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Department of Internal Medicine №2

METHODOLOGICAL MATERIALS ON CARDIOLOGY

the main professional educational program of higher education - a program of a specialist in a specialty
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Methodological materials are intended for teaching 4th year students (7.8 semesters) of the Faculty of General Medicine of the Federal State Budgetary Educational Institution of Higher Education SOGMA of the Ministry of Health of the Russian Federation in the discipline "Faculty therapy".

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CONTENT

RHEUMATISM

Rheumatism (Sokolsky-Buyo disease) is a systemic inflammatory disease of the connective tissue with a predominant localization of the process in the cardiovascular system, which develops in connection with an acute infection caused by (β-hemolytic and group A streptococcus in predisposed individuals, mainly in children and adolescents 7 -15 years.

Abroad, the term "acute rheumatic fever" is often used to denote acute rheumatism, which should be recognized as more correct.

Prevalence... Rheumatism is recorded in all climatogeographic zones. According to the generalized data of the WHO (1989), the incidence of rheumatism in economically developed countries began to decline at the beginning of the 20th century, taking the most pronounced rate over the past 30 years (the period of intensive introduction of antibiotics, as well as the prevention of rheumatism), and now these areas are 5 per 100,000 population per year. RC Willams (1994) cites similar data.

However, rheumatism has not been completely eradicated, as evidenced by outbreaks of the disease that have taken place in the United States and other countries of the world in the last decade.

In the Russian Federation, the primary incidence of rheumatism over the past 2-3 years has increased from 0.05 to 0.08 per 1000 population, and among the child contingent this indicator has increased even more intensively - from 0.06 to 0.16 per 1000 children.

The indicated increase in the incidence of rheumatic fever confirms the postulate of J. Rotta that rheumatism will not disappear as long as group A streptococcus circulates, and the population of our planet cannot be rid of this group streptococcus over the next several decades.

The presented prevalence of rheumatic fever and rheumatic heart defects caused by them high temporary and persistent disability of the adult contingent of patients indicates the social significance of this problem.

Because of this, this pathology continues to attract close attention of researchers.

Etiology and pathogenesis...streptococci can secrete a wide range of somatic and extracellular substances with toxic and antigenic properties. These include streptolysins O and B, streptokinase and hyaluronidase, proteins and deoxyribonuclease B, etc. These substances are capable of damaging various cells and tissues of the body, thereby exerting a pathogenetic effect.

Indirect confirmation of the importance of streptococcal infection in rheumatism should be considered the detection in the vast majority of patients of various anti-streptococcal antibodies - antistreptolysin-O (ASL-O), antistreptogyaluronidase (ASH), antistreptokinase (ASK), antideoxyribonuclease B (antiDNAase B).

However, streptococcal exposure alone is not enough for the development of the disease.

For its occurrence, an individual hyperimmune reaction of the body to streptococcal antigens and the duration of this response are needed. The absence of rheumatism in young children suggests that it is for the development of the disease that the repeated exposure of group A streptococci on the child's body is necessary.

It has also been established that the patient's predisposition is of great importance for the occurrence of rheumatism. So, rheumatism occurs only in 0.3-3% of children who have had an acute streptococcal infection.

The predisposition to rheumatism is a complex concept that includes the synthesis of a number of terms. It is not limited only to the special reactivity of anti-streptococcal immunity.

At the beginning of this century, the founder of the pediatric school A.A.Kisel pointed out the role of seminal predisposition in the onset of rheumatic fever, as evidenced by familial aggregation of rheumatism, significantly exceeding the prevalence of the disease in the population.

It was found that the frequency of "family" rheumatism is undoubtedly influenced by environmental (primarily streptococcal infection) and genetic factors.

L. I. Benevolenskaya et al. (1989) as the most adequate offer a multifactorial model of rheumatism, based on the joint participation in the formation of susceptibility to the disease of a large number of genetic and environmental factors.

The role of genetic predisposition is confirmed by the more frequent development of rheumatism in siblings of a patient from a family in which one of the parents suffers from rheumatism, as well as a higher prevalence of the disease among monozygotic twins than heterozygous twins (Benevolenskaya L.I. et al., 1989).

The study of genetic markers revealed that among patients with rheumatism, people with blood groups A (II), B (III) and "nonsecretors" AB and H are more common.

Interesting data were obtained when evaluating dermatoglyphics as a genetic marker. The general regularity of this indicator in patients with rheumatism is the redistribution of the patterned phenomenon on the fingers.

The system of histocompatibility antigens in rheumatism is being intensively studied. A series of studies has been completed. At the same time, a certain pattern of associations with HLA antigens, characteristic for each specific study, was revealed. So, according to the observations of N. Yu. Goryaeva (1986), with rheumatism in patients of Russian nationality, HLA-A11, B35, DR5 and DR7 prevail. It has been shown that in patients with valvular heart disease, the frequency of HLA-A3 carriage is increased; if the aortic valve is damaged, the HLA-B15 antigen is present. Some researchers have noted an increase in the content of HLA and DR4 in rheumatism. Thus, rheumatism is characterized by a wide variability in the association of histocompatibility antigens.

In recent years, the hypothesis has been discussed that the alloantigen of B-lymphocytes, determined using monoclonal antibodies D8 / 17, is associated with susceptibility to rheumatism and can be considered as a genetic marker that determines the hereditary predisposition to the disease. The place of antiphospholipid antibodies to the development of various clinical manifestations of rheumatic fever is being studied.

It has been established that in the complex pathogenesis of rheumatism, a large role is played by immune inflammation, as well as immunopathological processes, in which streptococcal antigens and anti-streptococcal antibodies are most actively involved. In this case, the concept of the role of cross-reacting antigens - antigenic components of streptococcus and body tissues - has the greatest confirmation.

The hypothesis of "antigenic mimicry" between somatic fragments of group A streptococci and human myocardial antigens was recognized, as well as the concept of a similarity between the streptococcus wall and the glycoprotein contained in the human heart valves. The pathogenetic significance of these cross-reactions is unclear, but the interaction between the microorganism and the microorganism may explain the development of myocarditis and valvulitis.

The role of circulating anticardial antibodies and immune complexes in the pathogenesis of the disease was discussed. A series of studies was devoted to the study of cellular immunity disorders, which revealed changes in the quantitative ratio of T- and B-lymphocytes and their functional activity. There have been reports criticizing the concept of an autoimmune nature of rheumatic fever. Currently, new directions are being developed in the study of the pathogenesis of post-streptococcal diseases, including rheumatism (Totolyan A.A., 1988; Nasonov E.L., 1991, etc.).

So, in the development of the disease, streptococcus plays a large role, which has a multifaceted effect, which is realized only in a predisposed organism and determines a variety of clinical manifestations.

Pathological picture... The development of the rheumatic process is accompanied by a variety of morphological changes. It has been established that the primary and predominant lesion of the connective tissue, primarily the heart, is the main one. There are 4 stages of development of the pathological process in rheumatism: mucoid swelling, fibrinoid changes, proliferative reactions and the phase of sclerosis.

The possibility of reverse development of the pathological process at the stage of mucoid

swelling should be considered of fundamental importance.

Fibrinoid changes represent an irreversible phase of connective tissue disorganization. The proliferative stage is manifested by the formation of rheumatic, ashoff-talalaevsky (named after the authors who described it) granulomas.

Rheumatic granuloma consists of large, irregularly shaped basophilic cells of histiocytic origin, giant multinucleated cells of myogenic origin with eosinophilic cytoplasm, as well as lymphoid, plasma and mast cells. Typical rheumatic granulomas are nosological, occurring only in the heart. They are most often located in the perivascular connective tissue or in the interstitium of the myocardium (mainly of the left ventricle), papillary muscle, septum, as well as in the endocardium, less often in the outer shell of the vessels. The cycle of granuloma formation and scarring takes 3-4 months on average.

Currently, granulomas in pathological examination are found much less frequently than previously noted, which is apparently associated with a change in the clinical and morphological "appearance" of rheumatism, with the so-called pathomorphosis of the disease.

An important morphological substrate of heart damage in rheumatic heart disease is a nonspecific inflammatory reaction. It consists of edema of intermuscular connective tissue, fibrin sweating, infiltration with cellular elements, mainly polymorphonuclear leukocytes and lymphocytes.

According to the studies of M.A. Skvortsov (1946), it is the nonspecific exudative-proliferative component of inflammation that determines the clinical manifestations of the disease and correlates directly with the activity of the pathological process and the severity of carditis. These patterns are especially pronounced in childhood.

The favorite localization of the pathological process is the mitral valve, less often the aortic and tricuspid. It is in children that the clinical symptoms of valvulitis of the mitral and aortic valves often develop.

The outcome of the above processes is cardiosclerosis. Among the nonspecific, but important for the pathogenesis and morphogenesis of rheumatism are changes in the vessels of the microvasculature, which are found in all organs.

Serous membranes are constantly involved in the process, especially with high activity of rheumatism, causing a picture of serous, serous-fibrous inflammation.

In articular tissues with clinically pronounced rheumatic polyarthritis, manifestations of disorganization of connective tissue, exudative inflammation, and vasculitis are observed. A feature of rheumatic joint damage is the reversibility of the process not only in the phase of mucoid swelling, but also in the initial stages of fibrinoid changes.

At the heart of the defeat of the nervous system is the involvement of the cerebral vessels in the rheumatic process. The pathological substrate of small chorea is a change in the cells of the striatum, subthalamic nuclei of the cerebral cortex and cerebellum.

Damage to the skin and subcutaneous tissue is manifested by signs of vasculitis, endotheliosis and focal inflammatory infiltration.

Thus, the basis of rheumatism is a process that is complex in its structure, in which, against the background of impaired reactivity of the body, inflammatory and proliferative changes of different intensity are played out, causing a variety of clinical manifestations of the disease.

Clinical picture... Acute rheumatic fever most often occurs in school and adolescence, much less often in preschoolers. In children of the first three years, it practically does not occur. Gender does not have a significant effect on the incidence of rheumatism in children, although girls are slightly more likely to get sick.

According to the observations of domestic and foreign researchers, rheumatism is characterized by a significant variety of clinical manifestations and variability of the course.

Outstanding pediatrician A.A.Kisel (1939) gave a brilliant description of the main manifestations of rheumatism, calling them the absolute symptom complex of the disease. These include: polyarthritis, heart disease, chorea, erythema annularis, rheumatic nodules.

Polyarthritis, carditis, chorea can occur in isolation, but more often in various combinations with each other.

Rheumatic arthritis... Articular syndrome is observed in 2/3 of children with rheumatism for the first time, and in about half of patients with a second attack of the disease. Transient oligoarthritis, less often monoarthritis, should be considered the predominant form of lesion in modern conditions. Classic polyarthritis in recent years is less common. The main symptoms of rheumatoid arthritis are acute onset, fever, joint pain, swelling, limited movement, possibly fever and redness of the skin above them, that is, it is usually reactive in nature. Rheumatic arthritis is characterized by the involvement in the process of large and medium joints, most often the knee and ankle, the symmetry of the lesion, volatility, the rapid reverse development of the pathological process (especially against the background of anti-inflammatory therapy).

A feature of the modern course of articular syndrome in children is often both a reduced nature and the possibility of prolonged arthritis.

Rheumatoid arthritis is most often combined with carditis, less often it occurs in isolation.

*Rheumatic carditis.*Leading in the clinical picture of rheumatism, which determines the severity of its course and prognosis, is heart damage - rheumatic heart disease. The latter occurs in 70-85% of children with rheumatism for the first time, and not at all more often with recurrent rheumatic heart disease. The symptomatology of rheumatic heart disease is largely determined by the predominant lesion of one or another shell of the heart - the myocardium, endocardium and pericardium. However, due to the difficulties in differentiating the lesions of individual membranes of the heart in clinical practice, the term "rheumatic heart disease" has become widespread. Diagnosis of the latter is based on subjective and objective data.

According to the observations of the majority of pediatricians, subjective complaints in childhood often fade into the background, and only 4-6% of children, upon careful questioning, note discomfort in the region of the heart at the onset of the disease. Complaints of increased fatigue, especially after school hours, are presented by 12-15% of patients.

One of the earliest clinical symptoms of a new-onset rheumatic process in most children (60-70%) is a violation of the general condition and an increase in temperature. Simultaneously with these signs or after them, the first manifestations of incipient carditis may appear.

Early symptoms of primary rheumatic heart disease include heart rate abnormalities in the form of tachycardia (30-40%) or bradycardia (20-30%). In some children (30-40%), the heart rate at the onset of the disease has no noticeable deviations from the norm.

The characteristic clinical signs of the disease include the expansion of the boundaries of the heart, detected in 80-85% of patients. The latter, as a rule, is moderate and occurs mainly to the left. Percussion expansion of the boundaries of the heart is confirmed by X-ray examination.

An important diagnostic sign of primary rheumatic heart disease should be considered a weakening of heart sounds, which is detected in the vast majority of patients and is reflected in phonocardiographic examination in the form of a decrease in amplitude, deformation, widening and depletion of high-frequency oscillations of mainly 1 tone.

Relatively often, with primary rheumatic heart disease, it is possible to detect additional III (45-75%) and less often IV (15-25%) tones, while the frequency of their detection, as a rule, correlates with the severity of carditis.

The most common symptom of primary rheumatic heart disease is the appearance of a systolic murmur. Depending on the predominant damage to the myocardium or endocardium of the valves, systolic murmur has different localization, intensity, duration, timbre and conductivity. So, with myocarditis, the noise is usually weak or moderate, it is better heard at the V point, less often on the pulmonary artery; outside the heart area, as a rule, is not provided. According to PCG data, for patients with myocarditis, the most typical systolic murmur, oval-subsiding shape, medium-amplitude and medium-frequency, recorded immediately after the I tone, mainly in the V point in the pulmonary artery.

On an ECG in rheumatic myocarditis, dysfunction of the sinus-atrial node is quite often recorded, which is reflected in the form of tachycardia, bradycardia and respiratory arrhythmia.

Less often, especially in the early stages of the development of rheumatic heart disease, rhythm disturbances are detected: migration of the pacemaker, interference with dissociation. Along with this, there may be a violation of atrioventricular conduction I and much less often II degree. On the ECG, violations of bioelectrical processes in the ventricular myocardium are often recorded.

X-ray examination in most children with rheumatic myocarditis reveals signs of a decrease in contractile function and myocardial tone.

The priority of domestic pediatricians was an attempt to isolate the component of valvular lesion in the general clinical picture of primary rheumatic heart disease. Of particular importance in its recognition is the qualitative characteristic of the newly appeared noise. So, with endomyocarditis with mitral valve lesions, systolic murmur most often has a blowing character, is characterized by duration, is best heard in the zone of the mitral valve projection (apex of the heart, point V), is often carried out to the left outside the heart region, and intensifies after exercise. The blowing timbre of systolic murmur, its apical localization give grounds already in a relatively early period of the disease to suspect endomyocarditis with damage to the mitral valve. On the PCG, this noise, as a rule, is recorded in the high-frequency spectrum in the form of pansystolic murmur or prolonged protosystolic murmur, usually has a small amplitude and the epicenter of registration at the apex. This qualitative characteristic of the noise contributes to its correct interpretation and interpretation as endocardial.

Ultrasound examination of the heart helps to objectify the signs of valvular lesion. With valvulitis of the mitral valve in 75% of children, thickening and "shaggy" of the echo signal from the leaflets and chords of the valve is revealed. In about 1/3 of patients, there is a limitation of the mobility of the posterior valve leaflet, a decrease in systolic excursion of the closed mitral leaflets. Often, a slight prolapse of the leaflets at the end of systole is found, indicating a lesion of the subvalvular apparatus.

In subsequent years, using a new generation of equipment, namely with the help of Doppler echocardiography (DEHOKG), the DEHOKG criteria of rheumatic endocarditis were identified, which can be successfully used in any age group.

Rheumatic endocarditis of the mitral valve is characterized by the following symptoms:

- marginal clavate thickening of the anterior mitral valve;

- hypokinesia of the posterior mitral leaflet;
- mitral regurgitation;
- passing dome-shaped diastolic bend of the anterior mitral valve.

Simultaneously with the systolic murmur in the mitral region, a mesodiastolic murmur can be heard, which is more often recorded on the PCG than on auscultation. Appearing in the midst of rheumatic heart disease, mesodiastolic murmur under the influence of active treatment quickly disappears. Monitoring its dynamics allows us to classify this noise among the main signs of primary rheumatic heart disease.

When electrocardiographic examination in children with severe mitral valvulitis, there are signs of acute overload of the left atrium with mitralization of the P wave, in some patients these changes are combined with the initial symptoms of an increase in the left ventricle.

X-ray examination of children with mitral valvulitis makes it possible to ascertain the presence of the so-called mitral configuration of the heart by the auricle of the left atrium, an increase in the size of both left cardiac chambers; in some cases, there are signs of impaired pulmonary hemodynamics in the venous bed of the lungs.

Of great diagnostic significance, despite the relative rarity (3-5%), is listening to the "pouring" diastolic noise along the left edge of the sternum, recorded on the PCG in the form of high-frequency, protodiastolic murmur and indicative of aortic valve lesion.

Ultrasound examination with aortic valve valvulitis reveals small-amplitude diastolic flutter of the mitral cusps in 50% of children. In some patients, a thickening of the echo signal from the aortic valve leaflets is found.

On the ECG with aortic valve valvulitis, signs of diastolic overload of the left ventricle are often recorded.

Radiographically, valvulitis of the aortic valve is characterized by a tendency towards a horizontal position and aortic configuration of the heart, a predominant increase in the left ventricle, a relative increase in its pulsation and aorta.

In primary rheumatic heart disease, pericardial friction noise may appear, however, clinically, the latter is determined extremely rarely in the modern course of the disease (1-2%). At the same time, during X-ray examination of the patient, signs of limited adhesive pleuropericarditis can be detected somewhat more often.

As for circulatory insufficiency, the latter is rarely observed in the modern course of primary rheumatic heart disease and is usually stage I and much less often stage II (according to the classification of ND Strazhesko and V.Kh. Vasilenko, 1935).

One of the most important clinical criteria confirming the presence of primary rheumatic heart disease in a child is the positive dynamics of its clinical and paraclinical manifestations under the influence of active antirheumatic therapy. So, in the overwhelming majority of children, on the background of treatment, normalization of the heart rate, restoration of sonority of tones, a decrease in the intensity of systolic and diastolic murmurs, a reduction in the boundaries of the heart, disappearance of symptoms of circulatory insufficiency occurs. Dynamic study of the evolution of disease symptoms is of great importance for the diagnosis of primary rheumatic heart disease.

The accumulated medical experience shows that it is important not only to diagnose primary rheumatic heart disease, but also to clarify the degree of its severity, since a differentiated assessment of the severity of cardiac changes makes it possible to prescribe adequate pathogenetic treatment and carry out all the necessary therapeutic and preventive measures in the future.

To characterize rheumatic heart disease in children, as well as in adults, one can be guided by the most important definitions proposed by A. I. Nesterov (1969 - 1973): bright, moderate and mild rheumatic heart disease.

For a differentiated assessment of the severity of carditis, a wide range of clinical and instrumental-graphic research methods are used. The group with bright carditis can include children who have pronounced pathological changes in the form of pancarditis or endomyocarditis with involvement of the valvular apparatus of the heart in the pathological process, characterized by a distinct expansion of the borders of the heart and congestive heart failure (circulatory failure of

stage II-III); the second group (with moderate carditis) consists of patients with myocarditis or endomyocarditis without clear signs of valvular lesion, with a moderate expansion of the borders of the heart, without congestive heart failure (circulatory failure not higher than stage I),

Shifts in paraclinical parameters directly correlate with the severity of carditis.

In the conditions of modern reality (80-90-ies) in the overwhelming majority of patients after the 1st attack of rheumatism, moderate and mild carditis is detected, less often there are distinct changes in the heart. However, in the domestic and foreign literature of recent years, there have been reports of a possible severe course of primary rheumatic heart disease.

It has been established that the outcome of rheumatism is determined by the frequency of the formation of heart disease. The percentage of cases of heart defect formation after primary rheumatic heart disease has decreased by 2.5 times and is currently 20-25%. There is a direct relationship between the severity of rheumatic heart disease and the incidence of heart disease.

With the help of ultrasound, it is possible to identify commissural mitral stenosis already in the outcome of primary rheumatic heart disease, which, as a rule, does not have clinical manifestations, but is determined only by echoscanning.

In about 7-10% of children, after suffering from primary rheumatic heart disease, mitral valve prolapse develops.

A feature of the formed heart defects should be considered a slower (compared to previous decades) rate of their occurrence, an unsharp degree of their severity and stable compensation over a number of years.

Recurrent rheumatic heart disease often develops against the background of acquired heart disease, and often in children with an almost intact heart, that is, without a valve defect formed during a previous attack.

A characteristic manifestation of the disease should be considered the detection of new noises or an increase in their intensity, a change in the sonority of tones, the appearance of signs of circulatory failure.

As in primary rheumatic heart disease, auscultatory data are supplemented by the results of electrophysiological, X-ray and ultrasound studies.

A feature of recurrent rheumatic heart disease is an increase in the number of cases of subsequent formation of heart disease, especially in adolescence.

*Chorea.*This disease, being the lot of childhood, occurs in 12-17% of patients with rheumatism. Chorea mainly affects girls between the ages of 6 and 15.

The disease begins gradually with the appearance of an unstable mood, asthenia of the child, tearfulness, irritability. Later, the main symptom complex of chorea is added, characterized by hyperkinesis, discoordination of movements, and a decrease in muscle tone.

Hyperkinesis is manifested by erratic, non-stereotypical, violent movements of various muscle groups and are accompanied by a violation of handwriting, slurred speech, and awkwardness of movements. It is difficult for a child to bring a spoon to his mouth, drink and eat on his own.

Hyperkinesis intensifies with excitement, disappears during sleep, more often bilateral, less often unilateral (hemichorea). Implementation of coordination problems is difficult. Having closed his eyes, the child cannot, after spreading his hands, precisely touch the tip of the nose with his index finger and hold his tongue out for a long time (> 15 s), has difficulty inflating his cheeks and glass of teeth.

In the supine position, putting the heel of one leg on the knee of the other, sliding movements cannot touch the big toe.

