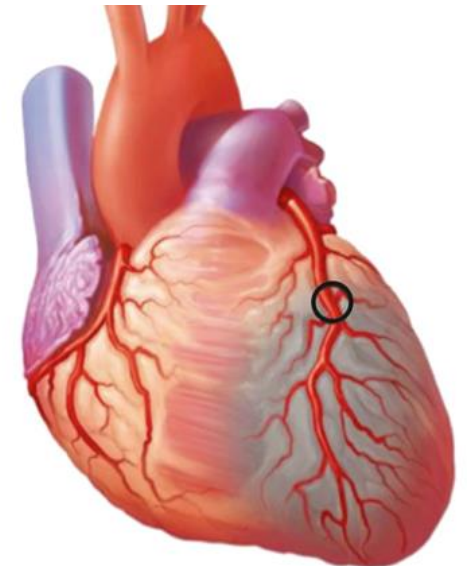
It is almost always associated with rupture of an atherosclerotic plaque and partial or complete thrombosis of the infarct-related artery.



Plaque rupture/erosion with occlusive thrombus



Plaque rupture/erosion with non-occlusive thrombus



The significance of ECG in acute myocardial infarction



- ECG is widely used for diagnosis and differential diagnosis of acute myocardial infarction (AMI).
- It gives the following information about AMI:
 - localization of AMI and estimation of the infarct-related coronary artery (which ECG leads are abnormal)
 - Size/square of AMI (how many ECG leads are abnormal)
 - Depth of AMI (subendocardial, transmural, etc)- presence of Q-wave, Q/R ratio,
 - “Age” of AMI and healing (evolution of AMI)

ECG in ACS: key points -1



ECG in ACS is useful for:

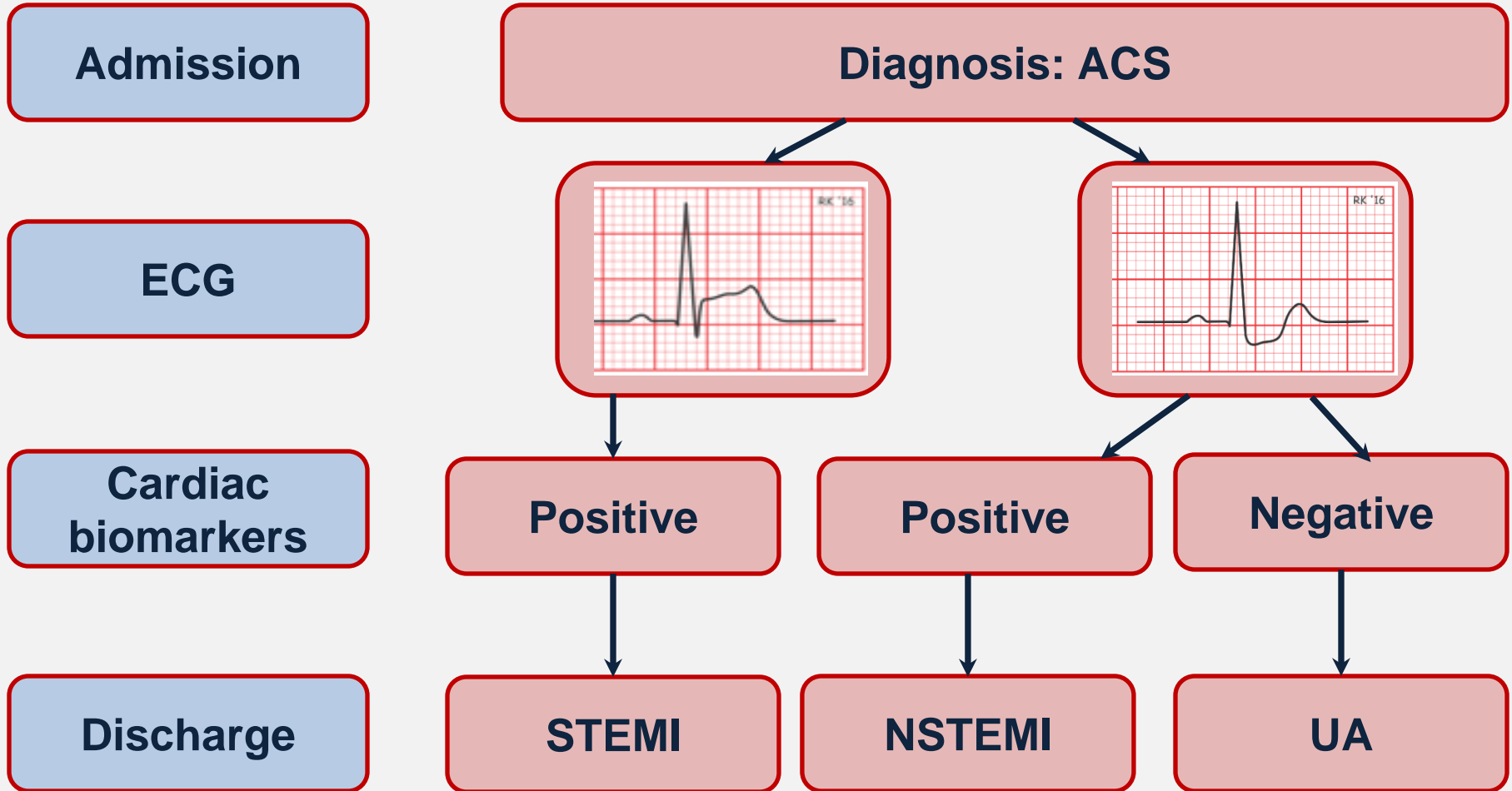
- Detection of complications of AMI (acute pericarditis, Dressler's syndrome, arrhythmias and conduction disorders, aneurysm, etc.)
- Determination of treatment tactics (ACS with ST elevation can be treated by thrombolytics, while in case of ACS without ST elevation it is contraindicated)
- Dynamic monitoring of patient during treatment and in determining the efficacy of reperfusion therapy (ST segment resolution)

ECG in ACS: key points -2



- ECG should be obtained within 10min interval of first medical contact.
- If possible ECG with the patient suspected of having ACS should be compared with his previous ECGs.
- ECG changes in ACS have dynamic character - serial ECG should be taken for continuous monitoring.
- Sometimes ECG of patients with ACS may be normal - it doesn't rule out myocardial ischemia; patients with characteristic chest pain should be monitored continuously with the ECG and cardiac biomarkers control.

Diagnostic approach in ACS



STEMI vs NSTEMI



- STEMI is associated with complete occlusion of the coronary artery (this results in transmural ischemia and injury). Depending on the time from the heart attack onset ECG helps to reveal signs like peaked T wave, ST segment elevation, decreased voltage of R wave, inversion of T wave, appearance of pathological Q wave.
- NSTEMI is caused by partial (incomplete) coronary artery occlusion. This results in a reduction of coronary blood flow and causes ischemia of subendocardium.
- The ECG may show signs of subendocardial ischemia - ST segment depression and/or T wave inversion.

Signs of acute myocardial ischemia



ST elevation:

New ST segment elevations in at least 2 contiguous leads:

- Men age ≥ 40 yrs: ≥ 2 mm in V2-V3 and ≥ 1 mm in all other leads;
- Men age < 40 yrs: $\geq 2,5$ mm in V2-V3 and ≥ 1 mm in all other leads;
- Women (any age): $\geq 1,5$ mm in V2-V3 and ≥ 1 mm in all other leads;

ST depression and T wave changes:

- New horizontal or downsloping ST-depression ≥ 0.5 mm in two contiguous leads and/or T inversion > 1 mm in two contiguous leads with prominent R wave or R/S ratio > 1 .

Q wave: key points

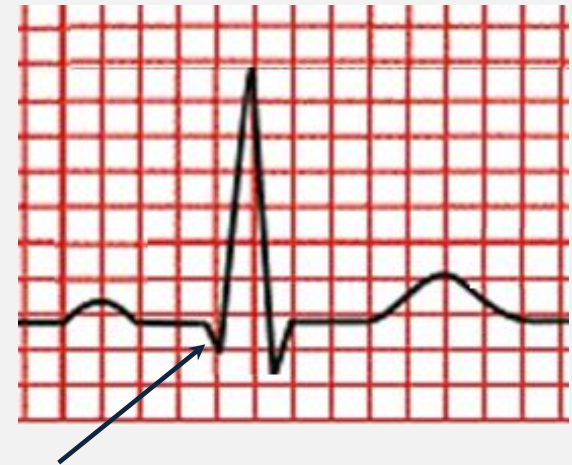


- Q wave is any negative deflection that precedes an R wave.
- Q wave can be physiological (seen at normal conditions) and pathological.
- Presence of pathological Q wave indicates the necrosis of myocardial tissue.
- It can be seen in ongoing MI or it can be an evidence of atherosclerosis as a result of previous MI.
- Evaluation of clinical manifestation and dynamic changes in ECG, imaging/laboratory tests is necessary to make a conclusion about the “age” of the necrosis.

Features of physiological Q wave



- The physiological Q wave represents the normal left-to-right depolarization of the interventricular septum.
- Small 'septal' Q waves are typically seen in the left-sided leads (I, aVL, V5 and V6).



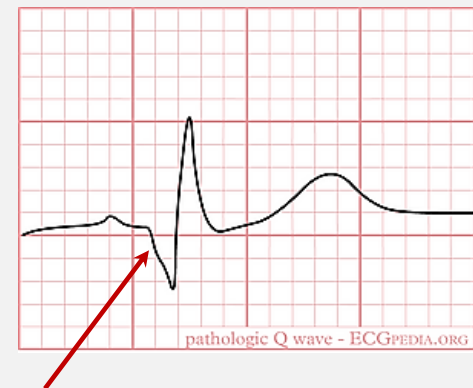
Physiological Q wave

Features of pathological Q wave



- The depth of the pathological Q wave should be at least 25% of the depth of the associated R wave and duration $>0,04$ sec.
- The Q wave should appear in at least 2 contiguous leads
- Any Q wave in leads V1- V3 with a duration of $>0,02$ seconds is likely to be pathological.

