CORONARY ARTERY DISEASE (CHD): ANGINA PECTORIS, MYOCARDIAL INFARCTION. CLINIC, DIAGNOSTICS, CLASSIFICATION, PRINCIPLES OF TREATMENT.

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ISCHEMIC HEART DISEASE (CHD) IS

a disease based on insufficiency of coronary blood supply, myocardial ischemia caused by atherosclerosis, atherothrombosis and (or) spasm of the coronary arteries.

MYOCARDIAL ISCHEMIA IS

a restriction of coronary blood supply, and as a result, a restriction of oxygen delivery to the heart muscle. Ischemic heart disease - insufficient nutrition of the heart muscle.

THUS, ISCHEMIC HEART DISEASE (CHD) IS

acute or chronic heart disease

due to a decrease in myocardial blood supply

due to a pathological process in the coronary artery system

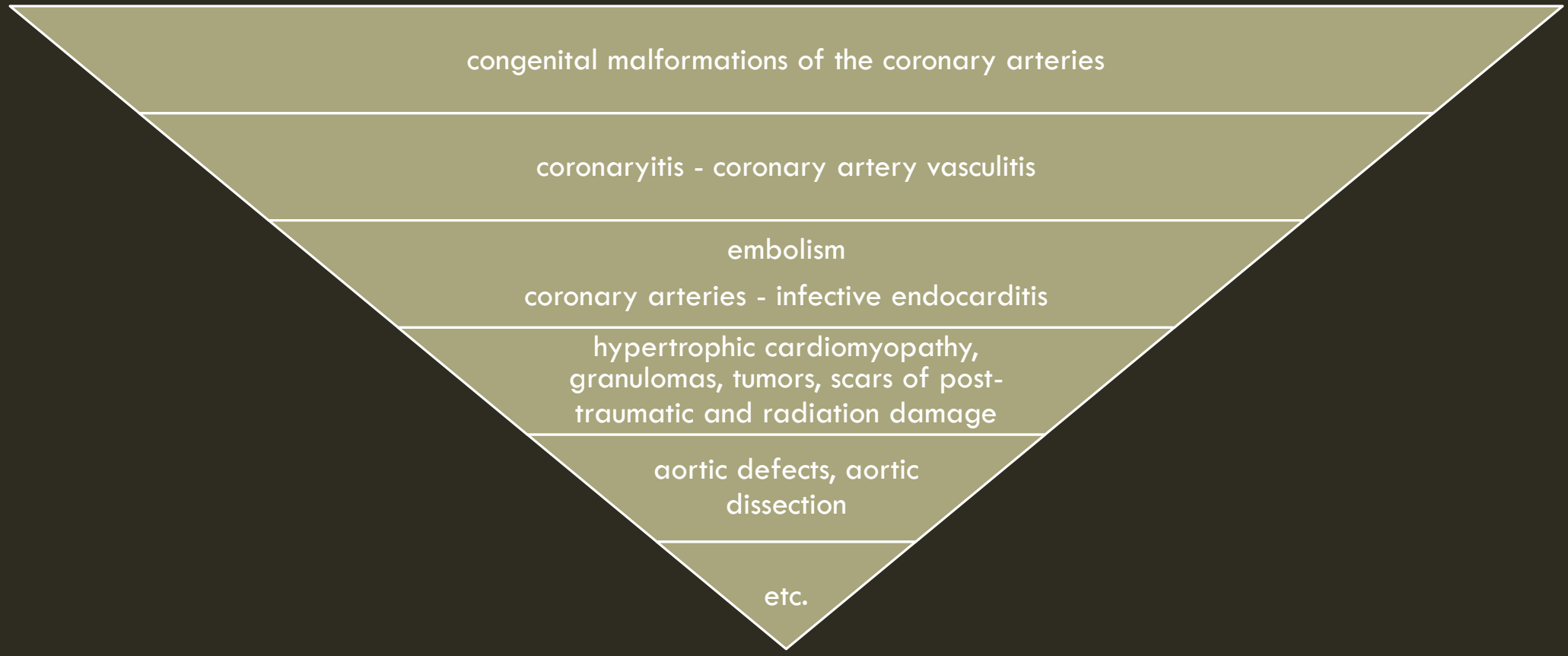
Acute coronary syndrome is a group of symptoms and signs that suggest an acute myocardial infarction or unstable angina. This term is used in establishing a preliminary diagnosis.

The European Society of Cardiology (ESC, 2019) defines chronic coronary heart disease as "a dynamic process of atherosclerotic plaque accumulation and functional changes in coronary circulation that can be altered through lifestyle adjustments, pharmacological therapy and revascularization, leading to stabilization or regression of the disease.

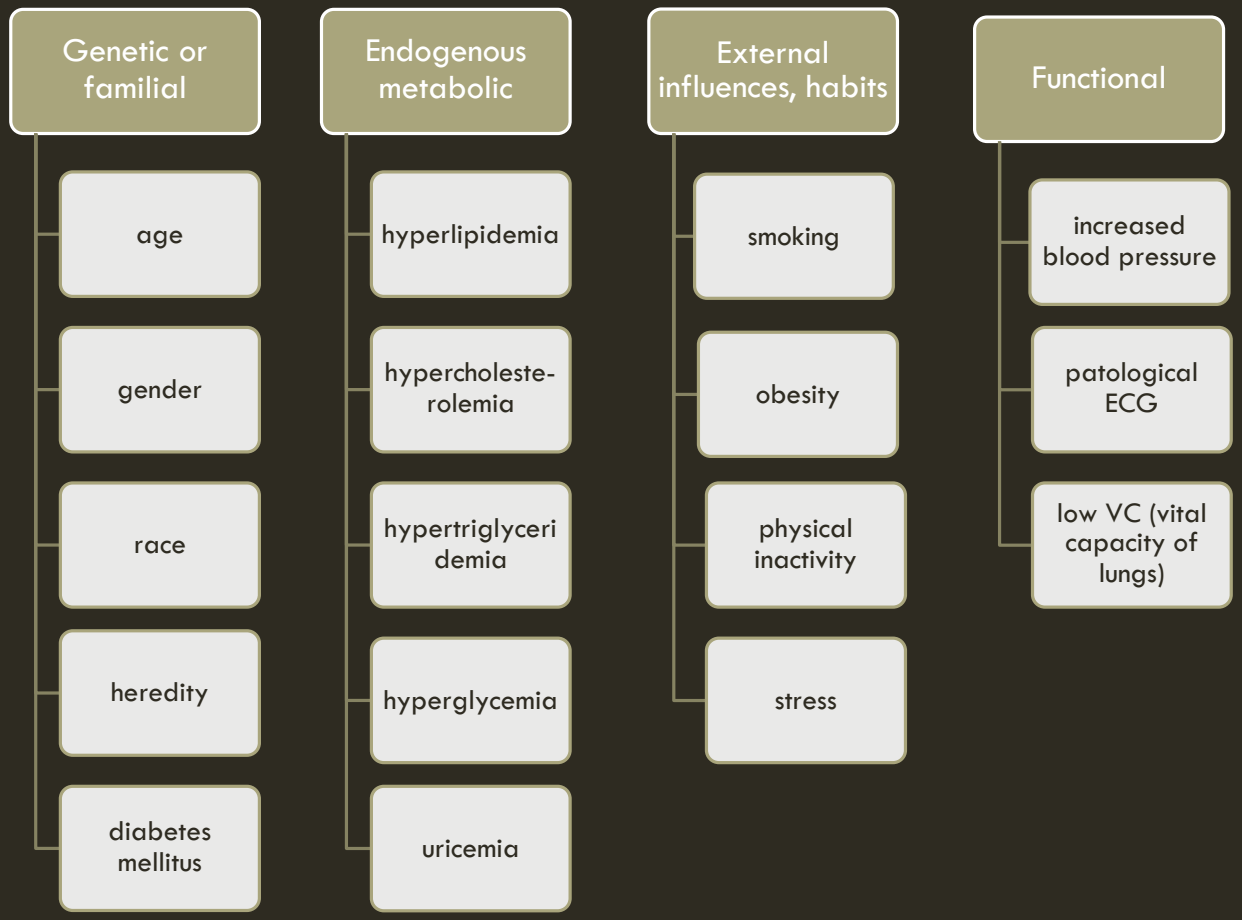
ETIOLOGY OF ISCHEMIC HEART DISEASE

Atherosclerosis of the coronary arteries – 95 %	Blood clots of the coronary arteries	Coronary artery spasm
Increased myocardial oxygen demand	Impossibility of an adequate increase in myocardial perfusion	Severe LV hypertrophy