The doctor is able to detect even minor hyperkinesis if he holds the child's hands in his hands for a long time.

With a pronounced form of chorea, the symptom of "flabby shoulders" is positive (when the patient is lifted by the armpits, the head sinks deep into the shoulders); there is a retraction of the epigastric region during inhalation, a delay in the reverse flexion of the lower leg when checking the knee reflex.

There are patients with severe muscular hypotension.

Chorea minor is often accompanied by symptoms of vegetative dystonia.

In the conditions of modern reality, the severity of clinical manifestations of chorea has decreased, practically no "choreic storm" and "paralytic form" are found.

Against the background of adequate therapy, the manifestations of chorea disappear after 1-2 months, however, in some children, the pathological process is delayed for a longer time.

Annular erythema(annular rash). It is observed in 7-10% of children at the height of a rheumatic attack. Clinically, it manifests itself as pale pink ring-shaped rashes, usually not accompanied by itching or other subjective sensations, does not rise above the level of the skin, and disappears with pressure. It is mainly localized on the skin of the trunk, less often on the arms and legs. Ring-shaped erythema usually quickly disappears, sometimes delayed for a longer period.

Children may have an annular rash.

According to modern views, ring-shaped erythema can occur not only in rheumatism, but also in other diseases (toxic-allergic form of chronic tonsillitis, allergic conditions).

*Rheumatic nodules.*In recent years, rheumatic nodules have been observed very rarely, mainly in children with recurrent rheumatic heart disease. They are round, dense, varying in size from a few millimeters to 1-2 cm, painless formations. The predominant localization is at the places of attachment of the tendons, above the bony surfaces and protrusions, in the area of the knee, elbow, metacarpophalangeal joints, and the occipital bone. The cycle of reverse development is on average 1-2 months, without residual effects.

Internal organ damage... In children with the modern course of rheumatism, internal organs are rarely affected, this manifests itself mainly in the form of an abdominal syndrome, which is noted in 5-7% of patients, as a rule, at the onset of a rheumatic attack. It is manifested by abdominal pain, which can be different in their severity and localization. Against the background of antirheumatic treatment, as a rule, there is a rapid, reverse development of symptoms.

Damage to the lungs (rheumatic pneumonia, pulmonary vasculitis and pleurisy), kidney (glomerulonephritis) and other organs, observed mainly in the severe course of the first attack and recurrent rheumatic heart disease, is now extremely rare.

Laboratory research... Based on the signs of the leading role of group A streptococcus in the development of rheumatism, this disease is considered as an immunological problem, great importance in its early diagnosis is given to shifts in immunological parameters. According to the observations of most researchers, the frequency of detection of streptococcal antigen in blood serum in the early stages of primary rheumatic heart disease is 60-75%, an increase in ASL-0 titers is observed in 70-85% of children, ASH - in 80-90% of patients.

A reflection of autoimmune processes is the detection of anticardial autoantibodies that have a pronounced cross-activity with group A streptococcus.In 1/3 of children in the early stage of the disease, circulating antibodies to connective tissue antigens, namely to structural glycoproteins and a soluble fraction of the main substance of connective tissue, are found.

The study of humoral parameters of immunity indicates an increase in all classes of immunoglobulins (A, M and G).

Circulating immune complexes are often detected. There are also shifts in the indicators of inflammatory activity. In the overwhelming majority of children with primary rheumatism, there is an increase in ESR, an increase in seromucoid indicators, dysproteinemia with a decrease in the amount of albumin and an increase in the level of globulin fractions due to an increase in γ-globulins, there is a tendency to leukocytosis.

Laboratory indicators, as a rule, have a direct relationship with the degree of activity of the rheumatic process, with the exception of chorea, when, despite severe clinical manifestations, they can remain within normal values.

The clinical picture of rheumatism in adults has been studied in detail by domestic and foreign researchers, the evolution of the disease in a favorable direction is noted.

Despite this, the main clinical syndrome of rheumatism in the adult contingent of patients remains rheumatic heart disease, which is observed in 90% of patients with primary and in 100%

with age-related rheumatism. In this case, pronounced rheumatic heart disease is usually found in acute and subacute course of primary rheumatism.

The symptom complex of primary rheumatic heart disease, regardless of its severity, along with objective data, is often accompanied by asthenia of the patient, subjective complaints of shortness of breath, palpitations, interruptions, cardialgia.

The formation of a heart defect after one attack occurs in 39-45% of cases. Moreover, the maximum frequency of their occurrence is observed during the first three years from the onset of the disease.

As new exacerbations appear, cardiac pathology progresses and recurrent rheumatic heart disease occurs against the background of combined and combined heart defects.

The incidence of joint damage in primary rheumatism in adults is 70-75% and significantly decreases with relapses of the disease. A feature of the articular syndrome, according to most researchers, should be considered the frequent involvement of the sacroiliac joints in the pathological process.

Chorea was observed in the anamnesis only in 11-13% of patients who fell ill in childhood and adolescence.

It should be emphasized that in this age group, the evolution of the disease in a favorable direction is also noted.

A characteristic feature of modern rheumatism in adults should be considered an increase in the latent form.

In elderly and senile people, primary rheumatism practically does not occur, however, relapses of the disease that began at a young age are observed. They are most often manifested by viscerites, mainly mild rheumatic heart disease or polyarthritis, rheumatic nodules.

Classification and nomenclature of rheumatism... Currently, the generally accepted classification and nomenclature of rheumatism, proposed by A.I. Nesterov and approved at a special symposium of the All-Union Antirheumatic Committee in 1964. It was compiled taking into account the following factors: 1) the phase of the disease (active and inactive) with a specification of the degree of activity of the pathological process (I, II, III); 2) clinical and anatomical characteristics of damage to the heart and other organs; 3) the nature of the course of the disease; 4) the state of blood circulation. The inactive phase of the disease includes the consequences and residual manifestations of cardiac (myocardiosclerosis, heart disease) and extracardiac lesions.

The merit in highlighting the degree of activity of the pathological process belongs to A.I. Nesterov (1964). His assessment is based on the characteristics of clinical, functional and laboratory parameters. There is a directly proportional relationship between the severity of the exudative component of inflammation, the clinical manifestations of the disease and the degree of activity of the rheumatic process.

The maximum activity is characterized by vivid clinical manifestations, with a minimum degree, mild symptoms are noted. Similar correlations are found with functional and laboratory parameters.

Differentiated determination of the degree of activity played a significant role in the characterization of the pathological process, the appointment of adequate treatment specifically for each patient, which undoubtedly contributed to an increase in the effectiveness of the therapy.

Clinical and anatomical characteristics of heart disease in the active phase of the disease include primary rheumatic heart disease against the background of valvular disease and rheumatism without obvious cardiac changes. The last heading mainly refers to childhood, since it is in children that rheumatic arthritis or chorea can proceed in isolation without cardiac pathology.

Among the lesions of other organs and systems in the active phase of rheumatism, polyarthritis, serositis, chorea, vasculitis, skin lesions, etc. are distinguished.

When assessing the nature of rheumatism, both the temporal characteristics of the process and the entire complex of clinical manifestations of the disease are taken into account. The duration of an attack in acute and subacute course is on average 1.5-2 months, with a protracted duration it is

delayed up to 4-5 months.

The continuously recurrent variant is characterized by a wave-like course and is usually characteristic of recurrent rheumatism with a formed heart defect. Latent includes a variant of the chronic course in which it is not possible to detect clinical and laboratory indicators of activity. Latent rheumatism can be primary or secondary.

In prognostic terms, the fact that each subsequent attack copies the previous one in its clinical manifestations, the degree of activity and the nature of the course is of great importance.

Insufficiency of blood circulation provides for the allocation of stage 5 according to M.D. Strazhesko and V.Kh. Vasilenko (1935).

3 decades have passed since the adoption of the classification. Over the years, there have been significant changes in the clinical picture and course of rheumatism. A more favorable nature of the disease is noted with a decrease in the severity of the nonspecific component of inflammation, which is reflected in the predominance of a moderate degree of activity of the pathological process, an increase in the frequency of mild carditis without congestive heart failure, the prevalence, predominance of acute and subacute variants of the course, a significant decrease in the percentage of formation of valvular heart defects in the outcome diseases.

In most children, acute rheumatic fever ends with complete recovery.

The continuously recurrent nature of rheumatism in the conditions of modern reality practically does not occur.

Table.

Working classification and nomenclature of rheumatism

Phase and degree of activity of rheumatism	Clinical and anatomical lesion characteristic hearts	Clinical and anatomical characteristic of damage to other organs and systems	The nature of the flow	Circulatory failure (N)
Active: Activity degree I, II, III	Primary rheumatic heart disease without valve defects Recurrent rheumatic heart disease (what) Rheumatism without obvious changes in the heart	Polyarthritis, serositis (pleurisy, peritonitis, abdominal syndrome) Chorea, encephalitis, meningoencephalitis, cerebral vasculitis, neuropsychiatric disorders. Vasculitis, nephritis, hepatitis, pneumonia, skin lesions, iritis, iridocyclitis, thyroiditis	Acute, subacute. Prolonged, continuously relapsing. Latent	H0 H1 H2A H2B H3
Inactive	Myocardiosclerosis is rheumatic. Heart disease (what)	Consequences and residual effects of transferred extracardiac lesions		

All of the above prompts to continue the work (which is being carried out at the present time) to improve the specified classification, reflecting in it the features of the modern course of rheumatism.

Diagnostics... The polymorphism of the clinical manifestations of the disease creates undoubted difficulties in its recognition. Since the time of G.I. Sokolsky and JB Boulliaud did not stop studying and improving the diagnostic criteria for rheumatism.

The priority in the development of diagnostic signs of this disease belongs to the largest Russian pediatrician A.A. Kisel (1940). Based on the accumulated medical experience, as well as

using the richest personal observations, he described the diagnostic signs of rheumatism - polyarthritis, heart disease, chorea, erythema annular, rheumatic nodules - and called them the absolute symptom complex of rheumatism.

Somewhat later, the criteria for the recognition of rheumatic fever were formulated and published by the American researcher TD Jones (1944).

Described by A.A. Kisel, as well as TD Jones, the diagnostic criteria for rheumatism have been successfully used by pediatricians and therapists for a number of years. However, these criteria could not cover the entire variety of clinical manifestations of the disease, and it is quite natural for many researchers to strive to further deepen and clarify them. So, the diagnostic signs of rheumatism were modified by the American Heart Association in 1955 and 1965.

Significant additions to the diagnostic criteria for rheumatism were made by A.I. Nesterov (1963, 1966, 1973), which in our country are called the Kisel-Jones-Nesterov criteria.

Kisel-Jones-Nesterov diagnostic criteria for rheumatism

Main manifestations:

1. Carditis
2. Polyarthritis
3. Chorea
4. Subcutaneous nodes
5. Annular erythema
6. Rheumatic history (connection with a previous nasopharyngeal (streptococcal) infection, the presence of a patient with rheumatism in the family)
7. Evidence ex juvantibus - improvement in the course of the disease under the influence of 3-5 days of antirheumatic treatment.

Additional manifestations:

A. General:

1. Temperature increase
2. Adynamia, fatigue, weakness
3. Pallor of the skin
4. Sweating
5. Nosebleeds
6. Abdominal syndrome

B. Special (mainly laboratory parameters):

1. Leukocytosis (neutrophilic)
2. Dysproteinemia:
    - increased ESR
    - hyperfibrinogemia
    - the emergence of C-reactive protein
    - increased levels of α2 and γ-globulins
    - increased levels of serum mucoproteins, lipoproteins
3. Pathological serological parameters: streptococcal antigen in the blood, increased titers of ASL-O, ASA, ASG.
4. Increased capillary permeability

In such a profound and generalized form, the Kisel-Jones-Nesterov criteria were adopted by rheumatologists.

Discussing the issues of diagnosing rheumatism, all researchers unanimously note the difficulties that arise especially when recognizing the early manifestations of the disease, which explains the rather high percentage of diagnostic errors.

Based on the generalization of the achievements of modern theoretical and practical rheumatology A.I. Nesterov formulated early diagnostic signs of rheumatism, combined into 3 main syndromes: clinical and epidemiological, clinical and immunological, and cardiovascular.

Clinical and epidemiological syndrome includes anamnestically clearly detectable streptococcal infection on the eve of the first symptoms of rheumatism, as well as streptococcal "environment" of the sick in the immediate vicinity of the patient - at home, at school, at work.

Clinical and immunological syndrome summarizes clinical and laboratory parameters. Clinical include an unmotivated delay in the restoration of vigor and full performance following a nasopharyngeal infection, fatigue after a habitual exercise, sweating, previously unusual for the patient, low-grade fever, arthralgia, palpitations.

Laboratory indicators primarily reflect the state of general immunological and inflammatory activity.

Cardiovascular syndrome is based on the generalization of subjective and objective parameters, which are determined during clinical and instrumental examination of the patient, confirming the presence of carditis and other extracardial localizations of the rheumatic process.

In 1982, the criteria for rheumatism were revised again by the American Rheumatological Association, which the WHO study group (1989) recommends to use for the diagnosis of acute rheumatism.

Criteria for the diagnosis of rheumatism	
Big criteria	Small criteria
Carditis	*Clinical:*
Polyarthritis	Prior rheumatism or rheumatic heart disease
Chorea	Arthralgia
Annular erythema	Fever
Subcutaneous rheumatic nodules	*Laboratory:*
	Increased ESR, C-reactive protein, leukocytosis
Data confirming the transferred streptococcal infection	
Increased titer of anti-streptococcal antibodies, ASL-O (antistreptolysin O)	
Others:	
Sowing from the throat of group A streptococcus	
Recently transferred scarlet fever	

The presence of two large or one large and two small criteria indicates a high likelihood of acute rheumatism in the presence of confirmation of data on the transferred infection caused by the streptococcus of troupe A.

As the accumulated long-term experience shows, none of the above diagnostic criteria for rheumatism is specific for this disease, which is why the search for new approaches to the recognition of this pathology continues.

Differential diagnosis... Polymorphism of clinical manifestations of rheumatism, variability of its course often (30-45%) leads to diagnostic errors, due to which a large group of similar conditions is included in the circle of differentiable diseases.

Rheumatic arthritis most often should be differentiated from reactive arthritis, especially after a nasopharyngeal infection. Rheumatoid arthritis is characterized by a "light interval" after a streptococcal infection, equal on average to 10-14 days, acute or subacute onset of the disease, often with an increase in temperature, volatile arthritis, its rapid reverse development, a clear increase in laboratory parameters (increased ESR, high persistent titers of anti-streptococcal antibodies), a frequent combination with other manifestations of the disease (carditis, chorea).

The differential diagnosis of rheumatoid arthritis is carried out with arthritis in other rheumatic diseases (the debut of juvenile rheumatoid arthritis, juvenile ankylosing spondylitis, systemic lupus erythematosus, hemorrhagic vasculitis, with serum sickness).

The qualitative characteristic of the articular syndrome in combination with other clinical manifestations of the disease allows a correct diagnosis to be made.

Carditis. The most frequent source of diagnostic errors in the recognition of rheumatic heart disease in children in modern conditions are functional cardiopathies, non-rheumatic carditis, mitral valve prolapse syndrome, infective endocarditis, congenital heart defects. Early diagnostic signs of primary rheumatic heart disease include:

1. The predominant occurrence of the disease in childhood and adolescence; a close relationship of its development with a previous nasopharyngeal infection;
2. The presence of an interval (2-3 weeks) between the end of the last nasopharyngeal infection and the onset of the disease, less often - a prolonged recovery after a nasopharyngeal infection;
3. Frequent increase in temperature at the onset of the disease;
4. Arthritis or arthralgia;
5. Auscultatory (clinical) and functional signs of carditis;
6. Shifts in acute phase inflammatory and immunological tests;
7. Positive dynamics of clinical and paraclinical indicators under the influence of antirheumatic treatment.

For functional cardiopathy, the following manifestations are characteristic: the presence of chronic foci of infection, the frequent connection of the disease with a variety of stressful influences, the presence of emotionally colored complaints in a child and especially in adolescents in the absence of objective signs of cardiac pathology, the presence of recurrent vegetative-vascular crises, normal values of laboratory parameters of inflammatory activity, a distinct effect of sedatives.

Functional cardiopathies sometimes have to be differentiated from recurrent protracted rheumatic heart disease in middle-aged women who were mistakenly diagnosed with rheumatism in childhood. The absence of a formed heart defect with indications of frequent "attacks of rheumatism" in childhood and the predominance of subjective manifestations over objective manifestations in the clinical picture of the disease make it possible to diagnose functional cardiopathy.

Non-rheumatic carditis have their own clinical symptoms and are characterized by a number of features: the presence of previous allergic diseases, a fairly frequent and close relationship with a predominantly viral nasopharyngeal infection, the subsequent short "light" interval, the abundance and persistence of cardiac complaints, the presence of symptoms of asthenia, vegetative dystonia, thermoregulation disorders in the onset of the disease, changes in the ECG, mainly in the form of extrasystole; minimal shifts in laboratory parameters of inflammatory activity, slow positive dynamics of clinical and electrocardiographic changes under the influence of therapy, i.e., their torpidity.

Idiopathic mitral valve prolapse (MVP) is characterized by a peculiar symptom complex: the prevalence of girls who often have such phenotypic features as asthenic physique, gracefulness, signs of dysplasticity of connective tissue, accidental detection of heart pathology, mainly in school and adolescence during medical examination when adolescents are transferred to an adult clinic ; presence of cardiac complaints, detection of the classic triad of changes - apical systolic murmur, systolic clicks and typical electrocardiographic changes. Attention is drawn to the qualitative characteristics of systolic murmur in MVP. This is a late systolic murmur, better detected in the standing position, significantly reduced lying, increasing after exercise.

The MVP syndrome is reflected during echocardiographic examination in the form of prolapse of the posterior, less often the anterior, or both mitral cusps. In contrast to the rheumatic process, MVP syndrome is characterized by the persistence of cardiac changes.

Often, infective endocarditis is a source of misdiagnosis when recognizing recurrent

rheumatic heart disease against the background of a formed heart defect, especially aortic (and in children and primary rheumatic heart disease). The latter is characterized by: the presence of weakness, sweating, chills, pain in bones and muscles, arthralgia, arthritis is possible. Infective endocarditis, as a rule, is accompanied by prolonged intermittent fever with chills, sweats, pallor of the skin, the appearance of Lukin's symptom, a tendency to thromboembolism, vasculitis, splenomegaly. Anemization, gammaglobulinemia, and the detection of bacteremia play an important role.

The most frequent masks of activity in the adult contingent of patients can be congestive heart failure, intercurrent infection, thromboembolism of small branches of the pulmonary artery, rhythm disturbances.

Certain congenital heart defects are also included in the range of differentiable conditions in children.

*Chorea.*Functional tics are most often the source of diagnostic errors in the recognition of chorea in children. The clinical picture of the latter is varied. Their feature should be considered the stereotyped movement. The most frequently observed tics of the face, involving various of its muscles and manifested by twitching or frowning of eyebrows, blinking, closing the eyes, twitching the wings of the nose, stereotypical chewing movements, sniffing the nose, etc. The course of tics is often chronic, possibly spontaneous cessation.

The range of differentiable states also includes hyperkinesis that occurs with systemic lupus erythematosus, thyrotoxicosis, brain tumors, and antiphospholipid syndrome. For this reason, these conditions should be included in the circle of differentiable diseases.

Treatment... Comprehensive treatment of rheumatism includes, possibly, the earlier prescription of combined drug therapy aimed at individual pathogenetic links and restoration of the impaired reactivity of the body, as well as a stubborn fight against streptococcal infection, both in the active and inactive phases of the disease.

The main task of complex therapy should be considered not only to suppress the activity of the rheumatic process, but also to prevent the formation of heart disease in patients with primary rheumatic heart disease, i.e., to achieve practical recovery of the patient. In this regard, the greatest prospects open up in childhood, when the disease begins. In patients with recurrent carditis, such therapy involves restoring the general condition and preventing the progression of existing heart changes.

Domestic clinicians have formulated the main links of the complex staged therapy of rheumatism, which includes: 1) inpatient treatment; 2) follow-up care in a local rheumatological sanatorium; 3) dispensary observation.

In our country, the method of complex therapy proposed by A.I.Nesterov has found wide application, including the simultaneous administration of small doses of GCS in combination with non-steroidal (NSAID) drugs. This combination therapy leads to good therapeutic results, while reducing the toxic effect of drugs on the patient's body.

The study of the mechanism of action of antirheumatic drugs showed that non-steroidal drugs are characterized by qualitatively special points of application in the body that are absent in hormones, due to which they not only enhance the action of hormones, but expand the sphere of their influence.

The initial daily doses of corticosteroids fluctuate depending on the severity of the condition, the severity of changes in the heart and the age of the child within the following limits: prednisolone - from 15 to 25 mg, triamsinolone - from 10 to 16 mg, dexamethasone - from 1.5 to 3.75 mg. The initial daily dose of GCS is prescribed at the rate of 0.7-0.8 mg, but not more than 1 mg of prednisolone per 1 kg of the child's weight, taking into account the physiological biorhythm of the adrenal cortex; it is recommended to use 1/2 - 1/3 of the daily dose of the drug in the morning. In recent decades, prednisone has been preferred.

For adults, prednisone is prescribed at the rate of 20-30 mg per day. The concept of treatment is the same. The initial dose of corticosteroids is gradually reduced (every 5-7 days by 2.5 mg) and is completely canceled. The course of treatment with corticosteroids lasts an average of 1.5

months, after which for 1 month. patients receive non-steroidal anti-inflammatory drugs.

Taking into account the streptococcal etiology of rheumatism and the possibility of exacerbation of foci of chronic infection, penicillin or its analogues is prescribed simultaneously with GCS for the first 10-14 days at 750,000-1,000,000 IU per day for children and 1,500,000 IU for adults. In the presence of multiple and often exacerbating foci of infection, the course of penicillin therapy is lengthened, and according to indications, patients are already in the hospital transferred to bicillin injections.

Taking into account the bright anti-inflammatory and antiallergic effect of GCS, all authors unanimously come to the conclusion that hormonal preparations are indicated for patients with a pronounced exudative component of inflammation, that is, with pronounced carditis, with a maximum or moderate degree of activity of the rheumatic process, with acute and less often - the subacute course of the disease.