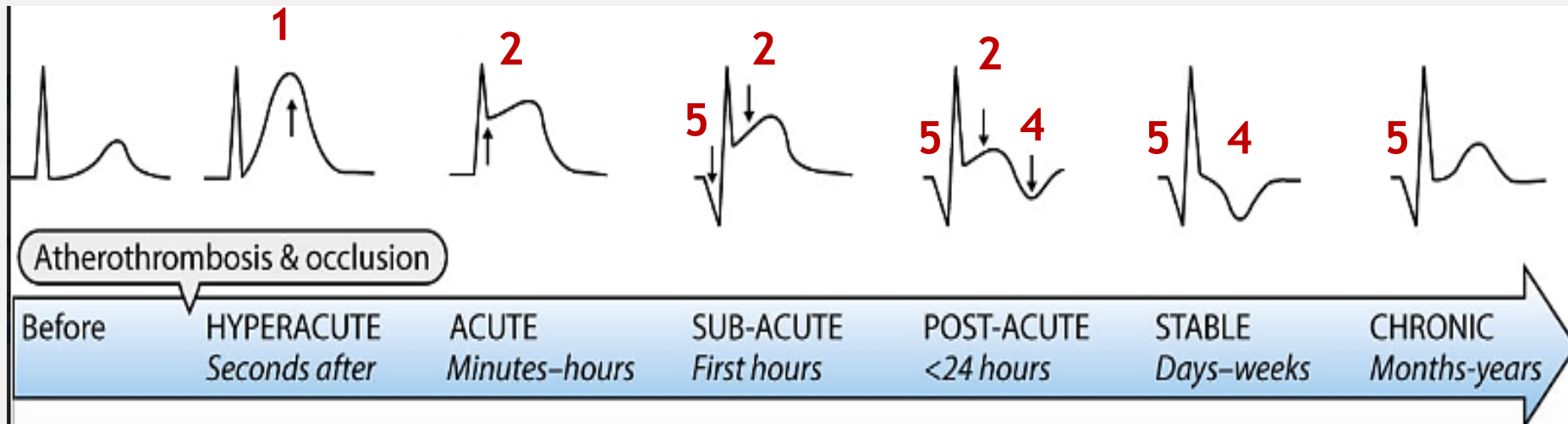
Pathological Q wave



Dynamic ECG changes in MI (STEMI)

1. Peaked hyperacute T waves;
2. ST elevation
3. ST depression;
4. T wave inversion;
5. Pathological Q wave.

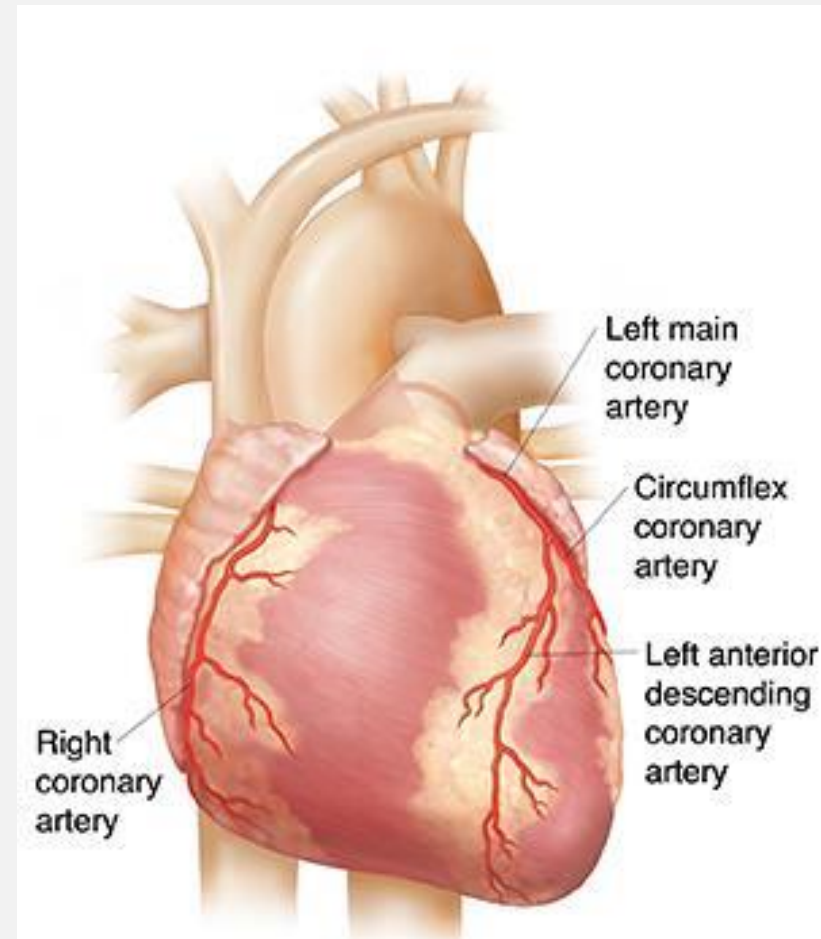
Evolution of ECG changes in STEMI (ST-elevation MI)



Site of infarction

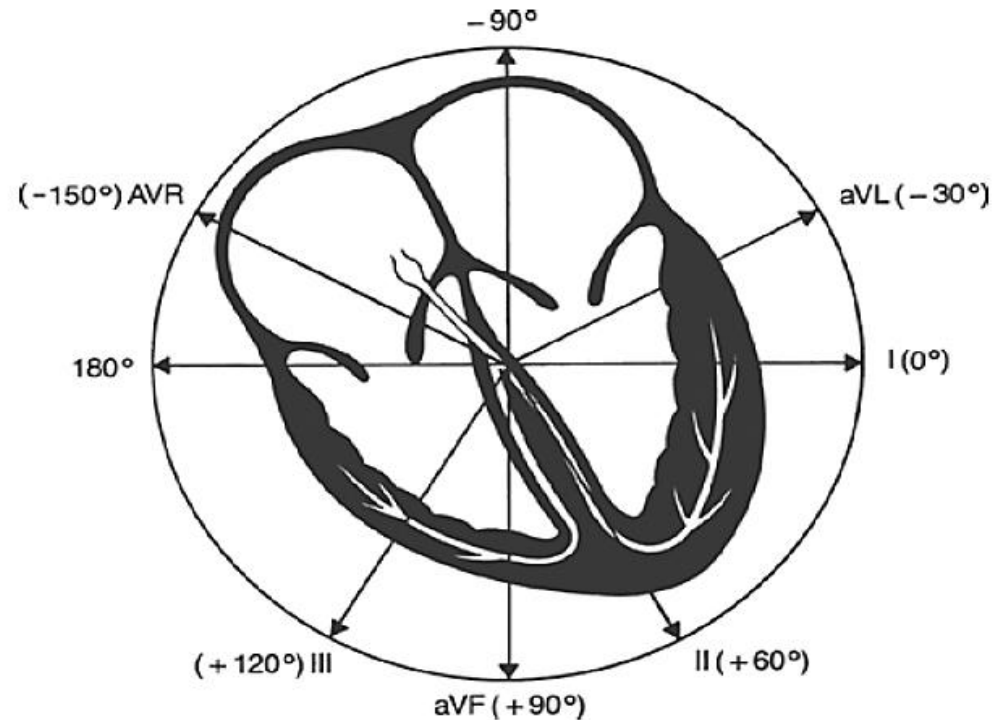
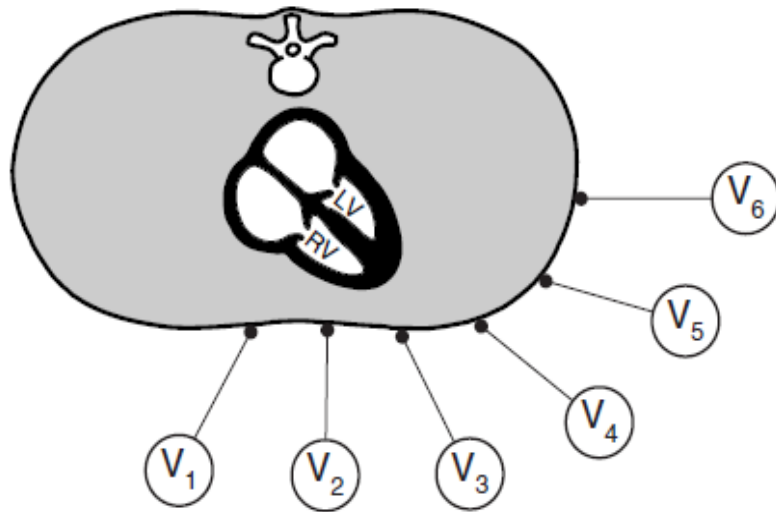


- Occlusion of the circumflex branch of the left coronary artery - a lateral wall infarction;
- occlusion of the anterior descending branch of the left coronary artery - an anterior wall infarction.
- occlusion of the right coronary artery or one of its branches - posterior or inferior wall infarctions. Right ventricular infarctions can also result from right coronary artery occlusion, can accompany inferior infarctions.



