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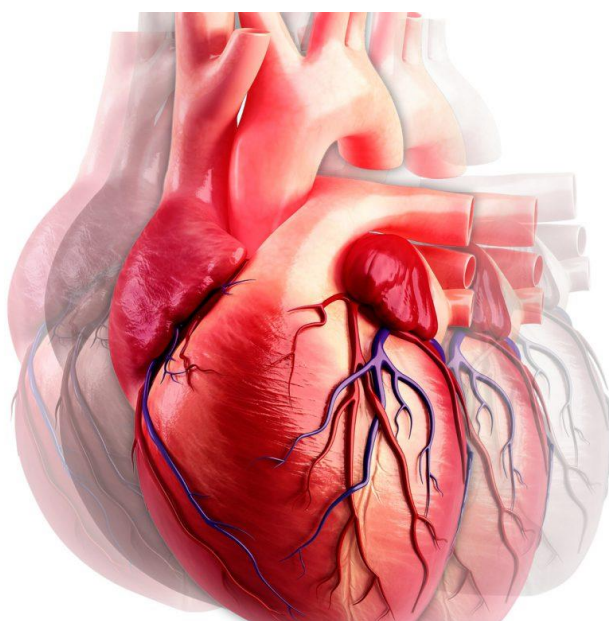
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**СТАБИЛЬНАЯ СТЕНОКАРДИЯ НАПРЯЖЕНИЯ:
ДИАГНОСТИКА, ЛЕЧЕНИЕ И ПРОФИЛАКТИКА
НА АМБУЛАТОРНОМ ЭТАПЕ**

Учебно-методическое пособие

**STABLE ANGINA PECTORIS: DIAGNOSIS, TREATMENT
AND PREVENTION AT THE OUTPATIENT STAGE**

Educational and methodical manual



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В учебно-методическом пособии подробно освещается тема «ИБС. Стабильная стенокардия напряжения», описаны этиологические факторы, патогенез, основные методы диагностики и лечения стабильной стенокардии напряжения с акцентом на амбулаторно-поликлинический этап ведения данной нозологии. В учебно-методическом пособии изложен теоретический материал в том числе по профилактике и особенностям диспансерного наблюдения пациентов со стабильной стенокардией напряжения, приводятся контрольные вопросы.

Пособие предназначено для иностранных студентов медицинских вузов.

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STABLE ANGINA PECTORIS

Stable angina pectoris is a term denoting discomfort or pain in the chest, having a pressing or compressive character and, as a rule, typical localization (behind the sternum) and irradiation (in the left arm, left half of the neck and lower jaw, left shoulder blade), relieved at rest or after taking nitrates, corresponding myocardial ischemia is associated with coronary heart disease.

The ICD-10 code is I20.8

EPIDEMIOLOGY

Prevalence: Stable angina is a common disease in Russia, especially among the elderly. According to the National Population Health Survey of Russia conducted in 2019, approximately 5.2% of men and 3.3% of women suffered from stable angina. Only half of the patients are aware of the presence of the disease and, accordingly, receive treatment. In this regard, the problem of prevention and early diagnosis of angina pectoris is very important for primary care physicians, not only for therapists and cardiologists, but also for other specialists.

ETIOLOGY

The supply of oxygen to the myocardium is directly related to the state of coronary blood flow. Stable angina, as a rule, is a consequence of atherosclerotic lesions of the coronary arteries, the mechanism of development of which is largely determined by the influence of risk factors. Identification and correction of risk factors for cardiovascular diseases (CVD) is an essential component of the work of a primary care physician, regardless of the presence or absence of clinical manifestations of coronary heart disease in patients, since they can contribute not only to the development, but also to the progression of the disease, which significantly worsens the prognosis. There are a number of risk factors for CVD.

- **Age.** The likelihood of CVD increases in men over 45 years of age and women over 55 years of age.

- **Gender.** Men have a higher predisposition to CVD than women.

- **Smoking.** Regardless of the number of cigarettes the patient smokes, the doctor should strongly recommend that the patient stop smoking.

- **Arterial hypertension.** According to numerous domestic and foreign studies, normalization of blood pressure significantly reduces the risk of developing CVD.

- **Nutrition factor and lipid metabolism disorders.** Excessive consumption of animal fats, salt, and dietary cholesterol can contribute to the development of atherosclerosis. The detection of dyslipidemia, especially in young people, will undoubtedly contribute to the earlier prevention of cardiovascular complications.

- **Diabetes mellitus and impaired glucose tolerance.** Patients suffering from diabetes mellitus, regardless of its type, are classified as having a high 10-year risk of death from CVD.

- **Overweight.** The presence of abdominal obesity: a waist circumference of more than 102 cm in men and more than 88 cm in women is associated with an increased incidence of cardiovascular complications. To assess the optimal body weight, it is very important to monitor the body mass index (BMI): weight (kg)/height (m²). Normally, the BMI is 18-25 kg/m².

- **Low physical activity.**

- **Alcohol abuse.**

- **Increased heart rate (HR).** According to the data obtained during the BEAUTIFUL-2008 study, an increase in heart rate of more than 70 beats per minute in patients with coronary heart disease contributes to an

increased risk of myocardial infarction and other cardiovascular complications.