When choosing one or another method of treatment, the degree of heart damage is of great importance. So, if the patient has a bright and moderately pronounced carditis, the advisability of using complex hormonal and drug treatment is beyond doubt. At the same time, in patients with diffuse endomyocarditis, often with a tendency to a protracted course, there is a need to increase the initial daily dose of hormones and lengthen the course of treatment with them.

With a minimal degree of activity of the rheumatic process, sluggish or latent course of the disease, hormonal drugs do not have pronounced therapeutic activity. With these options for the course, the use of NSAIDs is more justified.

Along with the bright positive effect of steroid hormones, many authors note their possible side effects, the clinical signs of which are varied and are expressed by a transient increase in blood pressure, excessive fat deposition, hypertrichosis, skin changes (dryness, acne, age spots, etc.), menstrual disorders cycle, changes in the functions of the nervous system and gastrointestinal tract, etc.

In the treatment of protracted and continuously recurrent forms of rheumatism, as well as primary rheumatic heart disease with damage to the valve apparatus, drugs of the quinoline series are widely used: delagil, plaquenil.

Delagil is prescribed depending on the age of the child in the following doses: children from 3 to 7 years old - 0.06-0.08 g each, from 7 to 10 years old - 0.08-0.25 g each, over 10 years old - 0.125 g each -0.25 g; adults - 0.25 g per day. The drug will be given once a day after dinner. These drugs are used simultaneously with hormones or are connected to the ongoing therapy later, when a tendency towards a protracted course of the process is revealed. The duration of their intake is from several months to 1-2 years. Plaquenil is preferred in children.

Along with corticosteroids in the treatment of rheumatism, the isolated prescription of various nonsteroidal drugs is widely used.

The daily dose of indomethacin is prescribed at the rate of 2-3 mg per 1 kg of the child's body weight with an even distribution of c. throughout the day. For adults, indomethacin is prescribed in a dose of 100 - 150 mg. The course of treatment is 1-1.5 months, if necessary, it is extended to 3-5 months. until the indicators of inflammatory activity are completely normalized.

Diclofenac sodium (Ortofen, Voltaren, etc.) is prescribed at the rate of 2-3 mg per 1 kg of the child's body weight per day, for adults 100-150 mg. The course of treatment lasts 1-1.5 months. The therapeutic effect can be regarded as good.

Studies assessing the therapeutic efficacy of various antirheumatic drugs (corticosteroids, indomethacin, voltaren) have shown that non-steroidal anti-inflammatory drugs for rheumatism in children (as opposed to adults) are noticeably inferior to hormonal therapy, especially in the presence of carditis of varying severity.

It should be emphasized that there is still no specific remedy in the fight against rheumatism. Widely used antirheumatic drugs should only be considered pathogenic.

One of the important components of therapy in a hospital setting, especially in childhood, is the remediation of streptococcal foci. Chronic tonsillitis is most often diagnosed in children. Conservative treatment for inflammation of the tonsils does not always lead to the desired result.

With decompensated chronic tonsillitis, it is necessary to resort to tonsillectomy, which is carried out in the subacute period of the disease, on average after 2-2.5 months. from the start of the attack.

The second important link in the comprehensive rehabilitation treatment of patients with rheumatism is a local rheumatological sanatorium for children and a cardiological sanatorium for adults. The task of the sanatorium is to achieve a final decrease in activity and full compensation of the rheumatic process, as well as restoration of the functional capacity of the cardiovascular system, by applying an appropriate therapeutic and motor regimen, a number of therapeutic and preventive measures.

Patients entering the sanatorium directly from the hospital are recommended to continue using antirheumatic drugs for one month, and according to indications, a longer period. Patients with chronic foci of infection are sanitized.

The third stage of complex rehabilitation therapy is dispensary observation of patients who have had rheumatic heart disease, including regular periodic examination, the appointment of general health-improving measures, hardening procedures, dosed physical exercises, the implementation of secondary prevention of relapse of the disease.

When the activity of the rheumatic process subsides (6-8 months after the attack), spa factors play an important role in the complex of rehabilitation measures.

At the stage of prophylactic medical examination, the issues of working capacity and employment of patients are resolved; together with cardiac surgeons, the issues of surgical correction of defects are discussed.

Observations carried out by a number of researchers have shown that a timely started complex hormonal and drug therapy, followed by the use of true staged treatment and further preventive measures, contributes to the overwhelming majority of patients with primary rheumatic heart disease (80-85%) not only suppressing the activity of the rheumatic process, but also preventing the formation of a defect. heart, that is, practical recovery or complete rehabilitation, which primarily applies to a sick child. In patients with recurrent carditis, this therapy helps prevent the progression of existing heart changes.

All this testifies to the indisputable success achieved in the treatment of rheumatism in children, however, the lack of a therapeutic effect in a number of patients with primary and recurrent rheumatic heart disease, as well as the formation of valvular heart disease in 1/5 of patients who have undergone primary rheumatic heart disease, as well as a large percentage of acquired defects. hearts in adults are forced to look for new ways to further improve therapeutic measures.

Prevention. According to the accumulated long-term experience of rheumatologists, existing methodological instructions, as well as WHO recommendations (1989), the program for the prevention of rheumatism and the fight against relapses of the disease includes primary and secondary prevention.

Primary prevention includes:

1. Measures to increase the level of natural immunity, primarily children and their adaptive mechanisms. These include measures to ensure the correct physical development of the child: hardening from the first months of life; high-grade fortified food; maximum use of fresh air; rational physical education and sports; combating overcrowding in dwellings, schools, children's institutions, vocational schools, carrying out especially a complex of sanitary and hygienic measures that reduce the possibility of streptococcal infection of groups, especially children.

2. Vigorous measures to combat streptococcal infection. This is the treatment of upper respiratory tract infections caused by group A streptococci.The most effective remedy is penicillin, which is prescribed parenterally in 750,000 units for preschoolers, 1,000,000 - 1,500,000 units for school-age patients and 1,500,000 - 2,000,000 units for adults within 10-14 days or during the first 5 days, followed by the introduction of Bicillin-5 at a dose of 750,000 - 1,500,000 IU per day twice with an interval of 5 days. For oral administration, the dose of phenoxymethylpenicillin or its analogs: oxacillin, ampicillin is 500,000 IU per day for preschool children and 1,000,000 IU for school-age patients and adults.

According to the WHO recommendations (1989), an effective drug in the treatment of nasopharyngeal infection is oral acid-fast penicillin - V-phenoxymethylpenicillin - smallpox, which is prescribed in the same doses as phenoxymethylpenicillin. For patients with allergies, erythromycin is an acceptable alternative. Other broad-spectrum antibiotics (eg, cephalosporin) can also effectively eliminate group A streptococcus from the upper respiratory tract.

A new direction in the primary prevention of rheumatism is the development of methods for predicting the disease.

In this regard, it should be considered extremely important to identify persons predisposed to the development of rheumatic fever and to conduct targeted prophylaxis in them.

In recent years, the concept of risk factors has been successfully developed in rheumatology, which serves as the basis for the implementation of prevention (Benevolenskaya L.I. et al., 1989). According to these authors, the risk factors for the development of rheumatism include:

- the presence of rheumatism or diffuse connective tissue diseases, as well as congenital connective tissue inferiority in first-degree relatives;
- female;
- age 7-15 years;
- suffered acute streptococcal infection and frequent nasopharyngeal infections.

A number of researchers attribute the carriage of some genetic markers to risk factors. According to the observations of N.A. Shostak (1995), the detection of the B-cell marker D8 / 17 in healthy individuals and primarily in the relatives of the proband is a specific marker of susceptibility to rheumatism.

The formation of risk groups makes it possible to narrow the circle of persons subject to primary prevention as much as possible. Dispensary observation of them with the implementation of a set of measures will be the basis for reducing the primary incidence of rheumatism.

Secondary prevention, aimed at preventing relapses and progression of the disease in children and adults who have had rheumatism, consists in the regular administration of bicillin (prolonged action penicillin).

By now, a great deal of experience has been accumulated, indicating the high effectiveness of bicillin for the prevention of recurrence of rheumatism in children. The most optimal is year-round prophylaxis carried out monthly. The latter is assigned to all children who have had reliable rheumatism during the previous 5 years. Year-round prophylaxis is carried out with the help of Bicillin-5 at a dose of 1,500,000 IU once every 4 weeks. school children, adolescents and adults. For preschool patients, Bicildin-5 is administered at a dose of 750,000 IU once every 2 weeks.

According to the current instructions for the prevention of recurrence of rheumatism and WHO recommendations (1989), patients at high risk of recurrence of the rheumatic process, Bicillin-5 should be administered 1 time in 3 weeks. in a dose of 1 500 000 IU for school-age children and adults, and 750 000 IU 1 time in 10 days for children of preschool age.

One of the promising long-acting antibiotics is benzathine benzylpenicillin - retarpen, oxtencillin.

For the implementation of secondary prevention, daily oral administration of antibiotics (penicillin and its analogues) is also possible.

Taking into account that the largest number of relapses occurs in the first 5 years after the previous attack, the duration of secondary prevention is at least 5 years and is set individually for each patient. Children who have had primary rheumatic heart disease (arthritis or chorea) without heart damage are given year-round prophylaxis over the next 3 years, and seasonal (in spring and autumn) prophylaxis for the next 2 years. Patients who have had primary or recurrent rheumatism with heart disease, especially in the presence of signs of a developing or formed heart defect, year-round prophylaxis should be carried out for at least 5 years, for children - up to 18 years of age, and, if necessary, longer.

For pregnant women who have had rheumatism or have active manifestations of it, Bicillin-5 is prescribed from 8-10 weeks of gestation until delivery; the duration of prophylaxis in the postpartum period depends on the activity and characteristics of the course of the rheumatic

process.

Simultaneously with the implementation of secondary prophylaxis for patients with rheumatism with acute respiratory infections, tonsillitis, pharyngitis, it is recommended to carry out current prophylaxis. The latter provides for the appointment of a 10-day course of treatment with penicillin. Penicillin is also prescribed to patients before and after tonsillectomy and other surgical interventions.

According to numerous observations, during bicillin prophylaxis in 0.7-5% of patients, side reactions occur, mainly in the form of allergic manifestations. Among the latter, anaphylactic shock is extremely severe, due to which all precautions must be taken in persons with hypersensitivity to penicillin.

The accumulated long-term experience shows that bicillin prophylaxis along with a complex of other measures is a highly effective means of preventing recurrence of rheumatism in children and adults.

AORTIC FAULTS

Insufficiency of the aortic valve

Etiology... Rheumatic fever. Infective endocarditis. Syphilis. Atherosclerosis. Systemic lupus erythematosus. Rheumatoid arthritis. Prolapse. Injury.

Hemodynamic disorders... During diastole, there is a reverse flow of blood from the aorta to the left ventricle. If regurgitation is insignificant, significant hemodynamic disturbances do not occur. With severe regurgitation, the left ventricle is overfilled with blood from the left atrium and its additional portion from the aorta. Loading the volume of the left ventricle stretches the muscle fibers. In accordance with the Frank-Starling law, the force of ventricular contractions increases, which, provided that the myocardium is in good condition, leads to an increase in systolic ejection.

The left ventricle operates in a hyperfunctional mode, which leads to hypertrophy of cardiomyocytes with their subsequent degeneration. A short period of tonogenic dilatation of the left ventricle with an increase in the outflow pathway is quickly replaced by a period of myogenic dilatation with an increase in the inflow path. There is an expansion of the cavity of the left ventricle, a relative insufficiency of the mitral valve is formed. Left ventricular heart failure with hypertension of the pulmonary circulation develops. Due to the resistance load, the right ventricle undergoes stages of hypertrophy with tonogenic dilatation, and then dystrophy of myogenic dilatation. Symptoms of right ventricular heart failure appear.

Mention should be made of the reflex expansion of peripheral arterioles due to massive irritation of the aortic and carotid zones of the baroreceptors with a large volume of blood ejected by the left ventricle during systole. The reflex is biologically expedient, because of its existence, the end-diastolic pressure in the aorta decreases, and this contributes to an increase in systolic output.

Since the main role in the compensation of the defect is played by the powerful left ventricle, heart failure in such patients develops late. However, once it has arisen, decompensation immediately becomes refractory to therapy due to depletion of compensatory-adaptive mechanisms.

Clinic. At an outpatient appointment, the doctor may meet with the following options for complaints of patients with aortic insufficiency:

- a feeling of pulsation in the head, in the vessels of the neck. This symptom complex is caused by sharp changes in blood pressure during a one-cardiac cycle;

- tinnitus, dizziness with a sudden change in body position, transient visual disturbances, less often cerebral syncope with a short-term fainting. The listed symptoms occur with a significantly pronounced valve defect with a large volume of regurgitation, which makes compensatory reflex reactions inconsistent, as a result of which the blood filling of the cerebral vessels during diastole becomes inadequate to metabolic demand;
- cardialgia of various types. Pain in the region of the heart is often aching, pulling, prolonged. They are explained by relative coronary insufficiency due to inadequacy of blood flow to a large mass of hypertrophied myocardium;

- shortness of breath of varying severity up to paroxysmal, tachycardia. These are symptoms of left ventricular heart failure. Our own experience shows that patients with aortic insufficiency rarely live to see the development of biventricular heart failure.

Finally, in many patients with mild aortic valve insufficiency, complaints may be completely absent or limited to a feeling of pulsation in the vessels of the neck, head and palpitations during physical exertion. These symptoms are characteristic not only of aortic insufficiency, but also of hyperkinetic heart syndrome in other diseases. They can occur in healthy, detrained individuals, in athletes at submaximal loads. They are caused by massive irritation of the aortic and carotid reflex zones and adequate peripheral vasodilation.

On examination- moderately pronounced pallor, in the later stages in combination with acrocyanosis. Musset's symptom is relatively specific for this defect - head shaking to the beat of the pulse, "dance of carotids", pulsation of the pupils, pulsation of the uvula, pulsation of the vessels of the nail bed - Quincke's capillary pulse.

The left ventricular impulse is visible to the eye, displaced in the 6-7th intercostal space. On palpation, it is strong, uplifting, its domed area increases to 6-8 cm2. The push is determined in 6 - 7 intercostal spaces. Behind the xiphoid process, the pulsation of the aorta is palpable.

Percussion data. The characteristic aortic configuration of the heart with an accentuated "waist" (heart in the form of a "duck" or "boot"). At later stages - mitralization of the heart with a shift of the upper border upwards, right - to the right. Formation of a "bull heart".

Auscultation. The first tone at the apex is quiet due to the prolapse of the aortic valve composite. Weakening of the 2nd tone in the aorta for the same reason. At the apex of the heart, a pathological 3rd tone is often heard due to the stretching of the left ventricle at the beginning of diastole ("blow" of a large blood volume).

Protodiastolic murmur on the aorta, in the Botkin area, at the apex of the heart - a classic decrescendo regurgitation murmur associated with the 1st tone. Typically, the murmur is conducted along the blood stream from the aortic aortic point down and to the left. Austin-Flint functional diastolic murmur is heard at the apex of the heart in the meso-diastole due to eddies of blood currents from the aorta and from the left atrium or in the pre-systole due to the relative narrowing of the left atrioventricular opening by the mitral valve leaflet, which takes a horizontal position due to the high pressure on it from the blood flow from the aorta than from the left atrium. Misinterpretation of this noise is a common source of overdiagnosis of mitral stenosis.

A systolic murmur in the aorta is associated with two causes. The first is the swirling of blood in the aorta due to its expansion. I.A.Kassirsky considered the second reason more significant. These are swirls of blood around the compacted short, deformed valves. Systolic murmur on the aorta with "pure" aortic insufficiency is so constant that IA Kassirsky aptly designated it as an accompanying one.

A systolic murmur at the apex of the heart can be wired from the aorta or be a murmur of relative mitral regurgitation.

The pulse is fast and high. Blood pressure - high systolic, low diastolic, high pulse. At

auscultation of blood vessels - double tone of Traube, double noise of Vinogradov - Durozier.

X-ray examination... In dorsoventral and oblique projections - bulging and lengthening of the left ventricular arch, rounding of the apex. Deep, high-amplitude pulsation of the left ventricle and aorta. The aortic shadow is dilated.

Electrocardiogram. Classic syndrome of left ventricular hypertrophy: RV5.6 wave, SV1.2 wave, depression of the S interval - TV5.6; shift of the transition zone to the right; tooth TV5,6 biphasic or negative.

Phonocardiogram... Decrease in the amplitude of the 2nd tone on the aorta, 1st tone at the apex. 3rd tone at the top. Diastolic murmur in the aorta, in the Botkin's zone, at the apex of the type of decreasing, starting immediately after the 1st tone. "Accompanying" systolic murmur over the base of the heart takes 1/3 - 1/2 systole. It is low-amplitude, decreasing. At the apex there is a systolic murmur of relative mitral insufficiency associated with the 1st tone, and diastolic, more often presystolic (not increasing to the 1st tone!) Austin-Flint murmur.

Echocardiogram. An increase in the size of the cavity of the left ventricle, the ascending aorta.

Assessment of the degree of aortic regurgitation (N.M. Mukharlyamov et al.).

1st degree.A slight expansion of the border of the heart to the left (by 0.5 cm), increased apical impulse, pulsation of the carotid arteries. According to auscultatory-phonocardiographic data, there is a protodiastolic murmur of low intensity at the Botkin-Erb point, often determined only by auscultation (PCG may not be recorded). ECG is normal, less often with signs of left ventricular hypertrophy in the corresponding leads. On the echocardiogram, the anteroposterior size of the left ventricle is normal or slightly increased (systolic up to 4.5 cm, diastolic up to 6 cm). Increase in the amplitude of contraction of the interventricular septum and the wall of the left ventricle.

2nd degree. Expansion of the border of the heart to the left and down (by 0.5 - 1.5 cm), increased pulsation of the heart, carotid arteries, "capillary pulse". According to auscultatory-phonocardiographic data, there is a diastolic murmur starting immediately after the 2nd tone and spreading throughout the entire diastole, of medium intensity at the Botkin-Erb point and in the second intercostal space to the right of the sternum, the 2nd tone on the aorta may be moderately weakened. On the ECG, signs of left ventricular hypertrophy, more often in the form of a combination of an increased amplitude of the tooth in the corresponding leads with changes in the terminal part of the ventricular complex. On the echocardiogram, an increase in the anteroposterior size of the left ventricle (systolic up to 5.5 cm, diastolic up to 7 cm). A pronounced increase in the amplitude of contraction of the interventricular septum and the wall of the left ventricle.

3rd degree. Significant expansion of the borders of the heart to the left and downward (more than 2 cm); pronounced left ventricular impulse, "carotid dance", capillary pulsation and other characteristic symptoms of this defect. According to auscultatory-phonocardiographic data, an intense continuous diastolic murmur in all points of the heart, most pronounced in the aorta. 2 tone is sharply weakened. On the ECG, signs of left ventricular hypertrophy in the form of a combination of an increased amplitude of the R wave in the corresponding leads with changes in the terminal part of the ventricular complex. Echocardiographic signs of significant dilatation of the left ventricular cavity (an increase in anteroposterior size in systole - more than 5.5 cm, in diastole by more than 7 cm), a pronounced increase in the amplitude of movement of the interventricular septum and ventricular wall.

Insufficiency of the aortic valve is characterized by the appearance of oscillations of the anterior cusp of the mitral valve, caused by blood regression. However, there is usually no correspondence between the severity of the oscillation and the degree of aortic regurgitation.

Differential diagnosis... In case of insufficiency of the pulmonary artery valve, a protodiastolic murmur is heard at the base of the heart, however, unlike the murmur of aortic insufficiency, its epicenter is located in the 2nd - 3rd intercostal spaces to the left of the sternum.

Correct diagnosis helps to put other symptoms of pulmonary valve insufficiency: right ventricular heart failure syndrome, epigastric pulsation, displacement of the right border of relative cardiac dullness to the right, electrocardiographic signs of right ventricular hypertrophy.

Diastolic murmur of relative insufficiency of the pulmonary artery valve (Graham-Still murmur) is mild in nature, of medium intensity, it is better heard in the 2nd - 3rd intercostal spaces to the left of the sternum, often accompanied by systolic murmur of low and medium intensity. The most common cause is mitral stenosis with pulmonary hypertension. Radiographically, such patients show an enlargement of the pulmonary artery. In addition to mitral stenosis, Graham-Still murmur can be heard in other diseases accompanied by hypertension of the pulmonary circulation: chronic nonspecific lung diseases, primary pulmonary emphysema, Aerz-Arrilag disease, congenital heart defects. We observed a patient with a long-term tuberculous history, in whom, against the background of post-tuberculous pneumosclerosis, emphysema of the lungs developed a picture of severe respiratory failure of the restrictive type, right ventricular heart failure. Diastolic and systolic murmurs were heard on the base of the heart. The diastolic murmur was so intense that it was the reason for the exclusion of congenital heart defects. Autopsy revealed aneurysmal dilatation of the pulmonary artery with relative insufficiency of the pulmonary valve. This confirmed the correctness of the lifetime interpretation of noise as Graham-Still noise. Autopsy revealed aneurysmal expansion of the pulmonary artery with relative insufficiency of the pulmonary valve. This confirmed the correctness of the lifetime interpretation of noise as Graham-Still noise. Autopsy revealed aneurysmal dilatation of the pulmonary artery with relative insufficiency of the pulmonary valve. This confirmed the correctness of the lifetime interpretation of noise as Graham-Still noise.

Mitral stenosis with protodiastolic murmur beginning with a click of the mitral valve opening must be distinguished from aortic regurgitation with protodiastolic murmur, 3rd tone. Mitral stenosis occurs with signs of left atrial and right ventricular hypertrophy, aortic insufficiency is accompanied by left ventricular hypertrophy. Diagnostic difficulties are eliminated after a thorough analysis of the phonocardiogram, echocardiographic examination.

Hyperkinetic cardiac syndrome is characterized by a feeling of pulsation in the head and neck. With it, a fast and high pulse, high pulse pressure are found. A systolic murmur from the base of the heart is conducted into the carotid arteries. However, there is no direct sign of aortic insufficiency - a diastolic murmur in the aorta.