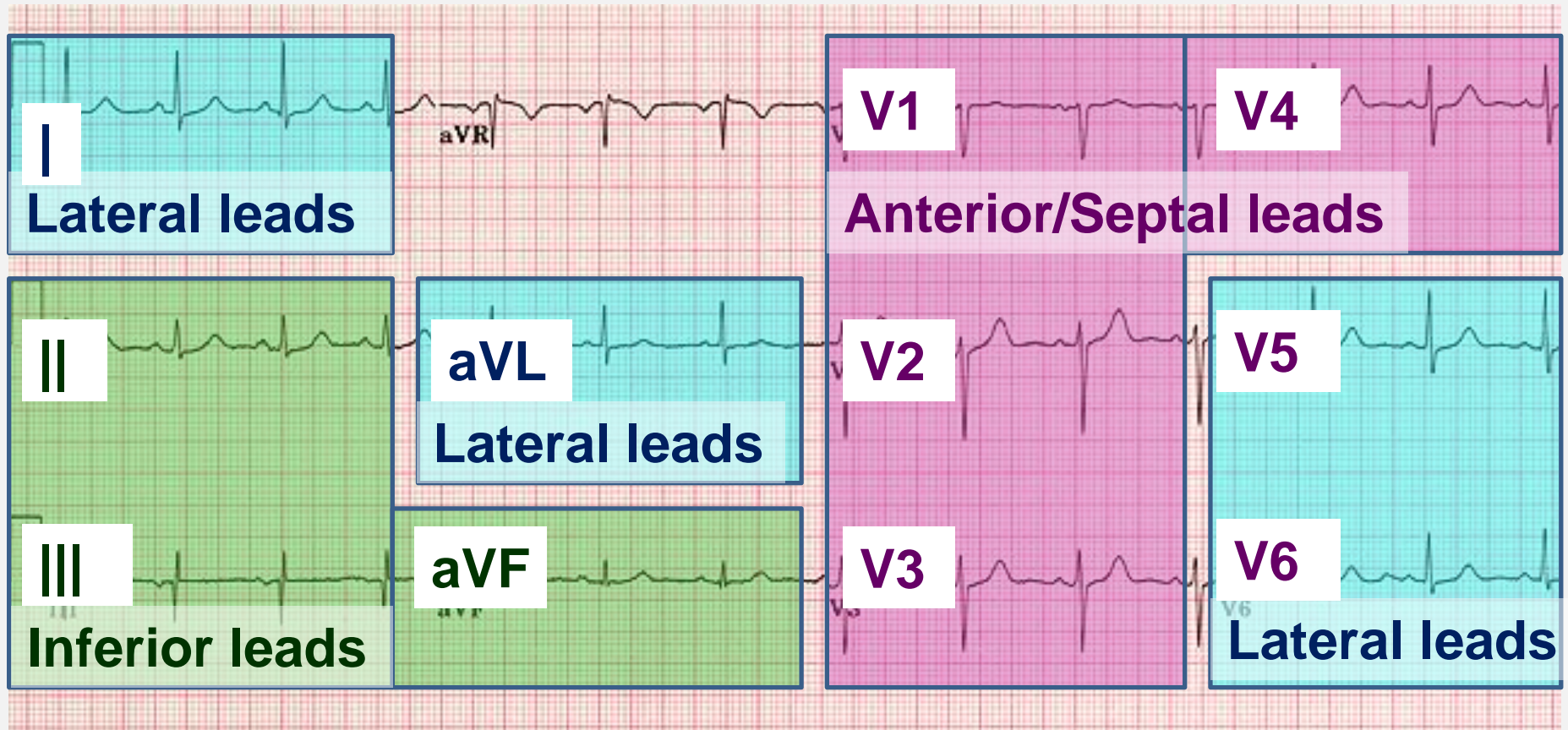
<https://www.mountrnittany.org/assets/images/krames/334577.jpg>

Leads and its angles



- Leads V1, V2 - septal leads, V3, V4 - anterior leads
- Leads II, III, and AVF - inferior leads;
- Leads I, AVL, V5, V6 - lateral leads;
- AVR;

ECG leads



Reciprocal changes-1



Ischemic ST elevations are often characterized by the presence of reciprocal changes (ST depressions) in the opposite leads.

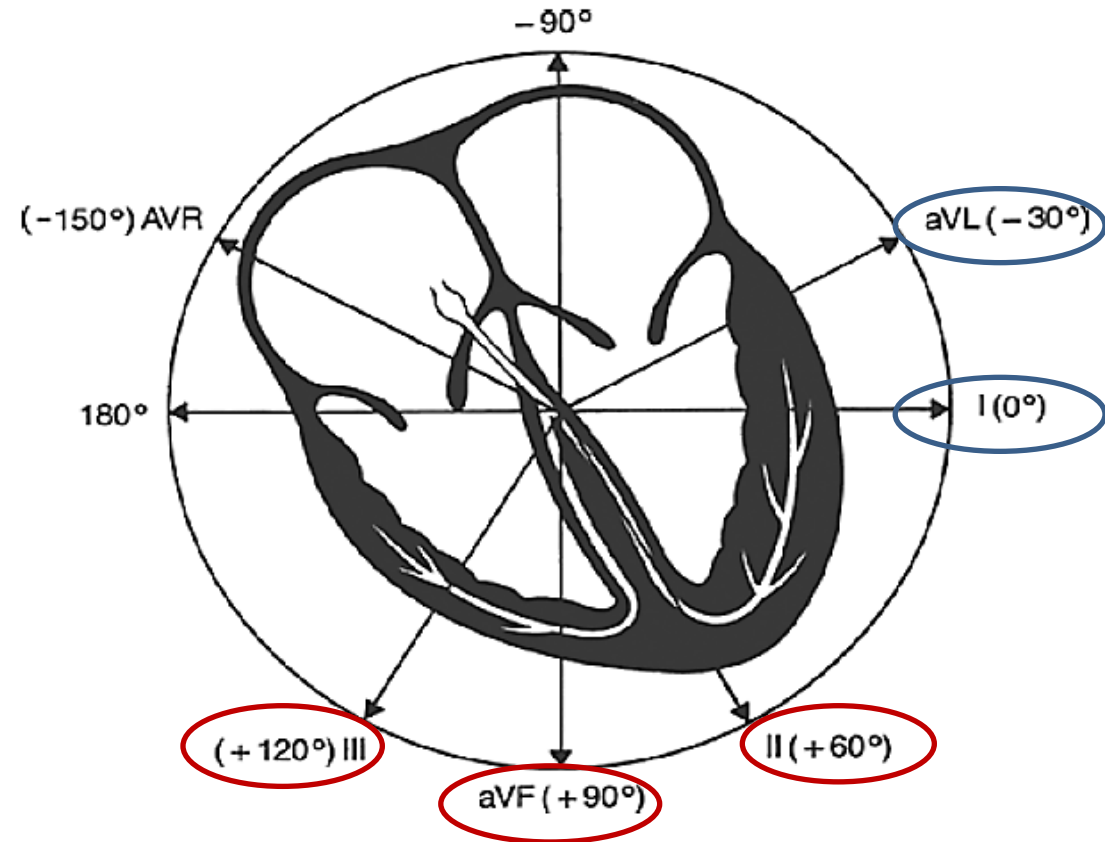
Reciprocal ST depressions are so called “mirror reflections” of ST elevations. Their features are:

- Associated with transmural ischemia
- Strongly connected with ST elevations and are seen only along with ST elevations; when ST elevations are gone (due to dynamic evolution of ECG changes in STEMI) ST depressions are not longer seen.
- Can be seen in opposite to ST elevations leads (leads with opposite angles). For example in case of inferior STEMI ST elevations will be seen in inferior leads accompanied with reciprocal ST depressions in anterior and/or lateral leads and vice versa.

Reciprocal changes-2



- If **ST elevation** is seen in inferior leads
- **Reciprocal ST depression** will be seen in lateral leads (opposite angle of observation).
- And vice versa