- **Heredity** - early development of coronary heart disease in close relatives (up to 55 years in men, up to 65 years in women).

- **In women, the development of premature menopause, as well as long-term use of hormonal contraceptives**, can lead to the development of coronary insufficiency.

In recent years, much attention has been paid to the study of such risk factors for the development of coronary heart disease and its complications as psychosocial stress, increased levels of C-reactive protein and fibrinogen, hyperhomocysteinemia, disorders of the hemostatic system, endothelial dysfunction, changes in the ankle-shoulder index. Anginal symptoms are considered stable if they manifest for more than 6 weeks without significant deterioration of the patient's condition. Usually, anginal symptoms appear in conditions associated with an increase in myocardial oxygen demand with limited oxygen delivery due to atherosclerotic narrowing of the coronary artery. The level of stress that causes an angina attack is an important criterion in determining the severity of coronary heart disease. In some cases, the cause of clinical manifestations of angina pectoris are coronary spasm and endothelial dysfunction.

Patients with Stable angina pectoris are divided into functional classes (FC) depending on the tolerance of physical activity and taking into account the classification of the Canadian Association of Cardiology.

PATHOGENESIS

Three main mechanisms are involved in the formation of angina pectoris:

- Atherosclerotic lesion of the coronary arteries, which makes it impossible to meet the increased needs of the myocardium (fixed coronary obstruction - stable angina pectoris).

- Transient vascular thrombosis accompanied by the formation of platelet aggregates (unstable angina pectoris).

- Decrease in coronary blood flow due to spasm or increased tone of the coronary arteries (dynamic obstruction).

The morphological substrate of angina pectoris is the formation of atherosclerotic narrowing of the coronary arteries (in 95% of patients). Angina pectoris appears during physical exertion or stressful situations, in the presence of narrowing of the lumen of the coronary artery by at least 50-70%.

CLASSIFICATION

The classification of the severity of angina pectoris was proposed by the Canadian Association of Cardiologists in 1976. There are four Functional classes of Stable angina pectoris.

- **Class I** - regular physical activity does not cause an attack of angina pectoris. An angina attack develops as a result of very intense, rapid or prolonged physical activity.

- **Class II** - a slight restriction on daily activity. An angina attack occurs when walking or climbing stairs quickly, walking or climbing stairs after eating, in cold or windy weather, with emotional stress or only for several hours after waking up, walking more than 200 m (two blocks) on flat terrain and climbing more than one flight of ordinary steps on average at a pace and under normal conditions.

- **Class III** - severe restriction of daily activity. An attack of angina pectoris occurs when walking at a normal pace on flat terrain at a distance of 100-200 m (one or two blocks) or climbing one flight.

- **Class IV** - inability to perform any physical activity without discomfort. Angina pectoris can occur at rest.

THE CLINICAL PICTURE

Angina pectoris is characterized by compressive, burning, pressing paroxysmal (2-3 minutes, no more than 20 minutes) pain behind the sternum or to the left of the sternum, which occurs during physical exertion and passes at rest or immediately after taking nitroglycerin.

Attacks of pain occur most often at the moment of physical or emotional stress, accompanied by vegetative disorders, a feeling of "fear of death", stiffness of the patient, a feeling of suffocation. Sometimes patients experience pain not in the heart, but in the places of its irradiation (shoulder, lower jaw, interscapular region). Some patients may experience paroxysmal shortness of breath, which is considered the equivalent of pain.

In addition, it should be remembered that there are a number of conditions that can exacerbate myocardial ischemia and, accordingly, pain. They are divided into:

- **non-cardiac** - arterial hypertension, disorders of rheological and coagulating properties of blood, chronic lung diseases, fever, thyrotoxicosis;
- **cardiac** - congenital and acquired heart defects, tachycardia, left ventricular dysfunction.

Upon examination of the patient, the general condition is usually satisfactory. There may be signs of impaired lipid metabolism (xanthomas, xanthelasmas, marginal corneal opacity in the form of "older arcs"), as well as signs of atherosclerosis of the peripheral arteries. On palpation, a slight left displacement of the apical (left ventricular) thrust can be detected, as well as pathological pulsation in the precordial region, which indicates the presence of a heart aneurysm. A slight displacement (within 0.5-1 cm) of the left border of relative cardiac dullness, a weakening of the 1st tone in the apex zone and an accent of the 2nd tone in the aortic zone are often detected; the 3rd or 4th cardiac tones, systolic murmur at the apex of the heart can be heard.

DIAGNOSTICS

The clinical diagnosis is based on:

- data obtained from a detailed patient survey;
- a thorough study of the anamnesis.

All other research methods are used to confirm or exclude the diagnosis, clarify the severity of the disease and prognosis.

When studying the medical history, life history, and family history, it is necessary to carefully evaluate all the risk factors for developing CVD available to the patient and listed above, which greatly optimizes further tactics for the diagnosis, treatment, and prevention of CVD.