Etiological diagnosis...

Infective endocarditis occurs more often in middle-aged men. History of rheumatic fever, heart surgery. Aortic insufficiency is accompanied by fever, which is relieved by high doses of antibiotics in combination with low and medium doses of glucocorticoids. Splenomegaly is common. With a long course of the disease - exhaustion, "drum fingers", thromboembolism, kidney and brain infarctions, pulmonary embolism.

Tertiary syphilis occurs with damage to the ascending aorta and aortic valves. SV Shestakov back in the 60s, paid attention to the peculiarity of the clinical symptoms of aortic insufficiency of syphilitic genesis. This is the preservation of the second tone above the aorta due to an increase in the vibration of its ascending section and the rarity of peripheral symptoms (rapid pulse; "carotid dance") due to the destruction of the aortic reflex zone as a result of specific inflammation. X-ray data are very characteristic - expansion of the ascending aorta, signs of its aneurysm.

"Pure" aortic insufficiency of rheumatic genesis is relatively rare. More often in the outcome of rheumatic fever, a combined aortic defect is formed. It is possible to think about aortic insufficiency in rheumatism if it is possible to identify direct signs of this disease - carditis, polyarthritis, combined mitral heart disease, laboratory markers of the transferred and (or) current streptococcal infection caused by group A beta-hemolytic streptococcus.

Ankylosing spondylitis (ankylosing spondylitis) and rheumatoid arthritis with visceritis have a fairly characteristic clinical picture. The detection of aortic insufficiency syndrome in such patients makes the etiological diagnosis reliable.

In systemic lupus erythematosus, aortic valve insufficiency is usually the outcome of

Liebman-Sachs endocarditis. Less commonly, the defect is formed as a result of myxomatous degeneration of the aortic tissues, thinning of the valve leaflets. It is necessary to think about the lupus genesis of aortic insufficiency if the clinical picture of the defect is detected in a woman of childbearing age without rheumatic fever and infective endocarditis in history, with "unmotivated" hyperthermia, benign polyserositis, nephropathy, "butterfly" on the face, capillaritis, positive LE-phenomenon. Fever, visceritis are controlled by large doses (60 - 80 mg / day) of glucocorticoids.

Atherosclerotic aortic insufficiency is diagnosed in elderly people, as a rule, for a number of years suffering from ischemic heart disease and hypertension. The second tone on the aorta in such patients is preserved and sometimes even strengthened due to its compaction.

In case of a traumatic defect, there must be a causal situation in the anamnesis (car accident, fall from a height). The defect is diagnosed on the basis of a clear chronological relationship between trauma and the appearance of a diastolic murmur in the aorta.

Aortic valve prolapse can be associated with mitral valve prolapse, but it can also be isolated in Marfan syndrome. The clinical presentation of aortic insufficiency in a patient with the typical appearance characteristic of Marfan syndrome makes the diagnosis of aortic valve prolapse probable. To verify the diagnosis, echocardiography is necessary, which reveals the displacement of the leaflet during diastole towards the outflow tract of the left ventricle relative to the line drawn from the place of attachment of the leaflets of the aortic valve to the fibrous ring of the aorta (KI Korytnikov, 1986).

We had to observe patient C, 53 years old, with a dissecting aneurysm of the ascending aorta, who, after an attack of severe precordial pain, developed a diastolic murmur in the aorta. The patient's typical appearance for Marfan syndrome, the clinical picture of the disease made it possible to diagnose a rupture of the prolapsed aortic valve cusps. At the section, the diagnosis was confirmed.

Treatment... Conservative treatment is carried out on a syndromic basis. With heart failure - glycosides, saluretics, peripheral vasodilators, nitrates. Indications for surgical treatment - aortic valve replacement with a ball prosthesis: left ventricular heart failure, cerebral syncope, ECG - left ventricular hypertrophy syndrome in combination with a decrease in diastolic pressure to 40 mm Hg. Art. and below.

STENOSIS OF THE AORTIC ORAL

Etiology. Rheumatic fever. Atherosclerosis. Congenital malformation - subvalvular, valvular, supravalvular stenosis.

Violation of hemodynamics... In healthy people, the diameter of the aortic opening is 30 + _5 mm. Hemodynamic disturbances appear when the orifice diameter is less than 20 mm. Stenosis of the aortic ostium results in resistance loading during left ventricular systole. The left ventricular myocardium operates in a hyperfunctional mode. This leads to its tonogenic dilatation with an increase in the outflow pathway, from the apex to the orifice of the aorta. Dystrophic changes gradually develop in the myocardium of the left ventricle with its myogenic dilatation. With myogenic dilation, the inflow path of the left ventricle increases, from the mitral valve to the apex. Formed relative insufficiency of the mitral valve. Regurgitation of blood from the left ventricle to the left atrium leads to its hypertrophy, dilatation and decompensation, stagnation in the pulmonary circulation. In some cases,

With stenosis of the aortic orifice, coronary blood flow decreases. This is caused by a low systolic ejection into the aorta, high intramyocardial pressure, a mismatch between the oxygen demand of the hypertrophied myocardium and the real possibilities of blood supply.

Clinic. With mild aortic stenosis for many years, patients' complaints and objective signs of heart failure may be absent. With moderate and pronounced stenosis, the doctor can meet with three

groups of patients' complaints. Approximately half of the patients have cardialgias of various types. Pain in the region of the heart can be angina pectoris or prolonged aching. They are caused by absolute or relative insufficiency of coronary blood flow. The second group of complaints is caused by low systolic output. This is a headache, dizziness, peculiar episodes of "lightheadedness" with a feeling of weakness, instability, dizziness, fear of losing consciousness. The third group of complaints is the symptoms of left ventricular heart failure. These are shortness of breath and palpitations during exercise, weakness, fatigue, at later stages - paroxysms of nocturnal dyspnea. In rare cases, symptoms of left ventricular failure can be combined with signs of right ventricular - pain in the right hypochondrium, swelling of the legs.

*On examination*patients draw attention to the pallor of the skin (due to a small shock ejection and compensatory vasoconstriction). Visually and palpation is determined by a strong, displaced to the left, left ventricular impulse. In most patients, systolic tremors are palpable in the 2nd intercostal space to the right of the sternum. The same tremor can be detected behind the handle of the sternum, on the carotid arteries.

Percussionat the initial stages, only the displacement of the left border of relative cardiac dullness to the left is determined. At later stages, during mitralization of the defect, the upper limit of relative cardiac dullness shifts upward, - the right one - to the right.

On auscultationdetermined weakened 1st tone at the apex, 2nd tone on the aorta. The systolic murmur on the aorta is usually rough and prolonged. I.A.Kassirsky and V.E. Nezlin described it as scraping and rumbling. The place of the best listening to the noise is the second intercostal space to the right of the sternum. However, in some patients, the noise has an epicenter under the handle of the sternum or in the 1st intercostal space on the right. The main characterological feature of systolic murmur in the aorta is its conduction through the blood flow to the carotid arteries. Less intense systolic murmur at the apex of the heart. It can be not only wired from the aorta, but also independent, due to the relative or organic insufficiency of the mitral valve.

The pulse in patients with severe aortic stenosis is small and slow, in the absence of heart failure, it is rare. Blood pressure: a decrease in systolic numbers, an increase in diastolic. With cardiac decompensation, symptomatic "mild" arterial hypertension often develops.

X-ray diagnostics. With "pure" aortic stenosis, due to the concentric nature of left ventricular hypertrophy, the heart does not increase sharply in diameter and does not assume the typical aortic configuration characteristic of aortic insufficiency. Stenosis of the aortic opening is characterized by lengthening of the left ventricular arch, rounding of the apex. The ascending aorta is dilated, postgenotic dilatation. A significant radiological symptom is calcification of the aortic valve.

Electrocardiogram... The horizontal position of the electrical axis. Sign of left ventricular hypertrophy.

Phonocardiogram. Decrease in the amplitude of the 1st tone at the apex of the heart, the 2nd tone on the aorta. A rhomboid or fusiform systolic murmur, not associated with the 1st tone, is most pronounced in the aorta.

Echocardiography. Structural changes and separation of the aortic valve leaflets. Increased thickness of the left ventricular myocardium.

Assessment of the severity of the defect (N.M. Mukharlyamov et al).

1st degree... Slight expansion of the border of the heart to the left and downward by 0.5 - 1 cm; increased apical impulse. According to auscultatory-phonocardiographic data, a rhomboid-shaped systolic murmur is determined with localization in the second intercostal space to the right of the sternum from small to medium amplitude with a peak in the first half of systole. ECGs are normal or with signs of slight left ventricular hypertrophy in the form of an isolated increase in the R wave in the corresponding leads. On the echocardiogram, there is a slight decrease in the degree of opening of the aortic valve cusps, a slight fibrosis of the cusps. Increase in the wall thickness of the left ventricle up to 1.2 cm.

2nd degree. Expansion of the borders of the heart to the left and down (up to 2 cm), increased diffuse apical impulse, pain in the region of the heart with moderate physical exertion, sometimes at rest. Auscultatory-phonocardiographic data are characterized by a rhomboid systolic murmur in the second intercostal space to the right of the sternum, conducted to the apex of the heart and to the vessels of the neck; average noise amplitude; the peak of the noise in the middle of the systole, 2 tone can be attenuated. On the ECG, signs of left ventricular hypertrophy, more often in the form of an isolated increase in the R wave in the corresponding leads. On the echocardiogram, a moderately pronounced decrease in the degree of opening of the aortic valve cusps, an increase in the wall thickness of the left ventricle up to 1.5 cm.

3rd degree. Significant expansion of the borders of the heart to the left and downward, increased cardiac impulse, frequent pain in the region of the heart at rest. According to auscultatory-phonocardiographic data, a rhomboid systolic murmur in the second intercostal space to the right of the sternum, conducted to all points of the heart, to the vessels of the neck, mainly to the right and to the back. Noise of large amplitude, peak of noise in the second half of systole; 2 tone is significantly weakened. Systolic tremor is determined. On the ECG, signs of severe left ventricular hypertrophy in the form of a combination of an increased amplitude of the R wave with changes in the terminal part of the ventricular complex. On the echocardiogram, a significant decrease in the degree of opening of the aortic valve cusps, an increase in the anteroposterior size of the left ventricle (systolic more than 4 cm), diastolic more than 6 cm (and myocardial thickness more than 1.6 cm).

Differential diagnosis.

Coarctation of the aortaproceeds with arterial hypertension of the upper half of the body, arterial hypotension of its lower half. A systolic murmur is heard above the aorta, carried out into the interscapular space. X-ray examination determines the expansion of the shadow of the aorta, strengthening of the shadow of the ascending aorta, weakening - descending. Usur ribs can be detected.

In cardiac surgery centers, during angiocardiography, it is possible to visualize the coarctation of the aorta and to clarify the place of narrowing.

Differential diagnostic criteria for aortic valve stenosis and hypertrophic cardiomyopathy with obstruction of the left ventricular outflow tract are given in the section "Diseases of the heart muscle".

Atherosclerosis of the aortamay be accompanied by a rough systolic murmur in the aorta with conduction to the carotid arteries. Radiographically in such patients, the aortic shadow with its calcification is revealed. On the phonocardiogram, the noise is not diamond-shaped. Echocardiography allows you to make sure that there is no damage to the valve cusps.

Treatment. Conservative treatment is carried out on a syndromic basis. With heart failure, cardiac glycosides, saluretics, peripheral vasodilators are prescribed. Anginal syndrome is an indication for the appointment of nitrates and calcium antagonists.

Surgical treatment (implantation of an artificial aortic valve) is indicated for aortic stenosis of 2-3 degrees in the presence of cerebral syncope, anginal syndrome, and left ventricular heart failure.

COMBINED AORTIC FAILURE

The etiology of the defect in the overwhelming majority of cases is rheumatic. The clinic is characterized by a combination of symptoms of the "pure" defects described above. Auscultatory symptoms - systolic ejection murmur and early diastolic aortic murmur - allow the physician-first contact to diagnose affectio aortae at an outpatient appointment. The question of the prevalence of

stenosis or insufficiency of the aortic valve is decided after a comprehensive clinical and instrumental examination of the patient in the cardiology center. Indications for surgical treatment are also determined there.

Examination of disability for valvular heart disease

A certificate of incapacity for work is issued taking into account the etiological factor (activation of a rheumatic or septic process, destabilization of the course of ischemic heart disease, etc.), the stage of heart failure, the presence or absence of arrhythmias and heart block. Organopathology, background and concomitant diseases, individually personal indicators are assessed in detail. It is obligatory to take into account the social factor - the "professional route", the duration and intensity of physical and psycho-emotional stress.

The duration of labor loss with an active rheumatic process, on average, ranges from 1.5 to 2 months. Patients with stage 2 A congestive heart failure (2 f.cl.), As a rule, are hospitalized for a period of at least 3-4 weeks, at 2 B stage (3 f.cl.), These periods are prolonged to 4-6 weeks. The terms of hospitalization are lengthened in the presence of arrhythmias, thromboembolic complications, acute heart failure, severe illnesses - diabetes mellitus, chronic nonspecific lung diseases, hepatitis, etc. If it is possible to stabilize heart failure at the level of the 1st stage (1 f.cl.), Most patients recognized as able-bodied. Persons engaged in heavy physical labor are employed through the VKK or are recognized as invalids of the 3rd group.

With chronic heart failure 2 A tbsp. (2 ph. Cells) the majority of patients, except for a narrow contingent of persons with great intellectual potential (editors, scientists, writers) are recognized as invalids of the 2nd group due to the loss of professional ability to work.

Patients with valvular heart disease requiring surgical treatment, before and after surgery, are disabled for 2-4 months. The further fate of patients is determined by the outcome of the operation. Disability issues are resolved individually, depending on the presence or absence of activation of the rheumatic process, arrhythmias, the stage of heart failure, and the patient's social status. Determining the length of stay on sick leave after cardiac surgery and expert assessment of partial or complete disability is the prerogative of specialized cardiological ICCs at cardiological dispensaries and specialized cardiological VTEKs.

Clinical examination

Patients with valvular heart disease are observed by a family doctor or a local therapist in consultation with a rheumatologist and a cardiologist on a nosological basis in the groups "rheumatic fever", "rheumatic arthritis", "ankylosing spondylitis", "systemic lupus erythematosus", etc.

Frequency of inspections - at least 4 times a year. The scope of the examination - a clinical analysis of blood, urine, x-ray examination of the chest organs, ECG, PCG, echocardiography, biochemical blood tests - acute phase reactions, glycosaminoglycans. If necessary - consultations of an ENT specialist, dentist, ophthalmologist, neurologist. According to indications - consultation of a cardiac surgeon.

The complex of therapeutic measures is determined by the underlying disease, the leading clinical symptom complexes - heart failure, arrhythmias, etc.

Criteria for the effectiveness of clinical examination: a decrease in the frequency of relapses of the disease, a decrease in the period of temporary disability, the number of patients who have become disabled.

MITRAL FAULTS

Mitral stenosis

Mitral stenosis is a common acquired heart disease. It can be observed in "pure" form or in combination with mitral valve insufficiency.

Etiology. Almost all cases of mitral stenosis are a consequence of rheumatism. Quite often, the history of such patients (up to 30-60% of cases) does not show obvious rheumatic attacks, nevertheless, there should be no doubt about the rheumatic origin of the defect.

Mitral stenosis usually develops at a young age and is more common in women.

Pathogenesis. Hemodynamic changes... In humans, the area of the left atrioventricular opening ranges from 4-6 cm2. It has a significant reserve of area, so only a decrease in it by more than half can cause noticeable hemodynamic changes.

The narrowed mitral opening serves as an obstacle to the expulsion of blood from the left atrium; therefore, a number of compensated mechanisms are activated to ensure normal blood supply to the left ventricle.

In the atrial cavity, the pressure rises (from 5 mm Hg to 25 mm Hg). This increase in pressure leads to an increase in the left atrium-left ventricle pressure difference, thereby facilitating the passage of blood through the narrowed mitral foramen. The left atrial systole is lengthened and blood flows into the left ventricle for a longer time. These two mechanisms - an increase in pressure in the left atrium and lengthening of the left atrial systole at first compensate for the negative effect of the narrowed mitral opening on intracardiac hemodynamics.

The progressive decrease in the area of the opening causes a further increase in pressure in the left atrial cavity, which simultaneously leads to a retrograde increase in pressure in the pulmonary veins and capillaries. In some patients (30%), a further increase in pressure in the left atrium and pulmonary veins due to irritation of the baroreceptors causes a reflex narrowing of the arterioles (Kitaev's reflex). This protective reflex protects the pulmonary capillaries from excessive pressure increase and sweating of the liquid part of the blood into the alveolar cavity. Subsequently, prolonged spasm of arterioles leads to the development of morphological changes. This creates a second barrier to blood flow, thereby increasing the load on the right ventricle. As a result, its hyperfunction and hypertrophy reach pronounced degrees. A significant increase in pressure in the pulmonary artery and the right ventricle makes it difficult to empty the right atrium, which is also facilitated by a decrease in the cavity of the ventricle due to its pronounced hypertrophy. Difficulty in expelling blood from the right atrium causes an increase in pressure in its cavity and the development of hypertrophy of its myocardium.

Incomplete emptying of the right ventricle during systole leads to an increase in diastolic pressure in its cavity. The developing dilatation of the right ventricle and the relative insufficiency of the tricuspid valve somewhat reduce the pressure in the pulmonary artery, but the load on the right atrium increases even more. As a result, decompensation develops in a large circle.

Clinic. From the analysis of the pathophysiological features of the defect, it follows that the clinical picture of the disease at different stages of its development will differ in some features. Nevertheless, in all patients with mitral stenosis, objective signs should be observed, depending solely on the characteristics of the valve lesion.

Complaints. If mitral stenosis is not pronounced sharply and is compensated by the increased work of the left atrium, then patients may not complain. They are able to perform quite significant physical activity. With an increase in pressure in a small circle, there are complaints of shortness of breath during exercise. Another complaint is cough, dry or with a small amount of mucous sputum, often mixed with blood. With high pulmonary hypertension, patients often complain of rapid fatigue, weakness, because there is no adequate increase in cardiac output.

With the appearance of congestion in a small circle, patients with physical exertion often

complain of palpitations. Sometimes angina pain. Their cause may be: 1) stretching of the left atrium; 2) stretching of the pulmonary artery; 3) compression of the left coronary artery by an enlarged left atrium.

Objective data.

Inspection. The appearance of a patient with moderately pronounced stenosis does not present any peculiarities. With an increase in the symptom of pulmonary hypertension, a typical facies mitralis is observed: against the background of pale skin, a sharply outlined "mitral" blush of the cheeks with a somewhat cyanotic shade.

Visually, the region of the heart bulges out - there is a "heart hump" This symptom is associated with hypertrophy and dilatation of the right ventricle and with its increased impacts on the anterior chest wall.

Noteworthy is the absence of an apical impulse, since the left ventricle is pushed aside by the hypertrophied right ventricle.

Palpation. If, after preliminary physical exertion, the patient is laid on his left side, then when holding his breath in the expiratory phase at the apex of the heart, diastolic tremor can be determined - "cat's purr". This symptom is caused by low-frequency fluctuations of blood when it passes through the narrowed mitral opening. In the 2nd intercostal space to the left of the sternum, palpation with the palm of the hand during the exhalation phase can determine the strengthening (accent) of the II tone. Nesterov BC (1971) describes the symptom of "two hammers": if the hand is placed on the heart area so that the palm is projected onto the apex, and the fingers are projected onto the area of the second intercostal space to the left of the sternum, then clapping 1 tone is felt by the palm as the first "hammer" , and the accented II tone is perceived by the fingers of the hand as a blow of the second "hammer".

In the upper part of the epigastrium, a pulsation can be observed, depending on the increased work of the hypertrophied right ventricle: during inspiration, this pulsation increases sharply, since the blood flow to the right ventricle increases.

Percussion. With percussion, the relative dullness of the heart is increased upward due to the left atrial appendage and to the right due to the right atrium.

Auscultation... It gives the most significant signs for the diagnosis, since the detected phenomena are directly related to impaired blood flow through the mitral foramen.

Tone I is amplified (clapping). This depends on the fact that in the preceding diastole the left ventricle is not filled with sufficient blood and contracts rather quickly. At the apex, the tone of the opening of the mitral valve (click of the opening) is also heard immediately after the II tone. The clapping I tone in combination with the II tone and the opening tone will create a characteristic melody at the top of the heart - "quail rhythm".

Diastolic murmur is a characteristic auscultatory symptom in mitral stenosis. The murmur is associated with the movement of blood through the narrowed mitral opening due to the pressure gradient from the left atrium to the left ventricle.

Pulse is usually not an indicator of characteristic changes. The pulse is slightly less than normal filling as a result of a decrease in cardiac output.

R-logic exploration... The purpose of this study is to more accurately determine the enlarged sections of the heart chambers, as well as to find out the state of the vessels of the small circle.

ECGturns out to be very valuable in the diagnosis of mitral stenosis and assessment of the stage of its course. The purpose of the ECG is to detect hypertrophy of the left atrium and right ventricle, the presence of rhythm disturbances.

Signs of left atrial hypertrophy: 1) two-apical P wave in lead I, AVL, V4-6; 2) in lead V1, there is a sharp increase in the amplitude and duration of the second phase of the P wave; 3) an increase in the time of the internal deflection of the P wave by more than 0.06 sec.

Signs of right ventricular hypertrophy: 1) deviation of the electrical axis of the heart to the right in combination with a shift in the ST interval and a change in the T wave in AVF, III; 2) the R

wave increases in the right chest leads, and the S wave increases in the left chest leads.

FCG- graphical recording of heart sounds and cardiac murmurs. The value of PCG increases in conditions when, during auscultation, it is difficult to attribute the audible murmur to a particular phase of the cardiac cycle.

ECHO-KG is currently essential for the diagnosis of mitral stenosis.

Diagnostics.

Direct signs: 1) clapping I tone; 2) the tone of the opening of the mitral valve (click of the opening); 3) diastolic murmur; 4) diastolic tremor (palpation); 5) ECHO-KG - signs of mitral stenosis.