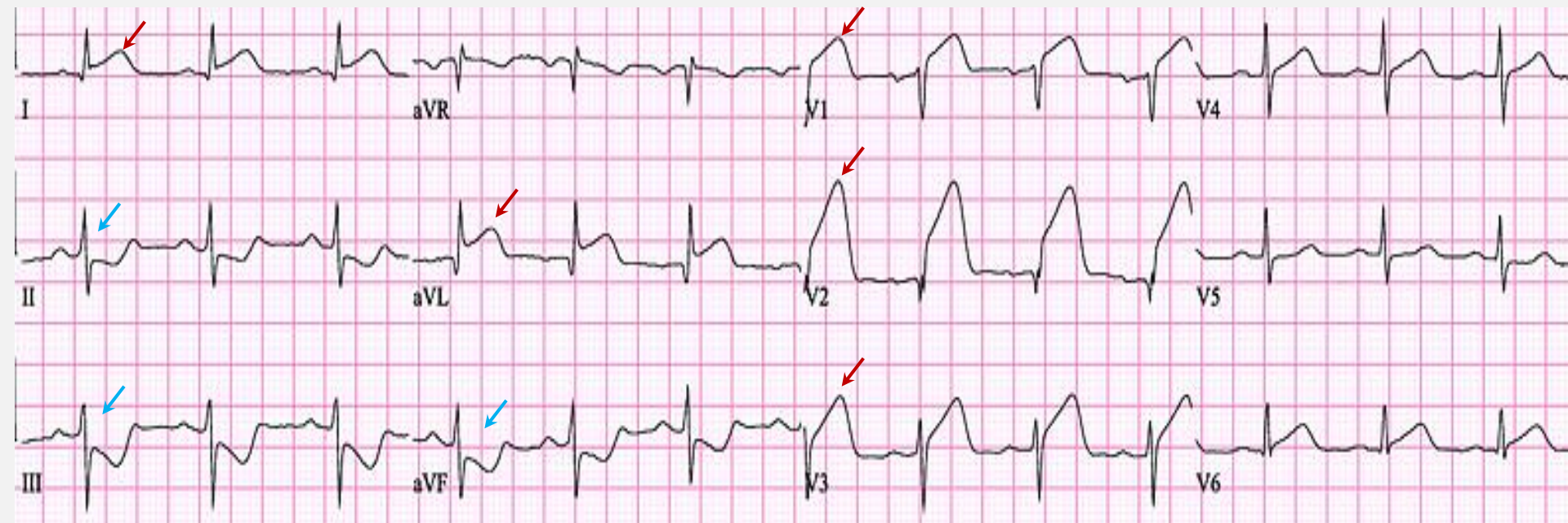


Reciprocal changes -3



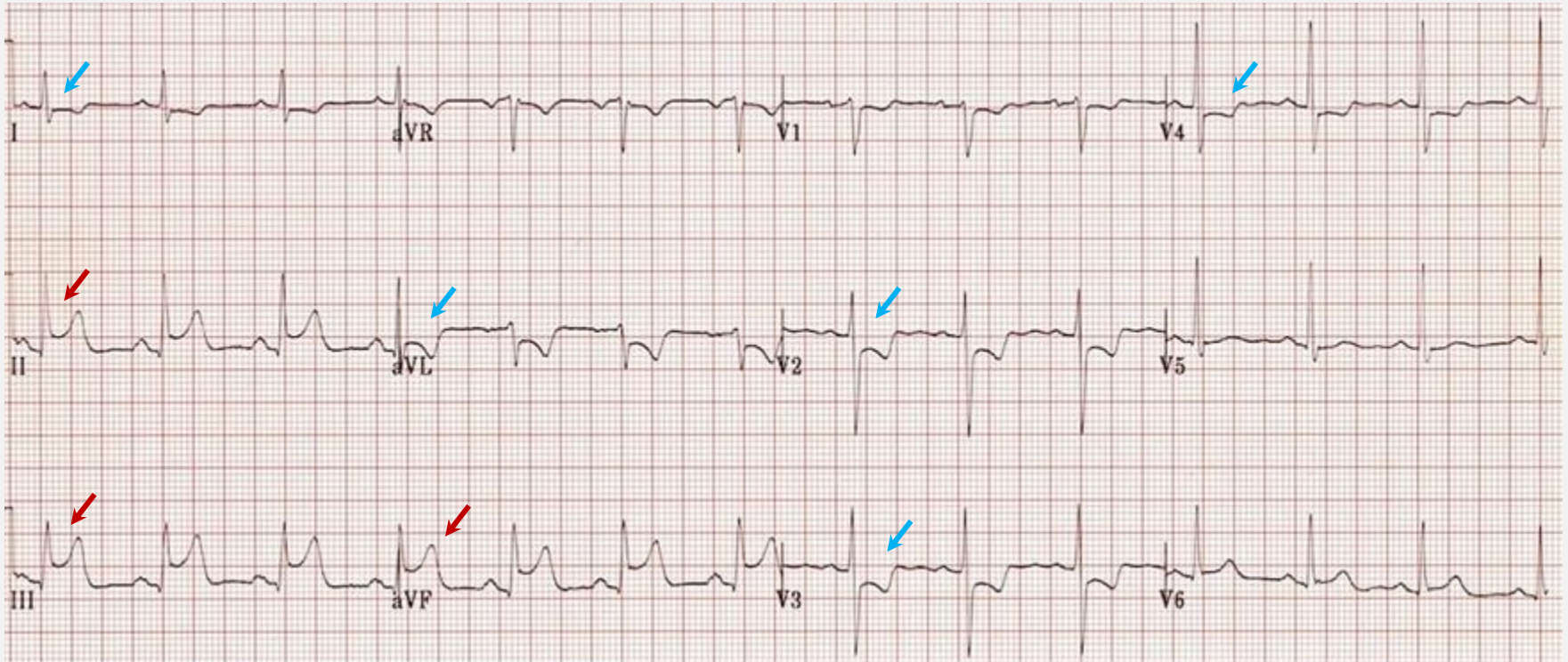
- Sometimes reciprocal changes in STEMI may be missing due to absence of ECG lead with an opposite angle of observation or if current of injury is not strong enough to be detected at the opposite site of observation

STEMI



- This is acute period of anterolateral STEMI (ST elevation in anteroseptal (V1-V3) and high lateral (I, aVL) leads - look at red arrows, with reciprocal ST depression in inferior leads II, III, aVF) - look at blue arrows.

STEMI



This is inferior STEMI (ST elevation in leads II,III, aVF - red arrows, with reciprocal ST depression in anterior leads V2,V3,V4 and lateral I,aVL - blue arrows).

NSTEMI



In case of NSTEMI an ECG will show the following characteristics:

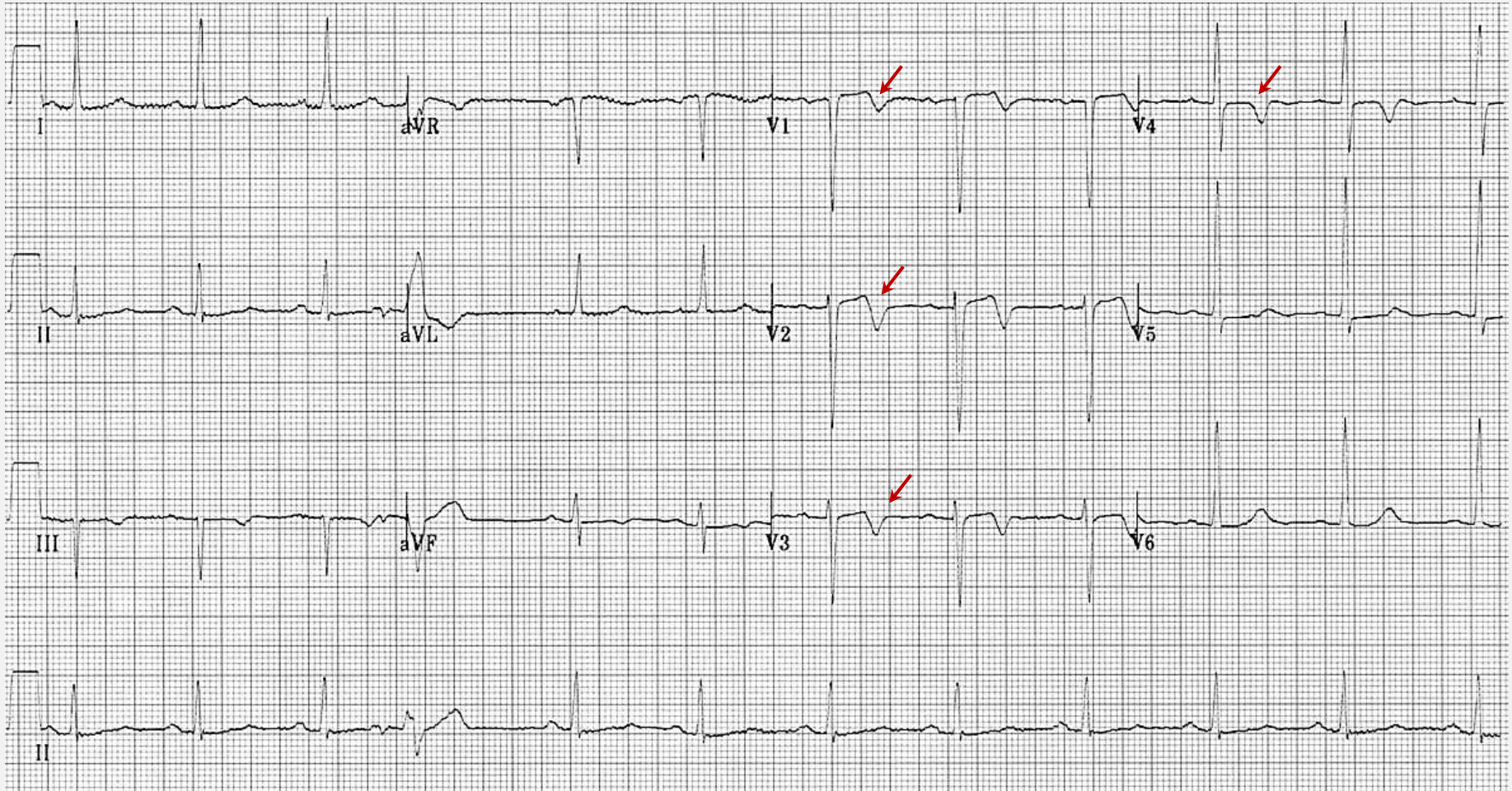
- depression of ST wave (horizontal or downsloping ST depression ≥ 0.5 mm at the J-point in ≥ 2 contiguous leads indicates myocardial ischaemia)
- T-wave flattening or inversion (at least 1mm deep).
- no progression to Q wave.

Unstable angina



- ECG may show ST-T abnormalities, or may be normal (only typical chest pain is present) but laboratory markers of myocardial necrosis are negative!