A mandatory laboratory test is a general blood test (CBC); it allows you to exclude anemia and erythrocytosis. Additional laboratory tests:

- determination of blood glucose levels in case of suspected diabetes mellitus;
- conducting a glucose-loaded sample in case of impaired glucose tolerance;
- determination of cholesterol levels;
- determination of the level of low and high density lipoproteins, triglycerides;
- determination of the level of aspartate aminotransferase (AST) and alanine aminotransferase (ALT).

Instrumental diagnosis of Stable angina pectoris includes electrocardiography (ECG), echocardiography (EchoCG), daily ECG monitoring, ECG under load - bicycle ergometry, coronary angiography. All studies, except ECG, are classified as additional; they also include examinations by a cardiologist and a cardiac surgeon. Bicycle ErgoMetry is performed only in patients with Stable angina pectoris I and II FC. If it is impossible to

perform bicycle ergometry or if it is uninformative, it is possible to perform transesophageal atrial electrical stimulation. In addition, if appropriate indications are available, an outpatient cardiologist can refer an outpatient patient to an appropriate center for stress imaging studies, computed tomography or positron emission tomography of the myocardium.

Electrocardiography (ECG)

In all patients with suspected angina pectoris, an ECG in 12 leads should be recorded at rest outside the pain attack and at the moment of pain. Normal ECG results at rest are not uncommon even in patients with very severe angina pectoris. However, on an ECG at rest, such signs of coronary heart disease can be detected as a past myocardial infarction, hypertrophy of the left ventricle, conduction system disorders and cardiac arrhythmias.

Load test (Stress test)

A stress test in the practice of a polyclinic doctor is the most common research method, as it allows you to clarify the diagnosis of coronary heart disease, determine treatment tactics, its effectiveness and prognosis assessment, including the need to refer a patient to medical-social Expert Commission. The test results are considered positive if a horizontal or obliquely downward decrease in the ST segment by 0.1 mV is registered in any lead, or if a characteristic attack of angina pectoris occurs.

Absolute contraindications to the test:

- acute stage of a heart attack (within 7 days of its onset);
- unstable angina pectoris;
- acute cerebrovascular accident;
- acute thrombophlebitis;
- pulmonary embolism;

- severe heart failure III-IV FC (according to the classification of the New York Cardiological Association);
- severe pulmonary insufficiency;
- fever.

This test should not be performed in the presence of tachyarrhythmia, complete LBBB, high degree of sinoatrial and atrioventricular blockages, diseases of the musculoskeletal system, obliterating lesions of the arteries of the lower extremities.

According to the results of the stress test, FC can be determined in patients with coronary heart disease (Table 1).

Table 1

Functional class of coronary heart disease according to the results of a stress test

The results of the stress test	Functional classes			
	I	II	III	IV
HR*SBP/100 HR – heart rate, SBP – systolic blood pressure	≥ 278	218-277	151-217	≤ 150
Power of the last stage of the load, W	>125	75-100	50	25
Maximum oxygen consumption, ME (metabolic equivalent) 1 ME = 3.5 ml O ₂ /min per 1 kg of body weight	≥ 7	4,0-6,9	2,0-3,9	<2

HOLTER-ECG

Daily monitoring is necessary to detect changes in the ECG during episodes of chest pain during daily exercise and to diagnose pain-free myocardial ischemia. A decrease in the ST segment with a total duration of 60

minutes or more is one of the indications for surgical treatment of angina pectoris (Society of Specialists in Emergency Cardiology, 2013). 24 hour ECG monitoring helps to evaluate the effectiveness of treatment, identify heart rhythm and conduction disorders, and vasospastic angina pectoris.

ECHOCARDIOGRAPHY

Echocardiography at rest is of great importance in the differential diagnosis of non-coronary chest pain that occurs with aortic valve stenosis and hypertrophic cardiomyopathy. This study makes it possible to analyze both the morphology and function of cardiac structures, and to stratify the risk of patients with heart failure. If latent myocardial ischemia is suspected, the polyclinic doctor may refer the patient to a specialized center for echocardiography during physical activity (stress echocardiography). If it is impossible to carry out tests with physical activity, pharmacological tests with dobutamine, methylergomethrin, dipyridamole, adenosine are used. Most often they are carried out in a hospital setting.

CORONAROGRAPHY

This method occupies a central place in the management of patients with Stable Angina, as it allows you to assess the degree and type of damage to the coronary arteries, and choose the optimal management tactics for the patient. Currently, hemodynamically significant is a narrowing of the vessel lumen by more than 50%, which, as a rule, already has clinical manifestations. Indications for coronary angiography are determined by a cardiac surgeon. Coronarography is performed only in a hospital setting.

FEATURES OF DIAGNOSIS OF STABLE ANGINA PECTORIS AT THE OUTPATIENT STAGE

When contacting a doctor, young people should pay special attention to identifying such risk factors as familial hereditary hyperlipidemia, a history of CVD, diabetes mellitus, metabolic syndrome, arterial hypertension, smoking, congenital heart disease and cardiomyopathy.