Indirect signs: 1) R-logical and ECHO-KG-signs of an increase in the left atrium; 2) ECG - left atrial hypertrophy; 3) shortness of breath on exertion; 4) attacks of cardiac asthma; 5) pulsation in the epigastrium due to the right ventricle; 6) R-logical and ECHO-KG - signs of right ventricular hypertrophy.

Treatment. There are no specific conservative treatments for mitral stenosis. Circulatory insufficiency is treated according to conventional methods (cardiac glycosides, diuretics, potassium preparations). With an active rheumatic process - antirheumatic drugs, antibiotics. The surgical method of treatment is commissurotomy.

Mitral insufficiency.

There are relative mitral insufficiency, which occurs due to a variety of reasons: heart disease leading to hemodynamic overload of the left ventricle (arterial hypertension, coarctation of the aorta), dilated cardiomyopathy, left ventricular aneurysm, after extensive myocardial infarction. In all these situations, "mitralization" develops with the expansion of the cavity of the left ventricle and the annulus fibrosus. The next cause of relative mitral insufficiency is calcification of the valve ring, which can be combined with the deposition of calcium salts on the wall of the left ventricle, this process is observed in old age.

Mitral valve prolapse syndrome is an excessive movement of the mitral leaflets into the left atrial cavity during ventricular systole.

Etiology. In most cases, the cause of the organic form of mitral insufficiency is rheumatism (up to 75%), much less often - atherosclerosis, prolonged septic endocarditis.

Pathogenesis and changes in hemodynamics... Incomplete closure of the leaflets of the mitral valve causes backflow of blood from the ventricle to the atrium during ventricular systole. The degree of regurgitation determines the severity of the defect.

Due to the throwing of part of the blood into the left atrium, more than normal amount of blood accumulates in it. An excess amount of blood in the left atrium stretches its walls and, during the presystole, more than usual enters the left ventricle. The increased blood flow to the left ventricle causes dilatation and hypertrophy. The developing dilatation of the left ventricle in this case is not evidence of a decrease in its contractile function, which is a compensatory reaction to the intake of an increased amount of blood.

Left ventricular hypertrophy is not significantly developed, since the resistance exerted by the left ventricle during the expulsion of blood by it is not increased.

The left atrium also experiences volume overload as it receives an increased amount of blood. The defect is compensated for a long time by a powerful left ventricle.

Subsequently, with the weakening of the left atrium under the influence of powerful impulses of the regurgitation wave, the atrial myocardium loses its tone. The pressure in the left atrial cavity rises and then is retrogradely transmitted to the pulmonary veins. Passive pulmonary hypertension occurs.

Clinical picture... Mitral insufficiency goes through a number of stages in its

development, each of which has a certain clinical picture. At the same time, regardless of the severity and characteristics of the clinic, all patients will have direct signs of defect.

Complaints... There are no complaints at the stage of compensation for defects. With a decrease in the contractile function of the left ventricle, patients complain of shortness of breath during exercise and palpitations. With an increase in congestion and a small circle, attacks of cardiac asthma, as well as shortness of breath at rest, may appear.

Some patients develop a cough, often with an admixture of croca (hemoptysis).

With an increase in symptoms of right ventricular failure, edema and pain appear in the right hypochondrium due to an increase in the liver and a stretching of the capsule. Pain in the region of the heart, aching, stitching, pressing.

Objective research. Inspection... The patient's appearance was unremarkable. With a high degree of insufficiency and an increase in stagnation in a small circle, acrocyanosis can be noted, up to the typical facies mitralis.

When viewed and **palpation**the area of the heart of the pathology is not noted if the regurgitation is small. With significant regurgitation, there is a "heart hump" - due to severe left ventricular hypertrophy. "Heart hump" is usually left-sided localization.

Percussionthere is one degree or another increase in relative dullness to the left (dilatation and hypertrophy of the left ventricle). An increase upward due to a hypertrophied and dilated left atrium is typical for cases of severe regurgitation.

Auscultation of the heart gives the most informative signs, since changes in tones and the appearance of noise are associated with a violation of blood flow through the mitral valve.

Tone 1 is weakened or completely absent. This is due to a violation of the mitral valve slamming mechanism (absence of a "closed valve period"). In addition, the weakening of the I tone may be due to the layering of oscillations caused by the regurgitation wave on it.

The accent of the II tone over the pulmonary artery is usually moderately pronounced and occurs with the development of congestion in the small circle. Often at the apex of the heart, a third tone is heard, which is an increase in the physiological third tone of the heart. As you know, the III tone can normally be determined in young people and children. With mitral insufficiency, the III tone occurs due to the fact that the increased amount of blood coming from the left atrium increases the vibrations of the walls of the ventricle.

The most characteristic auscultatory symptom in mitral regurgitation is systolic murmur. This noise occurs due to the passage of a reverse wave of blood (regurgitation wave) from the left ventricle into the left atrium through a relatively narrow opening between the loosely closed leaflets of the mitral valve. The intensity of systolic murmur varies widely and is associated with the severity of the valve defect. The systolic murmur in the apex of the heart is best heard. However, if the patient is turned on the left side, then the place of the best listening is shifted lateral, closer to the anterior and even median axillary line. The louder and longer the systolic murmur, the more severe the mitral regurgitation. The noise is directed to the left axillary region.

Pulse and blood pressure usually does not change.

R-logic exploration... In the anteroposterior projection, there is a rounding of the 4th arc on the left contour of the heart due to dilatation and hypertrophy of the left ventricle. In addition, the enlargement of the left atrium causes the bulging of the 3rd arch of the left contour. The enlargement of the left atrium is especially clearly detected in the first oblique or left lateral projection, where this part of the heart displaces the contrasted esophagus along an arc of a large radius. The overall dimensions of the heart are increased, in cases of severe defect - to a very significant extent.

ECG.ECG - signs of left ventricular hypertrophy: 1) in V5, 6 increases the R wave; 2) in V1,2 and the S wave increases; 3) in V5.6, changes in the terminal part of the ventricular complex can be observed in the form of a downward shift of the ST interval and changes in the T wave.

FKT. Allows you to give a fairly detailed description of the systolic murmur. **ECHO-KG** - there is discordance of the anterior and posterior cusps, signs of fibrosis of the anterior cusp,

turbulent systolic blood flow in the left ventricular cavity.

Diagnostics.

Direct symptoms:

1. Systolic murmur at the apex.
2. Weakening of 1 tone and often the presence of a III heart tone.
3. ECHO-KG - turbulent systolic blood flow into the left atrial cavity.

Indirect signs:

1. Left ventricular enlargement
2. Left atrial enlargement
3. The appearance on the electrocardiogram of a high additional "regurgitation wave" due to a high ejection of blood from the left ventricle into the left atrium during systole.

Complications.

Rhythm disturbances (atrial fibrillation).

Hemoptysis and cardiac asthma are less common than with mitral stenosis and occur more easily.

Forecast. Depends on the severity of the valve defect and on the state of the myocardium.

Treatment.

There are no specific conservative treatments for mitral valve disease. Developing circulatory failure is treated according to conventional methods (cardiac glycosides, diuretics, potassium preparations).

The indication for the surgical method of treatment is the value of regurgitation of at least 40% of the stroke volume of blood. Performing an operation - mitral valve replacement.

HYPERTONIC DISEASE

Hypertension (HD) (essential arterial hypertension) is a disease whose leading symptom is arterial hypertension, not associated with any known cause.

Etiology.

The main etiological factor is recognized by many researchers as neuropsychic overstrain that occurs both after short-term acute and after long-term "chronic" effects. Primary functional disorders occur in the cerebral cortex and especially in the centers of the hypothalamic region that regulate blood pressure.

The emergence and development of hypertension largely depends on a complex of predisposing factors: heredity, imbalance of nervous processes, psychoneurosis, inflammatory processes in the brain, anatomical and metabolic disorders after a concussion, etc.

Pathogenesis

1) Hemodynamics:

The mechanisms that regulate local blood circulation and the systems that control the integrative parameters of hemodynamics form a complex mosaic, in which there are many unexplored links, interacting forward and backward connections. The higher hierarchical links of the complex chain of regulation are responsible for the integral response of the CVS, for the preservation of its functional stability and physiological economy. They provide a balance of

pressor and depressor effects. Hypertension can result from a disturbance in any of the regulation links.

2) Structural changes in the arteries:

A decrease in the caliber of small arteries as a result of smooth muscle contraction leads to a sharp deformation of the vascular wall, the internal elastic membrane gathers like an accordion, and endothelial cells are functionally impaired. With long-term preservation of a high tone, morphological restructuring takes place, fixing new relationships: a narrow lumen of a vessel and an excess thickness of its wall.

3) The main links of pathogenesis:

The development of arterial hypertension in most diseases is due to three main mechanisms at the level of the kidneys. The kidneys play a major role in the regulation of blood pressure, excreting and storing Na and water, forming pressor and depressant substances. These pathogenetic mechanisms are:

1. delay Na and pods;
2. activation of the pressor system;
3. decreased function of the depressor system.

Retention of Na and Water. In kidney disease, sodium and water retention is caused by renal impairment - a decrease in blood perfusion, glomerular filtration rate and an increase in sodium reabsorption during its passage through the nephron. A decrease in sodium and water excretion by the kidneys leads to the development of hypervolemia, an increase in the BCC, as well as an increase in the level of sodium in the vascular wall with its swelling and increased sensitivity to the pressor effects of angiotensin and catecholamines. In turn, an increase in the intravascular level leads to the accumulation of Ca in the smooth muscle cells of the vascular wall with an increase in contractility and vascular tone, followed by an increase in OPS. This mechanism, with the leading role of overhydration, hypervolemia and increased cardiac output, is of primary importance in the development of hypertension with acute HM, in acute and chronic renal failure.

Pressor system activation... The pressor system of the body is represented by the renin-angiotensin-aldosterone system (RAAS) and the sympathetic-adrenal system. This system regulates the BCC and the activity of vasoconstrictor factors. Violation of the RAAS activity can lead to an increase in blood pressure, an increase in BCC, or to a concentration of vasoconstrictor factors. The main components of the renin-angiotensin system are the enzymes renin and kininase 2 (an angiotensin converting enzyme) and hormones - angiotensin 1,2,3. Renin - is formed in the juxtaglomerular apparatus (JUGA) of the kidney glomeruli. The stimuli for increased renin secretion are lowering the pressure in the arterial system of the kidneys (shock, blood loss), hypovolemia, sodium deficiency in food, and taking urolithiasis. Under the action of renin, the hormone angiotensin 1 is formed from the angiotensinogen produced in the liver, passing under the influence of the angiotensin-converting enzyme (kininase 2) in the lungs, kidneys and plasma into angiotensins 2. Angiotensin 2 causes systemic spasm of arterioles with an increase in both OPS and renal vascular resistance, enhances sodium reabsorption, acting directly on the renal tubules, and also enhances secretion of aldosterone. Angiotensin 3 is a metabolite of angiotensin 2; it is characterized by a weakly expressed pressor effect, but to a large extent stimulates the secretion of aldosterone by the adrenal cortex. Increased plasma renin activity (ARP) plays a role in the development of hypertension in diseases in which the kidneys are functionally preserved, but the JGA region is ischemic. Renin-dependent hypertension is observed in some patients with end-stage renal failure treated with systematic hemodialysis.

Renin in the body can be found in two main forms:

- low molecular weight active renin, which is determined by radioimmunoassay;
- high molecular weight inactive renin.

The detection of high plasma renin activity in a number of patients is due to increased activation of total renin. The low activity of renin in patients with hypertension is possibly due to the fact that most of the total amount of renin in these patients is in an inactive state.

Aldosterone - the hormone of the adrenal gland zone, stimulates the reabsorption of Na + ions and the release of K ions from the body. The release of aldosterone is regulated by ACTH, the concentration of ions and RAS.

In clinical practice, increased secretion of aldosterone contributes to the development of hypertension in all cases of activation of the RAS, as well as in primary hyperaldosteronism (tumors of the adrenal glomerular zone).

The pressor effect of aldosterone is associated with its effect on the cell membrane, with an increase in its permeability to Na. Retention of sodium increases the sensitivity of the vascular wall to pressor influences.

Decreased function of the depressor system... This system includes prostaglandins, the kallikrein-kinin system. Renal prostaglandins, mainly PGE2, prostacyclins (PCI2), reduce the tone of the arteries, reduce their response to vasopressor substances, are powerful natriuretics and hydrouretics. The end products of the kallikrein-kinin system - bradykinins and kalladin - also have pronounced vasodilating properties; excretion of kallikrenin can serve as an indicator of vasodilation and activity of the natriuretic system. The death of the renal parenchyma leads to a decrease in the depressive function of the kidneys.

I. Classification by blood pressure

Category	Systolic blood pressure mm Hg	Diastolic blood pressure mm Hg
Normal blood pressure	<130	<85
The upper limit of the norm	130-139	85-89
Hypertension		
Stage I (mild)		
Stage II (moderate)	140-159	90-99
Stage III (severe)	160-179	100-109
Stage IV	180-209	110-119
(very heavy)	210 and higher	120 and higher

If systolic and diastolic pressures fall into different categories, the higher category should be selected for individual BP classification. For example, the blood pressure level is 160/92 mm Hg. should be classified as stage II, and 180/120 mm Hg. - as stage IV.

II. In the classification of arterial hypertension, adopted in Europe in 1993, the stage of arterial hypertension depends on the degree of damage to the target organs.

Stages of arterial hypertension	Target organ damage
Stage I	There are no objective signs of target organ damage
Stage II	There is at least one of the following signs of target organ damage: • left ventricular hypertrophy; • generalized or local lesion of the retinal arteries; • proteinuria and / or a slight increase in blood creatinine (1.2-2 mg / dl); • ultrasound or radiological evidence of the presence of an atherosclerotic plaque (aorta, carotid, iliac, or femoral arteries).
Stage III	The presence of a complex of signs of target organ damage: A heart: • angina pectoris, myocardial infarction;

	• heart failure. Brain: • transient violation of cerebral circulation; • stroke; • hypertensive encephalopathy. Kidneys: • plasma creatinine level> 2 mg / dl; • renal failure. Ocular fundus: • hemorrhages and exudations with or without papilla edema. Vessels: • dissecting aortic aneurysm; • occlusive lesions of the arteries.

Clinical symptoms

1. Subjective manifestations: pain and interruptions in the region of the heart, headaches, dizziness, decreased visual acuity, flickering of spots, circles, flies before the eyes, shortness of breath when walking,
2. With the development of severe cardiosclerosis and circulatory failure - acrocyanosis, pasty legs and feet, with severe left ventricular failure - attacks of suffocation, hemoptysis.
3. Blood pressure is higher than 160/90 mm Hg.
4. The pulse in the early stages of the disease is not significantly changed, in the later stages - increased filling and tension, sometimes arrhythmic.
5. The left border of the heart is enlarged, with auscultation in the initial stages - an increase in the I tone above the apex of the heart, later - its weakening, an accent of the II tone above the aorta. With an increase in heart failure - the rhythm of the gallop.
6. Clinical manifestations of brain and kidney damage in the expressed stages of the disease.

Clinical options:

1. *Hyperkinetic option*develops mainly in the early stages and is characterized by palpitations and pain in the region of the heart, a feeling of pulsation in the head, headaches; sweating, redness of the face; chills-like tremor; high but labile blood pressure; an increase in MO with a relatively small or even normal PS.
2. *Volume (sodium) dependent hyporenine variant*with signs of water retention, it is manifested mainly by swelling of the face and hands; constant dull enough intense headaches in the occipital region; numbness of fingers and toes; the connection of these symptoms and an increase in blood pressure with the intake of salt, water in the evening; more often a decrease in the content of renin, aldosterone in the blood; a distinct clinical effect of taking saluretics.
3. *Hyperrenal (angiotensin-dependent) vasoconstrictor variant* characterized by a high level of blood pressure, its stable nature, high blood levels of renin, aldosterone, angiotensin-II.
4. *Malignant variant (rapidly progressive):* manifests itself in extremely high blood pressure, resistant to conventional antihypertensive therapy, rapid progression

of severe disorders of the kidneys (development of chronic renal failure), brain (severe hypertensive encephalopathy, stroke), fundus vessels, often rapidly fatal (1-2 years after the onset first symptoms in the absence of active targeted treatment).

5. *Benign option:* characterized by slow progression, undulating alternation of periods of deterioration and improvement, damage to the heart, cerebral vessels, retina and kidneys at the stage of blood pressure stabilization; the effectiveness of treatment, a clear staging of the course; the development of complications in the later stages of the disease.

Diagnostics

Laboratory data:

1. *UAC*: with a prolonged course of hypertension, an increase in the content of erythrocytes, hemoglobin and hematocrit indicators is possible ("hypertensive polycythemia").
2. *TANK*: the addition of atherosclerosis leads to hyperlipoproteinemia of types II and IV but Fredriksen, with the development of chronic renal failure - an increase in the level of creatinine, urea.
3. *OAM*: with the development of nephroangiosclerosis and chronic renal failure - proteinuria, microhematuria, cylindruria, hypo-, isostenuria in the sample according to Zimnitsky.

Instrumental research

1. *ECG*;
2. *X-ray examination of the heart*... During the period of initial concentric hypertrophy, only the rounding of the apex of the left ventricle is revealed. With a more pronounced, but still moderate increase in the size of the left ventricle, the apex of the heart descends slightly downward, and then moves to the left. With hypertrophy and dilatation of the "inflow pathways", the left ventricle enlarges posteriorly, narrowing the retrocardial space. In the later stages, all parts of the heart are enlarged.
3. *Echocardiography* reveals an increase in the left ventricle.

HYPERTONIC CRISE (HK)

Definition

HA is one of the most frequent and severe complications of hypertension and symptomatic arterial hypertension, characterized by an acute increase in blood pressure to individually high numbers and a sharp exacerbation of the symptoms of the disease with a predominance of cerebral and cardiovascular disorders.

ON THE. Ratner (1958) identifies the following types of crises:

1. Hypertensive crisis of the first type is associated with the release of adrenaline into the bloodstream and develops more often in the early stages of hypertension, usually lasts up to 2-3 hours, relatively quickly stops. Characterized by a sharp headache, dizziness, the appearance of "fog" before the eyes, general anxiety, a feeling of heat, a feeling of pulsation and trembling throughout the body, stabbing pain in the region of the heart. The skin of the face, neck, chest becomes covered with red spots, sweat. Sometimes the crisis ends with an urgent urge to urinate. PS increases by 20-50 beats per minute, systolic blood pressure rises by 80-100 mm Hg, diastolic by 30-50 mm Hg. At the time of a crisis, a small amount of protein, erythrocytes may appear in the urine, the content of glucose and leukocytes may increase in the blood. With this type of crisis, MO (hyperkinetic type) increases significantly.

2. Hypertensive crisis of the second typeassociated with the release of norepinephrine into the bloodstream, characterized by a more gradual development, severe course, long duration (up to several days), develops mainly in the late stages of GB. Characterized by a sharp headache, dizziness, transient visual and hearing impairment, constricting pain in the heart, palpitations, often transient paresis, parasthesia, a state of stunned consciousness, confusion. Blood pressure is very high, diastolic blood pressure rises up to 140-160 mm Hg are possible, PS is significantly increased, MO can be reduced (hypokinetic crisis). After a crisis, a relatively large amount of protein, casts, erythrocytes is excreted in the urine.

3. Complicated hypertensive crisis- characterized by a sharp increase in blood pressure, acute coronary insufficiency, cardiac asthma, pulmonary edema or acute cerebrovascular accident, cerebral edema. Transient blindness, deafness, aphasia, symptoms of irritation of the meninges are possible. In the most severe cases, convulsions and loss of consciousness are possible.

A.P. Golikov (1985), in accordance with the type of hemodynamics, distinguishes:

1. **Hyperkinetic type**- characterized by an increase in cardiac output (stroke and minute volumes) with normal or decreased PS. Develops in the early stages of GB.
2. **Hypokinetic type**- characterized by a significant increase in total PS, a decrease in minute and stroke volumes. It develops in stage II-III GB.
3. **Eukinetic type**- characterized by an increase in the total PS and normal MO and develops more often in patients with hypertension II - III stage. against the background of a significantly increased initial pressure.

In order to provide effective care, it is most expedient to divide hypertensive crises into 2 large groups (Gifford et al. 1991).

Crisis I - in conditions requiring an immediate decrease in blood pressure (within 1 hour).

- hypertensive encephalopathy;
- acute left ventricular failure;
- acute aortic dissection;
- eclampsia;
- postcoronary arterial bypass;
- some cases of hypertension, combined with an increase in the level of catecholamines circulating in the blood;
- hypertension with intracerebral hemorrhage;
- acute subarachnoid hemorrhage;
- acute heart attacks (strokes) of the brain;
- unstable angina pectoris or acute period of myocardial infarction.

Crisis II - in conditions requiring a decrease in blood pressure within 12-24 hours.

- high diastolic hypertension (140 mm Hg) without complications;
- malignant arterial hypertension without complications;
- hypertension in the postoperative period.

Treatment program for hypertension

1. Elimination of negative psychoemotional and psychosocial stressful situations.

2. Non-drug methods of treatment. The effect of non-drug treatment is assessed after 2-4 months.

2.1. Health food. The most pathogenetically substantiated in hypertension is the hyponatrium diet No. 10.

The main principles are:

- anti-atherosclerotic diet;
- reducing the intake of free liquid to 1-1.5 liters. per day;

- strict compliance of the energy value of the diet with the energy consumption of the body;
- exclusion of products that excite the central nervous system and the cardiovascular system;
- reducing the content of table salt, "ideal" -2-5 g per day;
- reducing the consumption of saturated fats and enriching the diet with unsaturated fats;
- enrichment of the diet with foods containing magnesium and potassium.

2.2. Normalization of body weight.

2.3. Limiting alcohol consumption and smoking cessation.

2.4. Regular dynamic physical activity.

You should train at least 3 times a week, each time for at least 30 minutes, with a heart rate of 65-70% of the maximum.

Patients suffering from both coronary artery disease at the same time, as well as patients over 40 years of age with risk factors for ischemic heart disease, should be tested with physical activity before starting training.