NSTEMI

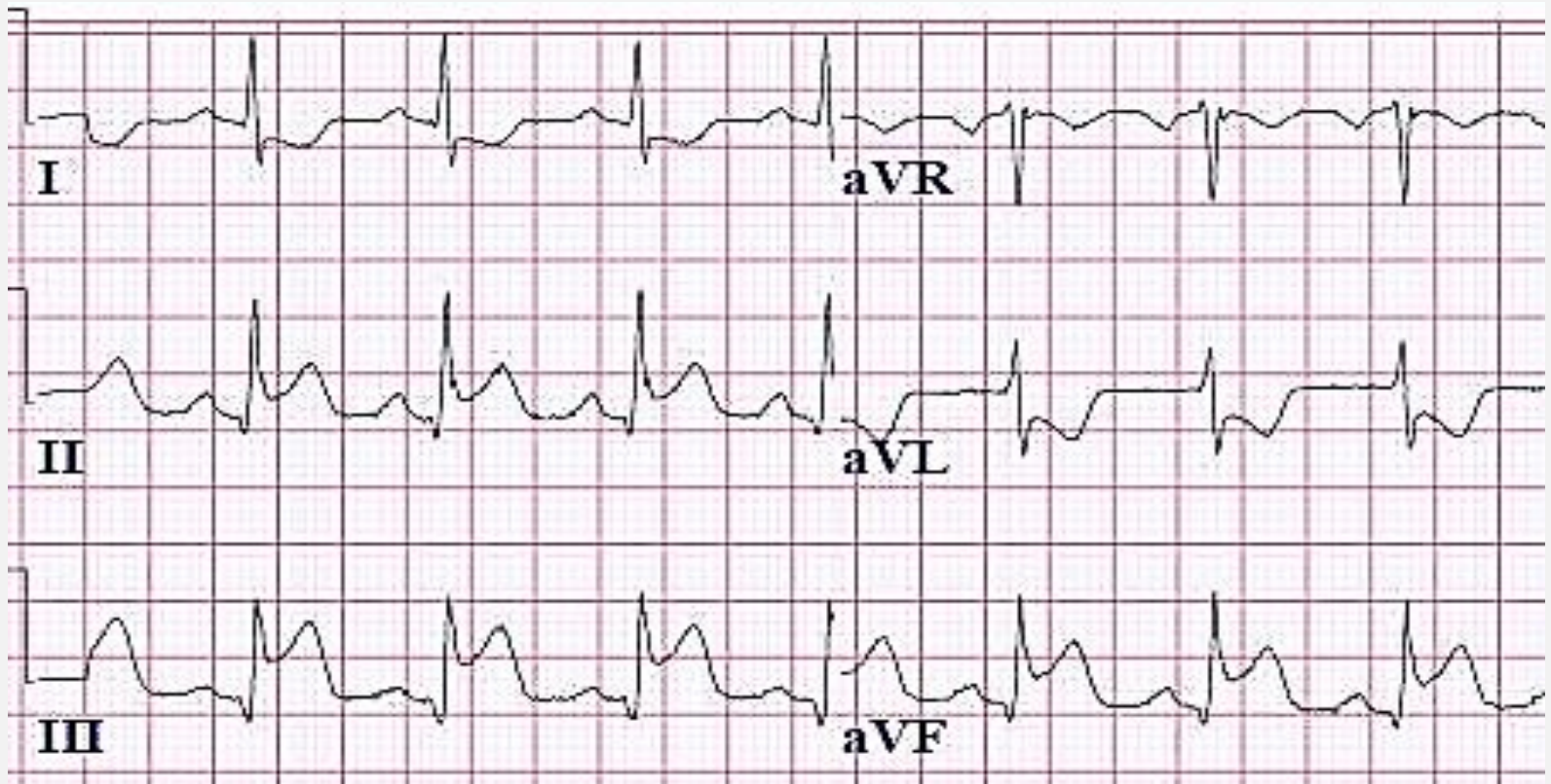


There are abnormal T waves in V1-4 – biphasic in V1-3, inverted in V4 (red arrows). Note left axis deviation, signs of left ventricular hypertrophy.

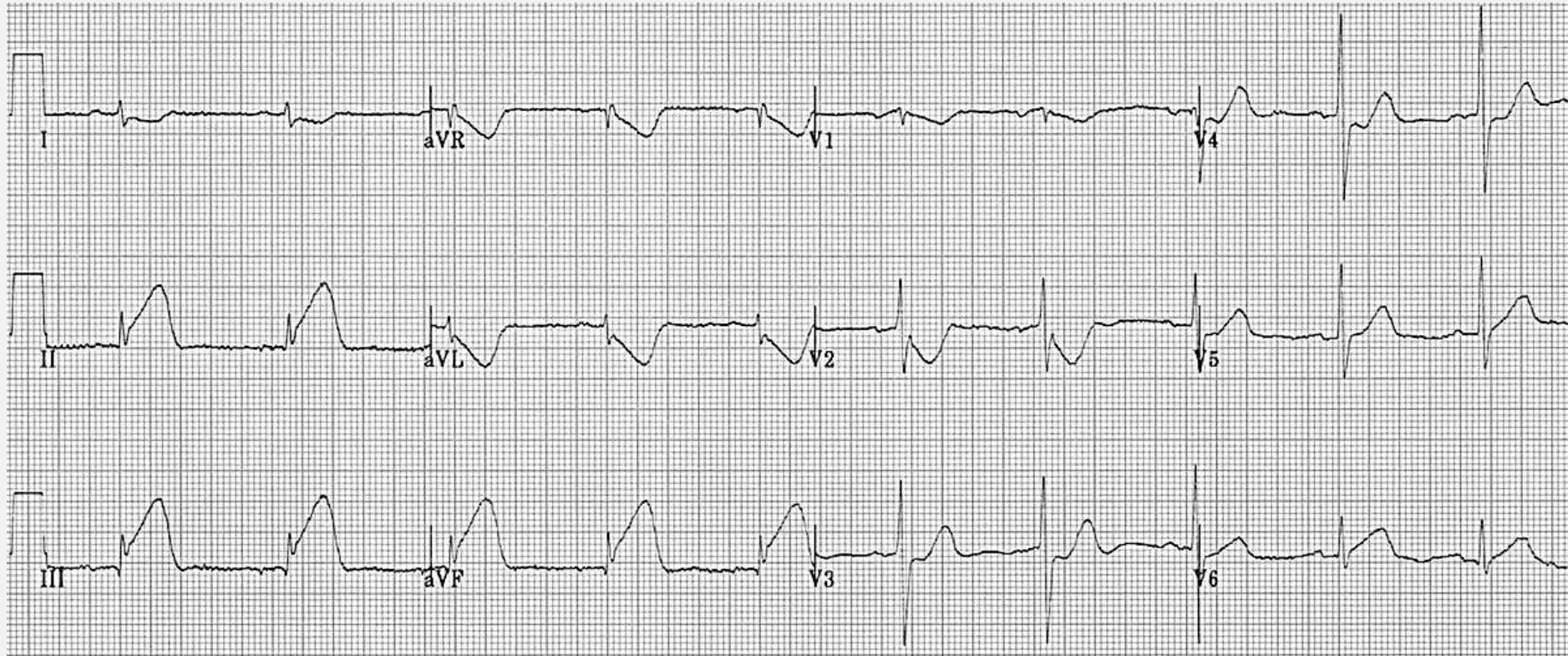
Practical part



Decode an ECG. Try to find all the repolarization abnormalities.

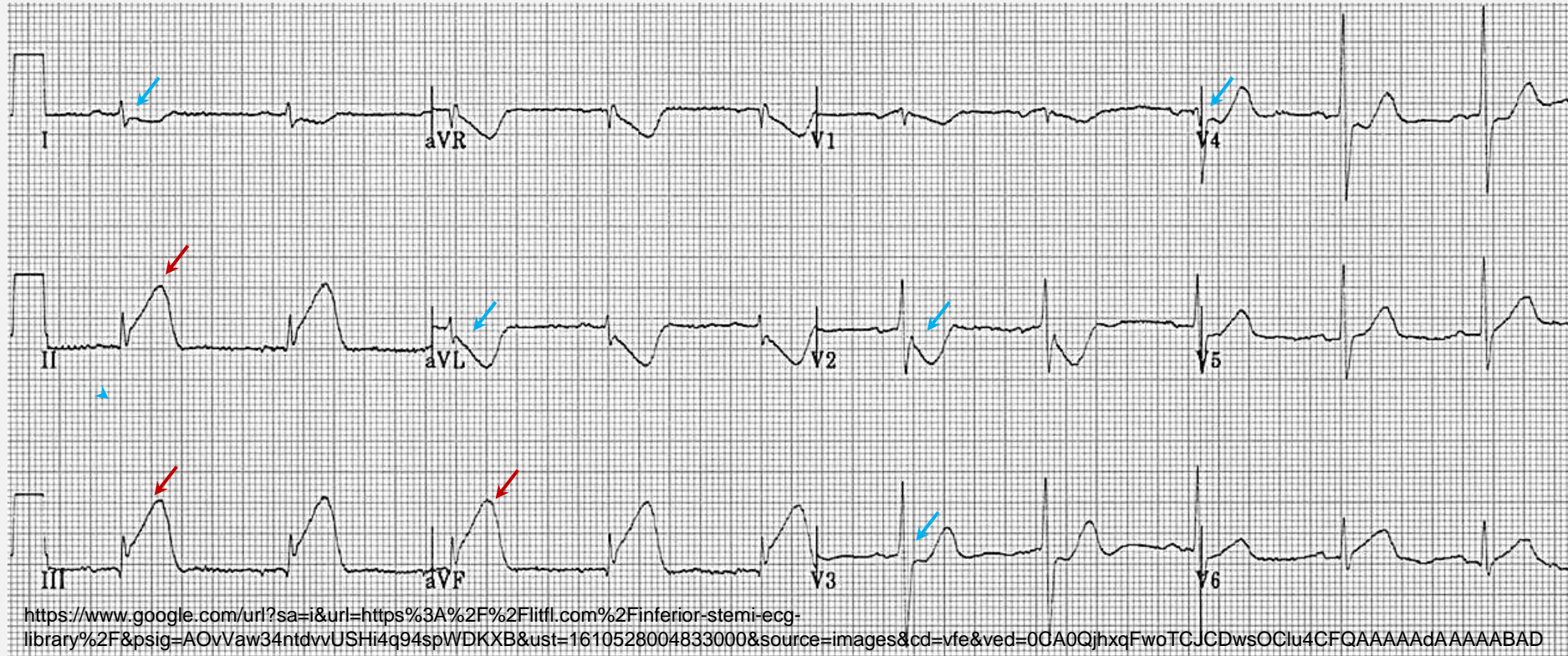


Practical part



- The patient is admitted to the department with ischemic chest pain during the past 3 hours not relieved by nitroglycerin. Decode an ECG and give your conclusion.