OTHER REASONS OF IHD



RISK FACTORS FOR ISCHEMIC HEART DISEASE



MULTIPLE RISK FACTORS

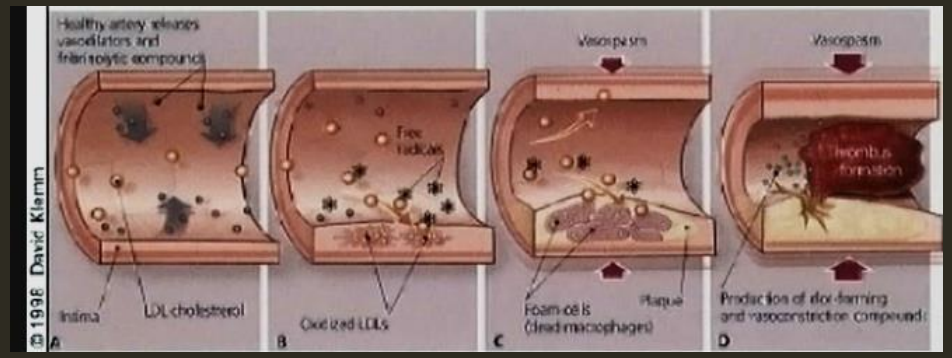
Lipid metabolism disorders	• An increase in serum cholesterol by 15% increases the risk of coronary heart disease by 35%.
Arterial hypertension	• For patients with blood pressure above 180 mm Hg. Art., the risk of coronary lesions is 8 times higher than for persons with normotension
Smoking	• Decrease in coronary blood flow, myocardial hypoxia; rhythm disturbances, coronary spasm, accelerated platelet aggregation
Hypodynamia	• The risk of coronary insufficiency is 3 times higher
Obesity	• The probability of CHD increases by 14% with an excess of weight of 10%
Gender	• At a young age in men, the incidence is much higher. After menopause, the incidence is about the same in men and women
Genetic predisposition	• A burdened family history was noted in 57% of patients with coronary artery disease

CLINICAL CLASSIFICATION OF CHD

1. Sudden cardiac death
2. Stenocardia
3. Myocardial infarction
4. Postinfarction cardiosclerosis
5. Violations of heart rhythm or conduction
6. Heart failure
7. "Mute" (painless) form of ischemic heart disease

THE MAIN PATHOGENETIC LINKS ARE ACUTE AND CHRONIC CORONARY INSUFFICIENCY

Narrowing of proximal (epicardial) CA by atherosclerotic plaque with limited coronary blood flow or its functional reserve and the inability to adequately dilate blood vessels in response to increased myocardial oxygen demand



Pronounced spasm of the coronary artery

Thrombosis of the coronary artery with the possible formation of microthrombi in the microcirculatory vascular bed

Microvascular dysfunction (syndrome X)

FACTORS THAT AGGRAVATE MYOCARDIAL ISCHEMIA

- Causes of increased tone and spasm of CA are increased synthesis of vasoconstrictors (endothelin, thromboxane A2, angiotensin II, serotonin).
- Decreased concentrations of vasodilators (nitric oxide, prostacyclin, endothelium-relaxing factor)
- Increased platelet aggregation and activity.

FACTORS THAT INCREASE MYOCARDIAL OXYGEN DEMAND:

- Tachycardia
- AH
- Hypothyroidism
- Heart failure.
- Valve heart defects.
- Use of catecholamines, sympathomimetic (cocaine) intoxication, bronchodilators, tricyclic antidepressants.

FACTORS THAT CAUSE A DECREASE IN OXYGEN DELIVERY TO THE HEART MUSCLE:

- Anemia, polycythemia.
- Hypoxemia: asthma, chronic obstructive pulmonary disease, sleep apnea.
- Hypotension.
- Bradycardia.
- Carbon monoxide poisoning.

WHAT IS ANGINA PECTORIS (STENOCARDIA)?

Angina is chest pain caused by damage to the arteries of the heart. Pain is a sign that the heart is not getting enough oxygen. With angina pectoris, pain manifests itself as an uncomfortable feeling of compression, compression, distention in the center of the chest. Pain can radiate to the neck, shoulder, lower jaw, back and left arm.

ANGINA PECTORIS (CLASSIFICATION OF THE CANADIAN CARDIOVASCULAR SOCIETY)

I functional class (I FC)	• Normal physical activity, such as walking or climbing stairs, does not cause angina. Angina pectoris occurs with strenuous or fast, or prolonged exertion during work or outdoor activities.
II functional class (II FC)	• Slight limitation of normal activity: brisk walking or climbing stairs, walking uphill, walking or climbing stairs after eating, in cold winds or when emotionally stressed, or in the first few hours after waking up. Walk more than two blocks (more than 500 m) and climb more than one flight of a normal staircase at a normal pace and under normal conditions.
III functional class (III FC)	• Severe limitation of normal physical activity: walking a distance of one or two blocks (100-150m) and climbing less than one flight of an ordinary staircase at a normal pace.
IV functional class (IV FC)	• Angina pectoris occurs with small physical exertion, when walking on level ground at a distance of less than 100 m. The occurrence of angina attacks at rest is characteristic. There may be seizures that awaken the patient at night, after eating, during the transition from vertical to horizontal position (increased venous blood flow to the heart).

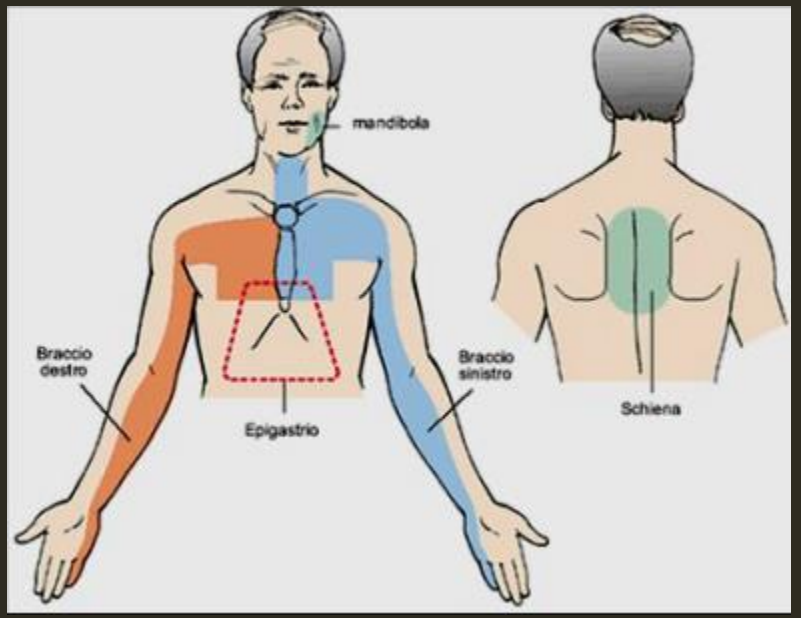
ANGINA ATTACK

The pain is cutting, pressing, burning, squeezing

Difficult to express discomfort - heaviness, compression, tightness

Sometimes the patient can deny the presence of pain

Typical localization of pain	•Retrosternal localization of pain
Typical irradiation of pain	•In the left shoulder and arm
Possible localization of pain	•The pain begins inside the chest behind the sternum and from here spreads in all directions
Possible irradiation of pain	•In the shoulder blade, neck, face, jaw, teeth, in the right shoulder and scapula. The more severe the attack, the wider the area of irradiation
Intensity and duration	•The duration of an anginal attack is almost always more than one minute, but less than 15 minutes (most often 2-5 minutes)
The structure of a pain attack	•The pains increase gradually in the form of sequential ever increasing attacks of burning and compression. Having reached its climax, the pains quickly disappear
Clenched fist symptom	•The patient, to describe his sensations, puts his fist or palm, or two palms on the sternum



