MINISTRY OF HIGHER EDUCATION, SCIENCE AND INNOVATIONS OF THE REPUBLIC OF UZBEKISTAN
MINISTRY OF HEALTHCARE OF THE REPUBLIC OF UZBEKISTAN
TASHKENT MEDICAL ACADEMY
DEPARTMENT OF INTERNAL DISEASES IN FAMILY MEDICINE №2

EDUCATIONAL-METHODICAL COMPLEX
on the module

AMBULATORY-POLYCLINIC THERAPY

Field of knowledge: 500000 – Health care and social security

Branch of education: 510000 – Health care

Direction of education: 5510100 – General medicine

5111000 – Professional education

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CONTENTS

INTRODUCTION

The subject of "Ambulatory polyclinic therapy" provides students with practical and independent knowledge, skills, control methods of knowledge assessment using modern technologies and literature, as well as clinical thinking, justification of disease and its symptoms in general practitioners, formation of knowledge and skills, including: teaching students timely and early diagnosis of diseases with a syndromic approach, teaching students comparative diagnosis of diseases with certain syndromes, further important knowledge and practical skills improvement (data collection, determination of the patient's problem and objective examination, as well as reasonable recommendation of laboratory-instrumental examination methods, counseling skills, teaching students to make a reasonable choice of patient management tactics, targeted treatment to students - performs the tasks of teaching the implementation of preventive measures, teaching students the principles of dispensary observation and monitoring in the conditions of a rural medical center, a rural family polyclinic (RFP) and a family polyclinic (FP).

This complex was created for the 6th course of the subject "Ambulatory polyclinic therapy", and it is intended for professors-teachers of the department of Internal diseases in family medicine № 2 and 6th year students of the Faculty of Medical pedagogy.

LECTURE TOPIC: FUNDAMENTALS OF FAMILY MEDICINE. HISTORY OF FAMILY MEDICINE. DEVELOPMENT IN UZBEKISTAN.

TRAINING TECHNOLOGY

<table>
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<tr><td>Lecture plan</td><td>1. History of family medicine
2. Prerequisites for the development of general medical practice in Uzbekistan
3. The main directions of reforms in the health care of Uzbekistan at the present stage
4. Rural medical stations
5. Principles of family medicine
6. Preventive work of a family doctor
7. The specifics of the work of a general practitioner</td></tr>
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1. Introduce students to the basics of family medicine
2. Familiarize yourself with the stages of healthcare reform in Uzbekistan at the present stage
3. Tell about the work of rural medical centers, about their purpose
4.Teach the basic principles of family medicine
5.Teach students the stages of GP preventive work
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OP needs to know:
1. Basic principles of family medicine
2. Categories of services provided in SVPs
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4. Prevention steps
5. The volume of medical services provided by GPs</td></tr>
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Conditions for the educational process	Audience

TECHNOLOGICAL CARD LECTURES

Stages, time	**Activity**	
	Teacher	**Students**
Stage 1 Introduction (5 minutes)	1. Tells about the topic of the lecture, its purpose and plan	1. Listen
Stage 2 Actualization (improvement) of knowledge (20 minutes)	2.1. In order to increase the actualization - the importance of students' knowledge asks questions: 1. Define the terms family medicine, general practitioner 2. List the categories of services provided in SVPs 3. List the documents on the basis of which the GP works 4. List the types of SVPs depending on the number of people served Conducts a survey 2.2. Showing on the screen, offers students to get acquainted with the goals and objectives of the lecture. Slide #1, #2	2.1. Answer questions asked 2.2. Study slide number 1 2.3. Study slide number 2
Stage 3 Main part (informational) (55 min)	3.1. Introduces students to the lecture material, the importance of the topic and the principles of the formation of an intelligent cultural personality, in particular the GP. In order to increase the actualization of knowledge, he conducts a quick survey of students: 1. For 1 point of the lecture plan: list the basic principles of family medicine	3.1. Together they analyze the listened lecture material, ask questions

	2. On the 2nd point of the lecture plan: tell us the categories of services provided in the SVP 3. For point 3 of the lecture plan: list the main regulatory documentation on the basis of which the GP works 4. According to the 4th point of the lecture plan: tell the main stages of prevention On the 5th point of the lecture plan: The volume of medical services provided by GPs Stopping at the important points of the lecture, he suggests writing down the main points in a notebook	Key points are written in a notebook.
<u>Stage 4</u> Final (10 min)	4.1. Asking questions: 1. What is the difference between the work of a general practitioner and a subspecialist 2. List the main categories of services 3. Which surveys are the prerogative of the SVP 4. Name the main stages of prevention 4.2. Gives a task for independent work of students: Tactics of a General Practitioner in the Most Common Pathologies	4.1. Answer questions 4.2. Listen, write

1.1 Lecture: "Foundations of family medicine"

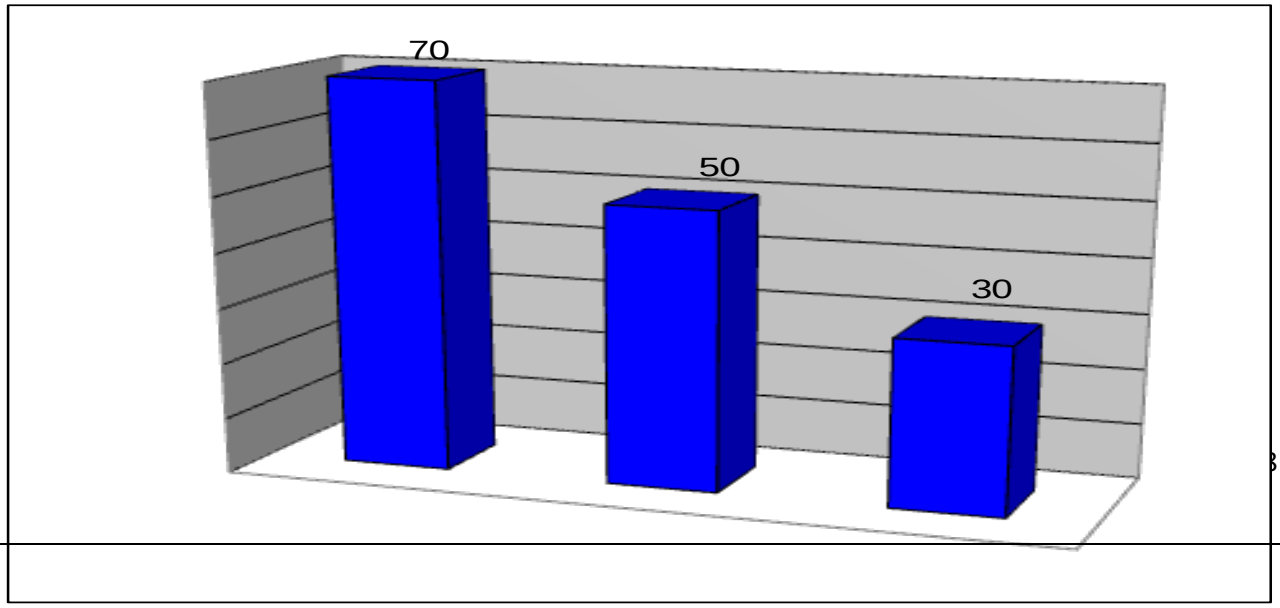
Auguste Rodin once said: “In my youth I considered the nose, lips and expressions separately. I was ignorant, I should have looked at everything as a whole.
In recent years, general practice has attracted increasing attention from governments in many countries as an economical and effective way to provide primary health care, allowing you to suspect the disease in time and start treatment.
The place of a general practitioner in the healthcare system is unique - he directly communicates with the patient and his family, takes responsibility for the health of those observed, provides treatment and prevention, using the latest achievements of medical science, and coordinates the efforts of all health services.
Prerequisites for the development of general medical practice in Uzbekistan:

- Great Britain is the founder of general medical practice.
- In 1948, the National Health System was created, providing comprehensive free medical care to the entire population of Great Britain.
- At the end of the 70s of the last century, physicians in Western Europe and the United States realized that increased specialization moves medical care away from the specific needs of the population and increases the cost of the healthcare system to meet these needs.

The list of medical care provided by different parts of health care in England:

- Assistance provided by general practitioners on an outpatient basis - 90% of patients.
- Assistance provided in hospitals - 9-10% of patients.
- Assistance provided in specialized centers - 1% of patients.
- Given this situation, in 1978 the Declaration of Alma-Ata was adopted, which placed primary health care at the forefront of health policy, giving general medical practice an important role.

PHC is an integral part of the national health care system and at the same time acts as the main component of the overall social and economic development of society
• The reason for this is the smaller number of family doctors, rural medical centers.
Primary health care is the first step in the contact between individuals, the family, the community of people and the national health system, bringing medical care as close as possible to the place of residence and work, forming the first element of a continuum of health care.



- The percentage relationship between the number of general practitioners and the cost of health care costs in terms of Gross Domestic Product of individual countries: although the US spends the largest amount in the world on health care, 15% (1.55 trillion dollars - data for 2002) of its GDP nevertheless remain in 12th place among 13 countries where medicine is considered the most developed in the world.

Thus, in our country, the Decree of the President of the Republic "On the state program for reforming the healthcare system of the Republic of Uzbekistan in 1998-2005" was adopted. (UP - 2107 dated 10.11.98). The priority direction of the reform was the improvement of PHC, incl. introduction and strengthening of GENERAL MEDICAL PRACTICE.

Reforming primary health care in Uzbekistan

The main directions of reforms in health care are:

- A fundamentally new structure of the healthcare system, aimed at creating equal conditions for receiving primary medical care in urban and rural areas;
- A new practical approach to the issues of motherhood and childhood, aimed at creating conditions for the birth and upbringing of a healthy generation;
- The main directions of reforms in health care are: Creation of a fundamentally new system of emergency medical care for the population at all territorial levels;
- Rejection of old stereotypes, significant expansion of financial sources of the industry, incl. through the development of paid and private health care;
- Optimization of the industry's financing system, primarily through the concentration of budgetary funds in health care, outpatient treatment and prevention, instead of inefficient use of expensive hospital beds.

The development of a general practitioner model includes:

- The chosen model of general medical practice should correspond to the customs, traditions and mentality of people, as well as other features of the country.
- Family medicine, introduced in different countries without appropriate changes, usually does not function properly.
    - One idea, many implementations. The network of rural healthcare facilities has been restructured, the existing multi-stage system has been replaced by a 2-stage system

Today, more than 3,100 rural medical centers operate in the Republic. A RURAL MEDICAL POINT can be:

1st type - up to 1500 people;
2nd type - up to 3500 people;
3rd type - up to 6000 people;
4th type - up to 10,000 people and more served population

The quality of medical care for the population largely depends on the level of professionalism of medical workers and reforms in healthcare, which could not but be reflected in the improvement of the system of training and retraining of personnel. Family medicine is a separate, one-of-a-kind specialty built on special, basic principles that distinguish it from other medical specialties.

Principles of family medicine

1. COMPREHENSIVENESS - there is no such category of complaints or problems that would not be taken into account; all problems related to human health are considered; family medicine does not make restrictions, but unites the solution of medical problems into a single whole with all aspects of the patient's life
2. DURATION OF FOLLOW-UP - a family doctor observes his patient at all stages of life from birth to death; long-term observation creates an opportunity to identify risk factors, control over preventive and therapeutic measures; the effectiveness of the work of the GP is improved.
3. INTEGRATION - the inclusion of all aspects of medicine: curative, rehabilitative, preventive, human health protection in society as a whole.
4. COORDINATION - if necessary, the family doctor consults with narrow specialists or refers the patient to inpatient treatment; at the same time, it does not stop monitoring him and exercises control over his treatment; coordinates all stages of patient treatment at different levels of the healthcare system.
5. CONFIDENT RELATIONSHIP - the duration of observation contributes to a trusting relationship between the doctor and the patient; the family doctor protects the rights and dignity of the patient; the patient feels that he is respected, heard and understood.
6. TEAM WORK - family doctor, nurse, all medical staff work as a single team; the task of the family doctor is to organize the work of each team member according to his knowledge and skills in the interests of the patient
7. ACCESSIBILITY OF RECEIVING PRIMARY MEDICAL CARE - each family should have its own family doctor; work should be organized in such a way as to provide the patient with easy access to medical care; the provision of emergency care at any time of the day should be thought out.
8. RESPONSIBILITY OF THE PATIENT FOR HIS HEALTH, THE HEALTH OF HIS FAMILY MEMBERS - AND COOPERATION WITH HIM - the family doctor and the patient work as partners, making efforts to achieve the best results; the patient becomes an active participant in the restoration and preservation of his health and the health of his family.
9. PREVENTIVE WORK - one of the main activities of a family doctor and his team; family medicine focuses on maintaining a healthy lifestyle, on the early diagnosis of diseases for the systematic maintenance of health and conducting health education among the population.

Practical steps for all types of prevention

PRIMARY prevention - carrying out a number of measures to prevent the disease: a) promotion of healthy lifestyles among the population; b) active early detection of risk factors that predetermine the possibility of developing the

disease and their correction. SECONDARY prevention: a) active detection of diseases at early stages of development (preventive examinations, screening); b) non-drug correction and timely adequate drug treatment of a newly diagnosed disease with drugs of proven efficacy. TERTIARY prevention: a) prevention of acute and chronic complications, timely examination of patients, monitoring of necessary laboratory and instrumental studies, continued correction of existing risk factors and basic treatment with drugs of proven efficacy, dynamic observation; b) continuation of treatment and high-quality rehabilitation of an existing complication.

10. REDUCING THE COST OF MEDICAL CARE TOGETHER WITH INCREASING ITS QUALITY - if the patient is regularly and effectively observed by a family doctor, then a large number of diseases can be prevented or detected and cured at an early stage, resulting in a significant reduction in costs.

GENERAL PRACTITIONER - FAMILY PHYSICIAN - ethen a specialist providing quality PHC to the attached population, regardless of gender, age, nationality, race, religion, social status and type of disease. He is a qualified doctor with deep and consistent knowledge, skills to prevent and treat the most common problems, pathological conditions and diseases among the population, timely, competently and purposefully exercising a certain amount of REQUIRED practical skills. The work of a family doctor requires a wide variety of knowledge, skills and abilities; this is also clinical experience in order to correctly assess the situation; and knowledge of pathophysiology, to understand the reasons for the deterioration or improvement of the patient's condition; and clinical trial data to assess prognosis and adjust treatment; knowledge of the deontology and psychology of the patient, to help him make arrangements in case of loss of independence. All this knowledge will be needed at the same time: to know one thing in such a situation is absolutely not enough; to feel confident, a general practitioner must constantly take care of the completeness of his knowledge; then at the right time he will be able to select what the patient needs and solve his problems at a high level.

The specifics of the work of the GP:

- It consists in the need to solve for each patient the most difficult task of clinical medicine: WHAT WITH THE PATIENT?
- This is the cardinal difference between the methodology of thinking of a family doctor and a “narrow specialist”.
- The latter decides the question: is the patient mine or not mine?
- If the patient is “not mine”, then interest in him is lost, he is “transferred” to a specialist of a different profile.
- For a family doctor, the patient is “ALWAYS mine”, but for a short time he may need the help of other specialists (Which specialists? And when? - the GP decides, for this he needs both knowledge, skills, and CLINICAL EXPERIENCE).
- The role of the GP in solving the problems of the patient is unique, as he, following the principles of family medicine, is in constant coordination and integration with the population.

Why a family doctor and not a narrow specialist? We believe that the future belongs to family medicine! The way it is: economical; meets the needs of the patient; diagnoses quickly and correctly; focuses on prevention; treats in the context of the family; treats the patient as a whole. I am not against narrow specialists, but the problem is different - when one says that he is a brilliant cardiologist, and the other, for example, a great gastroenterologist, it turns out that our unfortunate body is divided into parts. But how can one divide if the body is a single whole?!Turning to the district police officer, we are sent to see various narrow specialists, and we find ourselves in some kind of vicious circle, and even if we manage to get out of this, we are prescribed at least 10 drugs. You can't take 10 types of medicines at once, besides, it hits your pocket very hard. But this process can be regulated by a family doctor. He serves all family members, regardless of age, gender and stage of a particular disease, understands all common diseases among the population (and most importantly, he knows how to prevent or detect diseases in the early stages). At the same time, in case of difficulty, the family doctor can involve a narrow specialist, he does not ignore them. By referring his patient to a narrow specialist, the family doctor is sure that he will return to him and the family doctor will be able to adjust the prescription, which was appointed to him by a narrow specialist, taking into account the characteristics of his body or chronic diseases. This is the advantage of a family doctor, this person becomes a confidant of the family.

Communication between the family doctor and the patient. The ability to build relationships with the patient is necessary for a doctor of any specialty. Without a trusting relationship, neither diagnosis, nor treatment, nor prevention is possible. A conversation with a patient allows the doctor to solve a number of problems - to strengthen trusting relationships, obtain the data necessary for a diagnosis, develop a treatment plan, provide the patient with the necessary information, convince him to give up bad habits and lead a healthy lifestyle. The general practitioner should possess the following qualities: high awareness, the ability to hear the unsaid, the ability to ask the right questions that require a detailed answer, the ability to show the patient that he is highly valued and respected, the ability to understand what the patient feels and knows, the ability to facilitate decision making for the patient, the ability to win over, the ability to understand the position of the patient,

In conclusion, I would like to quote the words of Antoine Saint-Exupery, who said:

- "I believe the day will come when man will give himself into the hands of chemists and physicists. Without asking him anything, they will take the blood, consult the table and cure with one pill.
- And yet, if I get sick, I will turn to some old doctor. He will look at me out of the corner of his eye, feel my pulse and stomach, listen. Then he rubs his chin and smiles at me to better remove the pain. Of course I admire science, but I doubly admire WISDOM."

List of used literature

1.A. Gadaev "Umumiy amalyot vrachlari uchun maruzalar tuplami", 2011

2.J.Murta “Reference book of the general practitioner”, 1991
3. J. Nobel “General medical practice according to John Nobel”,2001
4. Order No. 80 dated March 23, 2009 of the Ministry of Health of the Republic of Uzbekistan
5. Normative documents on the activities of agriculture
medical center, 2009
6. *www.emcmos.ru/ru/departments/69/*
7.family-med.web.-3.ru/medicina/
8.www.ty-doktor.ru/abst_family_medicine

LECTURE TOPIC: «CHEST PAIN SYNDROME. DIFFERENTIAL DIAGNOSIS OF CORONARY PAIN IN THE CHEST. FEATURES OF THE COURSE OF IHD, MYOCARDIAL INFARCTION. TREATMENT. PREVENTION. »

TRAINING TECHNOLOGY

Number of students-	Time - 2 hours
Form of the lesson	Lecture - visualization
Lecture plan	1. Blood supply to the heart. Definition of the concept of vascular atherosclerosis 2. Causes and risk factors leading to the development of atherosclerosis with subsequent development of coronary heart disease 3. IHD classification 4. The concept, definition and clinical course of various types of coronary artery disease 5. Features of the course of acute myocardial infarction, GP tactics, emergency care 6. Diagnosis of diseases accompanied by chest pain 7. Principles of treatment, prevention and clinical examination of patients with coronary heart disease
The purpose of the lesson: to familiarize students with the etiology, pathogenesis of diseases accompanied by coronary chest pain, to acquaint students with the modern classification of coronary artery disease, to teach the principles of diagnosis, treatment, prevention and medical examination of diseases accompanied by coronary chest pain, to familiarize with the features of the course, tactics of GP in case of myocardial infarction	
Pedagogical tasks: 1. Strengthen and deepen students' knowledge of diseases accompanied by	The results of the educational process: The GP needs to know: 1. Risk Factors for Coronary Heart Disease

coronary chest pain, familiarize MI with risk factors 2. To teach students to correctly establish a diagnosis in accordance with the modern classification 3. To teach students the ability to differentiate diseases accompanied by coronary chest pain 4. To acquaint students with the features of the course of myocardial infarction, the provision of emergency care, the tactics of GP 5. To teach students the management of patients with coronary chest pain, treatment and prevention steps	2. Diseases accompanied by coronary pain in the chest 3. IHD classification 4. Principles of differential diagnosis of diseases accompanied by coronarogenic pain in the chest 5. Tactics of management, principles of treatment of patients with myocardial infarction 6. Principles of prevention and medical examination of patients with coronarogenic chest pain
Teaching methodology	Lecture text, videos, questionnaires, questions, “yes-no” technique
Form of study	Laser projector, visual materials, special technical equipment, display of MIatic patients, ECG of patients
Means of education	team
Conditions for the educational process	Audience

TECHNOLOGICAL CARD LECTURES

Stages, time	Activity	
	Teacher	students
Stage 1 Introduction (5 minutes)	1. Tells about the topic of the lecture, its purpose and plan	1. Listen
Stage 2 Knowledge update (20 minutes)	2.1. In order to increase the actualization of students' knowledge, asks questions: 1. Define the terms atherosclerosis, ischemic heart disease? 2. List the diseases accompanied by pain in the chest	2.1. Answer questions asked

	3. List risk factors leading to chest pain 4. List the groups of drugs used to treat IHD 5. What is myocardial infarction? Clinical variants of the course of myocardial infarction. GP tactics Conducts a survey 2.2. Showing on the screen invites students to familiarize MIselves with the goals and objectives of the lecture. Slide #1, #2	2.2. Study slide number 1 2.3. Study slide number 2
<u>Stage 3</u> Main part (informational) (55 min)	3.1. Introduces students to the lecture material, the importance of the topic and the principles of the formation of an intelligent cultural personality, in particular the GP. In order to increase the actualization of knowledge, he conducts a quick survey of students: 1. According to 1 point of the lecture plan: tell the blood supply to the heart. Define the concept of atherosclerosis 2. According to paragraph 2 of the lecture plan: List the causes and risk factors leading to the development of atherosclerosis with the subsequent development of coronary heart disease 3. On the 3rd point of the lecture plan: Classification of coronary artery disease 4. According to the 4th point of the lecture plan: Define coronary artery disease and tell the clinical course of various types of coronary heart disease	3.1. Together they analyze the listened lecture material, ask questions Key points are written in a notebook.

	5. According to the 5th point of the lecture plan: Features of the course of acute myocardial infarction, GP tactics, emergency care 6. According to the 6th point of the lecture plan: Diagnosis of diseases accompanied by pain in the chest 7. According to paragraph 7 of the lecture plan: Principles of treatment, prevention and medical examination of patients with coronary heart disease Stopping at the important points of the lecture, he suggests writing down the main points in a notebook	
Stage 4 Final (10 min)	4.1. Asking questions: 1. List the most common diseases accompanied by coronary pain in the chest 2. Tell the modern classification of coronary heart disease 3. Tell the main key points of the clinical course of various types of coronary artery disease 4. Name the basic principles of treatment, prevention and rehabilitation of patients with acute myocardial infarction 4.2. Gives a task for independent work of students: Atypical forms of the clinical course of myocardial infarction, complications	4.1. Answer questions 4.2. Listen, write

Ischemic heart disease (IHD) is a myocardial disease caused by an acute or chronic discrepancy between myocardial oxygen demand and the real coronary blood supply to the heart muscle, which is expressed in the development of ischemia, ischemic damage, necrosis and scar fields in the myocardium and is accompanied by a violation of systolic and / or diastolic function hearts. ischemic heart disease is

one of the most common diseases of the cardiovascular system in all economically developed countries. According to prospective studies, ischemic heart disease about 5–8% of men aged 20 to 44 and 18–24.5% of men aged 45 to 69 are affected. Prevalence ischemic heart disease in women it is slightly less and in the older age group it usually does not exceed 13-15%. To share ischemic heart disease accounts for more than half of all deaths from cardiovascular disease. The Russian Federation has one of the highest prevalence and mortality rates in Europe from ischemic heart disease. It should also take into account the great socio-economic significance ischemic heart disease, which leads to relatively early disability and disability of patients.

Morphological basis ischemic heart disease more than 95–97% of cases is atherosclerosis of the coronary arteries (CA). Atherosclerotic plaques narrowing the lumen of the coronary vessels are localized mainly in the proximal (epicardial)CA, mainly in the region of their mouth. At the same time, intramural coronary vessels, at least macroscopically, remain intact.

Risk factors of ischemic heart disease. Mention should also be made of the significance in the formation ischemic heart disease risk factors (FR) identical to the RF of atherosclerosis. Recall that the most significant of MI are:

1. Non-modifiable (unchangeable) risk factors: age over 50–60 years; gender (male); burdened heredity.

2. Modifiable (changeable): dyslipidemia (increased levels of cholesterol, triglycerides and atherogenic lipoproteins in the blood and / or a decrease in the content of anti-atherogenic HDL); arterial hypertension (AG); smoking; obesity; carbohydrate metabolism disorders (hyperglycemia, diabetes mellitus); hypodynamia;

irrational nutrition; hyperhomocysteinemia, etc.

It has now been proven that such risk factors have the greatest prognostic value. ischemic heart disease like dyslipidemia, AG smoking, obesity and diabetes.

Working classification ischemic heart disease

1.Sudden cardiac death (primary cardiac arrest).

2.Angina.

2.1. Stable exertional angina (indicating the functional class from I to IV).

2.2. Unstable angina:

2.2.1. New onset angina (AFS).*

2.2.2. Progressive angina (PS).

2.2.3. Early postinfarction or postoperative angina pectoris.

2.3. Spontaneous (vasospastic, variant, Prinzmetal) angina.**

3.Painless myocardial ischemia.**

4.Microvascular angina ("syndrome X").

5.Myocardial infarction.

5.1. Q-wave myocardial infarction (large-focal, transmural).

5.2. Myocardial infarction without Q wave (small focal).

6.Postinfarction cardiosclerosis.

7.Heart failure (indicating the form and stage).

8.Cardiac arrhythmia and conduction disorders (indicating the form).
Note:* sometimes for the first time angina pectoris from the very beginning has a stable course; ** Some cases of silent myocardial ischemia, as well as severe attacks of spontaneous angina pectoris, can be classified as unstable angina.

Stable exertional angina is one of the most common clinical forms ischemic heart disease. It occurs, as a rule, against the background of stenosing coronary atherosclerosis in the presence of large epicardial coronary arteries "uncomplicated" atherosclerotic plaque, which has a dense and durable connective tissue capsule. In these cases, the onset of exertional angina is usually provoked by an increase in myocardial oxygen demand, which is not accompanied by an adequate expansion of the resistive CA(arteriole). Less important is the spasm of the coronary vessels. Recall that among the factors contributing to the increase in myocardial oxygen demand include:

- increase in heart rate (heart rate);
- increased inotropism (contractility) of the heart muscle, more often associated with an increase in activity SAS;
- increase in afterload and, accordingly, systolic pressure in the cavity LV(for example, when increasing AP-arterial pressure);
- increased preload and end-diastolic volume LV;
- increase in myocardial mass LV(hypertrophy of the heart muscle).

Thus, exertional angina is provoked not only by physical activity (fast walking, running, climbing stairs), but also by any other factors that increase myocardial oxygen demand: emotional stress, stress, increased AP, increased venous flow to the heart, heart failure (volume overload LV), tachycardia of any origin, etc.
The first clinical manifestations ischemic heart disease may be different. According to the Framingham Study, approximately 40% of men and 56% of women present with stable angina pectoris. In these cases, angina often begins gradually, and its intensity slowly increases. From the onset of the disease, it usually takes several weeks or months before the patient sees a doctor.

Clinical picture. The pain syndrome in angina pectoris is paroxysmal in nature, appearing, as a rule, against the background of a relatively favorable condition of the patient, which makes the patient immediately pay attention to it, often causing marked anxiety and fear.
In typical cases, the pain is localized behind the sternum, usually in the region of its upper and middle thirds. Less commonly, pain occurs in the region of the apex of the heart, to the left of the sternum in the II-V intercostal space, under the left shoulder blade, or even in the left arm, collarbone or in the left half of the lower jaw (atypical localization of pain).
The nature of the pain is usually burning, squeezing, pressing. Sometimes patients describe angina as "a feeling of discomfort in the chest." During an attack of angina pectoris, patients, as a rule, are laconic and indicate the localization of pain with a palm or fist pressed to the sternum (Levin's symptom)

New onset angina is diagnosed in those cases when attacks of angina pectoris of exertion and / or rest first appeared in a patient no more than 1 month ago. At

first, attacks may resemble the pain of stable exertional angina. In typical cases, pain attacks occur against the background of physical or psycho-emotional stress, especially in cold and windy weather, are localized behind the sternum, radiate to the left arm, shoulder blade, shoulder. The duration of pain, as a rule, does not exceed 1-5 minutes. Pain is relieved by nitroglycerin and/or cessation of the precipitating factors.

However, very soon, some patients with new onset angina notice that attacks of anginal pain are repeated more and more often and become more intense and prolonged. Over a short period of time (1-4 weeks), they lead to a marked decrease in exercise tolerance. Sometimes pains appear at rest and are accompanied by a feeling of lack of air, severe weakness, sweating, dizziness. It should be remembered that each such anginal attack in patients with the described unstable course of new onset angina may result in the development MI or even sudden death.

Strictly speaking, new onset angina is not synonymous with unstable angina pectoris and may not always be associated with complicated atherosclerotic plaque. In these cases, new onset angina is characterized by a more “calm” stable course: angina attacks, although they recur, are usually provoked by significant physical or psycho-emotional stress, the tolerance of patients does not decrease for a long time. At the same time, slowly developing stenosis occurs from the very beginning. CA, which at a certain stage becomes only hemodynamically significant and is manifested for the first time by angina pectoris, which immediately becomes stable. After 1–1.5 months from the onset of the disease, such a course ischemic heart disease regarded as stable exertional angina.

Nevertheless, the nature of the further course of new onset angina(stable or unstable) in a patient with recent anginal attacks is very difficult to predict. Therefore, each such case requires the doctor to pay close attention to the development of all the symptoms of the disease. There is no doubt that every patient with new onset angina should be observed in a cardiological hospital, where he can be provided with timely medical care and an appropriate examination.

Considering the features of the pain syndrome in new onset angina, both stable and unstable, one should keep in mind the relatively high incidence in these patients, especially in young people, of atypical angina, resembling a clinic of autonomic dysfunction. Pain in the region of the heart can have a significant duration, often localized not behind the sternum, but in the region of the apex of the heart, to the left of the sternum or in the epigastrium, often provoked not by physical activity, but appear spontaneously, which emphasizes the importance of dynamic occlusion of the coronary artery due to its spasm.

Progressive exertional angina always regarded as unstable angina-UA. It occurs, as a rule, in patients with a more or less long history of stable exertional angina, indicating an "exacerbation" of the disease. To diagnose UA, one should focus primarily on the qualitatively changed nature of the pain syndrome:

1. Patients experience a significant increase in the duration and intensity of angina attacks.

2. Pain attacks are provoked by less and less physical or psycho-emotional stress, indicating a change in the functional class of angina pectoris (IIIFC).
3. Attacks of rest angina pectoris join angina pectoris (if they were not there before) (IVFC).
4. The effectiveness of nitroglycerin and other antianginal drugs previously used by patients for the relief or prevention of angina pectoris is noticeably reduced.

rest angina

Severe and prolonged attacks of rest angina (if they were not previously detected in this patient) are one of the most dangerous clinical variants of UA in terms of prognosis. According to some reports, the frequency of development MI within 1-2 months from the moment of occurrence of the first such anginal attacks reaches 40-50%, and mortality - 11-15%.

In fact, this form can be detected both in patients with progressive exertional angina (in cases where angina pectoris is accompanied by rest angina), and in patients with new onset angina and Prinzmetal's angina. It is also possible the sudden appearance of such pain attacks against the background of a relatively stable course of the disease.

This form of UA is clinically manifested by repeated and severe attacks of rest angina lasting more than 15–20 minutes. Intense retrosternal pain is often accompanied by acute weakness, sweating, shortness of breath, transient rhythm and conduction disturbances, and / or a sudden decrease in AP. The pain usually appears at rest, without previous exertion, and becomes increasingly refractory to nitroglycerin. For its relief, the use of narcotic analgesics is often required. At the same time, exercise tolerance drops sharply.

Severe and frequent attacks of spontaneous (variant) Prinzmetal's angina also refer to a very unfavorable prognostic form of UA. According to some authors, 1/4 - 1/2 of patients with spontaneous variant angina pectoris develop over the next 2-3 months MI or sudden cardiac death.

Early postinfarction angina

Early postinfarction angina pectoris occurring within 48 hours to 2 weeks from the onset of acute MI, according to its prognostic value also refers to UA. The resumption of anginal attacks in the early postinfarction period significantly worsens both the immediate and long-term prognosis. MI: by the end of the first year, the frequency of relapses MI reaches 50%, and lethality - 17%. Early postinfarction angina usually indicates incomplete thrombolysis in the occluded CA and about the ongoing formation of a thrombus, including in other vascular areas, if there is a multivessel lesion CA.

Clinically, anginal pain in these patients is not much different from ordinary angina pectoris, and there are both cases of severe angina pectoris at rest and / or low exertion, refractory to antianginal therapy, and cases of mild anginal pain provoked only by excessive physical exertion. However, in all these cases, the early resumption of angina pectoris in a patient with MI regarded as UA.

Thus, a thorough analysis of the main characteristics of the pain syndrome and other anamnestic data in most cases makes it possible to distinguish among patients ischemic heart disease patients with unstable disease.

The term "unstable angina" (UA) proposed by N. Fovler and S. Conty et al. is currently used to refer to the most severe period of the course ischemic heart disease, which is characterized by rapid progression of coronary insufficiency and a high risk of developing MI and sudden cardiac death (up to 15–20% within 1 year). Selection of this form ischemic heart disease, undoubtedly, is of great practical importance, as it directs the doctor to the earliest possible detection and intensive treatment of patients with a high risk of fatal complications.

The following clinical forms are classified as unstable angina pectoris.

1.For the first time, angina pectoris of an unstable course (within 1 month after the onset of the first attack of angina pectoris).

2.Progressive angina pectoris (a sudden increase in the frequency, severity, duration of angina attacks in response to the usual physical activity for this patient, a decrease in the effectiveness of nitroglycerin and other drugs previously successfully used by the patient).

3.Severe and prolonged attacks of rest angina (more than 15–20 minutes), including severe cases of spontaneous (variant) angina pectoris.

4.Early postinfarction and postoperative (after coronary artery bypass grafting, transluminal angioplasty, etc.) angina pectoris.

In some cases, other forms can be attributed to the National Assembly ischemic heart disease described in the previous chapter, for example MI and microvascular form of angina pectoris, severe and prolonged attacks of Prinzmetal's vasospastic angina pectoris.

The term “acute coronary syndrome” was introduced into clinical practice in the late 1980s, when it became clear that the use of certain active methods of treatment (for example, thrombolytic therapy or primary coronary angioplasty) should be decided before establishing the final diagnosis - the presence of or lack MI (Russian recommendations of the committee of experts VNOK, 2001). In other words, acute coronary syndrome is only a preliminary diagnosis that helps to choose the optimal tactics for managing patients in the very first hours from the onset of an exacerbation of the disease, when to accurately confirm or reject the diagnosis. MI or UA is not possible.

Depending on the results of the initial clinical examination and registration ECG in 12 leads, patients with acute coronary syndromes can be assigned to one of two categories of patients with an exacerbation ischemic heart disease:

1.Acute coronary syndrome with persistent elevation of the RS–T segment or “new”, first-time blocade of the left bundle branch block.

2.Acute coronary syndrome without persistent RS–T elevation.

It was found that more than 2/3 of patients with acute coronary syndrome with persistent RS–T segment elevation or “new” block develop MI, and in the vast majority of cases – transmural MI with Q wave (Savantto et al., 1999). Only in a

small percentage of cases, the outcome of this form of acute coronary syndrome is NS. Therefore, the main goal of treating these patients, even before establishing an accurate diagnosis MI, is the fastest possible and complete restoration of coronary blood flow using thrombolytic therapy or primary angioplasty (see below).

Acute coronary syndrome without persistent RS–T elevation. Patients with chest pain and/or sudden onset ECG changes indicative of acute myocardial ischemia are classified as acute coronary syndromes without persistent RS-T elevation. In these patients, the resting ECG may show persistent or transient RS–T depression and/or T wave inversion, but no persistent RS–T rise. In some cases ECG may turn out to be little changed, while in other cases, on the contrary, the diagnosis of this category of acute coronary syndrome can be made in the presence of painless ischemic changes.

The occurrence of acute myocardial ischemia in these patients is based on the formation of a non-occlusive parietal, predominantly platelet ("white") thrombus, and, as a rule, in the area of the complicated atherosclerotic plaque. Subsequently, in most patients with acute coronary syndrome without a persistent rise in the RS–T segment either unstable angina or acute MI without Q wave. These two forms ischemic heart disease (UA and MI without Q wave) differ from each other in the absence or presence of markers of necrosis (elevated levels of troponins, CK-MB and myoglobin).

It has been established that in patients with acute coronary syndrome without a persistent rise in the RS–T segment, the use of thrombolytic therapy is ineffective. Treatment of such patients should be aimed at eliminating severe myocardial ischemia and preventing the process of further thrombosis.

The main goal of treating patients with acute coronary syndrome without a persistent rise in the RS–T segment, which later transforms into UA, is to reduce the risk of MI and sudden death and reducing the consequences of acute widespread myocardial ischemia LV(rhythm and conduction disturbances, progression HF and etc.). According to the recommendations of the European Society of Cardiology (2000) and the Russian recommendations of the expert committee of VNOK (2001), the following drug and non-drug effects can be used for this purpose.

1. Anti-ischemic (antianginal)LS:
   - β-blockers;
   - nitrates;
   - blockers of slow calcium channels.
2. Antithrombin drugs:
   - heparins (unfractionated and low molecular weight);]
   - direct inhibitors of thrombin.
3. Antiplatelet agents:
   - aspirin;
   - receptor antagonists to adenosine diphosphate (thienopyridines);
   - blockers of glycoprotein IIb/IIIa platelet receptors.
4. Coronary revascularization:
   - transluminal coronary angioplasty;

- aorto-coronary bypass.

Treatment of patients with the listed methods should be started immediately, as soon as the clinical data and the results of an ECG study at rest give the impression of the presence of an acute coronary syndrome without a persistent rise in the RS–T segment (the most characteristic of UA) and a clinical assessment of the risk of occurrence MI and sudden death. With the resumption of pain attacks, nitroglycerin is indicated, including its intravenous administration, and, if necessary, the use of intravenous infusions of β-blockers and narcotic analgesics.

It should be emphasized that in acute coronary syndrome without a persistent rise in the RS–T segment, i.e. in the vast majority of patients with UA, the use of thrombolytic therapy is not recommended, because, while increasing the risk of hemorrhagic complications, this method of treatment does not reduce mortality and the incidence of MI, and according to some data even increases MI. A completely different approach can be traced in relation to patients with acute coronary syndrome with persistent RS-T segment elevation or "new" bundle branch block, in which this method of treatment is the method of choice.

In the absence of contraindications, β-blockers are recommended to be prescribed to all patients with NS. The positive effect of these LS due mainly to their negative inotropic and chronotropic effects. A decrease in myocardial oxygen demand and a decrease in heart rate caused by β-blockers is usually accompanied by a clear anti-ischemic effect and reduces the risk of subsequent development by about 13%.MI.

Since there is no evidence of greater effectiveness of one or another β-blocker in the treatment of NS, the choice of a particular drug should be based primarily on taking into account the individual clinical situation: the presence of concomitant diseases (AG or arterial hypotension, diabetes mellitus, lung diseases, etc.), the patient has signs of dysfunction LV, the presence of sinus tachycardia or bradycardia, etc. We only recall that in the treatment of patients with NS, the appointment of β-blockers should be avoided in severe acute CH, concomitant bronchial asthma, severe arterial hypotension, sinus bradycardia (< 50-55 beats per minute) and AV blocCAde II, III and I degree (at least when P-Q (R) > 0.24 s). With caution, drugs are used for chronic lung diseases; in these cases, it is advisable to use short-acting cardioselective β 1-blockers in reduced doses.

Patients at high risk of developing MI and sudden death (see above), intravenous drip administration of β-blockers is indicated, followed by their transfer to oral administration of these drugs. It should be remembered that in patients with a tendency to spasm CA β-blockers should be used with great caution and are contraindicated in Prinzmetal's vasospastic angina. The exception is β-blockers with additional vasodilating properties (carvedilol).

Nitrates are used in patients with UA primarily for the purpose of hemodynamic unloading LV and reduction or relief of disease symptoms (eg, persistent pain in the region of the heart, shortness of breath, and other signs of left ventricular failure). In the latter case, nitrates are prescribed parenterally, gradually increasing the dose until the symptoms of the disease are relieved or side effects appear (headaches, arterial hypotension).

The initial rate of intravenous infusion of nitroglycerin solution is 10 μg/min. It then increases by 10 μg/min every 3–5 min until a reaction occurs. AP or changes in symptoms. If the pain in the heart and other signs of ischemia decrease or disappear, then the rate of increasing the dose of the drug does not increase. If there is a corresponding reaction AP, and the symptoms of acute myocardial ischemia persist, then the rate of drug administration is increased at large time intervals and under constant monitoring AP. Special care is required when the systolic AP becomes below 110 mm Hg. Art. or in a patient with arterial hypertension - 25% below baseline. If pain and other signs of myocardial ischemia do not occur within 12 hours, you should try to reduce the dose and start switching to other dosage forms of nitrates (orally, buccally, transcutaneously), taking into account the possibility of side effects and the presence of contraindications to the use of nitrates.

Blockers of slow calcium channels (calcium antagonists) are prescribed to patients with UA mainly for symptomatic purposes, since, having a vasodilating effect (especially drugs of the nifedipine group and diltiazem) and reducing myocardial contractility and its oxygen demand (verapamil and diltiazem), they still do not prevent the development of acute MI and do not reduce the mortality of patients with NS. Calcium antagonists are especially indicated in patients with Prinzmetal's variant vasospastic angina, as well as in the presence of contraindications to the appointment of β-blockers (for example, with concomitant bronchial asthma).

Verapamil and diltiazem are indicated in patients with signs of hyperactivation SAS, sinus tachycardia and the need to reduce myocardial oxygen demand. Drugs of the nifedipine group, which have the most pronounced vasodilating effect, are indicated for concomitant AH and Prinzmetal's vasospastic angina. When using calcium antagonists of the nifedipine group, drugs with a prolonged effect should be used, since there is evidence that the systematic use of short-acting nifedipine drugs may be accompanied by an increase in mortality of patients ischemic heart disease.

The main goal of treating patients with UA is to prevent further progression of coronary thrombosis and the development of acute MI. Currently, several groups are used for this purpose preparations, affecting the key links in the process of thrombosis:

1. Antiplatelet drugs:
   - aspirin;
   - receptor antagonists to adenosine diphosphate (thienopyridines);
   - blockers of glycoprotein IIb/IIIa platelet receptors.
2. Antithrombin drugs:
   - indirect thrombin inhibitors (unfractionated and low molecular weight heparins);
   - direct inhibitors of thrombin (hirudin, etc.).

Antiplatelet drugs. All patients with UA are recommended to prescribe aspirin (acetylsalicylic acid), which is the “gold standard” of antiplatelet therapy. As with stable formsischemic heart disease, it is advisable to prescribe small doses of aspirin (75–325 mg per day), since it is at this dosage that aspirin inhibits platelet

cyclooxygenase and, preventing the formation of thromboxane A2, has almost no effect on the metabolism of arachidonic acid in the vascular endothelium and does not reduce the production of prostacyclin. Patients with acute coronary syndrome should start taking aspirin as soon as possible. For example, at the first contact of an ambulance doctor with a patient or upon admission to a hospital, along with measures to relieve pain, the patient is recommended to chew 1 uncoated aspirin tablet (250-500 mg). Subsequently, they switch to taking aspirin orally 1 time per day (75-325 mg).

Thienopyridines. In patients with intolerance to aspirin, patients with NS can be prescribed ticlopidine (ticlid) or clopidogrel, which effectively inhibit platelet adhesion and aggregation. They exhibit antagonistic properties AP F-receptors of platelets and, according to some data, inhibit the activity of IIb / IIIa receptors. Since ticlopidine has a large number of side effects, including the occurrence of neutropenia and thrombocytopenia, it is preferable to use clopidogrel, which is associated with significantly fewer adverse events. It should be remembered that the therapeutic effect of ticlopidine and clopidogrel does not occur immediately; recommend at the beginning of treatment the so-called loading doses of theseLS, for example, for clopidogrel - 300 mg per day once, followed by 75 mg per day.

Since the mechanisms of action of aspirin and clopidogrel are different, a combination of these two is possible.LS, which in patients with NS is more effective in preventing MI and sudden death compared with aspirin alone. In the absence of contraindications in patients with UA from the first days of the disease, it is recommended to use just such a combination of aspirin and clopidogrel. Curantyl (dipyridamole), which also has a pronounced antiplatelet effect, is recommended for use in patients with stable exertional angina I–IIFC. Patients with UA and MI Curantyl is not usually prescribed, although there are reports of the successful use of this drug in some patients with NS.

Platelet glycoprotein IIb / IIIa receptor blockers are a relatively new group of antiplatelet drugs that prevent the formation of a platelet thrombus at the final stage of this process. Recall that activated IIb/IIIa platelet receptors interact with the fibrinogen molecule, contributing to the formation of a dense platelet clot, as if penetrated by fibrin threads. Currently, three drugs belonging to this class of antiplatelet agents have passed comprehensive clinical trials.LS:

- abciximab (monoclonal antibodies to glycoprotein IIb / IIIa platelet receptors;
- eptifibatide (synthetic cyclic peptide);
- tirofibatide (non-peptide selective IIb/IIIa platelet receptor blocker).

All three drugs are administered intravenously. Blockers of IIb/IIIa platelet receptors for oral use (orbofiban, sibrofiban, etc.) have also been created. Clinical trials of infusion blockers IIb/IIIa platelet receptors have shown that the addition of these drugs to heparin therapy in patients with UA reduces the risk of MI and mortality from cardiovascular causes. Especially shown is their use during coronary angioplasty and coronary artery bypass surgery.

Antithrombin agents

Practically in all patients with acute coronary syndrome, including patients with MI, there is a need to use antithrombin drugs. These include:

- indirect thrombin inhibitors (unfractionated and low molecular weight heparins);
- direct inhibitors of thrombin (hirudin, etc.).

Unfractionated heparin has been successfully used for many years to treat patients with acute MI. It turned out that its use is indicated in patients with UA. Heparin is a natural anticoagulant factor produced by mast cells. Heparin has an anticoagulant effect mainly due to its binding to antithrombin III, which is a natural inhibitor of thrombin. When combined with heparin, the activity of antithrombin III increases 700 times, and it inhibits some key activated blood coagulation factors: IIa, VIIa, Xa, XIa, XIIa Heparin also increases the activity of lipoprotein lipase, reduces the concentration of cholesterol and LDL, has anti-inflammatory, immunosuppressive, hypoglycemic and diuretic action (VG Kukes). Heparin can reduce SVR due to the expansion of resistive vessels, eliminates spasm CA. When using large doses, it can cause thrombocytopenia and leukopenia, increases the content of lymphocytes, monocytes and eosinophils.

In NS, heparin is used to prevent further thrombosis. CA. According to the recommendations of the European Society of Cardiology (2000) and VNOK of the Russian Federation (2001), in patients with acute coronary syndrome, unfractionated heparin is administered only intravenously - initially by bolus, in the form of a bolus at a dose of 60–80 U / kg (but not more than 5000 IU), and then with a long-term (48–72 h) intravenous infusion at a dose of 12–18 U/kg/h (but not more than 1250 U/kg/h). The APTT indicator should serve as a means of control. Doses of heparin are adjusted in such a way that 6 hours after the start of administration, the APTT is 1.5–2.5 times higher than the control (normal) indicator of this laboratory and subsequently steadfastly kept at this “therapeutic” level. If the indicated APTT level is determined in 2 consecutive measurements, the next measurement can be taken after 24 hours.

myocardial infarction (MI**)**is an ischemic necrosis of the heart muscle, which develops as a result of acute insufficiency of the coronary circulation.

MI is one of the most common causes of death and disability of the population, both in our country and abroad. In the Russian Federation annually MI develops in 0.2-0.6% of men aged 40 to 59 years. In men of the older age group (60–64 g), the incidence MI even higher and reaches 1.7% per year. Women get sick MI 2.5–5 times less often than men, especially in young and middle age, which is usually associated with a later (about 10 years) development of atherosclerosis in MI. After the onset of menopause (over the age of 55–60 years), the difference in the incidence of men and women decreases significantly.

In recent years, there has been an increase in the incidence MI especially among young and middle-aged people. Despite the widespread decline in hospital mortality from MI, the overall mortality from this disease is still high, reaching 30-50% of the total number of cases. Moreover, most of the deaths occur at the prehospital stage.

Modern classification MI provides for its division:

- by the size and depth of damage to the heart muscle;
- by the nature of the course of the disease;
- by localization MI;
- according to the stage of the disease;
- according to the presence of complications MI.

1. According to the size and depth of damage to the heart muscle, transmural and non-transmural are distinguished MI.

With transmural MI (myocardial infarction with a Q wave) the focus of necrosis captures either the entire thickness of the heart muscle from subendocardial before subepicardial layers of the myocardium), or most of it, which is reflected on the surface ECG in the form of the formation of a pathological Q wave or a QS complex in several electrocardiographic leads. Hence the synonym for transmural MI- "Q wave myocardial infarction". As a rule, such damage to the heart muscle is quite extensive and the focus of necrosis extends to 2 or more segments.LV(large focal MI).

With non-transmural MI (myocardial infarction without Q wave) the focus of necrosis captures only subendocardial or intramural departments LVand is not accompanied by pathological changes in the QRS complex ("myocardial infarction without a Q wave"). For a long time in the domestic literature to designate MI without a Q wave, the term "small focal MI". Indeed, in most casesMIwithout a Q wave is significantly less in extent than a transmural infarction, although there are often cases of extensivesubendocardial MIspanning several segmentsLV, but affecting onlysubendocardialmyocardial layers.

2. According to the nature of the course of the disease, primary, repeated and recurrent MI.

Primary MI diagnosed in the absence of anamnestic and instrumental signs of pastMI.

Repeated MI is diagnosed in those cases when a patient who has documented information about the pastMI, there are reliable signs of a new focus of necrosis, more often formed in the pool of other CA in periods exceeding 28 days from the date of the previous heart attack.

With recurrentMIclinical, laboratory and instrumental signs of the formation of new foci of necrosis appear in terms from 72 hours (3 days) to 28 days after developmentMI, i.e. until the end of the main processes of its scarring.

3. By localizationMIallocate:

- anterior septal (anterior septal);
- anteroapical;
- anterolateral;
- anterobasal (high anterior);
- common anterior (septal, apical and lateral);
- posterior diaphragmatic (lower);
- posterolateral;
- posterobasal;
- common posterior;

- MIright stomach.

MImore often localized inLV, hitting its front, back, side walls and/orinterventricular septum(VZHP), which depends on the location of critical stenosis or occlusion of one or anotherCA. Circulatory disorders inLADLCAmay lead to the developmentMIanterior septal region, apex and much less often - posterior diaphragmatic (lower) wallLV. The cessation of blood flowOVLCAaccompanied by the appearance of anterobasal, lateral or posterior basalMI(with occlusion of the distal partsOVLCA). In case of circulatory disorders in the pool PCAmay develop posterior diaphragmatic (with damage to the proximal PCA) or posterobasalMI(with occlusion of the distal parts of the PCA). Isolated right ventricular infarction is relatively rare. Often there is a combinationMIdifferent localization.