When diagnosing angina pectoris in elderly patients, it is necessary to take into account the possibility of atypical manifestations of Stable Angina pectoris against the background of a polymorbid condition, carefully conduct a clinical examination, use methods such as daily monitoring, echocardiography, transesophageal atrial electrical stimulation, pharmacological tests in the diagnosis of Stable Angina pectoris. There are no absolute contraindications to coronary angiography in the elderly. The frequency of side effects when prescribing medications in elderly patients increases significantly, therefore, the polyclinic doctor should be more careful about the choice of tactics for their treatment.

Despite the fact that, on average, the incidence of coronary heart disease in men is higher in the population than in women, the possibility of developing CVD in the latter, especially in the postmenopausal period, cannot be underestimated. This is due to a higher incidence of atypical CVD symptoms and a large number of concomitant diseases over the age of 45-50 years, especially when combined with diabetes mellitus and Arterial hypertension, as well as with an increase in the number of cardiovascular complications that occur during this period of life.

The development of CVD is also significantly influenced by such independent risk factors as diabetes mellitus and hypertension. It should be borne in mind that in diabetes mellitus, pain may be absent, and normalization of blood pressure contributes to a significant reduction in mortality from CVD.

DIFFERENTIAL DIAGNOSIS

Recognition of angina pectoris is a reliable way to diagnose coronary heart disease. Some doctors neglect detailed questioning, preferring complex instrumental studies. However, only qualified questioning of the patient and the collection of anamnesis in 80% of cases makes it possible to correctly diagnose coronary heart disease. Table 2 presents a clinical algorithm for classifying chest pain (Diamond A.I., 1983). Relatively often,

angina pectoris is the only clinical sign of the disease, when neither examination nor examination can reveal deviations from the norm.

Table 2

Clinical algorithm for classifying heart pain

Typical angina pectoris (defined)	It corresponds to all three presented characteristics:
	• discomfort behind the sternum of characteristic quality and duration;
	• occurs during physical exertion or emotional stress;
	• passes at rest and/or a few minutes after taking nitrates
Atypical angina pectoris (probable)	Corresponds to two of these characteristics
Non-anginal pain in the chest	Corresponds to only one of these characteristics or does not correspond at all

Table 3 presents the main diseases and conditions that can cause chest pain.

Angina pectoris as a form of coronary heart disease should be differentiated from angina pectoris as a syndrome of other diseases that are nosologically unrelated to coronary heart disease. We are talking about nodular periarteritis, rheumatism, sepsis, syphilis, etc. In these diseases, coronary artery damage is not associated with atherosclerosis. Let's briefly focus on the most common diseases in outpatient practice.

Table 3

Causes of chest pain (Russian recommendations of experts of the All-Russian Scientific Society of Cardiology, 2009)

Cardiovascular ischemic (non- coronary)	Pulmonary	Gastrointestinal	Mental	Other
<ul style="list-style-type: none"> • Delaminating aortic aneurysm; • pericarditis; • cardiomyopathy; • aortic stenosis; • pulmonary embolism; • myocarditis; • mitral valve prolapse 	<ul style="list-style-type: none"> • Pleurisy; • pneumothorax; • pneumonia; • lung cancer 	<p>Diseases of the esophagus:</p> <ul style="list-style-type: none"> • esophagitis; • esophageal spasm; • hernia of the esophageal orifice of the diaphragm; • cancer of the esophagus. <p>Gastrointestinal and biliary diseases:</p> <ul style="list-style-type: none"> • cardiac gastric cancer; • gastric ulcer; • intestinal colic; • cholecystitis; • pancreatitis; • biliary colic 	<p>States of anxiety:</p> <ul style="list-style-type: none"> • somatoform autonomic dysfunction; • hyperventilation; • panic disorders; • primary phobia; • psychogenic cardialgia. <p>Affective states:</p> <ul style="list-style-type: none"> • depression 	<p>Chest:</p> <ul style="list-style-type: none"> • osteochondrosis of the thoracic spine; • fibrosis; • injury to the ribs and sternum; • sternoclavicular arthritis; • intercostal neuralgia; • shingles ("before the rash stage")

Chest pain of myocardial origin can occur with mitral valve prolapse, mitral heart disease, hypertrophic cardiomyopathy, myocarditis, pathological sports heart, alcoholic myocardial damage and a number of other diseases.

Mitral valve prolapse is one of the most common valvular heart defects, occurring, as a rule, at a younger age, more often in women. Chest pain is not typical for myocardial ischemia, it is often stabbing, prolonged and not associated with physical activity. Asthenoneurotic syndrome is often inherent in such patients. A mesosystolic click, heard best at the apex, is a classic symptom of mitral valve prolapse.

Systolic murmur follows a click if mitral regurgitation occurs. Echocardiography is crucial in the diagnosis of mitral valve prolapse.

Pain in the heart area is a common occurrence in people suffering from **alcoholism**. The ECG reveals nonspecific changes in the ST segment and the T wave. Radiography reveals the expansion of the boundaries of the heart. With echocardiography, dilation of the left ventricle is established. The diagnosis of alcoholic cardiomyopathy is facilitated with simultaneous signs of liver damage, and vegetative disorders often occur.