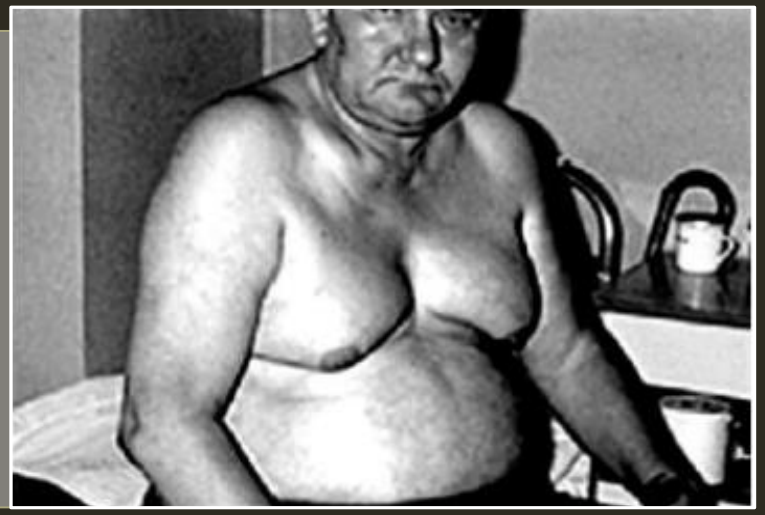
Collection of complaints and medical history remains a key element in the diagnosis of angina. In most cases, this makes it possible to establish an accurate diagnosis, and physical examination and instrumental methods of examination are necessary only to confirm or rule out alternative pathology.

- Shortness of breath
- Rhythm disturbances
- Medical history - consideration of risk factors

INTERICTAL EXAMINATION

Identifying signs
indicating the existence
ATHEROSCLEROSIS (for example -
xanthelasm) and identification
risk factors for ischemic heart disease -
heredity, obesity,
arterial hypertension,
physical inactivity, smoking, etc.

Heart examination - sometimes
left shift, widening and
weakening of the apical
impulse on palpation,
expansion of borders to the left at
percussion, weakening the SI above the
apex of the heart, accent SII above
aorta on auscultation, with
exploration of peripheral
arteries - their hardening and
tortuosity



THE MAIN INSTRUMENTAL AND LABORATORY METHODS OF RESEARCH OF CORONARY HEART DISEASE:

ECG in 12 leads at rest

ECG in dynamics

(during the attack, 2 hours after its end and 24 hours later)

The absence of changes on the ECG at rest does not rule out the diagnosis of coronary heart disease

Functional stress tests

or Holter ECG - if necessary (suspected arrhythmia, vasospastic angina)

Identify transient signs of myocardial ischemia

Standard blood test - general blood and urine test, fasting glucose, creatinine, lipid profile (total cholesterol, low-density lipoprotein cholesterol, triglycerides)

EchoCG

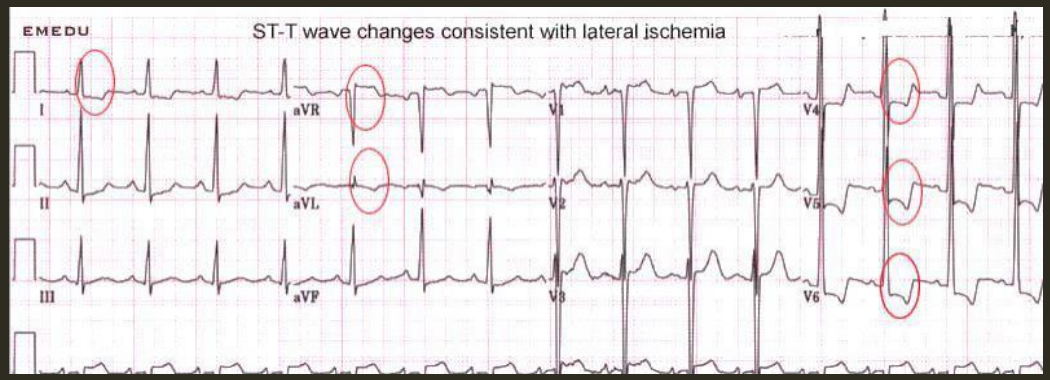
MRI of the heart (with non-informative echocardiography)

Chest radiography (if necessary)

Myocardial scintigraphy with thallium-201

Coronoventriculography

CHANGES IN THE ECG WITH AN ATTACK OF ANGINA



T wave inversion/violation of repolarization

- Increased amplitude of the T wave (high "coronary" T waves).
- The apex of the T wave becomes pointed.
- The T wave is reduced, flattened.
- Wave T two-phase with an initial negative phase.
- T wave becomes negative (T wave inversion)
- T wave changes may be isolated and not combined with depression of the S-T segment

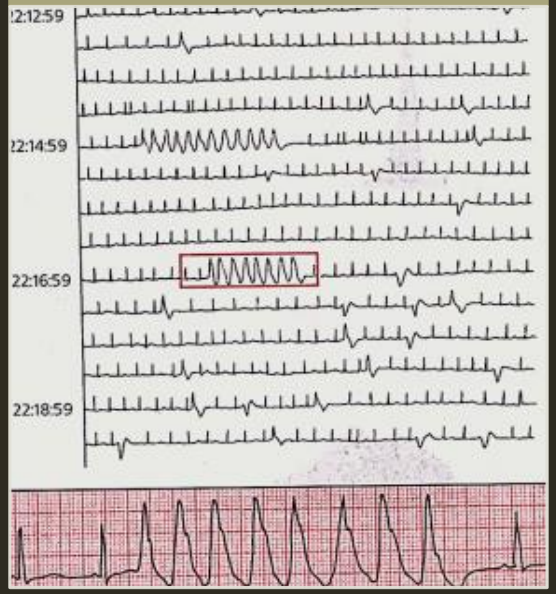
S – T segment depression/ subendocardial ischemia

- The S-T segment will move downward by 1-2 mm, having a horizontal direction (horizontal depression of the S-T segment)
- The S-T segment will shift downward by 1-2 mm, having an obliquely descending direction (obliquely descending depression of the S-T segment)

S-T segment rise/transmural ischemia

- The S-T segment will move upward by 1–2 mm (elevation) in severe ischemia

Ventricular tachycardia/electrical instability of the myocardium due to ischemia



COMPLICATIONS OF STENOCARDIA

- Myocardial infarction
- Sudden cardiac death
- Acute vascular insufficiency
- Rhythm and conduction disturbances
- Chronic HF

PRINCIPLES OF ANGINA PECTORIS TREATMENT

Non-drug

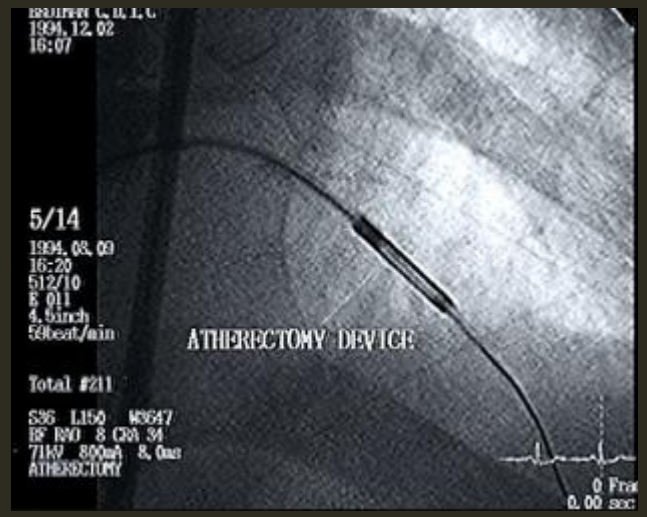
- correction of risk factors
- lifestyle modification

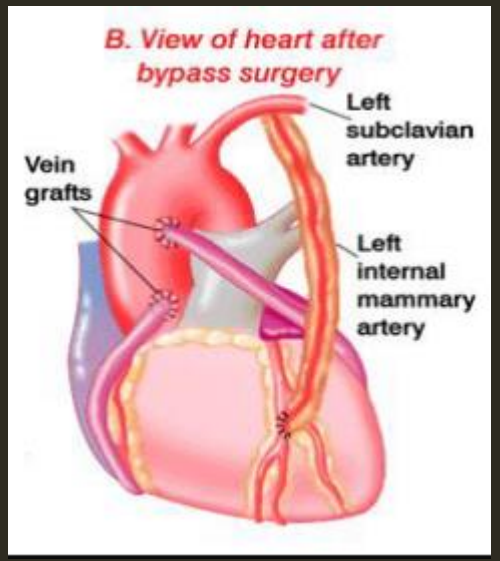
Medication

- in the interictal period and during attacks of angina pectoris (mainly nitrates, beta adrenergic blockers, calcium antagonists, hypolipidemic, antiplatelet agents)

Surgical

- coronary angioplasty
- shunting





MYOCARDIAL INFARCTION (MI)

(MI) — It is ischemic necrosis of a cardiac muscle, developing as a result of acute insufficiency of coronary blood circulation

Classification:

On size and depth of defeat (Q, not Q MI)

On character of a course (primary, repeated, relapsing)

On localization

On a stage

On presence of complications

ACUTE MI

The diagnosis is established with indication of the date of occurrence (up to 28 days): localization ventricle (PN); primary, recurrent, recurrent (it is not necessary to note the size and location if there are difficulties in ECG diagnosis):

Acute myocardial infarction with the presence of a pathological Q wave.