4. According to the stage of the course of the disease, they distinguish
   - the most acute period - up to 2 hours from the onsetMI;
   - acute period - up to 10 days from the onsetMI;
   - subacute period - from 10 days to the end of 4-8 weeks;
   - postinfarction period - usually after 4-8 weeks.
5. Among the most common complicationsMIrelate:
   - acute left ventricular failure (pulmonary edema);
   - cardiogenic shock;
   - ventricular and supraventricular arrhythmias;
   - conduction disorders (SA- blocCAde, AV blocCAde, blocCAde of the legs of the bundle of His);
   - acute aneurysmLV;
   - external and internal myocardial ruptures, cardiac tamponade;
   - aseptic pericarditis (epistenocarditis);
   - thromboembolism.

Physical research. The purpose of a physical examination of a patient with MI is not so much to establish a diagnosis of MI, which is confirmed mainly by laboratory and ECG data, but to assess the functional state of the cardiovascular system and timely diagnosis of severe complications of a heart attack.

Inspection. During a general examination, in the first minutes or 1–1.5 hours that have passed from the onset of the disease, attention is drawn to the pronounced excitement and motor restlessness of patients experiencing severe pain at this time. They try to change position, sometimes even walk around the room, sit in a chair, go to bed in search of a position that alleviates suffering. This feature, characteristic of patients with MI, distinguishes MI from patients with angina pectoris, who seem to freeze in place during a painful attack. After the relief of pain syndrome, the excitation of patients with developing MI, as a rule, disappears. The exception is cases of progressive left ventricular failure, accompanied by sudden onset and rapidly increasing shortness of breath and suffocation. On examination, pallor of the skin is often noted, cold extremities and severe sweating, indicating the possible development of acute vascular

insufficiency (short-term reflex pain shock) or the initial clinical manifestations of true cardiogenic shock. In most cases, cyanosis of the lips is determined. Severe cyanosis, the position of orthopnea in combination with moist fine bubbling rales in the lower parts of the lungs and an increase in the frequency of respiratory movements indicate the presence of acute left ventricular failure.

Palpation and percussion of the heart. On palpation of the heart, local tenderness in the left precordial region may be noted. With percussion, it is not possible to identify any noticeable expansion of the LV cavity in the most acute period of the disease. The exception is patients with diseases preceding a heart attack, accompanied by LV dilatation (AH, atherosclerotic and post-infarction cardiosclerosis, etc.).

Auscultation of the heart. In the most acute period of MI in patients, several auscultatory phenomena can be detected:

1. Weakening and muffled tone I at the apex, associated with a decrease in contractility of the ischemic LV myocardium.
2. Weakening of the II tone, due to a slowdown in the early diastolic relaxation of the left ventricle or a decrease in pressure in the aorta. With an increase in pressure in the pulmonary artery, due to venous congestion in the pulmonary circulation, the emphasis of the II tone on the pulmonary artery is determined. Finally, in some patients with myocardial infarction and severe atherosclerotic aortic thickening, an accent of II tone on the aorta can be heard.
3. Sinus tachycardia, which in the first 2-3 hours from the onset of MI indicates, rather, not the presence of heart failure, but a pronounced activation of the SAS that occurs against the background of pain stress.
4. Sinus bradycardia, sometimes detected in patients with MI, is associated, on the contrary, with the predominance of the activity of the parasympathetic nervous system and the inhibition of automatism of the SA node, which is especially often observed in MI of the posterior phrenic region of the left ventricle. In addition, in more rare cases, bradycardia may be due to SA-block or AV block II and even III degree. Blood pressure in the first minutes and hours of MI may increase, which is often associated with increased activity of the SAS, increased concentration of catecholamines in the blood, resulting from pain and psycho-emotional stress. With the development of acute vascular insufficiency, blood pressure decreases, mainly due to systolic blood pressure. At the same time, there is a decrease in filling, tension and magnitude of the arterial pulse, as well as its increase.

Laboratory confirmation of acuteMIbased on the identification of: 1) non-specific indicators of tissue necrosis and inflammatory response of the myocardium; 2) hyperfermentemia; and 3) an increase in the content of myoglobin and troponins in the blood.

Nonspecific reaction of the body to the occurrence of acuteMIIt is associated primarily with the breakdown of muscle fibers, the absorption of protein breakdown products into the blood, and local aseptic inflammation of the heart

muscle, which develops mainly in the peri-infarction zone. The main clinical and laboratory signs reflecting these processes are:

1. An increase in body temperature (from subfebrile numbers to 38.5–39 ° C).
2. Leukocytosis, usually not exceeding 12-15 x 109 / l.
3. Aneosinophilia.
4. A small stab shift of the blood formula to the left.
5. Increase in ESR.

All patients with suspected developing Q-wave MI, i.e. patients with acute coronary syndrome and persistent elevation of the RS-T segment should be immediately hospitalized in the intensive care unit of specialized cardiology departments. Basic therapy, which is carried out in all patients with Q-wave MI, regardless of the presence or absence of certain complications, includes the following measures:

- pain relief (analgesia);
- thrombolytic therapy (of course, taking into account individual indications and contraindications);
- antithrombotic and antiplatelet therapy;
- oxygen therapy;
- use of anti-ischemicLS;
- the use of ACE inhibitors and angiotensin II receptor antagonists.

Thus, the basic principles of basic therapyMIwith Q wave (treatment of uncomplicatedMI) in many ways resemble those in patients with unstable angina pectoris.

DIFFERENTIAL DIAGNOSIS OF NON-CORONARY PAIN IN THE CHEST

In the practice of a general practitioner, one often has to deal with various situations accompanied by pain in the chest, which often require the doctor to quickly, effectively help patients. What should be the help in various specific situations depends on what kind of pathology the patient has - does it threaten the patient's life? What causes these pains? In this lecture, we will focus on some diseases that are accompanied by pain in the chest of a non-coronary nature. First of all, exclude the most dangerous diseases that threaten the life of the patient:

- myocardial infarction
- Spontaneous Pneumothorax
- TELA
- Dissecting aortic aneurysm

Only by excluding them, you should look for other causes of chest pain. So let's consider separately some pathologies accompanied by pain in the chest.

Acute dissecting aortic aneurysm. An aneurysm is a local saccular bulging of the aortic wall or diffuse expansion of the entire aorta by more than 2 times compared to the norm. The main causes: atherosclerosis, trauma, Marfan's syndrome, syphilis, cystic median necrosis.Acute dissection is the most common medical emergency associated with aortic disease. More commonly seen in men. Clinically, delamination is manifested by sudden intense pain in the chest or in the

back with irradiation along the aorta. The pain is characterized by an undulating course, which indicates further dissection of the aorta. In this regard, the localization and irradiation of pain gradually changes. The patient's condition is severe, resembling shock, but blood pressure is initially elevated (high blood pressure is one of the main causes of aortic dissection). With the spread of dissection, the development of hemipericardium with cardiac tamponade, aortic insufficiency due to aortic valve rupture, and ischemia of various organs is possible. Often there is asymmetry of the pulse and blood pressure in the upper and lower extremities. The diagnosis is confirmed by dynamic radiography (expansion of the aortic shadow, double contour), computed tomography, magnetic resonance imaging, echocardiography, dopplerography, aortography. Transesophageal echocardiography has a high diagnostic value in stratification of the thoracic region. The sensitivity and specificity of the method reaches 90%.

Forms of the course of aortic dissection: acute - hours, subacute - days (rarely 2-4 weeks), chronic - months. Without treatment, 70% of patients die in the first 2 weeks, 50% of survivors die within a year. The most common cause of death is aortic rupture.

Surgical treatment - emergency prosthetic aorta. Before the start of the operation and if acute delamination is suspected, medical treatment is immediately indicated. With high blood pressure, beta-blockers are prescribed, first intravenously, then orally. Next, sodium nitroprusside is needed. With normal blood pressure, monotherapy with beta-blockers is carried out in order to reduce the contractility of the left ventricle.

CARDIOMYOPATHY (CMP)- a group of diseases of the heart muscle of unknown etiology. They are also called primary or idiopathic cardiomyopathies. Secondary cardiomyopathies include diseases whose etiology is known. These are myocardial lesions in systemic diseases, alcohol poisoning, heart defects and other pathological conditions.There are three forms of primary cardiomyopathy: dilated, hypertrophic, and restrictive. A presumably causal role is assigned to various damaging factors: toxic, metabolic, and infectious. There are indications of immune disorders in dilated cardiomyopathy and the hereditary nature of the disease in hypertrophic cardiomyopathy with autosomal dominant inheritance.

DILATED CARDIOMYOPATHY (DCM)- a disease characterized by an increase in the size of the heart, systolic dysfunction and congestive heart failure.

clinical picture. Symptoms usually develop gradually. Sometimes the disease proceeds for months and even years asymptomatically, only with dilatation of the left ventricle, and is detected by radiography or echocardiography. The first signs of the disease are increased fatigue and weakness, then shortness of breath joins, initially during exercise, and in later cases - cardiac asthma, orthopnea. Peripheral edema and hepatomegaly are late and optional signs. Very frequent cardialgia and much less often - angina pectoris. A significant place in the clinical picture is occupied by thromboembolic complications that develop as a result of separation of intracardiac thrombi, as well as from the veins of the lower extremities. Sudden thromboembolism in the pulmonary artery system may also be the first symptom of

dilated cardiomyopathy. On examination - cold skin, alternating pulse, decrease in systolic and pulse pressure, expansion of the boundaries of the heart most often to the left, left ventricular pulsation, and in later cases - right ventricular. Auscultatory-systolic murmur due to mitral or, to a lesser extent, tricuspid regurgitation, gallop rhythm, cardiac arrhythmia. With severe dilatation of the left ventricle and a significant decrease in ejection fraction, the prognosis is unfavorable. The diagnosis is established on the basis of chest X-ray data, ECG, echocardiography, radionuclide ventriculography, coronary angiography, myocardial biopsy. X-rays show left ventricular enlargement, often cardiomegaly. There are signs of venous congestion in the lungs, interstitial and even alveolar edema, pleural effusion. ECG shows sinus tachycardia sometimes a variety of atrial and ventricular arrhythmias, as well as violations of intraventricular and atrioventricular conduction. A pathological Q wave may be detected, reflecting extensive non-coronary myocardial fibrosis, changes in the ST segment and T wave. Echocardiography shows an increase in the left ventricle with an increase in end-systolic and end-diastolic volumes, a decrease in ejection fraction, mitral and tricuspid regurgitation. Diffuse myocardial damage is characteristic. Radionuclide ventriculography confirms diffuse hypokinesia of the myocardial walls, an increase in heart volume, a decrease in ejection fraction; coronary angiography reveals normal vessels, it is necessary for patients with a pathological Q wave on the ECG for the differential diagnosis of myocardial infarction. In the study of biopsy material, extensive interstitial or perivascular fibrosis is determined, sometimes foci of calcifications. However, there are no specific diagnostic signs that are unique to dilated cardiomyopathy. Treatment. Medical treatment is aimed at combating heart failure. Its basic principles do not differ from the treatment of heart failure of any etiology and consist in controlling the content of sodium and fluid in the body, reducing preload and afterload, and improving the pumping function of the heart. Patients are advised to limit physical activity and reduce salt intake. Due to the risk of pulmonary and systemic thromboembolism in patients with DCM with mural thrombi in the left ventricle, as well as with atrial fibrillation and reduced ejection fraction (<20%), anticoagulant therapy with drugs such as warfarin should be used starting with a dose of 10 mg / day under constant monitoring of prothrombin time. For the treatment of cardiac arrhythmias in DCM, it is best to use the correction of hypoxia, electrolyte disorders and acid-base disorders (potassium preparations, antioxidants), as well as to combat heart failure. The use of antiarrhythmic drugs in patients with DCM did not increase life expectancy, although recently there have been reports of a beneficial effect with the use of β-selective adrenergic blockers (such as metoprolol-lopresor) or β-antagonists with vasodilating properties (bucindolol or carvedilol). In addition to antiarrhythmic action, they improve hemodynamic functions. In addition, carvedilol has an anti-apoptotic effect. In cases of sustained ventricular tachycardia, in the event of syncope or episodes of sudden death, the installation of an automatic implantable defibrillator is indicated. If heart failure is refractory to drug therapy, it is necessary to decide on heart transplantation.

HYPERTROPHIC CARDIOMYOPATHY- a myocardial disease characterized by asymmetric or symmetrical hypertrophy of the left ventricular myocardium with the obligatory involvement of the interventricular septum in the hypertrophic process. clinical picture. The disease is more common at a young age. Symptoms vary from asymptomatic forms to severe clinical manifestations and sudden death. In asymptomatic forms, a prerequisite for a detailed examination of patients is an accidentally detected systolic heart murmur or ECG changes. Patients with a family history of hypertrophic cardiomyopathy or sudden death deserve special attention. One of the most common symptoms is a variety of cardialgia: from rare stabbing pains to typical angina pectoris. The latter is a consequence of relative coronary insufficiency due to a mismatch between muscle mass and perfusion and depends on the degree of hypertrophy, or mechanical compression of the coronary arteries by the hypertrophied myocardium, as well as on impaired diastolic filling of intramural vessels due to impaired myocardial relaxation. The second most common symptom is arrhythmic syndrome (from 30 to 70%). The range of arrhythmias is extremely wide: these are disorders of atrioventricular conduction, and ventricular arrhythmias of various gradations, including paroxysms of ventricular tachycardia, and supraventricular arrhythmias, including atrial fibrillation, although the latter is not typical for this disease. According to the high frequency of arrhythmias, palpitations and interruptions in the work of the heart are a frequent complaint of patients with hypertrophic cardiomyopathy. One of the leading symptoms is fainting. Their occurrence is associated both with episodes of arrhythmia (paroxysms of ventricular and supraventricular tachycardia, complete atrioventricular block) and with low ejection syndrome. The frequency of syncope and pre-syncope conditions varies significantly - from multiple daily to single ones throughout life. Less commonly, patients with hypertrophic cardiomyopathy have signs of circulatory failure. Of these, shortness of breath is most often noted, while edema and liver enlargement are observed very rarely. On examination, an increase in the size of the heart is found, however, weakening of the first tone at the apex and the presence of systolic murmurs of various nature and intensity, more often along the left edge of the sternum, in the III-IV intercostal space, are not always considered typical. It is associated with obstruction of the left ventricular outflow tract due to hypertrophy of the upper third of the interventricular septum with anterior systolic movement of the anterior leaflet of the mitral valve and mesosystolic occlusion of the aortic valve. The diagnosis of hypertrophic cardiomyopathy is verified using electrocardiography, echocardiography, and chest x-ray. If it is necessary to detail the degree of damage to the myocardium and coronary arteries, sounding of the cavities of the heart and coronary angiography, dopplerography and biopsy of the endomyocardium are performed. The presence of a pathological Q wave on the ECG is considered a characteristic sign of hypertrophic cardiomyopathy. Usually it is determined in II, III, aVF and left chest leads, at the same time the R wave does not grow in the right chest leads. In some patients, an abnormal Q wave is recorded, starting from the right chest leads, while the ventricular complex may take the form of QS - these changes are most characteristic of asymmetric hypertrophy of the

interventricular septum. A typical ECG sign of the apical form of hypertrophic cardiomyopathy is giant negative T waves in the chest leads. Rhythm and conduction disturbances, described earlier, may be absent on a standard ECG, but, as a rule, are detected during its daily monitoring. Echocardiography is the main method for diagnosing hypertrophic cardiomyopathy. Thickening of the interventricular septum in its different parts more than 13 mm in combination with its hypokinesia (range of motion less than 3 mm) is considered a classic sign. Anterior systolic movement of the anterior leaflet of the mitral valve and partial mesosystolic covering of the aortic valve are also characteristic, a decrease in the cavity of the left ventricle in diastole. Radiography of the heart reveals typical signs of left ventricular hypertrophy, sometimes signs of left atrial enlargement. But in some patients, the radiograph does not differ from the normal one. Probing of the heart cavities and coronary angiography help to clarify the nature and degree of intracardiac hemodynamic disorders (to determine the pressure gradient between the left ventricle and the aorta, an increase in the end diastolic pressure in the left ventricle) and coronary circulation. For the same purpose, radioisotope research methods can also be used. Dopplerography (color Doppler scanning) allows you to non-invasively obtain fairly accurate information about the state of intracardiac hemodynamics. Biopsy of the endomyocardium examines 5 morphological features: a) short fibers, interrupted by connective tissue; b) large ugly nuclei; c) fibrosis; d) degenerating muscle with disappearance of myofibrils; e) chaotic arrangement of muscle fibers with turbulence.

Classification. In accordance with the localization of myocardial hypertrophy, the following morphological variants of hypertrophic cardiomyopathy are distinguished: 1. Idiopathic hypertrophic subaortic stenosis with disproportionate hypertrophy of the interventricular septum, obstruction of the outflow tract of the left ventricle, thickening of the endocardium under the aortic valve, thickening and paradoxical movement of the anterior leaflet of the mitral valve to the septum in systole. 2. Asymmetric septal hypertrophy without changes in the portal and mitral valves and without obstruction of the outflow tract of the left ventricle.

3. Apical hypertrophic cardiomyopathy with limitation of the hypertrophy zone by the apex area. 4. Symmetric hypertrophic cardiomyopathy with concentric hypertrophy of the left ventricular myocardium. Differential diagnosis is carried out with ischemic heart disease, valvular stenosis of the aortic orifice, pulmonary artery stenosis, ventricular septal defect, mitral insufficiency, and at the beginning of the disease - with neurocirculatory dystonia. The use of these research methods allows to exclude these diseases. Treatment of patients with hypertrophic cardiomyopathy is aimed at reducing the symptoms of the cardiovascular system with the help of drug therapy, and in case of resistance to it, surgical intervention is indicated. Of the medications, β-blockers and verapamil have found the greatest use in the treatment of hypertrophic cardiomyopathy. Their appointment is necessary both at the stage of clinical manifestations (pain, arrhythmia, shortness of breath), and in asymptomatic course due to their action to delay the progression of the disease by reducing the intraventricular pressure gradient and improving ventricular diastolic function. The

drugs are shown in usual doses; propranolol 120-160 mg/day, verapamil 240-480 mg/day. Perhaps the appointment and selective p-blockers. Patients with dangerous cardiac arrhythmias are justified in the appointment of amiodarone. Nitrates and vasodilators should be avoided because of the risk of increased left ventricular obstruction. Patients with atrial fibrillation need anticoagulant therapy, moreover, it should be continuous starting from the moment of registration of this rhythm disturbance (aspirin or warfarin is prescribed). Surgical treatment in the form of partial muscle resection in the basal part of the interventricular septum (myotomy-myectomy) is recommended for patients with severe clinical manifestations and a gradient in the outflow tract exceeding 50 mm Hg. Art., and also if drug therapy no longer has the desired result.

RESTRICTIVE CARDIOMYOPATHY- a disease characterized by a violation of the diastolic function of the heart as a result of morphological changes in the endocardium, subendocardium and myocardium. In this case, the size of the heart is usually not increased, and sometimes reduced. Endomyocardial fibrosis and Leffler's fibroplastic eosinophilic parietal endocarditis, previously designated as independent nosological forms, are now considered as different stages of the same disease. Sometimes the name "endomyocardial disease" occurs. Primary for this disease is pronounced eosinophilic leukocytosis (sometimes called leukemia), reaching from 36 to 75%. Regardless of the cause of eosinophilia, 3 stages are distinguished in the development of heart disease: necrotic, thrombotic, fibrotic. As a result, a sharp thickening of the endocardium and obliteration of the ventricular cavities with fibrous tissue and thrombotic masses develop. More often, both ventricles are affected (in 50-70% of cases), but there is also an isolated lesion of the right or left ventricle with approximately the same frequency. clinical picture. The main manifestations of the disease are associated with heart failure, arrhythmia and embolism. The first signs of the disease are nonspecific: weakness, shortness of breath, decreased exercise tolerance. Pain in the region of the heart is relatively rare. In the future, clinical manifestations are determined by right or left ventricular failure, but even with a combination of damage to the left and right heart, right ventricular symptoms, as a rule, predominate. Often there is recurrent ascites, liver enlargement (sometimes without peripheral edema), pronounced cyanosis of the face, swelling of the cervical veins. Regardless of the prevalence of insufficiency, a pericardial effusion is detected, often fluid in the pleural cavity. Accordingly, heart sounds are muffled, usually tachycardia, lowering blood pressure. The murmur of mitral insufficiency, gallop rhythm is often auscultated. Thromboembolic pneumonia is a common complication of restrictive cardiomyopathy. Electrocardiography, chest x-ray, echocardiography, angiocardiography, cardiac catheterization and endocardial biopsy, blood tests are methods used to confirm the diagnosis of restrictive cardiomyopathy. According to the ECG, low QRS and T voltages are detected, especially with pericardial effusion, various arrhythmias, blockades of conduction. With right ventricular endomyocardial fibrosis, 75% of patients have pathological Q waves in V1-V2 leads, negative - T and a decrease in the ST segment, sometimes high right atrial P waves. pulmonary circulation,

pericardial effusion, in patients with left ventricular - an increase in the left atrium, stagnation in the pulmonary circulation. In both forms, linear calcification is sometimes detected near the apex and in the region of the outflow tract. Echocardiography reveals endocardial thickening, obliteration of the ventricular cavity, paradoxical septal movement, and pericardial effusion; diastolic opening of the pulmonary valve is often observed. Angiocardiography confirms a change in size, uneven contours of the ventricles, obliteration of the apex of the heart, expansion of the outflow tract, and a decrease in cardiac output. The use of endocardial biopsy is justified in differential diagnosis with sarcoidosis, myocarditis, and amyloidosis of the heart. Anemia, varying degrees of eosinophilia are recorded in blood tests. Differential diagnosis is carried out with constrictive pericarditis, pericarditis of any etiology, atrial myxoma, sarcoidosis, myocarditis, amyloidosis of the heart and other types of idiopathic cardiomyopathy. Unlike hypertrophic and dilated cardiomyopathy, restrictive cardiomyopathy is not characterized by cardiomegaly, the size of the heart is usually small, and obliteration of the ventricular cavities in the apical region is characteristic. Most often it is necessary to differentiate with constrictive pericarditis.

Treatment. There is currently no effective treatment for restrictive cardiomyopathy. Justified cautious use of diuretics in cases of stagnation in the pulmonary and systemic circulation and digoxin in cases of reduced contractility of the left ventricle. Drugs with a positive inotropic effect are ineffective, and vasodilators should be used with great care to avoid deterioration of ventricular filling due to excessive reduction in preload. A high ventricular filling pressure is required to maintain adequate cardiac output in restrictive cardiomyopathy. There is no convincing evidence to support the use of calcium channel blockers in this pathology, although they may be able to increase ventricular compliance in diastole.

ALCOHOLIC CARDIOMYOPATHY develops with many years of regular alcohol abuse. When the so-called "beer" heart occurs, the cobalt contained in beer plays a decisive role in myocardial damage, and the clinical symptoms differ from those in alcoholic heart damage. In chronic alcoholism, heart damage occurs at the subcellular level, intracellular calcium transport is disrupted, which leads to disruption of myocardial relaxation processes. The clinical picture already at an early stage of alcoholic cardiomyopathy is manifested by palpitations, extrasystole, and sometimes atrial fibrillation. A gallop rhythm may be heard. Other signs of chronic alcoholism are also revealed - hand tremor, severe sweating. In the later period, there is an increase in the size of the heart, signs of cardiac decompensation. On the ECG, there is a reduced or negative T wave. Treatment is aimed at the complete exclusion of alcoholic beverages, the intake of B vitamins and other drugs that improve myocardial metabolism. In heart failure, cardiac glycosides, diuretics, antiarrhythmics are used. If there is no severe heart failure, small doses of beta-blockers are prescribed. Other forms of secondary cardiomyopathy in infectious and parasitic diseases, amyloidosis, etc. are discussed in the relevant chapters. If there is no severe heart failure, small doses of beta-blockers are prescribed. Other forms of secondary cardiomyopathy in infectious and parasitic diseases, amyloidosis, etc. are

discussed in the relevant chapters. If there is no severe heart failure, small doses of beta-blockers are prescribed. Other forms of secondary cardiomyopathy in infectious and parasitic diseases, amyloidosis, etc. are discussed in the relevant chapters.

MYOCARDIODYSTROPHY- a group of non-coronary and non-rheumatic diseases of the myocardium, characterized by a violation of metabolic processes in the heart muscle and certain structural changes that occur under the influence of extracardiac causes. Among them, according to V. Kh. Vasilenko (1983) and N. R. Paleev (1991), myocardial dystrophy in anemia, malnutrition and obesity, vitamin deficiency, kidney and liver damage, disorders of certain types of metabolism, diseases of the endocrine system, systemic diseases, intoxications, physical overexertion, infections. The pathophysiological mechanism consists in the development of energy deficiency in conditions of adaptive hyperfunction of the myocardium, aimed at maintaining an adequate level of functioning of the cardiovascular system in a situation of prolonged action of extracardiac causes. As a result, myocardial damage occurs and a picture of myocardial dystrophy develops. Changes that occur in the heart muscle are nonspecific. When the action of the extracardiac factor is eliminated, changes in the heart muscle are reversible. In the development of myocardial dystrophy, 3 stages are distinguished. The first stage is characterized by adaptive hyperfunction of the myocardium, as a rule, with a hyperkinetic variant of blood circulation resulting from dysfunction of regulatory systems - an increase in the influence of the sympathoadrenal and suppression of the influence of the parasympathetic nervous system. Stage II is characterized by the formation of metabolic and structural changes, leading to a violation of the function of the heart. At stage III, severe pathological changes in metabolism, the structure and function of the heart muscle develop, accompanied by circulatory failure and cardiac arrhythmias. The clinical picture, in accordance with the stages of development of myocardial dystrophy, to a certain extent depends on its stage. In the early stages of the formation of myocardial dystrophy, complaints of a cardiac nature may be absent. Rapid fatigue, decreased performance, poor exercise tolerance can be regarded as manifestations of the underlying disease. Most often, patients note cardialgia, localized in the apex of the heart, long-term, not having a clear connection with physical activity at the time of its implementation, not eliminated after taking nitroglycerin. At the same time, both physical and emotional overload often provoke cardialgia in such patients, but more often after a while. Sometimes pain can be unreasonable. In addition, many patients are concerned about the feeling of lack of air, shortness of breath, palpitations. In II-III stages of myocardial dystrophy, edema, shortness of breath at rest, rhythm and conduction disturbances may appear. With an objective examination at the beginning of the development of myocardial dystrophy, a weakening of 1 tone above the apex of the heart, a short systolic murmur, and tachycardia are determined. Subsequently, a gallop rhythm may be formed due to the occurrence of a pathological III tone, rhythm and conduction disturbances (extrasystole, atrial fibrillation, intraventricular and atrioventricular blockade) are often detected, and circulatory failure appears. Certain

clinical manifestations are due to the extracardiac pathology that led to the development of myocardial dystrophy. So, with myxedema, the size of the heart can significantly increase, the movements of its walls are sluggish and slow; with thyrotoxicosis, atrial fibrillation can develop very early; with anemia, symptoms of valvular heart disease can be simulated - a systolic murmur typical of mitral, tricuspid and aortic valve insufficiency can be heard; with disovarian diseases, patients often complain of “hot flashes”, a feeling of heat, sweating, paresthesia in the limbs, etc. The ECG often reveals a decrease and flattening of the T wave, most pronounced in the right chest leads (V, 3), less often in the left. Sometimes the ST segment is displaced, intraventricular conduction is disturbed. If such changes are established on the ECG, pharmacological tests (potassium, obsidian) are performed for the purpose of diagnosis. In the case of such changes with intracellular potassium deficiency, ingestion of 4-6 g of potassium chloride can normalize the ECG. With an excessive effect of catecholamines on the myocardium, normalization of the ECG is possible and within 1-1.5 hours after taking 60-80 mg of obsidan (anaprilin, inderal). However, it should be noted that the data of pharmacological tests should not be absolutized. Only the totality of the results of clinical, instrumental methods and dynamic observation makes it possible to diagnose myocardial dystrophy. Treatment should be aimed at eliminating the underlying process that caused dystrophic changes in the myocardium. In addition, pharmacotherapy is prescribed that affects the metabolic processes in the heart muscle, normalizes the electrolyte balance (potassium, magnesium preparations), improves microcirculation (dipyridamole, teonikol, aspirin), affecting the catecholamine balance (anaprilin, obzidan, inderal). At the same time, antiarrhythmic therapy, treatment of heart failure is carried out.

PERICARDITIS -inflammation of the sheets of the pericardial sac (epicardium and pericardium), which occurs as a complication of various diseases and is very rarely an independent disease. Currently, the main causes of pericarditis are connective tissue diseases, tuberculosis, bacterial and viral infections, postpericardiotomy syndrome associated with cardiac surgery, pericarditis during tumor processes, postinfarction, uremic.At autopsy, pericarditis is detected, according to various authors, in 3-10% of cases. The frequency of pericarditis detected clinically is much lower, since in many patients it is almost asymptomatic. clinical picture. Pericardial disease usually manifests in one of 3 clinical forms: acute dry or effusion, chronic effusion, and constrictive. At the beginning of the inflammatory process, pericarditis, as a rule, is dry due to the deposition of fibrin on the affected epicardium. The most important sign of it is retrosternal pain, usually sharp, cutting, but it can also be dull, pressing. The pain is aggravated by deep breathing, coughing, turning the torso, in the position on the back and left side, relieved by sitting and leaning forward. It is not relieved or stopped by taking nitroglycerin. Pain often radiates to the left supraclavicular region, neck, shoulders. The appearance of pain in most cases is preceded by an increase in body temperature (a characteristic sign for differential diagnosis with myocardial infarction), general weakness, fatigue, myalgia. Pericardial rub is the most important objective symptom

of the disease. Often it is determined only with careful listening, pressing the stethoscope on the chest and in the position of the patient lying on his stomach, if the patient leans on his elbows and knees, in a state of deep inspiration, or if the patient leans forward. Pericardial friction is often transient and may disappear within hours of onset. Sometimes pericarditis is accompanied by extrasystole, atrial fibrillation and other arrhythmias. The appearance of pain in most cases is preceded by an increase in body temperature (a characteristic sign for differential diagnosis with myocardial infarction), general weakness, fatigue, myalgia. Pericardial rub is the most important objective symptom of the disease. Often it is determined only with careful listening, pressing the stethoscope on the chest and in the position of the patient lying on his stomach, if the patient leans on his elbows and knees, in a state of deep inspiration, or if the patient leans forward. Pericardial friction is often transient and may disappear within hours of onset. Sometimes pericarditis is accompanied by extrasystole, atrial fibrillation and other arrhythmias. The appearance of pain in most cases is preceded by an increase in body temperature (a characteristic sign for differential diagnosis with myocardial infarction), general weakness, fatigue, myalgia. Pericardial rub is the most important objective symptom of the disease. Often it is determined only with careful listening, pressing the stethoscope on the chest and in the position of the patient lying on his stomach, if the patient leans on his elbows and knees, in a state of deep inspiration, or if the patient leans forward. Pericardial friction is often transient and may disappear within hours of onset. Sometimes pericarditis is accompanied by extrasystole, atrial fibrillation and other arrhythmias. Often it is determined only with careful listening, pressing the stethoscope on the chest and in the position of the patient lying on his stomach, if the patient leans on his elbows and knees, in a state of deep inspiration, or if the patient leans forward. Pericardial friction is often transient and may disappear within hours of onset. Sometimes pericarditis is accompanied by extrasystole, atrial fibrillation and other arrhythmias. Often it is determined only with careful listening, pressing the stethoscope on the chest and in the position of the patient lying on his stomach, if the patient leans on his elbows and knees, in a state of deep inspiration, or if the patient leans forward. Pericardial friction is often transient and may disappear within hours of onset. Sometimes pericarditis is accompanied by extrasystole, atrial fibrillation and other arrhythmias.

The effusion in the pericardium appears almost simultaneously with the deposition of fibrin, but at first, due to the pronounced absorption capacity of the pericardium, it is insignificant and most often accumulates gradually. Normally, the heart sac contains about 25-35 ml of fluid, the accumulation of effusion helps to reduce pain in the region of the heart and leads to shortness of breath, tachycardia, jugular veins that do not collapse on inspiration, cyanosis, and sometimes temporary disturbances of consciousness. The area of cardiac dullness increases, the apex beat is not detected in most cases, the tones become more deaf, the pericardial friction noise disappears. An increase in the amount of effusion can lead to cardiac tamponade and the appearance of a paradoxical pulse (decrease in the amplitude of the pulse or its complete disappearance on inspiration), best felt on the carotid or femoral artery.

The pallor of the skin, the cyanosis of the lips, nose, and ears increase, and there is swelling of the face and neck (“Stokes' collar”). Sometimes predominant overflow of the veins and swelling of one and 1 hands, more often the left, develops due to compression of the innominate vein by fluid in the upper pericardial sinuses. In the future, the liver increases and becomes bogged down, especially its left lobe. Ascites and edema are formed on the legs and lower back. The hallmark of pericarditis is that there is usually no congestion in the lungs. The final stage of the progression of acute pericarditis may be constrictive pericarditis, but often it develops initially, characterized by a sharp thickening and compaction of the heart shirt. This leads to a decrease in the distensibility of the heart and the filling of its chambers, followed by overflow of peripheral veins with blood. Stagnation in the systemic circulation is the main clinical symptom of constrictive (adhesive) pericarditis. Patients complain of shortness of breath, fatigue, weakness, a sharp expansion of the cervical veins. An enlarged liver with ascites and peripheral edema is revealed. The venous pressure rises sharply (usually more than 250 mm of water. Art.). Heart sounds are muffled, an additional tone is often heard 0.1-0.12 s after the second tone, sometimes a systolic click, splitting of the 2nd tone due to early closure of the aortic valve with a decrease in systolic ejection. As a rule, a paradoxical pulse is determined, tachycardia is characteristic, which increases with the slightest load. Compressive pericarditis is characterized by Beck's triad: high venous pressure, ascites, small quiet heart. Constrictive pericarditis occurs chronically with gradual progression of heart failure. In the development of chronic constrictive pericarditis, 3 stages are distinguished: initial, severe and dystrophic. In the initial stage, there is weakness, shortness of breath when walking, venous pressure rises only after exertion. For the stage of pronounced phenomena, the appearance of ascites is typical. The combination of hypertension syndrome in the system of the superior vena cava and the syndrome of impaired hepatic and portal circulation is also characteristic, the ratio of which, in contrast to cases of pericardial tamponade, does not depend on the position of the patient's body. The dystrophic stage is characterized by the development of hypoproteinemia. At this stage of the process, along with ascites and effusion in the pleural cavities, edema forms on the lower extremities, genitals, body, face, and hands. This is due to hypoproteinemia. An ECG study plays an important role in the diagnosis of pericarditis. On the ECG with dry pericarditis, a concordant position of the ST segment is found in 2 or 3 standard leads, especially in lead II and V2_6, without significant changes in the ORS complex. As acute phenomena subside, the ST segment returns to the isoline with the appearance of a small negative T wave. When an effusion appears, the voltage of the QRS complex decreases. In cases of constrictive pericarditis, it decreases even more, often a deep and wide Q wave is formed. Changes in repolarization are typical, signs of left atrial overload and atrial fibrillation are not uncommon. With the appearance of an effusion, the voltage of the QRS complex decreases. In cases of constrictive pericarditis, it decreases even more, often a deep and wide Q wave is formed. Changes in repolarization are typical, signs of left atrial overload and atrial fibrillation are not uncommon. With the appearance of an effusion, the voltage of the QRS complex

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Echocardiography at the initial stage reveals a thickening of the pericardium or a small amount of fluid in the pericardial cavity. With effusion pericarditis, additional fluid is clearly defined, and its amount can be established. For constrictive pericarditis, it is typical to receive 2 independent echo signals corresponding to the visceral and parietal sheets of the pericardium, limiting the movement of the posterior wall of the left ventricle. Radiologically, an increase in the cardiac shadow, a change in its contours (smoothing of the waist), a weakening of the heart pulsation, and a congestive expansion of the root vessels are established. In cases of development of constrictive pericarditis, the size of the heart is normal or even reduced, only the left atrium is somewhat enlarged. A typical sign is pericardial calcification, a sharp weakening or absence of heart pulsation.

Pericardial puncture allows not only to confirm the presence of effusion in the cavity of the heart shirt, but also to determine its nature, to distinguish pericarditis from hydropericardium (transudate), chilo- and hemopericardium, to conduct a detailed cytological examination of the exudate, to put bacteriological, immunological and biochemical tests. Classification. According to the etiological classification, there are 3 groups of pericarditis: 1. Pericarditis caused by exposure to an infectious agent (bacterial, tuberculous, rheumatic, viral and rickettsial, fungal, with protozoal invasion). 2. Aseptic pericarditis: allergic, with connective tissue diseases (systemic lupus erythematosus, rheumatoid arthritis), traumatic, autoimmune (post-infarction, post-commissurotomy, etc.), with blood diseases, malignant tumors, deep metabolic disorders (uremic, gouty). Differential diagnosis is carried out with acute myocardial infarction, pneumonia, pleurisy, pulmonary embolism, dissecting aortic aneurysm, restrictive cardiomyopathy, cirrhosis of the liver, tricuspid valve stenosis, mitral stenosis, vena cava insufficiency syndrome in mediastinal tumors. Treatment is strictly differentiated depending on the etiology of the disease and its form. With infectious pericarditis, antibiotic therapy is prescribed, taking into account the tolerability of the drug and the sensitivity of the microflora. In the treatment of tuberculous pericarditis, a combination of 3 drugs is usually used: rifampin - 600 mg, isoniazid -300 and ethambutol - 50 mg / kg of body weight daily. In cases of dry or exudative pericarditis with an unexplained etiology and the absence of active inflammatory foci, antibiotic therapy is usually not prescribed. If the pericarditis is purulent or the heart shirt is affected due to sepsis, purulent focus or pneumonia, antibiotics are required. In this case, it is recommended to introduce antibiotics into the cavity of the heart shirt after the maximum possible extraction of effusion through the catheter and washing the cavity. Treatment of allergic, autoimmune and recurrent pericarditis begins with the appointment of non-hormonal anti-inflammatory and antihistamine drugs (Voltaren, diclofenac, metindol, plaquenil, diphenhydramine, suprastin). If there is no effect, steroid hormones are indicated, and in some cases immunosuppressants (azathioprine, colchicine). With pericarditis, associated with rheumatic diseases, systemic lupus erythematosus, the

use of steroids is justified at the earliest stages of development. The same approach is used for postinfarction pericarditis (Dressler's syndrome). NSAIDs are given first, such as aspirin 650 mg orally every 6–8 hours or indomethacin 25–50 mg orally every 4–8 hours. doses. In cases of acute pericarditis in the initial stages of large-focal myocardial infarction, it is recommended to prescribe only aspirin. The use of other non-steroidal or glucocorticoid anti-inflammatory drugs is contraindicated, as they can slow down scar formation and increase the likelihood of myocardial rupture. Anticoagulants for infarcted pericarditis, if possible, should not be prescribed because of the threat of hemorrhagic pericarditis with subsequent cardiac tamponade. If the tumor nature of pericarditis is established and malignant neoplasm cells are found in the effusion, cytostatics are re-introduced into the cavity, preferably thioTEF (50 mg each). With dialysis pericarditis, the number of hemodialysis sessions increases to 6-7 per week. If this fails or there is evidence of cardiac tamponade, pericardectomy or drainage of the pericardial cavity is indicated. In cases of compressive pericarditis, patients should be kept under constant observation with repeated echocardiography to evaluate the effectiveness of anti-inflammatory treatment. If the volume of the pericardial effusion decreases and signs of cardiac tamponade disappear, pericardiocentesis is not required. If such resolution of the disease does not occur, there are indications for the removal of fluid from the pericardial cavity. With constrictive pericarditis, surgical treatment is performed, the volume of which is determined by the prevalence of the compressive capsule, the degree of growth of the connective tissue, and the severity of calcium deposits. Most often, the task of the surgeon is to free the ventricles from the squeezing capsule, starting from the left. When the heart is released from the right ventricle, intraoperative pulmonary edema may occur with a fatal outcome. Expansion of the volume of surgical intervention sharply increases the risk of injury to thin-walled parts of the heart and large veins. As a symptomatic treatment for pericarditis, cardiac glycosides, diuretics, and angiotensin-converting enzyme inhibitors are prescribed. there are indications for the removal of fluid from the pericardial cavity. With constrictive pericarditis, surgical treatment is performed, the volume of which is determined by the prevalence of the compressive capsule, the degree of growth of the connective tissue, and the severity of calcium deposits. Most often, the task of the surgeon is to free the ventricles from the squeezing capsule, starting from the left. When the heart is released from the right ventricle, intraoperative pulmonary edema may occur with a fatal outcome. Expansion of the volume of surgical intervention sharply increases the risk of injury to thin-walled parts of the heart and large veins. As a symptomatic treatment for pericarditis, cardiac glycosides, diuretics, and angiotensin-converting enzyme inhibitors are prescribed. there are indications for the removal of fluid from the pericardial cavity. With constrictive pericarditis, surgical treatment is performed, the volume of which is determined by the prevalence of the compressive capsule, the degree of growth of the connective tissue, and the severity of calcium deposits. Most often, the task of the surgeon is to free the ventricles from the squeezing capsule, starting from the left. When the heart is released from the right ventricle, intraoperative pulmonary edema may occur with

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PLEURITIS -this is an infectious or aseptic inflammatory process of various etiologies in the pleura, accompanied by the formation of fibrin deposits on their surface and (or) the accumulation of liquid (serous, purulent, hemorrhagic,

chylous, etc.) exudate in the pleural cavity. Pleurisy can be primary and secondary. Pleurisy is considered primary in cases where the local inflammatory process and the general reaction of the body associated with it are the main signs of the disease. The vast majority of pleurisy is a secondary process and occurs in the presence of purulent-inflammatory processes in adjacent (pulmonary tuberculosis, pneumonia, mediastinitis, liver abscess, subdiaphragmatic abscess, paranephritis, pancreatitis, etc.) or distant (osteomyelitis, otitis media, sinusitis, etc.) organs and fabrics. Depending on the presence or absence of effusion, pleurisy is divided into dry (fibrinous) and effusion (exudative). Adhesive and cicatricial changes in the pleural cavity are often referred to as adhesive, ossifying pleurisy. However, it is more correct to consider them as the outcome of inflammation. Free effusion in the pleural cavity is observed in the absence of adhesions between the sheets of the visceral and parietal pleura and can be located typically and atypically. Encapsulated pleurisy occurs when there are adhesions between the pleural sheets. Their localization is very diverse. Clinical picture and diagnosis. Clinically pronounced dry pleurisy usually begins with sudden pain in one or the other half of the chest, fever and cough. There are common phenomena - weakness, malaise, poor appetite, night sweats, chills. When examining a patient, attention is drawn to his posture, facial expressions and the nature of breathing. So, with unilateral pleurisy, the patient spares the affected side: restricts respiratory movements, prefers the position of lying on the sore side, not moving; presses the hand on the affected side in a sitting or standing position.

During auscultation over the area of dry pleurisy, one can hear weakened breathing and pleural friction noise, different in timbre and duration, localized or diffuse. The rubbing noise of the pleura, at first very gentle, later becomes more rough, reminiscent of the scratching of new skin or rough scratching. The clinical picture of exudative (effusion) pleurisy usually depends on the diseases of which it was a complication. With clinically pronounced exudative pleurisy, typical complaints of patients are shortness of breath, a feeling of heaviness in the chest and cough. As the effusion accumulates, shortness of breath and a feeling of heaviness in the chest increase, although there is no strict dependence of these complaints on the amount of fluid in the pleural cavity. On examination, one can observe the characteristic forced position of the patient - lying on his sore side. With very large effusions or symptoms of respiratory and heart failure, patients take a semi-sitting position. Respiratory excursions of the diseased side of the chest are limited (the chest on the side of the lesion lags behind when breathing). The increased pressure of the fluid accumulated in the pleural cavity increases the volume of the corresponding half of the chest, and also leads to bulging and expansion of the intercostal spaces. If the amount of pleural fluid exceeds 300-500 ml, then dullness of percussion sound (with massive effusions), weakening or disappearance of voice trembling and respiratory noises are noted on the affected side. The blunting zone has a curved upper border (Damuazo line). With left-sided pleurisy, Traube's semilunar space disappears. Often the pleural puncture becomes the decisive method of diagnosis. The appearance of the pleural fluid, its cellular composition are evaluated, a biochemical

and bacteriological study is performed. X-ray examination with dry pleurisy reveals only limited mobility of the diaphragm on the affected side; later, there is a slight diffuse darkening of the pulmonary field, due to pleural adhesions, moorings. X-ray display of pleural effusion depends on its amount, the state of the pleural cavity (for example, adhesions, moorings, etc.) and the position of the patient's torso (vertical or horizontal). On radiographs of the lungs, darkening is determined from slight to total, depending on the size of the effusion. Very great diagnostic value is thoracoscopy with biopsy of the pleura, which allows you to identify the tuberculous or tumor origin of pleurisy. Treatment. Treatment of pleurisy should be comprehensive and aimed primarily at eliminating the underlying process that led to its development. Symptomatic treatment aims to relieve pain, accelerate the resorption of fibrin, prevent the formation of extensive moorings and adhesions in the pleural cavity. The danger of converting serous exudate into purulent or encysted exudate necessitates regular pleural punctures (after 1-2 yes) with its maximum evacuation. This tactic gives the best anatomical and functional outcomes, since the lung fully expands and obliteration does not develop. With purulent pleurisy, the exudate is evacuated, the cavity is drained and washed with antiseptic solutions, antibiotics are administered intrapleurally. With intoxication, severe shortness of breath, In violation of the activity of the heart, plasma-substituting solutions, oxygen inhalations, and cardiac glycosides are used intravenously. To reduce pain (especially in patients with dry pleurisy), you can use distraction therapy with tight bandaging of the lower chest, lubricating the affected side with iodine tincture in the form of a grid. As the exudate resolves, after the disappearance of pain, normalization of body temperature and ESR, patients are recommended to exercise exercise therapy with the inclusion of breathing exercises to prevent pleural adhesions. In the absence of contraindications, physiotherapy (sollux, inductothermy, electrophoresis) and sanatorium-resort (local sanatoriums, the southern coast of Crimea, the Black Sea coast of the Caucasus, etc.) treatment is carried out. To reduce pain (especially in patients with dry pleurisy), you can use distraction therapy with tight bandaging of the lower chest, lubricating the affected side with iodine tincture in the form of a grid. As the exudate resolves, after the disappearance of pain, normalization of body temperature and ESR, patients are recommended to exercise exercise therapy with the inclusion of breathing exercises to prevent pleural adhesions. In the absence of contraindications, physiotherapy (sollux, inductothermy, electrophoresis) and sanatorium-resort (local sanatoriums, the southern coast of Crimea, the Black Sea coast of the Caucasus, etc.) treatment is carried out. To reduce pain (especially in patients with dry pleurisy), you can use distraction therapy with tight bandaging of the lower chest, lubricating the affected side with iodine tincture in the form of a grid. As the exudate resolves, after the disappearance of pain, normalization of body temperature and ESR, patients are recommended to exercise exercise therapy with the inclusion of breathing exercises to prevent pleural adhesions. In the absence of contraindications, physiotherapy (sollux, inductothermy, electrophoresis) and sanatorium-resort (local sanatoriums, the southern coast of Crimea, the Black Sea coast of the Caucasus, etc.) treatment is

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Diseases of the esophagus - achalasia (non-opening) of the cardia-AK (cardiospasm, chiatospasm, functional obstruction of the cardia)is a violation of the motor function of the smooth muscle of the esophagus, in which its lower sphincter becomes hypertensive, does not relax when swallowing, and the peristalsis of the esophagus is replaced by its abnormal contractions. AK is a relatively rare disease, with an incidence of 7 cases per 100,000 population. The etiology has not been established. It is believed that the cause of the disease is psycho-emotional disorders and stressful situations, prolonged use of cold (ice) drinks and ice cream.

Pathogenesis. At the heart of AK is a violation of the innervation of the smooth muscles of the esophageal wall and the lower esophageal sphincter, which leads to the shutdown of the physiological opening of the cardia during swallowing, an increase in the tone and motility of the esophagus and food retention in it. clinical picture. The main signs of AK are dysphagia, regurgitation, and chest pain when swallowing. Dysphagia is episodic at first, in severe cases it is observed at every meal, especially dry or poorly chewed, aggravated by excitement. To facilitate the passage of food, patients drink water or milk, take deep breaths, arch the body back, which in some cases helps. Regurgitation is manifested by regurgitation of saliva, mucus and food debris accumulated in the esophagus, which occurs when the body is tilted, with a full esophagus or at night, during sleep. Nocturnal regurgitation of food into the oral cavity with its leakage into the respiratory tract is accompanied by symptoms of “night cough”, “wet pillow”, “night vomiting”, etc. Aspiration pneumonia and bronchitis often develop. Pain behind the sternum occurs when swallowing or outside the meal. They disappear after regurgitation or passage of food into the stomach. The full picture of AK is chronic progressive dysphagia and weight loss over several months or years. Diagnosis. An X-ray examination does not detect air in the stomach. The levels of air and fluid in the standing position of the patient indicate food retention in the esophagus. Swallowing barium shows the expansion of the esophagus, and in severe cases, the esophagus looks like a sigmoid colon. Peristalsis in the lower 2/3 of the esophagus is abnormal, its terminal part is persistently coracoidally narrowed and is represented by a non-relaxing lower esophageal sphincter. Reception 1-2 tab. Nitroglycerin relaxes the sphincter and the contents of the esophagus pass into the stomach. With the help of manometry, normal or increased pressure in the lower esophageal sphincter is determined, and

when swallowing, it does not relax. Endoscopy of the esophagus reveals an enlarged cavity and signs of chronic esophagitis (hyperemia, edema, erosion, increased bleeding). This method allows you to exclude cancer of the esophagus. Complications: repeated aspiration pneumonia and chronic bronchitis. With the help of manometry, normal or increased pressure in the lower esophageal sphincter is determined, and when swallowing, it does not relax. Endoscopy of the esophagus reveals an enlarged cavity and signs of chronic esophagitis (hyperemia, edema, erosion, increased bleeding). This method allows you to exclude cancer of the esophagus. Complications: repeated aspiration pneumonia and chronic bronchitis. With the help of manometry, normal or increased pressure in the lower esophageal sphincter is determined, and when swallowing, it does not relax. Endoscopy of the esophagus reveals an enlarged cavity and signs of chronic esophagitis (hyperemia, edema, erosion, increased bleeding). This method allows you to exclude cancer of the esophagus. Complications: repeated aspiration pneumonia and chronic bronchitis.

Treatment. Drug treatment consists in taking sedatives, nitrates (nitroglycerin, sustak, nitrong, nitro-mac, nitrosorbide, isoket, erinite, etc.), anticholinergic drugs (gastrocepin, metacin, platifillin, etc.) and calcium ion antagonists (nifedipine - according to 10-20 mg sublingually 20 minutes before meals). Buscopan is also used (1 tablet 3 times a day), non-absorbable antacids (phosphalgagel, maalox, almagel, etc.). With the ineffectiveness of conservative treatment resort to cardiodilatation. When carried out by an experienced specialist, this method is effective in about 85% of cases. Possible complications of cardiodilatation include esophageal perforation and bleeding.

hiatal hernia- displacement through the esophageal opening of the diaphragm into the posterior mediastinum of some abdominal organs (most often the cardia of the stomach). It ranks third in frequency after peptic ulcer and cholecystitis. It is found slightly more often in women than in men.