Pain syndrome in the heart area with myocarditis can last for hours, days. When diagnosing, it is necessary to take into account the connection with the infection, an increase in body temperature, leukocytosis; possible damage to muscles, joints and lymph nodes. With prolonged course, rhythm and conduction disorders, cardiomegaly and heart failure develop. On the ECG, changes in the amplitude of the R wave, depression of the ST segment and inversion of the T wave are most often noted.

Chest pain in **pericarditis** resembles angina pectoris, but lasts from a day or more, is not stopped by nitroglycerin, increases with breathing, weakens in a sitting position and increases in a supine position. On examination, it is possible to detect pericardial friction noise, cyanosis, swelling of the cervical veins, an increase in the boundaries of the heart, a weakening of tones; on an ECG, an elevation of the ST segment in all leads, an inversion of the T wave in leads V1-V3. Echocardiography data is of great importance.

In the differential diagnosis of angina pectoris with valvular heart defects or cardiomyopathies, clinical data and echocardiography are of leading importance.

Heart pain of psychogenic origin occurs with somatoform autonomic dysfunction. One of the varieties of this dysfunction can be considered dishormonal cardiomyopathy, which occurs in women in the premenopausal and menopausal periods.

Dishormonal cardiomyopathy in some cases occurs in young women, as a rule, with gynecological pathology (more often with uterine fibroids). Heart pain resembles somatoform autonomic dysfunction. It is not associated with physical exertion, but neuropsychiatric stress clearly causes or increases pain. Many patients experience hot flashes to the head, numbness of the fingers, and a feeling of lack of air. On an ECG, flattening or inversion of the T teeth is often noticeable in many leads, more often in the thoracic ones, which indicates the diffusivity of the process in the myocardium. The test with bicycle ergometry is negative or is not brought up to diagnostic criteria.

Cardialgic syndrome in somatoform autonomic dysfunction is long-lasting and persistent. The pain is stabbing or aching, localized in the region of the apex of the heart, relieved by taking levomentol solution in mentholizalate (Validol) or Valocordin, sedatives. In addition to cardiac syndrome, patients are diagnosed with tachycardial, neurotic, vegetative-dystonic, asthenic syndromes and respiratory disorders syndrome. The test with bicycle ergometry in most patients is negative.

Angina pectoris must be differentiated from a variety of non-cardiac diseases: sternocarbon arthritis (Titze syndrome), shingles, sliding rib syndrome and very frequent cervical-thoracic radiculitis associated with osteochondrosis of the cervical and thoracic spine, as well as various lesions of ribs and cartilage, myositis and traumatic chest lesions.

Vertebrogenic cardialgia is a rather intense prolonged pain behind the sternum and in the left half of the chest. The pain is associated with the position of the body (there are no short bouts of pain during physical exertion), increases with palpation of the spine, intercostal spaces and shoulder girdle muscles, weakens under the influence of anti-inflammatory and analgesic drugs. There is no effect from the use of nitroglycerin. However, the presence of pronounced symptoms of cervical-thoracic osteochondrosis with the phenomena of radiculalgia in no case makes it possible to exclude coronary heart disease. To exclude coronary heart disease, it is necessary to conduct a full range of diagnostic studies, regardless of the severity of vertebrogenic pathology. Radiological signs and computed tomography (CT) data provide some assistance in diagnosis.

Chest pain can be caused by diseases of the gastrointestinal tract, especially the esophagus. Esophagitis, cardiospasm, diverticula, and esophageal tumors can cause pain behind the sternum. A common cause of cardialgia is a hernia of the esophageal orifice of the diaphragm, less often - peptic ulcer, stomach tumor, diseases of the gallbladder, pancreas, intestines. Often, to confirm these diseases, it is necessary to resort to the help of instrumental research methods: ultrasound, endoscopic and radiological, including CT.

In case of hernia of the esophageal orifice of the diaphragm, one of the causes of pain is concomitant **peptic esophagitis**. The pain is localized in the epigastric region or above the lower third of the sternum, often accompanied by a burning sensation, increased salivation. Pain from irritation of the diaphragmatic nerve increases due to eating, the transition from a vertical position to a horizontal one. The diagnosis is assisted by an X-ray examination in the patient's lying position or with the head end of the couch lowered.

Differential diagnosis of angina pectoris requires the exclusion of lung and pleural diseases. Chest pain may be a manifestation of thromboembolism of the branches of the pulmonary artery, as well as a sign of

pneumothorax, pneumomediastinum or pleurisy. Chest pain occurs in bronchial asthma, chronic bronchitis and primary pulmonary hypertension. In such situations, X-ray examinations, angiography, bronchoscopic examination with biopsy, CT are highly informative research methods.

EXAMPLES OF DIAGNOSIS FORMULATION

1. Coronary heart disease. Stable angina pectoris, FC III. Postinfarction cardiosclerosis (date). Left Bundle Branch Block. Chronic heart failure (CHF) with a reduced ejection fraction (35%), stage II B, FC IV.

2. Coronary heart disease. Stable angina pectoris, FC II. CHF with preserved ejection fraction (61%), stage I, FC I.

TREATMENT

Treatment of angina pectoris has two goals:

- improving the quality of life of patients;
- increasing the life expectancy of patients.