Practical part



- **Conclusion - prolonged ischemic chest pain not relieved by nitrates indicates ACS. On ECG -ST elevation in leads II, III, avF (red arrows), reciprocal ST depression in leads I, avL, V2-V4 (blue arrows). It is inferior STEMI.**

Posterior MI

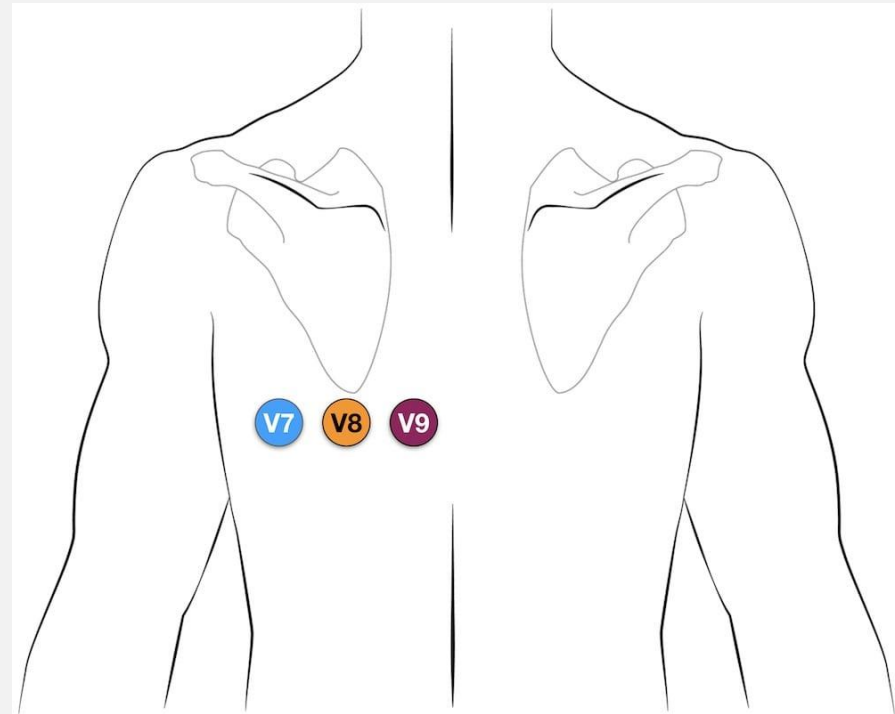


- Posterior infarction typically results from an occlusion of the right coronary artery.
- Because none of the standard leads overlie the posterior wall (thus we cannot visualize ST elevation and Q wave), the diagnosis requires finding reciprocal changes in the anterior leads.
- The normal QRS complex in lead V1 consists of a small R wave and a deep S wave; therefore, the presence of a tall R wave, particularly with accompanying ST segment depression can be considered as reciprocal changes of posterior STEMI.
- Another method of detection of posterior STEMI - is placement of additional posterior leads.

Posterior leads



- **V7** - Left posterior axillary line, in the same horizontal plane as V6.
- **V8** - Tip of the left scapula, in the same horizontal plane as V6.
- **V9** - Left paraspinal region, in the same horizontal plane as V6.



<https://litfl.com/wp-content/uploads/2018/08/Posterior-leads-V7-V8-V9-ECG-placement-768x614.jpg>

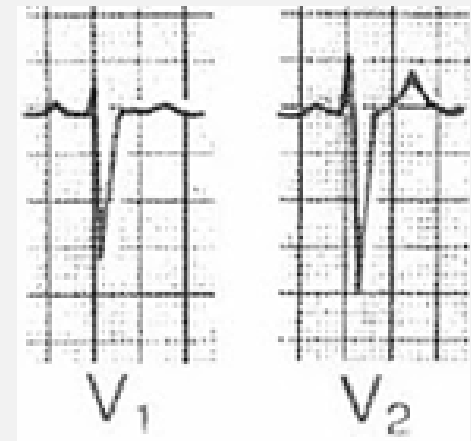
<https://litfl.com/posterior-myocardial-infarction-ecg-library/>

Posterior STEMI

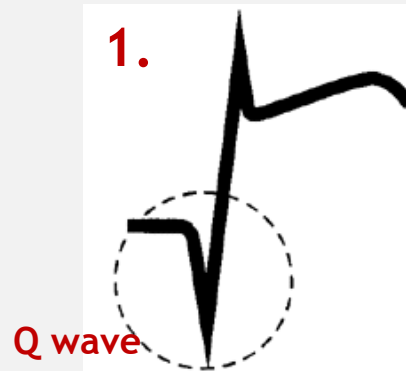


- The normal QRS complex in lead V1 consists of a small R wave and a deep S wave; therefore, the presence of a tall R wave, particularly with accompanying ST segment depression can be considered as reciprocal changes of posterior STEMI.

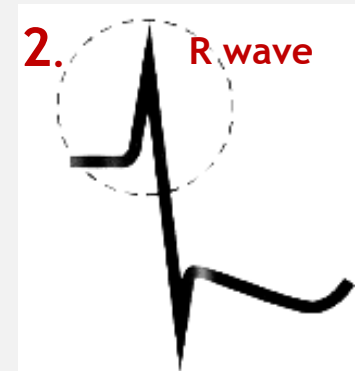
Normal QRS in leads V1, V2



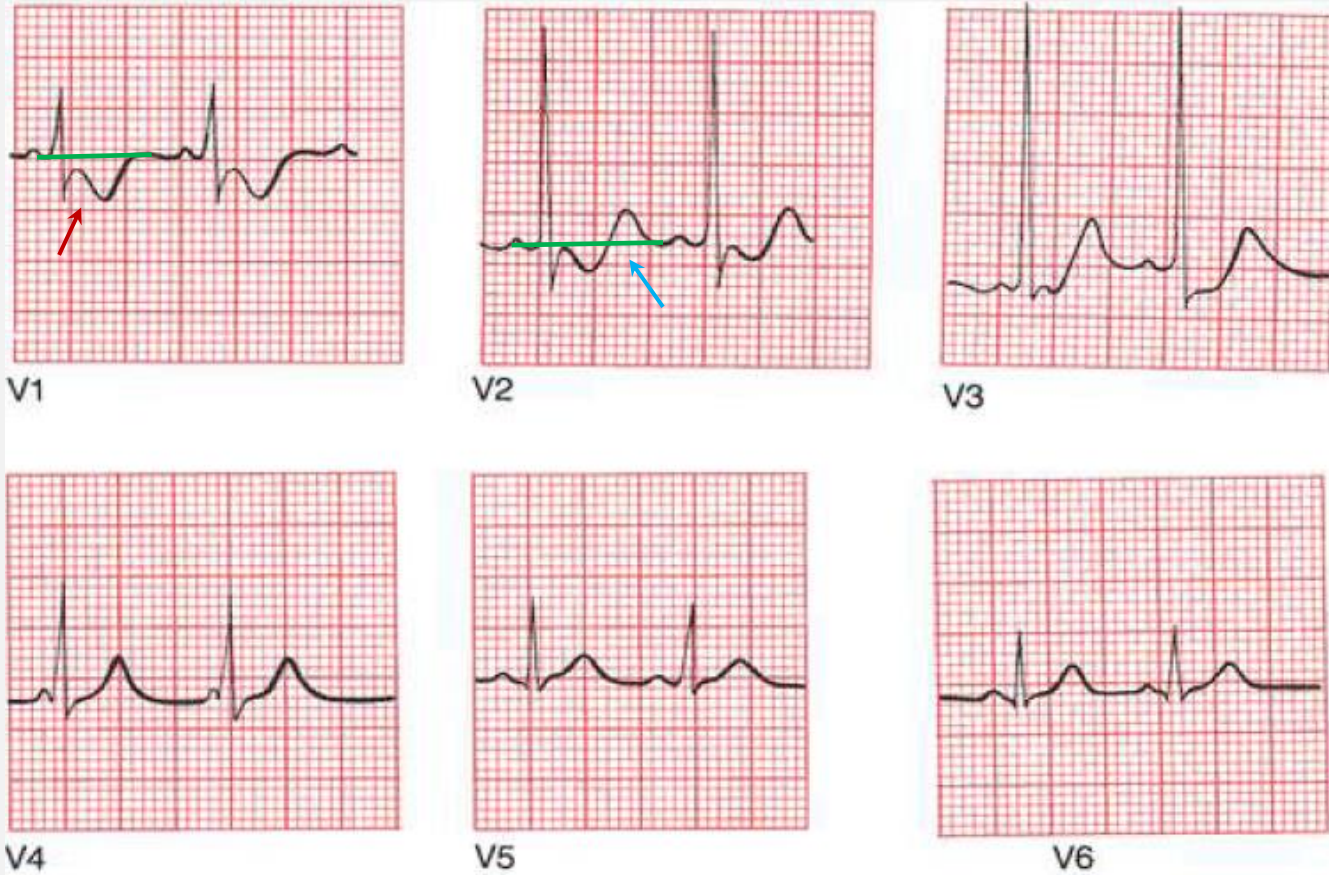
1. Q wave with ST elevation in posterior leads



2. Tall R wave with ST depression in V1, V2 - reciprocal changes (mirror reflexion) of posterior leads



Posterior STEMI



- Tall R waves in leads V1, V2; ST depression with T wave inversion in lead V1 (red arrow), and bifasic T wave in V2 (blue arrow)- reciprocal changes indicating posterior STEMI.

MI in LBBB



It is difficult to diagnose acute MI in the presence of left bundle branch block (LBBB).

A new LBBB is always pathological and can appear as a result of acute MI.