Etiology and pathogenesis. In the formation of hernias, a sharp increase in intra-abdominal pressure, congenital underdevelopment of connective tissue structures that strengthen the esophagus, or their dystrophic changes in the elderly, shortening of the esophagus, a decrease in muscle tone, the disappearance of adipose tissue under the diaphragm, kyphosis of the thoracic spine, etc. are important.axial (sliding) hernias, in which the abdominal segment of the esophagus and the cardia of the stomach enter the chest cavity, and paraesophageal (more rare), when the abdominal segment of the esophagus and the cardia remain under the diaphragm, and part of the stomach or other abdominal organs enter the chest. The clinical picture is determined by the symptoms of insufficiency of the cardia and reflux esophagitis: belching, regurgitation of gastric contents, heartburn (especially when bending over or lying down, weakening in an upright position) are disturbing; dysphagia, aerophagia, rapid satiety during meals, persistent hiccups, vomiting with blood, iron deficiency anemia are not uncommon. Shortness of breath, palpitations, reflex angina may be noted. Paraesophageal hernias are more often asymptomatic or may be accompanied by pain in the epigastric region and infringement.

Complications: bleeding of varying intensity, causing anemia; erosion and ulcers, cicatricial changes in the esophagus, infringement (especially with paraesophageal hernia), aspiration pulmonary complications, arrhythmia, etc. Diagnosis. In most cases, a hernia can be detected by X-ray examination in the horizontal position of the patient, with straining. Esophagoscopy reveals an axial hernia and associated esophagitis. The decrease in pressure in the region of the lower esophageal sphincter, revealed during esophagotonokymography, is taken into account. Treatment is mostly conservative. It consists in prescribing a sparing diet within the extended table No. 1, astringents (bismuth nitrate 0.25-0.5 g 2-3 times a day; silver nitrate (0.12 g - 200 ml) 1 tablespoon 3 times a day 15 minutes before meals) and antacids (Almagel 1-2 tsp. spoons 3-4 times a day 30 minutes before meals; vikalin 1-2 tablets. 3 times a day, 1 hour after meals, crushed in 1/2 cup of warm water; magnesium oxide 0.5-1 g 3-4 times a day 1 hour after meals, etc.). If necessary, antispasmodics are used (no-shpa 1-2 tablets 2-3 times a day, papaverine 0.04 g 2-3 times a day) and anticholinergics (atropine sulfate 1 table 2-3 times a day before meals, platifillin 1 table 2-3 times a day before meals, metacin 0.002-0.005 g 2-3 times a day). When serious complications are attached, surgical treatment is performed, which consists in lowering the hernial sac into the abdominal cavity, fixing the cardia and fundoplication. It is not recommended to work associated with the inclination of the body and the tension of the abdominal press. Sleep should be with a raised headboard (on 2-3 pillows), mainly on the right side.

Esophageal cancer (EC)ranks 5th among all cancerous tumors in adult men. This tumor has an uneven frequency in different countries and regions. The zone of its especially high frequency covers Central Asia, Kazakhstan, Yakutia. In European countries, RP is relatively rare: men - 6, women - 2 cases per 100 thousand population.

Etiology. The use of strong alcoholic beverages, too hot or spicy food, smoking, malnutrition, a small amount of fresh vegetables and fruits in the diet are among the predisposing factors for this type of cancer. Chronic esophagitis, polyps of the esophagus, cicatricial strictures, achalasia of the cardia, Plummsr Vinson's syndrome (iron deficiency in the body) matter.

Pathogenesis. Strong alcoholic drinks, hot food and drink injure the mucous membrane of the esophagus, facilitate the contact of carcinogens with it. Chronic esophagitis creates conditions for the implementation of the carcinogenic effect of agents contained in tobacco smoke and supplied with food and water. An esophageal polyp poses a real danger of turning into cancer. Cicatricial changes in the esophagus are alarming in terms of their malignant degeneration. Pathomorphology. RP is almost always primary and is located in places of physiological narrowing. In 90% of cases, it is represented by squamous cell carcinoma and in 10% by adenocarcinoma. Metastasis is observed in 50% of cases and occurs along the lymph flow.

clinical picture. The patient progresses dysphagia and weight loss. Dysphagia begins when taking dense, then semi-liquid and liquid food. When the tumor spreads to the periesophageal tissues, chest pains appear. Bleeding from the

tumor is usually small, but can sometimes be significant. Due to discomfort when swallowing, patients limit themselves to food and lose weight. If the tumor grows into the surrounding tissues, the pain syndrome becomes excruciating and especially worries the patient at night. When the recurrent nerves are involved in the pathological process, hoarseness of the voice occurs, and when the trachea and bronchi are affected, a painful cough, shortness of breath, aspiration pneumonia and lung abscess occur. When the tumor spreads to the supraclavicular region, enlarged lymph nodes can be determined in it, and with liver damage, its increase. Diagnosis. Patients with persistent dysphagia and (or) with a decrease in body weight for a short time should be carefully examined. Esophagography is most important for diagnosis: dilatation of the esophageal lumen can occur early in the disease. Tumor ulcerations should be distinguished from peptic ulcers in the section of the esophagus lined with columnar epithelium. Any ulcers located outside this zone are suspicious for RP. In all cases, if RP is suspected, an endoscopic examination is performed. Multiple biopsy and cytology help in the diagnosis. Computed tomography is necessary to detect the spread of the tumor to the mediastinum and peri-aortic intra-abdominal lymph nodes. Blood changes are characterized by signs of anemia, thrombocytosis, increased ESR. There is blood in the stool. Treatment is carried out by oncologists. Surgical, radiation, medicinal and combined methods are used. The prognosis is unfavorable in most cases. Prevention consists in stopping smoking, alcohol and hot tea abuse, rational nutrition, timely detection and treatment of precancerous diseases.

esophagitis- inflammation of the mucous membrane of the esophagus of various etiologies. There are acute and chronic esophagitis (reflux esophagitis). Acute esophagitis. Etiology. Acute esophagitis may be the result of various damaging factors (thermal, chemical, mechanical), acute inflammatory diseases of the oral cavity, gastrointestinal tract, infectious diseases (typhoid, influenza, diphtheria), and allergic reactions on the esophageal mucosa. Pathomorphology. Depending on the intensity of the lesion, they differcatarrhal, edematous, erosive, pseudomembranous, hemorrhagic, exfoliative, necrotic and phlegmonous forms. The lesion may be focal or diffuse. clinical picture. Mild forms are asymptomatic. In more severe forms, there is a burning sensation behind the sternum during eating, pain along the esophagus, aggravated by swallowing, salivation, belching, regurgitation. With erosive and hemorrhagic forms, hematemesis, melena are possible, with exfoliative - fever. Phlegmonous and necrotic esophagitis is characterized by intense pain, vomiting, and a severe general condition. Complications: possible bleeding, perforation, mediastinitis, strictures.

Diagnosis. Anamnestic data are taken into account, the results of esophagoscopy - hyperemia, erosion, ulcers, necrosis. The prognosis is determined by the nature and prevalence of the lesion, concomitant complications. With phlegmonous and necrotic forms - unfavorable.

Treatment. Apply cold drink, 0.25% solution of novocaine inside, vegetable oil (200 ml) with anesthesin (2 g) 1 tbsp. spoon every hour. Pain syndrome is removed by subcutaneous injection of 1-2 ml of 1% morphine solution. To prevent possible spasm of the esophagus, 1 ml of a 0.1% solution of atropine and 2 ml of a

2% solution of papaverine are injected subcutaneously. Prophylactic administration of antibiotics in massive doses begins. A starvation diet is recommended. Subsequently, a mechanically and chemically sparing diet is prescribed, in severe cases - parenteral nutrition. In case of exposure to strong alkalis or acids, the stomach is washed with a weak solution of soda or a solution of acetic or citric acids. In all cases, the underlying disease is treated and, if possible, the pathogenetic factors of esophagitis are eliminated.

Osteoporosis of the thoracic spine. For him, the most significant are: - childless and small women, - women of fragile physique, premature menopause, long-term use of corticosteroids, other diseases (thyrotoxicosis, Itsenko-Cushing's disease or syndrome, type I diabetes, pathology of the liver and kidneys); low body mass index <19 kg/m2. Awkwardness and discomfort in the spine. Weak and moderate pain in the chest and spine. Pain worsens with prolonged sitting, standing, walking

Spondyloarthropathies. Characteristic combination of pain in the chest with uveitis and arthralgia (including history).

Age of onset before 40 years, slow, gradual increase in pain, duration of pain over 3 months, morning stiffness, decrease in pain after exercise and movement

Pain in the chest also occurs when the bone-costal structures of the chest are affected: with Tietze's syndrome, costo-sternal syndrome (costochondritis, costosternal chondrodynia), anterior costal syndrome, xifoidalgia

Pain in the chest with tumors of the spinal cord. Characteristic symptoms: sudden pain or gradually increasing, bilateral or unilateral pain, pain decreases with movement, often appears at rest at night, sensory disturbances in the zone of radicular innervation.

Herpes zoster. Shingles (herpes zoster) is a sporadic disease resulting from the activation of a latent varicella-zoster virus.

It is characterized by inflammation of the posterior roots of the spinal cord and intervertebral ganglia.In most cases, the onset is acute. Body temperature can rise to 38-39 ° C; its rise is accompanied by general toxic reactions (headache, malaise, chilling).

In the zone of innervation of one or more spinal ganglia, skin rashes appear with pain and other subjective sensations characteristic of them.

Burning, paroxysmal unilateral chest pains are characteristic, which intensify at night and are often accompanied by pronounced emotional reactions.

The appearance of an erythematous papule and vesicle surrounded by a halo of hyperemia.

Psychogenic chest pain.

It is a common variant of pain, which consists in the fact that the phenomenon of pain itself, being the leading one in the clinical picture at some stagediseases, is simultaneously in the structure of various affective and vegetative disorders.

Psychogenic painin the chest is most often localized in the zone of the apex of the heart, the precordial region and the region of the left nipple.

Most often, psychogenic chest pains are observed in: anxiety-hypochondriacal disorders, phobic disorders, anxiety-panic disorders, depression.

The main criteria for psychogenic pain: The predominance of multiple and prolonged pain.Regardless of the absence or presence of an organic cause of pain, the patient's complaints far exceed those that are possible for this
organic find. The existence of a temporary connection between a psychogenic problem and the development or increase of pain syndrome. There may be aching, stabbing, pressing, squeezing, burning or throbbing pain, prolonged, coinciding with periods of fatigue and great emotional stress. There may also be short-term pain, not associated with physical activity.

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LECTURE TOPIC: "SYNDROME OF ARTERIAL HYPERTENSION AND HEADACHE. DIFFERENTIAL DIAGNOSIS OF ARTERIAL HYPERTENSION."

TRAINING TECHNOLOGY

Number of students-	Time - 2 hours
Form of the lesson	Lecture - visualization
Lecture plan	Definition of terms arterial hypertension and essential arterial hypertension 1. Causes and risk factors leading to high blood pressure Various pathogenetic mechanisms of blood pressure rise 2. Classification of hypertension (BP norms, stages, degrees, risk stratification)

	3. General differential diagnostic signs of symptomatic hypertension and hypertension 4. Clinical manifestations and course of diseases occurring arterial hypertension 5. Diagnosis of diseases accompanied by arterial hypertension 6. Principles of treatment, prevention and medical examination of patients with arterial hypertension
Purpose of the lesson: to familiarize students with the etiology, pathogenesis of diseases accompanied by arterial hypertension, to acquaint students with the modern classification of hypertension, to teach the principles of diagnosis, treatment, prevention and clinical examination of diseases accompanied by hypertension	
Pedagogical tasks: 1. Strengthen and deepen students' knowledge of diseases accompanied by an increase in blood pressure 2. To teach students to correctly establish a diagnosis in accordance with the modern classification 3. To teach students the ability to differentiate diseases accompanied by hypertension 4. Familiarize students with the stages of prevention of patients with hypertension	**The results of the educational process:** **OP needs to know**: 1. Risk factors leading to increased blood pressure. 2. Diseases associated with arterial hypertension. 3. Principles of differential diagnosis of diseases accompanied by arterial hypertension. 4. Principles of treatment. 5. Principles of prevention and medical examination of patients with hypertension
Teaching methodology	Lecture text, videos, questionnaires, questions, “yes-no” technique
Form of study	Laser projector, visual materials, special technical equipment, presentation of thematic patients
Means of education	Team

Conditions for the educational process	Audience

TECHNOLOGICAL CARD LECTURES

Stages, time	**Activity**	
	Teacher	**students**
Stage 1 Introduction (5 minutes)	1. Tells about the topic of the lecture, its purpose and plan	1. Listen
Stage 2 Actualization (increasing the importance) of knowledge (20 minutes)	2.1. In order to increase the actualization (increase in importance) of students' knowledge, asks questions: 1. Define the terms symptomatic arterial hypertension and essential hypertension? What is the difference? 2. List the diseases accompanied by arterial hypertension? 3. List the risk factors leading to an increase in blood pressure? 4. List the groups of drugs used to treat hypertension? Conducts a survey 2.2. Showing on the screen, offers students to get acquainted with the goals and objectives of the lecture. Slide #1	2.1. Answer questions asked 2.2. Study slide number 1 2.3. Study slide number 2
Stage 3	3.1. Introduces students to the lecture material, the importance of	3.1. Together they analyze the listened

Main part (informational) (55 min)	the topic and the principles of the formation of an intelligent cultural personality, in particular, a GP teacher. In order to increase the actualization of knowledge, he conducts a quick survey of students: 1. According to 1 point of the lecture plan: Define the terms arterial hypertension and essential arterial hypertension? 2. According to paragraph 2 of the lecture plan: List the causes and risk factors leading to an increase in blood pressure 3. According to the 3rd point of the lecture plan: List the various pathogenetic mechanisms of the rise in blood pressure? 4. According to the 4th point of the lecture plan: Tell the main points of the classification of hypertension (BP norms, stages, degrees, risk stratification) 5. According to the 5th point of the lecture plan: List the general differential diagnostic signs of symptomatic hypertension and hypertension 6. According to paragraph 6 of the lecture plan: List the main clinical manifestations and course of diseases that occur with arterial hypertension 7. According to paragraph 7 of the lecture plan: What are the main methods for diagnosing diseases accompanied by arterial hypertension 8. According to paragraph 8 of the lecture plan: List the basic	lecture material, ask questions Key points are written in a notebook.

	principles of treatment, prevention and medical examination of patients with arterial hypertension Stopping at the important points of the lecture, he suggests writing down the main points in a notebook	
Stage 4 final (10 min)	4.1. Asking questions: 1. List the most common diseases accompanied by an increase in blood pressure 2. Tell the modern classification of hypertension 3. Name the basic principles of treatment, prevention of patients with arterial hypertension 4.2. Gives a task for independent work of students: Symptomatic arterial hypertension associated with kidney pathology	4.1. Answer questions 4.2. Listen, write

Arterial hypertension (AH) is one of the main risk factors for stroke, myocardial infarction, and heart and kidney failure. The prevalence of hypertension is 20–40% in the adult population of many economically developed countries of the world, and among the elderly, its frequency exceeds 50%. Currently, there is no doubt the need for long-term, essentially lifelong drug therapy for hypertension. With a decrease in blood pressure (BP) by only 13/6 mm Hg. Art. the risk of developing a cerebral stroke is reduced by an average of 40%, and myocardial infarction - by 16%.

Hypertension (AH) or essential arterial hypertension is a disease in which there is an increase in blood pressure not associated with a primary organic lesion of an organ and systems. A very common disease, most often occurring in old age.
Distinguish: optimal blood pressure < 120/80 mm Hg. Art., normal blood pressure < 120-129 / 80-84 mm Hg. Art., increased, normal blood pressure 130-139 / 85-89 mm Hg. Art. The diagnosis of arterial hypertension is made when blood pressure is detected above 140/90 mm Hg, at least twice during repeated visits to the doctor

Classification of hypertension by stages (WHO, 1996)

1 stage: At this stage, there are no objective signs of damage to target organs. The only manifestation of the disease is the syndrome of arterial hypertension.
2 stage: At least one sign of target organ damage:

- LV hypertrophy
- proteinuria 30-300 mg/day. creatininemia 115-133 µmol/l (for men and 107-124 µmol/l for women)
- generalized or focal narrowing of the retinal arteries
- atherosclerotic vascular disease

3 stage: At this stage, in addition to the syndrome of arterial hypertension and signs of target organ damage, associated clinical conditions are detected: angina pectoris, MI, HF, TNMK, stroke, encephalopathy, dementia, CHF, changes in the fundus (hemorrhage, swelling of the papilla of the ON disc, exudation, atrophy)
To date, there are three degrees of AH:
1 degree AH:140-159/90-99 mmHg Art.
2 degree arterial hypertension:160-179/100-109 mmHg
3 degree arterial hypertension:more than 180/110 mm Hg. Art.
Isolated SAG:> 140 < 90 mmHg
In the presence of hypertension in a patient, in addition to its degree of increase, the degree of risk is also assessed.
When determining the degree of risk, the following are taken into account: gender, age, cholesterol numbers in the blood, obesity, the presence of diseases with hypertension in relatives, smoking, a sedentary lifestyle, target organ damage.

Risk stratification of hypertension

FR, POM	AG 1 degree	AG 2 degrees	AG 3 degrees
No	low risk	moderate risk	high risk
1-2 FR	moderate risk	moderate risk	Very high risk
More than 3 FR or POM	high risk	high risk	Very high risk

AKS, SD	Very high risk	Very high risk	Very high risk

Etiology: not clear. But there are factors that contribute to the development of HHD.

1. Nervous-psychic traumatization. emotional stress.
2. Hereditary - constitutional features.
3. Occupational hazards, constant strain of vision, attention.
4. Features of nutrition (abuse of table salt)
5. Age-related restructuring of the hypothalamic - pituitary system.
6. Skull trauma. 7. Intoxication (smoking, alcohol).

Pathogenesis:

1. Increased activity of the sympathetic-adrenal system.
2. Activation of the renin-angiotensin mechanism
3. Increased production of prostaglandin F2 and cyclic nucleotides.
4. Increased production of antidiuretic hormone.

Clinical picture: Complaints of patients depend on the stage and form of HHD. At the beginning of the disease, subjective sensations of the patient may appear in the form of occasional headaches, dizziness, general weakness, fatigue, irritability, and insomnia. Sometimes patients do not make any complaints, and only a random measurement of blood pressure shows its increase. In the future, when blood pressure becomes stable and higher, complaints become more prominent, shortness of breath appears when walking, pain in the heart, palpitations, headaches occur more often.

With sharp rises in pressure that occur after excitement, overload at work, patients may develop the phenomena of a cerebral crisis, manifested in the form of a feeling of heaviness and pressure in the head or severe headaches, accompanied by dizziness, nausea, vomiting, and sometimes visual impairment. The cause of such a crisis is a sharp spasm of cerebral vessels.

Pain in the region of the heart can occur in the form of angina attacks and outside the cerebral crisis. This happens in cases where atherosclerosis of the coronary vessels of the heart develops with HHD.

In the later stages, when the phenomena of heart failure occur, patients complain of pronounced shortness of breath, which manifests itself in the form of typical attacks of cardiac asthma. Their occurrence is associated with insufficiency of the left ventricle. In severe forms of HHD, patients often develop visual impairment due to vascular changes in the retina of the fundus. The appearance of the patient is varied. In some cases, the skin and mucous membranes are normal, in others, the patient's face becomes red (red hypertension), and more often pale due to a sharp spasm of arterioles (pale hypertension). On palpation of the heart, an increased apex beat is determined, indicating hypertrophy of the left ventricle. In the early periods of the

disease, only LV hypertrophy is noted for a long time without a significant expansion of the boundaries of the heart. In the future, when LV dilatation joins hypertrophy, the increased apical impulse shifts outward from the mid-clavicular line, sometimes reaching the anterior axillary line. Percussion and X-ray in these cases, an increase in the boundaries of the heart to the left is revealed. During auscultation of the heart in the early stages of the disease, usually only the accent of the second tone on the aorta, later, with the development of sclerotic processes in the aorta, a small systolic murmur is heard. In the later stages, when LV expansion occurs, a systolic murmur is also heard at the apex, which occurs due to functional insufficiency of the mitral valve. Both systolic and diastolic pressures usually rise but especially great importance is attached to the figures of diastolic pressure, a significant increase in which indicates the severity of the course of HHD. The ECG reveals signs characteristic of LV hypertrophy, often combined with symptoms of coronary insufficiency. From the side of the fundus, there is a narrowing of the arteries and dilation of the veins (Salus symptom). In severe forms of HHD, hemorrhages and degenerative changes in the area of the corpus luteum and optic nerve often appear, which can lead to severe visual impairment, up to blindness. From the side of the fundus, there is a narrowing of the arteries and dilation of the veins (Salus symptom). In severe forms of HHD, hemorrhages and degenerative changes in the area of the corpus luteum and optic nerve often appear, which can lead to severe visual impairment, up to blindness. From the side of the fundus, there is a narrowing of the arteries and dilation of the veins (Salus symptom). In severe forms of HHD, hemorrhages and degenerative changes in the area of the corpus luteum and optic nerve often appear, which can lead to severe visual impairment, up to blindness.

Complications:

1. <u>Cardiac:</u>development of chronic coronary artery disease. Acute heart failure. development of an aortic aneurysm.
2. <u>Cerebral:</u>Reduced vision, development of atherosclerosis of cerebral vessels, dynamic and organic disorders of cerebral circulation.
3. <u>Renal:</u>hypertensive nephrosclerosis. Chronic renal failure.

Differential diagnosis.

Symptomatic arterial hypertension accounts for 6-9% of all cases of increased blood pressure. Differential diagnosis of symptomatic hypertension is important, since the nature of therapeutic measures and the determination of the prognosis of the disease depend on the correct diagnosis. Syndrome of malignant arterial hypertension: pallor of the skin; BP 240/130- 300/170 mmHg and higher; ineffectiveness of antihypertensive drugs; III-IV degree of retinopathy (edema of the optic nerve and hemorrhages); the rapid deterioration of the general condition of the patient, the rapid development of organic changes in the brain, heart and renal arteries; neutrophilic leukocytosis in the blood, increased ESR, thrombocytopenia, anemia of moderate severity; rapidly progressive renal failure; proteinuria, hematuria, cylindruria, isostenuria in urinalysis,

Signs that suggest the presence of symptomatic hypertension:age < 40 years; inefficiency of antihypertensive therapy; malignant or progressive nature of

hypertension; good tolerability of hypertension, few complaints; stability of blood pressure, predominant increase in diastolic blood pressure, alarming history: - nephropathy, cystitis, edema, renal colic, - "out of program" disorders: muscle weakness, vascular murmurs, paroxysms of increased blood pressure; paradoxical reaction to some drugs.

Atherosclerotic lesions of the renal arteries are the most common cause of renovascular arterial hypertension (about 70%). It usually develops in men over 50 years of age.

The diagnosis is established on the basis of the detection of prolonged systolic or systolic-diastolic murmur over the projection of the renal arteries (in the epigastrium 2-3 cm above the navel, and also at this level to the right and left of the midline of the abdomen). Noise is detected in approximately 50-60% of patients. The diagnosis is verified by objective research methods: isotope renography, excretory urography, computed tomography, abdominal aortography, renal vein catheterization - an increase in the content of renin in the venous blood of the affected kidney. Aortography data are decisive not only in the final diagnosis, but also in the choice of treatment method (balloon angioplasty, surgical correction of stenosis).

It is believed that angioplasty or surgical correction is indicated for: 1) poorly treatable hypertension; 2) deterioration of kidney function against the background of drug treatment; 3) drug intolerance; 4) the young age of the patient.

In cases of non-functioning kidney - nephrectomy. Therapeutic tactics do not differ from those for HHD.

Fibromuscular dysplasia of the renal arteries occurs in 10-20% of patients with renovascular hypertension. This disease occurs in women 4-5 times more often than in men, and hypertension with it usually develops at a young age (before 40 years). More often this pathology is congenital.

Diagnosis is carried out by the same methods and on the basis of the same signs as in atherosclerotic hypertension. Only in cases of angiography, stenoses in the renal arteries are detected in the form of a string of beads or pearls. Treatment. Balloon angioplasty is the method of choice (high success rate and low risk of re-stenosis).

Nonspecific aortoarteritis (panarteritis, pulseless disease, Takayasu's syndrome, etc.) leads to stenosis of the aorta and main arteries and to ischemia of the affected organ.

The syndrome of vasorenal hypertension is observed in half of the patients and is due to the involvement of the mouths of the renal arteries in the process. This increases mainly diastolic pressure to 100-160 mm Hg. Art. and systolic - up to 180-250 mm Hg. Art.

Blood pressure is measured in both the upper and lower extremities. Asymmetry of blood pressure, as well as the presence of systolic-diastolic murmur over the projection of the renal arteries, are typical clinical signs of this pathology. The final diagnosis of the syndrome of renovascular hypertension is established by aortography.

Treatment of hypertension is the same as for hypertension. With the development of stenosis and occlusion of the main arteries, which sharply disrupt the hemodynamics of an organ or region, reconstructive vascular operations are indicated.

Chronic glomerulonephritis. The hypertensive form of chronic glomerulonephritis is one of the most common causes of symptomatic renal hypertension (about 30-40%). The pathogenetic mechanisms of hypertension in this disease are based on the activation of the renin-angiotensin system, a decrease in the ability of the kidney to produce vasodilator and natriuretic substances, which leads to an increase in the reabsorption of sodium and water. As nephrosclerosis progresses, renoprival mechanisms of the pathogenesis of hypertension join.

In chronic glomerulonephritis, much more often than in patients with HHD, there is a stabilization of blood pressure at high levels, and in the absence of adequate therapy, an outcome in malignant hypertension.

The diagnosis of chronic glomerulonephritis is established on the basis of anamnestic indications of previously transferred acute glomerulonephritis or nephropathy of pregnancy, repeated tonsillitis and other diseases caused by streptococcus, pain in the lumbar region. In the process of examining such patients, a pale edematous face (“braitika”) is noted. Repeated urine tests are most informative, and changes in the urine are detected before an increase in blood pressure or with very moderate hypertension. Most often they are manifested by slight proteinuria (in 98% of cases), less often - erythrocyturia (in 60% of cases) and cylindruria (in 40-50% of cases). Additional information in the diagnosis can be obtained by ultrasound of the kidneys - narrowing of the cortical layer with an unchanged pelvicalyceal system. The diagnosis is verified by puncture biopsy of the kidneys.

Treatment. Along with the therapy of the underlying disease, medications are prescribed: loop diuretics, ACE inhibitors, calcium antagonists. With resistance, alpha-blockers and / or labetalol. In cases where more intensive treatment is needed, minoxidil can be added. Hemodialysis or kidney transplantation may be required to lower blood pressure in end-stage renal disease.

Chronic pyelonephritis is the most common cause of SAH. In the process of autopsy, chronic pyelonephritis is detected in 6% if there are indications of hypertension during life.The pathogenesis of hypertension in pyelonephritis does not differ significantly from that in glomerulonephritis. The predominant localization of morphological changes in the medulla of the kidneys suggests a greater involvement in the occurrence of AH of a decrease in the depressor function of the kidneys. AH in chronic pyelonephritis is relatively benign. When making a diagnosis, attention should be paid to the identification of risk factors for chronic pyelonephritis, a history of dysuric disorders, including in childhood and adolescence, pain and lumbar region of a dull or aching nature, unmotivated fever. Patients with chronic pyelonephritis attract attention with pallor of the skin, paraorbital edema and “bluish” circles under the eyes. Quite often at such patients the nocturia is observed. In laboratory studies of urine, hypoisosnuria, moderate proteinuria (in 75% of cases), pyuria (in 50% of cases), less often hematuria (in 30% of cases) are most often

detected. However, in many patients with acute exacerbations, there are no changes in the urine. With urine pospas, growth of more than 100,000 colonies per 1 ml of urine or the isolation of the same pathogen in cases of repeated crops is considered diagnostically significant, even if the number of colonies does not reach 100,000 per 1 ml of urine. With pyelonephritis, unilateral changes often predominate, so certain diagnostic information can be obtained as a result of a radiographic study. Methods for verifying the diagnosis are ultrasound examination of the kidneys and excretory infusion urography, less often a kidney biopsy. Treatment. Adequate therapy of chronic pyelonephritis is carried out. Drug treatment is the same as for chronic glomerulonephritis. Avoid the appointment of non-steroidal anti-inflammatory drugs (suppress the synthesis of renal vasodilators prostaglandinop), potassium-sparing diuretics and potassium preparations.

Phvochromocytoma is a tumor, usually benign, consisting of chromaffin cells and producing catecholamines (adrenaline and norepinephrine). In 90% of cases, pheochromocytoma is localized in the adrenal medulla, more often on the right. Paraganglioma - extra-adrenal chromaffin tissue - can be located in the gates of the kidneys, bladder, along the aorta (thoracic and abdominal).

The pathogenesis of arterial hypertension in pheochromocytoma is due to the release of a significant amount of catecholaminone, which leads to an increase in peripheral resistance. In some cases, hypnotic hypnosis in pheochromocytoma is paroxysmal in nature. It is believed that the inclusion of the renin-angiotensin-aldosterone system contributes to the development of a permanent form of arterial hypertension. During the period of crisis conditions with pheochromocytoma, blood pressure rises suddenly, within a few seconds it reaches a very high level (250-300 / 150-130 mm Hg), pronounced tachycardia, pallor of the face, cold sweat, vision is impaired. There is a strong thirst, the urge to urinate, the level of sugar in the blood rises. In the blood - leukocytosis. Crises can be provoked by a cold test, deep palpation of the abdomen, bringing the lower extremities to the stomach, taking dopegyt, reserpine, clonidine. The latter can be used for differential diagnosis. When taking 0.3 mg of clonidine in persons without pheochromocytoma, the level of catecholamines in the blood (after 2-3 hours) and urine (when taking the drug at 21 hours, urine is collected in the range from 21 to 7 hours) decreases sharply. In patients with a tumor, the content of catecholamines in the blood and urine does not change. The assumption of the presence of pheochromocytoma is confirmed by the determination of increased excretion of catecholamines and their metabolites in daily urine: adrenaline (more than 50 mcg), norepinephrine (more than 100-150 mcg), vanillindelic acid (more than 6 mcg), including within 3 hours after the next crisis . The diagnosis is verified using computed tomography, ultrasound. In recent years, scintigraphy with labeled 131J, an analogue of guanethidine, which is selectively captured by the tumor, has been increasingly used. Treatment. Emergency treatment is carried out with phentolamine (radical treatment - surgical removal of the tumor. If surgical removal is not possible,

Primary aldosteronism (Conn's syndrome) is manifested by clinically stable arterial hypertension, more often of the diastolic type due to an increase in aldosterone

synthesis in the glomerular layer of the adrenal cortex. The basis of this pathology in most cases is a solitary adenoma of the adrenal cortex (aldosteroma). The disease is more common in women.As a result of increased secretion of aldosterone, there is an increased retention of sodium (its ions) in the renal tubules and the accumulation of interstitial fluid - polyuria occurs. At the same time, there is an increased excretion of potassium ions, both in the renal tubules and in the intestines, salivary and sweat glands. Extracellular alkalosis occurs, renin secretion is suppressed. Low-renin volume (sodium)-dependent high arterial hypertension develops, especially with bilateral damage to the adrenal glands. Arterial hypertension is combined with such important diagnostic features as muscle weakness, especially in the muscles of the legs. Sometimes there are paroxysmal paralysis of the muscles of the legs, lasting from several hours to several days, spasms and contractures in the muscles of the legs, paresthesia, numbness. In the diagnosis of primary aldosteronism and its differential diagnosis, one should take into account the level of potassium (hypokalemia) and sodium in the blood serum, the state of acid-base balance, daily diuresis, which can range from 2 to 7 liters per day, urine density, usually significantly reduced, nocturia, isosthenuria , alkaline urine. Reduced or no plasma renin activity and increased urinary aldosterone excretion are characteristic features of primary aldosteronism. Hypokalemia can be confirmed by a hypothiazide test. From pharmacological tests, aldosterone antagonists (veroshpiron 100 mg / day for 4-5 weeks) can still be used to confirm the diagnosis, resulting in a decrease in diastolic blood pressure by at least 20 mm Hg. Art.

Treatment. For bilateral adrenal hyperplasia, potassium-sparing diuretics (spironolactone, amiloride, or triamterene) with or without nifedipine are indicated. With adrenal adenoma - surgical resection after preoperative treatment with spironolactone (veroshpiron, aldactone).

Arterial hypertension is often observed in such endocrine diseases as Itsenko-Cushing's syndrome (treatment - hypophysectomy, irradiation with heavy particles, adjuvant therapy with mibotan, parlodel), thyrotoxicosis (treatment - surgical or medication - Mercazolil, (beta-blockers), hypothyroidism (treatment alpha-thyroxine), acromegaly (treatment - transsphenoidal removal of an eosinophilic pituitary adenoma, radiation or adjuvant therapy with bromocriptine).

Hemodynamic arterial hypertension (coarctation of the aorta, aortic valve insufficiency) is treated surgically with the help of surgical correction of the vascular defect.

In the neuroendocrine form of the hypothalamic syndrome, arterial hypertension is treated with peritol (12 mg per day) or parlodel (5 mg per day).

Non-drug therapy: andlifestyle changes, dosed physical activity, weight loss, reduction of salt intake to 4.5 g per day, diet, reduction of alcohol consumption to 20-30 g of pure ethanol per day, smoking cessation, auto-training.

Medical therapy:

β-blockers, calcium antagonists, diuretics, ACE inhibitors, central alpha2-agonists, alpha-blockers, angiotensin II receptor antagonists, potassium channel activators.

- Monotherapy may be the initial treatment for moderately elevated blood pressure, with a low or moderate risk of complications.
- A combination of two drugs at low doses is reasonable as an initial treatment if BP is elevated to grade 2 or 3, and also if patients have a moderate increase in BP, but there is a high or very high risk of complications.
- In some cases, blood pressure control is not achieved with the use of two drugs. In these cases, a combination of three or more agents is required.
- In uncomplicated hypertension or in elderly patients, antihypertensive therapy should be given in stages.

MECHANISMS OF ACTION OF MAIN ANTIHYPOTENSIVE DRUGS

Group	Mechanism of action
ACE inhibitor	Inhibits ANF, which helps to reduce the effect of angiotensin II on blood vessels
Angiotensin receptor antagonists	weakening the effects of angiotensin II, which are mediated by AT1-angiotensin receptors
calcium antagonists	Reduces calcium entry into vascular smooth muscle cells
Beta blockers	competitive inhibitor of the binding of the mediator of the sympathetic nervous system to beta-adrenergic receptors.
Diuretics	SVR, natriuresis - fluid volume
Agonists imidazoline receptors	central action. Excitation of imidazoline receptors leads to suppression of the activity of sympathetic neurons of the spinal cord, which is accompanied by inhibition of the activity of the sympathetic nervous system with a decrease in the release of catecholamines, a decrease in sympathetic impulses to the vessels and the heart.
Alpha blockers	block alpha-adrenergic receptors in the muscular wall of blood vessels, as a result of which they expand

Drug treatment with ACE inhibitors can be prescribed for:

- AH + diabetes mellitus, AH high risk and very high risk + diabetes mellitus, AH high risk and very high risk + NK, CHF, AH high risk and very high risk + LV hypertrophy, AH high risk and very high risk + CKD, AH high risk and very high risk + dyslipedemia, hypertension high risk and very high risk + diabetic nephropathy, renovascular hypertension ("renal" hypertension), asymptomatic left ventricular dysfunction after myocardial infarction.

Drug treatment with calcium antagonists can be prescribed for:

AH + CAD, AH with high risk and very high risk + NC; AH high risk and very high risk + LV hypertrophy - when combined with ACE inhibitors; AH high risk and very high risk + dyslipidemia when combined with ACE inhibitors; pulmonary hypertension, hypertension in pregnancy.

Primary prevention of hypertension

- Measures aimed at raising the educational level of the population, shaping people's attitudes towards a healthy lifestyle and creating conditions for its implementation (renunciation of bad habits, rational nutrition, adherence to a rest regimen, prevention of physical inactivity, prevention of stressful situations, prevention of obesity and its treatment, correction dyslipedemia.

Secondary prevention of hypertension

- Preventive examination and tonometry
- Combating identified risk factors for hypertension in the population;
- Taking on "D" accounting of newly diagnosed patients;
- Treatment of hypertensive patients with drugs of proven efficacy, with individual selection of the group and dose, while observing the continuity and duration of antihypertensive drugs.
- Maintaining the target level of blood pressure
- Maintaining the quality of life.

Tertiary prevention of hypertension

-Prevention of complications

- Prevention of diabetes mellitus and coronary artery disease
- Psychological support

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BRONCHO-OBSTRUCTIVE SYNDROME. DIFFERENTIAL DIAGNOSIS IN BRONCHIAL OBSTRUCTIVE SYNDROME. SYNDROMES OF COUGH WITH SPUTUM AND HEMOPTYSIS. DIFFERENTIAL DIAGNOSIS OF COUGH WITH SPUTUM AND HEMOPTYSIS.

TRAINING TECHNOLOGY

Number of students-	Time - 2 hours
Form of the lesson	Lecture - visualization
Lecture plan	1. Definition of terms of broncho-obstructive syndrome (BOS). *2.* Causes and risk factors leading to biofeedback. *3.* 3. Various pathogenetic mechanisms of biofeedback. *4.* BOS classification. *5.* General differential diagnostic signs of biofeedback. *6.* Clinical manifestations and course of BOS diseases. *7.* Diagnosis of diseases accompanied by broncho-obstructive syndrome. *8.* Principles of treatment, prevention and clinical examination of patients with biofeedback.
Purpose of the lesson: to acquaint students with the etiology, pathogenesis of diseases accompanied by broncho-obstructive syndrome, to acquaint students with the modern classification of broncho-obstructive syndrome, to teach the principles of diagnosis, treatment, prevention and medical examination of diseases accompanied by biofeedback.	
Pedagogical tasks: 1. Strengthen and deepen students' knowledge of diseases accompanied by broncho-obstructive syndrome. 2. To teach students to correctly establish a diagnosis in accordance with the modern classification	**The results of the educational process:** **OP needs to know**: *1.* Risk factors leading to biofeedback. *2.* Diseases accompanied by BOS. *3.* Principles of differential diagnosis of diseases accompanied by broncho-obstructive syndrome. *4.* Principles of treatment. *5.* Principles of prevention and clinical examination of patients with biofeedback.

3. To teach students the ability to differentiate diseases accompanied by biofeedback. 4. Familiarize students with the stages of prevention of patients with biofeedback.	
Teaching methodology	Lecture text, videos, questionnaires, questions, “yes-no” technique
Form of study	Laser projector, visual materials, special technical equipment, presentation of thematic patients
Means of education	team
Conditions for the educational process	Audience

TECHNOLOGICAL CARD LECTURES

Stages, time	**Activity**	
	Teacher	**students**
Stage 1 Introduction (5 minutes)	1. Tells about the topic of the lecture, its purpose and plan	1. Listen
Stage 2 Actualization (increasing the importance) of knowledge (20 minutes)	2.1. In order to increase the actualization (increase in importance) of students' knowledge, asks questions: 1. Define the terms BOS? 2. List the diseases accompanied by biofeedback. 3. List the risk factors leading to biofeedback. 4. List the groups of drugs used to treat broncho-obstructive syndrome. Conducts a survey 2.2. Showing on the screen, offers students to get acquainted with the goals and objectives of the lecture. Slide #1	2.1. Answer questions asked 2.2. Study slide number 1 2.3. Study slide number 2
Stage 3 Main part (informational) (55 min)	3.1. Introduces students to the lecture material, the importance of the topic and the principles of the formation of an intelligent cultural personality, in particular, a GP teacher. In order to increase the actualization of knowledge, he conducts a quick survey of students:	3.1. Together they analyze the listened lecture material, ask questions

	1. According to 1 point of the lecture plan: Give a definition of the terms broncho-obstructive syndrome? *2.* According to paragraph 2 of the lecture plan: List the causes and risk factors leading to biofeedback. *3.* According to the 3rd point of the lecture plan: List the various pathogenetic mechanisms of broncho-obstructive syndrome? *4.* According to the 4th point of the lecture plan: Tell the main points of the BOS classification. *5.* According to paragraph 5 of the lecture plan: List the general differential diagnostic signs of broncho-obstructive syndrome. *6.* According to paragraph 6 of the lecture plan: List the main clinical manifestations and the course of diseases that occur with broncho-obstructive syndrome. *7.* According to paragraph 7 of the lecture plan: What are the main methods for diagnosing diseases accompanied by biofeedback. *8.* According to paragraph 8 of the lecture plan: List the basic principles of treatment, prevention and medical examination of patients with broncho-obstructive syndrome. Stopping at the important points of the lecture, he suggests writing down the main points in a notebook	Key points are written in a notebook.
<u>Stage 4</u> final (10 min)	4.1. Asking questions: 1. List the most common diseases accompanied by broncho-obstructive syndrome. 2. Tell the modern classification of biofeedback. 3. Name the basic principles of treatment and prevention of patients with broncho-obstructive syndrome.	4.1. Answer questions 4.2. Listen, write

	4.2. Gives a task for independent work of students: Broncho-obstructive syndrome.	

Chronic obstructive pulmonary disease -a collective concept that unites a group of chronic diseases of the respiratory system: chronic obstructive bronchitis (COB), pulmonary emphysema (EL), severe bronchial asthma (BA). COPD is also considered as a symptom complex with signs of terminal respiratory failure FEV1 <1.5 l or 30% of the expected value, i.e. progression of the disease, leading to the loss of the reversible component of bronchial obstruction, cor pulmonale. Causes of COPD: about 90% of COPD, about 1% of emphysema, about 10% of severe BA.

"Chronic obstructive pulmonary disease (COPD) is a collective term that combines chronic environmentally mediated inflammatory diseases of the respiratory system with a predominant lesion of the distal respiratory tract with partially reversible bronchial obstruction, which are characterized by progression and increasing chronic respiratory failure."

Classification of COPD by severity

Severity: mild: Main clinical signs: intermittent cough, shortness of breath only with intense physical exertion or absent. Functional indicators: FEV1> 70% of the expected values. Volumetric indicators are normal.

Moderate: Main clinical signs: persistent cough, most pronounced in the morning, scanty sputum, shortness of breath with moderate exertion. Scattered dry rales. Functional indicators: FEV1 - 50-69% of the expected values, an increase in residual lung capacity, transient episodes of hypoxia, signs of overload of the right heart.

Severe: Main clinical signs: persistent cough, dyspnea at rest, cyanosis, participation of accessory muscles in breathing, remote wheezing, signs of right ventricular failure. Functional indicators: FEV1 less than 50% of the expected values, hypoxia, hypercapnia, signs of cor pulmonale, fatigue of the respiratory muscles, erythrocytosis.

COPD Risk Factors

External factors: smoking, occupational hazards, ambient air pollution, low socioeconomic status, adenovirus infection, vitamin C deficiency. Internal factors: a-antitrypsin deficiency, prematurity, bronchial hyperreactivity, familial nature of the disease, genetic predisposition.

Causes of COPD: about 90% of COPD, about 1% of emphysema, about 10% of severe BA.

BRONCHIAL ASTHMA (BA) is a chronic persistent inflammatory disease of the airways (mainly bronchi) with their reversible obstruction, manifested by asthma attacks or status asthmaticus. Patients with asthma are characterized by high reactivity (hyperreactivity) of the bronchi in response to the action of various stimuli, which in healthy people does not cause a bronchospastic reaction.

As a result of the increase in the number of patients with chronic lung diseases, the allergization of the population, air pollution, the widespread use of antibiotics,

vaccines, serums, etc., there is an increase in the incidence of bronchial asthma, which sometimes causes death. The prevalence and form of asthma are influenced by the climate and natural features of the region.

Etiology and pathogenesis.AD is an etiologically and pathogenetically heterogeneous disease. The etiological factors of AD are schematically divided into exogenous and endogenous. The most common exogenous factors include: 1) allergization of the population (urbanization); 2) air pollution; 3) the introduction of chemistry into agriculture, industry and everyday life; 4) widespread use of antibiotics, vaccines, sera, etc.; 5) climatic and natural features of the region (for example, a cold and damp climate, an abundance of flowering plants, dustiness, etc.). Endogenous etiological factors of asthma are various infectious and inflammatory diseases of the respiratory organs (acute pneumonia, acute and chronic bronchitis), congenital or acquired defects and disorders of the immune, endocrine, neuropsychic, neurohumoral and other body systems. AD can be based on various specific (immunological) and (or) non-specific (non-immunological), congenital or acquired pathogenetic mechanisms. The main manifestation of BA is more or less pronounced paroxysmal disorders of bronchial patency, i.e. reversible (spontaneously or as a result of treatment) bronchial obstruction. Some patients with severe BA develop persistent, refractory to conventional therapy bronchial obstruction syndrome - status asthmaticus. The most common causes of status asthmaticus are acute infections and exacerbations of chronic infections of the upper respiratory tract, bronchopulmonary apparatus, viral diseases, unjustified dose reduction or withdrawal of glucocorticosteroids, overdose of sympathomimetic drugs, excessive use of sleeping pills,

Clinical picture and diagnosis. The clinical picture of asthma is diverse: from rare mild manifestations to prolonged and painful asthma attacks. Asthmatic attacks occupy a central place in the clinical picture of asthma. They usually come on at night, often unexpectedly, sometimes after unclear precursors in the form of a feeling of tightness in the chest, shortness of breath. At the beginning of the disease, asthma attacks are mostly short - from a few minutes to half an hour; in the future, they become longer, persistent, dragging on for a whole day and sometimes turning into an asthmatic status. Often during an attack (usually towards the end) with a cough, a small amount of mucous, viscous sputum is released. In grayish lumps, it is sometimes possible to see spirally twisted curls of mucus (Kurschmann spirals) and casts of small bronchi. For all clinical and pathogenetic variants, mild, moderate and severe BA are distinguished. Mild episodic course of BA: • asthma attacks are short-term and occur no more than 1-2 times a week; • nocturnal symptoms no more than 2 times a month; • in the interictal period, the state of health is not disturbed; • indicators of the function of external respiration - PSV or FEV, - not less than 80% of the due ones; • daily fluctuations in PSV or FEV do not exceed 20%; • after inhalation of a bronchodilator (with an exacerbation), the parameters of PSV and FEV return to normal values. Moderate course of BA: • daily symptoms; • the need for daily intake of short-acting p2-agonists; • nocturnal symptoms more than once a week; • exacerbations disrupt physical activity and sleep; • PEF or FEV is 60-80%

of the proper values; • daily fluctuations in PSV more than 30%. Severe BA: • symptoms are constant with frequent exacerbations; • physical activity is limited; • frequent nocturnal symptoms; • indicators of PSV or FEV| less than 60% of due; • daily fluctuations in PSV more than 30%. Patients with any severity (even mild) can develop severe and even life-threatening exacerbations.

A generally accepted etiological classification of bronchial asthma still does not exist due to the variety of causes that cause it, and the frequent combined effects of various factors. The following main forms of the disease are distinguished: allergic, non-allergic and mixed. Allergic AD is based on an immunological mechanism with excessive production of IgE. This leads to a massive release of mediators by the mast cell at any contact of the patient with the "guilty" allergen. Allergic asthma often occurs in people with a burdened family or personal allergic history, usually begins in childhood, in such patients - positive skin and provocative tests with non-infectious allergens, increased levels of general and specific IgE, there are other allergic manifestations (allergic rhinitis, conjunctivitis, atonic dermatitis). With non-allergic bronchial asthma, it is not possible to detect sensitization to a specific allergen. The onset of the disease occurs at a more mature age, and the triggering factor, as well as the "culprit" of the exacerbation, is most often a respiratory viral infection. A few days after the onset of a viral disease, shortness of breath, coughing, and asthma attacks appear, which can persist from several days to several months. Patients whose disease has signs of allergic and non-allergic asthma belong to the group of mixed asthma. Reliable diagnosis of BA is possible only by excluding secondary bronchospastic syndrome (BS). Secondary BS is most common in diseases of heteroallergic genesis (anaphylactic shock, serum sickness),

Treatment. The overall treatment program for a patient with asthma should include: 1) an educational program; 2) assessing and monitoring the severity of the disease; 3) exclusion of factors that provoke an exacerbation of the disease, or control over them; 4) development of an individual scheme of drug treatment; 5) development of a treatment plan for exacerbation of the disease, emergency treatment for an asthma attack and (or) status asthmaticus; 6) dispensary observation. Successful treatment of patients with asthma largely depends on the timely elimination of contact with allergens or reduction of their influence. Elimination of the allergen is quite effective in controlling the severity of the disease. The individual scheme of drug treatment is determined primarily by the severity of asthma. The prescription of medicines is carried out in the form of four steps with an increasing bronchodilatory and anti-inflammatory effect. The stage of treatment corresponds to the severity of BA: the first - mild BA, the second and (or) the third - moderate BA, the fourth - severe BA. The basis of basic anti-inflammatory therapy is non-steroidal anti-inflammatory drugs (cromoglycate and nedocromil sodium) and inhaled corticosteroids. Cromoglycate and nedocromil sodium are used for mild and some forms of moderate asthma. Stage 1. Mild episodic course of the disease. At this stage, there is no need to prescribe basic anti-inflammatory therapy and treatment consists of elimination measures (avoid triggers) and relief of seizures with the help of (short-acting 52-agonists. Perhaps the prophylactic use of p2-agonists or sodium cromoglycate before

exercise or exposure to other provoking factors. Stage 2. Mild persistent BA. This condition requires constant anti-inflammatory therapy, otherwise the disease will progress. Basic treatment begins with cromoglycate or nedocromil (especially at a young age) sodium. With insufficient effect, you should switch to inhaled glucocorticosteroids in low doses or combine them with cromolyns. An alternative to these drugs at this stage may be theophylline of prolonged action (serum concentration 5-15 μg / ml) or antileukotriene drugs (zafirlukast, zileuton). Short-acting p2-agonists are recommended for the relief of asthma attacks. Their frequent intake (daily) indicates the need to strengthen basic therapy. Stage 3. Moderate course of BA. Anti-inflammatory basic therapy in these patients includes inhaled glucocorticosteroids in medium doses or a combination of low doses of these drugs with long-acting p2-agonists (inhaled or oral). It is possible to replace long-acting p2-agonists with prolonged theophylline. As an alternative, anticholinergics (ipratropium bromide) or their combination with a P2 agonist can be used, especially in case of poor tolerance of p2 agonists or concomitant chronic obstructive bronchitis, as well as in the elderly. The insufficient effect of the ongoing basic anti-inflammatory therapy may require the use of high doses of inhaled glucocorticoids at this stage. Step 4 Severe BA. Anti-inflammatory therapy consists of: • regular use of inhaled glucocorticoids in high doses; • long-term use of oral corticosteroids (usually not more than 60 mg of prednisolone per day); • taking long-acting bronchodilators (long-acting p2-agonists or long-acting theophylline). With severe adverse reactions to these bronchodilators, it is possible to replace them or combine them with anticholinergics (ipratropium bromide). Short-acting p2-agonists are used as a symptomatic remedy for seizures, but not more than 3-4 times a day (the possibility of overdose). If more frequent use of p2-agonists is required, control of basic anti-inflammatory therapy is required. An additional treatment program for infectious-dependent allergic asthma includes immunotherapy aimed at increasing the body's resistance to respiratory tract infections, as well as the use of some modern antiviral and immunomodulatory agents. The latter include T-activin, to a lesser extent thymalin, decaris, in some forms interferon, drugs of the immunoglobulin group sometimes help. It is advisable to conduct immunotherapy with heterogeneous bacterial vaccines or mucopolysaccharide complexes, bacterial waste products (pyrogenal, prodigiosan). In the acute phase of the disease, it is necessary to eliminate the inflammatory process in the respiratory organs. For this purpose, antibiotics, sulfonamides, phytoncides are prescribed, as well as salicylates and other non-steroidal anti-inflammatory drugs. Symptomatic treatment is of no small importance in the complex therapy of patients with BA. So, to improve the drainage function of the bronchial tree and reduce the viscosity of sputum, expectorants are used. Secretolytic drugs are recommended that act directly on the bronchial mucosa (essential oils, iodides, sodium bicarbonate, etc.); secretomotor drugs (reflexively - through the stomach and vomiting center - increase bronchial secretion: infusion of thermopsis, marshmallow root, coltsfoot leaves, plantain) and mucolytics (trypsin, chymopsin, ribonuclease, mukaltin, bromhexine, bisolvone, etc.). expectorants are used to improve the drainage function of the bronchial tree and reduce the viscosity

of sputum. Secretolytic drugs are recommended that act directly on the bronchial mucosa (essential oils, iodides, sodium bicarbonate, etc.); secretomotor drugs (reflexively - through the stomach and vomiting center - increase bronchial secretion: infusion of thermopsis, marshmallow root, coltsfoot leaves, plantain) and mucolytics (trypsin, chymopsin, ribonuclease, mukaltin, bromhexine, bisolvone, etc.). expectorants are used to improve the drainage function of the bronchial tree and reduce the viscosity of sputum. Secretolytic drugs are recommended that act directly on the bronchial mucosa (essential oils, iodides, sodium bicarbonate, etc.); secretomotor drugs (reflexively - through the stomach and vomiting center - increase bronchial secretion: infusion of thermopsis, marshmallow root, coltsfoot leaves, plantain) and mucolytics (trypsin, chymopsin, ribonuclease, mukaltin, bromhexine, bisolvone, etc.).); secretomotor drugs (reflexively - through the stomach and vomiting center - increase bronchial secretion: infusion of thermopsis, marshmallow root, coltsfoot leaves, plantain) and mucolytics (trypsin, chymopsin, ribonuclease, mukaltin, bromhexine, bisolvone, etc.).); secretomotor drugs (reflexively - through the stomach and vomiting center - increase bronchial secretion: infusion of thermopsis, marshmallow root, coltsfoot leaves, plantain) and mucolytics (trypsin, chymopsin, ribonuclease, mukaltin, bromhexine, bisolvone, etc.).