Non-drug treatment

First of all, a lifestyle change is necessary:

- reduction of body weight in case of obesity in order to achieve optimal BMI;
- giving up smoking and alcohol abuse;
- rational nutrition;
- regular physical activity.

Medical treatment

Drugs that improve the prognosis.

Antiplatelet drugs (antiplatelet agents) should be taken constantly. It is necessary to take into account their ulcerogenic effect.

- acetylsalicylic acid 75-150 mg/day.

• clopidogrel at 75 mg / day; take no more than a year. Patients after coronary angioplasty are recommended to combine with acetylsalicylic acid.

Hypolipidemic agents are prescribed if diet and lifestyle changes do not lead to the target values of blood lipids that are desirable for patients with stable angina pectoris, or when ultrasound examination (ultrasound) reveals atherosclerotic plaques in the vessels. Statins are prescribed for continuous use in hyperlipoproteinemia types IIa, IIb, III.

• atorvastatin 10-20 mg / day, in the absence of effect - 40 mg / day, with aggressive therapy - 80 mg / day;

- rosuvastatin 5-40 mg / day.

To control the side effects of these drugs, the levels of AST, ALT and creatine kinase in the blood are examined. With an increase in enzyme activity of 3 times or more, it is recommended to stop taking statins.

In the absence of the expected effect of statins, a new class of lipid-lowering drugs can be prescribed: an inhibitor of cholesterol absorption in the intestine - ezetimibe. It can be prescribed either independently or in addition to statins:

- ezetimibe 10 mg /day;

With an increase in triglycerides and a decrease in high-density lipoprotein (HDL) cholesterol, fibroic acid derivatives (fibrates) can be prescribed, for example fenofibrate (145 mg / day). It is better to take the drug in the morning. A combination of fibrates with statins is possible, a side effect of such a combined intake is myopathy. It is necessary to carefully monitor the level of liver enzymes and creatine phosphokinase.

Nicotinic acid can be prescribed to reduce the level of low-density lipoprotein (LDL) cholesterol, triglycerides and increase HDL cholesterol:

- nicotinic acid 2-4 mg/day;
- enduracin (nicotinic acid) 500 mg 3 times a day.

According to the GISSI study, the use of omega-3 polyunsaturated fatty acids, for example Omacor (omega-3 triglycerides) at a dose of 1-4 g/day, reduces the risk of developing CVD.

Angiotensin converting enzyme (ACE) inhibitors

The use of this group of drugs is justified in patients with CHF, including in combination with diabetes mellitus, hypertension, CHF and myocardial infarction. This was confirmed by the studies of EUROPA and HOPE. Assign:

- perindopril 2.5-10 mg /day;
- ramipril 2.5-10 mg / day.

Recently, the world literature has been actively discussing the possibility of using targeted therapy - monoclonal antibodies that reduce cholesterol and LDL levels by inhibiting the PCSK9 enzyme (evolocumab and alirocumab).

Drugs that improve the quality of life of patients with stable angina pectoris

Antianginal (anti-ischemic) drugs

This group includes beta-blockers, α -, β -blockers, blockers of slow calcium channels, nitrates. It is in this sequence that it is recommended to prescribe these classes of medicines.

Beta-adrenoblockers

Basic requirements:

- selectivity;
- lack of internal sympathomimetic activity;

- a single dose during the day.

The optimal dose of drugs corresponds to the achieved heart rate of 50-60 per minute.

The most recommended drugs:

- bisoprolol 5-10 mg 1 time per day;
- metoprolol 50-100 mg 1-2 times a day;
- nebivolol 2.5-5 mg 1 time per day;
- carvedilol 25-50 mg 2 times a day.

If this group of drugs is intolerant or if there are contraindications to them, **slow calcium channel blockers (CCBs)** can be prescribed.

In patients with CHF, this group of drugs helps to increase exercise tolerance and reduce the frequency of painful angina attacks.

Absolute contraindications to the appointment of beta-blockers:

- bronchial asthma;
- severe bradycardia at rest;
- pronounced manifestations of obliterating atherosclerosis of the arteries of the lower extremities;
- the terminal stage of CHF.

When using them, sinus bradycardia, various heart blockages, arterial hypotension, weakness, and sleep disorders may occur.

Slow Calcium Channel Blockers (CCBs)

Medicines of this group are recommended to be prescribed in the form of prolonged forms.

From dihydropyridine blockers of slow calcium channels recommended:

- amlodipine 5-10 mg / day;
- felodipine 5-10 mg / day.

The mechanism of action of these drugs is peripheral vasodilation; as a result, these drugs contribute to the development of tachycardia. They can be prescribed to patients with sinus node weakness syndrome, impaired atrioventricular conduction, and pronounced sinus bradycardia.

Non-dihydropyridine blockers of slow calcium channels are recommended:

- verapamil 120-480 mg/day;
- viltiazem 120-320 mg/day.

These drugs slow down atrioventricular conduction, therefore they are indicated for supraventricular arrhythmias, but are contraindicated in sinus node weakness syndrome and impaired atrioventricular conduction. In addition, according to INVEST research, verapamil reduces the risk of developing diabetes mellitus.

Nitrates

It is recommended to prescribe the following nitrate preparations.