2.5. Psychorelaxation, rational psychotherapy.

To combat psychoemotional stress, the following methods are used:

- behavioral therapy;
- rational psychotherapy aimed at reducing psychophysiological reactivity;
- training in the skills of an adequate response to a stressful impact;
- psycho-relaxation therapy.

2.6. Acupuncture.

Contributes to the normalization of the tone of the vasomotor center, sympathetic nervous system, endocrine system, which leads to a decrease in blood pressure.

2.7. Acupressure.

The acupressure is based on the same principle of influencing pathologically active points only with the tip of the finger.

2.8. Physiotherapy treatment.

This type of therapy for arterial hypertension is carried out differentially, depending on the stage.

2.9. Hypoxic training.

The mechanism of the hypotensive action of adaptation to hypoxia is as follows:

- decrease in total peripheral resistance;
- a decrease in the activity of the renin-angiotensin-aldosterone system;
- increased blood levels of atrial natriuretic hormone, which inhibits the activity of the renin-angiotensin-aldosterone system and has a hypotensive effect.

3. Drug antihypertensive therapy.

The main groups of antihypertensive drugs are currently considered 4 groups: β-blockers, diuretics, calcium antagonists, ACE inhibitors. When choosing antihypertensive drugs, the ability of drugs to influence the level of atherogenic lipoproteins in the blood is taken into account. It should also take into account the age of patients, the severity of concomitant coronary artery disease.

3.1. Treatment with β-blockers

It is believed that the most significant mechanism of the hypotensive action of β-blockers is a decrease in heart rate and minute blood volume.

Main representatives:

Propranolol(anaprilin, inderal, obzidan) is a noncardioselective β-blocker. Initially, it is prescribed at 40 mg x 2 r per day, a decrease in blood pressure is possible on the 5-7th day of treatment. In the absence of a hypotensive effect every 5 days, increase the dose by 20 mg and bring it to the individual effective dose (i.e., 80 mg x 4 r per day). After achieving the effect, the dose is

gradually reduced and switched to a maintenance dose.

Nadolol (korgard)- non-cardioselective β-blocker of prolonged action without intrinsic sympathomimetic activity. Treatment begins with taking 40 mg once a day, then you can increase the daily dose by 40 mg every week and bring it to 240 mg (less often - 320 mg).

Atenolol (tenormin)- cardioselective β-blocker. At the beginning of treatment, it is prescribed in a daily dose of 50 mg. In the absence of a hypotensive effect, the daily dose can be increased after 2 weeks to 200 mg.

For the treatment of hypertension, it is advisable to use β-blockers with vasodilating properties. B-blockers with vasodilating properties include:

- non-cardioselective (pindolol, dilevalol, labetolol, proxodolol, carteolol);
- cardioselective (carvedilol, prisidilol, bevantolol).

Indications for long-term monotherapy of hypertension with β-blockers:

1. AH with the presence of left ventricular myocardial hypertrophy.
2. AH in young patients with active lifestyles.
3. Combination of hypertension with exertional angina.
4. Long-term treatment of hypertensive patients who have suffered transmural myocardial infarction.
5. AH in combination with cardiac arrhythmias, primarily supraventricular, as well as sinus tachycardia.

3.2. Diuretic treatment.

For the treatment of hypertension, the following groups of drugs are used:

1. Thiazide and thiazide-like diuretics.

Most often, these diuretics are used in patients with mild to moderate hypertension. Of these drugs, only hydrochlorothiazide is listed on the List of Vital Drugs.

*Hydrochlorothiazide (hypothiazide, dihydrochlorothiazide, esidrex).*With high hypertension, start with a dose of 50-100 mg once a day in the morning. With mild and moderate hypertension with 25 mg - in the morning. Maintenance dose 12.5-25 mg in the morning 1-3 times a week.

Of the thiazide-like diuretics, the most commonly used are:

Chlorthalidone (hygroton, oxodoline). A daily dose of 25-50 mg is also used. In case of insufficient hypotensive effect, the dose is increased to 100 mg per day in 1-2 doses.

Clopamide (Brinaldix) - a daily dose of 20-60 mg.

2. Loop diuretics

Usually used in hypertensive patients with resistance to thiazide diuretics, for the relief of hypertensive crises, with severe renal failure.

Furosemide- the initial dose is 20-40 mg 2 times a day, but the maximum daily dose is not more than 360 mg. In hypertensive crises accompanied by pulmonary edema, the dose is 100-200 mg IV.

Ethacrynic acid (uregit). The daily dose is 25 to 100 mg.

3. Potassium-sparing diuretics... Most often used:

Spironolactone (veroshpiron, aldactone) - the initial daily dose is 50-100 mg, then the daily dose is doubled every 2 weeks, reaching a maximum of 400 mg.

Amiloride - it is prescribed 5-10 mg once a day.

4. Uricosuric diuretics.

Indacrinone - a daily dose of 40 to 200 mg.

Ticrinafen - a daily dose of 30-480 mg.

They are used when arterial hypertension is combined with gout.

5. Diuretics with vasodilating properties.

Indapamide hemihydrate (arifon) - it is recommended to use in a dose of 2.5 mg once a day for any degree of severity of hypertension, after 1-2 months the dose can be increased to 5 mg per

day.

Indications for the predominant use of diuretics as antihypertensive drugs.

1. Volume-dependent hyporenin variant of GB, in women in the pre- and menopause.
2. High stable hypertension, in which sodium and water retention is noted.
3. Combination of hypertension with congestive heart failure, obstructive bronchial diseases.
4. The combination of hypertension with renal failure.

3.3. Treatment with calcium antagonists.

1. Calcium antagonists of the 1st generation.

Nifedipine (adalat, corinfar, cordafen)... The short-acting form of nifedipine can be used to relieve hypertensive crises and treat hypertension in doses not exceeding 40 mg per day. Slow-release tablets and capsules are prescribed 20-30 mg once a day, the maximum dose is 120 mg / day.

Verapamil (isoptin, finoptin) in the usual dosage form - an initial dose of 80 mg 3 times a day. Maximum - 360-480 mg. Extended forms - the initial dose is 120-180 mg once a day, the maximum is 360 mg / day.

Diltiazem (Dilzem, Cardizem, Cardil). In the usual form, an initial dose of 30 mg 3 times a day, a maximum of 360 mg. Long-term action - 120 mg initial dose, maximum - 360 mg / day.

2. Calcium antagonists of the 2nd generation.

The most widespread are the second generation calcium antagonists - dihydropyridine derivatives.

Nicardipine (cardin) - initial dose of 30 mg 2 times a day, maximum 120 mg.

Amlodipine (Norvasc) - an initial dose of 5 mg, a maximum of 10 mg.

Felodipine (plendil) - an initial dose of 5 mg / day, a maximum of 20 mg / day.

Indications for preferential administration of calcium antagonists in hypertension...

1. Combination of hypertension with exertional angina.
2. AH in persons who have had myocardial infarction without a Q wave.
3. Combination of hypertension and cerebrovascular insufficiency.
4. Combination of hypertension with severe dyslipidemia.
5. Combination of hypertension with chronic obstructive bronchial diseases.
6. AH in patients with diabetic nephropathy.
7. The presence of chronic renal failure in patients with hypertension.
8. The combination of hypertension with cardiac arrhythmias.

3.4. Treatment with ACE inhibitors.

For the treatment of GB, the following are often used:

Captopril (kapoten, tensiomin) - the initial dose is 12.5-25 mg 2-3 times a day, the maximum dose is 200-300 mg.

Enapril (enap, renitek) the initial dose is 5 mg, the maximum dose is 20-40 mg / day.

Perindopril (Prestarium, Coverex, Coversil) - an initial dose of 2-4 mg once a day, in the absence of a hypotensive effect - 8 mg per day.

Indications for the predominant appointment of ACE inhibitors in arterial hypertension

1. Combination of hypertension with congestive circulatory failure.
2. Combination of hypertension with ischemic heart disease, including after myocardial infarction.
3. AH in diabetic nephropathy.
4. Combination of hypertension with chronic obstructive bronchial diseases.
5. Combination of hypertension with impaired glucose tolerance or diabetes mellitus.
6. Severe hyperlipidemia in patients with hypertension.

3.5. Angiotensin receptor antagonists - II.

Losartan (kosaar)- an initial dose of 50-100 mg once a day. Indications for use of the drug are the same as for ACE inhibitors. Angiotensin II receptor blocking drugs are still in clinical use.

3.6. Direct vasodilators.

Hydralazine (apressin) - the initial dose is 10 mg 2-4 times a day, the maximum is 300 mg / day.

Dihydralazine (nepressol)- as part of adelfan. Prescribe 1-4 tablets per day.

3.7. α-blockers.

Prazosin (minipress, pratsiol)- an initial dose of 0.5-1 mg at bedtime. The maximum dose is 20 mg.

2nd generation postsynaptic α-blockers

Terazosin (chitrin) - the initial dose is 1 mg / day, the maximum dose is 20 mg once a day.

Ebrantil (urapidil) - the initial dose is 30 mg 2 times a day, the maximum dose is 180 mg / day.

3.8. centrally acting α2-agonists

Clonidine (clonidine, katapresan, gemiton) - the initial dose is 0.075-0.1 mg 2 times a day, the maximum is 0.45 mg. As monotherapy, clonidine is effective in 50-60% of patients with mild to moderate hypertension.

Methyldopa (dopegit, aldomet) - initial dose 0.25 g 2-3 times a day, maximum daily dose - 2 g.

Moxonidine (Zinc) - initial dose 0.2 mg, maximum -0.4 mg once a day.

3.9. Sympatholytics

1. Rauwolfia alkaloids.

Reserpine - the initial dose is 0.1-0.25 mg, the maximum dose is 0.3-0.5 mg / day.

Raunatin (rauvazan)- the initial dose is 2-4 mg. It is used for mild hypertension.

2. Guanethidine (ismelin, isobarin) - the initial dose is 12.5-25 mg, the average daily dose is from 50 to 100 mg.

3.10. Potassium channel activators

Nicorandil - inside 20 mg 2-3 times a day

Minoxidil - the initial dose is 2-2.5 mg 2 times a day, the maximum dose is 40 mg.

3.11. Vasoactive prostaglandins and stimulants of prostacyclin synthesis.

Pikletanin - a daily dose of 50-75 mg in 2-3 doses.

STEP THERAPY for GB

Step-by-step treatment program for hypertension (report of the WHO Expert Committee, 1980)

Stage I B-blocker II stage B-blocker + apressin or prazosin III stage	Stage I saluretic Stage II saluretic + B-blocker or reserpine, or dopegit III stage

B-blocker + apressin or prazosin + saluretic IV stage Add octadine or minoxidil to stage III treatment	saluretic + B-blocker or reserpine + apressin IV stage Add octadine or minoxidil to the stage III treatment

Stepwise treatment of hypertension according to N.A. Mazuru (1988).

1. Non-drug treatment (diet, weight loss, salt restriction, alcohol elimination, normalization of sleep).
2. Monotherapy with one of the drugs (clonidine, β-blocker, calcium antagonist, rauwolfia drugs, prazosin, labetolol).
3. The use of drugs of two groups.
4. Prescribing three drugs from three groups.
5. Simultaneous use of drugs from 4 groups.

Treatment of mild (I st.) And moderate (II st) arterial hypertension.

At stages 1 and 2 of hypertension, treatment begins with monotherapy. The drugs of 6 groups were determined by means of the 1st line: thiazide diuretics. β-blockers, calcium antagonists, ACE inhibitors, α-blockers, α and β-blockers.

If after 1-3 months of monotherapy the desired antihypertensive effect is not obtained, you can proceed as follows:

- carry out monotherapy with the same drug, but increase the dose to the maximum;
- continue monotherapy, but replace the drug with another;
- go to the III stage of treatment - combined antihypertensive therapy, adding a drug from another main group.

When the required antihypertensive effect is obtained, you can return to monotherapy (stage I).

Treatment of severe (III stage) and very pronounced (IV stage) arterial hypertension

At the 3rd and 4th stages of hypertension, in many patients it is possible to achieve an optimal hypotensive effect when monotherapy is carried out in adequate daily doses. However, most often it is necessary to start treatment with a combination of two (stage II) or three drugs (stage III). In the most severe cases, in the absence of an effect on stage III after 8-12 weeks. go to the IV stage.

Currently, a stepwise treatment regimen for PH is not considered mandatory because each patient has a number of individual characteristics of the course of the disease, which requires the differentiated use of antihypertensive drugs.

Ischemic heart disease. STENOCARDIA

Definition. IHD - acute or chronic heart damage caused by a decrease or cessation of blood supply to the myocardium due to an atherosclerotic process in the coronary arteries, which disrupts the balance between coronary blood flow and myocardial oxygen demand

IHD risk factors:

- male sex (men earlier and more often than women suffer from coronary artery

disease)
- age (risk of coronary heart disease at the age of 40)
- hereditary predisposition (parents have coronary artery disease, hypertension and their complications up to 55 years of age)
- dysproteinemia: hypercholesterolemia (fasting cholesterol level - 6.5 mmol / l), triglyceridemia (2.3 mmol / l or more), hypo α - cholesterolemia (0.9 mmol / l)
- arterial hypertension: blood pressure - 160 / 95ml. and more
- overweight
- smoking (1 cigarette per day)
- physical inactivity - not active leisure
- stress
- diabetes

Etiology.

Atherosclerosis of the coronary arteries in 97% of cases (Chazov E.I. 1971)
Spasm of the coronary arteries.

Pathogenesis.

1. Obstruction of coronary arteries by atherosclerotic process.
2. Decrease in the adequacy of the expansion of the coronary arteries under the influence of local metabolic factors with an increase in myocardial oxygen demand.
3. The role of endothelial factors.

Endothelin- vasoconstrictor factors, stimulates platelet aggregation. Prostacyclin - Produced in the coronary arteries in the endothelium. It has vasodilating properties, inhibits platelet aggregation. Endothelial relaxation factor is nitric oxide (NO), which is produced in endothelial cells.

4. Increased platelet aggregation. The production of thromboxane (vasoconstrictor) by thrombocytes is increased, and the balance between thromboxane and prostacyclin is normal. With ischemic heart disease, the balance is disturbed, the activity of thromboxane increases.
5. Increased myocardial oxygen demand.
6. Lack of collateral circulation.
7. Increasing the activity of lipid peroxidation, which contributes to platelet aggregation.

IHD classification (VKNTS AMN 1984)

I. Sudden coronary death.
II. Angina pectoris.
A. Exertional angina
- first emerging
- stable angina pectoris with an indication of the functional class
- progressive angina

B. Spontaneous angina
III. Myocardial infarction.
A. Large focal
B. Small focal
IV. Postinfarction cardiosclerosis.
V. Cardiac arrhythmias
Vi. Heart failure.

Methods for the diagnosis of ischemic heart disease.

Correct and timely diagnosis of ischemic heart disease allows not only to start treatment on time and improve the patient's quality of life, but also to influence the prognosis of his life. The "rule of five fingers" by P. Harvey still retains its meaning: 50% of the diagnosis in cardiology is the result of questioning

30% - physical examination data

10% -ECG

5% - R - graphy

5% - laboratory data

Thus, the doctor receives 80% of the diagnostics by direct examination.

Patient interview... To detail complaints, a thorough collection of anamnesis of the disease, to find out the risk factors for ischemic heart disease.

ECG- relatively low specificity. Even in patients with a pronounced clinical picture of coronary artery disease, there may be no changes. On the other hand, changes similar to ischemic ones can be recorded on the ECG and in other conditions (with left ventricular hypertrophy, hypokalemia). ECG allows to identify only pronounced organic disorders (scar), or rhythm disturbances.

Exercise test- a fairly accessible, relatively inexpensive non-invasive method for assessing the nature of pain in the chest. Bicycle ergometry (VEM) and treadmia are officially approved procedures for determining exercise tolerance. The only reliable sign of ischemia is a decrease in ST by 1 mm or more and the appearance of clinical signs of myocardial ischemia.

24-hour ECG monitoring - allows you to identify painless ischemia, rhythm disturbances (paroxysms of arrhythmias).

ECHO-KG - evaluates the contractility of the myocardium, reveals various defects, mitral valve prolapse, cardiomyopathy.

Stress-ECHO-KG - determination of ischemia during exercise.

Transoesophageal heart stimulation - ischemia can be caused by the imposition of a certain heart rate, but it is not physiological and therefore is rarely used.

Myocardial scintigraphy- carried out during an exercise test. Zones of decrease or absence of radioactivity (cold foci) reflect the localization and spread of zones of ischemia disturbance.

Coronary angiography - a new ability to accurately determine atherosclerotic changes.

Intracoronary ultrasound diagnostics of heart vessels - allows you to examine vessels in d = 1.8-2 mm.

Angina pectoris

"Angina pectoris" - Geberden first used this term in 1768. And for 140 years it was the only term used to define coronary heart disease, until the concept of myocardial infarction was defined.

Angina pectoris is paroxysmal pain in the region of the heart, which is one of the forms of ischemic heart disease.

Classification of stable angina pectoris.

Options class I - chest pain occurs with high intensity loads.

Class II - pain occurs when walking (more than 2 km.), when climbing stairs (more than 1 floor), after eating.

III class - pain occurs when walking on level ground at the usual pace at a distance of 1-2 blocks, 1st floor of the stairs.

IV class - rest angina.

Unstable angina is referred to as first-onset (up to 1 month)

- progressive

- postinfarction angina (2 weeks of acute myocardial infarction, accompanied by pain)
- Prinzmetal's angina.

Patients with unstable angina pectoris should be hospitalized because the risk of sudden death is very high.

Etiology and pathogenesis.

The widespread point of view that atherosclerosis of the coronary arteries is the cause of angina pectoris is erroneous.

For the correct diagnosis of coronary artery disease, one should know that atherosclerosis of the coronary arteries is not an obligatory component of it and that there is a form of ischemic heart disease that occurs with completely unchanged coronary arteries and is manifested by acute myocardial infarction, sudden death.

In recent years, there has been an evolution of views on the role of coronary spasm in the origin of angina pectoris. Through the stage of denying the possibility of spasm, the reality of this phenomenon in the pathogenesis of angina pectoris has been scientifically proven. It turned out that an almost totally stenotic coronary artery can spasm in the segment where there is no atherosclerotic lesion. The recognition of the spastic theory was facilitated by the effective therapy of angina pectoris with nitrates and calcium antagonists.

Clinic.

The main symptom is pain. Its most frequent localization is behind the sternum with irradiation to the left arm, scapula, neck, and sometimes to the left half of the jaw. The nature of the pain is constricting, burning. Some patients perceive pain as the strongest, unbearable, while experiencing a feeling of fear, lasting 5-10 minutes, sometimes up to 20 minutes. Occurs after physical exertion, passes at rest. Pain is relieved by taking nitroglycerin. A biochemical blood test does not reveal any abnormalities. At the height of the painful attack, ECG changes can be detected: flattening of the z.T. up to the transition to negative or, conversely, an increase to a giant pointed.

Diagnostics and differential diagnostics.

Diagnosing angina is not easy. Pain in the region of the heart can be felt in diseases not associated with damage to the cardiovascular system. Therefore, true cardialgias are distinguished, i.e. associated with heart damage and the so-called pseudocardialgia, when painful sensations in the region of the heart are not associated with its defeat.

Differential diagnosis is carried out with gastrointestinal stone disease, diaphragmatic hernia, left-sided infarction pneumonia, cervicothoracic radiculitis, intercostal neuralgia, neuroses.

Common signs of pseudocardialgia:

- pain is not related to physical activity
- no typical localization
- often due to a certain posture
- no effect from nitroglycerin

Differential diagnosis with MI

Nitro drugs are not effective for MI. The presence of myocardial infarction is confirmed by an increase in body temperature, the development of leukocytosis, characteristic changes in the ECG: deep h. Q, ST rise above the isoline, negative ZT, increased blood levels of CPK, MV-CPK, LDH enzymes.

Treatment

I. Diet with restriction of foods rich in cholesterol, saturated fatty acids (fatty meats, fish, spinach).

II. Treatment with antianginal drugs

a) nitrates
b) B-blockers
c) antagonists of Ca
d) potassium channel activators

In patients with coronary artery disease, the production of endothelium-relaxing factor (NO) by the coronary arteries is reduced, nitrates make up for this deficiency. Moreover, they improve collateral blood flow, reduce platelet aggregation, and improve microcirculation.

a) Three organic nitrates are vital:

1. Nitroglycerin (tablets, drops, inhalation form, capsules, buccal forms, cutaneous forms, for intravenous administration).

2. Isosorbide dinitrate (tablets, solutions for intravenous administration, aerosol, ointments, buccal forms).

Nitrosorbide 10 mg.
Isoket 20 mg.
Cardiket 20 mg.
Izosobit 20 mg.
The duration of action is 3-5 hours.
Prolonged forms
Isoket-retard 40-60 mg.
Cardiket-retard 20-40-60 mg.
Valid until 15-18 o'clock. Take 1x2 p.
3. Isosorbide 5-mononitrate
MonoMak 20-40 mg.
Monosit 20 mg.
Olikard-retard 40-50-60 mg.
Efoks 20-60 mg.
They act for up to 24 hours. In small doses, they do not cause tolerance.

The main contraindications for taking nitrates:

- glaucoma
- increased intracranial pressure
- stroke
- arterial hypotension

Group of synonyms: Molsidomin 0.002 g. Korvaton 0.002 g. Sydnofarm
Do not induce tolerance.

b) B-blockers

Used for the treatment of coronary artery disease since 1964. Black for his work in the field of creating B-blockers was awarded the Nobel Prize in 1988. These drugs have the following properties:

Reduce myocardial oxygen demand by reducing heart rate, systemic blood pressure and myocardial contractility.

Oxygen delivery to the myocardium is increased due to increased collateral blood flow.

Cardioselectivity (B1 - selectivity) - the ability of B-blockers to selectively block myocardial B1 receptors and not affect B2 receptors (bronchi, insulin secretion), allows them to be taken by patients with coronary artery disease and arterial hypertension with chronic bronchitis and diabetes mellitus.

Anaprillin - 10-20 mg.
Atenolol - 50-100 mg
Bisoprolol - 2.5 mg.
Trazicor - 20 mg.