Oxygen therapy is used to improve arterial oxygen saturation. The patient breathes a 24-28% oxygen-air mixture through the nose using a T-shaped tube. Effective non-drug methods of symptomatic treatment of patients with asthma are breathing exercises, chest massage, postural drainage, and acupuncture. With an intractable attack of suffocation in the conditions of the intensive care unit, plasmapheresis, therapeutic bronchoscopy, and artificial ventilation of the lungs (controlled breathing) are used. Emergency care for asthma attacks and status asthmaticus. A mild attack of suffocation can be stopped by ingestion of tablets of eufillin, theofedrine or anthastman, no-shpy, papaverine or halidor, 30-60 drops of solutan. Distractions (talking with the patient, hot foot bath, mustard plasters or cans on the back). Sometimes, one inhalation of adrenaline derivatives is enough to stop an attack - isoprenaline (Novodrin, Euspiran), orciprenaline (asthmopent, alupent), hexaprenaline (ipradol), ventolin (salbutamol), berotek (fenoterol). A moderate asthma attack is stopped in most cases with a 0.1% solution of adrenaline administered at a dose of 0.5-1 ml subcutaneously or as an aerosol. The action of adrenaline occurs after 2-3 minutes and lasts about 1 hour, so some patients need to re-introduce it (up to 10 times a day). To preserve the bronchospasmolytic action, it is advisable to administer a 5% solution of ephedrine at a dose of 0.5-1 ml subcutaneously or intramuscularly simultaneously with adrenaline; its action comes later and lasts several hours. Injected simultaneously with adrenaline, ephedrine enhances and prolongs the action of adrenaline. If there are contraindications to the administration of adrenaline (hypersensitivity), with hypertension and coronary artery disease, some of its derivatives can be used - orciprenaline (alupent), hexaprenaline (ipradol), ventolin (salbutamol), berotek (fenoterol). Alupent is used in the form of injections (0.5 mg), aerosol (2% and 5% solutions, up to 1 mg). Modern bronchospasmolytics (berotek, ventolin) are used in the form of an aerosol.

In the absence of the effect of adrenaline and its derivatives, aminophylline (aminophylline, diafillin) is used in the form of a 2.4% solution, 10 ml intravenously slowly. With side effects of aminophylline (nausea, vomiting, pain in the heart), solutions are used simultaneously: 2% - papaverine, 2 ml, 2% - noshpy, 2 ml, 1% - dibazole, 4-6 ml. With nocturnal attacks of suffocation and bradycardia, the patient's condition can be significantly improved by subcutaneous injection of 1 ml of a 0.2% solution of platifillin or 0.5-1 ml of a 0.1% solution of atropine, in some patients, especially with pain, inhalation of nitrous oxide gives a good effect in a mixture with oxygen using an apparatus for gas anesthesia. In case of a severe asthma attack that is not amenable to the above therapy and threatens to turn into a hypercapnic coma, it is necessary to call a specialized ambulance team. At the prehospital stage, the patient should be given intravenous adrenaline, eufillin in the usual dosage, alupent - 0.5 mg in 1 ml of sodium chloride solution slowly over 5 minutes or 5-20 mg in 250 mg of 5% glucose solution at a rate of 10-15 drops per minute, ipradol - 1-2 ampoules intravenously for 5 minutes.

Status asthmaticus (AS) is the most severe complication of asthma. It is an acute respiratory failure due to airway obstruction refractory to sympathomimetic and aminophylline therapy. Emergency care for AS consists of three mandatory components: oxygen therapy, infusion and drug (eufillin and its analogues, glucocorticosteroid hormones) therapy.

Oxygen therapy is carried out in the form of continuous insufflation of an oxygen-air mixture with a relatively low oxygen content (30-40%); infusion therapy - heparinized solution of 5% glucose, Ringer's solution, polyglucin, reopoliglyukin. The total volume of infusion therapy is 3-3.5 liters on the 1st day, in subsequent days - at the rate of 1.6 l / m2 of body surface. Drug treatment of AS is carried out with the most narrowed range of drugs, including the complete rejection of adrenostimulants. 15 ml of a 2.4% solution of eufillin in an isotonic solution of glucose or sodium chloride are administered intravenously for 4-6 minutes, together with 5000 IU of heparin. The daily dose of aminophylline is 1.5-2 g. Prednisolone is administered intravenously: from 30 to 90 mg. In case of decompensation, along with the above measures, therapeutic bronchoscopy with segmental bronchial lavage is performed, the patient is transferred to artificial lung ventilation. During the remission of BA, much attention should be paid to non-drug treatment: hypoallergenic diet, physiotherapy exercises, swimming, massage, physiotherapy, spa treatment (Southern coast of Crimea, Kislovodsk, high-mountainous regions of Elbrus region, etc.).

CHRONIC OBSTRUCTIVE BRONCHITIS (COB) -chronic diffuse non-allergic inflammation of the bronchi, leading to a progressive obstructive impairment of pulmonary ventilation and gas exchange and manifested by cough, sputum production and shortness of breath not associated with damage to other organs and systems.

ETIOLOGY AND PATHOGENESIS. Etiological factors are smoking (active and passive), air pollution, occupational hazards, a1-antitrypsin deficiency, viral infections, bronchial hyperreactivity. The main pathogenetic factors are dysfunction

of the local bronchopulmonary protection system, structural restructuring of the bronchial mucosa (hypertrophy of the mucous and serous glands, replacement of the ciliated epithelium with goblet cells), the development of the classical pathogenetic triad (hypercrinia, dyscrinia, mucostasis), and the release of inflammatory mediators and cytokines.

Clinical picture and diagnosis. Most often, patients with COB complain of cough, shortness of breath and sputum production. An increase in body temperature and hemoptysis are much less common. Shortness of breath is the most characteristic symptom of COB. It is predominantly expiratory in nature, occurs with significant physical exertion, but gradually, as the disease progresses, it becomes permanent. Characterized by shortness of breath in the morning, accompanied by a hacking cough and wheezing. For patients with COB, shortness of breath is the main cause of deterioration in the quality of life. Cough in COB is protracted, often hacking with sputum difficult to separate, especially in the morning. The presence of obstruction leads to the progression of COB, pulmonary emphysema, the development of cor pulmonale, the occurrence of atelectasis of complex origin and, as a result of the latter, to pneumonia. In the blood, even during the period of exacerbation of the disease, changes may be absent. Sometimes moderate leukocytosis, a shift of the leukocyte formula to the left, a slight increase in ESR are determined. Radiological symptoms in most patients are not detected for a long time. In some patients, radiographs show diffuse, uneven amplification and deformation, as well as a change in the contours of the lung pattern due to mesh peribronchial pneumosclerosis, with emphysema - an increase in the transparency of the lung fields. The obstructive nature of chronic bronchitis is confirmed by the data of a functional study (pneumotachometry, spirography). Diagnostic criteria for COB: actually bronchial obstruction (clinical manifestations and a decrease in FEV1 less than 84% or a decrease in the TIFFNO index below 88% of the proper values); irreversibility or partial reversibility of bronchial obstruction, variability of FEV1 values by less than 12% during the day; stably confirmed bronchial obstruction; age is usually over 50 years; physical and radiological signs of emphysema; steady progression of the disease in the absence of adequate treatment; detection of the disease is usually in smokers. over 50 years; physical and radiological signs of emphysema; steady progression of the disease in the absence of adequate treatment; detection of the disease is usually in smokers. over 50 years; physical and radiological signs of emphysema; steady progression of the disease in the absence of adequate treatment; detection of the disease is usually in smokers.

Treatment of patients with COB should begin as early as possible. It is important to eliminate all factors that cause irritation of the bronchial mucosa (smoking, unsuitable working conditions, climatic zone, etc.).

Therapeutic measures should first of all be aimed at eliminating the inflammatory process and respiratory infection, hyposensitization, correction of secondary immunodeficiency states and reduced nonspecific resistance of the organism, improvement of bronchial patency, correction of hypoxia. It is necessary to sanitize chronic foci of nasopharyngeal infection, to ensure free breathing through the nose.

The inflammatory process is stopped by non-steroidal anti-inflammatory drugs (aspirin, ibuprofen, reopyrin, etc.). With exacerbation of COB with purulent sputum, antibacterial drugs should be prescribed, which are effective primarily against influenza bacillus, pneumococcus and gram-negative microbes. After each endobronchial sanitation procedure, positional drainage and vibration massage of the chest are performed. To restore bronchial patency, selective stimulants of p2-adrenergic receptors (berotec, bricanil, salbutamol), acetylcholine blockers (atrovent), methylxanthine preparations (eufillin, long-acting theophyllines - teopek, durofilin, etc.), calcium channel blockers (corinfar) are prescribed. Combined drugs are effective - theofedrin, solutan, berodual, etc. Expectorants (infusions of thermopsis, coltsfoot, wild rosemary, thyme, plantain, marshmallow root decoction, 3% potassium iodide solution, etc.) contribute to better sputum discharge, inhalation alkaline solutions, plentiful hot drink, intake of alkaline mineral waters. With viscous sputum, inhalations of enzymes are prescribed - trypsin, chymotrypsin, chymopsin, ribonuclease, deoxyribonuclease. Mucolytic agents are also successfully used to thin sputum: 10% solution of acetylcysteine 2 ml intramuscularly or as an aerosol inhalation 3 times a day, bromhexip (bisolvon) orally, parenterally or as an aerosol inhalation 4-7 mg 2-3 times a day day. The drainage function of the bronchi in patients with purulent chronic bronchitis can be improved with the help of therapeutic bronchoscopy (2-4 procedures with an interval of 3-7 days) with intratracheal administration of drugs (antibiotics; mucolytic drugs - acetylcysteine, chymotrypsin, trypsin, ribonuclease; bronchodilators - ephedrine, naphthyzinum; antiseptic solutions - furacilin, furagin, etc.). To stop bronchospasm, an UHF electric field is prescribed, followed by electrophoresis of platifillin (0.1% solution), aminophylline (5% solution). To improve sputum discharge, iodine electrophoresis (5% solution) with inductothermia, trypsin or pancreatin electrophoresis, etc. are used. The use of various distractions (mustard plasters, cans, warm foot baths, etc.) has not lost its significance. When the phenomena of exacerbation of the disease subside, usually from the 4th-6th day from the onset of the disease, physiotherapeutic procedures are prescribed (sollux, UV radiation, UHF currents, electrophoresis of novocaine, calcium chloride on the chest). To improve bronchial patency and restore the drainage function of the bronchi, chest massage and physiotherapy exercises, including postural drainage, are prescribed. Sanatorium-and-spa treatment for chronic bronchitis is carried out in the warm, dry season at the climatic resorts of the southern coast of Crimea, the middle mountains (Kislovodsk, Teberda) or in local sanatoriums (Bobruisk, Naroch). The use of various distractions (mustard plasters, cans, warm foot baths, etc.) has not lost its significance. When the phenomena of exacerbation of the disease subside, usually from the 4th-6th day from the onset of the disease, physiotherapeutic procedures are prescribed (sollux, UV radiation, UHF currents, electrophoresis of novocaine, calcium chloride on the chest). To improve bronchial patency and restore the drainage function of the bronchi, chest massage and physiotherapy exercises, including postural drainage, are prescribed. Sanatorium-and-spa treatment for chronic bronchitis is carried out in the warm, dry season at the climatic resorts of the southern coast of Crimea, the middle

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EMPHYSEMA OF THE LUNG. In practical medicine, the term “pulmonary emphysema” refers to any increase in the airiness of the lungs, which can be functional (acute swelling of the lungs), compensatory (vicarious), senile (atrophic), chronic substantial (obstructive), etc. The most common forms of pulmonary emphysema are divided into two large groups - non-obstructive and obstructive pulmonary emphysema. With non-obstructive emphysema, there are no obstruction phenomena of terminal bronchioles and small bronchi, and with obstructive emphysema, ventilation disorders caused by collapse of terminal bronchioles and (or) obstruction of small bronchi form part of the disease potential. Non-obstructive pulmonary emphysema includes compensatory and senile, and to obstructive - functional and chronic substantive emphysema as an independent form of chronic obstructive pulmonary disease. An emphysematous lung resembles worn,

overstretched rubber, which, having lost its elastic properties, cannot be reduced to its former volume. An emphysematous lung does not collapse, and therefore, occupies a larger volume than a healthy one.

Pathogenesis. In most cases, pulmonary emphysema is secondary and is the result of diffuse lung diseases, primarily chronic obstructive bronchitis (obstructive, centrilobular, or centriacinar, pulmonary emphysema). The emphysematous process affects the alveoli located near the respiratory bronchioles, since the inflammatory-dystrophic process passes from the respiratory bronchioles to the alveoli adjacent to them. Emphysema of the lungs can develop without previous lung disease (primary diffuse, idiopathic or panacinar). The occurrence of primary emphysema is primarily associated with a hereditary deficiency of the alpha-1 protease inhibitor (alpha-1-antitrypsin), disorders of the pulmonary microcirculation, changes in the properties of the surfactant, smoking, etc. are also of some importance. Primary emphysema with hereditary alpha-1 antitrypsin deficiency occurs in young people, therefore it is called juvenile emphysema. In recent years, there has been a significant increase in COPD patients with alpha-1 antitrypsin deficiency. Primary panacinar (panlobular) emphysema evenly affects all the alveoli that make up the lung lobule. The functioning surface of the lung is reduced.

Clinical picture and diagnosis. The clinic of pulmonary emphysema is mainly limited to the symptom complex of increased airiness of the lung tissue, as well as symptoms of respiratory and heart failure. Clinical manifestations of chronic substantive (true) pulmonary emphysema noticeably “lag behind” pathomorphological ones, therefore, already fully formed emphysema is diagnosed by clinical signs. The main complaints of patients with emphysema are shortness of breath and fatigue. Patients with emphysema, suffering from chronic bronchitis, also complain of a dry cough, aggravated by shortness of breath. Patients with emphysema are outwardly characterized by raised shoulders, a short neck, a barrel-shaped chest, squeezed from the sides, with intercostal spaces that retract when inhaled, and acrocyanosis. The chest bulges out. The intercostal spaces are dilated and even protrude, the supraclavicular regions are smoothed or bulge, the respiratory excursion of the chest decreases. Auxiliary muscles are involved in the act of breathing: on inspiration - sternocleidomastoid and scalene muscles, on exhalation - anterior serratus and abdominal muscles. On exhalation, patients cover their mouths, puffing out their cheeks (puffing). Percussion defines a “box” sound, limited mobility of the pulmonary edge, low standing and a decrease in the mobility of the diaphragm, weakened breathing with increased exhalation. Scattered dry, sometimes moist, small bubbling rales are heard. The boundaries of the heart are determined with difficulty. In patients with emphysema, body weight decreases, which is explained by the predominance of catabolic processes, which mainly affect the alveoli and, to a lesser extent, other tissues. With severe respiratory failure in peripheral blood, erythrocytosis and an increase in hemoglobin may be noted. In patients with primary panacinar pulmonary emphysema, the level of alpha-1-antitrypsin in the blood serum is lowered. The most characteristic radiological symptoms of primary panacinar emphysema are: a homogeneous increase in the

transparency of the lung fields and depletion of the lung pattern, especially in the lower sections, low standing of the diaphragm and a “hanging heart”. With secondary centriacinar emphysema, the transparency of their lower sections is significantly reduced due to peribronchitis and other changes. The diaphragm usually does not move downwards, since the total volume of the lungs is not significantly changed. In patients with primary panacinar pulmonary emphysema, the level of alpha-1-antitrypsin in the blood serum is lowered. The most characteristic radiological symptoms of primary panacinar emphysema are: a homogeneous increase in the transparency of the lung fields and depletion of the lung pattern, especially in the lower sections, low standing of the diaphragm and a “hanging heart”. With secondary centriacinar emphysema, the transparency of their lower sections is significantly reduced due to peribronchitis and other changes. The diaphragm usually does not move downwards, since the total volume of the lungs is not significantly changed. In patients with primary panacinar pulmonary emphysema, the level of alpha-1-antitrypsin in the blood serum is lowered. The most characteristic radiological symptoms of primary panacinar emphysema are: a homogeneous increase in the transparency of the lung fields and depletion of the lung pattern, especially in the lower sections, low standing of the diaphragm and a “hanging heart”. With secondary centriacinar emphysema, the transparency of their lower sections is significantly reduced due to peribronchitis and other changes. The diaphragm usually does not move downwards, since the total volume of the lungs is not significantly changed. With secondary centriacinar emphysema, the transparency of their lower sections is significantly reduced due to peribronchitis and other changes. The diaphragm usually does not move downwards, since the total volume of the lungs is not significantly changed. With secondary centriacinar emphysema, the transparency of their lower sections is significantly reduced due to peribronchitis and other changes. The diaphragm usually does not move downwards, since the total volume of the lungs is not significantly changed.

Treatment.A complete cure for pulmonary emphysema is impossible due to its steady progressive course and the irreversibility of structural changes in the lung tissue. Therefore, the treatment of emphysema is mainly aimed at combating the disease that caused its development (for example, chronic bronchitis, respiratory failure, pulmonary hypertension and chronic decompensated cor pulmonale). In the presence of chronic obstructive bronchitis, expectorants and cough-calming agents (thermopsis, mucolytics, codeine), aerosol therapy, tracheobronchial infusions of antimicrobial solutions, enzymes are prescribed; with bronchospasm - bronchospasmolytics (eufillin); with decompensated pulmonary heart - cardiac glycosides (corglicon), drugs that unload the pulmonary artery system (lasix). Do not use respiratory stimulants since the respiratory center in patients with emphysema remains functionally intact. Eufillin is most effective when administered intravenously (2.4% solution of 5 ml in isotonic sodium chloride solution 2-3 times a day in repeated 10-day courses). physical activity, training in rational breathing with the participation of the diaphragm. It is recommended to work in a warm room with good ventilation and clean air. Physical therapy can be

of great benefit, which strengthens the respiratory muscles and ensures a full exhalation. Particular attention is paid to breathing exercises. Chest massage, aerotherapy walks in the fresh air with a gradual lengthening of the route are also prescribed.

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LECTURE TOPIC: "DIFFERENTIAL DIAGNOSIS OF EDEMATOUS SYNDROME. MODERN METHODS OF TREATMENT FOR CHF. PREVENTION."

TRAINING TECHNOLOGY

Number of students -	Time - 2 hours
Form of the lesson	Lecture - show and demonstrate
Lecture plan	1. Give an explanation for the edematous syndrome 2. The main causes of edematous syndrome 3. The pathogenesis of edematous syndrome 4. Clinical signs of edematous syndrome 5. Characteristic signs of edematous syndrome in diseases of cardiovascular diseases 6. CHF classification 7. Characteristic signs of edematous syndrome in kidney disease 8. Characteristic signs of edematous syndrome in liver diseases 9. Diseases accompanying false edema and their clinical signs 10. Diseases accompanied by regional edematous syndrome 11. GP tactics in diagnosing NS 12. Treatment of nephrotic syndrome

Purpose of the lesson: To provide students with information about edematous syndrome, classification of nephrotic syndrome, the main diagnostic criteria for diseases associated with nephrotic syndrome, complications, diagnosis and training in prevention and emergency measures.	
Pedagogical tasks: 1. Provide complete information to students about the classification, clinical signs and factors leading to edematous syndrome. To deepen their knowledge of diagnosis and emergency care for edematous syndrome. 2. Explain the principles of differential diagnosis of diseases accompanied by edematous syndrome 3. To achieve independent mastery of practical skills in edematous syndrome and diseases accompanied by edematous syndrome 4. Explain to students the principles of prevention	***Learning outcomes:*** **OP needs to know**: 1. Be able to diagnose edematous syndrome 2. To know the clinical signs of the main diseases accompanied by edematous syndrome 3. To acquire knowledge of the main causes leading to edematous syndrome 4. To be able to determine the diagnostic criteria for diseases accompanied by edematous syndrome 5. To be able to determine the diagnostic criteria for diseases accompanied by regional edematous syndrome 6. Principles of diagnosis and treatment of diseases accompanied by edematous syndrome 7. Prevention and rehabilitation of diseases accompanied by edematous syndrome
Teaching Methods	The text of the lecture, videos, brainstorming, solving situational problems, competitions between groups, “yes-no” technique
Training uniform	Laser projector, visual materials, communication with specialized equipment
Training aids	Team
learning tool	Audience

TECHNOLOGICAL CARD LECTURES

Stages, time	**Activity**	
	teacher	**students**
Stage 1 **Login(5min)**	1. Tell the topic, goals and expected plans	1. Listen
2nd stage	2.1. Ask questions to students to improve their knowledge	2.1. Answer questions

Improve knowledge (20 minutes)	1. Edema syndrome syndrome, what do you mean? 2. List the group of diseases accompanied by edematous syndrome? 3. What are the main signs of edematous syndrome in cardiovascular diseases? 4. Classification of chronic heart failure 5. The main signs of edematous syndrome in kidney diseases 6. The main signs of edematous syndrome in liver diseases Conducts a quick survey 2.2. Offers to get acquainted with the purpose of the lecture by showing on the screen. Gives explanations of the information presented at the lecture Slide 1, 2,...	2.2. #1 Familiarize yourself with the slides 2.3. No. 2 get acquainted with the slides
Stage 3 **Main stage (informative) (55 minutes)**	3.1. To acquaint students with the content of the lecture and methods for the development of interpersonal relationships. Improves students' knowledge of the topic of the project and conducts "brainstorming" 1 - question of the plan: Give an explanation for the edematous syndrome? 2 - a question of the plan: list the main signs of edematous syndrome in diseases of the cardiovascular disease? 3 - the question of the plan: the main causes of nephrotic syndrome? 4 - question of the plan: Classification of chronic heart failure 5 - question of the plan: Characteristic signs of regional edema? 6 - question of the plan: Characteristic signs of inflammatory edema? 7 - a question of the plan: the principles of treatment of edematous syndrome? Offer to stop and write down the main points of the lecture	3.1. Asking questions and discuss the given materials Write down the main points
Stage 4 **Final (10 minutes)**	4.1. The question is asked: 1. Explain the edematous syndrome 2. Variants of edematous syndrome 3. The main causes of general edematous syndrome	4.1. answer questions

	4. Diseases accompanied by regional oedema syndrome 5. treatment of edematous syndrome based on the underlying disease 4.2. Give tasks for independent work: Treatment of diseases accompanied by regional edematous syndrome	4.2. listening, recording

Edema syndrome- excessive accumulation of fluid in the tissues of the body and serous cavities, manifested by an increase in the volume of tissues and a change in the capacity of the serous cavity, a change in the physical properties and a violation of the functions of edematous organs and tissues.

Edema syndrome is an excessive accumulation of fluid in body tissues and serous cavities, manifested by an increase in tissue volume and a change in the capacity of the serous cavity, a change in physical properties and a violation of the functions of edematous organs and tissues.

On palpation, a pasty consistency of the skin is felt, after pressing with a finger, a hole remains on it. If, after pressing a finger on the edematous skin, there is no fossa, then the edema can be attributed to false.

There are local (localized) edema associated with fluid retention in a limited area of the tissues of the body or organ, and general (generalized) - a manifestation of the positive water balance of the body as a whole. Generalized edema includes edema in heart failure, cirrhosis of the liver, nephrotic and nephritic, dropsy of pregnancy, cachexic and idiopathic, as well as as a result of chronic loss of potassium by the body during the abuse of laxatives. Contribute to the appearance of edema or accelerate their development can: phenylbutazone, pyrozolone derivatives, mineralocorticoids, androgens, estrogens, licorice root preparations.

Edema is one of the most common symptoms of somatic pathology and occurs in a number of diseases and pathological conditions. Currently, there is no generally accepted classification of edema, but the presented clinical classification of edema summarizes the main characteristics of this symptom:

The main causes of the development of edematous syndrome

General edema:

- ❖ Heart disease
- ❖ kidney disease
- ❖ Liver disease
- ❖ Hypoproteinemia
- ❖ Idiopathic edema (Parchon's syndrome).

local edema

Venous edema:

- ➢ Acute deep vein thrombosis;
- ➢ Chronic venous insufficiency;
- ➢ Venous obstruction

Lymphedema:

1. Idiopathic:

a) congenital;
b) early.
2. Inflammatory.
3. Obstructive.
Fat edema.

Other types of edema:

- traumatic
- Endocrine
- Myxedema.
- Neurogenic edema
- Hypothalamic edema.
- Trophedem Mezha.
- Complex regional pain syndrome (reflex sympathetic dystrophy).

Edema caused by medication:

- Hormones (GCS, female sex hormones, estrogens, progesterone, testosterone).
- Antihypertensive drugs (rauwolfia alkaloids, β-blockers, calcium tubule blockers, etc.)
- Non-steroidal anti-inflammatory drugs (butadione, naproxen, ibuprofen, indomethacin).
- Other medicines (MAO inhibitors, antidepressants, midantan).
- False edema
- Myxedema
- Systemic scleroderma.
- Dermatomyositis

Edema mechanisms

The pathophysiological mechanisms of edema are a decrease in oncotic pressure (i.e., pressure formed due to the osmotic activity of albumin) and osmotic pressure of blood plasma (pressure due to plasma osmolarity, one of its components is oncotic pressure). Also, edema develops with an increase in the hydrostatic pressure of blood in the capillary. The opposing forces P guide are R onk, R osm and R t. Tissue pressure is the sum of the osmolarity of the interstitial space and the pressure of the interstitial tissue on the capillary wall. An important factor in the appearance of edema is an increase in the permeability of the capillary wall, which develops during hypoxia, hypercapnia, acidosis, inflammation, and an increase in P guide.

In connection with the above, the listed pathological processes trigger different pathophysiological mechanisms for the development of edema. This is reflected in the nature of differential diagnosis and therapeutic measures. The diagnostic task in the form of edema is formed on the basis of the listed main causes of edema, pathophysiological mechanisms and clinical classification. In order to determine whether the selected pathophysiological mechanism is appropriate, it is necessary to keep in mind the diagnostic algorithm. Of course, each of the pathological conditions

that lead to the formation of edema has its own clinical picture, which to reflect in the algorithm means to make it cumbersome and non-functional.

Factors contributing to the development of edema are:

- a decrease in tissue pressure when the connective tissue is depleted of collagen with an increase in its friability, for example, when hyaluronidase is released, which is observed in inflammatory and toxic edema

- low pressure in the pleural cavity facilitates the development of hydrothorax with general edema in patients with circulatory failure.

-positive water balance of the body is based on excess retention of sodium by the kidneys. The emerging hyperosmia of the extracellular space causes an increase in the secretion of vasopressin, which enhances the reabsorption of water in the renal tubules and leads to its excessive retention in the body. Rarely, edema is based on primary hypersecretion of vasopressin. Hypersecretion of aldosterone caused by hypovolemia or decreased cardiac output is considered to be the main cause of sodium accumulation in edematous syndrome. The associated decrease in renal blood flow increases the secretion of renin by the kidneys, increases the formation of angiotensin II, which stimulates the secretion of aldosterone. As a result of sodium reabsorption in the distal nephron, the osmotic pressure of the extracellular fluid increases,

Thus, the main factors leading to disruption of the local water balance can be the following:

1. Increase in hydrostatic pressure in capillaries.
2. Decreased oncotic pressure of blood plasma.
3. Increased oncotic pressure of the interstitial fluid.
4. Decreased tissue mechanical pressure.
5. Increased capillary permeability.
6. Violation of the outflow of lymph.

Causes of edematous syndrome

Chronic heart failure

Heart failure is a pathological condition in which cardiac output does not meet the needs of the body due to a decrease in the pumping function of the heart.

Symptoms of heart failure:

Causes of heart failure:

The cause may be an overload of the heart with an increased volume of blood and (and) pressure (with heart defects, hypertension, cor pulmonale, etc.), as well as a decrease in the contractile function of the myocardium with a decrease in its mass (myocardial infarction, postinfarction cardiosclerosis, cardiac aneurysm, etc.).), degenerative changes (with amyloidosis, hemochromatosis, etc.), myocarditis, cardiomyopathy, myocardial dystrophy of various etiologies.

Cardiac edema as a manifestation of heart failure

In patients with chronic heart failure, heart diseases (malformations, atherosclerotic cardiosclerosis, myocardial infarction, heart aneurysm, congestive cardiomyopathy, etc.) are detected. The expansion of the boundaries of the heart of one degree or another, hepatomegaly are determined. Arrhythmias, especially atrial

fibrillation, are often noted. The position of orthopnea, oliguria, nocturia, congestion in the lungs, swelling of the jugular veins are characteristic. The development of edema is usually preceded by shortness of breath. Edema grows slowly, usually spreading from the bottom up. They are symmetrical, slightly displaced. The pronounced dependence of edema on the position of the body is taken into account: the appearance on the legs in walking patients and on the lower back in bedridden patients. Edema is usually worse in the evening. They are characterized by a doughy consistency; when pressed, a fossa that does not disappear for a long time remains. The skin over the edema area is cold, cyanotic. With the prolonged existence of edema, trophic changes in the skin, cracks, and dermatitis occur. In severe cases (anasarca), external edema is combined with ascites, hydrothorax, more often right-sided, less often with hydropericardium.

In chronic heart failure, edema is associated with an increase in P guide and an increase in the permeability of the vascular wall due to circulatory hypoxia. Since the increase in P guide is due to a decrease in the contractile activity of the right ventricle, hypertension in the vena cava system leads to an increase in pressure in the venules and capillaries of the microvasculature. Peripheral edema is caused by isolated right ventricular heart failure or its combination with left ventricular failure. In addition to hemodynamic causes, the development of secondary hyperaldosteronism with water and sodium retention in heart failure should be pointed out, which is a factor that independently causes or increases edema.

Since the main role in the genesis of edema is played by the hydrostatic component, they are observed in those parts of the body in which the P guide is positionally higher (lower limbs). Edema of the legs tends to decrease in the prone position, while swelling of the face and upper limbs increases. Edema usually improves with loop diuretics. Perhaps the development of anasarca, as well as abdominal and parenchymal edema, often associated with peripheral edema. Edema in heart failure is always accompanied by its other symptoms in the form of general weakness, shortness of breath, and malaise. In history, as a rule, there is a chronic heart disease (arterial hypertension, coronary heart disease, cardiomyopathy, congenital or acquired heart disease, etc.). Shortness of breath worsens with walking and lessens with rest. Echocardioscopy reveals signs of systolic dysfunction of the left ventricular myocardium in the form of a decrease in ejection fraction, an increase in end-systolic and diastolic volumes, and a decrease in stroke volume. In the study of blood, hypoalbuminemia is not observed, and in the study of urine - proteinuria, which makes it possible to exclude nephrotic syndrome or protein starvation as causes of edematous syndrome. However, the phenomenon of congestive kidney is known, which develops in chronic heart failure and manifests itself as a slight proteinuria with a decrease in renal function, which is undulating and increases or decreases as signs of heart failure decompensation increase or stop. This pathological condition is not always easy to differentiate from glomerulonephritis on the background of heart failure. With a congestive kidney (secondary nephropathy against the background of chronic heart failure), there is no nephrotic syndrome, hematuria is extremely rare, and the explanation for its appearance often

lies in the plane of iatrogenic pathology (drug interstitial nephritis). The latter also needs to be differentiated from a congestive kidney. The presence of isolated erythrocyturia, the absence of increased edema, the presence of a causative drug or a combination of them (NSAIDs, analgesics, aminoglycosides), as well as the restoration of renal function after discontinuation of these drugs allows differential diagnosis. and the explanation for its appearance often lies in the plane of iatrogenic pathology (drug interstitial nephritis). The latter also needs to be differentiated from a congestive kidney. The presence of isolated erythrocyturia, the absence of increased edema, the presence of a causative drug or a combination of them (NSAIDs, analgesics, aminoglycosides), as well as the restoration of renal function after discontinuation of these drugs allows differential diagnosis. and the explanation for its appearance often lies in the plane of iatrogenic pathology (drug interstitial nephritis). The latter also needs to be differentiated from a congestive kidney. The presence of isolated erythrocyturia, the absence of increased edema, the presence of a causative drug or a combination of them (NSAIDs, analgesics, aminoglycosides), as well as the restoration of renal function after discontinuation of these drugs allows differential diagnosis.

Not all pathological conditions are accompanied by the development of edema, but their appearance against the background of chronic heart failure explains their combination with edematous syndrome.

Moreover, in some cases, the genesis of edematous and urinary syndromes is explained by one disease. For example, in a patient suffering from infective endocarditis, valvular heart disease develops with the development of severe heart failure and, as a result, edematous and urinary syndromes (congestive kidney), kidney damage proceeds according to the type of glomerulonephritis caused by an immunocomplex mechanism, leading to the formation of a urinary syndrome and introducing contribution to the development of edema. In this case, puncture nephrobiopsy does not always allow for differential diagnosis, and due to the severity of the condition and the presence of contraindications for biopsy, the latter seems hardly possible.

HNK classification according to N.D. Strazhesko and V.Kh. Vasilenko

Stage I	**Latent HF manifested by shortness of breath, palpitations, fatigue only during exercise. At rest hemodynamics is not disturbed.**
Stage II A	**Signs of heart failure at rest are moderate, exercise tolerance is reduced, there are disorders in the systemic or pulmonary circulation, asthma attacks, radiographic, and in some cases ECG signs of secondary pulmonary hypertension, peripheral edema and a moderate increase in liver size are observed.**

Stage II B	**Pronounced signs of CHF at rest, severe hemodynamic disturbances in the systemic and pulmonary circulation.**
Stage III	**Terminal-with slight movement or even at rest, severe violations of hemodynamics, metabolism, irreversible degenerative changes in organs and tissues.**

HNK classification of the New York Heart Association (NYHA)

I-class	**Patients with heart disease, but without restrictions on physical activity. Asymptomatic dysfunction of the left ventricle.**
II-class	**Patients with heart disease causing slight limitation of physical activity. mild heart failure.**
III-class	**Patients with heart disease causing significant limitation of physical activity. Moderate heart failure.**
IV-class	**Patients with heart disease in whom even minimal physical activity causes discomfort. Severe heart failure.**

Edema associated with kidney disease

Hypooncotic edema may occur with hypoproteinemia (less than 50 g/l). At the same time, albumin deficiency (less than 25 g/l), which has a significantly higher osmotic activity than globulins, is of particular importance.

Edema in nephrotic syndrome appears primarily in places with the most loose subcutaneous tissue: on the face (especially in the eyelids), on the anterior abdominal wall, in the genital area. Shortness of breath is not typical. There is no dependence of edema on the position of the body. Gradually, edema can reach the degree of anasarca. Often they are accompanied by ascites, less often by hydrothorax. Oliguria is optional. Characterized by high proteinuria (daily - more than 3 g), hypoproteinemia, dysproteinemia, hyperlipidemia, often - an abundance of cylinders in the urine (granular, fatty, waxy) and fat-transformed renal epithelium. With an unclear cause of nephrotic syndrome, a kidney biopsy is indicated.

Nephritic edema is most often observed in acute or chronic glomerulonephritis (without nephrotic syndrome), occurs due to a decrease in the filtration charge of sodium and an increase in the permeability of the capillary wall. The rapid development of edema is characteristic (in a few days). However, they can sometimes appear earlier than changes in the urine. Edema is moderately expressed, localized in places with the most loose fiber (eyelids, face), stronger - in the morning, rather soft and mobile. The skin over the edema area is warm and pale. Trophic changes are not typical. Often, edema is combined with oliguria, arterial hypertension. Almost always there is a urinary syndrome (moderate proteinuria, hematuria, cylindruria). There are no laboratory signs of nephrotic syndrome. Nephrotic syndrome

The genesis of edema in nephrotic syndrome is associated with a decrease in plasma oncotic pressure due to a decrease in albumin concentration. These edemas are also called protein-free. Characteristic are their symmetry, as well as localization on the lower extremities. The latter is observed in patients who most of the time are in a standing or sitting position with their legs down. Edematous tissues tend to form in those parts of the body in which the venous blood pressure is positionally increased. Edemas are able to migrate positionally, i.e. depending on the position of the patient's body, the hands, face and neck, body can swell. With a long prone position, swelling of the lower extremities decreases, swelling of the upper extremities and face appears. Quite often intensity of hypostases has asymmetric character. With prolonged lying on the side, the limbs of the lateral side swell more. Obligatory companions of edema in nephrotic syndrome are high proteinuria and hypoalbuminemia. The absence of one of these signs casts doubt on the nephrotic syndrome.

Edema associated with kidney disease

- The formation of edema is accompanied by a decrease in diuresis (daily amount of urine up to 500 ml or less).
- Body weight increases in a short time.
- Edema appears in the morning and disappears during the day.
- Edema of the subcutaneous tissue at first is limited, local, later (sometimes quickly) the edema becomes widespread (anasarca). - The beginning of edema from the face (around the eyes, eyelids, cheeks, etc.) and its spread to the trunk and limbs is characteristic.
- There is accumulation of fluid in the cavities, often ascites and rarely hydrothorax.
- Often there is swelling of the internal organs (heart, liver, lungs, brain), which can create a certain clinical picture depending on the localization. - The rapid appearance of edema and also a rather rapid disappearance are characteristic.
- Edematous skin is pale, of normal temperature.
- The consistency of edema is soft, pasty.
- With large edema, signs of dystrophy of the skin and its appendages are visible: dryness, sometimes shiny, atrophic, peeling of the epidermis, brittleness, fading of hair and nails. - Edema quickly shifts when changing body positions.
- Leave a hole when pressed with a finger, which quickly returns to its previous position. - In most cases, shortness of breath usually does not occur. The liver is not enlarged.

Edema in diseases of the liver.

Edema caused by hypoalbuminemia in liver diseases can manifest itself in advanced stages of severe liver diseases (chronic hepatitis, cirrhosis) with a pronounced violation of the albumin-synthesizing function of the liver. More often in liver diseases, ascitic syndrome dominates (often in combination with right-sided hydrothorax).

Edema of hepatic origin. In the anamnesis, the transferred viral hepatitis, alcoholism, toxic effects are revealed; - There are no pronounced signs of impaired functioning of the heart and kidneys;

- Identify symptoms of chronic liver failure (cirrhosis), arterial spider hemangiomas, hepatic palms (erythema), gynecomastia and developed venous collaterals on the anterior abdominal wall;

- Edema is localized mainly in the abdominal cavity with the formation of ascites.

- Edema on the legs join relatively rarely;

- Occurs without clinical signs with a small amount of fluid;

With an increase in the amount of fluid, stretching of the abdominal cavity, an unpleasant sensation, anorexia, nausea, heartburn, pain in the right hypochondrium, and respiratory disorders are observed. Swelling or swelling of the lateral parts of the abdomen;

- Symptom of undulation or "splash noise" on auscultation;

- Shortening of percussion sound in the lateral parts of the abdomen with a change in the position of the patient or the appearance of dullness in the center of the abdomen with the knee-elbow position of the patient. - Ascites can be combined with swelling of the external genitalia, umbilical or inguinal hernia, pleural effusion, often right-sided.

In the blood, a change in the amount of bilirubin, enzymes.

Protein starvation

Edema and hypoalbuminemia in protein starvation are interrelated. Edema occurs in the case of protein or complete starvation, subject to free fluid intake. In this case, edema may appear already on the 3-5th day.

When conducting differential diagnosis, it is of great importance to interview the patient with the construction of a food diary, as well as the presence of hypoalbuminemia in the absence of proteinuria. Edema is symmetrical. Most often located on the legs. Normalization of nutrition or intravenous drip in severe cases of albumin solution is accompanied by relief of edema. The effect of furosemide is low and increases only after the infusion of albumin solution.

Enteropathy with increased protein loss.

*Primary exudative enteropathy*characterized by increased loss of protein through the ectatic lymphatic vessels of the small intestine mucosa.

Main clinical manifestations:

- pronounced massive hypoproteinemic often asymmetric edema;

- swelling appears on the legs, arms, face, lower back;

- Sometimes anasarca;

- Ascites and hydrothorax develop quite quickly;

- Diarrhea appears before edema, often simultaneously with them.

Stool without mucus, blood;

- Patients are concerned about pain in the abdomen, polyfecal matter, constant bloating, rumbling, increased flatulence;

*With secondary exudative enteropathy*leading signs - increasing protein deficiency with a significant decrease in body weight, edematous - ascitic syndrome, dystrophic changes in all organs and systems of the body.

Hyperaldosteronism

Hyperaldosteronism is common in clinical practice and is mainly associated with three main problems: the use of diuretics, CHF and nephrotic syndrome. In this case, hyperaldosteronism is designated as secondary. There is also Kohn's syndrome, caused by a benign tumor of the adrenal gland (aldosteroma) with hyperproduction of aldosterone. In this case, primary hyperaldosteronism takes place. Removal of the tumor is accompanied by relief of the clinic of the disease.

In the diagnosis of hyperaldosteronism, the determination of the level of aldosterone in the blood is used. When taking blood in the supine position, the concentration of aldosterone is normally 8-172, when taken in a standing position - 30-355 mg / ml.

In primary hyperaldosteronism, the concentration of aldosterone in the blood exceeds the normal 5-10 times. There is a retention of sodium and water, increased excretion of potassium in the urine. Edema is symmetrical. Swelling of the face, limbs. Arterial hypertension often develops.

Unlike primary hyperaldosteronism, secondary hyperaldosteronism does not lead to the development of hypertension syndrome. The edematous syndrome (pastosity of the face, fingers and toes) is much less pronounced. Hyperaldosteronism is not an independent cause of anasarca, however, severe edema, for example, in nephrotic syndrome or chronic heart failure, usually occurs with secondary hyperaldosteronism. This is due to many reasons. Chief among them are the use of loop diuretics to combat edema with the development of hyponatremia, the formation of hypovolemia (nephrotic syndrome), circulatory hypoxia (chronic heart failure). They are a powerful stimulus for the secretion of renin, which activates the conversion of angiotensinogen to angiotensin I, which leads to an increase in the concentration of angiotensin II and aldosterone. The secretion of aldosterone also increases directly under the influence of these factors.

When taking diuretics, especially loop diuretics (furosemide, torasemide, ethacrynic acid), hyponatriosis is observed, which is a powerful stimulus for aldosterone secretion. In this regard, the daily intake of furosemide in the same dose is accompanied by a gradual decrease in diuresis, despite the absence of complete relief of hyperhydration phenomena. In pseudo-Bartter's syndrome, the main pathogenetic link in developing changes due to prolonged abuse of furosemide is hyperaldosteronism.

Allergic edema

- Allergic edema appears in the form of the so-called Quincke's edema, sometimes together with urticaria and other allergic diseases (bronchial asthma, vasomotor rhinitis, hay fever).

- Urticaria and angioedema are more common in girls. - A few minutes after contact with the allergen, the patient develops erythema on the skin, then urticaria, severely itchy elements appear.

Skin rashes are very diverse in nature: nodular, blisters of various sizes and shapes.

- When reacting to food before the appearance of typical rashes, the patient feels tingling of the tongue, lips, palate, swelling in these places, often sharp pains in the abdomen. There may be perioral and perianal dermatitis.

- Often observed phenomena of conjunctivitis, less often - shortness of breath due to swelling of the larynx. Sometimes patients develop vomiting, collapse, anaphylactic shock.

Myxedematous edema

- Characteristic for severe primary or secondary hypothyroidism and are associated with a lack of thyroid hormones in the body.

- This leads to an increase in the hypophilicity of tissues that swell more than normal.

- Characterized by: drowsiness, lethargy, memory loss, slowing down of speech, movements, fatigue, decreased performance, arthralgia.

- Swelling of the face and extremities, fossa does not remain when pressed.

The nature of the edematous fluid resembles mucus (mucous edema - myxedema).

- The skin is dry, pale, with a yellowish tint.

- Breakage and loss of hair on the head, lateral third of the eyebrows.

- The tongue is thickened, along the edges there are impressions from the teeth.

- Hoarseness of voice.

- Decreased body temperature.

- Bradycardia, deafness of heart sounds, less often normal heart rate, rarely tachycardia.

- A tendency to hypotension, in 10-20% of patients - arterial hypertension, which, as a rule, decreases or disappears during therapy with thyroid drugs.

- Hypochromic and IDA often develop.

- Some patients may develop lactorrhea and amenorrhea.

Inflammatory edema are local, for example, around a boil, carbuncle, phlegmon, and other purulent foci. The mechanism of their occurrence is associated with the leakage of fluid from the vessels into the focus of inflammation, in which hydrogen ions accumulate in large quantities. A distinctive feature of inflammatory edema is skin hyperemia, pain at rest or on palpation, and an increase in local temperature. They usually develop on one leg or arm. Changes in urine are not accompanied, with the exception of a small transient proteinuria (in the form of traces), as a result of high body temperature and intoxication.

Edema syndrome in diseases of the joints

In diseases of the joints, edematous syndrome has a very specific picture. Unlike all other variants of limb edema, “articular” is local in nature. It appears in the area of the affected joint, without spreading in the distal or proximal direction. The manifestation of edema is clearly associated with the appearance of other symptoms - pain in the joint, significantly aggravated by its flexion and extension, limitation of the volume of active and passive movements. Many patients note the so-called starting problems - joint stiffness in the morning after a night's rest, which disappears after 10–20 minutes of movement. Regression of edema is observed as inflammation subsides, with the next acute episode, edematous syndrome reappears. In some patients with gonarthrosis, swelling or pastosity of the lower leg and supramallear region is possible.

Edema occurring in osteoarticular diseases:

-It is almost always local and occurs in the area of the affected joint in the acute period of the disease, combined with severe pain and limitation of movement in the affected joint. After the course of treatment, the edema disappears, although with a long course and frequent exacerbations, the deformation of the surrounding tissues ("pseudo-edema") becomes permanent.

Edema of pregnant women

With a normal pregnancy, swelling of the ankles in the later stages are quite common. The development of dropsy in pregnant women is associated with impaired water-salt metabolism and blood circulation in the system of capillaries and precapillaries as a result of altered neuroendocrine regulation. Usually dropsy of pregnancy is detected after the 30th week of pregnancy, rarely - earlier. Noteworthy is a more significant increase in body weight (1-2 kg per week, while in normal pregnancy it is 300-400 g per week). Initially, edema appears on the feet and legs, then rises higher. Even with large edema, ascites and hydrothorax are not observed. The general condition remains satisfactory. Unlike nephropathy in pregnant women, there are no changes in the urine (in particular, there is no proteinuria) and arterial hypertension. The prognosis is usually favorable. In rare cases, a transition to nephropathy of pregnant women is possible.

Cyclic edema

Cyclic edema in the so-called premenstrual syndrome is apparently due to hormonal imbalance (excess estrogen and lack of progesterone), which affects vascular permeability and promotes sodium and water retention. Often revealed insufficiency of the corpus luteum and anovulatory cycles. In the second half of the cycle, small swelling of the legs and feet usually appear. Possible swollen gums. Sometimes dizziness is noted, which is associated with swelling of the structures of the inner ear. In addition, weakness, irritability, headaches, sleep disturbance (insomnia or excessive drowsiness), and sometimes a depressive state are observed. After the cessation of menstruation, these phenomena quickly decrease and disappear.

Edema associated with impaired venous outflow

Venous edema

-WITHassociated with acute and chronic deep vein thrombosis of the extremities.

- Unilateral lesions.
- Edema appears suddenly, often against the background of complete health.
- For acute thrombosis, soreness on palpation of the edema area with the appearance of a fossa with pressure is typical.
- Edema covers both the lower leg and thigh.
- In the first days, the edema is of an increasing nature, accompanied by bursting pains in the limbs
- It proceeds with an increase in the venous pattern on the thigh and in the inguinal region.
- After a few weeks, the swelling decreases but almost never completely disappears.

Varicose veins of the lower extremities are widespread among the population. It is much more common in women over 40 years of age. Often, other signs of congenital weakness of the connective tissue in the body are found: hernias of different localization, hemorrhoids, flat feet. In most cases, the veins of the lower extremities, branches of the great saphenous vein are involved. Characterized by a slow, gradual development of the disease. Initially, there is a feeling of heaviness in the legs, sometimes - aching pain, convulsive muscle contractions at night. After a long walk or standing, pastosity of the legs and feet is noted. Later, excruciating skin itching may join. Swelling of the legs is more pronounced in the evening, during the night they decrease. Pain is more often unilateral, and with a bilateral process, it is asymmetric. At a late stage of the disease, trophic ulcers, dermatitis, eczema, recurrent erysipelas. Edema becomes dense due to induration of subcutaneous tissue, increases with the complication of the process with thrombophlebitis, lymphangitis. The diagnosis is usually made on examination. The difference in the volume of the limbs (measured with a centimeter tape) and the asymmetry of skin temperature are assessed. To assess the functional ability of the valvular apparatus of the veins (communication and deep), the Trendelenburg–Troyanov, Perthes, Prett, and other tests are used, which are described in detail in surgical manuals.

Thrombosis of the deep veins of the legs (often the anterior and posterior tibial veins, venous sinuses of the soleus muscle, etc.) is predominantly secondary (with diseases of the superficial veins), less often primary. The presence of venous adhesions, septa, compression of the veins by tendon-ligament formations contribute to the primary lesion. Characterized by pain in the calf muscles, which are aggravated by movements in the ankle joint. There is a moderate swelling of the lower leg in the ankle area (to clarify the presence of edema, the circumferences of both legs should be measured in symmetrical areas). There is a local increase in body temperature, pain on palpation of the muscles of the leg.

The following diagnostic symptoms are checked:

1) Homans: in the position of the patient lying down, the movement of the foot in the back direction causes pain in the calf muscles;

2) Moses: with deep vein thrombophlebitis, pain occurs when the lower leg is compressed in the anteroposterior direction and does not appear with compression in the lateral direction (the latter is characteristic of myositis and inflammatory diseases of the subcutaneous tissue);

3) Lowenberg: when the middle third of the lower leg is compressed with a sphygmomanometer cuff, pain occurs at a pressure below 150 mm, while in a healthy person, slight pain is noted only at a pressure of 180 mm or more.