Acute MI without pathological Q wave

Acute MI is uncertain

Recurrent MI (up to 28 days, diagnosed with repeated increase followed by a natural decrease in the level of cardiospecific enzymes)

Repeated IM

ACS with stable elevation or without ST segment elevation on the ECG. This is a preliminary diagnosis before establishing the presence of heart damage due to myocardial ischemia or its absence (unstable angina). On the ECG, elevation or depression of the ST segment reflects ischemia before the development of myocardial necrosis or sudden cardiac death (up to 3 days). However, in some patients with clinical symptoms of corticosteroids changes in the ECG may be absent.

THE CLINIC OF MI – DEPENDS ON A STAGE OF MI AND PRESENCE OF COMPLICATIONS

Painful syndrome

Syndrome of arrhythmias

Syndrome of circulatory insufficiency

Allocate some clinical variants of the beginning of MI:

Painful (anginous) beginning variant (status anginosus)	Asthmatic variant (status asthmatics)	Abdominal variant (status gastralgicus)	Arrhythmic a variant	Cerebrovascular a variant	A few symptoms or asymptomatic beginning

PAINFUL SYNDROME IN ACS

Prolonged (more than 20 minutes) anginal pain at rest, the onset of severe angina for the first time in life (III functional class according to the classification of the Canadian Society of Cardiovascular Diseases).

- Prolonged pain is observed in 80% of patients

Or recent destabilization of pre-stable angina with at least functional class (progressive angina)

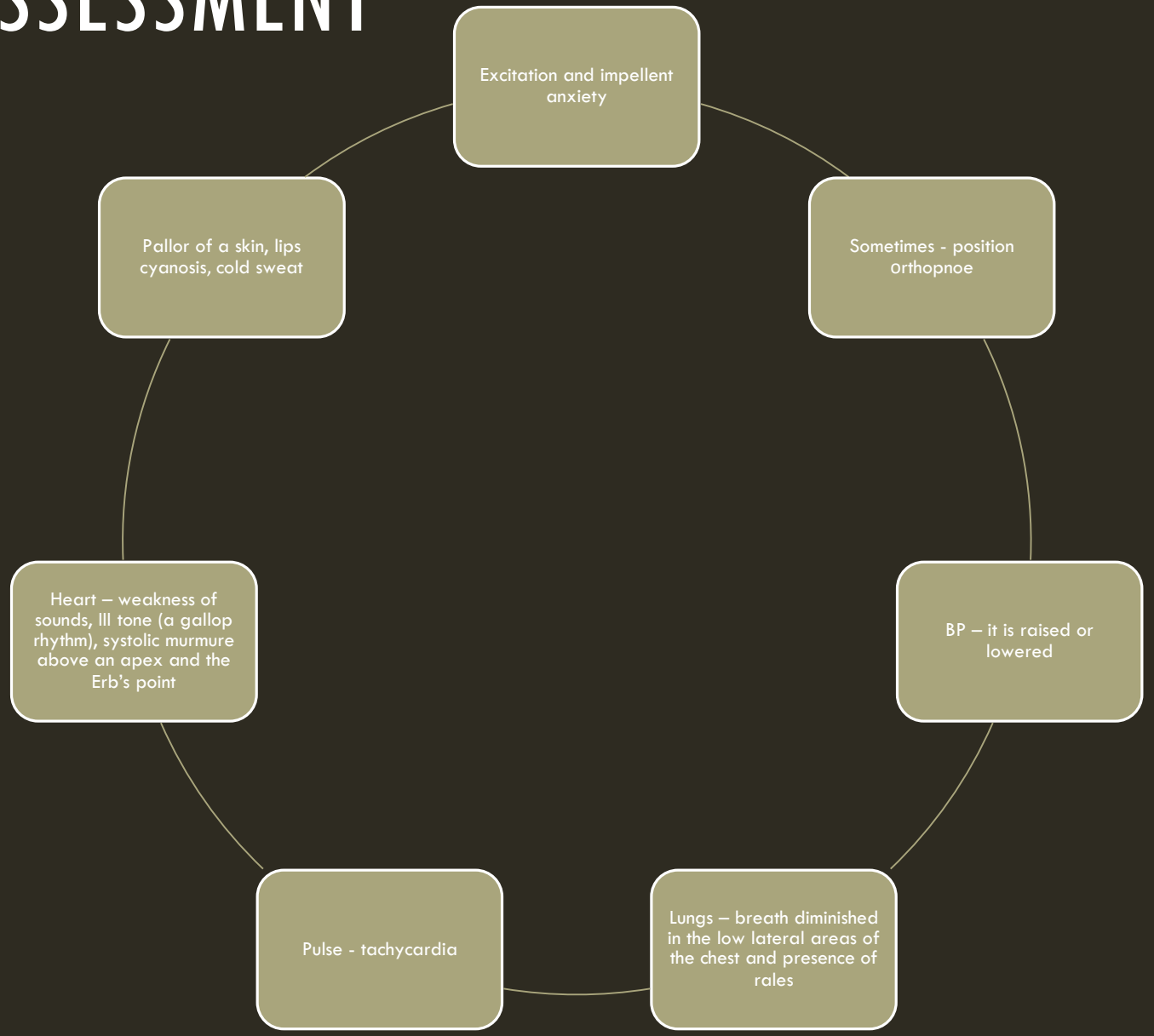
- de novo or progressive angina - is only observed in 20%

PHYSICAL ASSESSMENT

- The purpose of physical assessment of MI is not so much establishment of the diagnosis of MI (which basically proves to be true the data of laboratory and an analysis of electrocardiogram) how many an estimation of a condition of cardiovascular system and timely diagnostics of complications of MI.

- The purpose of the examination is to exclude the presence of non-cardiac causes of chest pain, the possibility of non-ischemic heart disorders (pericarditis, valvular diseases), potential favorable extracardiac factors, pneumothorax and, finally, signs of hemodynamic instability and left ventricular dysfunction (LV).

DATA OF ASSESSMENT



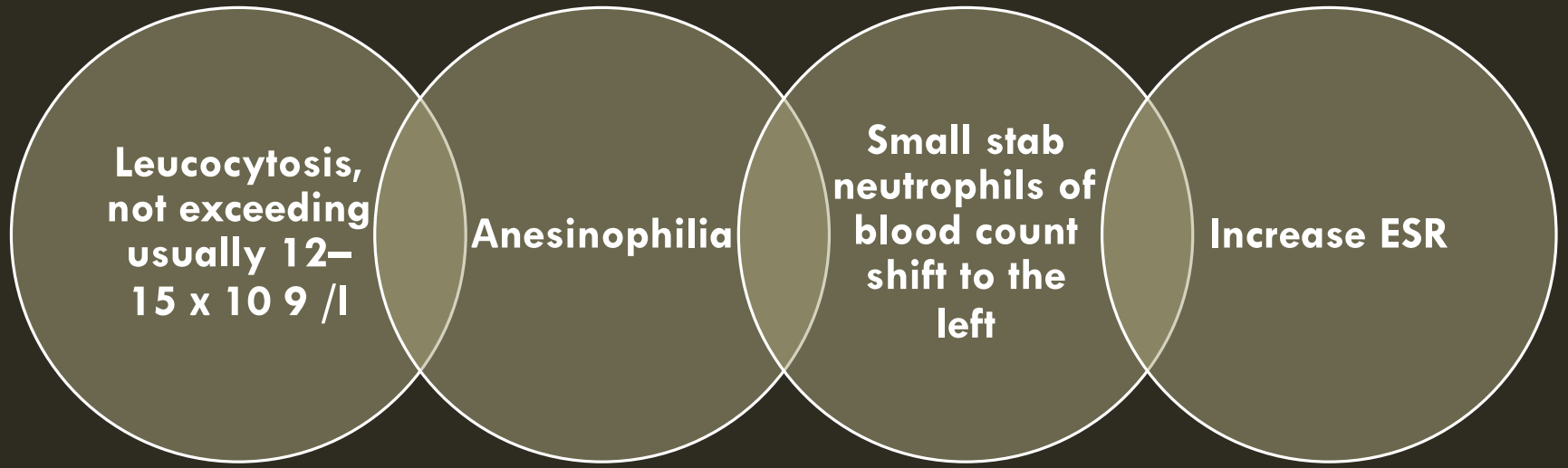
LABORATORY DIAGNOSTICS OF MI

Markers of necrosis of myocardium	Troponins I and T (2-4 hours after the beginning of anginous attack),
	CPhK (MB “myocardial band” fraction – in 4-6 hour)
	AST (in 12-14 hours), LDH1 (12-24)