Korgard - 20-40 mg.
Wisken - 5-10 mg.
Cordanum - 50 mg.
Practical recommendations for the use of B-blockers

1. The initial dose of B-blockers should be small, and the multiplicity depends on the duration of the drug's action.
2. The dose should be selected individually, focusing on the clinical effect, heart rate, blood pressure, heart rate in an upright position should be 55-60 v, blood pressure - at least 100 mm height.
3. After the onset of the antianginal effect, it is necessary to gradually reduce the dose of the drug and select a supporting one.
4. It is inappropriate to prescribe large doses of B-blockers for a long time due to the development of side effects.
5. It should not be canceled suddenly after prolonged therapy, as unstable angina pectoris, MI may develop.
6. Side effects and contraindications (bradycardia, hypotension) should be taken into account, they can cause increases in triglycerides, HDL cholesterol, erythema, psoriasis, and an itchy rash may appear.

Contraindications to the appointment.

- acute heart failure (pulmonary edema of cardiogenic shock)
- congestive heart failure
- av blockade II-III degree
- bradycardia iss I 50 in 1
- hypotension (BP I 100 mm Hg)
- insulin dependent diabetes mellitus

c) Ca antagonists

This group of drugs expands coronary arteries, eliminates coronary spasm, expands collaterals, reduces myocardial oxygen demand, inhibits platelet aggregation, and exhibits angiatherogenic properties.

I. Derivatives of diphenylalkylamine: verapamil (isoptin, finoptin) 40-80-120 mg., 2 ml. 0.25%

II. Benzodiazetine derivatives: diltiazem (cardil, dilzem, dilacor) - 60-90-120 mg.

III. Dihydropyridine derivatives: nifedipine (corinfar, cordafen, cordipine), isradipine (lomir), norvasc (amlodipine) felodipine (plendil).

With stable exertional angina, the daily doses are as follows:
Verapamil - 240 mg.
Diltiazem - 240-360 mg.
Nifedipine - 30-60 mg.

Side effects: headache, dizziness, redness of the face, swelling of the feet, legs, constipation, diarrhea, dermatitis, S. Lyell.

Contraindications: sick sinus syndrome, arterial hypotension,

It is preferable to prescribe Ca + antagonists in patients with angina pectoris combined with arterial hypertension, in young people with hyperlipidemia, in patients with concomitant bronchial obstruction.

d) Potassium channel activators activate potassium channels, thereby blocking the flow of Ca + into the cell and expanding the coronary arteries.

They cause a nitrate-like effect, which also leads to vasodilation. Reduce platelet aggregation and the production of free radicals in the myocardium.

Nicorandil - 10-20 mg x 2p.
Minoxidil - 10-20 mg x 2p.
Diazoxide - 10-20 mg. x 2p
Pinacidil - 10-20 mg. x 2p.

Gradual therapy of free patients with stable exertional angina.

F.K. I - drug therapy, as a rule, is not carried out, normalization of the work regime, everyday life, elimination of stress, antiatherogenic diet, elimination of risk factors. For pain - nitroglycerin under the tongue. Physical training shown.

F.K. II - all of the above, as well as monotherapy with one drug from 3 groups of antianginal drugs for 1 week. If there is no effect, they switch to stage II (treatment with 2 drugs) B-blockers + Ca + antagonists, nitrates + Ca + antagonists, nitrates + B-blockers.

F.K. III - treatment starts from stage II, in the absence of effect, after a week it goes to stage III - a combination of 3 drugs: (prolonged-action nitrates + B-blockers + Ca + antagonists). It is advisable to add antiplatelet agents to these drugs. It should be remembered about the development of tolerance to nitrates and prescribe them for 3-4 weeks, followed by a break for 7 days, after which the sensitivity of the coronary arteries to nitrates is restored and they can be prescribed again.

F. K. IV. Treatment with antianginal agents can be immediately prescribed from stage III, as described above, in addition, treatment of CHF, metabolic therapy, anticoagulants, antiplatelet agents.

III. Correction of plasma lipid composition.

1. Drugs that prevent the formation of atherogenic lipids:
   - statins (lovastatin, zocor, pravastatin)
   - fibrates (clofibrate, fenofibrate)
   - a nicotinic acid
   - probucol 500 mg. x 2 p.
2. Drugs inhibiting the absorption of cholesterol in the intestine:
   - bile acid sequestrants (cholestyramine, colestipol)
   - guarem
3. Physiological correctors of lipid metabolism containing essential phospholipids:
   - essential
   - lipostabil

The goal of lipid-lowering therapy in patients with coronary artery disease is to reduce and maintain cholesterol, LPPP at a level of less than 2.6 mmol / liter. The task of the attending physicians is to achieve a decrease in plasma cholesterol levels to 5.2 mmol / liter.

IV. Elimination of risk factors.

V. Metabolic therapy:

Preduct, mildronate. oliphene.

MYOCARDIAL INFARCTION

Myocardial infarction (MI) is an acute disease caused by the occurrence of one or more foci of ischemic necrosis in the heart muscle due to absolute or relative insufficiency of coronary blood flow.

In Russia, the first description of the clinical picture of myocardial infarction complicated by cardiogenic shock and heart rupture dates back to 1878 (K. Knopf). In 1892, in England, W. Osler suggested a direct connection between myocardial necrosis and damage to the coronary arteries of the heart. In the same year, V.M. Krenig described in detail the clinical picture of episthenocarditis pericarditis, which is, as is known, a complication of myocardial infarction.

However, the true history of the study of MI begins with the works of V.P. Obraztsov and N.D. Strazhesko (1909), who described for the first time in the world a detailed clinical picture of its various forms and causes in the form of coronary artery thrombosis. In recent years, the priority of Russian scientists has also been recognized in the United States, where for a long time the description of the MI clinic was associated with the works of Herrick (1912). In the study of myocardial infarction, an important role was played by the electrocardiographic method of research.

Etiology. The development of myocardial infarction is associated either with complete occlusion of the coronary artery (thrombus, embolus, atherosclerotic plaque), or with an acute discrepancy between the volume of blood supply through the coronary vessels and myocardial oxygen requirements. In more than 90% of cases, the cause of myocardial infarction is the cessation of blood flow to a site of the heart muscle through the coronary arteries sharply altered by atherosclerosis.

Factors contributing to the onset of myocardial infarction include an anomaly in the development of the network of coronary vessels, insufficiency of collateral connections between vessels and dysfunctions of their functions, increased thrombus-forming properties of blood, and disturbed microcirculation.

In turn, a number of factors can contribute to the appearance of these disorders, which determine the possibility of MI. These are the so-called risk factors. These include old age, arterial hypertension, lipoid metabolism disorders, carbohydrate metabolism disorders, obesity, genetic predisposition, smoking, physical inactivity, stress.

According to the Institute of Cardiology IM A. L. Masnikov VKNTs, in almost 90% of patients admitted to the clinic with myocardial infarction, it was possible to trace the relationship of its occurrence with the previous first stress or "stressful" situation.

A number of authors point out the possibility of the influence of nervous overstrain on an increase in the thrombus-forming properties of blood. The incidence of MI remains high and increases with age. So, according to IA Mazur (1985), for men aged 20-29 years, it is 0.08 per 1000 people / year, 30-39 years - 0.76; 40-49 years old -2.13; 50-59 years old -5.81; 60-64 years - 17.12. In women under the age of 50, myocardial infarction occurs 5-6 times less often than in men.

Epidemiological observations also confirm the importance of tobacco as a risk factor for the development of myocardial infarction. It turned out that among men 30-60 years old, MI developed 3.5 times more often in smokers. This is associated with an increase in the thrombus-forming properties of the blood, and with spasm of the coronary vessels, and with an increase in the content of carbon monoxide in the blood, as well as in myocardial oxygen demand (Astrur P. et al., 1987; Kannel WB et. Al., 1985) ...

In the domestic literature, the case of MI is usually divided depending on:

- the size of the lesion focus (large, - finely focal);
- spread of necrosis deep into the heart muscle (trans, - intramural, subendocardial, subepicardial);
- localizations of the lesion (anterior, lower, posteriorbasal, apical, lateral and septal regions of the left ventricle).

Recently, a number of authors have expressed an opinion about the inaccuracy of using the terms "transmural" and "subendocardial" MI. Previously, it was assumed that the presence of a pathological Q wave indicates a penetrating, transmural lesion of the heart muscle. A number of works have shown that the pathological Q wave on the ECG reflects not only the depth of myocardial damage, but also a number of other changes in the heart muscle. In this regard, P. Spodick proposed the terms "myocardial infarction with Q wave" and "Myocardial infarction without Q wave", as simpler and more convenient for use, with which many authors have now agreed.

Most often, myocardial infarction develops in the anterior wall of the left ventricle, i.e. in the blood supply basin of the anterior descending branch of the left coronary artery most often affected by atherosclerosis.

Clinic of myocardial infarction.

The clinical course of myocardial infarction is largely determined by the localization and extent of myocardial necrosis.

Large focal MI is a typical classic form of myocardial infarction, in which extensive necrotic changes involve all layers of the heart muscle.

The typical course of MI includes 5 periods:

1. Prodromal period, or the period of precursors ("preinfarction state"). In foreign literature, the term "unstable angina" corresponds to it. It lasts from several minutes to 30 days. For this condition, there are: a) the first-onset angina pectoris; b) progressive angina pectoris; c) postinfarction angina. During this period of the disease, dynamic ECG changes may be observed, indicating ischemia or damage to the heart muscle, however, in about 30% of patients, pathological signs on the ECG are absent.

2. The sharpest period... It lasts several minutes or hours from the onset of anginal status until the appearance of signs of necrosis of the heart muscle on the ECG. In this period, according to the main clinical manifestations of the disease, the following variants of myocardial infarction, described for the first time in the world by Obraztsov and Strazhesko, are distinguished:

- anginal (Status anginosus),
- asthmatic (Status asthmaticus),
- abdominal (Status gastralgicus),
- cerebrovascular,
- arrhythmic,
- asymptomatic (painless).

The most common variant of the onset of myocardial infarction - anginal - is manifested by severe pain syndrome (in 95%). Patients describe the onset of pain as strong compression, compression, heaviness. The pain is perceived as "dagger", tearing, burning.

Localization anginal pain - usually behind the sternum in the depths of the chest, less often in the left half of the chest.

Irradiatesanginal pain, usually in the left shoulder blade, shoulder, forearm. More often than with angina pectoris, the pain is widely reflected in both shoulder blades, both shoulders, and neck.

The onset of anginal pain in myocardial infarction is sudden, often at night or in the early morning hours, lasting several hours.

The duration of pain with anterior MI is usually longer than with localized necrosis on the inferior wall.

Ending pain. Repeated sublingual nitroglycerin administration does not relieve anginal pain in myocardial infarction. Decreased only by narcotic drugs.

3. Acute period. Corresponds to the final formation of the necrosis focus. Lasts about 10 days. Anginal pain with the end of necrotization subsides and if it occurs again, then only in cases of recurrence of myocardial infarction or early postinfarction angina. On the 2-4th day, the appearance of pericardial pain associated with the development of reactive aseptic inflammation of the pericardium - episthenocardial pericarditis, is possible. During this period, there are signs of resorption of necrotic masses and aseptic inflammation in the tissues adjacent to the zone of necrosis. A few hours after the onset of the disease, a febrile reaction occurs (38.5 ° C). Neutrophilic leukocytosis appears (10x109 / l - 12x109 / l), which decreases by the 7th day. ESR gradually increases. This is described in the literature as a crossover phenomenon. In the blood, the activity of a number of enzymes increases: creatine phosphokinase (CPK) and its MB - fraction,

ENZYME	Peak of activity	Duration of increased activity
KFK	1st day	3-4 days
AST	2nd day	3-4 days

LDH	3-6 days	1-2 weeks

With extensive MI, the activity of enzymes can increase many times higher than normal, for example, CPK - 20-30 times, ACT - 10 times.

Hyperglycemia is often observed, as well as a violation of the ratio of protein fractions in the blood (a decrease in albumin and an increase in globulins).

The ECG retains the picture of a monophasic curve, characteristic of MI changes in the ST segment, T and R waves.

On auscultation, the heart sounds are muffled, the rhythm of the gallop can be heard. Systolic murmur - as a manifestation of myocardial insufficiency, is heard at the apex of the heart. With pericarditis, a pericardial rubbing noise is detected.

4. Pre-acute period. Lasts about 2 months. This period is more favorable than the previous ones, because the main complications that cause high mortality in myocardial infarction develop, as a rule, on the 1st day of illness. During this period, most of the pain in the region of the heart is absent. The manifestation of heart failure decreases, although it may persist and increase. Rhythm disturbances are much less common - in 35-40%. During this period, tachycardia disappears, in some cases, systolic murmur ceases to be heard. The manifestations of the resorption-necrotic syndrome gradually disappear. By the end of the first week, the temperature will return to normal. If this does not happen, one should think about the addition of complications - pneumonia, thromboendocarditis, Dressler's syndrome.

Leukocytosis gradually decreases and at the end of the first week the number of leukocytes normalizes, eosinophils reappear. Blood sugar levels return to normal within a few days after the onset of myocardial infarction. The activity of CPK decreases by the 3rd day, LDH5 - by 10-14 days, ACT - by 3-5 days it is normalized. The ECG shows characteristic dynamics.

5. Postinfarction period- the time of complete scarring of the focus of necrosis and consolidation of the scar. During this period, the cardiovascular system adapts to new conditions of functioning, characterized by the exclusion of a certain part of the myocardium from the contractile function.

The most common complications are rhythm disturbances, chronic heart failure. The ECG shows signs of a formed scar.

Diagnosis of myocardial infarction

The basis for the diagnosis of myocardial infarction, especially in the first hours of the disease, is a thoroughly collected analysis of pain syndrome, taking into account the anamnesis indicating the presence of coronary artery disease, corresponding risk factors, and in the future, the appearance of dynamic ECG changes and an increase in enzyme activity or the content of cardiospecific proteins in the blood. The diagnosis of MI in the typical classical course of the disease is not difficult. It is much more difficult to recognize myocardial infarction in atypical and erased course. ECG registration and enzyme diagnostics help to make the final diagnosis.

ECG - diagnostics

ECG changes in myocardial infarction depend on its shape, location and stage. According to ECG signs, MI is conventionally divided into transmural, large-focal, subendocardial, intramural and small-focal. Some authors identify only 2 types of myocardial infarction with pathological Q wave.

With transmural necrosis, the entire myocardium under the electrode loses its ability to excite, as a result of which the shape of the ECG in the direct leads is determined by the depolarization vector of the opposite wall, forming the QS complex.

In case of large-focal MI, as a result of the loss of the vector of the affected part, the

myocardium, a pathological Q wave is formed.

With subendocardial myocardial infarction, the pathological Q wave does not form. Changes in the ST segment, T wave.

There are 4 main types of MI localization:

front - in which direct changes are recorded in leads V1 -V4;

lower (posterior diaphragmatic) - with direct changes in leads I, III, AVF;

lateral - with direct changes in leads I, AVL, V5 - V6;

posterior-basal - reciprocal changes are recorded in V1 - V2 (high Z. R, depression ST, high Z. T). Direct changes can only be in additional leads V7-V9.

Topical diagnosis of myocardial infarction

Localization of IM	ECG leads with direct signs	ECG leads with reciprocal signs
Anterior and antero-septal	V1 - V4	III, AVF
Anterobasal	AVL, V2 - V4	III, AVF, V1 - V2
Anterior spread	I, II, AVL, V1 - V6	III, AVF
Side	I, II, AVL, V5 - V6	V1
Posterior diaphragmatic (lower)	II, III, AVF	I, AVL, V2 - V5
Circular apical	II, III, AVF, V3 - V6	AVR, V1 - V2
Posterior basal	V7 - V9	V1 - V3

ECG - stages of myocardial infarction

1. The most acute stage (stage of damage)... Already at this stage, a necrosis zone is formed in the center of the damage zone. The ECG records the rise of the ST segment above the isoline, in the form of a monophasic curve. If necrosis in stage I has not yet formed, then there is no pathological h on the ECG. Q. If already in this stage a necrosis zone has formed in the center of the damage zone, then h appears on the ECG. Q, or QS.
2. Stage of development of MI (acute).Arising already in stage I, the zone of necrosis increases in depth and prevalence. This stage lasts up to 2 weeks. The ST segment is located above the isoline. The zone of necrosis leads to the appearance of pathological s on the ECG. Q, or QS. In dynamics, there is a gradual approach of the ST segment to the isoline and an increase in the inversion of the T wave, which stands out from the monophasic curve. At the end of the acute stage, the ST segment is on the isoline, the T wave is deep, negative.
3. Subacute stage... The area of damage disappears. The zone of necrosis is stabilized. The true size of myocardial infarction can be judged precisely at this stage, when additional death of a part of the muscle fibers that were in the state of damage occurs. The duration of this stage is 3-8 weeks. At this stage, z are registered. Q, or QS, the ST segment on the isoline, negative T. Diagnosis of an ECG in this period is reduced to a gradual decrease in the degree of T wave inversion and changes in the QRS complex.
4. Cicatricial.For this stage, in the case of large-focal MI, the presence of pathological h is characteristic. Q, decrease in amplitude h. R, ST segment on the isoline, stable shape of the T wave. Signs of cicatricial changes on the ECG can persist for life.

Differential diagnosis of myocardial infarction

There are two types of errors in the diagnosis of MI:

MI is mistakenly regarded as a disease, or, conversely, one or another disease is mistaken for MI. And more often it occurs with an atypical course of myocardial infarction. Most often it is necessary to differentiate from a prolonged attack of angina pectoris, PE (pulmonary embolism),

acute diseases of the abdominal organs, dissecting aortic aneurysms, and sometimes from spontaneous pneumothorax.

In all cases, for diagnosis, a serious help is enzyme diagnostics, ECG.

Treatment of myocardial infarction

Emergency care for uncomplicated large-focal myocardial infarction should be aimed at:

- pain relief (narcotic analgesics, antipsychotics);
-
- restoration of coronary blood flow (thrombolytic drugs, anticoagulants, antiplatelet agents);limiting the size of necrosis (B-blockers, nitroglycerin);
- prevention of early complications (arrhythmias).

Anesthesia

This must be done as quickly and completely as possible. The generally accepted method of pain relief is intravenous fractional administration of morphine and neuroleptanalgesia.

Positive effects narcotic analgesics - removal and reduction of pain, elimination of anxiety and fear, reduction of pre- and afterload.

Negative effects - oppression of respiration and contractility of the heart, increased activity of the gag reflex.

Usually morphine is injected in 2-3 stages in a total dose of 1 ml of a 1% solution, only intravenously. Do not use in elderly, debilitated patients with signs of respiratory depression. Morphine is categorically contraindicated in arterial hypotension.

Promedolhas a relatively weak analgesic effect, moderately inhibits breathing. Enter in a dose of 1 ml of a 2% solution intravenously slowly in two stages. The action begins in 3-5 minutes. and lasts about 2 hours.

Analgin. In myocardial infarction, it can be indicated either to potentiate the action of narcotic analgesics, or independently with initially mild pain in elderly patients.

Restoration of coronary blood flow. Thrombolytic therapy.

Indications:

- anginal pain persisting without supporting factors for more than 30 minutes. and not inferior to repeated intake of nitroglycerin;
- ECG: ST elevation by 1 mm or more, at least in 2 adjacent leads;
- the first 6 hours of the disease.

The use of thrombolytic therapy for myocardial infarction without H. Q is ineffective.

Contraindications:

- recent (within 10 days) bleeding;
- surgical interventions within the last 2 months;
- hemorrhagic diathesis of any etiology;
- dissecting aortic aneurysm;
- malignant neoplasms;
- pregnancy.

Choosing a drug... For thrombolytic therapy, streptokinase, urokinase, tissue plasminogen activator are used. All thrombolytic drugs activate plasminogen, a key enzyme of the fibrinolytic system. As a result, plasminogen is converted into an active fibrinolytic enzyme - plasmin, which converts fibrin into a soluble state.

During the first 3 hours of myocardial infarction, the effectiveness of different thrombolytic agents is approximately the same. Later, tissue plasminogen activator and prourokenase are more active.

Streptokinase(streptase) is injected intravenously at a dose of 1,500,000 IU per 100 ml. isotonic sodium chloride solution for 30 minutes. Before the administration of streptokinase

intravenously, 30 mg is usually given by injection. prednisolone.

Heparin... If thrombolytic therapy has not been given, heparin should be used from the first hours of MI in combination with aspirin. Heparin is especially indicated in cases of myocardial infarction without Q, but with ST depression, with unstable angina pectoris or an increased risk of thrombotic complications in patients:

- repeated MI;
- congestive heart failure;
- aneurysm of the heart;
- deep vein thrombosis of the leg;
- atrial fibrillation.

At the beginning, 5000-10000 units are injected intravenously, then they switch to drip infusion of the drug. After stabilization of the state, heparin is prescribed in 5000 units. after 6 hours subcutaneously, on average within 3-5 days.

The dose of the drug is recommended to be selected in such a way that for 2-3 days to maintain the coagulation time 1.5-2 times higher than the initial one. The heparin dose is gradually reduced.

Low molecular weight heparins... Encouraging results obtained with their application. So, for example, sulodexin (wessel duet f) 600 subcutaneously.

Acetylsalicylic acid(aspirin) as a direct antiplatelet agent is indicated in the 1st day of myocardial infarction, regardless of whether thrombolytic therapy was carried out or not. Aspirin is prescribed inside 125-200 mg / day.

Limiting the size of the necrosis.

It is possible to limit the zone of ischemia and necrosis by:

- restoration of coronary blood flow and collateral circulation;
- hemodynamic unloading of the heart.

Restoration of coronary blood flow is carried out by thrombolytic therapy, as well as by surgical methods (coronary artery bypass grafting, coronary angioplasty). For the purpose of hemodynamic unloading of the heart, the method of aortic balloon counterpulsation is used, as well as treatment with peripheral vasodilators and B-blockers.

The use of nitrates.

Nitrates have an antianginal effect, reduce the anterior and afterload on the left ventricle, reduce myocardial oxygen demand, and increase coronary blood flow.