Postthrombophlebitic syndrome is a consequence of acute deep vein thrombosis (after which partial recanalization of the lumen of the vein remains), as well as phlebosclerosis and insufficiency of venous valves, leading to chronic impairment of venous outflow. In connection with venous hypertension in the affected veins, there is a pathological discharge of blood into the saphenous veins with their secondary varicose veins. Most often, the deep veins of the legs are affected, less often - the iliac-femoral venous segment. Among patients, women aged 30–60 years predominate. Edema of the legs is most pronounced on the shins, much less on the thighs. They increase in the evening, with prolonged standing, decrease in the supine position, especially with a raised leg. Swelling is considered small if the circumference of the lower leg is increased by 2 cm compared to a healthy leg, moderate severity - by 2-4 cm, a sharp edema gives an increase of more than 4 cm. The edema is accompanied by a feeling of heaviness, fullness in the leg, pulling pains, aggravated by physical exertion. When pressed, the hole usually does not remain. The skin of the lower leg atrophies, the hairline disappears. Diffuse or patchy brown pigmentation of the skin is visible, especially in the lower third of the lower leg, as well as cyanosis. As a rule, there is an unsharply pronounced secondary varicose expansion of the superficial veins. In contrast to primary varicose veins in post-thrombophlebitis syndrome, varicose veins are insignificant, and trophic disorders (up to trophic ulcers) are pronounced. When pressed, the hole usually does not remain. The skin of the lower leg atrophies, the hairline disappears. Diffuse or patchy brown pigmentation of the skin is visible, especially in the lower third of the lower leg, as well as cyanosis. As a rule, there is an unsharply pronounced secondary varicose expansion of the superficial veins. In contrast to primary varicose veins in post-thrombophlebitis syndrome, varicose veins are insignificant, and trophic disorders (up to trophic ulcers) are pronounced. When pressed, the hole usually does not remain. The skin of the lower leg atrophies, the hairline disappears. Diffuse or patchy brown pigmentation of the skin is visible, especially in the lower third of the lower leg, as well as cyanosis. As a rule, there is an unsharply pronounced secondary varicose expansion of the superficial veins. In contrast to primary varicose veins in post-thrombophlebitis syndrome, varicose veins are insignificant, and trophic disorders (up to trophic ulcers) are pronounced.

Lymphostasis

Primary (idiopathic) elephantiasis is a genetically determined defect of the lymphatic vessels of the legs at the collector level. This disease is more common in young women. Puffiness may appear first on one leg; then both legs are involved in the process. But the asymmetry usually persists. The process goes through three stages in succession: lymphedema (mild lymphatic edema); transitional, with the gradual development of fibrotic changes in the distal part of the limb; fibrodemas (with total tissue fibrosis). In the first stage, edema is unstable. They are aggravated in hot weather and with prolonged standing, decrease in the supine position. The consistency is doughy, when pressed, a hole remains. The skin is not changed, easily shifted, pale. Further, the distal parts of the limb are compacted, the skin thickens, poorly displaced, when pressed, there is no longer a hole. With further progression, hyperkeratosis, papillomatosis, tissue growth in the form of shapeless bumps ("pillows"), separated by deep folds, join. Characterized by increased sweating and hypertrichosis on the affected side. Complications join: lymphorrhea in case of skin lesions (in this case, edema may decrease), dermatitis, trophic ulcers, the development of purulent-septic infection is possible.

Secondary elephantiasis develops after recurrent erysipelas, transferred lymphadenitis and lymphangitis, pyoderma, chronic vaginitis and proctitis, with compression of the lymphatic vessels by scars after mechanical injuries, deep burns. This complication is also possible after radiation therapy and during surgical removal of collector lymphatic vessels and lymph nodes in cancer patients. Clinical manifestations are similar to those observed in primary elephantiasis. An appropriate history is essential for diagnosis. Lymphography revealed tortuosity of the lymphatic vessels. Often multiple lymphangiectasias are found.

Idiopathic edema

Idiopathic edema occurs more often in middle-aged women (35–50 years old) who are prone to overweight and autonomic disorders, especially in menopause. Idiopathic edema is usually small, soft, appears on the feet and legs by the end of the day, in the morning it can be on the eyelids and fingers (it is difficult to put on a ring), more pronounced in hot weather. Periods of fluid retention may spontaneously change to periods of profuse diuresis with the onset of general weakness. In most cases, swelling goes away on its own. Small doses of aldosterone antagonists (veroshpiron) are effective.

Chronic venous insufficiency

CVI is currently the most urgent problem in therapeutic practice due to the high prevalence of chronic venous diseases (CVD) of the lower extremities, both among the working population and among the elderly and senile. The incidence of chronic venous pathology in the adult population as a whole is approximately 30%, reaching 80% in older age groups. Unfortunately, in real practice there is a certain stereotype of the action of a general practitioner in the case when a patient presents with complaints characteristic of a venous disease: the patient immediately receives a referral to a surgeon, phlebologist, etc. At the same time, only 10-15% of patients

with CVI need surgical treatment, and the basis of the treatment program for CVD is conservative means: elastic compression and pharmacotherapy.

The development of CVD is based on violations of the tone and structural restructuring of the walls of the veins of the lower extremities as a result of a chronic aseptic inflammatory process, the formation of valve failure and damage to the surface tissues of the lower extremities as a result of leukocyte aggression. These features of the pathogenesis of CVD explain the fact that surgery is not a solution to problems in most patients. Surgical intervention, unfortunately, cannot restore venous tone, remove edema, and prevent the negative consequences of leukocyte activation. In this regard, therapeutic agents that affect the main mechanisms of the pathogenesis of the disease become relevant.

Lipedema

- The etiology is not known.

- We can talk about an inherited defect of the subcutaneous tissue.

- Lipedema is found only in women. A similar picture can also be observed in their relatives in the descending or ascending line.

- A symmetrical increase in the volume of subcutaneous adipose tissue is characteristic only on the legs. - The volume and shape of the thigh and foot remain unchanged.

- Signs of CVI are not detected.

- Has an orthostatic character.

- Increases before menstruation, prolonged sitting, bathing in warm water, uncontrolled use of salt.

- Palpation of the lower leg often causes pain.

Algorithms of action of the therapist in case of detection in a patient with CVD

1. Determine the need for surgical intervention.

2. If there are contraindications to surgical treatment or the patient refuses surgery, the therapist should prescribe conservative treatment and recommend dispensary observation.

3. If CVD variants requiring surgical care are identified and there are no contraindications to surgery, the therapist should refer the patient to the polyclinic surgeon.

4. In case of an acute situation (varicothrombophlebitis, deep vein thrombosis), the therapist should urgently send the patient for a consultation with a surgeon. The latter, when confirming the diagnosis, ensures the hospitalization of the patient in a surgical hospital.

In the treatment of edematous syndrome, the following plan must be followed:

1. Treatment of the underlying disease.

2. Rational order of treatment: Creating an optimal physical and psychological environment for patients at home and at work.

3. Therapeutic nutrition: Diet - complete, quickly digestible, rich in proteins, vitamins, potassium. With a large fluid retention and arterial hypertension, the amount of salt and water is limited.

Treatment of renal edema.

-Glucocorticosteroids, cytostatics, pulse therapy, plasmapheresis, hemosorption; - Symptomatic treatment: ACE inhibitors, diuretics (hypothiazide, furosemide, uregit); - In a nephrotic crisis to prevent the development of hypovolemia and to restore circulating blood (in / in reopoliglyukin, dextrans, hemodez, protein solutions).

Treatment of hepatic edema.

- Diuretics: spironolactone, furosemide
- Protein preparations - fresh frozen plasma, albumin; e- Abdominal paracentesis; - Ascitosorption; uh
- Ultrafiltration of blood;
- Surgical treatment.

Treatment of exudative hypoproteinemic enteropathy

- Reducing protein loss;
- Elimination of diarrhea;
- Treatment of dysbacteriosis and insufficient absorption syndrome;
- Correction of metabolic disorders;
- Protein hydrolysates, anabolic hormones, calcium, iron, vitamins;
- If necessary, antibiotics, corticosteroids;
- Thiazide and potassium-sparing diuretics.

Methods of treatment of chronic venous insufficiency.

The vast majority of patients with chronic venous insufficiency are shown long-term, regular compression therapy, on an outpatient basis - preferably with the help of medical compression stockings. Such knitwear is divided into preventive and therapeutic (depending on the amount of pressure exerted in the supra-ankle region), is selected according to individual measurements and is sold in pharmacies or specialized stores. A contraindication to the use of compression agents are chronic obliterating lesions of the arteries of the extremities with a decrease in regional systolic pressure on the tibial arteries below 80 mm Hg. Art. (according to ultrasound data). The use of this method of treatment can be difficult during the period of exacerbation of arthrosis of the knee and ankle joints, in the hot season, with individual intolerance to compression products.

Pharmacotherapy of CVD is aimed at normalizing the structure and function of the venous wall, as well as stopping leukocyte aggression, one of the leading causes of trophic complications in this pathology. Indications for pharmacotherapy of CVD are presented in Table. 3.

In most clinical situations, the ideal treatment for patients with CVD is Detralex, a universal protector of the venous wall and valvular apparatus, which has the ability to suppress the adhesion of leukocytes in the lumen of capillaries, increase

the number of functioning lymphatic vessels and accelerate lymph transport, reduce blood viscosity and the speed of erythrocytes.

The effectiveness of Detralex (a micronized fraction of flavonoids) in relation to the main symptoms of CVD (feelings of heaviness, fever and pain in the legs, swelling of the extremities and trophic ulcers up to 10 cm in size) has been proven in numerous randomized multicenter placebo-controlled trials (Evidence level A) and confirmed in International guidelines for the treatment of CVD of the lower extremities, adopted in 2007.

The ease of use of Detralex (2 tablets 1 time per day in the evening), the versatility of the standard dosage and course of treatment, and good tolerance allow us to recommend this drug for widespread use in the practice of therapeutic specialists as an initial remedy already in the initial detection of patients with venous and lymphatic edema.

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LECTURE TOPIC: "JOINT SYNDROME. DIFFERENTIAL DIAGNOSIS IN ARTICULAR SYNDROME. INDIVIDUAL APPROACH TO TREATMENT. PREVENTION."
TRAINING TECHNOLOGY

<table>
<tr><td>Number of students -</td><td>Time - 2 hours</td></tr>
<tr><td>Form of the lesson</td><td>Lecture - visualization</td></tr>
<tr><td>Lecture plan</td><td>1. Features of the articular syndrome
2. The main diseases accompanied by articular syndrome
3. Features of the course of diseases occurring with articular syndrome
4. Diagnosis of diseases accompanied by articular syndrome.
5. Principles of treatment, prevention and clinical examination of patients with articular syndrome in the practice of general practitioners.</td></tr>
<tr><td colspan="2">The purpose of the lesson: to familiarize students with the etiology, pathogenesis of diseases accompanied by articular syndrome, to teach the principles of diagnosis, treatment, prevention and medical examination of diseases accompanied by articular syndrome.</td></tr>
<tr><td>Pedagogical tasks:
1. Strengthen and deepen students' knowledge of diseases accompanied by articular syndrome
2. To teach students to correctly diagnose in accordance with the modern classification of diseases
3. To teach students the ability to differentiate diseases accompanied by articular syndrome
4. To acquaint students with the peculiarities of the course of diseases accompanied by articular syndrome,
5. To teach students the management of patients with articular syndrome in the practice of GP.</td><td>The results of the educational process:
The GP needs to know:
1) Features of the articular syndrome
2) The main diseases accompanied by articular syndrome
3) Features of the course of diseases occurring with articular syndrome
4) Diagnosis of diseases accompanied by articular syndrome.
5) Principles of treatment, prevention and clinical examination of patients with articular syndrome in the practice of general practitioners.</td></tr>
</table>

Teaching methodology	Lecture text, videos, questionnaires, questions, “yes-no” technique
Form of study	Laser projector, visual materials, special technical equipment, presentation of thematic patients, X-ray images of patients
Means of education	Team
Conditions for the educational process	Auditorium

TECHNOLOGICAL CARD LECTURES

Stages, time	Activity	
	Teacher	students
Stage 1 Introduction (5 minutes)	1. Tells about the topic of the lecture, its purpose and plan	1. Listen
Stage 2 Actualization (improvement) of students' knowledge (20 minutes)	2.1. In order to increase the actualization (increase in importance) of students' knowledge, asks questions: *1)* List the diseases that may be accompanied by articular syndrome? *2)* List the objective signs that could indicate the presence of articular syndrome? *3)* List diseases that are accompanied by significant morning stiffness? Conducts a survey (or conducts a survey) 2.2. Showing on the screen, offers students to get acquainted with the goals and objectives of the lecture. Slide #1, #2	2.1. Answer questions asked 2.2. Study slide number 1
Stage 3 Main part (informational) (55 min)	3.1. Introduces students to the lecture material, the importance of the topic and the principles of the formation of an intelligent cultural personality, in particular the GP. In order to increase the actualization of knowledge, he conducts a quick survey of students:	3.1. Together they analyze the listened lecture material, ask questions

	1) According to 1 point of the lecture plan: tell the features of the articular syndrome *2)* According to paragraph 2 of the lecture plan: List the main diseases accompanied by articular syndrome *3)* According to the 3rd point of the lecture plan: Features of the course of diseases that occur with articular syndrome *4)* On the 4th point of the lecture plan: Diagnosis of diseases accompanied by articular syndrome. *5)* On the 5th point of the lecture plan: Principles of treatment, prevention and clinical examination of patients with articular syndrome in the practice of GPs. Stopping at the important points of the lecture, he suggests writing down the main points in a notebook	Key points are written in a notebook.
<u>Stage 4</u> Final (10 min)	4.1. Asking questions: 1. List the most common diseases, articular syndrome 2. Tell the main key points of the clinical course of various diseases accompanied by articular syndrome 4. Name the basic principles of treatment, prevention and rehabilitation of patients with articular syndrome. 4.2. Gives a task for independent work of students: Basic principles of primary prevention of diseases with articular syndrome associated with infection.	4.1. Answer questions 4.2. Listen, write

Differential diagnosis of articular syndrome

Articular syndrome is an almost universal manifestation of rheumatic diseases; its differential diagnosis underlies the definition of the nosological form, and therefore serves as a rationale for choosing a therapeutic approach. In the advanced stages of the disease, when there are organic changes in organs and tissues, the diagnostic problem is greatly simplified. Serious analysis is required in the debut, often presented exclusively by arthralgias.

Currently, rheumatologists use the following classification, where all forms of rheumatic diseases are divided into the following:

I. Rheumatism
II. Diffuse connective tissue diseases
III. Systemic vasculitis
IV. Rheumatoid arthritis
V. juvenile arthritis
VI. Ankylosing spondylitis (Bechterew's disease)
VII. Arthritis associated with spondyloarthritis
VIII. Arthritis associated with infection
IX. Microcrystalline arthritis
X. Osteoarthritis
XI. Other joint diseases
XII. Arthropathy in non-rheumatic diseases
XIII. Diseases of extra-articular soft tissues
XIV. Diseases of bones, cartilage and osteochondropathy

Examination of patients complaining of arthralgia aims to identify which structures of the musculoskeletal system are the source of pain or dysfunction. Joints are made up of surfaces of articular cartilage, bone, ligaments, and synovium. The joint space is not an empty space, as it seems to us when examining radiographs, but is represented by articular cartilage, transparent to x-rays, therefore, it is possible to assess the degree of cartilage destruction radiologically by measuring the distance between two bone surfaces. Cartilage differs from bone in a more elastic composition, has a lower coefficient of friction and, most importantly, does not have the regenerative abilities of bone. Therefore, cartilage damage should be considered as an irreversible process.

A defect or loss of cartilage can occur in two ways:

- mechanical abrasion, as is the case with osteoarthritis;
- erosion as a consequence of inflammatory synovitis in rheumatoid arthritis or other rheumatic diseases.

Synovia extends between the osteocartilaginous borders on both sides; normally, it does not cover the articular cartilage. Its surface is represented by one or two layers of synoviocytes capable of morphological adaptation, reflecting the function performed by the cell at the moment - synthetic or phagocytic. The histological picture of the initial stage of inflammatory synovitis is similar in most diseases. For example, ankylosing spondylitis cannot be distinguished from

rheumatoid arthritis by synovial biopsy. Only in some situations, such as tuberculous arthritis, the diagnosis can be based on biopsy data.

With the course of rheumatoid arthritis, the process acquires the following characteristic features: erosive synovitis develops, corroding both cartilage and bone. Hypertrophied during the inflammatory process, synovial villi are attached to the adjacent edge of the articular cartilage, creeping onto its surface. This inflammatory tissue replaces the cartilage. This cartilage replacement tissue is known as pannus and is made up of fibrous tissue infiltrated with chronic inflammatory cells, including mast cells. At the edge of the articular cartilage, the pannus replaces the bone tissue, causing the occurrence of erosions that are detected by x-ray examination. Pannus can also penetrate the subchondral bone plate and grow into the subchondral bone.

Because cartilage breaks down faster, progressive erosive synovitis appears on x-ray first as cartilage loss, then as periarticular bone erosion. It is important to note that persistent synovitis prevents the formation of osteophytes in the vicinity of the decreasing cartilage tissue. So, radiological signs of chronic persistent synovitis are:

- cartilage loss;
- usuration of the bone adjacent to the places of thinning of the cartilaginous plates;
- no signs of osteophytes.

As the disease progresses, the vascularization of the synovium decreases (compared to earlier stages), which, on the one hand, is associated with fibrosis as a stage in the evolution of the disease, and, on the other hand, with immobility developing due to fibrosis. The so-called "burnt out rheumatoid arthritis" is formed. This expression can be considered unsuccessful, since, although physical examination reveals the absence of hyperthermia, effusion and hyperemia, patients still have morning stiffness, an increase in ESR and, more importantly, bone usuration increases with dynamic radiological observation.

Degenerative joint diseases have completely different characteristics.

- *No synovitis. (This, by the way, explains the low effectiveness of anti-inflammatory drugs in this disease.)*
- *Cartilage defect is localized in places of mechanical damage. Often there is a neighborhood of almost normal cartilage with areas where the cartilage is completely worn out.*
- *The appearance of osteophytes in the neighborhood of areas of cartilage defects.*

Only in the later stages of osteoarthritis, pain syndrome can occur both during exercise and at rest and disrupt night sleep. Since the cartilage does not have a regenerative ability, once the symptom complex has arisen, it tends to progress. However, in cases of normal household loads, the cartilage wears out very gradually and the deterioration increases gradually, over the years. The symptoms of synovitis persist at rest and are only accentuated during exercise.

Morning stiffness, characteristic of rheumatoid arthritis, ankylosing spondylitis, and other systemic diseases, usually lasts at least two hours. This

symptom is associated with a physiological drop in the level of corticosteroids in the blood in the pre-morning hours and with the accumulation of cytokines from the inflammatory fluid during sleep. Morning stiffness in osteoarthritis is transient, lasts no more than 20 minutes and does not coincide with objective symptoms. The duration of morning stiffness in systemic rheumatic diseases is directly dependent on the severity of inflammatory reactions. For example, one of the important criteria for remission of rheumatoid arthritis is the complete disappearance of morning stiffness.

With the exception of gout, synovitis is a manifestation of systemic diseases, and patients show signs of a generalized process. Osteoporosis, on the contrary, is formed due to local mechanical influences and, of course, is not accompanied by systemicity.

Osteoarthritis affects almost exclusively weight-bearing joints - hip, knee, first metatarsophalangeal. Polyarthrosis, as a rule, is familial and is caused by a genetic inferiority of the cartilage and ligamentous apparatus. The appearance of defigurations in the elbows, metacarpophalangeal, radiocarpal joints should be considered as a manifestation of inflammatory reactions.

As soon as degenerative joint disease begins to manifest itself clinically, its typical signs can be detected on the radiograph. At the same time, the initial stages of synovitis are X-ray negative. Usuration of the bones is visible only with a far advanced process. Differential diagnostic characteristics of the articular syndrome in osteoarthritis and systemic synovitis are presented in the table.

With many systemic diseases, the diagnosis becomes obvious only after a few months, when a classic symptom complex is formed. In the early stages there are always significant diagnostic difficulties. However, there are certain characteristic variations of openings:

- acute monoarthritis,
- migratory arthritis,
- intermittent arthritis,
- spreading arthritis.

Acute monoarthritis most often occurs with septic lesions and synovitis, with microcrystalline arthritis. Both diagnoses are verified quite easily with the help of a diagnostic puncture by a cultural or crystallological analysis of the synovial fluid.

The term "migratory" arthritis is used in cases where inflammation in the initially affected joint subsides completely and the process resumes in the following. This variant is quite rare and is characteristic of rheumatism and gonococcal arthritis.

Intermittent flare-ups of arthritis after a long period of remission occur in gout, spondylitis, psoriatic arthritis, and arthritis associated with intestinal infection.

Spreading arthritis is the most nonspecific: in this case, with persistent inflammation in the initially affected joint, all new joints are involved in the process.

When making a diagnosis, it is very important to take into account family history data, for example, information about the presence of Heberden's nodes, gout, spondylitis, SLE, hemochromatosis in the family.

Physical examination of the joints should take into account three parameters: soreness (sensitivity), swelling, mobility. Synovitis is characterized by pain (sensitivity) throughout the joint. If the pain is localized only in a certain area (point) of the joint, one should think of a local, local cause of its occurrence, such as bursitis, tendovaginitis, or a fracture. Bone crepitus and osteophyte formation is a cardinal feature of degenerative joint disease. Whereas effusion and synovial thickening are typical of synovitis. It is important to remember that soft tissue swelling is not detectable on physical examination of the axial joints and is rarely found in proximal joints such as the shoulder or hip. In addition to the protocol for studying the range of motion, it can be noted that whether there is a significant difference between passive and active volume of movement. This difference indicates that the lesion is due to muscle weakness, tendon rupture, or neurological disease, but not to bone block.

Analysis of the involvement of a specific joint in the process can be very important, since some joints are never affected in certain diseases and, conversely, for many nosologies there are typical localizations.

The temporomandibular joint, for example, is often involved in rheumatoid arthritis but is never affected in gout. The cervical spine is often affected in RA, spondylitis, and osteoarthritis, but never in gonococcal arthritis or gout. The joints of the larynx are affected in one third of all cases of rheumatoid arthritis and extremely rarely in other types of inflammatory joint lesions. The characteristic symptoms of inflammation of the joints of the larynx are sore throat, localized in the larynx and accompanied by a change in voice. Both signs can only be expressed for a few hours in the morning. Synovitis usually develops in unweighted joints of the upper extremities, while osteoporosis is not seen in the elbow, metacarpal, or wrist joints. Spondylitis is usually progresses from the sacroiliac joint up the spine, the localization of the lesion of which may be different. Rheumatoid arthritis, on the other hand, affects only the cervical region and does not cause back pain.

It has been observed that some joints are never affected in the onset of rheumatoid arthritis. These are the so-called articular exceptions - distal interphalangeal, metacarpophalangeal joint of the thumb, proximal interphalangeal fifth finger.

The study of the olecranon area is often very fruitful in the evaluation of rheumatic diseases, since rheumatoid nodules, gouty tophi or psoriatic plaques are most often localized here. Rheumatoid nodules are often also found in the iliac region, on the ears, along the spine, and may be indistinguishable from tofi on physical examination. However, rheumatoid nodules can be seen early in the disease, are very characteristic of the initial outbreak, and tend to decrease in size over time. Tophi, on the other hand, often occur earlier than several years after the patient has been clinically diagnosed. Sometimes a specific diagnosis requires a biopsy of the nodule or aspiration of the contents of tofus to identify the crystals. Gout is clearly diagnosed by the detection of uric acid crystals in the synovial fluid aspirated from the inflamed joint. Serum uric acid levels can only indicate a predisposition to gout.

When performing an X-ray examination, it should be remembered that:

1) osteoporosis is nonspecific and often results from immobility associated with pain;

2) narrowing of the joint space indicates the loss of cartilage;

3) new bone growths indicate osteosclerosis, are a sign of osteophytes and the absence of synovitis;

4) soft tissue edema is best diagnosed by physical examination.

It is important to remember that x-rays show bone, not cartilage or synovium, and because cartilage takes time to break down, x-rays usually lag behind the clinical picture by several weeks. More specific information appears after three to four years, when there is erosion (usuration) of the articular cartilage by granulation connective tissue - pannus.

The most informative laboratory test for rheumatoid arthritis is a latex test aimed at detecting rheumatoid factor.

What is the diagnostic value of the latex test?

Approximately 5% of healthy young people and 15% of the elderly have a positive latex test. Since rheumatoid arthritis affects approximately 1% of the population, it can be stated that only 15-20% of seropositive people suffer from rheumatoid arthritis. Only 85% of diagnosed patients are seropositive. Thus, it is obvious that the latex test does not confirm or exclude the presence of the disease. The latex test is also often positive in other systemic connective tissue diseases, as well as in some chronic inflammations such as tuberculosis, gout, and bacterial endocarditis.

Once detected, a positive titer of rheumatoid factor persists throughout the course of the disease. Therefore, there is no particular need for repeat studies in patients with overt rheumatoid arthritis. However, serological tests are usually negative in the initial stages and become positive as the disease progresses.

This reaction is of prognostic value. In general, in seropositive patients, the process proceeds unfavorably, often subcutaneous rheumatoid nodules are involved. It should be noted that the detection of rheumatoid factor increases with age. In general, the classic onset of the disease, manifested by a typical symptom complex, a high titer of rheumatoid factor, is characteristic of patients aged 55-65. In this group of patients, the process progresses rapidly, erosion of the articular surfaces develops early.

Negative results of the study of rheumatoid factor during long-term observation of a seriously ill patient force us to look for another disease that occurs with rheumatoid-like articular syndrome.

GENERAL PRACTITIONER TACTICS

Diagnosis in rheumatology, as in any other clinical discipline, is based on the analysis of the entire complex of clinical, laboratory and instrumental data. However, often a family doctor does not have a laboratory equipped with modern methods and the possibility of conducting the necessary X-ray examinations in the arsenal of a family doctor. Therefore, it is the primary stage of the diagnostic search

that becomes so important. And the first step in the correct interpretation of the articular syndrome is a thorough questioning and examination of the patient. Here you need the ability to "actively" identify complaints and symptoms, group them into syndromes, form a subsequent program of laboratory and instrumental examination methods. The general practitioner should remember that certain nosological forms have certain features of the articular syndrome. So,

- *Gender, age, profession, lifestyle of the patient.*
- *Daily dynamics of pain syndrome.*
- *Prevalence (monoarthritis, polyarthritis) and localization (large and / or small), symmetry of joint damage.*
- *Previous conditions (past infection, trauma, medication, gastronomic excesses, the presence of uveitis, iridocyclitis).*
- *General condition of the skin, nails (signs of psoriasis), auricles (the presence of tophi), signs of impaired metabolism, hypothyroidism, menopausal syndrome, etc.*

GENDER, AGE, PROFESSION, LIFESTYLE OF THE PATIENT

According to many authors, the following features are distinguished among the main gender differences. Males, according to statistics, are more susceptible to gout (the ratio of men and women according to different authors is 2-7:1), reactive arthritis associated with urogenital infection. Peripheral ankylosing spondylitis is three times more common in men than women. For women, the occurrence of rheumatoid arthritis, arthritis in systemic diseases of the connective tissue, polyosteoarthrosis, dyshormonal arthropathy is more common. Psoriatic arthritis often debuts at age

from 20 to 50 years, and men and women get sick equally often. With the development of arthritis at a young age, one should first of all pay attention to the presence of a history of infections (tonsilogenic, urogenital). At the same time, the peak incidence of rheumatoid arthritis occurs in women aged 40-50 years. Women suffer from rheumatoid arthritis much more often than men (according to generalized statistics - 3-5 times). When referring to a doctor in elderly people with articular syndrome, it is first necessary to exclude degenerative joint damage - polyosteoarthritis, the frequency of which increases sharply with age. Thus, radiographic manifestations of polyosteoarthrosis occur in 100% of people over 75 years of age, while clinical manifestations of the disease may be absent. It is necessary to remember such a disease that has become more frequent in recent years, like polymyalgia rheumatica. This pathology also affects people only in the second half of life, it is characterized by severe pain of stereotyped localization (neck, shoulder and pelvic girdle), movement disorders, a significant increase in laboratory parameters of inflammation, as well as the onset of remission when corticosteroids are prescribed in small doses. Profession and lifestyle can indirectly influence the predisposition to various types of as well as the onset of remission with the appointment of corticosteroids in small doses. Profession and lifestyle can indirectly influence the predisposition to various types of as well as the onset of remission with

the appointment of corticosteroids in small doses. Profession and lifestyle can indirectly influence the predisposition to various types of

joint diseases. According to some authors, wheeled transport drivers are more susceptible to ankylosing spondylitis, and rheumatoid arthritis is more common in people engaged in physical labor, often subject to hypothermia and other adverse environmental factors. Physical inactivity, overweight, gastronomic excesses leading to metabolic disorders can be combined with gout and osteoarthritis. In patients with gout, the frequency of the metabolic syndrome reaches 70%, and the degree of hyperuricemia increases due to an increase in the severity of its individual signs, for example, excess weight. In patients with gout without metabolic syndrome, the level of hyperuricemia is significantly lower than in those with it. Obesity may also be an independent risk factor for the early development and rapid progression of osteoarthritis.

DAILY DYNAMICS OF PAIN SYNDROME

An important step in the differential diagnosis of articular syndrome is the analysis of the dependence of pain intensity on the time of day and physical activity. And here it is necessary to take into account which (“inflammatory” or “degenerative”, mechanical) character of pain prevails in the patient. Most joint diseases can be divided into two groups: inflammatory (arthritis) and non-inflammatory (arthrosis). In arthritis, inflammation is most pronounced in the most vascularized part of the joint - the synovial membrane, in the absence of treatment, the process passes to the cartilage. With arthrosis, degenerative changes begin with cartilage, and the synovial membrane is involved in the pathological process a second time in the form of reactive synovitis. The main difference between "mechanical" and "inflammatory" pain is their change in relation to periods of rest and load of the joints. The "mechanical" pains characteristic of arthrosis appear or increase after exercise and decrease or disappear after periods of rest, while the "inflammatory" pains characteristic of arthritis have an opposite rhythm. Examples of such arthritis are reactive, rheumatoid, gouty and psoriatic arthritis. Arthritis pains are more disturbing for patients in the morning, after a night's sleep, they decrease or disappear after a warm-up, in the evening. However, it is necessary to focus on the difference between morning stiffness, characteristic of arthritis, and the so-called “starting” pains, that is, short-term pains at the beginning of the movement of a patient with arthrosis, at rest, regardless of the time of day. Morning stiffness is a subjective but very valuable diagnostic symptom common to all arthritis, especially rheumatoid arthritis. However, it should be noted that not all patients correctly understand what morning stiffness is. Some patients take for it the limitation of joint mobility, the “starting” pains of the first movements in osteoarthritis. Because the role of the doctor in the active identification of complaints of the patient is great. Clarifying questions help to identify this symptom: “In the morning after waking up, the movements in the joints are more limited and difficult than in the afternoon or in the evening?”; “How long after waking up do you feel pain and difficulty moving”; “In the morning you feel especially bad, there is a feeling of stiff, filled hands, can you clench your fingers into a fist after waking up?”. Patients often quite

characteristically describe their condition: "In the morning with great difficulty and only after taking the medicine / or warming up, the fingers become sensitive and clenching into a fist becomes possible." The duration of morning stiffness in rheumatoid arthritis is usually more than 30 minutes, and temporary gradations of morning stiffness in combination with other symptoms can be used to determine the degree of activity of rheumatoid arthritis: up to 1 hour - I degree, before noon - II degree, after 12 noon - III degree of activity.

PREVALENCE (MONOARTHRITIS, POLYARTHRITIS), LOCALIZATION (LARGE AND/OR SMALL), SYMMETRICITY OF JOINT LESIONS

The next step, subject to the assumption of arthritis, is the analysis of the localization of the lesion. It must be remembered that the features of clinical manifestations depend on the stage of the disease: if initial changes are detected, the correct diagnosis can be difficult, dynamic monitoring is necessary. When deploying the clinical picture, the manifestations of the disease are the most typical, with a far advanced process, the diagnosis can be made according to individual typical signs.

If the patient has an axial lesion of all three joints of one finger with a characteristic “sausage-like” change in the shape of the joint due to confluent tissue swelling or damage to the distal interphalangeal joints, then psoriatic arthritis should be suspected.

Psoriatic arthritis is a severe erosive arthritis with a chronic progressive course, often involving the spine or sacral joints in the pathological process. Diagnosis is not difficult if the patient has psoriatic skin plaques, there are nail lesions in the form of a “thimble”, as well as transverse and longitudinal striation of clouded nail plates. Quite often, psoriatic plaques can be located in “hidden” places for the patient (scalp, gluteal folds, umbilical ring). Therefore, suspecting psoriatic arthritis due to the peculiarities of the articular syndrome, the doctor should actively ask the patient about skin rashes (especially with peeling), the presence of psoriasis in close relatives and motivate himself to a thorough subsequent examination of possible plaque localization sites.

Even with symmetrical damage to the joints (unlike rheumatoid arthritis), their deformation is disordered: the axes of the joints are chaotically directed in different directions. With such an arrangement of the affected joints and the absence of visible plaques, they should be actively sought, especially on the scalp. Immunological examination usually does not detect rheumatoid factor.

The initial manifestations of the disease in the form of monoarthritis are often observed after an injury. Anamnestic data help to quickly resolve the issue of the nosological affiliation of arthritis, and magnetic resonance imaging of the joint - to clarify the integrity of intra-articular formations, ligaments, menisci, tendons.

Identification of acute monoarthritis, especially large joints, requires differential diagnosis with infectious arthritis of a specific nature (tuberculous, gonococcal). It is necessary to purposefully conduct a survey with the involvement of specialists of a narrow profile (phthisiatrician, urologist and venereologist).

With an asymmetric lesion of large and medium joints of the lower extremities and small joints of the feet, one of the seronegative arthritis can be suspected. Especially if there are concomitant "inflammatory" pains in the lumbosacral or thoracic spine.

In the case of a symmetrical lesion of the proximal (as opposed to distal in psoriasis) interphalangeal, metacarpophalangeal and metatarsophalangeal joints, the doctor may suggest that the patient has rheumatoid arthritis.

Most often it is necessary to resolve the issue of the etiology of acute arthritis in men. Complaints such as a pronounced intensity of pain and especially the sudden appearance of pain in the first metatarsophalangeal joint among full health, often at night, makes it logical to assume that the patient has gouty arthritis. In our clinical experience, most errors in the diagnosis of gout occur when the patient

applied for an appointment with arthritis of the joints of another localization (ankle, knee, joints of the upper extremities). The anamnesis data helps in making a diagnosis if the patient remembers that such arthritis was previously, but quickly (within 3-10 days) ended in complete recovery.

PREVIOUS STATUS

When questioning the patient, it is necessary to pay attention to the factors that precede the development of arthritis, since some of them may

cause this disease on its own.

With the development of arthritis in young people, the presence of a tonsilogenic infection should be excluded (after suffering a sore throat, scarlet fever, streptococcal pharyngitis). A preliminary diagnosis of acute rheumatic fever is possible in adolescents and young people with a migratory nature of joint damage, often large, passing spontaneously or with good effect when taking non-steroidal anti-inflammatory drugs. The articular syndrome is combined with a symptom complex of cardiac lesions (variable murmurs, symptoms of valvular heart disease). Disappearance of arthritis without residual changes is characteristic. The diagnosis is confirmed by an increase and subsequent dynamics of the level of streptococcal antibodies.

The risk of rheumatoid arthritis increases in the first 3 months after childbirth or abortion, which is explained by an increase in prolactin levels during this period. Prolactin binds to T-lymphocyte receptors and activates cells that play a critical role in the development of rheumatoid arthritis.

Preceding the development of an attack of gout can be excessive nutrition, especially when eating game, meat and fatty foods, alcohol intake, as well as long-term use of diuretics and antihypertensive drugs combined with diuretics. An attack of gout can be triggered by surgery, long walking, wearing tight shoes.

If a patient with articular syndrome has eye pathology in the form of uveitis, iridocyclitis, conjunctivitis; inflammatory process in the genitourinary tract, enterocolitis, it is necessary first of all to exclude ankylosing or reactive spondyloarthritis. It is also necessary to clarify with the patient the presence of contact with patients with tuberculosis, the possible consumption of milk and dairy products from cows with brucellosis. When identifying these signs, the doctor

should be aware of the possibility of the patient developing one of the seronegative spondyloarthritis (reactive arthritis, spondyloarthritis in Crohn's disease and ulcerative colitis).

GENERAL CONDITION OF SKIN, NAILS, SIGNS OF IMPAIRED METABOLISM

Of course, an important point in the differential diagnosis of articular syndrome is a thorough general examination and physical examination of the patient, including an assessment of not only the condition of the joints, but also the condition of the skin, nails, hairline, mucous membranes, auscultation of the heart and lungs, palpation of the liver and spleen.

Mucosal lesions (conjunctivitis, urethritis, balanitis annulare, painless erosions in the oral cavity) in combination with keratoderma of the soles of the feet and hands and enthesis lesions in the heels (sites of attachment of tendons and ligaments to bones near the joints) are diagnostic criteria for reactive arthritis associated with urogenital or intestinal infection.

It should be remembered the difficulties of diagnosing chronic gout, especially the form that occurs without an increase in the level of uric acid. In a number of patients, gout can occur without hyperuricemia, but at the same time, the detection of hyperuricemia makes the diagnosis of gout eligible without a characteristic syndrome of joint damage. The main diagnostic sign of chronic gout is tophi. Tophi are accumulations of uric acid salts in the form of formations of a solid consistency ranging in size from wheat grain to walnut.

The characteristic localization of tophi is the ear shell, the elbow area, fingers at the level of the distal phalanges. However, tophi can have a wide variety of localizations, for example, in our clinical practice, they were found in the region of the anterior abdominal wall.

Often the cause of articular syndrome are diseases of extra-articular soft tissues (skeletal muscles, tendons, ligaments, fascia, aponeuroses, synovial bags and entheses) due to metabolic and endocrine diseases. The importance of diagnosing these diseases is due to their high prevalence. In such cases, a thorough examination is necessary with the involvement of related specialists to exclude diabetes mellitus, thyroid pathology, hormonal disorders of the menopause and other diseases that can cause pain in the joints. The main feature of this kind of arthralgia is the absence of laboratory and radiological signs of joint pathology. Treatment of this category of patients is primarily aimed at treating the underlying disease.

It should be noted that there are no universal schemes for the differential diagnosis of articular syndrome, however, the individual treatment scheme for the patient will depend on the timely correct interpretation of the articular syndrome. In the case of gouty arthritis, this is a correction of metabolic disorders and taking antihyperuricemic therapy, with reactive arthritis - etiotropic antibiotic therapy, rheumatoid arthritis - therapy with basic anti-inflammatory drugs, osteoarthritis - drugs containing chondroitin sulfate and glucosamine sulfate. And the earlier the treatment regimen is selected for the patient and the transition from symptomatic treatment (pain relief with non-steroidal anti-inflammatory drugs) to etiotropic and

pathogenetic therapy is carried out, the more likely it is to avoid permanent disability, improve the patient's quality of life. It will also prevent the risk of such formidable complications as kidney damage in gout, the development of visceritis in rheumatoid arthritis, damage to the valvular apparatus of the heart in other types of ankylosing seronegative spondyloarthritis. careful collection of anamnesis, complaints, targeted physical examination are of particular importance, which is difficult to overestimate. And the correct interpretation of the data obtained at the initial stage of the diagnostic search will subsequently allow you to adequately select diagnostic and treatment schemes for each specific patient, to achieve success in ongoing therapy.

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LECTURE TOPIC: "SYNDROME OF HEPATOMEGALY AND JAUNDICE. DIFFERENTIAL DIAGNOSIS IN JAUNDICE AND HEPATOMEGALY . PREVENTION."

TRAINING TECHNOLOGY

<table>
<tr><td>Number of students-</td><td>Time - 2 hours</td></tr>
<tr><td>Form of the lesson</td><td>Lecture-Visualization</td></tr>
<tr><td>Lecture plan</td><td>6. Topographic anatomy, blood supply to the liver
7. Types of jaundice, the main diseases accompanied by jaundice
8. Differential diagnosis of jaundice
9. The concept, definition of hepatomegaly
10. The most common diseases accompanied by hepatomegaly
11. Differential diagnosis of diseases accompanied by hepatomegaly
12. Principles of treatment, prevention and patients with hepatomegaly</td></tr>
<tr><td colspan="2">The purpose of the lesson: to familiarize students with the mechanism of occurrence of hepatomegaly and jaundice in various diseases, to teach to differentiate various diseases accompanied by hepatomegaly and jaundice, to teach the tactics of GP in diseases accompanied by hepatomegaly and jaundice</td></tr>
<tr><td>Pedagogical tasks:
1. Strengthen and deepen students' knowledge of diseases accompanied by jaundice
2. Strengthen and deepen students' knowledge of diseases accompanied by hepatomegaly
3. To teach students the ability to differentiate diseases accompanied by hepatomegaly and jaundice
4. To acquaint students with the peculiarities of the course of diseases accompanied by jaundice and hepatomegaly.
5. To teach students the management of patients with</td><td>The results of the educational process:
The GP needs to know:
1. Types and pathogenesis of the development of jaundice
2. Diseases accompanied by hepatomegaly and jaundice
3. Principles of differential diagnosis of diseases accompanied by hepatomegaly and jaundice
4. Tactics of management, principles of treatment of patients with jaundice and hepatomegaly
5. Principles of prevention and medical examination of patients with hepatomegaly and jaundice</td></tr>
</table>

hepatomegaly and jaundice, the tactics of GP.	
Teaching methodology	Lecture text, videos, questionnaires, questions, “yes-no” technique
Form of study	Laser projector, visual materials, special technical equipment, presentation of thematic patients
Means of education	Team
Conditions for the educational process	Audience

TECHNOLOGICAL CARD LECTURES

Stages, time	Activity	
	Teacher	students
Stage 1 Introduction (5 minutes)	1. Tells about the topic of the lecture, its purpose and plan	1. Listen
Stage 2 Knowledge update (20 minutes)	2.1. In order to increase the actualization of students' knowledge, asks questions: 1. Define the terms jaundice, hepatomegaly? Types of jaundice 2. List the diseases accompanied by jaundice and hepatomegaly 3. Diagnosis of patients with hepatomegaly and jaundice 4. List the groups of drugs used to treat hepatitis, liver cirrhosis Conducts a survey 2.2. Showing on the screen invites students to familiarize themselves with the goals and objectives of the lecture. Slide #1, #2	2.1. Answer questions asked 2.2. Study slide number 1 2.3. Study slide number 2
Stage 3 Main part (informational) (55 min)	3.1. Introduces students to the lecture material, the importance of the topic and the principles of the formation of an intelligent cultural personality, in particular the GP. In order to increase the actualization of knowledge, he conducts a quick survey of students:	3.1. Together they analyze the listened lecture material, ask questions

	1. According to 1 point of the lecture plan: types and pathogenesis of the development of jaundice 2. According to the 2nd point of the lecture plan: diseases accompanied by hepatomegaly and jaundice 3. According to the 3rd point of the lecture plan: the principles of differential diagnosis of diseases accompanied by hepatomegaly and jaundice 4. According to the 4th point of the lecture plan: tactics of management, principles of treatment of patients with jaundice and hepatomegaly 5. According to the 5th point of the lecture plan: the principles of prevention and medical examination of patients with hepatomegaly and jaundice Stopping at the important points of the lecture, he suggests writing down the main points in a notebook	Key points are written in a notebook.
<u>Stage 4</u> Final (10 min)	4.1. Asking questions: 1. List the most common diseases accompanied by hepatomegaly and jaundice 2. Tell us the main key mechanisms for the development of various types of jaundice 3. Tell the main key mechanisms for the development of hepatomegaly in various diseases 4. Name the basic principles of treatment, prevention and rehabilitation of patients with jaundice and hepatomegaly 4.2. Gives a task for independent work of students:	4.1. Answer questions 4.2. Listen, write

	Diseases of liver accumulation (hemochromatosis, Wilson-Konovalov's disease) clinical course, treatment.	

There are several types of jaundice. Hemolytic (suprahepatic)-hexcessive destruction of red blood cells and increased production of bilirubin
Hepatic jaundice is a violation of the capture of bilirubin by liver cells and its binding to glucuronic acid. Subhepatic - the presence of an obstacle to the release of bilirubin with bile into the intestine and the reabsorption of bound bilirubin into the blood.
Hereditary microspherocytosis - a hereditary disease accompanied by extravascular hemolysis - in the cells of the reticuloendothelial system Anemia is moderate, but during crises the hemoglobin content is 20-25 g / l. Hereditary stomacytosis is characterized by a defect in erythrocyte membranes. 50 g / l during it), jaundice due to unbound bilirubin.The most common chronic liver disease is chronic hepatitis. It is characterized by an enlarged liver, pain or a feeling of heaviness in the right hypochondrium. In 95% of patients, liver enlargement is noted. Jaundice and pruritus are less common, but symptoms such as loss of appetite, nausea, and poor fat tolerance are common. As a rule, patients with chronic hepatitis can themselves distinguish such signs of the disease as flatulence, unstable stools, they feel weakness and decreased performance. Moderate yellowing of the skin and mucous membranes is also an external sign of a violation of the liver in case of chronic hepatitis.
Specialists also distinguish between inactive and active, benign, aggressive, progressive recurrent hepatitis. Diagnosis of liver diseases, in which laparoscopy or liver puncture is performed, can accurately distinguish between forms of hepatitis. Often "neglected" hepatitis leads to the development of cirrhosis of the liver. About 2 million people die from this dangerous disease every year in the world. Meanwhile, a person with liver disease always has a chance to prevent the development of serious complications.
Etiology and pathogenesis of obstructive jaundice. Currently, the causes of obstructive jaundice have been studied quite well. According to the etiological principle, they can be combined into five main groups: - congenital malformations of the bile ducts; - benign diseases of the biliary tract and pancreas; pudendal gland, which are etiologically associated with cholelithiasis (biliary duct stones, cicatricial structures of the ducts, stenosis of the major duodenal papilla (BDS), indurated pancreatitis);

- structures of the main bile ducts, developingas a result of an operating injury; - primary and secondary (metastatic) tumors of the organs of the hepatobiliary zone; - parasitic diseases of the liver and bile ducts.
A large group of diseases of the biliary system and pancreas is accompanied by the development of mechanical obstruction of the bile ducts, manifested by the appearance of icteric coloration of the skin and sclera in the patient, which erroneously led to the combination of all these diseases into one that entered the

clinic.
jaundice. It has now been established that jaundice is only a symptom of the disease, although long-term mechanical obstruction of the biliary tract leads, as a rule, to the fact that jaundice becomes jaundice-disease. The reason for this is the changes in the organs that develop as a result of cholestasis. Recently, the number of patients suffering from diseases of the main bile ducts has significantly increased. those and pancreas. The earlier the nature of patological process and a rational allowance for the restoration of bile outflow from the liver, the better the result and treatment of the patient. When collecting patient complaints, attention should be paid to the general signs of the disease. In addition to icteric staining of the skin and visible mucous membranes, the most common clinical symptom is pain, especially with subhepatic cholelithiasis. The nature of pain and their intensity largely depends on the disease that caused obstructive jaundice. - vomiting. Weight loss is more common with subhepatic jaundice caused by malignant neoplasms. Often there is an increase in body temperature. In most cases, it is associated with an infection of the biliary tract, less often with tumor decay. Prolonged subfebrile temperature is a differential diagnostic sign of obstructive jaundice, in contrast to viral hepatitis, An enlarged liver is a common symptom of prolonged obstructive jaundice. It occurs due to the overflow of the liver with congestive bile and, to some extent, the inflammatory process around the bile ducts. Enlargement of the gallbladder is a characteristic sign of tumors of the Vater nipple, the head of the pancreas and the terminal part of the common bile duct. With a thick abdominal wall, it is not always possible to palpate an enlarged gallbladder; in this case, laparoscopy or ultrasound helps. A characteristic sign of cholestasis is itching, which sometimes occurs even before the onset of jaundice. Persistent, debilitating itching, poorly amenable to therapy, is especially characteristic of obstructive jaundice of tumor genesis. The duration of jaundice is very different: from a few days with short-term blockage of the extrahepatic biliary tract, for example, with a calculus, to many months, with alveoccosis and tumors.

With gallstone diseasejaundice, as a rule, is preceded by attacks of pain in the right hypochondrium, accompanied by dyspeptic symptoms, primarily nausea and vomiting. Often during an attack, body temperature rises. Jaundice usually occurs after another attack, and if the stone does not pass into the duodenum, the attacks often recur.

Cancer of the head of the pancreas. Characteristic of this disease is the presence of complaints even before the appearance of jaundice (loss of appetite, weight loss, abdominal pain, itching of the skin). Jaundice occurs, as a rule, without a pronounced pain syndrome and progressively increases. Patients are concerned about dull pain in the epigastrium and right hypochondrium, often with irradiation to the back. Enlargement of the liver is observed in 75% of patients, Courvoisier's symptom is determined in 60-65% of patients, and with the help of laparoscopy, an increase in the gallbladder is noted in 100% sick. **gallbladder cancer**. It occurs more often in women, since the factor contributing to its occurrence is cholelithiasis. Before jaundice in patients, in most cases, there are

certain complaints, primarily due to the presence of gallstones in the bladder: attacks of acute pain in the right hypochondrium with an increase in body temperature. The latter are often associated with a violation of the diet (especially the intake of large amounts of fat). Later, weight loss is observed. With cancer of the gallbladder, jaundice progresses and, once started, is not eliminated without surgical treatment. Cancer of the extrahepatic bile ducts. It happens somewhat more often in women (55%). When the tumor is localized only in the right or left hepatic duct, the "non-draining lobe syndrome" develops. At the same time, signs of cholestasis are detected in the laboratory, often patients complain of itching, at the same time, jaundice, With the localization of the tumor in the common hepatic duct with the indicated signs of cholestasis, jaundice occurs, which steadily progresses. The gallbladder with such localization of the tumor is always collapsed, empty, which is well detected during laparoscopy.
Cancer of the major duodenal nipple. It is rare, as is gallbladder cancer. The disease quickly causes obstruction of the common bile duct and is manifested by severe jaundice and other signs of cholestasis. Sometimes, as a result of the collapse of the tumor or a decrease in edema or inflammation, jaundice decreases, then the base appears. When disintegration of the tumor often occurs intestinal bleeding. Due to the early manifestation of signs of the disease, surgical treatment is usually successful.

Intrahepatic jaundice of mechanical origin. Its most common causes are alveococcosis and primary or metastatic liver cancer. In alveococcosis, jaundice is preceded by a long period of growth, often over many years. alveococcal node. Only very rarely, when the node is located near the gates of the liver, jaundice can be the first sign of the disease. The usual manifestation of alveococcosis is dull pain in the right hypochondrium and a feeling of a foreign body, especially when the torso is tilted.) before the onset of jaundice. _Objective examination of the patient is carried out according to generally accepted rules. First of all, the general condition of the patient is assessed, the development of the subcutaneous fat layer, the color of the skin, etc.
A detailed examination of the abdomen is carried out: examination, percussion, palpation. If a formation in the abdominal cavity is palpated, a number of signs are revealed: size, surface nature, consistency, mobility, localization, relation to surrounding tissues. The data of the examination results should be given in full, for which questions are asked to the attending physician, and, if necessary, the conclusions of specialists (therapist, neuropathologist, infectious disease specialist, dermatologist, etc.) taken from the patient's history are given. In some cases, the preliminary diagnosis of obstructive jaundice is not difficult. This happens with advanced cancers of the biliopancreatic zone, when the tumor is palpated, distant metastases are determined, or Courvoisier's syndrome.