DYNAMIC OF LABORATORY MARKERS OF MIOCARDIAL INFARCTION

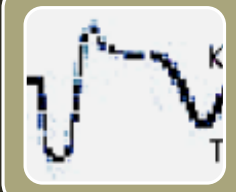
Markers	Norma	Time from onset of myocardial infarction		
		Baseline elevation hours	Peak elevation hours	Normalization days
Creatine kinase MB	0–4 ME/L	3–6	12–24	1.5–3
Lactate dehydrogenase	15–30 %	12–24	24–72	7–14
Aspartate aminotransferase	28–125 mmol/l	8–12	24–48	3–5
Troponin T, I	Less 0.1 mkg/l	3–12	12–48	3–16
Myoglobin	20–66 mkg/l	1–4	6–7	1

THE GENERAL ANALYSIS OF BLOOD – IS A FEW INFORMATIVE



ELECTROCARDIOGRAM DIAGNOSTICS — A BASIS — DIAGNOSE OF MI

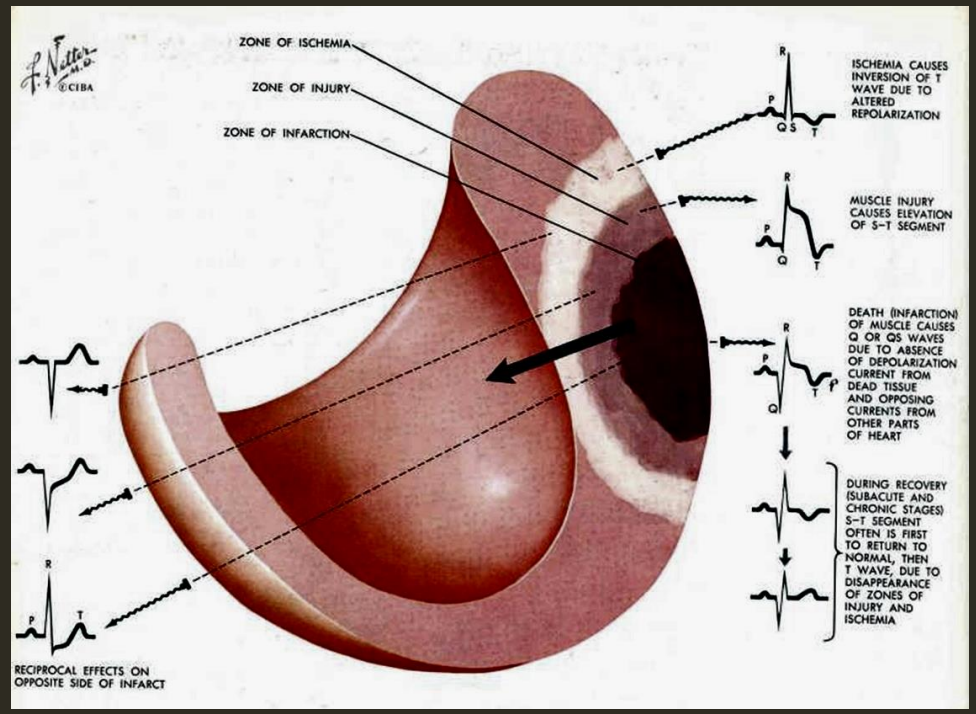
CHANGES IN THE ECG WITH MI



An ischemia Zone – coronary T

An injury Zone – elevation of segment ST

Pathological Q wave



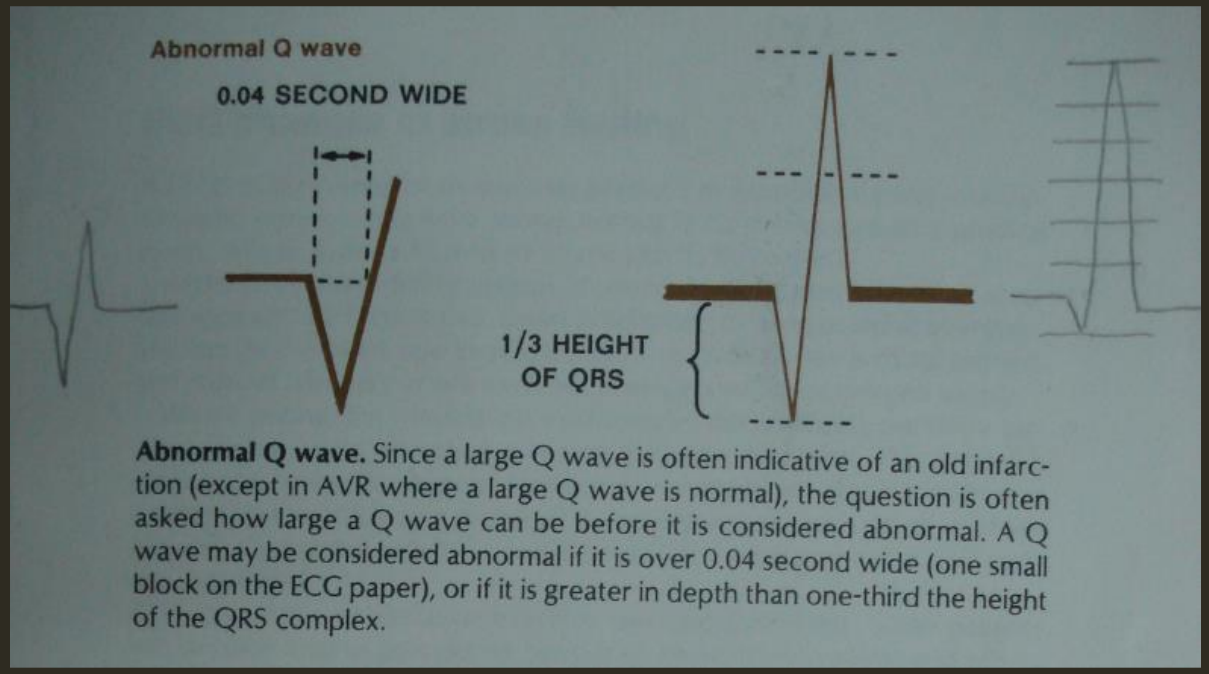
PATHOLOGICAL Q WAVE

Normal Q wave:

Depth of Q must be at I and II lead up to 15% of max. R at main leads

At III lead Q may touch to 60% of R, or R can be absent (wave QS) if horizontal EHA