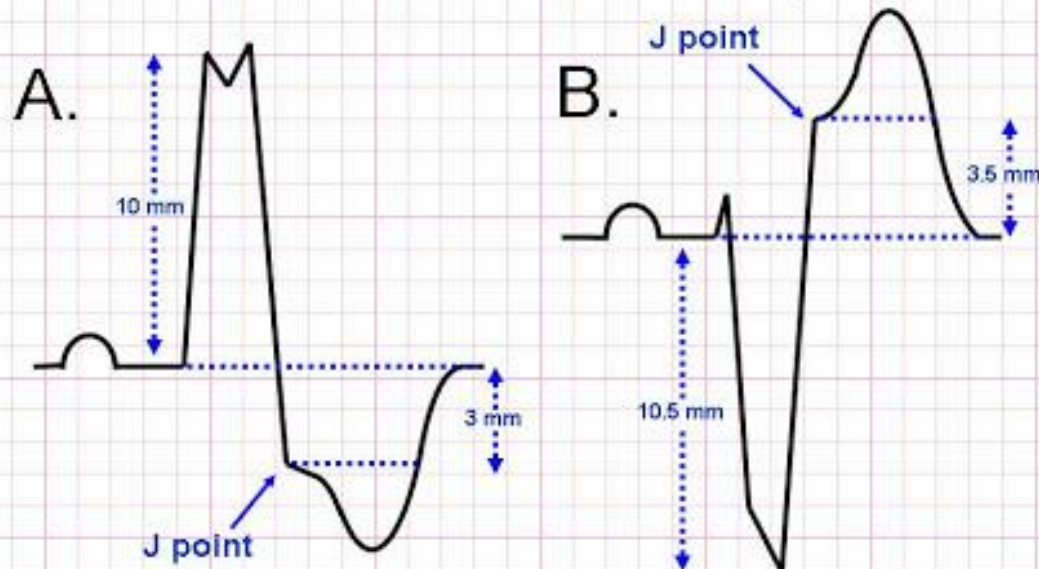
To diagnose MI in LBBB the following criteria can be used:

- Original Sgarbossa criteria;
- Smith-modified Sgarbossa criteria.

MI in LBBB-cont.



Excessive Discordance
ST-Segment Depression or Elevation
> 0.2 the QRS Complex



ems12lead.com

Abnormal for LBBB or Paced Rhythm

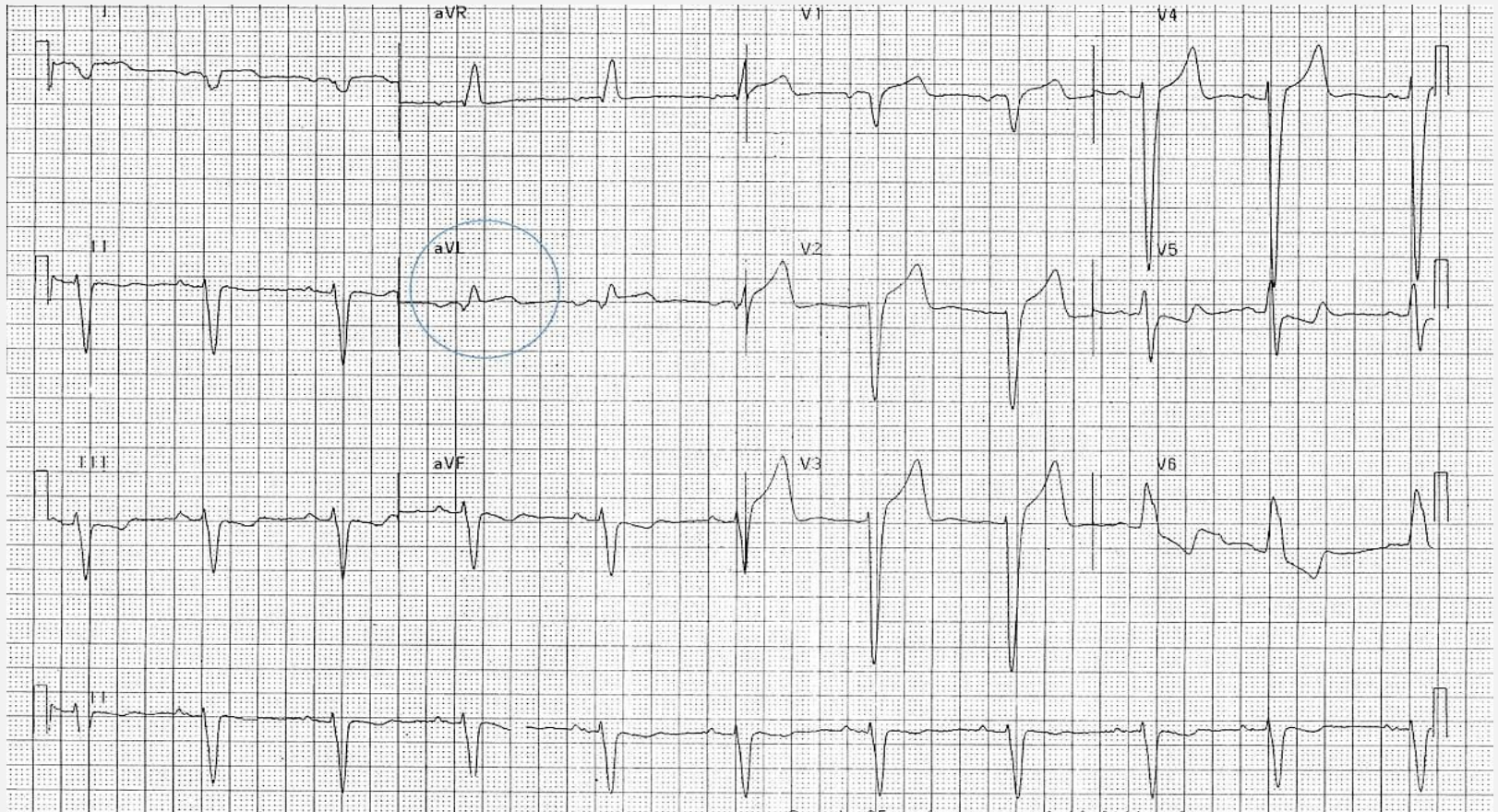
MI in LBBB-cont.



Smith-modified Sgarbossa criteria:

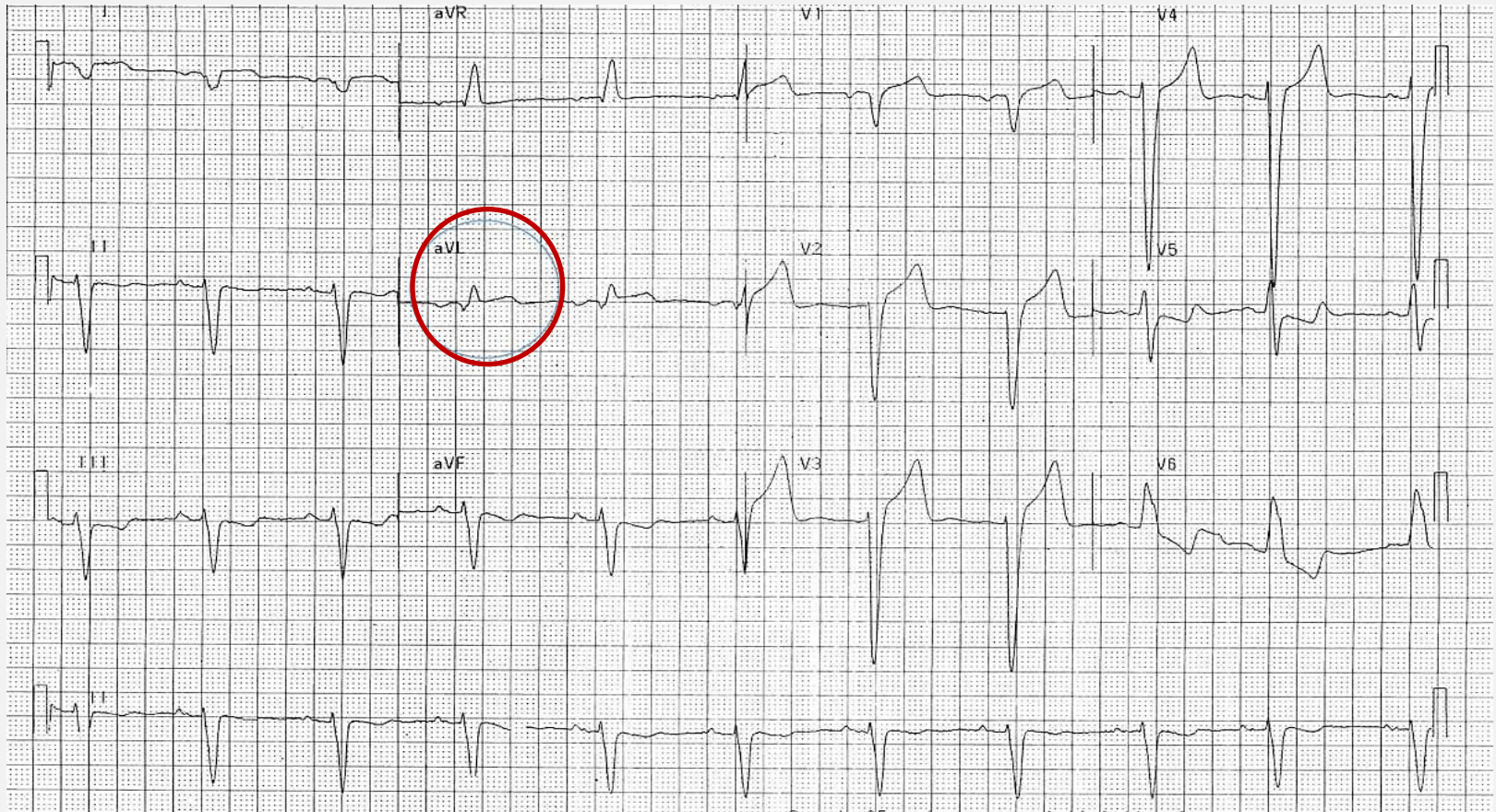
1. ≥ 1 lead with ≥ 1 mm of concordant ST elevation.
2. ≥ 1 lead of V1-V3 with ≥ 1 mm of concordant ST depression.
3. ≥ 1 lead anywhere with ≥ 1 mm ST segment elevation and proportionally excessive discordant ST segment elevation, as defined by $\geq 25\%$ of the depth of the preceding S-wave.