- nitroglycerin 0.5 mg at an attack;
- isosorbide dinitrate (Nitrosorbide 30 mg/day, Cardiket 40-120 mg / day);
- isosorbide mononitrate (Monocinque, Monosan 40-120 mg/day);
- molsidomine has a nitrate-like effect (4 mg 2-3 times a day), which can be used to prevent angina pectoris.

The main mechanisms of action of nitrates:

- venodilation, reduction of venous return to the heart, reduction of preload and myocardial oxygen demand;

- moderate expansion of arterioles in the large and small circulatory circles, reduced afterload on both ventricles of the heart;
- reduction of vasoconstriction and elimination of coronary artery spasm.

A common side effect of nitrates is headache, rebound syndrome upon withdrawal, and the development of addiction.

With Stable angina pectoris I and II FC, short-acting nitrates are prescribed 5-10 minutes before the expected physical exertion, which can cause an attack of angina pectoris, or moderately prolonged-acting drugs.

With Stable angina pectoris III and IV FC, only prolonged-acting nitrates are prescribed.

If-channel inhibitors

A new class for the treatment of Stable angina pectoris is if-channel inhibitors of the sino-atrial node, in particular ivabradine (5-7.5 mg 2 times a day). By contributing to the blockade of ion currents of the sinus-atrial node, ivabradine reduces heart rate and, according to the BEAUTIFUL study, reduces the risk of cardiovascular complications. The drug can be prescribed for intolerance to beta-blockers or even in combination with them.

Myocardial cytoprotectors, which have an antianginal effect, are prescribed for Stable angina pectoris of all FC. Slow-release trimetazidine is prescribed 35 mg 2 times a day.

In case of Stable Angina I FC, basic drug antianginal therapy is usually not performed; acetylsalicylic acid, trimetazidine are prescribed. In Stable angina pectoris II FC, antianginal therapy is performed with one of three anti-ischemic agents. When combined with hypertension, sinus tachycardia, arrhythmias, beta-blockers are preferred, since their beneficial effect has been proven to reduce the risk and frequency of recurrent heart attack. Slow calcium channel blockers are indicated for concomitant bron-

chial asthma, chronic obstructive pulmonary disease (COPD), hypertension, supraventricular arrhythmia (verapamil), obliterating atherosclerosis of the lower extremities, diabetes mellitus. They are recommended for young and middle-aged patients who lead an active lifestyle.

Nitrates are prescribed in the presence of contraindications to β -adrenoblockers and concomitant heart failure. They are preferred for elderly people who lead a sedentary lifestyle. In Stable angina pectoris III FC, two antianginal drugs are prescribed: β -adrenoblocker + CCBs; CCBs + nitrates, β -adrenoblocker + nitrates. If there is no effect, after a week you should switch to a combination of three drugs. In parallel, antiplatelet and statin therapy is carried out. In case of insufficient antianginal effect, it is advisable to prescribe trimetazidine. In case of Stable angina pectoris IV FC, combined therapy with drugs of all three main groups is performed. It is mandatory to prescribe antiplatelet agents and statins. Trimetazidine is used to enhance the antianginal effect. If you are intolerant of nitrates, molsidomine is prescribed. For long-term treatment of Stable angina pectoris, only prolonged forms of beta-blockers, CCBs and nitrates are used. Treatment is considered effective if angina attacks are eliminated or Stable angina is reduced by at least one FC.

INDICATIONS FOR HOSPITALIZATION

Angina pectoris is not an indication for treatment in a hospital setting. Hospitalization is necessary if unstable angina is suspected or if there is a prolonged angina attack with no effect from taking nitroglycerin (that is, if there is a suspicion of myocardial infarction).

PREVENTION AND REHABILITATION

Stable angina pectoris has the most favorable prognosis among all types of angina pectoris. The main method of prevention is the elimination of risk factors: dieting and weight loss, constant medication for the treatment of high blood pressure and complete cessation of smoking. An extremely important point is regular monitoring by a cardiologist.

Requirements for the regime of work, rest, rehabilitation

The main method of physical rehabilitation is metered-dose physical training. To control the level of physical activity, it is advisable to use heart rate. Approximately, it should be 10-12 per minute below the level at which anginal pain, shortness of breath, and fatigue appear.

- With Stable angina pectoris I FC, dosed physical training is carried out 4-5 times a week; training includes walking, skiing, swimming. The exercises are performed at a steady pace, but acceleration is acceptable. After 6-7 weeks, in the absence of angina attacks, physical activity can be increased by including short-distance jogging and sports games in training. A medical examination and an ECG are required every 6 months.

- With Stable angina pectoris II FC, dosed physical training is carried out 3-4 times a week. This is walking at a constant speed under the heart rate control lasts 45-60 minutes (minute accelerations are allowed). Daily walks are also recommended, during which the patient should walk 5-7 km. After 6-7 weeks of training, jogging for a few minutes can be recommended. After another 2-3 months, skiing and swimming are allowed. Once every 2 weeks, a doctor's monitoring and an ECG are required.