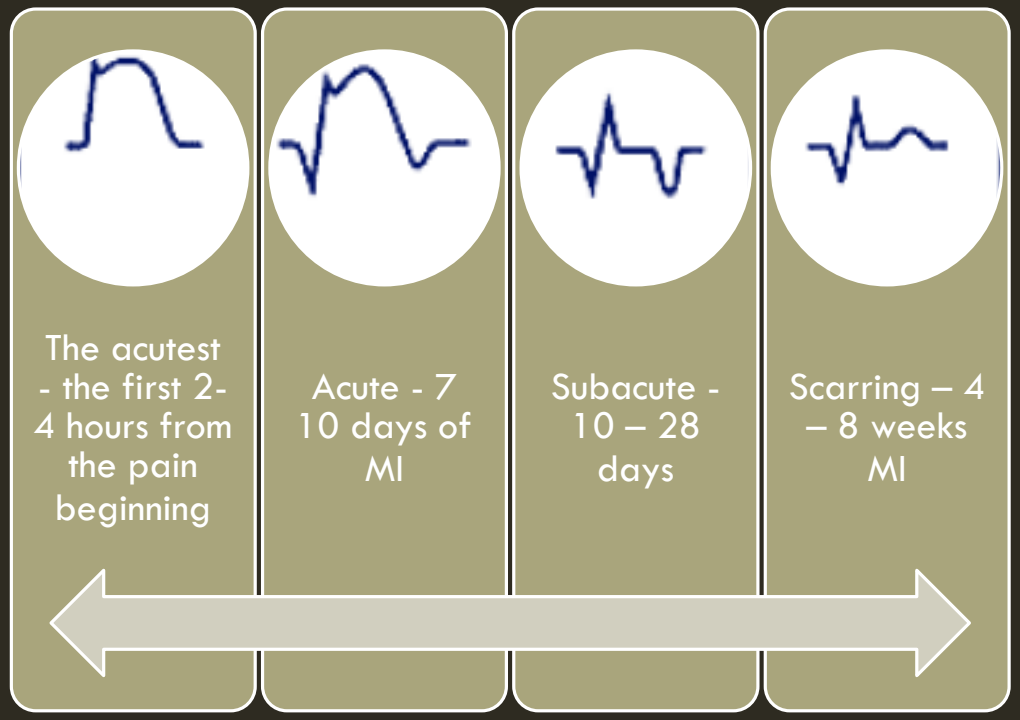
Q wave at V5-V6 usually pathological



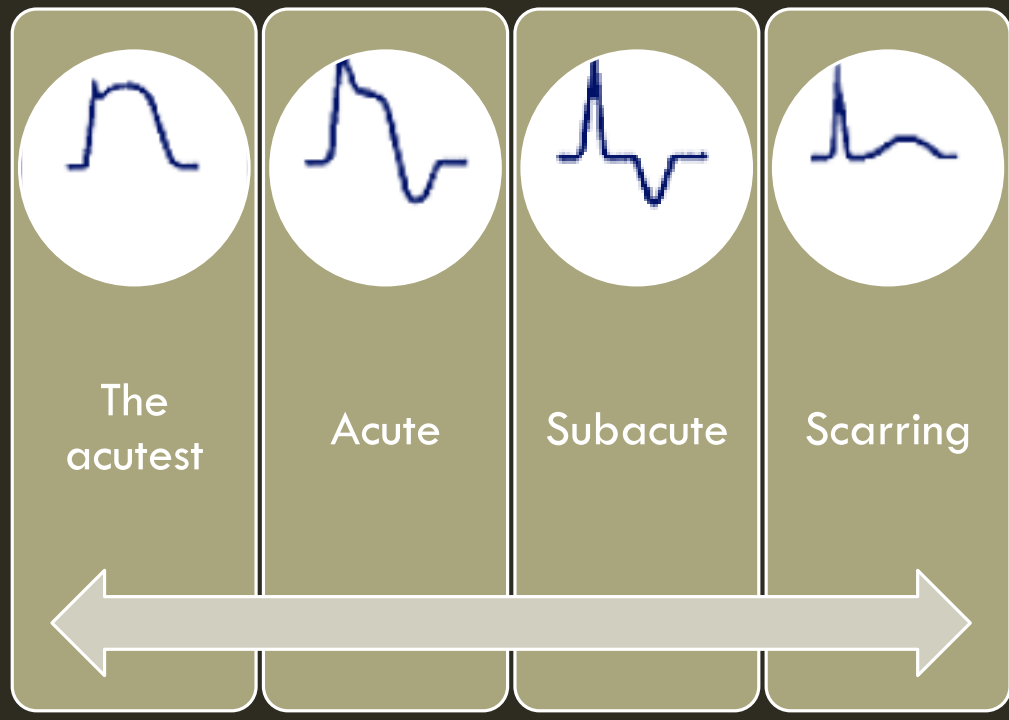
Abnormal Q wave. Since a large Q wave is often indicative of an old infarction (except in AVR where a large Q wave is normal), the question is often asked how large a Q wave can be before it is considered abnormal. A Q wave may be considered abnormal if it is over 0.04 second wide (one small block on the ECG paper), or if it is greater in depth than one-third the height of the QRS complex.