The position of the doctor is more difficult with the initial manifestations of cholestasis.

Laboratory methods. The vast majority of laboratory methods are of little use for the early diagnosis of mechanical and intrahepatic non-mechanical

cholestasis. In both cases, the main signs of cholestasis - an increase in the amount of cholesterol, conjugated bilirubin bile acids, alkaline phosphatase activity, leucine aminopeptidase, the appearance of pathological lipoprotein X in the blood - will take place. The most difficult is the early laboratory diagnosis of cholestasis. tic forms of viral hepatitis and mechanical cholestasis, since in the early stages of obstructive jaundice, especially with its rapid increase, there may be cytolysis phenomena, and indicator enzymes will be increased almost to the same extent as in viral hepatitis. Of course, modern methods for diagnosing liver diseases almost always make it possible to put diagnosis is correct. However, most of these methods are not always available to a practical doctor, some of them are invasive, and therefore the search for simple methods for diagnosing different types of cholestasis is justified.

Ultrasound diagnostics. The main component of the diagnosis is the study of the width of the bile ducts. Using this method, in 90% of cases it is possible to identify stones in the gallbladder. With the localization of calculi in the terminal section of the common bile duct, their detection is y x 20-30%, since the head of the pancreas and the 12 duodenum with gas are located there, which makes it difficult to identify stones. The expansion of the bile ducts is not always pronounced, which also makes it difficult to diagnose. Scanning the liver. Radioisotope scanning of the liver is especially advisable in cases where mechanical cholestasis is due to the presence of tumors or an alveococcosis node that compresses the main bile ducts inside the liver. For the diagnosis of subhepatic cholestasis, liver scanning is ineffective. Nevertheless, scanning should always be carried out in doubtful cases, when it is difficult to identify a mechanical obstruction in the extrahepatic biliary tract. Retrograde cholangiopancreatography (RCPG). This method, unlike the three previous ones, is invasive, complications are possible during its implementation, there are cases of lethality. At the same time, it is highly informative, surpassing the method of ultrasound diagnostics. ERCP makes it possible to establish the localization of the obstacle, and often its nature.

Percutaneous transhepatic cholangiography. In terms of information content, this method is not inferior to ERCP, but the number of complications with it is much greater (leakage of bile, bleeding into the abdominal cavity, peritonitis).

Needle biopsy. In subhepatic cholestasis, it is usually contraindicated, since high pressure in the biliary tree, as a rule, leads to the outflow of bile from the puncture hole into the abdominal cavity. Puncture biopsy is of great diagnostic value only when focal pathological formations are visible in the liver, the nature of which is unclear. In these cases, the needle biopsy should be targeted. Needle biopsy is strictly contraindicated in cases of suspected liver echinococcosis. _Differential diagnosis and establishment of the final diagnosis. Differential diagnosis should be carried out taking into account the data obtained. Most often with various forms of obstructive jaundice or hemolytic or parenchymal jaundice. When making a differential diagnosis of this cholestasis with other forms of obstructive jaundice, it should be remembered that the most common cholelithiasis, then pancreatic cancer, gallbladder cancer, extrahepatic bile duct cancer, cancer of the major duodenal

papilla, less often primary liver cancer and alveococcosis. It is necessary to recall the possibility of obstructive jaundice as a result of another disease due to compression of the biliary tract of the pancreas, or metastasis from other organs, preventing normal bile outflow. Further, the final diagnosis is formulated indicating the type of disease, its localization, type of jaundice and possible obstruction for bile outflow. For example, cholelithiasis, chronic calculous cholecystitis, choledocholithiasis, It is necessary to recall the possibility of obstructive jaundice as a result of another disease due to compression of the biliary tract of the pancreas, or metastasis from other organs, preventing normal bile outflow. Further, the final diagnosis is formulated indicating the type of disease, its localization, type of jaundice and possible obstruction for bile outflow. For example, cholelithiasis, chronic calculous cholecystitis, choledocholithiasis, It is necessary to recall the possibility of obstructive jaundice as a result of another disease due to compression of the biliary tract of the pancreas, or metastasis from other organs, preventing normal bile outflow. Further, the final diagnosis is formulated indicating the type of disease, its localization, type of jaundice and possible obstruction for bile outflow. For example, cholelithiasis, chronic calculous cholecystitis, choledocholithiasis,

mechanical jaundice. Further, the diagnosis of the patient is formed depending on all the diseases identified in him.

According to literature data, obstructive jaundice occurs more often in people over the age of 40 years. At the same time, the development of both calculous and tumor obstruction of the bile ducts is characteristic of the elderly and senile age, while at a young age the cause of obstructive jaundice is more often cholelithiasis. In the first days of the onset of obstructive jaundice, an increase in the level of aminotransferase activity is observed. This is due to a short-term (3-5 days) increase in the permeability of hepatocyte membranes and the release of indicator enzymes from them. Hyperbilirubinemia in obstructive jaundice is characterized by an increase in the blood of predominantly conjugated bilirubin and, to a lesser extent, unconjugated bilirubin. Since with obstructive jaundice in the first time after the development of obstruction of the bile ducts, the liver function is slightly impaired, the excretion of bile by the hepatocyte continues. The bilirubin secreted into the lumen of the bile ducts along with bile is absorbed through their wall and enters the spaces of Disse through communications in the bile capillaries. From the space of Disse, bilirubin enters the blood through the lymphatic system. A longer increase in pressure in the bile ducts leads to dysfunction of the liver cells, and paracholia occurs (the return of bilirubin from the hepatocyte to the blood). As hepatocyte function deteriorates, bilirubin uptake also suffers. and in the blood, the amount of unconjugated bilirubin increases. During this period, necrosis of hepatocytes can be observed, due to which the activity of aminotransferases increases again in the blood. Very rare causes of obstructive jaundice include a complicated duodenal ulcer, as well as an acute inflammatory process in the vermiform appendix, if it is located in the zone of the gate of the liver. The mechanical factor of blockage of the intrahepatic bile ducts can also occur in Botkin's disease, when the intralobular bile ducts are clogged with bile clots. Gallstone disease and mechanical obstruction of the hepatobiliary duct:

Cholestasis occurs most often due to the migration of stones from the gallbladder into the ducts. Stones formed in the ducts themselves are much less common (they are frequent in the countries of the East). During this period, necrosis of hepatocytes can be observed, due to which the activity of aminotransferases increases again in the blood. Very rare causes of obstructive jaundice include a complicated duodenal ulcer, as well as an acute inflammatory process in the vermiform appendix, if it is located in the zone of the gate of the liver. The mechanical factor of blockage of the intrahepatic bile ducts can also occur in Botkin's disease, when the intralobular bile ducts are clogged with bile clots. Gallstone disease and mechanical obstruction of the hepatobiliary duct: Cholestasis occurs most often due to the migration of stones from the gallbladder into the ducts. Stones formed in the ducts themselves are much less common (they are frequent in the countries of the East). During this period, necrosis of hepatocytes can be observed, due to which the activity of aminotransferases increases again in the blood. Very rare causes of obstructive jaundice include a complicated duodenal ulcer, as well as an acute inflammatory process in the appendix, if it is located in the zone of the liver gate. The mechanical factor of blockage of the intrahepatic bile ducts can also occur in Botkin's disease, when the intralobular bile ducts are clogged with bile clots. Gallstone disease and mechanical obstruction of the hepatobiliary duct: Cholestasis occurs most often due to the migration of stones from the gallbladder into the ducts. Stones formed in the ducts themselves are much less common (they are frequent in the countries of the East). Among the very rare causes of obstructive jaundice include a complicated duodenal ulcer, as well as an acute inflammatory process in the appendix, if it is located in the zone of the liver gate. The mechanical factor of blockage of the intrahepatic bile ducts can also occur in Botkin's disease, when the intralobular bile ducts are clogged with bile clots. Gallstone disease and mechanical obstruction of the hepatobiliary duct: Cholestasis occurs most often due to the migration of stones from the gallbladder into the ducts. Stones formed in the ducts themselves are much less common (they are frequent in the countries of the East). Among the very rare causes of obstructive jaundice include a complicated duodenal ulcer, as well as an acute inflammatory process in the appendix, if it is located in the zone of the liver gate. The mechanical factor of blockage of the intrahepatic bile ducts can also occur in Botkin's disease, when the intralobular bile ducts are clogged with bile clots. Gallstone disease and mechanical obstruction of the hepatobiliary duct: Cholestasis occurs most often due to the migration of stones from the gallbladder into the ducts. Stones formed in the ducts themselves are much less common (they are frequent in the countries of the East). The mechanical factor of blockage of the intrahepatic bile ducts can also occur in Botkin's disease, when the intralobular bile ducts are clogged with bile clots. Gallstone disease and mechanical obstruction of the hepatobiliary duct: Cholestasis occurs most often due to the migration of stones from the gallbladder into the ducts. Stones formed in the ducts themselves are much less common (they are frequent in the countries of the East). The mechanical factor of blockage of the intrahepatic bile ducts can also occur in Botkin's disease, when the intralobular bile ducts are clogged with bile clots. Gallstone disease and mechanical

obstruction of the hepatobiliary duct: Cholestasis occurs most often due to the migration of stones from the gallbladder into the ducts. Stones formed in the ducts themselves are much less common (they are frequent in the countries of the East). Obturation is formed due to the fact that a large stone cannot pass into the duodenum; no less frequent is a prolonged spasm of the sphincter of Oddi, as a result of which stones, even small ones, get stuck in the terminal section of the common bile duct for some time. Jaundice with cholestasis caused by cholelithiasis in 65% of cases, unstable. This is due to the fact that small stones after the elimination of sphincter spasm independently pass into the intestine. Large stones in this case can migrate in the proximal direction, remaining in the common bile duct, but allowing the evacuation of bile ("valve" stone).
Cirrhosis of the liveris a chronic progressive disease characterized by degeneration and necrosis (necrosis) of the liver tissue, signs of liver failure and portal hypertension (increased blood pressure in the portal vein of the liver); accompanied by proliferation of connective tissue and a deep violation of the structure and function of the liver.
The causes of liver cirrhosis are: chronic viral hepatitis: B, C, O (40% of all cases), alcohol abuse (50% of all cases), impaired immunity, metabolism, Konovalov-Wilson disease and other hereditary diseases, the effect of hepatotropic drugs and toxic substances, diseases of the biliary tract. The most common cause is adversely occurring alcoholic and viral hepatitis.
Due to inflammation or poisoning, the liver cells die and are replaced by scars and the liver decreases in size. As a result, its functional performance is reduced. In addition, the flow of blood through the portal vein of the liver decreases due to its narrowing, which transports nutrient-rich blood from the gastrointestinal tract to the liver. At the same time, blood pressure rises in the vein, which leads to the accumulation of fluid in the abdominal cavity and varicose veins of the esophagus, from which life-threatening bleeding is possible.
If the liver is no longer able to perform its tasks of detoxification, this leads to brain damage (encephalopathy).
Clinical signs depend on the stage of the disease. In the advanced stage, weight loss, general weakness, jaundice, enlarged spleen, and bleeding from the gastrointestinal tract are noted.
Liver cirrhosis corresponds to the next stage of chronic hepatitis morphogenesis. The main difference between liver cirrhosis and chronic hepatitis is the development of a diffuse inflammatory process with high fibroplastic activity and liver fibrosis. Widespread fibrosis leads to a restructuring of the normal architectonics of the hepatic lobules with the formation of multiple pseudolobules and intrahepatic vascular anastomoses. There is a nodular transformation of the liver parenchyma with the subsequent development of liver failure.
The classification of liver cirrhosis, as well as chronic hepatitis, is based mainly on morphological criteria. Liver cirrhosis is divided into small-nodular (micronodular) with node sizes up to 1-3 mm (less than 10 mm) and large-nodular (macronodular), in which the diameter of the nodes can reach 5 cm. These two morphological variants

of cirrhosis can also be considered as successive stages with the transformation of micronodular into macronodular. A combination of two types of nodes is possible, which corresponds to the development of mixed micromacronodular cirrhosis of the liver.

Cirrhosis of the liver can develop as a result of a variety of chronic diseases and pathological conditions. The main causes of liver cirrhosis are serum hepatitis B, C, D and alcoholic liver damage. The combined effect of these two factors is especially unfavorable. In some patients, the cause of liver cirrhosis remains unknown. However, due to the indication of specific markers of hepatitis viruses, the proportion of cryptogenic cirrhosis has decreased markedly.

Clinical manifestations of liver cirrhosis vary widely depending on the stage of the disease, the activity of the process, the presence of complications. The establishment of an etiological diagnosis, which is necessary for a differentiated therapy program, requires taking into account the entire set of clinical and epidemiological data and the results of laboratory tests.

HBV cirrhosis of the liver.According to its morphological characteristics, it is large-nodular. It is more often detected in men - the ratio of men and women is 3: 1, mainly over the age of 40 years. Very often, cirrhosis of the liver develops in chronic hepatitis B induced by mutant strains.

According to S.N. Sorinson, HBV-cirrhosis of the liver, unlike alcoholic, develops relatively more often in the first 5 years after acute hepatitis B. HBV-cirrhosis, like liver cirrhosis of other origin, is characterized by a long course with different rates of progression, repeated exacerbations arising under the influence of visible causes or spontaneously.

During laboratory examination, patients are diagnosed with anemia, usually of the hypochromic microcytic type, often in combination with a decrease in the number of platelets and leukocytes. These signs characterize the development of hypersplenism, which is understood as the deposition of blood in the spleen with an accelerated breakdown of formed elements. Hypersplenism is often, but not always, associated with marked splenomegaly.

Biliary cirrhosis withassociated with stagnation of bile - cholestasis. It occurs more often in women aged 50-60 years. Relatively benign course. It proceeds with skin itching, which may appear long before jaundice. Cholestasis can be primary and secondary, like cirrhosis: primary - the outcome of cholestatic hepatitis, secondary - the outcome of subhepatic cholestasis. In addition to pruritus, other signs of cholemia are also characteristic: bradycardia, hypotension, and a greenish color of the skin. Signs of hypercholesterolemia: xanthomatosis, steatorrhea. Cholestasis also leads to osteoporosis, an increase in bone fragility. The activity of alkaline phosphatase increases. Bilirubin direct up to 2 mg%. The liver is enlarged, dense. With secondary biliary cirrhosis - a previous or recurrent pain syndrome (usually a stone in the biliary tract). There are also chills, fever, and other signs of inflammation of the bile ducts. Enlarged painless liver, small nodular. The spleen never enlarges.

Adrenal block.Associated with a violation of the outflow of blood in the hepatic vein (thrombosis, congenital narrowing). Portal hypertension develops. Budd-Chiari

Syndrome. Clinical manifestations depend on how rapidly portal hypertension progresses.
Subhepatic block.Associated with damage to the portal vein (thrombosis, compression from the outside). Clinic - severe pain, fever, leukocytosis, rapid liver dysfunction.
Intrahepatic portal block- cirrhosis of the liver. If there is no clear etiological factor, then splenography is used (a contrast agent is injected into the spleen and after 6-7 seconds it is removed from the liver). If biliary cirrhosis occurs, the level of cholestasis (primary or secondary) should be established. Use cholangiography. Cirrhosis of accumulation (hemochromatosis)- pigmentary cirrhosis, bronze diabetes, is associated with a deficiency of an enzyme that binds iron to protein and, thereby, regulates its absorption. With this defect, iron is intensively absorbed in the intestine and, insufficiently binding to protein, begins to be deposited intensively in the liver, skin, pancreas, myocardium, and sometimes in the adrenal glands. In the liver, iron accumulates in RES cells - a large, finely nodular liver is observed. Inactivation of sex hormones is often disturbed. Signs of portal hypertension dominate. The skin has gray-dirty pigmentation, the same on mucous membranes, the skin becomes especially dark if iron is deposited in the adrenal glands - a symptom of adrenal insufficiency. With damage to the pancreas - diabetes mellitus syndrome. With cardiomyopathies, severe irreversible rhythm disturbances. Wilson-Konovalov disease- other accumulation cirrhosis (hepatolenticular degeneration). The basis of the disease is a violation of copper metabolism associated with a birth defect. The normal plasma concentration is 100-120 μg%, with 93% of this amount being in the form of ceruloplasmin and only 7% associated with serum albumin. Copper in ceruloplasmin is tightly bound. This process is carried out in the liver. Ceruloplasmin is an alpha-2 globulin and each molecule contains 8 copper atoms. It has been established by radioisotope methods that in hepatolenticular insufficiency, the disorder is associated with a genetic defect in the synthesis of ceruloplasmin, due to which its content is sharply reduced. In this case, copper cannot be stably bound and is deposited in the tissues. In some cases, the content of ceruloplasmin remains normal, but its structure changes (the ratio of fractions changes). Copper is especially tropic in Wilson-Konovalov syndrome to the liver, brain nuclei, kidneys, endocrine glands, and cornea. In this case, copper begins to act as a toxic agent, causing typical degenerative changes in these organs.
Clinic.It is a set of syndromes characteristic of damage to the liver and extrapyramidal nervous system. Along the course, acute and chronic forms are distinguished. The acute form is characteristic of an early age, develops at lightning speed and ends fatally, despite treatment. The chronic form is more common with a slow course and gradual development of symptoms. First of all, extra pyramidal muscle rigidity of the lower extremities appears (violation of gait and stability). A picture of parkinsonism is gradually formed, then the psyche changes (paranoid reactions, hysteria). Sometimes liver failure comes to the fore: an enlarged liver, the picture resembles cirrhosis or chronic active hepatitis. The differential sign is hypocupremia below 10 μg% or the level of copper is kept at the lower limit of

normal, a lot of copper is excreted in the urine - over 100 mcg / day. There may be a positive thymol test. An important symptom is the Kaiser-Fleischer ring (bluish green or brownish green around the periphery of the cornea due to copper deposition). Treatment is aimed at binding copper and removing it from the body: cuprenil (D-penicillinamine) is used, as well as unitiol + diet with the exception of copper (chocolate, cocoa, peas, liver, rye bread).

Hepatomegaly- pathological enlargement of the liver. Hepatomegaly of the liver is characterized by an increase in its size, is not an independent disease, but only a symptom of disturbances in the functioning of body systems. Liver hepatomegaly can appear not only in diseases occurring in the liver itself, but also in other organs. The functions of the liver are diverse, it neutralizes various toxins of endogenous and exogenous origin. It decomposes them into non-toxic components that are excreted naturally from the body along with urine and feces. Diseases, manifested mainly by an increase in the liver, can be divided into three groups.

Diseases of the liver and its vessels: acute viral hepatitis, chronic hepatitis B, C, D, autoimmune hepatitis, liver cirrhosis (latent form), liver echinococcosis, liver cancer, benign liver tumors, non-parasitic liver cysts, tuberculous granulomatosis, liver tuberculoma, Budd's disease -Chiari.

Storage diseases: fatty liver, hemochromatosis, hepatolenticular degeneration, amyloidosis.

Diseases of the cardiovascular system: constrictive pericarditis, circulatory failure II and III degree (“congestive liver”).

Especially pronounced hepatomegaly is observed in leukemia and other hemoblastoses, as a result of massive infiltration of the liver tissue with malignant leukemia or lymphoblastic cells, or as a result of the formation of foci of extramedullary (extramedullary) hematopoiesis in the liver. In these cases, the liver sometimes reaches an enormous size, occupying more than half of the abdominal cavity and sometimes reaching a weight of about 10-20 kg.

If the size of the liver according to 1. medioclavicularisdextra exceeds 12 cm or the left lobe is palpated in the epigastric region, they speak of an enlarged liver. It is important to exclude hepatic prolapse (for example, in chronic obstructive pulmonary disease or right lung distention) or other tissues in the right upper quadrant (enlarged gallbladder, kidney or bowel tumor). The size of the liver is best determined by CT or ultrasound. It is important to evaluate the contours and pattern of the tissue of the organ; An increase in certain areas of tissue; "stony" consistency suggests the presence of a tumor; pain on palpation indicates inflammation (hepatitis) or a rapid increase in the size of the organ (right heart failure, Budd-Chiarisyndrome disease), fatty infiltration.

Hepatomegaly of the liver can be observed in hepatitis, that is, inflammation occurring in the tissues of the liver itself, in cirrhosis, when normal tissue is replaced due to inflammation with connective fibrous tissue, with the development of tumor formations, with the formation of cysts, with parasitic invasions into the liver. Also, the liver can increase due to the accumulation of products of improper metabolism in it, which most often occurs due to hereditary causes of metabolic disorders. In

cardiovascular diseases, when blood stagnates in the veins, an increase in this organ can also be observed. Hepatomegaly of the liver in each of these cases will proceed differently.

If a person is healthy, then his liver will not protrude from under the costal arch, it will be soft and painless. If there are own liver diseases, such as hepatitis, then its own cells (hepatocytes) will be affected due to the influence of various factors, toxic substances, viruses. Due to the fact that inflammation occurs in the tissues, it will swell, increasing in size, becoming painful and dense. Part of the hepatocytes may be destroyed in this case, due to which the liver may again decrease in size, which indicates the transition of the disease to a severe stage. Cirrhosis of the liver develops, as a rule, after a person has had hepatitis, with chronic intoxication (for example, alcohol). At the same time, part of the hepatocytes also dies, being replaced by connective tissue. Another part of the body begins to increase in size, from which the liver noticeably enlarges, becomes painful, bumpy, hard. Hepatomegaly of the liver in cirrhosis, according to clinical symptoms, resembles hepatomegaly in the presence of tumors, cysts, parasites.

Enlargement of the liver may be one of the signs of the course of a hereditary chronic disease, which is based on a violation of the production of glycogen. This type of disease is called glycogenosis. Hepatomegaly begins due to the fact that with the active production of this substance, it begins to accumulate in the liver, which is why it increases. In addition to the liver, with glycogenosis, the kidneys and spleen evenly increase. Hepatomegaly also appears in the presence of another metabolic disease - hemochromatosis. It is caused by a violation of the enzyme systems that are responsible for the adequate absorption of iron in the body. At the same time, iron is absorbed in the intestines more than necessary, which is why it begins to be deposited in the internal organs, which causes a change in their size. At the same time, cirrhosis develops in the liver, it becomes hard and small-hilly.

In chronic cardiovascular insufficiency, the liver also suffers. In case of insufficiency, blood slowly moves along the systemic circulation, causing stagnation and swelling of organs and tissues, their lack of oxygen saturation. The liver tissue also swells, becomes dense, painful along the edge, hematomegaly appears. As a result of edema and compression, hepatocytes begin to collapse, which causes the replacement of these areas with connective tissue. The liver again becomes bumpy, dense. Liver tissue can be restored in some diseases. But in cases of severe cirrhosis, it is not possible to restore liver tissue.

The diagnostic process can be conditionally divided into three stages. The main task of the study of the first stage is to establish liver damage, if during a dispensary examination or when the patient consults a doctor, they find corresponding complaints or deviations in the objective status (hepatomegaly, jaundice, ascites). At the first stage of diagnosis, a purposefully and carefully collected anamnesis is of great importance. Particular attention should be paid to epidemiological data: the possibility of acute viral hepatitis, previous blood transfusions, surgery, injections, alcohol abuse, indications of tuberculosis, trauma in the heart and liver, hereditary

diseases. It is important to find out if fever, arthralgia preceded the enlargement of the liver, or if there is a dyspeptic syndrome.

Objective research. When examining a patient, the color of the skin and mucous membranes (jaundice of the sclera, skin pigmentation) is assessed, extrahepatic signs (spider veins, “hepatic” palms), gynecomastia, as well as pulsation of the jugular veins and hepatojugular phenomenon are detected. An enlarged liver in some cases is already visible when examining the abdomen as a tumor moving during breathing in the right hypochondrium or in the epigastric region. Prolonged and significant enlargement of the liver can lead to deformation of the chest, while the intercostal spaces are not filled. Percussion of the organ gives very approximate results. Normally, the upper limit of the absolute dullness of the liver corresponds to the position of the lower edge of the right lung. The lower limit of the absolute dullness of the liver in the horizontal position of the patient with a normosthenic form of the chest passes along L. axillarisanteriordextra on the X rib, along L. medioclavicularisdextra - along the lower edge of the right costal arch, along L. parasternalisdextra 2 cm below the lower edge of the right costal arch, along L. medianaanterior 3-6 cm below the edge of the xiphoid process. The position of the lower edge of the liver may vary depending on the constitution and shape of the chest, but these are mainly changes in L. mediana anterior. The border of the left lobe of the liver is determined in centimeters along I. parasternalissinistra below the edge of the costal arch and to the left of this line (along the costal arch). The dimensions of hepatic dullness between the upper and lower borders are 10-12 cm according to L. axillarisanteriordextra, 9-11 cm according to L. medioclavicularisdextra, according to L.

Palpation gives more definite data. In 88% of healthy people, the lower edge of the liver is palpable, it is soft, painless, even. With liver disease, it can be dense, sharp; in patients with cardiac stagnation - rounded; tuberous - with tumors and parasitic diseases.

Laboratory diagnostic methods at the first stage play the role of screening tests, they include biochemical and instrumental studies. It is practically expedient to determine the minimum of biochemical parameters: serum bilirubin; ALT and ACT; alkaline phosphatase; total protein and proteinogram; thymol test; prothrombin time; bilirubin and urobilinogen in urine.

Screening instrumental tests include ultrasound and liver scan with 198Au, 99mTc, KT. With their help, it is possible to confirm liver damage and distinguish between focal and diffuse pathology. Scanning with a colloidal solution of I98Au or 99mTc helps to determine the localization of focal liver diseases: primary and metastatic cancer, echinococcosis, abscess, benign tumors. Various focal lesions of the liver are detected on scans as zones with a sharply reduced accumulation of radionuclides, which looks like foci with sparse shading or its absence on black-and-white scans. In addition to defects in the accumulation of drugs, focal lesions are characterized by changes in the topographic position of the liver and its size, as well as changes in the structure of the hepatic segments. For hydatidosis echinococcosis, extensive defects in the accumulation of the radionuclide are typical with a significant shift in

the silhouette of the liver and underlined shading over the affected area. With metastases of malignant tumors in the liver, a “dystrophic” background of the hepatoscan is observed, which significantly limits the diagnostic capabilities of the method.

When indicating a focal lesion, ultrasound is performed to more clearly determine the nature of the lesion - a cyst, abscess or metastases. Of course, the combined use of echohepatography and liver scanning makes it possible to obtain more information. Echohepatography performed after scanning is carried out aimingly. According to most authors, with a comprehensive study, a reliable conclusion can be obtained in 80-90% of cases of massive liver metastases, single nodes and germination of a cancerous tumor in a limited area. The importance of combined radionuclide scanning and echography in patients with liver echinococcosis is especially great. In these cases, targeted echography of areas with reduced absorption of the radionuclide on the scintigram is performed. On the echogram, a cavity containing a liquid is found that is well passable for ultrasonic waves. CT is similar in diagnostic capabilities to ultrasound, more clearly registers the contours of the liver and tumor nodes in it, especially with a diameter of more than 3-4 cm, has diagnostic advantages in the pathological process at the border of the liver and neighboring organs, when it is important to establish its primary localization. However, with the help of screening methods, it is only possible to suggest focal or diffuse liver diseases, but a specific diagnosis is not established.

At the second stage of diagnosis, the main task is to clarify the nature of focal or diffuse liver damage, i.e. establish a nosological diagnosis. The methods used in solving this problem are called selective. For the diagnosis of focal liver diseases, laparoscopy, selective angiography, as well as precision puncture biopsy under ultrasound and CT control are of particular importance. Selective methods include the reaction of latex agglutination and the determination of a-fetoprotein in the Abelev-Tatarinov reaction.

However, it should be emphasized that α-fetoprotein is not a specific marker of hepatocellular carcinoma, since this protein can also be produced by other tumors. However, if the level of α-fetoprotein exceeds 1000 ng / ml in patients with hepatomegaly and focal education in the liver according to ultrasound and scanning, then we can confidently assume hepatocellular carcinoma. Selective celiacography (angiography of the celiac trunk) is advisable to use with conflicting results of ultrasound, scanning and a-fetoprotein values. Laparoscopic examination is especially valuable in the diagnosis of malignant liver disease. In primary liver cancer, nodes of various shapes and sizes of yellowish-pink color are found on the surface of the organ against the background of an increase in the corresponding lobe of the liver. Of decisive importance for the diagnosis in these cases are the data of targeted liver biopsy. However, laparoscopic diagnosis is possible only when focal lesions are localized on the surface of the liver. Cysts, abscesses, tumors located deep in the liver tissue are not detected during laparoscopy, only deformity and enlargement of the liver are noted. In these cases, a reliable diagnostic method is celiacography, which provides important information about the state of the arterial

blood supply to the liver and spleen and allows diagnosing malignant liver tumors. only deformation and enlargement of the liver are noted. In these cases, a reliable diagnostic method is celiacography, which provides important information about the state of the arterial blood supply to the liver and spleen and allows diagnosing malignant liver tumors. only deformation and enlargement of the liver are noted. In these cases, a reliable diagnostic method is celiacography, which provides important information about the state of the arterial blood supply to the liver and spleen and allows diagnosing malignant liver tumors.

Great opportunities in the diagnosis of focal diseases are inherent in ultrasound and CT with precision puncture biopsy, which makes it possible to obtain material for cytological examination. High titers of a-fetoprotein are characteristic of hepatoma. Latex agglutination reaction is used in the diagnosis of echinococcosis.

If a diffuse disease is suspected, a nosological diagnosis is possible only after examining markers of hepatitis B, C, D, E and puncture liver biopsy. In addition, if autoimmune hepatitis is suspected, serum antibodies are determined - smooth muscle (SMA), antinuclear (ANA), hepatic-renal microsomal (LKM1), etc. In some cases, liver puncture data are insufficient for diagnosis and one has to resort to laparoscopy with targeted liver biopsy .

At the third stage, the main task of the study is to detail the diagnosis. It is necessary to clarify the presence of replication of viruses B, C, D, the activity of the process, the stage of the disease, the presence of complications. It is important to determine the degree of portal hypertension and liver failure. A special examination is carried out with indications for surgical treatment and planning its volume. The nature of the impaired function, the degree of hepatocellular insufficiency, the activity of the process are assessed using a syndromic model of impaired biochemical and immunological tests. Using the polymerase chain reaction, DNA-HBV, PHK-HCV and HDV are determined. This stage of laboratory studies is the maximum program, with the help of which it is possible to most fully establish the functional reserves of the liver and determine the prognosis.

The activity of the inflammatory process is assessed according to the indications of biochemical and immunological studies (thymol test, y-globulins and serum aminotransferase activity, the level of immunoglobulins and antibodies), as well as according to the data of a puncture liver biopsy. With a viral infection, it is important to confirm or refute the presence of viral replication.

Differential diagnosis in hepatomegaly. When diagnosing diseases manifested by an increase in the liver, it should be remembered that a neoplasm of the gallbladder, colon, and right kidney can be taken as the edge of the right lobe of the liver; in addition, hepatomegaly must be distinguished from hepatoptosis. To differentiate the true enlargement of the liver from these conditions allows palpation of the liver in various positions and ultrasound. To recognize diseases that manifest predominantly hepatomegaly, it is important to remember their diagnostic criteria. It is necessary to start diagnosing with hepatomegaly by identifying a pathology that is dangerous for others - anicteric and erased forms of acute viral hepatitis. For the diagnosis of acute viral hepatitis, information on blood transfusion and its

preparations, parenteral manipulations, chronic hemodialysis, repeated injections, prolonged stay of the patient in the hospital, the patient's belonging to risk groups or an unfavorable epidemiological history. Biochemical examination of blood serum revealed an increase in the activity of aminotransferases, aldolase. A greater increase in ALT activity than ACT is characteristic. The criteria for reliable diagnosis is the detection of markers of hepatitis A - anti-HAV; hepatitis B - HBsAg, HBeAg, anti-HBcIgM; hepatitis C - anti-HCV; hepatitis D - anti-HDVIgM; hepatitis E - anti-HEVIgM(IgG); hepatitis G - RNA-HGV. The criteria for reliable diagnosis is the detection of markers of hepatitis A - anti-HAV; hepatitis B - HBsAg, HBeAg, anti-HBcIgM; hepatitis C - anti-HCV; hepatitis D - anti-HDVIgM; hepatitis E - anti-HEVIgM(IgG); hepatitis G - RNA-HGV. The criteria for reliable diagnosis is the detection of markers of hepatitis A - anti-HAV; hepatitis B - HBsAg, HBeAg, anti-HBcIgM; hepatitis C - anti-HCV; hepatitis D - anti-HDVIgM; hepatitis E - anti-HEVIgM(IgG); hepatitis G - RNA-HGV.

After exclusion of acute viral hepatitis, it is necessary to examine serum markers of viruses B, C, D, G to detect chronic viral hepatitis, a common cause of hepatomegaly syndrome. The history data and the results of the study of serological markers make it possible to determine the etiological variant of chronic viral hepatitis and establish the phase of virus replication. Examination of liver punctates reveals varying degrees of histological activity - from minimal to pronounced. Chronic hepatitis B may be asymptomatic, but more often there are signs of liver damage in some patients with systemic manifestations (arthralgia, urticarial rash, glomerulonephritis, etc.). The presence of hepatitis B virus markers in blood serum (HBsAg, HBeAg, anti-HBc IgM class) and liver punctures is characteristic when stained with orcein according to Shikata.

Chronic hepatitis C. Asymptomatic or oligosymptomatic course of the disease is characteristic. A typical fluctuation in the activity of aminotransferases is 1.5-5 times compared with the norm, however, it often does not reflect the histological activity of the disease and does not correlate with the stage of fibrosis. Serological markers are antibodies to HCV; RNA-HCV can be found in serum and liver tissue.

Chronic hepatitis D. Accession of HDV infection to hepatitis B leads to clinical exacerbation, deterioration of laboratory parameters, often associated with the histological picture of chronic hepatitis of moderate and severe activity. Presence of anti-HDVIgM or PHK-HDV in serum; HDAg can be found in infected hepatocytes.

Antiviral therapy plays a leading role in the treatment of chronic viral hepatitis. When determining the indications, the activity of the process, clinical, biochemical and histological changes in the liver are taken into account. Chronic hepatitis B. An indication for prescribing α-interferon preparations (IFN-2b, IFN-2a) is the presence of markers of virus replication (HBeAg, DNA-HBV). The drug is prescribed at 5-10 million IU 3 times a week subcutaneously or intramuscularly for 4-6 months. An alternative drug for the treatment of chronic hepatitis B is a viral DNA polymerase inhibitor - lamivudine at a dose of 100 mg / day for 12-18 months.

The drug is also effective in the treatment of patients with a mutant strain of HBV (HBeAg, DNA-HBV+). The presence of HDV infection reduces the possibility of antiviral therapy. The main difficulties arise in chronic HBV/HDV superinfection.
Chronic hepatitis C. The indication for antiviral therapy is the presence of RNA-HCV in serum and severe histological activity or fibrosis. Treatment with a-interferon is carried out at a dose of 3 million IU 3 times a week for 16-24 months. Persistent disappearance of RNA-HCV is observed in 25-30% of cases. Combination therapy with interferon with nucleoside analogues (α-interferon + rebetol) or reverse transcriptase inhibitors (α-interferon + rimantadine) can increase the effectiveness of therapy and achieve success in the presence of the lb-HCV genotype. The course of treatment is 6 months. with genotype lb - 12 months.

Autoimmune hepatitis is characterized by the presence of a significant titer of circulating tissue autoantibodies. There are no other known causes of chronic hepatitis in the history and examination, such as hepatotropic viruses, drugs, toxic substances, alcohol, and hereditary metabolic diseases (Wilson-Konovalov disease and α-antitrypsin deficiency). Usually the disease occurs in women aged 12-25 years or in menopause after 50 years. Other autoimmune disorders are also possible: arthralgia, fever, synovitis, glomerulonephritis.
Serum aminotransferase activity is usually increased by 10 times or more, polyclonal gamma globulinopathy is noted with a predominant increase in IgG levels. Type I is characterized by the presence of SMA in titers of 1:40 or higher, ANA of a homogeneous type; II type - the presence of anti-LKMl; III type - the presence of anti-SLA. Histological picture: inflammatory infiltrates of the portal tracts contain a large number of plasma cells; polybridge necrosis and pseudoglandular transformation of hepatic cells - the so-called rosettes - are often observed. Even at an early stage of the disease, signs of cirrhosis are often present.

Liver cirrhosis is a chronic progressive disease with severe functional liver failure and portal hypertension. In 33% of patients, severe clinical symptoms occur only at the stage of decompensation of the process. The first symptom is often an enlarged, indurated liver. For diagnosis, an indication of acute viral hepatitis or history of alcoholism, extrahepatic vascular signs, gynecomastia are important, and in a biochemical study - an increase in the content of gamma globulins, aminotransferase activity, alkaline phosphatase, a reduced content of albumin, prothrombin in blood serum; detection of serological markers of hepatitis B, C, D viruses. On the echogram, focal-diffuse acoustic inhomogeneity of the tissue, an increase in the diameter of the portal splenic vein and spleen are determined. A radionuclide study shows an active accumulation of 198Au in the spleen - a symptom of a scanning spleen. To confirm portal hypertension, gastroscopy is important, which reveals the expansion of the veins of the esophagus, stomach. If necessary, perform a liver biopsy.

Treatment. With compensated liver cirrhosis, load restriction, a balanced diet with enough protein and vitamins, and abstinence from alcohol are necessary. Restrict medication intake. In active viral cirrhosis B and C with viral replication, therapy with a-interferon improves functional performance and prevents the

development of hepatocarcinoma. In patients with active cirrhosis of the liver of an autoimmune nature, glucocorticosteroids are used. Patients with subcompensated cirrhosis of the liver need a sparing regimen, a diet with salt restriction, and the appointment of drugs that improve the metabolism of liver cells. In hepatocellular insufficiency with ascites, along with the restriction of sodium chloride in food, diuretics are prescribed; in patients with encephalopathy, along with the restriction of protein in food to 20-40 g / day, lactulose and drugs that enhance ammonia metabolism (hepa-merz, or ornithine aspartate) should be used). With progressive and difficult to control liver failure against the background of non-alcoholic cirrhosis of the liver under the age of 60 years, the possibility of liver transplantation is being discussed. Complications of portal hypertension require special therapy.

Accumulation diseases - fatty hepatosis, hemochromatosis, hepatocerebral dystrophy, liver amyloidosis may not have characteristic clinical symptoms in the initial stage, and hepatomegaly in these cases is the only visible sign of the disease. Ultrasound and CT are of decisive importance in the detection of fatty hepatosis.

In the diagnosis of hemochromatosis, a significant increase in the content of iron in the blood serum, increased iron saturation of transferrin, and a sharp increase in the level of serum ferritin provide significant assistance. For the diagnosis of hepatocerebral dystrophy, neurological symptoms are important: trembling-rigid syndrome or hyperkinesis. Examination of the cornea with a slit lamp reveals a Kaiser-Fleischer ring. Characterized by a reduced content of serum ceruloplasmin < 200 ng/l, an increase in the content of copper not associated with ceruloplasmin in the blood serum and an increase in copper excretion in the urine. However, reliable diagnostic criteria for storage diseases are the data of liver punctures.

When diagnosing heart diseases, manifested in the early stages by an increase in the liver, it is necessary to remember about constrictive pericarditis with predominant localization in the right ventricle. She has a history of tuberculosis, trauma, and a wound in the region of the heart. The first signs of heart compression occur among more or less long-term well-being and are characterized by a feeling of heaviness in the right hypochondrium, enlargement and hardening of the liver, mainly in the left lobe. Shortness of breath occurs only during physical exertion, the pulse is soft, small filling. Typically, an increase in venous pressure up to 250-300 mm of water. no increase in heart size with reduced pulsation on x-ray or echocardiography.

Budd-Chiari disease is characterized by an enlarged liver, accompanied by pain in the right hypochondrium, fever. Diagnosis is based on Doppler ultrasound data with an assessment of blood flow in the portal, hepatic veins and / or lower cavography and venohepatography.

LIST OF USED LITERATURE

1. Yu.R. Kovalev "Internal diseases in questions and answers" / 2004
2. F.I. Komarov "Guide to gastroenterology" / 1995
3.DanL.Longo. AnthoniS.Fauci - Harisons Gastroenterology and Hepatology
4. Haile T. Debas- Gastrointestinal Surgery

5.S.Keshav - The Gastrointestinal System At a Glance
6.6.doktor-lib.com/…/47-glava38-zheltuxa-…
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LECTURE TOPIC: "DIFFERENTIAL DIAGNOSIS IN PATHOLOGICAL URINARY SEDIMENT. DIFFERENTIAL DIAGNOSIS FOR PROTEINURIA , LEUKOCYTURIA , HEMATURIA, CYLINDRURIA ETC. PREVENTION."

TRAINING TECHNOLOGY

Number of students-	Time - 2 hours
Form of the lesson	Lecture - visualization
Lecture plan	1. Introduction to urinary sediment, definition of the term proteinuria, types of proteinuria. 2. Causes and risk factors leading to proteinuria, various pathogenetic mechanisms of proteinuria. 3. Clinical manifestations and course of diseases with proteinuria and urinary sediment pathology. 4. Urinary sediment: cylindruria, hematuria, leukocyturia in various pathological conditions. 5. Principles of treatment, prevention and medical examination of patients with proteinuria and urinary sediment pathology
Purpose of the lesson: to familiarize students with the etiology, pathogenesis of diseases accompanied by urinary sediment pathology, proteinuria, teach the principles of differential diagnosis, treatment, prevention and medical examination of diseases accompanied by proteinuria, urinary sediment pathology	
Pedagogical tasks: 1. Strengthen and deepen students' knowledge of diseases accompanied by proteinuria and urinary sediment pathology 2. To teach students the ability to differentiate diseases accompanied by	**The results of the educational process:** **OP needs to know**: 1. Diseases leading to changes in urinary sediment, proteinuria 2. Principles of differential diagnosis of diseases accompanied by proteinuria and urinary sediment pathology 3. Principles of treatment, prevention and clinical examination of patients with urinary sediment pathology, proteinuria

proteinuria and urinary sediment pathology 4. Familiarize students with the treatment, stages of prevention of patients with urinary sediment pathology	
Teaching methodology	Lecture text, videos, questionnaires, questions, “yes-no” technique
Form of study	Laser projector, visual materials, special technical equipment, presentation of thematic patients
Means of education	Team
Conditions for the educational process	Audience

TECHNOLOGICAL CARD LECTURES

Stages, time	**Activity**	
	Teacher	**students**
Stage 1 Introduction (5 minutes)	1. Tells about the topic of the lecture, its purpose and plan	1. Listen
Stage 2 Actualization (increasing the importance) of knowledge (20 minutes)	2.1. In order to increase the actualization (increase in importance) of students' knowledge, asks questions: 1. Define the terms proteinuria, hematuria, cylindruria, leukocyturia	2.1. Answer questions asked

	2. List the diseases accompanied by proteinuria, urinary sediment pathology 3. List the main differential diagnostic signs of diseases with proteinuria and urinary sediment pathology? 4. List the groups of drugs used to treat diseases accompanied by urinary sediment pathology Conducts a survey 2.2. Showing on the screen, offers students to get acquainted with the goals and objectives of the lecture. Slide №1,2	2.2. Study slide number 1 2.3. Study slide number 2
<u>Stage 3</u> Main part (informational) (55 min)	3.1. Introduces students to the lecture material, the importance of the topic and the principles of the formation of an intelligent cultural personality, in particular, a GP teacher. In order to increase the actualization of knowledge, he conducts a quick survey of students: 1. According to 1 point of the lecture plan: list the diseases that lead to a change in urinary sediment, proteinuria 2. According to the 2nd point of the lecture plan: the basic principles of differential diagnosis of diseases accompanied by	3.1. Together they analyze the listened lecture material, ask questions

	proteinuria and urinary sediment pathology 3. Principles of treatment, prevention and clinical examination of patients with urinary sediment pathology, proteinuria Stopping at the important points of the lecture, he suggests writing down the main points in a notebook	Key points are written in a notebook.
<u>Stage 4</u> final (10 min)	4.1. Asking questions: 1. List the most common diseases accompanied by proteinuria and urinary sediment pathology 2. Tell us the main differential diagnostic signs of diseases accompanied by urinary sediment pathology and proteinuria 3. Name the basic principles of treatment, prevention of patients 4.2. Gives a task for independent work of students: Hereditary tubulopathies	4.1. Answer questions 4.2. Listen, write

To date, a number of diseases are accompanied by changes in urine sediment. In particular, we will focus on diseases that occur with urinary sediment pathology and proteinuria.

One of the diseases that occur with severe proteinuria is multiple myeloma, which is characterized by the following symptoms:

- ✓ Accompanied by the production of pathological immunoglobulins of one clone, causing tubular obstruction.
- ✓ With this disease, proteinuria can reach up to 20 g / day;

Other clinical signs: bone pain; spontaneous fractures caused by destructive processes in the bones; hepato- or splenomegaly; parasthesia, anemia, bleeding; in the blood: severe dysproteinemia with an M-gradient, elevated levels of pathological immunoglobulins; in sternal punctate - plasma cells, accompanied by the production of pathological immunoglobulins of one clone, causing tubular obstruction, with this disease, proteinuria can reach up to 20 g / day; myeloma kidney: filling of the renal tubules with cylinders consisting of k andλ- chains of immunoglobulins, antibodies to them, Tamm-Horsfall protein, partly from albumin and fibrinogen; acute renal failure

Valdestrom's macroglobulinemia is a disease in which pathological IgM is synthesized and accumulates in the blood. Kidney damage occurs, but rarely (proteinuria frequency 15-20%), other clinical signs are most characteristic: splenohepatomegaly, hemorrhagic syndrome; a sharp increase in ESR; an isolated increase in the IgM fraction, osteoparosis is sometimes observed.

Intravascular hemolysis - transfusion of incompatible blood; exposure to hemolytic poisons and toxins; medicinal, immunological and traumatic injuries of erythrocytes. During hemolysis, free hemoglobin binds to plasma haptoglobulin. When the blood is saturated with haptoglobin, hemoglobin begins to be filtered by the glomeruli. The clinical picture of acute renal failure develops. In urine: PU, EU, LU, free hemoglobin.

Rhabdomyolysis (traumatic crush syndrome)accompanied by PU, myoglobinuria, increased levels in the blood: aminotransferase, creatine phosphokinase, hyperkalemia, hyperuricemia.

In the clinical picture, the leading ones are: swelling and pain in damaged muscles, oligo-, anuria with the clinical picture of acute renal failure.

Glomerular proteinuria (0.1-20 g / day) - due to damage to the basement membrane. The protein fraction is represented by albumin, transferrin, β-microglobulin, globulin. Glomerular proteinuria is observed: in acute and chronic glomerulonephritis; amyloidosis; diabetic glomerulosclerosis. Glomerulonephritis is very common, and therefore we will dwell on them in more detail.

In the practice of a doctor, glomerulonephritis (nephritis) is not as common as, for example, coronary heart disease, rheumatic diseases or chronic non-specific lung diseases. However, their medical and social significance is great, which is determined by the following provisions.

Firstly, only acute nephritis, relatively rare at the present time, ends (and even then in no more than half of the cases) with recovery; Chronic nephritis, especially subacute, steadily progresses towards chronic renal failure (CRF), often accompanied by edema (nephrotic syndrome) and severe hypertension, leading to disability even before the development of CRF.

Secondly, young, able-bodied men get sick more often (and get sick more severely).

Thirdly, although there are methods that allow replacing non-functioning kidneys (the so-called renal replacement therapy) - dialysis and transplantation, but in Russia they are far from available to everyone (the need is met by about 1/20), they are very expensive and have their own difficulties - attachment to devices, the need for

constant immunosuppression, etc. All this makes the problem of conservative therapy aimed at suppressing the activity of nephritis and inhibiting its progression an urgent problem.

Let us say right away that the term “nephritis” is currently fully consistent with the term “glomerulonephritis” (GN) and includes a group of morphologically heterogeneous immunoinflammatory diseases with a predominant lesion of the glomeruli, as well as involving tubules and interstitial (interstitial) tissue. GN are independent nosological forms, but can also occur in many systemic diseases, such as systemic lupus erythematosus, hemorrhagic vasculitis, infective endocarditis, etc. The progression of GN after exposure to various initial factors is determined by fairly stereotyped processes that gradually lead to sclerosis. This is the proliferation of mesangial cells and the accumulation of the mesangial matrix, as well as damage to the wall of the capillaries of the glomerulus. In the induction of GN, the leading place is given to immune factors of damage, reactions of humoral and/or cellular immunity; in further progression, inflammatory mediators and non-immune mechanisms - hemodynamic and metabolic - play an important role. At all stages of development, the persistence of the etiological factor plays a leading role, which, unfortunately, is known only in 1/10 patients.

Etiology: • Infections: bacterial (streptococcus, staphylococcus, etc.); viral (hepatitis B, hepatitis C, etc.); parasitic.

• Toxic substances (organic solvents, alcohol, lead, mercury, drugs, etc.).

• Exogenous non-infectious antigens that act with the involvement of immune mechanisms, including the type of atopy.

• Endogenous antigens (rare): DNA, tumor, uric acid.

Clinical Syndromes

• Urinary syndrome (proteinuria and / or hematuria).

• Nephrotic syndrome (edema, proteinuria above 3.5 g per day, hypoalbuminemia, hyperlipidemia).

arterial hypertension.

• Impaired kidney function.

Clinical types of glomerulonephritis. The main clinical types of GN are acute, chronic, and rapidly progressive.

Acute GN develops 6–12 days after infection, usually streptococcal (angina, tonsillitis, pyoderma); the most nephritogenic b-hemolytic streptococcus group A, especially strains 12 and 49. Characterized by hematuria (often gross hematuria), edema, oliguria, hypertension. In children, acute GN usually has a cyclic course, with a rapid onset, in most cases ends in recovery. In adults, an erased variant with changes in urine without common symptoms is more common, gradually taking on a chronic course. If a connection with streptococcal infection is proven, titers of antistreptococcal antibodies are elevated and sowing from the pharynx is positive, antibiotic treatment is indicated for 8-12 days. Chronic GN often develops slowly, with an imperceptible onset, less often there is a clear connection with acute nephritis.

Latent GN is the most common form of chronic GN, manifested only by changes in urine (proteinuria up to 2-3 g per day, slight erythrocyturia), sometimes mild arterial hypertension. The course is usually slowly progressive.

Hypertensive GN is latent GN with more pronounced hypertension and minimal urinary syndrome.

Hematuric GN is manifested by constant hematuria, often with episodes of gross hematuria, proteinuria does not exceed 1 g per day, the course is quite favorable.

Nephrotic GN is characterized primarily by nephrotic syndrome (NS), which is usually recurrent. In the absence of erythrocyturia and hypertension (which is commonly seen in children), the prognosis is relatively good. In adults, isolated NS is rare, the combination with erythrocyturia and / or hypertension significantly worsens the prognosis; when combined with hypertension, they speak of mixed GN.

Quite common clinical and morphological classification of chronic GN, which is based on morphological changes in the glomeruli of the kidneys, it includes 5 forms.