- With Stable angina pectoris III FC, dosed physical training is carried out in the form of slow walking without acceleration at a pace below the pain threshold. If the condition improves, you should move at a speed of 3-5 km / h for 20-60 minutes. In addition, it is recommended to perform breathing and light physical exercises 1 time every 5 days. Rest days are shown at least once every 5 days.

- With Stable angina pectoris IV FC, walking at a pace of 60-70 steps per minute and individually selected physical therapy (exercise therapy) in a gentle mode, lasting 15-20 minutes under heart rate control in a safe frequency zone are recommended.

Psychological rehabilitation

The most accessible are rational psychotherapy, group psychotherapy and autogenic training. It is possible to use tranquilizers and antidepressants.

Contraindicated working conditions

Regardless of the type of Stable angina pectoris, it is absolutely contraindicated:

- work associated with constant or episodic significant physical overstrain (loader, steelworker, bricklayer, hammer thrower, etc.);
- work associated with conditions that pose a danger to others in case of sudden termination (pilot, dispatcher);
- work at height and in extreme conditions;
- work related to the effects of vascular and neurotropic poisons (tobacco industry, contact with gasoline, increased carbon monoxide content).

In Stable angina pectoris II FC are contraindicated:

- energy costs above 4.2 kcal/min (milling, welder, turner, outerwear cutter, etc.);
- pronounced neuropsychic stress (large-volume administrative work, work on the conveyor);
- work in the field, away from populated areas;
- work related to permanent residence in adverse meteorological and microclimatic conditions.

If it is impossible to find rational employment, patients with Stable angina pectoris I and II FC are referred to the ICEC to determine the III disability group.

In patients with Stable angina pectoris III FC, the ability to work is significantly limited. They are mostly disabled people of group II. Employment is possible only in conditions of light physical and mental work.

Patients with Stable angina pectoris IV FC are unable to work.

Requirements for dietary prescriptions and restrictions

Principles of diet therapy:

- limiting the total fat intake to 70 g (of which 2/3 should be vegetable);
- a sharp decrease in the consumption of saturated fatty acids (animal fats, butter);
- increased consumption of foods enriched with polyunsaturated fatty acids (vegetable oil, poultry, seafood, fish);
- increased consumption of fiber and complex carbohydrates (vegetables, fruits), the amount of fiber in the diet should be 35 g/day;
- replacing butter with vegetable oil during cooking;
- a sharp decrease in the consumption of cholesterol-rich foods (liver, kidneys, brains, eggs, fatty meat, butter, pork and mutton fat);
- reducing the consumption of table salt to 5 g /day.

The sexual aspect of rehabilitation

The patient should be informed in a timely manner about the possibility of an angina attack at the time of sexual intercourse. To prevent such a situation, the patient should take antianginal drugs. When Stable angina pectoris is combined with erectile dysfunction, after consulting a doctor, phosphodiesterase inhibitors of type 5 can be used: sildenafil, vardenafil (but with mandatory consideration of contraindications, such as taking prolonged nitrates, low blood pressure).

DISEASE OUTCOMES AND COMPLICATIONS

The improvement is expressed in a decrease in the frequency of seizures, a decrease in the intensity of pain, and an increase in exercise tolerance. The time of achievement is 3 months. Stabilization is expressed in the absence of both positive and negative dynamics during the course of the disease; with a stable course throughout the year, a consultation with a cardiac surgeon is necessary to resolve the issue of coronary artery bypass grafting.

The progression of the disease is expressed in the transition to a higher FC, in the appearance of rhythm disturbances, signs of circulatory insufficiency, and the development of myocardial infarction. With progression, hospitalization in a hospital is necessary.

DISPENSARY SUPERVISION

All patients suffering from Stable angina pectoris should be registered at the dispensary. The frequency of visits to the doctor depends on the severity of angina pectoris. Laboratory and instrumental studies should be carried out at least once every 6 months.

FORECAST

With Stable angina pectoris I and II FC, improvement occurs in 65% of cases; stabilization occurs in 15%; and progression and development of myocardial infarction occurs in 10%. With Stable angina pectoris III and IV FC, improvement occurs only in 10% of cases; stabilization - in 75%; progression and development of myocardial infarction - in 5% of cases.

EXAMINATION OF DISABILITY

Criteria for temporary disability: increased frequency of seizures, occurrence of rhythm and conduction disorders. The estimated time of temporary disability in Stable angina pectoris II FC is 6-10 days. Patients with Stable angina pectoris I and II FC without significant rhythm disturbances

and heart failure of no more than stage IIA, working in acceptable professions, conditions and types of work, should be recognized as able-bodied.

CONTROL QUESTIONS

1. Definition and classification of stable angina pectoris?
2. Etiology and pathogenesis of stable angina pectoris?
3. The clinical manifestation of stable angina pectoris?
4. The main methods of diagnosis of stable angina pectoris?
5. Differential diagnosis of stable angina pectoris?
6. Treatment of stable angina pectoris?
7. Indications for hospitalization of patients with stable angina pectoris?
8. Prevention and rehabilitation of stable angina pectoris?
9. Features of dispensary supervision of patients with angina pectoris?
10. Examination of disability in stable angina pectoris?

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НА АМБУЛАТОРНОМ ЭТАПЕ**

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