STAGES OF MI - DIAGNOSE ON AN ELECTROCARDIOGRAM

Transmural myocardial infarction

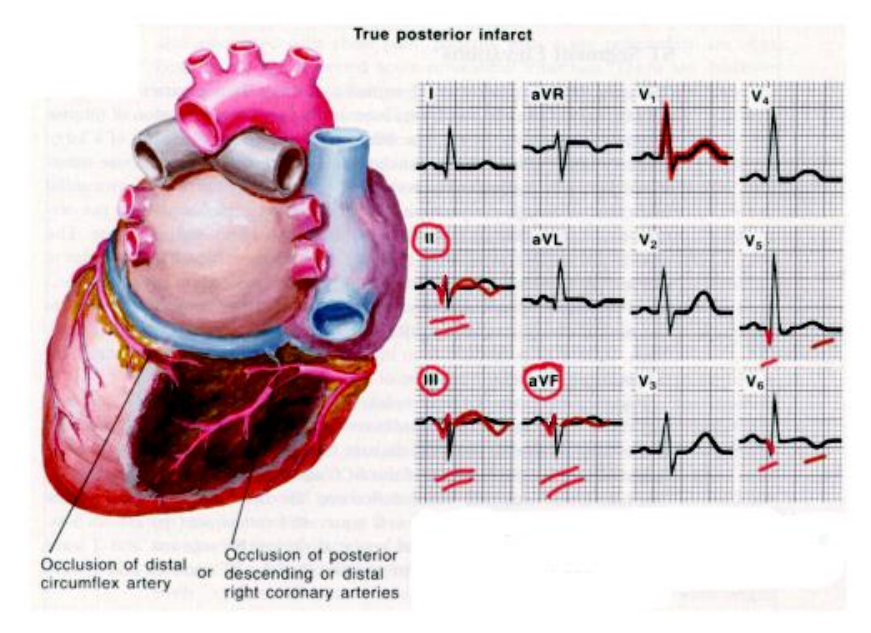


Subendocardial myocardial infarction

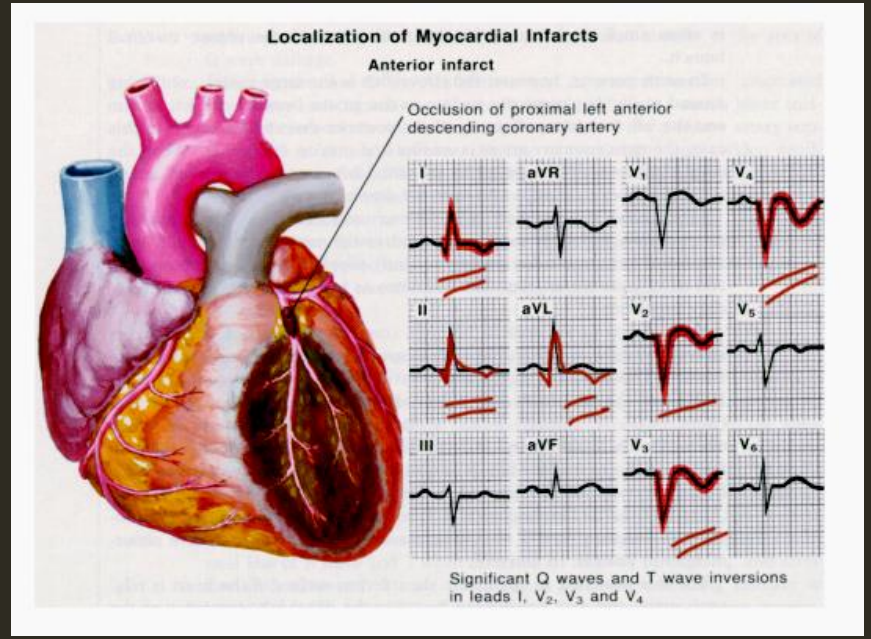


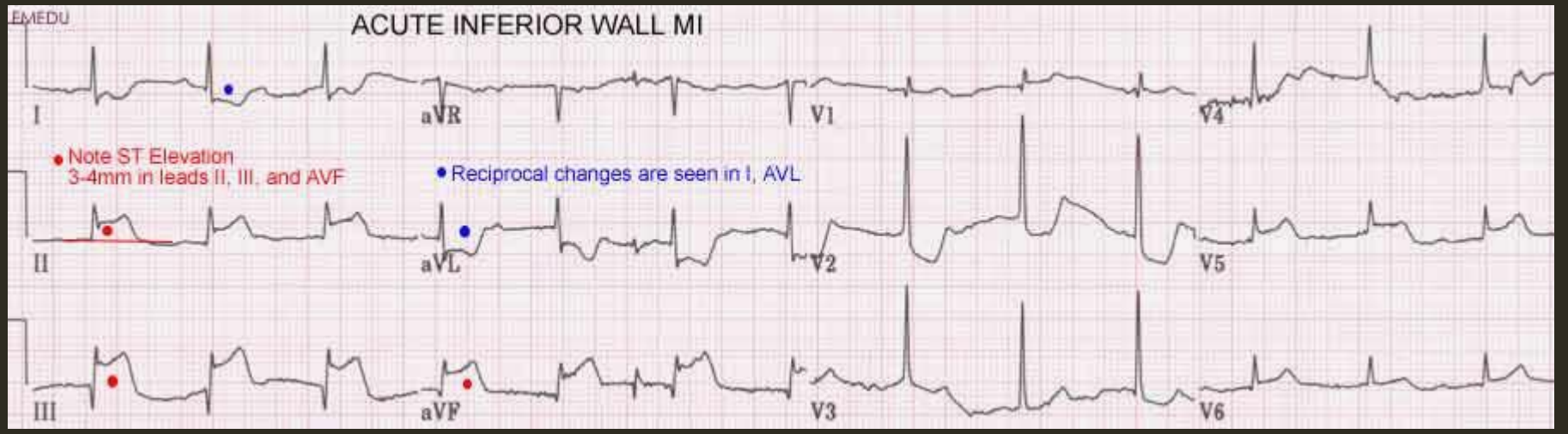
DIAGNOSE OF LOCALIZATION OF MI ON AN ELECTROCARDIOGRAM

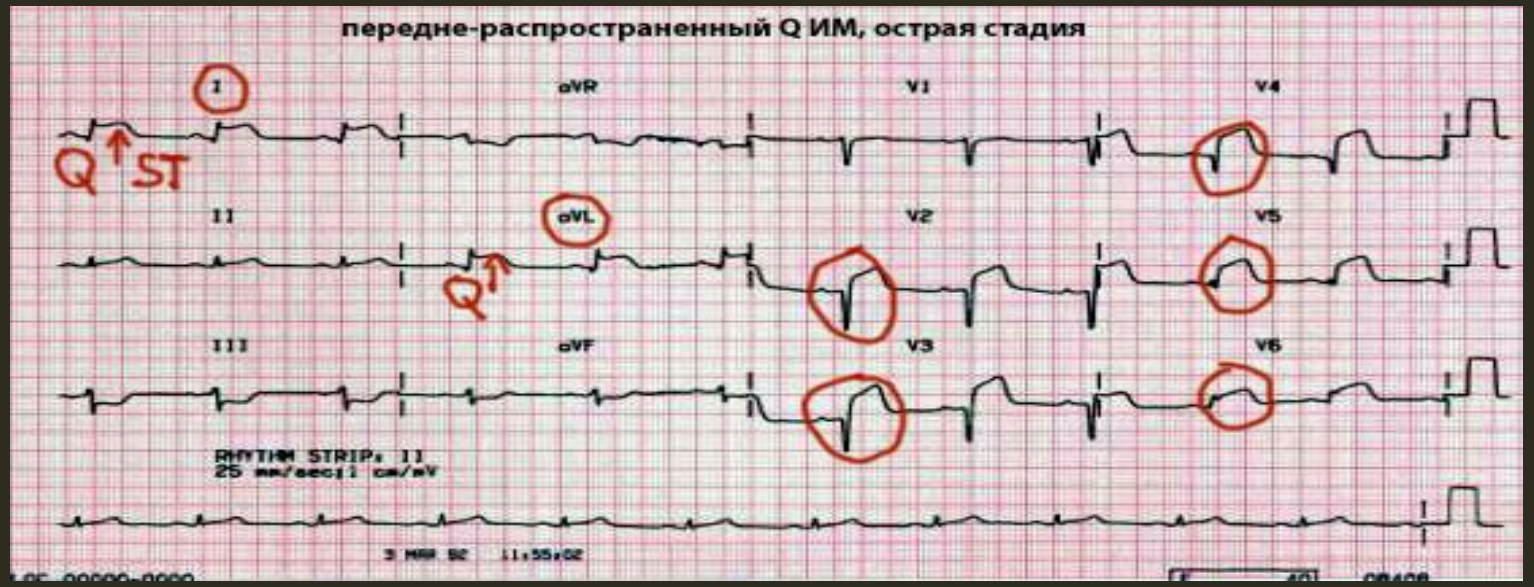
Posterior MI – pathological changes (Q, elevation of ST, coronary T) in leads II, III, aVF



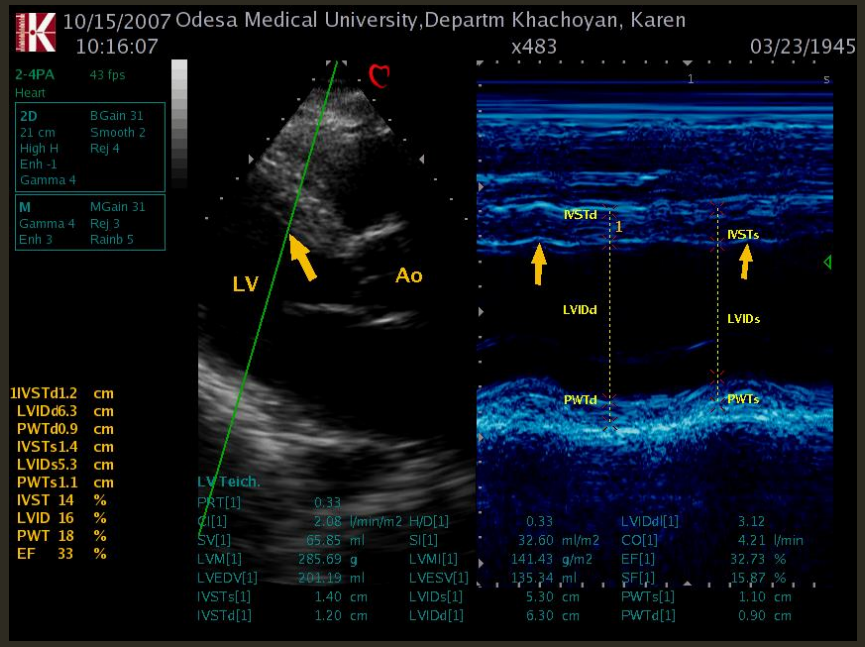
Anterior MI – pathological changes (Q, elevation of ST, coronary T) in leads I, II, aVL, chest (V2-6)

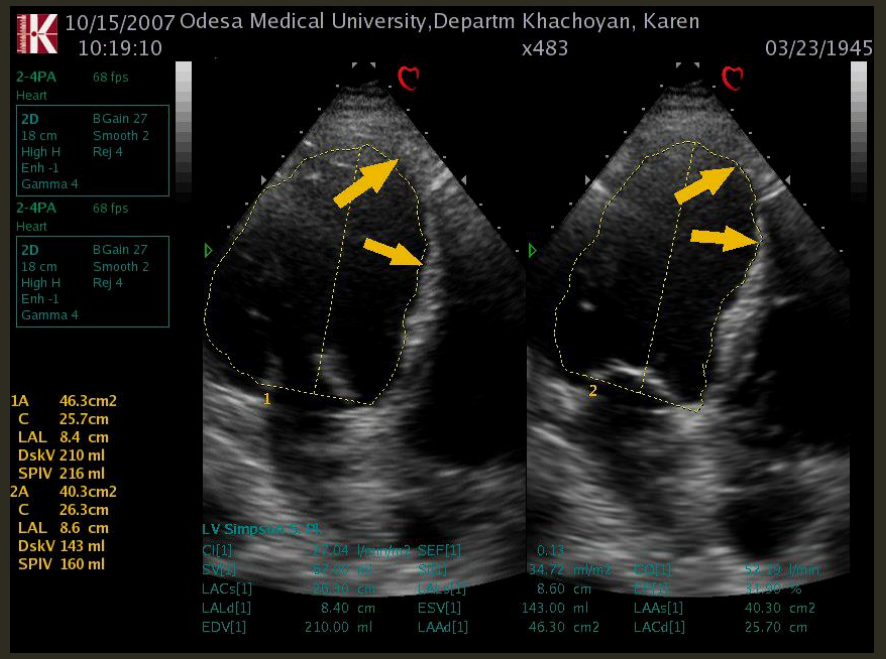



EMEDU
ACUTE INFERIOR WALL MI
I
aVR
V1
V4
Note ST Elevation
3-4mm in leads II, III, and AVF
Reciprocal changes are seen in I, AVL
II
aVL
V2
V5
III
aVF
V3
V6