Currently, it is customary to carry out intravenous infusion of nitroglycerin for 48-72 hours for all patients with MI with an adequate blood pressure of more than 100 mm Hg. (D. Alpert, G. Francis, 1994)

A.L. Syrkin (1991) recommends to inject nitroglycerin intravenously as follows: 2 ml of 1% nitroglycerin solution is diluted in 200 ml of isotonic sodium chloride solution. Dripping slowly. Isoket - 10 ml can be used instead of nitroglycerin. dissolve in 150 ml of saline. Along with this, tablet forms of nitrates are prescribed: nitrosorbide at 0.02 (2 t) x 4 p. per day, cardiket 20 mg x 3 r. in a day.

Nitrate therapy is highly effective and safe because it is physiological and is substitutional, because nitric oxide, which is part of nitrates, acts as an "endothelial hormone". And we know that when the endothelium is damaged by atherosclerosis, the release of this relaxation factor decreases.

The use of B-blockers.

Since 1964, Black was awarded the Nobel Prize (1988) for his work in the field of B-blockers.

They have a cardioprotective effect. Previously, their appointment reduces mortality by 25%. Have the following effect on MI:

- reduce myocardial oxygen demand and left ventricular wall tension by reducing heart rate and blood pressure;
- increase oxygen delivery to the myocardium due to increased collateral blood flow;
- have an antiarrhythmic effect.

Treatment with B-blockers can be started as soon as the patient is admitted to the hospital.
Timolol - 10 mg. x 3 p. per day,
Metoprolol 100 mg x 2 p. per day,
Atenolol - 50-100 mg per day,
Propranolol - 180-240 mg x 2 p. per day.
The therapy is continued for at least 12-18 months.

Prevention of life-threatening arrhythmias.

In the first 2-4 hours after the onset of clinical and ECG signs of myocardial infarction, death most often occurs from ventricular fibrillation. For prevention purposes, it is traditionally assumed to use lidocaine. Initially, it is recommended to inject 4-5 ml intravenously. 2% lidocaine solution, then establish an intravenous drip of 6 ml. 2% lidocaine solution in 200 ml. physical solution.

There is also a second technique: first, intravenously 6 ml. 2% solution, then 2-4 ml. 10% solution 4 times a day.

However, there is information indicating an increase in mortality by 38% and therefore it is now generally accepted that lidocaine in MI should not be used prophylactically. It is used only with the development of ventricular premature beats and tachycardia.

It has been established that intravenous administration of magnesium sulfate to patients with MI by 50% reduces the incidence of serious ventricular arrhythmias and halves mortality from MI.

The cardioprotective effect of MgSO4 in MI is due to its antiarrhythmic and antithrombotic effects.

Enter as follows: Dissolve 22 g of MgSO4 in 500 ml. 5% glucose solution and injected intravenously within 48 hours. You can use an ampoule 25% solution (10 ml. 25% solution of Mg S04 contains 2.5 g of the substance).

ACE inhibitors.

In connection with the activation of the renin-angiotensin-aldosterone system observed in MI, with extensive damage to the heart muscle, even without obvious signs of heart failure, the appointment of ACE inhibitors is indicated. Their use in patients from day 1 reduces mortality in patients with MI.

Treatment with metabolic cardioprotective agents.

Numerous studies have shown that intravenous administration of a "polarizing mixture" helps to reduce the size of necrosis.

The composition of the mixture: 250 ml 10% glucose solution, 100 ml 4% potassium chloride solution (or 10 ml Panangin), 6 - 8 units of insulin. The polarizing mixture is administered intravenously once a day.

Neoton - plays a key role in the energy supply of muscle contraction.

Antioxidant vitamin E - 1 ml is prescribed. 30% solution intramuscularly.

Lipid-lowering drugs xanthinol nicotinate 1 ml. in / m.

Normalization of the functional state of the central nervous system.

In the acute period of myocardial infarction, patients may experience a feeling of fear of death, anxiety, depression. Therefore, all patients are shown rational psychotherapy. It is possible to use tincture of valerian, motherwort herb, phenazepam.

Complications of MI.

- Cardiogenic shock;
- Cardiac asthma and pulmonary edema;
- Heart rhythm and conduction disorders;
- Thromboembolic complications;
- Aneurysm of the heart;
- Myocardial rupture;
- Pericarditis;
- Heart failure;
- Postinfarction angina.

INSUFFICIENCY OF BLOOD CIRCULATION

Insufficiency of blood circulation is a pathological condition in which the work of the cardiovascular system does not ensure the delivery of the required amount of blood and, consequently, oxygen to the organs and tissues.

The main reasons for the development of chronic heart failure

(Mukharlyamov N.M., 1978; Sidorenko B.A., Preobrazhensky D.V., 1995):

I. Myocardial damage
- myocarditis;
- dilated cardiomyopathy;
- myocardial dystrophy;
- IHD (atherosclerotic and postinfarction cardiosclerosis)

II. Overloading the heart muscle with pressure (systolic overload)
- arterial hypertension;
- aortic stenosis;
- narrowing of the orifice of the pulmonary artery

III. Volume overload of the heart muscle (diastolic overload)
- aortic or mitral regurgitation;
- ventricular septal defect;
- patent ductus arteriosus

IV. Combined myocardial overload
- a combination of reasons leading to volume and pressure overload;
- complex congenital and acquired heart defects

V. Violation of diastolic ventricular filling (diastolic failure)
- hypertrophic cardiomyopathy;
- hypertensive heart;
- restrictive cardiomyopathy;
- isolated mitral stenosis;
- exudative pericarditis;
- constructive pericarditis

VI. Conditions with high cardiac output

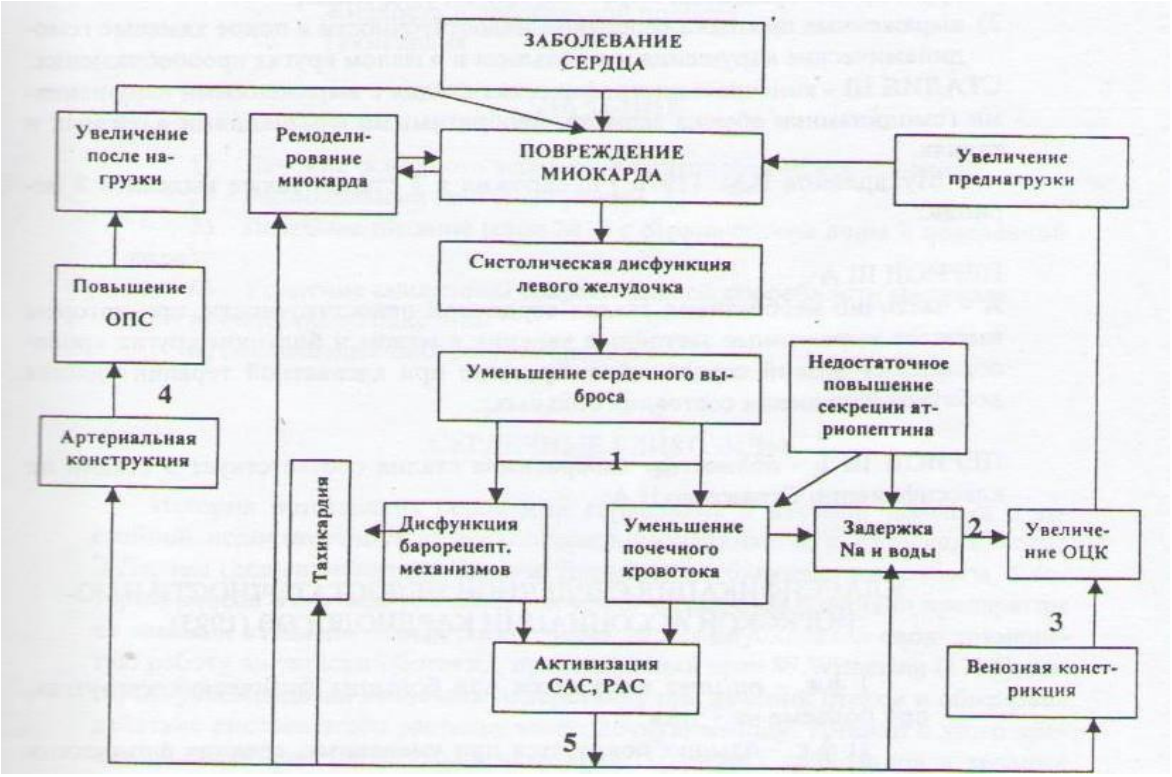
- thyrotoxicosis;
- severe anemia;
- massive obesity;
- cirrhosis of the liver

Basic concepts

1. Preload is the degree of diastolic filling of the left ventricle, it is determined by the tone of the veins, venous return of blood to the heart, pressure in the pulmonary circulation. Adequately reflects the preload end-diastolic pressure in the pulmonary artery.

2. Afterload is the systolic tension of the myocardium required for the expulsion of blood. According to Laplace's law, this is the pressure developed in the systole of the ventricles, multiplied by the radius of the ventricle. In practice, it is determined by intra-aortic resistance.

Scheme of the pathogenesis of chronic heart failure



Places of application of drug action:

1. Cardiac glycosides;
2. Diuretics
3. Venodilators;
4. Arterial vasodilators;
5. ACE inhibitors.

CLASSIFICATION OF HNK

(Strazhesko N.F., Vasilenko V.Kh.)

STAGE I - latent circulatory failure, manifested by shortness of breath, palpitations, fatigue, only with physical exertion. Hemodynamics is not disturbed at rest.

STAGE II

1) signs of insufficiency at rest are moderately expressed, exercise tolerance is reduced, there are disorders in the large or small circle of blood circulation, their severity is moderate;

2) pronounced signs of heart failure at rest, severe hemodynamic disturbances in the large and pulmonary circulation.

STAGE III - final: dystrophic stage with severe metabolic hemodynamic disorders, irreversible changes in organs and tissues.

Mukharlyamov N.M. (1978) proposed to distinguish 2 periods in 3 stages.

PERIOD IIIA

Partially irreversible stage of heart failure, in which there are pronounced congestion in the small and large circles of blood circulation, low cardiac output, but with adequate therapy it is possible to improve the condition of patients;

PERIOD IIIB - completely irreversible stage corresponds to stage 3 according to N.A. Strazhesko's classification.

NEW YORK ASSOCIATION OF CARDIOLOGISTS CLASSIFICATION OF HEART FAILURE (1995)

I f.c. - shortness of breath appears with great physical exertion, when climbing to the 3rd floor.

II f.c. - shortness of breath appears with moderate, medium physical exertion when climbing the 1st floor or brisk walking.

III f.c. - shortness of breath appears with minor physical exertion.

IV f.c. - shortness of breath appears at rest.

BASIC DIAGNOSTIC CRITERIA FOR INSUFFICIENCY OF BLOOD CIRCULATION.

- Paroxysmal nocturnal dyspnea
- Swollen neck veins
- Wheezing in the lungs
- Cardiomegaly
- Gallop rhythm
- Hepato-jugular reflux
- Acute pulmonary edema

Additional criteria:

- Swelling of the lower extremities
- Night cough
- Shortness of breath on exertion
- Hepatomegaly
- Pleural fluid
- Tachycardia

TREATMENT

1. Treatment of the underlying disease (etiological treatment)
2. Rational treatment regimen
3. Healing food (table number 10 with limitation of water and sodium chloride)

4. Strengthening the reduced contractility of the myocardium

a) cardiac glycosides
b) non-glycosyl inotropic agents

CARDIAC GLYCOSIDES

The history of the use of cardiac glycosides in the treatment of patients with congestive circulatory insufficiency begins in 1775, when E. Darwin (the grandfather of the famous Charles Darwin) published two works in which he described 6 cases of successful treatment of "cardiac dropsy" with a drug from "fresh stems" of foxglove. Ten years later, the English botanist, practicing physician W. Withering (1785) published his famous work, who recommended prescribing digitalis in the treatment of edema and substantiated the effect of the leaves of this plant on the heart muscle. It was from this time that the brilliant "career" of cardiac glycosides in the therapy of CNS began. More than 400 cardiac glycosides are currently known.

Mechanism of action:

- strengthen and shorten the systole;
- increase systolic output;
- lengthen diastole;
- reduce the activity of the sympathoadrenal system, the renin-angiotensin system;
- have a diuretic effect;
- reduce the reabsorption of Na and iodine in the renal tubules;
- oppress the automatism of the sinus node, which leads to a decrease in the heart rate.

The most commonly used:

Strofantin - 1 ml 0.05% intravenously
Korglikon -1 ml 0.06% intravenously
Digoxin -1 ml 0.025% intravenously
Celanide -2 ml 0.02% intravenously
0.25 mg tablet

Contraindications to the appointment of cardiac glycosides

- atrioventricular block II;
- intoxication with cardiac glycosides;
- sinus bradycardia less than 50 per minute;
- sick sinus syndrome;
- acute period of myocardial infarction (dangerous due to the possibility of arrhythmias).

METHODS OF CHF TREATMENT WITH CARDIAC GLYCOSIDES

When treating with cardiac glycosides, two periods are distinguished: a period of saturation and a period of maintenance therapy. The main objective of the saturation period is to achieve the individual saturation dose. Criteria for achieving a saturating therapeutic dose:

- decrease in heart rate up to 60-70 per minute;
- increase in daily urine output;
- reduction of edema;
- reduction of shortness of breath.

The task of the maintenance therapy period is to maintain the achieved full saturation with cardiac glycosides. For this, an amount of cardiac glycoside is injected daily, equal to the daily excretion of the drug.

TREATMENT WITH NON-GLYCOSIDE INOTROPIC MEDICINES

This group of drugs entered clinical practice in the 1980s. 2 groups of funds are used:

- stimulants (agonists) of B-adrenergic receptors (dopamine, dobutamine);
- phosphodiesterase inhibitors (amrinone, milrinone, piroximone).

The most commonly used:

Dopamine- endogenous catecholamine, increases contractility, increases renal blood flow. A special indication for the appointment of dopamine is the presence of bradycardia and hypotension in a decompensated patient.

Dopamine treatment: 40 mg of the drug (1 ampoule) is dissolved in 200 ml of 5% glucose solution or isotonic sodium chloride solution and injected intravenously.

Dobutamine - 20 ml of solution in a bottle.

Introduction method: 10 ml of water for injection or 5% glucose solution is injected into the bottle, then everything is transferred into a bottle with 500 ml of 5% glucose solution.

Currently, non-glycoside inotropic drugs are used primarily for the treatment of acute heart failure (including cardiogenic shock in patients with myocardial infarction). They are not an alternative to cardiac glycosides when long-term maintenance therapy is required.

5) Elimination of edematous syndrome. Treatment with diuretics.

CLASSIFICATION OF URINE PRODUCTS

Acting at the level of the glomerulus - aminophylline, theophylline, cardiac glucosides.

Acting at the level of the proximal tubule of the nephron - diacarb, fonurite, osmotic diuretics (mannitol, urea).

Acting at the level of Henle's loop are loop diuretics (furosemide, uregit), thiazide diuretics (hypothiazide, brinaldix, xypamide).

Acting at the level of the proximal tubule - indacrinone.

Acting at the level of the distal tubule - veroshpiron, soldaktol, triamterene, amiloride.

PRINCIPLES OF RATIONAL THERAPY WITH URINE

1. Diuretics should be prescribed only when the first symptoms and signs of edema syndrome appear.

2. In case of IIa degree of NK, diuretic therapy is recommended to start with small doses of thiazide diuretics (25 mg hypothiazide or 0.02 brinaldix, triampur K-1 tab.).

3. At IIb degree of NK, loop diuretics (furosemide, uregit, bumetanide) are effective as monotherapy or in combination with potassium-sparing diuretics.

4. At III degree of NK, the drugs of choice are loop diuretics, which are advisable to use for a long time in combination with potassium-sparing agents, for example, with verospiron.

5. Diuretic therapy should be carried out in such doses that the daily urine output does not exceed 2500 ml.

6. Thiazide, potassium-sparing diuretics should be used in the morning, preferably on an empty stomach.

Usually, the introduction is made in the following cases:

- when the patient develops acute left ventricular failure;
- with very severe chronic circulatory failure, when intestinal absorption is impaired and the intake of diuretics inside is ineffective.

At the initial, active stage of treatment of circulatory insufficiency, diuretics are

recommended to be used almost daily, since a break in diuretic therapy at the stage of pronounced edematous state increases water retention and delays the onset of the clinical effect. After the onset of a significant improvement in the condition, reduction of shortness of breath, edema, he switches to supportive therapy. After 2 days or even 1-2 times a week. Doses are individual.

6) Reduction of preload and post-load on the left ventricle (peripheral vasodilators)

a) venous vasodilators - nitrates, molsidomin, sydnopharm.

Nitrates

Three organic nitrates are vital:

- nitroglycerine;
- isosorbide dinitrate;
- isosorbide 5-mononitrate

Nitroglycerin for intravenous administration is available in the following dosage forms:

Perlinganite - in ampoules of 10 ml; in bottles of 50 ml.

Nitro pol-in ampoules of 5 and 25 ml; in bottles of 50 ml 0.1%.

Nitroglycerine - 1% solution 2 ml

Long-acting nitroglycerin preparations for oral administration. Produced depending on the content of nitroglycerin in them in 2 forms - mite and forte, the duration of action of the mite forms is 1.5-2 hours, forte lasts up to 6 hours.

	Mite (mg)	Forte (mg)
Sustak	2.6	6.4
Nitrong	2.6	6.5
Sustanit	2.6	6.5
Nitro Mac	2.5	4.5

Isosorbide dinitrate

Conventional-acting isosorbide dinitrate tablets are reference drugs for oral administration.

The domestic drug nitrosorbide, 10 mg in 1 tablet, as well as foreign drugs isodinite, isoket, cardiket in tablets or capsules of 20 mg are used. The action of the tablets begins in 20-30 minutes and lasts up to 3-5 hours. Prescribed at 10 mg 3-4 times a day. There are also extended release isosorbide dinitrate tablets and capsules. These include isoket - retard (40-60 mg), cardiket - retard (20, 40 and 60 mg). They are prescribed 2-3 times a day. The drugs of this group are also available in ampoules for intravenous administration. Isoket - 10 ml ampoule.

b) calcium antagonists

These are substances that inhibit the entry of calcium ions into cells through the "slow" calcium channels.

A drug	Average daily dose
1st generation	
Verapamil (finoptin)	240-480 mg, 3-4 times a day
Diltiazem (cardil)	60-90 mg, 3-4 times a day
Nifedipine (corinfar, cordafen)	10-20 mg, 3-4 times a day

2nd generation	
Amlodipine (Norvasc)	5-10 mg, once a day
Isradipine (lomir)	5-10 mg, once a day
Felodipine (plendil)	5-10 mg, once a day

c) ACE inhibitors

In August 1991, an editorial by a prominent American cardiologist, Professor E. Brownwald was published in an English medical journal, which "legally legalized" a new era in the treatment of patients with CHF - the era of angiotensin-converting enzyme (ACE) inhibitors.

The right to be called "remedy number 1" in the treatment of these patients with ACE inhibitors has earned due to the unique properties of affecting all "levels" of CHF. Another feature that distinguishes drugs of this class from traditional means of treating CHF is a wide range of indications for use, including not only CHF, but also arterial hypertension, myocardial infarction and even diabetic nephropathy.

Currently, more than three dozen ACEIs are known, which are combined into 3 groups:

- containing a SH-group (captopril);
- carboxyalkyl dipeptides (enalapril, ramipril);
- containing a phosphinyl group (fosinopril).

Indications for the appointment of ACE inhibitors in CHF:

- the initial stage of CHF;
- severe CHF, including those resistant to traditional treatment;
- CHF of any stage against the background of sinus rhythm in combination with diuretics;
- CHF of any stage and in combination with arterial and pulmonary hypertension;
- CHF of any stage in combination with diabetes mellitus.

METHODS OF TREATMENT OF ACE inhibitors

At the first appointment of ACE inhibitors, an acute drug test is carried out with the expected dose of the drug, usually the minimum, with the control of the general well-being of the patient, measuring blood pressure and heart rate during the day. The test allows you to determine the tolerance of the drug, the degree of blood pressure reduction, hemodynamic parameters.

With good tolerance, further dosage can be gradually increased.

Average therapeutic doses:

- for kapoten - 75 mg per day (25 mg 3 times a day);
- for enalapril - 10-20 mg per day;
- ramipril - 5 mg per day;
- for prestarium - 4 mg per day.

7) Decrease in increased activity of the sympathoadrenal system

As mentioned earlier, activation of the sympathoadrenal system plays an important role in the pathogenesis of CHF, which contributes not only to vasoconstriction, but also to the development of arrhythmias that worsen the prognosis for life.

In this regard, in recent years, the possibility of using B-adrenoreceptor blockers for the treatment of CHF has been discussed. Indications for the appointment of B-blockers:

- hypertensive type of hemodynamics;
- a combination of CHF and exertional angina;
- the presence of arrhythmias in CHF;

- combination of CHF with arterial hypertension.

This group of drugs has been used since 1964. Black was awarded the Nobel Prize in 1988 for his work on the creation of B-blockers.

It is necessary to start treatment with B-blockers with small doses (1/4 of the therapeutic dose), gradually increasing the dose:

- propranolol (anaprilin, obsidan) - 120 mg / day
- oxyprenolol (trazicor) - 120 mg / day
- atenolol (tenormin) - 100-200 mg / day.

When prescribing drugs in this group, remember about their selectivity. Cardioselective include atenolol, esmolol, bisoprolol, carvedilol, etc. Non-cardioselective include - anaprilin, timolol, trazicor, etc.

Contraindications: bronchial asthma, a tendency to bronchospasm, insulin-dependent diabetes mellitus, bradycardia and AV blockade.

Nevertheless, it should be considered that at present the question of the use of B-blockers in the treatment of CHF has not been finally resolved.

8) Metabolic and antioxidant therapy, the use of antihypoxants

In the complex therapy of CHF, the use of metabolic agents is advisable. The following drugs are recommended: multivitamins (duovit, oligovit), 1 tablet 3 times a day.

Retabolil - 1 ml / m once every 10 days;

Cocarboxylase is a coenzyme of vitamin B1. It is administered intramuscularly at 50-100 mg once a day, for 20-30 days;

Riboxin - 2 tab. 3 times a day or intravenously, 10 ml;

Neoton - improves myocardial contractility.

Vitamin E - 1 ml 10% IM for 20 days