Practical part



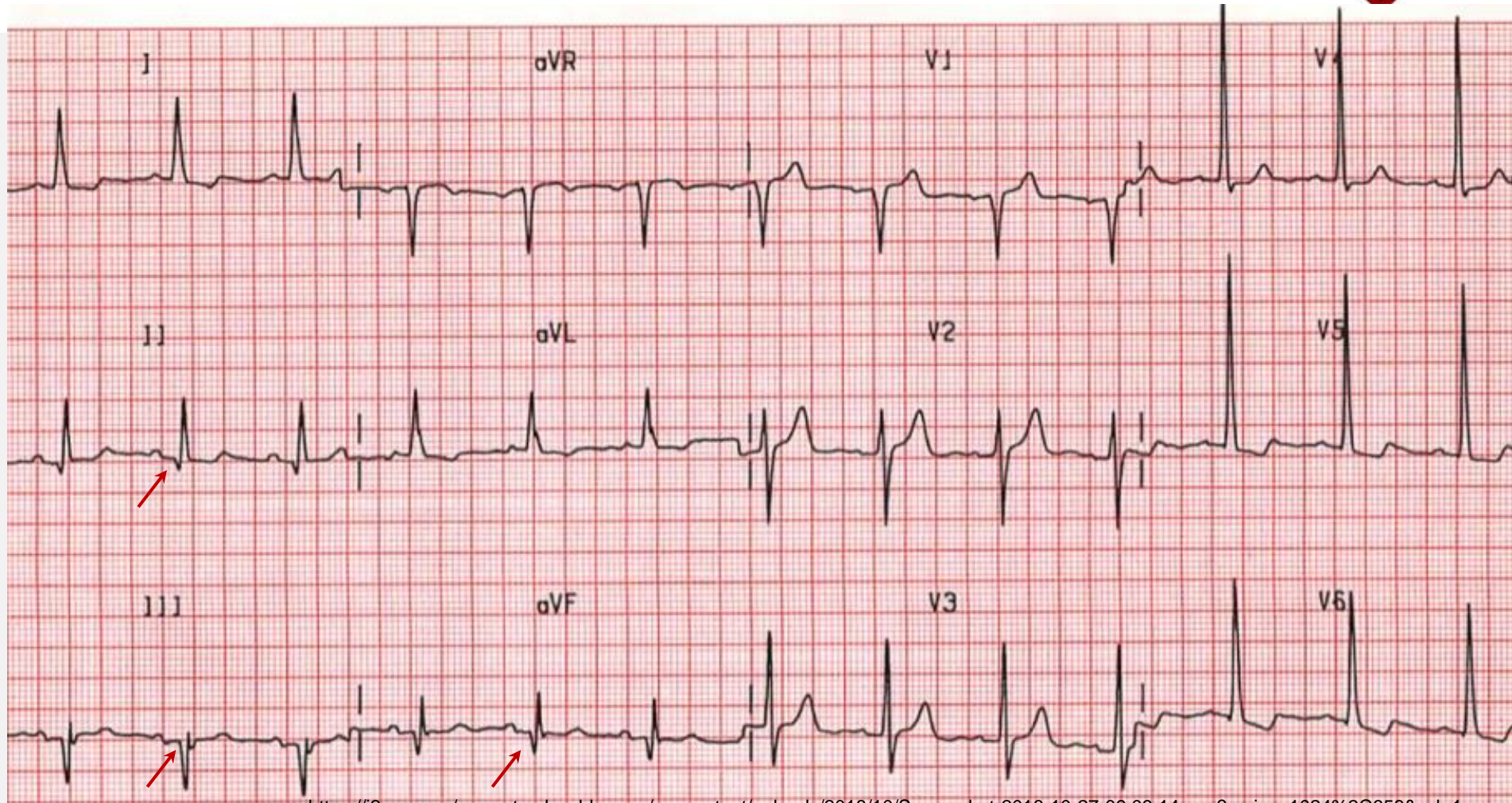
A patient presented with retrosternal chest pain and had elevated cardiac enzymes. Decode an ECG and give your conclusion.

Practical part



Answer: lateral MI (positive Sgarbossa criterion - there is 1mm concordant ST elevation in aVL (= 5 points)).

Old MI



<https://i2.wp.com/www.stemlynsblog.org/wp-content/uploads/2018/10/Screenshot-2018-10-27-09.09.14.png?resize=1024%2C658&ssl=1>

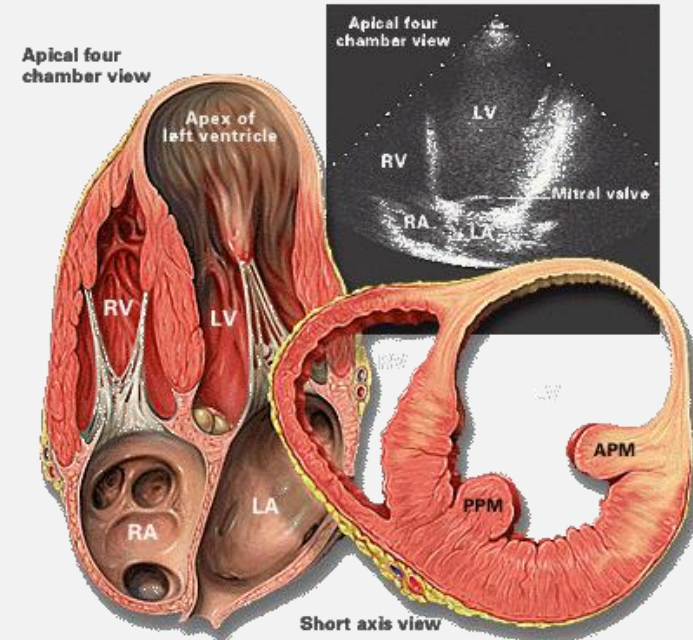
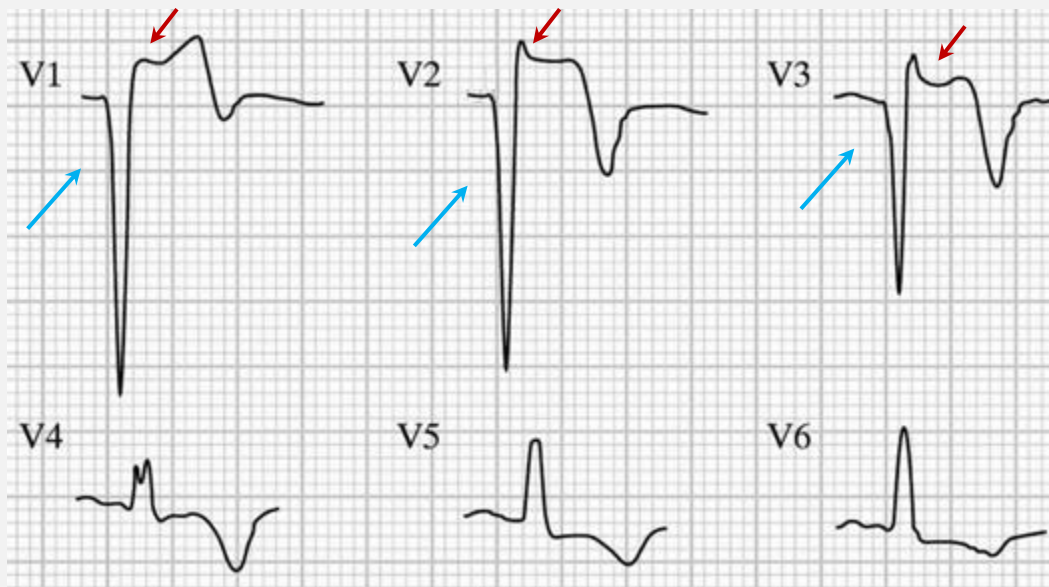
- There are pathological Q waves in leads II, III, avF with positive T waves indicative for old inferior STEMI.

Left ventricular aneurism



- If myocardial infarction is complicated with left ventricular aneurism (LVA) there'll be “frozen” ECG with permanent ST elevation occurring 2 weeks after a known transmural myocardial infarction (usually an anterior MI).
- LVA is usually seen in precordial leads (V1-V4 in case of apical aneurism) and is associated with the presence of Q waves indicating the previous anterior MI.
- Without known anamnesis of the patient (history of previous MI) ECG signs of cardiac aneurism may be confused with acute myocardial infarction.

Left ventricular aneurysm



- Note presence of ST elevation in leads V1-V3 (red arrows) along with deep Q waves in the same leads (blue arrows).

STEMI vs LVA



STEMI	LVA
New ST elevation on ECG compared with previous ECG	Usually develops after MI and stay unchanged afterwards
ST elevation will be present with reciprocal changes (ST depression)	ST elevation without reciprocal changes
ST elevation will change dynamically over the time	ST elevation is persistent without dynamic changes
Is associated with clinical picture of MI - prolonged ischemic chest pain, hemodynamic instability, etc	Without clinical picture of MI

**Thank you for your
attention!**