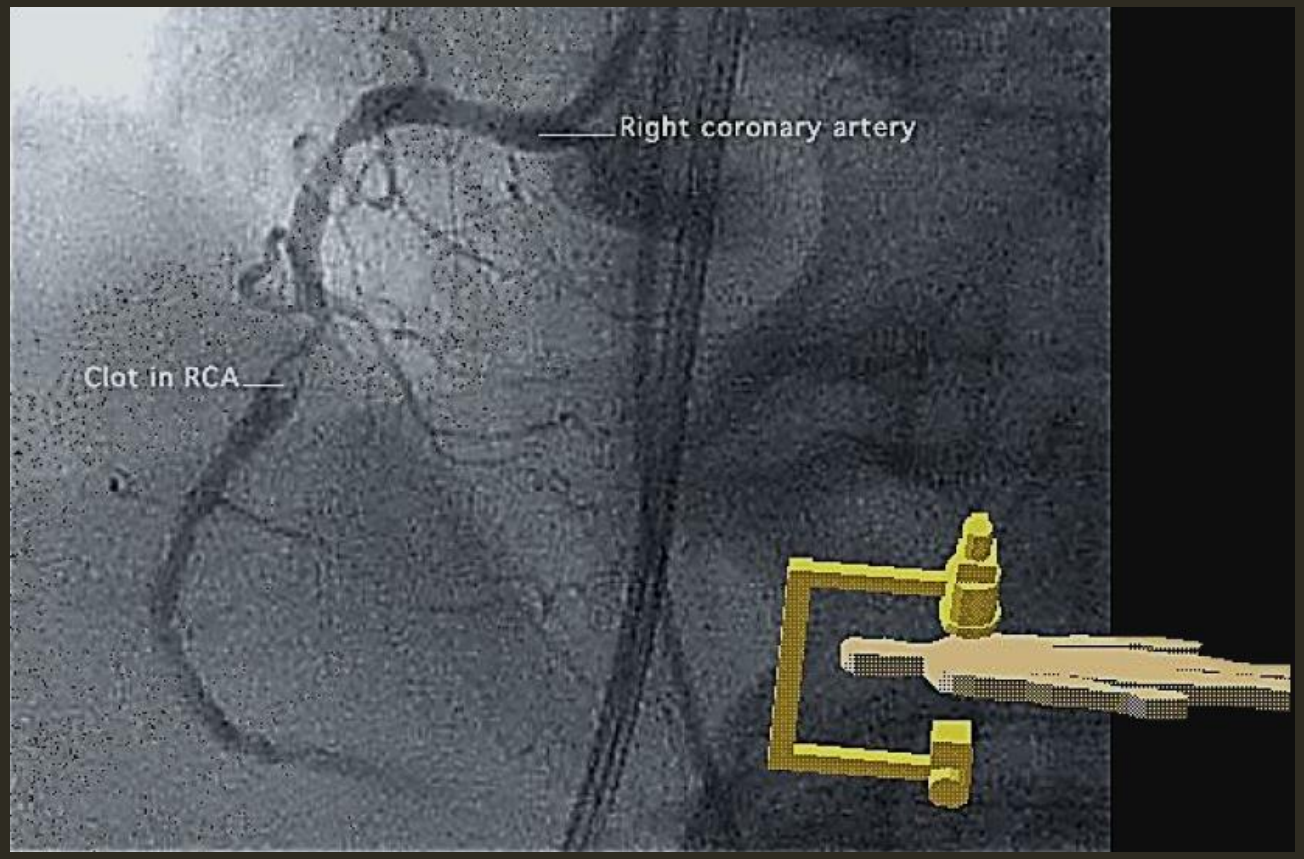

передне-распространенный Q ИМ, острая стадия
I
aVR
V1
V4
Q
ST
II
aVL
V2
V5
Q
III
aVF
V3
V6
RHYTHM STRIP: II
25 mm/sec; 1 cm/mV

OTHER METHODS OF DIAGNOSTICS - ECHOCG





ANGIOGRAPHY

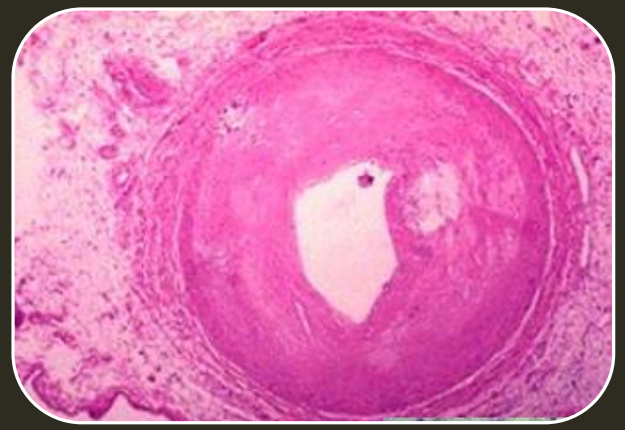


COMPLICATIONS OF MI

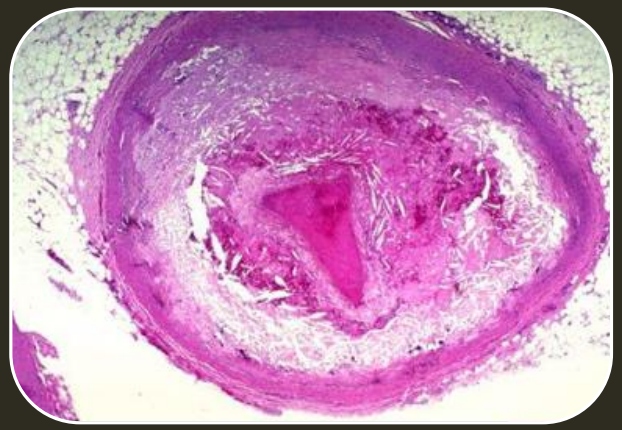
Rhythm disturbances	Heart failure (pulmonary edema)	Cardiogenic shock
LV aneurysm, myocardial rupture	Thromboembolic complications	Dressler's syndrome

TREATMENT PRINCIPLES FOR MI

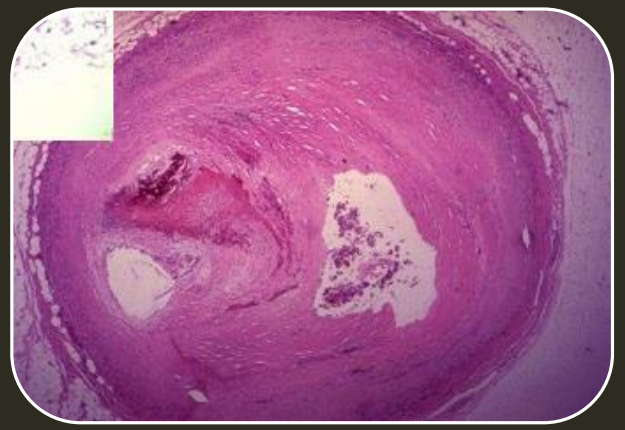
Early myocardial reperfusion	• in acute period of the disease (thrombolytics, antiplatelet agents, revascularization)
Limiting the zone of necrosis	• Limiting the zone of necrosis with hemodynamic unloading of the heart (nitrates, beta blockers, ACE inhibitors and etc.)
Prevention and treatment of complications	
Elimination of symptoms	• (pain, etc.)



Plaque in the lumen of coronary arteries



Artery thrombosis



Recanalization of arteries after thrombolytic therapy

THANK YOU FOR ATTENTION!