Minimal changes in the glomeruli are detected only with electron microscopy; with light microscopy, the glomeruli appear intact. This morphological form is observed more often in children, but also occurs in adults. Pronounced NS with massive edema is characteristic; erythrocyturia and arterial hypertension are rare. It is with this form that glucocorticoids are most effective, sometimes leading to the disappearance of edema in 1 week. The prognosis is quite favorable, CRF rarely develops. Focal-segmental glomerulosclerosis (FSGS) is sometimes difficult to distinguish from the previous form with light microscopy, however, in some of the glomeruli, sclerosis of individual capillary loops is detected; can develop with HIV infection, intravenous drug use. The clinical picture is characterized by persistent proteinuria or NS, usually in combination with erythrocyturia and arterial hypertension. The course is progressive, the prognosis is serious, it is one of the most unfavorable morphological variants, which rarely responds to active immunosuppressive therapy.

Membranous GN (membranous nephropathy) is characterized by diffuse thickening of the walls of glomerular capillaries with their splitting and doubling, massive deposition of immune complexes on the epithelial side of the glomerular basement membrane. In a third of patients, it is possible to establish a connection with known antigens - the hepatitis B virus, tumor, drugs. Therefore, patients with membranous nephropathy should be especially carefully examined in order to detect a tumor or hepatitis virus infection. The disease often develops in men, is characterized by proteinuria or NS, hematuria and arterial hypertension are observed in 15-30% of cases. The course is relatively favorable, especially in women, renal failure develops in only half of the patients.

Mesangioproliferative GN is the most common morphological type of GN that meets (unlike the previous variants) all the criteria for GN as an immunoinflammatory disease; characterized by proliferation of mesangial cells, expansion of the mesangium, deposition of immune complexes in the mesangium and under the endothelium. The clinical picture is characterized by proteinuria and / or hematuria, in some cases, NS and hypertension are noted. The current is relatively favorable.

As a separate option, mesangio-proliferative GN with deposition of immunoglobulin A in the glomeruli is isolated: IgA nephritis or Berger's disease. The disease develops at a young age, more often in men, the leading symptom is hematuria. In 50% of patients, recurrent gross hematuria occurs in the first days or even hours of febrile respiratory diseases. The prognosis is favorable, but worsens significantly with the addition of NS and / or hypertension. In some countries (such as Japan), IgA nephritis is the predominant type of nephritis. The last morphological variant of chronic GN is mesangiocapillary GN, which is characterized by a pronounced proliferation of mesangial cells with their spread and penetration into the glomerulus, which creates a characteristic glomerular lobulation with doubling of the basement membranes. May be associated with hepatitis C virus, as well as cryoglobulinemia. Proteinuria and hematuria are characteristic, NS and hypertension are frequent. This is an unfavorable variant of GN with a progressive course and the development of CRF, which responds poorly to therapy.

In addition to acute and chronic GN, there is a relatively rare rapidly progressive (subacute) GN (RPGN) with "crescents" in the glomeruli and rapidly progressive renal failure. RPGN can develop after infections, contact with organic solvents, but more often it is caused by systemic vasculitis and systemic lupus erythematosus. The prognosis is very serious, only massive immunosuppressive therapy can stop the progression.

Thus, most forms of GN are, to varying degrees, progressive diseases; the most reliable way to stop progression is to identify and eliminate the etiological factor. If it is impossible to establish the etiology, efforts should be aimed at suppressing the activity of the disease and stopping the exacerbation, which makes it possible to stabilize the course of the disease.

Treatment. To suppress the activity, immunosuppressive therapy is used: glucocorticoids, non-selective cytostatics and cyclosporine A.

Glucocorticoids (GCs), which have immunosuppressive and anti-inflammatory effects, have been the main means of pathogenetic therapy for nephritis for several decades. More commonly used orally - prednisolone in high (1-2 mg / kg per day) or moderately high (0.6-0.8 mg / kg per day) doses, daily in 2-3 doses or once in the morning, for a long time (1- 4 months), followed by a slow decline. An alternating regimen is also possible, when the patient takes a double daily dose once every other day in the morning. With high activity of renal inflammation, in order to quickly achieve very high concentrations of HA in the blood plasma, "pulse therapy" is indicated - intravenous administration of ultra-high doses (0.8-1.2 g) of methylprednisolone or prednisolone.

Cytostatics in the treatment of nephritis are used somewhat less frequently than GCs due to the greater severity of side effects. As a rule, alkylating compounds are used - cyclophosphamide (at a dose of 1.5-2 mg/kg per day) and chlorbutine (at a dose of 0.1-0.2 mg/kg per day); the antimetabolite azathioprine is less effective, although less toxic. The decrease in the number of leukocytes occurs within a few days or weeks. During this period, it is important to check the number of leukocytes in the peripheral blood every 2-3 days, so that if they decrease to the lower acceptable

level, the dose of the drug can be reduced or canceled. The most serious side effects of alkylating cytostatics are bone marrow suppression, infections, and gonadal failure. Other complications: hepatitis, alopecia, hemorrhagic cystitis, gastrointestinal disorders and an increased risk of developing tumors.

Of great interest is the selective immunosuppressant cyclosporin A (CsA), which has been used for a long time in transplantology, and in recent years in therapeutic nephrology. The initial dose of CsA for adults in the nephrology clinic is 3-5 mg/kg, for children - 6 mg/kg per day. In the future, the dose depends on tolerability, the presence of side effects and the concentration in the blood serum, which should be regularly checked.

CsA is indicated primarily for patients with minimal changes and FSGS with frequent recurrence of NS or steroid-resistant NS, with the development of complications of steroid and cytostatic therapy. The most serious complications of CsA treatment are hypertension and nephrotoxicity.

In the appointment of immunosuppressive therapy should be guided by the following provisions.

• With high GN activity, immunosuppressive therapy is always indicated.

• New-onset NS, especially without hematuria and hypertension, is always an indication for GC treatment.

• With rapidly progressive forms of nephritis (with a rapid increase in creatinine levels), it is imperative to prescribe immunosuppressants - large doses of HA and cytostatics orally and / or in the form of “pulses”.

Not only immunosuppressive therapy, but also the so-called non-immune nephroprotective therapy, the possibilities of which have expanded significantly over the past decade, can stabilize the course of GN, slow down its progression, and sometimes even lead to its regression. At the present stage, we can talk about four methods of nephroprotective therapy, the effect of which on the progression of GN has been proven or is being studied. These are: angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers; heparin sodium; dipyridamole; lipid-lowering drugs, especially statins.

It should be emphasized the importance of careful outpatient monitoring of patients, dietary recommendations, timely detection of exacerbations, for which therapists need knowledge of nephrological problems and timely referral of patients to nephrologists. Chronic pyelonephritis is a chronic non-specific infectious and inflammatory process that occurs in the pyelocaliceal system of the kidneys and tubulointerstitial zone.

The most common causative agents of chronic pyelonephritis are gram-negative bacteria of the intestinal group - Escherichia coli, enterococci, Proteus, Pseudomonas aeruginosa, less often staphylococci, streptococci. In the chronic course of the disease, there is often a change of pathogens or their pathological effect as part of microbial associations. The persistence of pathogens in the urinary tract is explained by the presence of L-forms of bacteria, which, under unfavorable conditions for the body, can turn into active forms. Most gram-negative bacteria - the main causative agents of pyelonephritis - have a kind of cilia (fimbria), the

receptors for which are some structures of the membranes of the cells of the urinary tract. This allows bacteria to attach to cells in the urinary tract (bacterial adhesion phenomenon).

The most common route of infection in the urinary tract is urogenic, rarely hematogenous. The most important factor in pathogenesis is a violation of urodynamics due to organic and functional factors that prevent the outflow of urine and increase the likelihood of infection. Organic causes include calculi, anomalies in the development of the urinary tract, pathological processes in the prostate gland. In men, the causes of urodynamic disorders are almost always organic. In women, functional causes predominate - due to certain changes in the hormonal background, in particular an increase in estrogen, the tone of the ureters decreases, their dyskinetic disorders appear.

The question of specific mechanisms of immunological disorders has not been finally resolved. The long-term, chronic course of pyelonephritis, the persistence of infection is explained by the lack of mechanisms, both local and general immunity. Immunological complexes are found in the renal tissue - antibodies connected to the fimbriae receptors of the bacteria that cause pyelonephritis. These immune complexes can cause immune inflammation in the interstitium and damage the tubular apparatus. Developing interstitial nephritis explains many manifestations of pyelonephritis, in particular, an early decrease in the concentration function of the kidneys, involvement in the process of glomeruli and vessels with the gradual development of chronic renal failure.

The main clinical manifestations of pyelonephritis.

There are clinical forms of chronic pyelonephritis - the most common - recurrent, as well as latent. Phases of the process - exacerbation, remission.

In the acute phase, an intoxication syndrome is expressed to some extent, due to an active inflammatory process in the urinary tract and interstitial tissue of the kidneys. It is manifested by fever, weakness, decreased performance, increased sweating. The pain syndrome is explained by stretching of the pelvis due to urodynamic disorders, an increase in the content of inflammatory mediators in the kidney tissue, and malnutrition of the renal tissue. Pain is usually unilateral, dull, aching, with urolithiasis it can be of the type of renal colic. The syndrome of impaired diuresis is manifested by both urinary disorders and urination disorders. Pyelonephritis is characterized by moderate polyuria due to damage to the tubules and a decrease in water reabsorption. Dysuric disorders are often observed - painful and frequent urination,

Urinary syndrome - prevails especially during exacerbation of the process of leukocyturia, there may be moderate proteinuria and hematuria. Leukocyturia is caused by an inflammatory process in the urinary tract; proteinuria and hematuria - interstitial nephritis.

At any stage of the process, it can be complicated by symptomatic arterial hypertension, which is explained, on the one hand, by an increase in the pressor function of the kidneys, and, on the other hand, by a decrease in its depressor function. The natural ending of chronic pyelonephritis is the development of chronic

renal failure due to sclerotic changes in the kidney parenchyma and a decrease in the mass of active nephrons. Chronic pyelonephritis - data of laboratory and instrumental research methods. In the examination of urine by the usual routine method, the most important symptom is leukocyturia, as well as hematuria and proteinuria. Leukocytes usually appear in large numbers during the exacerbation phase. In the phase of unexpressed exacerbation, remission, the number of leukocytes may not exceed the norm. In these situations, it is recommended to determine the number of formed elements in a certain time or in a certain volume. The most common tests are Addis-Kakovsky (number of formed elements per day) and Nechiporenko (determination in 1 ml). With pyelonephritis, the number of leukocytes per day is usually more than 2 million, in 1 ml - more than 2 thousand. Of great importance is the bacteriological examination of urine with the definition of the so-called. microbial number - with pyelonephritis, the microbial number of 100,000 or more has a diagnostic value. Radioisotope renography - the asymmetry of changes is determined. Of great importance is the bacteriological examination of urine with the definition of the so-called. microbial number - with pyelonephritis, the microbial number of 100,000 or more has a diagnostic value. Radioisotope renography - the asymmetry of changes is determined. Of great importance is the bacteriological examination of urine with the definition of the so-called. microbial number - with pyelonephritis, the microbial number of 100,000 or more has a diagnostic value. Radioisotope renography - the asymmetry of changes is determined.

In a functional study of the kidneys, in particular the Zimnitsky test, hypo- and isosthenuria can be detected already in the early stages, as a manifestation of the impaired concentration function of the tubules while the total kidney function is preserved, the latter is disturbed in the later stages of the development of the pathological process during the development of chronic renal failure.

Particularly important in the diagnosis of chronic pyelonephritis is attached to imaging methods - ultrasound, excretory urography.

Anomalies in the development of the kidneys, changes in the pelvis, calculi are detected sonographically. The cortico-renal index changes. X-ray urological examination can conditionally be considered a decisive method for diagnosing chronic pyelonephritis with a sufficiently long course of the process. The main radiological signs are expansion and deformation of the pelvis, spasm or expansion of the necks, changes in the structure of the cups.

A kidney biopsy is a direct method for diagnosing pyelonephritis, but only with positive results, since the pathological process in the kidneys with pyelonephritis is often focal.

The most characteristic morphological signs of pyelonephritis are large scars, lymphoid and histiocytic infiltrates in the interstitium, areas of tubular expansion, some of which are filled with colloidal masses - “thyroid-like” tubule transformation. In the later stages, the glomeruli are naturally affected.

AMYLOIDOSIS. Amyloidosis is a complex disorder of protein-carbohydrate metabolism, which leads to the formation of a special substance, amyloid, in the

internal organs and systems. Thanks to the latest research methods, the physical, chemical and antigenic properties of the amyloid substance have been studied in sufficient detail. However, despite the fact that more than 140 years have passed since the discovery of amyloid, many questions regarding the problem of amyloidosis still remain unresolved. With the help of intravital puncture biopsy and histochemical studies of punctures taken from organs affected by amyloidosis, it was established that the amyloid substance is a complex product of a protein nature - a glycoprotein in which proteins are strongly associated with polysaccharides and differ in amino acid composition from tissue and plasma proteins. The distribution of amyloidosis among the population of different countries has significant differences. So, according to pathoanatomical data, in the USA and some European countries, kidney amyloidosis is detected in 0.7%, while in Asian countries it is less common - up to 0.2%, which is explained by the peculiarities of the nutrition of the population of these countries, in particular, the low content in food diet of animal proteins and cholesterol. The high prevalence among the population of Spain (1.92%) and Portugal (1.43%) is due to the development of familial amyloidosis in these countries. It most often affects males aged 40-50 years, however, cases of kidney amyloidosis in children and even newborns are described. The causes of this disease are very diverse. It is known that many diseases can be complicated by the development of amyloidosis, but it can also be an independent disease.

In recent years, primary amyloidosis has become more common. Secondary amyloidosis develops as a result of chronic suppurative processes or infectious-allergic diseases, usually several years after the onset of the underlying disease. In this case, the deposition of amyloid occurs primarily in the affected organ, and then it accumulates in other parenchymal organs. Depending on the intensity of amyloid deposition in a particular organ, nephropathic, hepatopathic, cardiopathic, or mixed types of amyloidosis are distinguished.

The most common cause of secondary amyloidosis is pulmonary tuberculosis. Such an increase in amyloidosis can be explained by an increase in the life expectancy of patients with tuberculosis, as well as autoimmune reactions caused both by the disease itself and by the use of antimicrobial drugs. It should be borne in mind that amyloidosis in some cases occurs in people with inactive or cured pulmonary tuberculosis, but with severe pneumosclerosis, emphysema.

Among other diseases in the development of amyloidosis, purulent processes (bronchiectasis, lung abscess formation, osteomyelitis, etc.) are of great importance. The second place among the causes leading to the development of amyloidosis is rheumatoid arthritis, which, in 20% of cases, is complicated by amyloidosis. Approximately with the same frequency, amyloidosis occurs in patients with periodic illness, mainly the kidneys are affected. It can be caused by lymphogranulomatosis, tumors, multiple myeloma and other diseases. A number of theories have been proposed to explain the mechanism of development of amyloidosis.

According to the theory of dysproteinosis, or organoproteinosis, the amyloid substance is a product of perverted protein synthesis. In the blood plasma, globulin

protein fractions and abnormal proteins (paraproteins) accumulate, which, penetrating from the bloodstream into tissues, form amyloid substance in the cells of the active mesenchyme. According to this theory, the development of amyloidosis is usually preceded by dysproteinemia resulting from any chronic disease - tuberculosis, bronchiectasis, rheumatoid arthritis, malignant neoplasms, etc. At the same time, there is an increase in the content of globulin fractions in the blood, especially a2-globulins, and a decrease in albumin levels.

The theory of "cellular local genesis" explains the formation of amyloidosis by a perversion of the protein-synthetic function of the cells of the reticuloendothelial system. In this case, the synthesis of amyloid occurs as if in two phases. In the first - active, or preamyloid, as a result of the proliferation of elements of the reticuloendothelial system, the so-called pyroninophilic cells appear and the level of γ-globulins in the blood serum increases. In the second, proper amyloid phase, the cellular transformation of the reticuloendothelial system is suppressed and pyroninophilic cells are "depleted", the level of y-globulins decreases and the content of a- and b-globulins in the blood serum increases.

The immunological theory of the pathogenesis of amyloidosis links the formation of amyloid substance with autoimmune reactions. According to this theory, in many primary diseases, tissue decay products, leukocytes, and bacterial toxins accumulate, which can play the role of autoantigens, followed by the formation of autoantibodies. The reaction of the interaction of the antigen with the antibody leads to the development of amyloidosis and its deposition in the places of accumulation of antibodies, i.e., in the elements of the reticuloendothelial system.

There is no generally accepted classification of amyloidosis. The classification of V. V. Serov and I. A. Shamov is considered the most convenient for a practical doctor. In it, amyloidosis is grouped depending on the types, forms and types, as well as on the reasons leading to its development. According to this classification, there are:

1. Idiopathic (primary) amyloidosis: generalized (classic), nephropathic, neuropathic, cardiopathic, localized.
2. Hereditary (genetic) amyloidosis: periodic illness (familial Mediterranean fever); familial amyloidosis with fever, urticaria and deafness; familial amyloidosis with allergic manifestations, fever and nephropathy; familial neuropathic amyloidosis; familial cardiopathic amyloidosis.
3. Acquired (secondary): amyloidosis as a complication of chronic infections, collagen diseases and malignant tumors; paraamyloidosis (amyloidosis in paraproteinemic hemoblastoses).
4. Senile amyloidosis.
5. Local tumor-like amyloidosis.

Clinical manifestations of amyloidosis of the kidneys are very diverse: they depend on the localization of amyloid deposits in other organs, the duration of the disease, the severity of the violation of the structure and function of the affected organ, as well as the severity of the primary disease that caused the development of amyloidosis.

As already noted, in contrast to primary, hereditary and senile amyloidosis, secondary develops against the background of any chronic disease or after it. At the same time, according to available data, its appearance does not depend on the duration and severity of the underlying disease. Nevertheless, secondary amyloidosis often occurs several or many years after the underlying disease, although there are cases of amyloidosis developing 3 months after the onset of the underlying disease. It is impossible to accurately determine the timing of the end of the underlying disease and the onset of the development of amyloidosis in most cases, since it is usually recognized at a late stage of its development. Of great practical importance is the timely detection of amyloidosis at an early stage, when it is possible to reverse the development of this pathological process, while in the later stages it is practically irreversible or recovery is extremely rare. From this point of view, the differentiation of the course of amyloidosis of the kidneys by stages seems practically important. Four stages of renal amyloidosis are clinically distinguished: latent, proteinuric, nephrotic, and azotamic. The latent stage of amyloidosis is almost asymptomatic. When diagnosing it, it is necessary to pay attention to the symptoms of the underlying disease, which can be potentially dangerous in relation to the development of amyloidosis. The possibility of this factor increases in cases where the underlying disease is accompanied by the periodic occurrence of a small proteinuria. Symptoms such as an increase in the size of the liver and spleen should also be taken into account. From this point of view, the differentiation of the course of amyloidosis of the kidneys by stages seems practically important. Four stages of renal amyloidosis are clinically distinguished: latent, proteinuric, nephrotic, and azotamic. The latent stage of amyloidosis is almost asymptomatic. When diagnosing it, it is necessary to pay attention to the symptoms of the underlying disease, which can be potentially dangerous in relation to the development of amyloidosis. The possibility of this factor increases in cases where the underlying disease is accompanied by the periodic occurrence of a small proteinuria. Symptoms such as an increase in the size of the liver and spleen should also be taken into account. From this point of view, the differentiation of the course of amyloidosis of the kidneys by stages seems practically important. Four stages of renal amyloidosis are clinically distinguished: latent, proteinuric, nephrotic, and azotamic. The latent stage of amyloidosis is almost asymptomatic. When diagnosing it, it is necessary to pay attention to the symptoms of the underlying disease, which can be potentially dangerous in relation to the development of amyloidosis. The possibility of this factor increases in cases where the underlying disease is accompanied by the periodic occurrence of a small proteinuria. Symptoms such as an increase in the size of the liver and spleen should also be taken into account. The latent stage of amyloidosis is almost asymptomatic. When diagnosing it, it is necessary to pay attention to the symptoms of the underlying disease, which can be potentially dangerous in relation to the development of amyloidosis. The possibility of this factor increases in cases where the underlying disease is accompanied by the periodic occurrence of a small proteinuria. Symptoms such as an increase in the size of the liver and spleen should also be taken into account. The latent stage of

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The main clinical and laboratory sign of the latent stage is proteinuria, usually transient, unstable and insignificant. Occasionally, slight microhematuria and, even more rarely, minimal leukocyturia can be detected. Stable dysproteinemia is characteristic, which persists even with a favorable course of the underlying disease and is manifested by an increase in globulin fractions, mainly a2- and y-globulins. The level of glycoproteins and mucopolysaccharides, as well as fibrinogen increased to the upper limit of normal. Most patients have a significant and persistent increase in ESR in the absence of signs of exacerbation of the underlying disease. Kidney function at this stage does not suffer. In the biopsy specimen of the renal tissue, taken with the help of intravital puncture biopsy, an amyloid substance is found, which is located along the basement membranes of the rectus vessels,

The main clinical manifestation of the proteinuric stage of renal amyloidosis is persistent proteinuria, which is characterized by significant protein fluctuations (from 0.1 to 3.0 g / l) in the urine with microhematuria, cylindruria and occasionally leukocyturia. The most pronounced proteinuria is observed in secondary amyloidosis, although it is observed in primary and hereditary amyloidosis, but to a lesser extent. Constant loss of protein in the urine through the gastrointestinal tract, an increase in its breakdown in the body lead to the development of hypoproteinemia with hypoalbuminemia.

Significant changes in blood biochemical parameters are noted: severe dysproteinemia with hypoalbuminemia (up to 36.0%) and hyperglobulinemia in the form of an increase in fractions a1- (up to 9.0%), a2- (up to 15.0-16.0%) and y-globulins (up to 23.0-25.0%); hyperfibrinogenemia (up to 5.5 g / l), an increase in the content of sialic acids (up to 0.300) with normal or even low cholesterol concentrations. Significantly increased ESR, moderate anemia appears. The electrolyte balance changes, the amount of sodium and potassium decreases.

The main clinical manifestation of the nephrotic stage of amyloidosis is nephrotic syndrome, which is characterized by massive proteinuria, severe hypo- (up to 5.0-3.0 g / l) and dysproteinemia in the form of significant hypoalbuminemia (up to 20-30% and below), hyperalpha- 2-globulinemia (up to 20-30%) and hypergammaglobulinemia (up to 25%); hyperlipidemia, in particular hypercholesterolemia (up to 12.0 mmol / l or more), the presence in most patients (70-75%) of widespread pronounced edema, characterized by great resistance to diuretics. Characterized by hypotension, which is sometimes associated with damage to the adrenal glands amyloidosis. Anemia and a sharply accelerated ESR are noted. In addition to proteinuria, microhematuria, cylindruria and leukocyturia are often observed. The electrolyte balance of the blood is disturbed: the level of

sodium and potassium decreases; the content of b-lipoproteins and fibrinogen increases.

In the nephrotic stage, the symptoms of the underlying disease are mild, the clinic of nephrotic syndrome comes to the fore. Half of the patients have hepatomegaly and hepatolienal syndrome.

The azothemic stage of amyloidosis corresponds to the clinical picture of chronic renal failure, which does not differ significantly from that in other primary and secondary kidney diseases. Azotemic uremia, which is the main cause of death in this disease, is considered to be the end of CIN.

In renal amyloidosis, there may be no parallelism between the severity of chronic renal failure and the morphological picture of an amyloid-wrinkled kidney. Quite often at the patients who died from uremia, there are no signs of an amyloid-wrinkled kidney. Renal failure can develop and cause death in the proteinuric, nephrotic, and even latent stages of amyloidosis. Most often, the reasons for the rapid progression of renal failure in amyloidosis can be an exacerbation of the underlying disease, the addition of an intercurrent infection, or complications such as renal vein thrombosis, a sharp drop in blood pressure, and, consequently, glomerular filtration, for example, in adrenal amyloidosis, etc.

It is quite difficult to establish a diagnosis of renal amyloidosis in the early period of the disease, despite the fact that recently the diagnostic possibilities have increased significantly. In the recognition of secondary amyloidosis, an undoubted role is played by the nature, clinical picture, course and duration of the underlying disease preceding amyloidosis. It should be borne in mind that in some cases the primary disease may be latent or recovery may occur. Of paramount importance is the correct interpretation of the urinary syndrome. In this case, the most important initial diagnostic sign of amyloidosis is proteinuria, which steadily increases as amyloidosis progresses. Leukocyturia in amyloidosis is found quite often without a clinical picture of pyelonephritis, which is considered a valuable diagnostic feature, especially in the latent stage. Microhematuria in amyloidosis, according to E. M. Tareev, occurs in 11.5% of cases. The frequency and severity of cylindruria depend on the presence and severity of proteinuria. Hyaline casts are found more often than granular ones. In the advanced stage of secondary amyloidosis, birefringent lipids are detected in the urine. Dysproteinemia in secondary amyloidosis is a characteristic feature and depends both on the underlying disease and on the amyloidosis itself. Amyloidosis is characterized by an increase in the content of a2-globulins. In secondary amyloidosis, there is a regular increase in fibrinogen as the disease progresses. A certain relationship has even been established between the level of fibrinogen and the stage of amyloidosis: the higher the level of fibrinogen, the more reason to think about the late stage of amyloidosis. Hyperfibrinogenemia is a characteristic feature for some forms of hereditary amyloidosis, especially for amyloidosis in periodic illness, when a high level of fibrinogen is found regardless of the phase of the disease. Hyperhypidemia is detected in primary, secondary and hereditary amyloidosis. Severe hypercholesterolemia in the nephrotic and azothemic

stages of secondary amyloidosis is considered one of the criteria for diagnosing late stages of amyloidosis.

Edema occurs in the nephrotic stage of amyloidosis of the kidneys. However, in some cases they may occur at an earlier stage. So, their detection in the proteinuric stage is associated with the development of pulmonary heart failure caused by the underlying disease (with lung damage). Unlike nephrotic-type edema in glomerulonephritis, which develops rapidly at the onset of the disease, in renal amyloidosis, edema increases slowly. The occurrence of edematous syndrome at an early stage of amyloidosis of the kidneys can also be triggered by the addition of an intercurrent infection, drug intolerance, and other reasons. In all forms of amyloidosis of the kidneys, and especially often in the secondary, hepato- and splenomegaly are found. Enlargement of the liver is detected in 60%, spleen - in 24% of patients. Hepatomegaly in amyloidosis is due to a number of factors, including amyloid deposition and congestion. In the early stage of amyloid hepatomegaly, the deposition of amyloid substance in the liver occurs only in minimal amounts, therefore, there is no violation of its functional ability. In the advanced stages, the amyloid substance is deposited in significant quantities and disrupts the structural and functional ability of the liver, followed by the development of liver failure. The liver at the same time becomes dense, painful, jaundice, ascites sometimes develop. Enlargement of the spleen is found only in the advanced stage of amyloidosis. For the diagnosis of amyloidosis, the complement fixation reaction (CFR) is used with the blood serum of patients, while amyloid protein is used as an antigen. CSC with amyloid antigen is considered a highly sensitive diagnostic test. Special colorful tests (with Congo red, with methylene blue, with Evans's paint) also have a certain value in the diagnosis of amyloidosis. The most informative and reliable method for diagnosing amyloidosis is an intravital biopsy of organs and tissues. Detection of amyloid in organs makes it possible not only to confirm the diagnosis, but also to determine the stage of amyloidosis. This method allows in 87-100% of cases to establish amyloidosis of the kidneys. A kidney biopsy is especially valuable to identify the nature of the nephrotic syndrome: whether it is caused by amyloidosis of the kidneys, glomerulonephritis or other diseases, which is very important to know when choosing a method of treatment and determining the prognosis of the disease. Liver biopsy has a high diagnostic value in systemic amyloidosis. Spleen biopsy is not widely used because of the potential for bleeding. Biopsy of the colonic mucosa confirms the diagnosis of primary and secondary amyloidosis in about 70% of cases.

Biopsy of the gingival mucosa is the most accessible method for diagnosing amyloidosis, in addition, it does not give complications. However, its diagnostic value is not recognized by all; positive results in the presence of amyloidosis are obtained only in 40% of cases.

Amyloidosis of the kidneys can proceed in different ways. It depends on the localization and degree of amyloid deposition, involvement of other organs and systems in the process, on the nature and severity of the underlying disease. However, renal amyloidosis almost always has a chronic progressive course, which

ultimately leads to impaired renal function, the development of chronic renal failure and its final phase - azotemia uremia with a fatal outcome.

The life expectancy of patients with amyloidosis of the kidneys ranges from 1 to 3 years from the time of diagnosis. Cases are described when patients lived up to 10 years or more. The lethal outcome largely depends on the course of the underlying disease; in some cases, death occurs from the underlying disease that caused the development of amyloidosis. The outcome of amyloidosis depends on various complications: hemorrhages, thrombosis, intercurrent infections, etc. Recovery from amyloidosis is extremely rare if the diagnosis is made in the initial (early) stages of the disease, active treatment is started in a timely manner and the underlying disease is completely cured, which caused the development of amyloidosis.

With amyloidosis of the kidneys, treatment is still ineffective, especially with its late diagnosis. Scientifically, theoretically and experimentally substantiated treatment is mainly aimed at individual links in the pathogenesis of amyloidosis, i.e., the elimination of those factors that contribute to the formation of amyloid, the use of agents that inhibit the production and stimulate the resorption of amyloid.

In secondary amyloidosis, methods and means aimed at eliminating the main symptoms or completely curing the disease, the consequence of which it is, are of paramount importance. For this purpose, both conservative and radical (surgical) methods of treatment are used.

In the complex therapy of amyloidosis of the kidneys, a significant place is occupied by the diet. In the initial stage of amyloidosis, a low-protein diet (at the rate of 0.7 g of protein per 1 kg of body weight) with a high content of carbohydrates and rich in vitamins is recommended. The inclusion in the diet of fruits, berries, especially those containing a lot of vitamin C (blackcurrant, rosehip decoction, strawberries), as well as foods rich in potassium salts (unpeeled potatoes, rice, cabbage, apricots, apricots, raisins, oranges, bananas, figs) and etc.). It is expedient to carry out the daily requirement for proteins of animal origin by including raw liver in the diet. Zucchini, carrots, watermelons, melons, cucumbers are recommended. In order to increase the calorie content of food, marmalade, marshmallow, butter and sunflower oil are allowed. Limit the consumption of foods such as meat, eggs, beans, peas, beans, cocoa, halva, cheese. In the nephrotic stage, due to a significant loss of protein in the urine, a diet with a protein content of up to 1.5 g per 1 kg of body weight is recommended. In the presence of edema, table salt is limited, with massive edema up to 2-3 g per day and liquids up to 800-1000 ml, taking into account liquid dishes. Fats are allowed in normal amounts.

From the means of pathogenetic therapy of amyloidosis, desensitizing agents are used (diphenhydramine, pipolfen, suprastin, etc.), ascorbic acid, liver preparations and raw liver. For the same purpose, drugs of the 4-aminoquinoline series (delagil, hingamin, rezoquin, chloroquine, plaquenil) are prescribed, which inhibit the formation of mucopolysaccharides and nucleic acids, inhibit the enzyme systems of reticuloendothelial cells, change the content of sulfhydryl groups, i.e., affect some links of pathogenesis amyloidosis by reducing the synthesis of amyloid. These drugs are prescribed at 0.25-0.5 g per day after meals for many months. However, it should

be remembered that with prolonged use, side effects such as dyspepsia, allergic skin reactions, leukopenia, corneal clouding, and increased hematuria may develop.

Unitiol is also used as a therapeutic agent - a 5% solution of 5 ml intramuscularly, 1 time per day. The course of treatment is 30-40 injections. It is believed that it causes inhibition of the aggregation of amyloid protein substances in fibrillar structures and has a competitive effect with respect to SH-groups. This idea is based on experimental data (V. S. Rukosueva, 1975), according to which the body has a special factor that stimulates the production of a special soluble protein, the precursor of amyloid, by the cells of the reticuloendothelial system. Further transformation of this protein into amyloid fibrils occurs with the participation of disulfide bonds, which are affected by unithiol.

In the treatment of a periodic disease of the nephropathic type, complicated by amyloidosis, colchicine (colhamin) is recommended at a dose of 0.5-0.2 mg per day for 4-6 months. This drug prevents attacks of periodic illness and reduces proteinuria.

Currently, glucocorticosteroid hormones are not recommended for the treatment of amyloidosis. Anabolic steroids (nerobol, methandrostenolone, dianabol, etc.) have a therapeutic effect mainly through their positive effect on nitrogen metabolism. Their clinical effect is manifested by an increase in appetite, an increase in body weight, and an improvement in the general condition of patients.

Levamisole has an immunostimulating effect, in particular, it stimulates humoral and cellular immunity; in the experiment delays the progression of amyloidosis. It is recommended to take levamisole 150 mg 3 times a week. However, it is prescribed in combination with other drugs that increase amyloid resorption, since it cannot change the course of the disease on its own.

It has been established that liver preparations and raw liver prevent the development of amyloidosis in the experiment and give a good clinical effect. Therefore, in case of amyloidosis, it is recommended to take raw liver for a long time (years), and in case of poor tolerance - after cooking. The liver contains a powerful antioxidant system, so the positive effect is probably associated with the introduction into the body of a complete set of antioxidants close to endogenous (2 ml of sirepar corresponds to 40 g of raw liver). Treatment is carried out according to the following scheme: raw liver intake 1-2 months (100-150 g per day), 2-3 months intramuscular injections of sirepar 5 ml 2 times a week, 2-3 months liver intake after cooking, liver month " cocktail", and repeat the cycle again.

There are reports in the literature about the successful use of chronic hemodialysis and kidney transplantation in the end stage of renal amyloidosis. Cases are described when with the help of hemodialysis it was possible to prolong the life of patients up to 4 years or more.

To combat nephrotic syndrome, symptomatic agents are used, such as transfusion of native and dry plasma, plasma albumin, and diuretics are prescribed. In the absence of the effect of conventional diuretics, osmouretics are used - a 20% solution of mannitol, 200-400 ml, polyglucin, 500 ml daily. With the appearance of heart failure, cardiac glycosides are prescribed.

Patients with amyloidosis of the kidneys should be constantly under dispensary supervision. With a favorable course with preserved kidney function, a dispensary examination is carried out 1-2 times a year with the same amount of research as in chronic glomerulonephritis. In the presence of nephrotic syndrome - once a quarter. Sanatorium-resort treatment (Bayram-Ali, Bukhara) is recommended only in the latent and proteinuric stages in the absence of contraindications from other organs and systems.

Fanconi syndrome is a generalized tubulopathy of the proximal type, includes: proximal tubular acidosis with bicarbonaturia; proteinuria tubular type; polyuria; renal glucosuria; phosphaturia; hypophosphatemia; osteomalacia.

Primary Fanconi syndrome is a hereditary disease.

Clinic: physical and mental retardation, malnutrition, bone pain, rickets-like skeletal changes, polyuria, polydipsia, renal glucosuria. Secondary Fanconi syndrome - develops with amyloidosis, hyperparathyroidism, multiple myeloma, etc. Clinic: PU, deformation of the bones of the skeleton, bone pain, fractures, osteoparosis, polyuria, thirst, hypokalemic myasthenia gravis. Postrenal (extrarenal, false) Proteinuria is caused by the ingress of protein-rich inflammatory exudate into the urine in diseases of the urinary tract (prostatitis), with the decay of sperm, with prolonged stagnation of urine.

In terms of magnitude, this is insignificant (up to 1.0 g / day).

Minimal proteinuria - Loss of protein from 300 mg to 1 g / day. It is noted in: obstructive uropathy, tubulopathy, nephrolithiasis, polycystosis,

chronic interstitial nephritis.

Moderate PU (from 1 g / day to 3 g / day) with:acute urinary tract infection,

vesicoureteral reflux, acute tubular necrosis, chronic interstitial nephritis with arterial hypertension, hepatorenal syndrome, transplant rejection, primary and secondary glomerulonephritis (without nephrotic syndrome), proteinuric stage of amyloidosis,

High or nephrotic proteinuria (more than 3 g/day)

In the case of a combination of high proteinuria with hypoalbuminemia, they speak of an incomplete nephrotic syndrome.

If hyperlipidemia, hypoalbuminemia, dysproteinemia, edema and proteinuria more than 3.5 g/day are detected, they speak of a complete nephrotic syndrome.

According to the composition, selective and non-selective proteinuria is distinguished.

Selective proteinuria is the urinary excretion of a low molecular weight protein, mainly albumin. With highly selective proteinuria, low molecular weight fragments of albumin and globulin are detected in the urine. Histological studies of the kidneys reveal minimal changes.

With the selective type, the same proteins plus haptoglobins are determined.

Hematuria (erythrocyturia) - this urinary excretion of erythrocytes is more than the physiological norm, namely, more than 1 erythrocyte in the field of view of the microscope when examining the morning portion of urine after an appropriate toilet.

More precisely, hematuria is determined using quantitative methods for assessing the cellular composition of urine:
tryNechiporenko (more than 1 thousand erythrocytes in 1 ml of urine),
Addis-Kakovsky (more than 1 million erythrocytes per day).
According to the intensity of erythrocyte secretion, there are: ■ microhematuria (up to 100 erythrocytes in the field of view) ■ macrohematuria (over 100 erythrocytes in the field of view) By its nature, hematuria is divided into: ■ initial (at the beginning of the act of urination) ■ terminal (at the end of the act

Causes of hematuria:
Diseases of the parenchyma of the kidneys:
A. Glomerular diseases:
1. Primary: acute and chronic glomerulonephritis (including alcoholic), IgA - nephritis-Berge's disease;
2. Secondary: with SLE, hemorrhagic vasculitis, Wegener's granulomatosis, periarteritis nodosa, chronic active hepatitis, etc.
B. Infectious (infective endocarditis).
B. Tumors of the parenchyma of the kidneys (malignant and benign).
G. Hereditary diseases (Alport's syndrome).
Anomaly in the development of renal vessels (asymptomatic hematuria, due to renal venous hypertension, possible arteriovenous shunts, vasculitis).
3. Nephroptosis.
4. Extrarenal causes of hematuria (blood diseases, coagulation defect, DIC, overdose of anticoagulants, in the treatment of cytotoxic drugs, as well as systemic disease, usually manifested by hematuria, proteinuria, possible erythrocyte cilindruria).
Painful form of hematuria: - kidney injury, urolithiasis, sickle cell anemia, polycystic disease
Painless form of hematuria: glomerulonephritis (in this case, it can be combined with PU and CU); tumors.
Pain in the side or upper abdomen usually indicates the origin of hematuria from the upper half of the urinary tract. Pain radiating from the abdomen to the external genital organs is noted with obstruction of the ureter by blood clots that form with bleeding kidney cancer or sickle cell anemia, as well as papillary necrosis with diabetes mellitus, tuberculosis, etc.

-isolated hematuriaobserved in IgA nephritis in children and adults under 30 years of age, more often in men, accompanied by dull pain in the lumbar region and may recur on the background of pharyngitis. The level of IgA in the blood rises. - With infective endocarditis in 20% of cases, hematuria is due to the development of glomerulonephritis, 30-60% - kidney infarction. At the same time, hematuria is combined with fever of the correct type, positive blood culture, the formation of

heart disease, thromboembolic complications, splenomegaly, and an increase in ESR.Hematuria (erythrocyturia).

The pathogenesis of hematuria in various diseases of the kidneys and urinary tract varies. So, in patients with glomerulonephritis, the appearance of microhematuria is associated with an increase in the permeability of the basement membranes of the glomerular capillaries, an increase in the diameter of the pores in them, as a result, erythrocytes pass more freely and in excess of the norm per diapedesis through the glomerular filter. In severe and violent course of acute and exacerbation of chronic glomerulonephritis, severe hematuria (sometimes in the form of macrohematuria) may be due to a rupture in certain parts of the walls of the glomerular capillaries and the flow of blood from them into the cavity of the Shumlyansky-Bowman capsule, and then into the lumen of the tubules, where erythrocytes are not reabsorbed and are excreted in large quantities in the urine. In urolithiasis, the appearance of hematuria is associated with damage to the mucous membrane of the ureter or bladder by a calculus, especially if it has an uneven surface and sharp edges. In patients with kidney tuberculosis, with tumors of the kidneys and bladder, it occurs due to the destruction of the kidney tissue and damage to the vessels located in it. Its development in capillary toxicosis is due to an increase in the fragility and permeability of the wall of the glomerular capillaries.

Hematuria is short-term and transient, may appear periodically and have an intermittent course (intermittent hematuria). It can be persistent, stubborn, persist for many months and years. These features of hematuria are of great diagnostic value and should be assessed in conjunction with other clinical and laboratory findings when making a diagnosis.

Leukocyturia. Under the leukocyturia understand the excretion of leukocytes in the urine, the number of which exceeds the norm: in the general analysis of urine - more than 5-6 in the field of view, in the study of urine according to Nichiporenko - over 2.5-106 / l, and in the sample of Kakovsky - Addis - more than 4 ,0-106 days

This is a pathological phenomenon characterized by excessive (excessive) excretion of red blood cells in the urine. Like proteinuria, hematuria is one of the most important and common signs of various diseases of the kidneys and urinary tract. It almost always occurs in acute and chronic glomerulonephritis and is one of the most important diagnostic criteria for this disease. In many cases of glomerulonephritis, hematuria occupies a leading place among other clinical and laboratory signs, and sometimes it can be the only manifestation of this disease, which gives reason to distinguish the hematuric form in the clinical classification of glomerulonephritis.

In some classifications (G. Mazhdrakov, 1980, etc.), so-called primary hematurias are distinguished as independent nosological forms, in particular chronic recurrent hematuric nephritis (focal hematuric nephritis or benign hematuric nephritis), idiopathic recurrent hematuria with IgA-mesangial deposits (IgA -nephropathy) or recurrent hematuric syndrome.

Hematuria is a characteristic sign of interstitial nephritis. It can be caused by many drugs, including primarily sulfonamides, streptomycin, gentamicin, as well as analgesics (aspirin, analgin, phenacetin), butadione, etc.

We can talk about hematuria (erythrocyturia) in those cases when the number of erythrocytes excreted in the urine during the day exceeds 2 * 10 and reaches 5-15-100-106 or more. Depending on the intensity of erythrocyte excretion, microhematuria is distinguished, in which the color of urine does not change macroscopically, and macrohematuria, in which urine acquires the color of meat slops or becomes dark red.

With microhematuria, the number of erythrocytes varies from single to 10-15-100 per field of view, sometimes erythrocytes cover all fields of view with a thin layer, but the color of the urine has not yet changed. It is important that the presence of more than 1-3 erythrocytes in the urine in the field of view almost always indicates a pathology in the kidneys or urinary tract or a decrease in blood clotting ability (unless, of course, urine for analysis is collected after a thorough toilet of the external genital organs, especially in women, and if they do not have menstruation during urine collection).

With gross hematuria, erythrocytes cannot be counted and densely cover all fields of view under a microscope. Gross hematuria is most common and persists for a long time in tumors of the kidneys and bladder. It may be a consequence of the tuberculous process in the kidneys, but it is unstable, transient; observed with hemorrhagic vasculitis, an overdose of anticoagulants, during or after an attack of renal colic as a result of damage to the ureteral mucosa by a calculus, less often found in patients with acute and chronic glomerulonephritis and pyelonephritis.

Detection of LN always indicates the presence of an inflammatory process in the kidneys or urinary tract: tubulointerstitial nephritis, pyelonephritis, rejection of a transplanted kidney, urinary tract infection, cystitis, etc. Leukocyturia is also accompanied by prostatitis, kidney tuberculosis, hydronephrosis, urolithiasis and other urological diseases of the kidneys and urinary tract.

Cylindruria - this is the excretion of cylinders with urine, which are a “cast” formed in the lumen of the tubules from protein or cellular elements. Depending on which particles and in what quantity cover the protein cast of the cylinder, there are:

Hyaline -in all kidney disease, accompanied with proteinuria, is composed of clotted whey protein.

Grainy -are formed from degenerated cells of the epithelium of the proximal tubules.

waxy -are formed in the lumen of the distal tubules as a result of the death (dystrophy, atrophy) of the tubular epithelium of these departments.

Erythrocyte -with severe hematuria of various origins.

Leukocyte -with pyuria in patients with acute and especially purulent hr. pyelonephritis in the acute phase, etc.

Pigmented -with various kinds of hemoglobinuria (exposure to toxic substances, etc.).

List of used literature:

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LECTURE TOPIC: "ARRYTHMIA SYNDROME. DIFFERENTIAL DIAGNOSIS. TREATMENT. SUDDEN CORONARY DEATH. ETIOLOGY. PREVENTION. "

TRAINING TECHNOLOGY

Number of students -	Time - 2 hours
Form of the lesson	Lecture - visualization
Lecture plan	13. The conduction system of the heart. Definition of the concept of arrhythmia, blockade 14. Causes and conditions leading to arrhythmias and blockades 15. Classification of arrhythmias 16. The concept, definition and clinical course of various types of arrhythmias and blockades 17. Features of the treatment of various types of blockades and arrhythmias, GP tactics, emergency care
The purpose of the lesson: to familiarize students with the etiology, pathogenesis of diseases accompanied by the occurrence of arrhythmias and blockades, the modern classification of arrhythmias, to teach the principles of differential diagnosis and emergency care for various types of arrhythmias	
Pedagogical tasks: **1.Strengthen and deepen students' knowledge of diseases accompanied by arrhythmias and blockades** **2. To teach students to correctly establish a diagnosis in accordance with the modern classification** **3. Teach students the ability to differentiate different types of arrhythmias**	The results of the educational process: The GP needs to know: 1. The structure of the conduction system of the heart 2. Diseases accompanied by the occurrence of blockades and arrhythmias 3. Classification of arrhythmias 4. Principles of differential diagnosis of various types of arrhythmias 5. Tactics of management, principles of treatment of patients with various types of arrhythmias

4. Educate students on management, emergency care and treatment patients with arrhythmias	
Teaching methodology	Lecture text, videos, questionnaires, questions, “yes-no” technique
Form of study	Laser projector, visual materials, special technical equipment, display of thematic patients, ECG of patients
Means of education	Team
Conditions for the educational process	Audience

TECHNOLOGICAL CARD LECTURES

Stages, time	Activity	
	Teacher	students
Stage 1 Introduction (5 minutes)	1. Tells about the topic of the lecture, its purpose and plan	1. Listen
Stage 2 Knowledge update (20 minutes)	2.1. In order to increase the actualization of students' knowledge, asks questions: 1. Tell the structure of the conduction system of the heart 2. List the diseases accompanied by blockades and arrhythmias 3. List the groups of drugs used to treat blockades and arrhythmias Conducts a survey 2.2. Showing on the screen invites students to familiarize themselves with the goals and objectives of the lecture. Slide №1, №2	2.1. Answer questions asked 2.2. Study slide number 1 2.3. Study slide number 2
Stage 3 Main part (informational) (55 min)	3.1. Introduces students to the lecture material, the importance of the topic and the principles of the formation of an intelligent cultural personality, in particular the GP. In order to increase the actualization of knowledge, he conducts a quick survey of students:	3.1. Together they analyze the listened lecture material, ask questions

	According to 1 point of the lecture plan: tell the structure of the conduction system of the heart According to paragraph 2 of the lecture plan: list the diseases accompanied by the occurrence of blockades and arrhythmias On the 3rd point of the lecture plan: tell the classification of arrhythmias According to the 4th point of the lecture plan: list the differential diagnostic signs of various types of arrhythmias According to the 5th point of the lecture plan: tell the tactics of management, principles of treatment of patients with various types of arrhythmias Stopping at the important points of the lecture, he suggests writing down the main points in a notebook	Key points are written in a notebook.
<u>Stage 4</u> <u>Final (10 min)</u>	4.1. Asking questions: 1. List the most common diseases accompanied by arrhythmias and blockades 2. Tell the main diagnostic signs of arrhythmias (atrial fibrillation, extrasystole, blockade) 3. Name the basic principles of treatment, emergency care for various types of arrhythmias 4.2. Gives a task for independent work of students: Examine the ECG signs of blockades	4.1. Answer questions 4.2. Listen, write

ARRHYTHMIAS AND HEART BLOCK. Cardiac arrhythmia and conduction disorders are a large group of transient or permanent heart rhythm disorders, mainly arising from organic lesions of the cardiovascular system. They are caused by violations of the most important functions of the myocardium: automatism, excitability and conduction.

Of the organic lesions of the cardiovascular system, arrhythmias are most often found in ischemic heart disease, myocarditis, cardiomyopathies, heart defects, pathology of large vessels (thromboembolism of the pulmonary artery, aortic

aneurysms and tears, Takayasu's disease), hypertension, pericarditis, heart tumors. Arrhythmias are also observed in endocrinopathies (pheochromocytoma, thyrotoxicosis), drug intoxication (glycosides, catecholamines), acute infectious diseases, anemia and other pathological conditions.

Arrhythmias may be associated with features of the conduction system, as, for example, in cases of Wolff-Pahr Kinson-White syndrome.

Often, arrhythmias develop with electrolyte imbalances, especially potassium, calcium and magnesium.

Sometimes arrhythmias occur under the influence of excessive consumption of coffee, alcohol, smoking, most often with latent myocardial lesions. Some types of arrhythmias can also develop in healthy people in response to physical exertion or nervous tension.

The diagnosis of cardiac arrhythmias is based on clinical and electrocardiographic data. A healthy person is characterized by sinus rhythm.

Sinus tachycardia is diagnosed when the resting heart rate is more than 100 per minute while maintaining the correct sinus rhythm. The main causes are neurosis, thyrotoxicosis, heart failure, myocardial and rheumatic heart disease, intoxication, fever, anemia. In healthy people, it occurs during emotional and physical stress. As extracardiac causes of sinus tachycardia, there may be an imbalance in the tone of the autonomic nervous system with a predominance of sympathicotonia.

Clinically, sinus tachycardia is manifested by a sensation of palpitations, a feeling of heaviness behind the sternum, and sometimes shortness of breath. It, as a rule, gradually begins and gradually comes to an end unlike that at paroxysmal tachycardia. In ischemic heart disease, sinus tachycardia can cause anginal pain due to an increase in myocardial oxygen demand.

Diagnosis of sinus tachycardia is carried out according to ECG data - the presence of P waves of sinus origin, which precede each QRS complex, with a duration of the P-P interval less than 0.6 s and according to the results of vagal tests, which cause a gradual slowdown in the tachycardia rhythm, and in the case of paroxysms, abruptly break off attack or are ineffective.

In cases of severe sinus tachycardia, the duration of the electrical systole of the ventricles (Q-7) often decreases, the ST segment may shift below the isoline.

Treatment is aimed at eliminating the underlying cause: anemia, fever, thyrotoxicosis, etc. If tachycardia itself serves as a pathogenetic factor, for example, in angina pectoris, myocardial infarction, β-adrenergic receptor blockers are prescribed (propranolol orally, 10-40 mg every 6 hours or atenrlol 25-50 mg 2 times a day), calcium ion antagonists, verapamil groups (Isoptin, verapamil 40-80 mg 2-3 times a day). Often, sinus tachycardia is eliminated by vagotropic tests.

SINUS BRADYCARDIA is characterized by a slowing of the heart rate of sinoatrial origin below 60 beats per minute. Causes - an increase in the tone of the vagus nerve or a change in the function of the sinus node in a number of infections (influenza, typhoid fever), myocardial infarction (often posterior diaphragmatic), an increase in intracranial pressure, mycosedema, etc. Sinus bradycardia may be a consequence of drug treatment in cases of using β-blockers , quinidine-like drugs, cordarone,

verapamil, tranquilizers. In athletes, the rhythm frequency is in the range of 40-45 beats per 1 minute.

Clinically, it often does not manifest itself. Sometimes patients complain of a rare heart rhythm, weakness, a feeling of "fading" of the heart, dizziness. Excessive bradycardia can cause cerebral ischemia with syncope.

Diagnosed by ECG on the basis of normal sinus rhythm, in addition to a decrease in its frequency, a high peaked T wave is sometimes formed.

The heart rate in sinus bradycardia, in contrast to bradycardia caused by various types of blockades, increases in case of physical activity, injection of atropine.

Treatment in the absence of clinical manifestations is not required. If sinus bradycardia causes hemodynamic disturbances and other clinical manifestations, atropine (0.5–2.0 mg IV or s/C), isoproterenol (1–4 μg/min IV infusion) are prescribed. With mild bradycardia, belladonna preparations can be used. In the case of severe sinus bradycardia and the absence of the effect of drug treatment, pacing is performed.

SINUS ARRHYTHMIA is an irregular sinus rhythm characterized by a fluctuating frequency. Small fluctuations in frequency (the value of the RR intervals up to 0.1 s) are physiological and are usually associated with the act of breathing: when inhaling, the rhythm quickens somewhat, while exhaling, it slows down. Sinus arrhythmia, not associated with respiratory phases, indicates autonomic dysfunction or cardiovascular pathology. The difference between the RR intervals in such cases is 0.12 s or more.

Sinus arrhythmia in most cases does not cause discomfort, since it does not have a significant effect on hemodynamics, except when it is combined with severe sinus bradycardia. Diagnosis is by ECG based on normal sinus rhythm with a difference in R-R or R-R intervals. Of auxiliary importance for diagnosis is the disappearance of sinus arrhythmia after holding the breath and, conversely, the increase in arrhythmia against the background of deep breathing.

Special treatment for this type of arrhythmia is not required.

MIGRATING SUPRAVENTRICULAR RHYTHM is characterized by arrhythmia with different shape and polarity of the P waves, different duration of the P-R interval. It is based on the displacement of the source of impulse formation within the conduction system of the atria or from the sinoatrial node to the area of the atrioventricular junction or, conversely, the unequal rate of diastolic depolarization in the sinoatrial node, in specialized cells of the atria and atrioventricular junction.

When the tone of the vagus nerve changes, a migrating rhythm can occur in healthy people. In patients with organic heart diseases (myocarditis, heart defects, coronary heart disease), the migratory rhythm seems to be the result of the activation of the ectopic rhythm.

Clinically, the migration of the supraventricular rhythm is usually not manifested. The diagnosis is established using an ECG study: P waves of sinus origin alternate with right-left atrial teeth and precede the QRS complex; the value of the R-R intervals ranges from 0.12 to 0.20 s.

Treatment is directed at the underlying disease.

Rhythm of the atrioventricular junction (NODAL RHYTHM) occurs when the automatism of the sinoatrial node is suppressed and the impulse propagates retrogradely from the atrioventricular junction. As a result, a negative P wave is recorded on the ECG. It precedes the QRS complex, appears simultaneously with it or after it. Such a rhythm is more often recorded with organic pathology of the heart (myocarditis, coronary heart disease, myocardiopathies), as well as with intoxication with certain medications (glycosides, reserpine, quinidine, etc.). However, sometimes nodal rhythm can be periodically observed in healthy individuals with severe vagotonia.

clinical picture. Nodal rhythm in patients with heart disease can exacerbate the severity of their condition. Healthy people, as a rule, do not notice it. The rhythm of the atrioventricular connection is diagnosed only according to ECG data, in the presence of 3 or more nodal impulses in a row. The pulse rate with this rhythm is within 40-65 per 1 min.

Treatment of the underlying disease.

Extrasystole - premature contraction of the whole heart, only the atria or ventricles, caused by an impulse that occurs outside the sinus node. Accordingly, depending on the place of development, atrial, ventricular and outgoing extrasystoles are distinguished from the atrioventricular junction. The cause of extrasystole is inflammatory, dystrophic, sclerotic processes in the myocardium, lesions of the valvular apparatus of the heart, coronary artery disease, intoxication. Extrasystole also occurs with reflex effects from other organs (biliary and urolithiasis, diaphragmatic hernia, peptic ulcer, stomach disease, etc.).

Depending on the time of appearance, it is customary to distinguish between early, middle, late extrasystoles. Depending on the frequency, there are rare (5 or less per 1 min), medium (from 6 to 15) and frequent (more than 15 per 1 min). A group of two extrasystoles is called a steam room, of 3 or more - a paroxysm of tachycardia.

Early extrasystoles of type L to G are unfavorable in prognostic terms. Multiple, group (several extrasystoles occur in a row) and polytopic extrasystoles, indicating significant changes in the myocardium, should be included in this category.

clinical picture. Usually, with extrasystole, patients complain of a feeling of interruptions in the work of the heart, tremors and fading behind the sternum. In the case of prolonged allorhythmia (bigeminia, trigeminia), such complaints are often absent. In a number of patients, increased fatigue, shortness of breath, dizziness, and general weakness come to the fore.

On physical examination, extrasystole is defined as a premature beat followed by a compensatory pause.

Extrasystoles are diagnosed on the ECG by the premature appearance of an extrasystolic complex. At the same time, supraventricular extrasystoles have an unchanged form of the ventricular complex and an incomplete compensatory pause. With atrial extrasystole, a somewhat deformed P wave is sometimes noted. Extrasystoles from the atrioventricular connection, due to the retrograde propagation of the impulse to the atria, have a negative P wave. Ventricular extrasystoles are characterized by deformity, high amplitude of the ventricular complex, a width

exceeding 0.12 s, and a complete compensatory pause. The largest extrasystole wave is discordant to the ST segment, as well as to the T wave.
Interpolated (inserted) ventricular extrasystoles occur between 2 normal contractions, while the extrasystole appears very early.
Atrial extrasystoles and outgoing from the atrioventricular connection are called supraventricular.
The appearance on the ECG of extrasystoles with a different form of the ventricular complex (polytopic) indicates several ectopic foci. Polytopic and multiple extrasystoles are inherent in organic damage to the myocardium.
Differential diagnosis with ventricular extrasystoles is based on the presence of a deformed P wave in supraventricular extrasystoles and the absence of a QRS complex.
With supraventricular extrasystole, the P wave may become biphasic or negative, be in front of the OLU complex (with an impulse from the atrioventricular lead), and may also merge with the ORS complex. The occurrence of an extrasystole after each beat is called "bigeminy", after every second - "trigeminy", etc.
The appearance of monofocal extrasystoles such as bigeminia is more often observed against the background of sinus bradycardia. Polytopic (polyfocal) extrasystoles are observed in most cases in violation of electrolyte metabolism and acid-base balance.
Right ventricular extrasystoles are characterized by a high jagged R1-5 wave in the chest leads. With left ventricular extrasystole, there is a high RV in the right chest leads, deep SV in the left chest leads. To register episodically appearing extrasystoles, as well as extrasystoles that are paroxysmal in nature, Holter monitoring is most effective. In the case of using a conventional ECG for these purposes, the probability of registering extrasystoles increases when they are provoked by a Valsalva test, physical activity, in particular bicycle ergometry.
Treatment of extrasystoles is indicated in case of violation of the patient's well-being under its influence when it affects hemodynamics and with unfavorable prognostic extrasystoles that can lead to fatal arrhythmias (ventricular fibrillation or asystole).
Asymptomatic atrial extrasystoles without signs of sustained atrial tachycardia (that is, with a duration of paroxysm of less than 2 minutes) do not require antiarrhythmic therapy, except in cases of treatment of the underlying disease or elimination of provoking factors. It is necessary to exclude the influence of external arrhythmogenic factors (strong tea, coffee, smoking, drinking alcohol, the use of certain drugs - ephedrine, aminophylline, asthmapent, etc.).
With the development of extrasystole against the background of tachycardia and arterial hypertension, blockers of p-adrenergic receptors such as propranolol (anaprilin, inderal, obzidan 40-80 mg 2-3 times a day), atenolol (tenormin) 50-100 mg 2 times a day are indicated.
Atrial extrasystoles are best eliminated with antiarrhythmic drugs of class 1a (rhythmilen 100-200 mg 3 times a day, novocainamide 250-500 mg 3 times a day) and 1c (rhythm-norm 150-300 mg 3 times a day, etacizin 500 mg 3 times a day, allapinin 25 mg 3 times a day).

If there was a history of paroxysms of atrial fibrillation or atrial flutter during atrial extrasystole, drugs that depress AV conduction (digoxin, β-blockers, verapamil) should be simultaneously prescribed to slow down the contraction of the ventricles in case of paroxysm.

In cases of ventricular extrasystole, preference should be given to beta-blockers and class III antiarrhythmic drugs: amiodarone, cordarone at an initial dose of 600 mg per day in 3 doses, followed by a dose reduction of 200 mg every 5-6 days and a transition to a maintenance dose of 200 mg per day and sotalol 80-120 mg 2 times a day.

For emergency treatment of ventricular extrasystoles (with myocardial infarction), it is best to use intravenous administration of lidocaine or trimecaine at 40-120 mg (initially intravenously in a stream for 2-3 minutes, and then drip at a rate of 1-2 mg per 1 minute).

If there is no effect from individual drugs, several antiarrhythmic drugs are combined. The following combinations have been justified and tested in the clinic: cordarone, 100-200 mg 2-3 times a day + rhythmylen, 100 mg 2-4 times, or + ethacizin, 50 mg 2-3 times, or + ethmozine, 100 mg 2-3 times times; rhythmilene, 100 mg 3 times a day + ethmosin, 100 mg 3 times, or + allapinin, 25 mg 1-2 times, or + mexityl, 200 mg 2 times a day.

In the complex therapy of extrasystoles, it is advisable to include potassium and magnesium preparations (panangin or asparkam, 2 tablets 3 times after meals).

PAROXYSMAL TACHYCARDIAS are attacks of rapid heartbeat, usually from 140 to 220 beats per minute, with a sudden onset and end. An attack can last from a few seconds to hours or many days.

Different supraventricular paroxysmal tachycardia and ventricular. The first includes atrial and atrioventricular (AV) forms. The frequency of contractions - 200-300 per 1 min corresponds to flutter, and more than 300 - atrial fibrillation.

Supraventricular paroxysmal tachycardias are characterized by a regular rhythm and an unchanged ventricular complex in the absence of intraventricular blockade. According to the mechanism, ectopic and reciprocal (recurrent reentry type), atrial and AV tachycardias are distinguished.

Ventricular paroxysmal tachycardias, originating in the contractile myocardium of the ventricles or in the Purkinje fibers and bundle pedicles, occupy a special place, since they tend to go into ventricular fibrillation and to the appearance of severe hemodynamic disorders, including arrhythmogenic shock and pulmonary edema.

The reasons for the development are the same as for extrasystoles. Ventricular tachycardia can sometimes be the result of arrhythmogenic right ventricular dysplasia and digitalis intoxication.

clinical picture. During a paroxysm, patients feel a frequent heartbeat, often starting with a sharp push behind the sternum. In many cases, palpitations are accompanied by shortness of breath, pain in the region of the heart or behind the sternum, dizziness, and weakness. Arterial pressure decreases somewhat, and increases with sympathoadrenal crises. Such crises are also characterized by a feeling of fear, chills, frequent urination, lack of air. During an attack, patients are frightened, motor

restlessness is observed. The jugular veins are swollen, pulsate synchronously with the arterial pulse.

Auscultation reveals an equalization of the intensity of the I and II heart sounds, the pauses between the tones become the same (“pendulum rhythm”).

Long-term paroxysmal tachycardia can lead to heart failure, usually refractory to drug therapy. Heart failure develops especially rapidly with nodular and ventricular paroxysmal tachycardia due to a violation of the synchrony of the atria and ventricles. Against the background of an attack, signs of myocardial ischemia (decrease in the S-T interval) are often detected.

Diagnostics. The main method is electrocardiography. Informativeness increases with the use of transesophageal ECG recording, which makes it possible to identify the shape and localization of the atrial P wave. In cases of rare and short attacks, the diagnosis improves if daily ECG monitoring is used. The electrocardiographic signs of paroxysm of ventricular tachycardia include: the expansion of QRS complexes more than 0.12-0.14s against the background of tachycardia from 120 to 200 contractions per 1 min; following the P waves in a more rare sinus rhythm (better detected on the esophageal ECG); the phenomenon of complete and partial capture of the ventricles. With left ventricular tachycardia, the QRS complexes have the appearance typical of blockade of the right leg of the bundle of His, and with right ventricular tachycardia - for the blockade of the left leg.

Treatment. With paroxysm of supraventricular tachycardia, vagal tests are used: 1) massage of the carotid sinus, first on the right - 1-20 s, in the absence of effect - on the left; it is done carefully, while the activity of the heart is controlled (auscultatively or by ECG); the test should not be used in elderly patients, since cerebral circulation may be impaired (massage is contraindicated in the presence of noise on the carotid arteries and in violation of cerebral circulation); 2) moderate pressure on the eyeballs for a few seconds; 3) artificial induction of vomiting; 4) Valsalva test (deep breath with maximum exhalation with a pinched nose, closed mouth).

If there is no effect, the most effective is verapamil (finoptin, isoptin) intravenously slowly in a stream - 0.25% solution, 4 ml (10 mg), it is also possible to re-administer it after 20 minutes at the same dose (it is not recommended to administer verapamil while taking r blockers). A 1% solution of adenosine triphosphate (ATP) intravenously in a stream of 2-3 ml also has a fairly high efficiency.

With attacks of supraventricular tachycardia, blockers of p-adrenergic receptors are often used (intravenously slowly). Obzidan is administered at 1 mg over 1-2 minutes in a total dose of 3-10 mg (you must have a syringe with mezaton at the ready); cardiac glycosides are injected slowly in a stream with a 5% glucose solution or an isotonic sodium chloride solution (strophanthin - 0.25-0.5 ml, corglicon - 0.5-1 ml), aimalin -2.5-2 ml intravenously slowly for 5 min (to avoid severe complications), novocainamide intravenously slowly in a total dose of 0.5-1 g (in the absence of blockade of the bundle of His bundle and cardiac decompensation), cordarone - 300-450 mg

intravenously slowly in isotonic solution. Etmozin and etatsizin, as a rule, are used in a hospital, 2 ml of a 2.5% solution in physiological sodium chloride solution, slowly intravenously under the control of blood pressure and preferably an ECG. Combination therapy with beta-blockers and low doses of quinidine can be used. Quinidine is used in the first dose at a dose of 0.2 g, then 0.2 g every 2 hours (total dose - 1.2 g).

In order to prevent recurrence of paroxysms of supraventricular tachycardia, amiodarone (cordaron) and sotalol have justified themselves.

The main agent for the treatment of ventricular paroxysmal tachycardia is lidocaine, trimecaine, which are administered intravenously in a stream at a dose of 120 mg for 30 seconds, then drip at a rate of 2-3 mg / min for 12-24 hours. Lidocaine can also be used intramuscularly at 200- 400 mg. A drop in blood pressure that often occurs with this tachycardia requires the administration of pressor amines (norepinephrine, mezaton), which can help restore sinus rhythm.

A number of other drugs administered slowly intravenously are also effective, in particular, etmozin - 4 ml of a 2.5% solution (100 mg), etacizin - 2 ml of a 2.5% solution (50 mg), mexitil - 10 ml of a 2.5% solution (250 mg), obzidan up to 0.5 mg / kg of body weight, novocainamide, aimalin (giluritmal), cordarone in the previously indicated doses. Cardiac glycosides are contraindicated due to the risk of ventricular fibrillation.

The ineffectiveness of pharmacotherapy serves as an indication for electrical impulse therapy, which is inappropriate only for glycoside intoxication.

In case of paroxysmal ventricular tachycardia due to intoxication with cardiac glycosides, potassium preparations are used intravenously (panangin - 10-80 ml, difenin - 0.1 g 3 times a day, etmozine). It is advisable to correct hypomagnesemia, for this magnesium sulfate is prescribed - 4 ml of a 25% solution in 50-100 ml of isotonic sodium chloride solution intravenously.

For the prevention of ventricular tachycardia, the following drug therapy is necessary: cordarone 0.2 g 3 times a day, orally, or propafenone, 150-300 mg 2-3 times a day, orally, or novocainamide, 0.5 g 4-6 times a day, orally , or aymalin 50-100 mg 3 times a day orally, or disopyramide 0.2 g 3 times a day orally, or etacizin 50 mg 3 times a day orally. treatment.

Atrial fibrillation (Atrial fibrillation and flutter) is characterized by the presence of very frequent (more than 350 per 1 min) irregular (with flutter - regular) atrial impulses, leading to uncoordinated contractions of individual muscle fibers. In terms of prevalence, it ranks second after extrasystole. With this violation of the rhythm, there is no effective contraction of the atria. Frequent and irregular series of electrical impulses enter the ventricles, most of them are blocked in the atrioventricular junction, but often reach the ventricular myocardium, causing arrhythmic contractions.

With atrial flutter to the ventricles, every second, third impulses can be carried out - the so-called regular form of atrial flutter. If the conductivity of the atrioventricular connection changes, then the ventricles contract arrhythmically, as in atrial fibrillation.

Atrial fibrillation can be constant and paroxysmal. It is customary to distinguish between brali-, normo- and tachysystolic forms of atrial fibrillation, in which the heart rate at rest is 60 or less, 61-90 and more than 90 per minute, respectively.

Atrial fibrillation occurs against the background of various organic heart diseases: in the elderly against the background of coronary heart disease, in young people against the background of rheumatism with damage to the valvular apparatus of the heart or with congenital heart defects, myocarditis, myocardiopathy, thyrotoxicosis.

Clinical picture and diagnosis. The sensations of the patient and the violation of hemodynamics during atrial flutter largely depend on the form of atrioventricular conduction. When conducting 2:1 or 1:1 (rarely), a strong heartbeat, weakness, and cardiovascular insufficiency increase. The patient may not even notice the appearance of the 3:1 and 4:1 forms.

With atrial flutter, F waves are detected on the ECG, located at equal intervals close to each other. They are of the same height and width, their frequency is 200-350 per 1 min. The shape and width of the ventricular complexes are usually normal. The most common is atrioventricular block of varying degrees, and it is not always possible to establish the presence of one of the pair of atrial complexes due to its overlaying on the ventricular complex. In such a situation, atrial flutter can be mistaken for paroxysmal atrial tachycardia.

With atrial fibrillation, hemodynamic disturbance is due to the lack of coordinated contraction of the atria and ventricles due to arrhythmia. It has been established that in such a situation, the minute volume of the heart drops by 20-30%.

Subjective sensations of the patient depend on the frequency of contractions of the ventricles and their duration. With tachycardia (100-200 contractions per 1 min), patients complain of palpitations, weakness, shortness of breath, fatigue. In cases of bradyarrhythmic form (less than 60 contractions per 1 min), dizziness and fainting are noted. In the normoarrhythmic form (60-100 contractions per 1 min), there are often no complaints.

During the examination of the patient, an arrhythmia of heart contractions with varying intensity of tones and pulse waves, a deficit of pulse waves in relation to the heart rate are detected.

There are no P waves on the ECG, instead of them, continuously changing in shape, duration, amplitude and direction of the wave are determined. The distance between the QRS complex is not the same. Flutter waves are most clearly seen in lead V1.

Treatment. For the relief of paroxysmal atrial fibrillation, cardiac glycosides, p-blockers, novocainamide, verapamil (finoptin, isoptin), ethmozine, ethacizine, aymalin, quinidine are used. Cardiac glycosides are administered intravenously slowly in a stream in a 5% glucose solution or isotonic sodium chloride solution (0.05% solution of strophanthin - 0.25-0.5 ml, corglicon - 0.5-1 ml), obzidan 1 mg for 1 -2 min, total dose - 3-10 mg; at the same time, it is necessary to have a syringe with mezaton; when administered, control blood pressure. You can also use aymalin (2.5% solution - 2 ml intravenously slowly over 5 minutes), or novocainamide intravenously slowly in a total dose of 0.5-1 g (condition: no blockade of the bundle of His bundle and severe heart failure), or cordarone 6-9 ml (300-450 mg) without

dilution intravenously for 5-10 minutes. Verapamil (finoptin, isoptin) is administered at a dose of 5-10 mg intravenously by bolus, etmozin and ethacizin (usually in a hospital) - 2 ml of a 2.5% solution intravenously by bolus slowly or drip in isotonic sodium chloride solution. You can use quinidine (0.2 g every 2 hours, the total dose is 1.2 g). Combined therapy with β-blockers and cardiac glycosides, β-blockers and small doses of quinidine is also prescribed.

If there is no effect from pharmacotherapy, electrical impulse therapy is used.

With a constant form of atrial fibrillation, the therapeutic tactics are determined by the nature of the organic pathology of the heart, the severity of heart failure, and the heart rate.

In the case of normo- and bradysystolic forms of atrial fibrillation, the absence of cardiac decompensation, the underlying disease is treated, antiarrhythmic drugs are not used. In the tachysystolic form, treatment is aimed at slowing the heart rate or restoring sinus rhythm. Cardiac glycosides (digoxin, isolanide) are prescribed for oral administration in individually selected doses (with outpatient treatment - 1/2 tablet 3 times a day) under the control of heart rate, pulse deficit and ECG parameters. These drugs are used in combination with potassium preparations (panangin, asparkam, etc.). If necessary, a small dose of a β-blocker (trazicor, propranolol) is added.

As an antiarrhythmic agent, quinidine can be used after a trial dose (0.2 g every 2-2.5 hours) under ECG monitoring. When sinus rhythm is restored, maintenance therapy is subsequently prescribed (0.2 g every 6 hours). If the rhythm is not restored within 3-5 days under conditions of treatment with quinidine, the drug is canceled. You can try to restore the rhythm with Sotalex: 80-160 mg 2 times inside. If there are no contraindications, aspirin is prescribed at a dose of 0.1 g 1 time per day.

SYNDROME OF WEAKNESS (DYSFUNCTION) OF THE SINUS NODE (SYNDROME OF BRADY- AND TACHYCARDIA) is characterized by alternating periods of bradycardia and tachycardia, occurs due to a decrease in the number of specialized cells in the sinus node, proliferation of connective tissue. Organic changes in the myocardium play a role in the development of sick sinus syndrome (SSS) (with myocarditis, rheumatic heart disease, valvular heart disease, coronary disease, cardiomyopathy, etc.); intoxication with cardiac glycosides, quinidine; household poisoning with chlorophos, karbofos, poisonous mushrooms. Congenital or hereditary inferiority of the sinus node (idiopathic SSS) occurs in 40-50% of cases.

Clinical manifestations of SU dysfunction include dizziness, short-term loss or confusion of consciousness, darkening of the eyes, staggering, fainting (50-70% of cases), constant weakness, and fatigue. With the syndrome of bradycardia-tachycardia, the risk of intracardiac thrombi and thromboembolic complications increases, among which ischemic strokes are not uncommon. The extreme manifestations of SU dysfunctions are Morgagni-Adams-Stokes (MAC) attacks and sudden death.

Syncopal conditions caused by MAC attacks are characterized by suddenness, absence of pre-syncope reactions, severe pallor at the time of loss of consciousness

and reactive hyperemia of the skin after an attack, rapid recovery of the initial state of health. loss of consciousness
occur with a sudden decrease in heart rate less than 20 in 1 min or during asystole lasting more than 5-10 s.
Diagnostics. For SSSU, the following ECG signs are most characteristic:
constant sinus bradycardia with heart rate at rest less than 45-50 in 1 min; SU stop with sinus pauses longer than 2-2.5 s;
sinoauricular blockade with sinus pauses of more than 2-2.5 s;
slow recovery of SU function after electrical or pharmacological cardioversion, as well as in case of spontaneous cessation of an attack of supraventricular tachyarrhythmia (pause before the restoration of sinus rhythm for more than 1.6 s);
alternation of sinus bradycardia (pauses of 2.5-3 s) with paroxysms, atrial fibrillation or atrial tachycardia (bradycardia-tachycardia syndrome).
Holter monitoring is the most informative method for confirming and documenting the relationship between clinical and electrocardiographic manifestations of sinus dysfunction. In the process of evaluating the results of monitoring, the limit values of heart rate should be taken into account. In patients with SSSU, the maximum value of heart rate per day, as a rule, does not reach 90 per 1 min, and the minimum is less than 40 during the day and less than 30 during sleep.
To assess the function of the sinus node, the following tests are used:
1. Test with dosed physical activity (veloergometry, treadmill test, 10-20 squats). In persons with SSSU, the increase in heart rate in response to exercise does not exceed 20% of the initial value. With vegetative dysfunctions of the SU, the HR response is the same as in healthy people. If during exercise the sinus rate increases adequately, reaching 120 beats per 1 min or more, there is no need for special studies of the function of the SU.
2. Test with intravenous administration of atropine sulfate (0.02-0.03 mg/kg). It is necessary to record an ECG every 30 s after the administration of the drug with heart rate control until its maximum increase. In patients with SSSU, the heart rate does not reach 90 in 1 min, and the increase does not exceed 20% of the initial value. Often there is an accelerated escape rhythm from the atria or AV connection lasting more than 30 seconds. In persons with vagal dysfunction of the SU, an increase in heart rate to 90 or more in 1 min is noted.
A valuable method for studying the function of the sinus node is transesophageal EFI (electrophysiological studies). During the procedure, indicators are determined - the time to restore the function of the sinus node (VVFSU) and the corrected time to restore the function of the sinus node (KVVFSU), which normally do not exceed 1600 ms and 525 ms, respectively. An increase in the values of these indicators is characteristic of SU dysfunction.
Treatment. In the early stages of SSSU development, it is possible to achieve a short-term unstable increase in the rhythm by discontinuing drugs that slow down the heart rate and prescribing anticholinergics (atropine in drops) or sympatholytic agents (izadrin 5 mg starting from 1/4-1 /2 tablets, doses are gradually increased to prevent the occurrence of ectopic arrhythmias). In some cases, a temporary effect can be

obtained by prescribing belladonna preparations. In some patients, an effect was noted with the use of nifedipine, nicotinic acid, and in heart failure - ACE inhibitors. The main treatment for SSSU is continuous electrical stimulation of the heart.

FLUTTERING AND VENTRICULAR FIBRILLATION. Flutter - frequent regular activity of the ventricles (more than 250 contractions per 1 min), accompanied by a cessation of blood circulation; flickering (fibrillation) - frequent and erratic activity of the ventricles. In this case, the blood flow stops immediately. With paroxysmal flutter or ventricular fibrillation, syncope, attacks of Morgagni-Adams-Stokes occur. As a rule, this is a terminal arrhythmia in most patients who die from various serious diseases. The most common cause is acute coronary insufficiency.

Clinic and diagnostics. Since the onset of trembling or flickering of the ventricles, the pulse disappears, heart tones are not heard, blood pressure is not determined, the skin becomes pale with a bluish tint. Within 20-40 seconds, the patient loses consciousness, convulsions may appear, pupils dilate, breathing becomes noisy and frequent.

On the ECG during ventricular flutter, regular rhythmic waves are recorded, resembling a sinusoidal curve with a frequency of 180-250 per 1 min. Ri G teeth are not defined. In cases of ventricular fibrillation on the ECG, continuously changing in shape, duration, height and direction of the wave with a frequency of 130-150 per 1 min are observed.

Treatment. It is necessary to carry out electrical ventricular defibrillation, external heart massage as soon as possible, hit hard with a fist in the region of the heart or on the sternum. Electrical defibrillation starts with a maximum voltage of 7 kV (360 J). If there is no effect, the discharges are repeated, in the intervals between them an external heart massage and artificial ventilation of the lungs are done. IV or adrenaline - 0.5-1 mg, calcium chloride - 0.5-1 g, novocainamide - 250-500 mg, lidocaine -100 mg, obzidan - 5-10 mg are administered intracardiac as a bolus. The effectiveness of measures depends on the time after which they are started, and the possibility of electrical defibrillation of the heart.

The prognosis in most cases is unfavorable, especially when fibrillation occurs in patients with severe heart failure, cardiogenic shock.

VENTRICULAR ASYSTOLIA - a complete stop of the ventricles associated with the loss of their electrical activity. Most often this is the outcome of ventricular fibrillation. A straight line is recorded on the ECG.

Treatment differs little from that described. After defibrillation, adrenaline - 0.5-1 mg is administered intravenously as a bolus, then atropine - 1 mg, their administration is repeated every 5 minutes. Sodium bicarbonate should not be administered during cardiac arrest. Temporary electrical stimulation may be used.

SINOATRIAL (SINOAURICULAR) BLOCK (SAB) develop when the impulse from the sinus node (SA) to the atria is disturbed. It can be observed in cases of severe vagotonia, organic heart disease (CHD, myocarditis, cardiomyopathies, intoxication with glycosides, quinidine, hypokalemia).

There are 3 degrees of SAB. At I, the transition time of the impulse from the SA node to the atria is lengthened. It is not detected electrocardiographically.

With a blockade of the II degree, the entire cardiac contraction falls out - there is no P-QRST complex. A pause is recorded on the ECG, equal in duration to the double R-R interval. If a larger number of complexes fell out, then the pause will be equal, respectively, to their total duration. This may cause dizziness, a feeling of irregular activity of the heart, fainting.
The third degree of blockade (complete SAB) is actually asystole: not a single impulse from the SA node is conducted to the atria or is not formed in the sinus node. The activity of the heart is supported by the activation of underlying sources of rhythm.

Treatment. Therapy of the underlying disease. With severe hemodynamic disorders, atropine, belladonna, ephedrine, alupent are used. The appearance of fainting is an indication for cardiac pacing.
Atrioventricular block (AVB) is the slowing down or cessation of the conduction of impulses from the atria to the ventricles. Accordingly, a level of damage to the conduction system can occur in the atria, at the atrioventricular junction, and even in the ventricles. The causes of AVB are the same as for other conduction disorders. However, self-developing degenerative-sclerotic changes in the conduction system of the heart are also known, which lead to AVB in the elderly (Lenegre and Lev's diseases). AVB may be accompanied by a ventricular septal defect, Fallot's tetrad, aneurysm of the membranous part of the septum, etc.
There are 3 degrees of blockade. The first degree is characterized by a prolongation of the atrioventricular conduction time, the P-Q interval is equal to or greater than 0.22 s. At the II degree of AVB, 2 types of blockade according to Mobitz are distinguished. Type I Mobitz - gradual lengthening of the P-Q interval with the loss of one ventricular complex - the Samoilov-Wenckebach phenomenon. In type II Mobitz blockade, the progressive prolongation of the P-Q interval does not precede the prolapse of the ventricular complex. With this type, several ventricular complexes can fall out in a row, which leads to a significant decrease in heart rate, often with Morgagni-Adams-Stokes attacks.
With atrioventricular blockade of I and II degrees with periods of Samoilov-Wenckebach, there are no clinical manifestations. Dynamic monitoring of ECG data is of great importance.
Treatment. With first-degree AV block, if the P-Q interval does not exceed 400 ms and there are no clinical manifestations, no treatment is required. Second-degree Mobitz type I AV block without clinical manifestations also does not require treatment. In case of hemodynamic disturbances: atropine, 0.5-2.0 mg intravenously, then pacing. If AV blockade is caused by myocardial ischemia (the level of adenosine in the tissues increases), then the adenosine antagonist aminophylline is prescribed. In case of AV blockade II degree type Mobitz II, regardless of clinical manifestations, temporary, then permanent pacing (ECS) is indicated.
COMPLETE TRANSVERSE BLOCK (ATRIOVENTRICULAR BLOCK OF THE III DEGREE) is characterized by the complete absence of impulse conduction through the atrioventricular junction from the atria to the ventricles. The atria are

excited from the sinus node, the ventricles - under the influence of impulses from the atrioventricular junction below the site of blockade or from the centers of automatism of the third order. In this regard, the atria and ventricles are excited and reduced independently of each other. In this case, the rhythm of atrial contractions is correct and higher than the number of ventricular contractions.

The number of ventricular contractions depends on the location of the pacemaker. If it reaches (or exceeds) 45 in 1 min, it is considered that the pacemaker is located in the atrioventricular connection (proximal type of blockade). With this type, the impulse path through the ventricles is normal, since the complex *QRS not changed*. R-R distance is constant. Since the atria contract more frequently than the ventricles, the distance *P*-P < R-R.

Complete transverse blockade can be transient or permanent.

The combination of complete transverse blockade with atrial fibrillation or flutter is called the syndrome, or Frederick's phenomenon. With a decrease in heart rate to 20 or less in 1 min, periods of loss of consciousness with convulsions occur due to cerebral ischemia (attacks of Adams-Morgani-Stokes). If timely assistance is not provided, a fatal outcome may occur.

Clinically defined:

1. Audible atrial tones in a long diastolic pause. They are perceived from time to time as deaf sounds, designated "systole-echo".
2. Loud I tone at the top of the heart - cannon tone, described by academician N. D. Strazhesko. This loud I tone is heard regularly with an interval of 4-10 beats. It should be emphasized that the phenomenon of cannon tone is the most important diagnostic sign of complete atrioventricular block.

One of the criteria for a complete transverse blockade is a significant increase in systolic pressure. Physical activity and the introduction of atropine do not increase the heart rate.

ECG indicates that the atria and ventricles are excited independently of each other. With rapid atrial contraction in the correct rhythm, the ventricles contract within 40 times per 1 minute.

Complete atrioventricular block, characterized by pronounced bradycardia, can be acquired and congenital. Often it is asymptomatic, but more often patients are disturbed by dizziness, fainting, sometimes accompanied by convulsions, palpitations are noted only subjectively. First, patients have a pronounced bradycardia, which makes it possible to suspect a complete atrioventricular blockade. Cannon tone is heard. The final diagnosis is established by electrocardiographic data.

With incomplete atrioventricular blockade, only every second or less often every third or fourth atrial impulse is established with the help of an ECG (2:1, 3:1, etc.).

Often it is necessary to carry out a differential diagnosis between a complete atrioventricular block and an incomplete atrioventricular block of the II degree. You can distinguish them by recording an ECG after physical activity of the patient.

or administering atropine to him. With complete atrioventricular blockade, the correct alternation of the P waves and the QRST complex is eliminated, the P wave will occupy a different position in relation to the QRST complex.
With incomplete atrioventricular blockade, the P waves and the QRST complex will be located in the same ligament, the heart rate will be quickened.
Treatment. Permanent electrocardiographic stimulation (EX). If the causes of the blockade are reversible (for example, hyperkalemia), if the blockade occurs in the early postoperative period or with lower myocardial infarction, you can prescribe drugs that enhance the automatism of the ventricles: isadrin -5 mg (under the tongue) or Alupent infusion into a vein - 0.5- 1 ml of a 0.05% solution drip or jet slowly. But most often in such cases it is necessary to resort to a temporary pacemaker, especially with a replacement rhythm with wide QRS complexes.
Intraventricular blockade can develop in any part of the conduction system of the heart distal to the AV junction (from the bundle of His bundle to the Purkinje fibers). Main causes: ischemic heart disease, myocarditis, lesions of the valvular apparatus of the heart, cardiomyopathy. Blockade of the right leg can develop with cor pulmonale.
The main electrocardiographic sign of intraventricular block (IVB) is the widening of the ventricular complex. VZHB are complete when the QRS complex is equal to 0.12 s or wider, and incomplete - (QRS is wider than 0.09 s, does not exceed 0.12 s. With blockades of the branches of the His bundle from the side of the blocked leg, the ventricle is excited later, since the excitation impulse goes around the affected area.In the case of blockade of the left leg (LBN), the right ventricle will be the first to be excited, and in the case of blockade of the right leg (RBL) - the left ventricle.
Thus, along with an increase in the activation time of the ventricles, the usual course of excitation of the ventricular myocardium changes, which causes a significant deformation of the ventricular complex.
With complete LBBB on the ECG in leads V5_6, the QRS complex is represented by a wide R wave with a notch at the top or knee (ascending or descending). In V,_2 ventricular complexes have the form of QS with a wide tooth S. The electrical axis of the heart is deflected to the left and is located horizontally.
With RBN of the bundle of His, the main changes in the ventricular complex occur in the right chest leads: a split and notched QRS complex of the zsR, zsz, zSR types and a wide deep S wave in the left chest leads. The axis of the heart is usually deviated to the right, but a levogram is also possible.
The blockade of the terminal branches of the Purkinje fibers is diagnosed by a significant broadening of the complex
QRS, combined with a diffuse decrease in the amplitude of the ventricular complex. Blockades of the legs of the bundle of His do not require treatment by themselves, but they should be taken into account when prescribing drugs that slow down the conduction of an impulse in the pathway system. If the blockade of the left branch of the His bundle occurs during myocardial infarction, an electrode for a temporary pacemaker can be installed for 48-72 hours.

SYNDROME OF PREMATURE VENTRICULAR EXCITATION (WOLF-PARKINSON-WHITE, OR W-P-W), due to the presence of additional pathways along which the impulse propagates from the atria to the ventricles, is manifested on the ECG by a shortening of the P-Q interval to 0.08-0.11 with and widening of the QRS complex is more than normal (reaches 0.12-0.15 s). In this regard, the QRS complex resembles a bundle branch block. At the beginning of the QRS complex, an additional wave (D-wave) is recorded in the form of a “ladder”. Depending on the location of the D-wave, several variants of the syndrome are distinguished: a positive D-wave in lead V, - type A, a negative D-wave in V, - type B. Despite the shortening of the P-Q interval and the widening of the QRS complex, the total duration of the interval PQRS is usually within normal limits, that is, the QRS complex is widened as much as the P-Q interval is shortened.

W-P-W syndrome occurs in 0.15-0.20% of people, and 40-80% of them have various cardiac arrhythmias, mainly supraventricular tachycardias. Paroxysms of atrial fibrillation or flutter may occur (in about 10% of patients).

In 1/4 of persons with W-P-W syndrome, extrasystole is noted, mainly supraventricular. This pathology is more often observed in men and can manifest itself at any age.

Often there is a family predisposition. A combination of the W-P-W syndrome with congenital anomalies of the heart is possible. Its manifestation is promoted by neurocirculatory dystonia and hyperthyroidism.

Treatment. W-P-W syndrome, which is not accompanied by attacks of tachycardia, does not require treatment. If heart rhythm disturbances occur, and these are most often paroxysms of supraventricular tachycardia, the principles of treatment are the same as for similar tachyarrhythmias of another origin - vagotropic tests, intravenous administration of cardiac glycosides, P-adrenergic receptor blockers, isoptin, novocainamide. If there is no effect from pharmacotherapy, electrical defibrillation is performed. With frequent paroxysmal tachyarrhythmias, refractory to drug therapy, surgical treatment is performed: the intersection of additional pathways.

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LECTURE TOPIC: CARDOMEGALY AND HEART MURMUR SYNDROME. DIFFERENTIAL DIAGNOSIS OF CARDIOMEGALY AND HEART MURMURS.PREVENTION.

TRAINING TECHNOLOGY

Number of students-	Time - 2 hours
Form of the lesson	Lecture - visualization
Lecture plan	1. Anatomical structure of the valvular apparatus of the heart. 2. Causes leading to the appearance of murmurs in the heart. Diseases accompanied by murmurs in the heart 3. Concept, definition of organic and functional noise 4. Causes of development of congenital and acquired heart defects 5. Examination of patients with heart defects 6. Classification of congenital heart defects 7. Clinical course, diagnosis of certain types of congenital heart defects 8. Principles of treatment, prevention and clinical examination of patients with heart murmurs
The purpose of the lesson: to acquaint students with the etiology, pathogenesis of diseases manifested by heart murmurs; to teach to distinguish between heart murmurs in various diseases; recall the classification of congenital heart defects; to teach the principles of diagnosis, treatment, prevention and medical examination of diseases accompanied by heart murmurs, to familiarize with the features of the course, the tactics of GP in some congenital heart defects.	
Pedagogical tasks: 1. Strengthen and deepen students' knowledge of diseases accompanied by heart murmurs 2. To teach students to correctly establish a diagnosis in accordance with the modern classification 3. To teach students the ability to differentiate diseases accompanied by various heart murmurs 4. To acquaint students with the features of the course of	The results of the educational process: The GP needs to know: 1. Anatomical structure of the valvular apparatus of the heart. 2. Causes leading to the appearance of murmurs in the heart. Diseases accompanied by murmurs in the heart 3. Concept, definition of organic and functional noise 4. Causes of development of congenital and acquired heart defects 5. Methods of examination of patients with heart defects, clinical course of heart defects 6. Classification of congenital heart defects

some congenital heart defects, the tactics of GP 5.Teach students the management of patients with heart murmurs, treatment and prevention steps	7. Principles of treatment, prevention and clinical examination of patients with heart murmurs 8. Differential diagnostic signs of various diseases accompanied by heart murmurs
Teaching methodology	Lecture text, videos, questionnaires, questions, “yes-no” technique
Form of study	Laser projector, visual materials, special technical equipment, display of thematic patients, CDs with recordings of various heart murmurs
Means of education	Team
Conditions for the educational process	Audience

TECHNOLOGICAL CARD LECTURES

Stages, time	Activity	
	Teacher	students
Stage 1 Introduction (5 minutes)	1. Tells about the topic of the lecture, its purpose and plan	1. Listen
Stage 2 Knowledge update (20 minutes)	2.1. In order to increase the actualization of students' knowledge, asks questions: 1. What kind of noise do you know by origin? 2. List the diseases accompanied by murmurs in the heart area? 3. Tell the difference between functional and organic noise 4. Tell the classification of congenital heart defects Conducts a survey 2.2. Showing on the screen invites students to familiarize themselves with the goals and objectives of the lecture. Slide №1, №2	2.1. Answer questions asked 2.2. Study the slide №1 2.3. Study the slide №2
Stage 3 Main part (informational) (55 min)	3.1. Introduces students to the lecture material, the importance of the topic and the principles of the	3.1. Together they analyze the listened lecture material, ask questions

	formation of an intelligent cultural personality, in particular the GP. In order to increase the actualization of knowledge, quick survey of students: According to 1 point of the lecture plan: the anatomical structure of the valvular apparatus of the heart. On the 2nd point of the lecture plan: the reasons leading to the appearance of murmurs in the heart. According to the 3rd point of the lecture plan: diseases accompanied by murmurs in the heart area. On the 4th point of the lecture plan: the concept, definition of organic and functional noise. On the 5th point of the lecture plan: the causes of the development of congenital and acquired heart defects. According to the 6th point of the lecture plan: examination of patients with heart defects. According to the 7th point of the lecture plan: classification of congenital heart defects. According to the 8th point of the lecture plan: clinical course, diagnosis of certain types of congenital heart defects. According to point 9 of the lecture plan: principles of treatment, prevention and clinical examination of patients with heart murmurs. Stopping at the important points of the lecture, he suggests writing down the main points in a notebook	Key points are written in a notebook.
<u>Stage 4</u> <u>Final (10 min)</u>	4.1. Asking questions: 1. List the most common diseases accompanied by heart murmurs 2. Tell the modern classification of heart defects	4.1. Answer questions

	3. Tell the main key points of the clinical course of various types of congenital malformations. 4. Name the basic principles of treatment, prevention of patients with heart defects 4.2. Gives a task for independent work of students: Diseases accompanied by functional heart murmurs	 4.2. Listen, write

Determination of cardiac murmurs is of great differential diagnostic value, since their presence often confirms the presence of heart disease or other organic heart disease..

Heart defects (vitia cordis) are morphological changes in the valvular apparatus of the heart, leading to a violation of its function and hemodynamics, as well as congenital disorders in the development of the heart and large vessels.

Congenital malformations are formed during the fetal development of the fetus and in most cases are diagnosed in childhood. Congenital heart defects are often combined with other developmental defects.

In infective endocarditis, atherosclerosis, syphilis, the aortic valve is predominantly affected. Isolated aortic valve defects are more common in men. Aortic stenosis can develop in people with abnormalities in the structure of the aortic valve (bicuspid valve). A similar anomaly in the structure of the aortic valve, according to echocardiography and autopsies, is observed in 10-15% of people.

The basis of the clinical diagnosis of heart defects is still the usual clinical examination of the patient. Differential diagnosis of congenital and acquired heart defects is often helped by anamnestic information. It is especially important to ask the patient about the transferred attacks of rheumatism in childhood and adolescence. Patients with congenital heart defects in some cases lag behind in physical development, many of them have a history of frequent pneumonia and bronchitis. However, a number of patients with some and mild congenital heart defects survive to middle age, their physical development does not suffer.

Useful information for the diagnosis of heart defects can be obtained from an external examination of patients. Percussion reveals an increase in the size of the heart, especially with dilatation of its cavities. This method is difficult to determine the initial hypertrophy of the heart.

Auscultation remains an important method for diagnosing heart defects. In order to obtain maximum information at the same time, it is necessary to create conditions that ensure an increase in blood flow through the affected valve. This is achieved through physical activity and drug-induced slowing of the heart rate. The best place to listen to noises with mitral valve defects is the apex of the heart, with tricuspid valve defects - the lower edge of the body of the sternum, with aortic valve defects - the second intercostal space to the right or third to the left of the sternum. With

defects in the mitral valve, noises are conducted to the left axillary region, with stenosis of the aortic mouth, to the vessels of the neck.

Atrial and ventricular hypertrophy, cardiac arrhythmias can be detected using an ECG.

With radiography of the heart in three projections, the increase in individual cavities of the heart, the state of blood circulation in the small circle are more accurately determined.

Valuable diagnostic information can be obtained from echocardiography. It allows you to evaluate not only the anatomical features and contractility of the myocardium, but also the morphology, the function of individual structures of the heart. To date, echocardiography, performed on modern devices and by a qualified specialist in functional diagnostics, is the most informative method for examining the heart. This method of diagnosing heart defects has made available what was previously inaccessible.

Invasive methods for diagnosing heart defects (probing of the right and left parts of the heart with manometry, examination of the gas composition of blood from the cavities of the heart, contrast radiological methods) in the CIS countries are used only in cardiac surgery hospitals and under strict indications.

In the diagnosis of heart defects, a clinician requires a good knowledge of their semiotics, a correct analysis of subjective symptoms and objective data, a logical and comprehensive assessment of the results of clinical and paraclinical studies. It is not enough to establish the nature of the lesion of the valvular apparatus of the heart. It is important to correctly interpret the nature of the pathological process that led to the formation of the defect, to assess its activity and phase. If circulatory disorders occur, their cause should be found out: myocardial overload due to a defect, heart rhythm disturbance or exacerbation of the underlying pathological process. It should be remembered that the availability of modern instrumental methods for diagnosing heart defects will never replace the clinical thinking of a doctor.

Often, during auscultation of the heart in practically healthy people, noises are heard that are not organic. However, in such situations, problems may arise in the differential diagnosis with heart defects. The doctor needs to remember the main causes of functional (non-organic) heart murmurs and their distinguishing features from organic murmurs.

FUNCTIONAL (INORGANIC, INNOCENT, RANDOM) NOISES. There are many reasons and mechanisms for the formation of such noise in each case. Usually, functional murmurs are heard over the apex of the heart, at the Botkin point, or over the pulmonary artery. They occur in almost every second child and in almost a third of adult young people. Murmur at the base of the heart in children and young people appears due to the presence of a relative narrowing of the pulmonary artery. In childhood, the physiological predominance of the lumen of the cavity of the right ventricle over the diameter of the pulmonary artery is preserved.

Under the guise of aortic stenosis, congenital heart defects in young people, hyperkinetic cardiac syndrome is often hidden - a kind of functional (dysregulatory)

cardiovascular disorders. This functional disorder is most often found in young conscripts. The syndrome is based on an increase in the activity of myocardial β-adrenergic receptors, which entails cardiac hypertrophy with an increase in the volume and speed of blood ejection and a compensatory drop in total peripheral resistance. In such young men, a systolic murmur over the aorta (from weak to very noticeable) is heard, which is often carried out to the carotid arteries, especially the right one. On PCG, it has the shape of an asymmetric rhombus with a peak in the first half of systole. It's a high speed ejection noise. Stenosis of the aortic mouth is contradicted by the expressive features of hyperfunction of the heart: increased pulsation of the carotid arteries, rapid pulse, and increased pulse blood pressure.

Systolic murmurs in the heart in young people may occur due to changes in the tone of the papillary muscles, which is facilitated by the lability of the autonomic nervous system. In the formation of functional noise, the presence of additional (abnormal, false, “blind”) tendon chords attached to the mitral valve and papillary muscles may be important. One of the common causes of functional systolic murmur is a transient systolic ridge protruding into the lumen of the left ventricular outflow chamber as a result of systolic thickening or protrusion of the subaortic portion of the interventricular septum. In other words, a common cause of systolic murmur is the deformation of the contours of the cavity of the left ventricle, especially its outflow tract.

Inorganic noise may be due to the acceleration of blood flow in anemia, thyrotoxicosis in conditions of preserved contractile function of the ventricles. Systolic murmur is often heard in myocarditis, myocardial dystrophy of various origins, cardiosclerosis.

Functional noises are usually not very intense, their timbre is soft, blowing, they are very variable in intensity and duration with a change in body position. They are not carried out on the vessels of the neck in the direction of blood flow or in the axillary region. Such a murmur on FCG has a small amplitude and duration, more often it is located in the middle third of the systolic interval (mesosystolic murmur). The shape of the noise changes from one cardiac cycle to another, depending on the position of the body and phases of respiration. The amplitude of the I tone at the same time, with the exception of cases of myocarditis or cardiosclerosis, does not change.

During the examination, in some patients with MVP, symptoms of asthenia, a high arch of the upper palate, a sunken chest and scoliosis can be detected. On auscultation, a late systolic murmur and an additional tone (click) in the middle of systole are heard. These changes are well fixed on FKG. In 1/3 of patients, ECG changes are detected. They mainly concern T-wave inversion in leads II, III, and aVF. Possible prolongation of the Q-T interval, the presence of ventricular extrasystoles, other arrhythmias. Echocardiography plays a key role in the diagnosis of MVP.

ACQUIRED HEART DEFECTS. Mitral stenosis (narrowing of the left atrioventricular orifice, stenosis mltralis, stenosis ostii atrioventricularis sinistra). In humans, the area of the left atrioventricular opening ranges from 4-6 cm2. With mitral stenosis, this opening narrows. Due to the obstruction of blood flow from the

left atrium to the left ventricle, the blood pressure in the left atrium rises from 5 to 20-25 mm Hg. The systole of the left atrium is lengthened. Retrograde pressure increases in the pulmonary veins and capillaries. Reflexively, arterioles can also narrow (Kitaev's reflex), which leads to an increase in pressure in the pulmonary artery system. Functional spasm, and then anatomical changes in the vessels of the pulmonary circulation create the so-called second barrier to blood flow. The inclusion of the second barrier increases the load on the right ventricle. Its hypertrophy develops, and subsequently - decompensation in the systemic circulation. In the stage of compensation, patients do not complain. As the defect progresses, shortness of breath appears during exercise, and then at rest, cough, sometimes hemoptysis, palpitations, general weakness, and increased fatigue. Rarely there are aching or stabbing pains in the region of the heart that are not associated with physical activity. Extrasystole, as a rule, acts as a harbinger of atrial fibrillation. There is aphonia (Ortner's symptom) due to pressure of the enlarged left atrium on the recurrent nerve. During the examination, cyanosis of the lips, the tip of the nose, a blush of the cheeks with a somewhat cyanotic tint (facies mitralis) can be detected.

The apical impulse is weakened. Over the region of the apex of the heart with severe mitral stenosis, diastolic trembling (“cat's purr”) is determined. With hypertrophy of the right ventricle, a pulsation appears in the epigastric region, which intensifies on inspiration. Pulse and blood pressure did not change significantly. There is a tendency to decrease in pulse pressure. In the process of percussion, it is possible to determine the displacement of the boundaries of relative cardiac dullness up (left atrium) and to the right (right atrium). An increased (clapping) / tone is heard above the apex of the heart. Immediately after // the tone, a click (tone) of the opening of the mitral valve can be heard. Clapping tone I, tone II with the tone of the opening of the mitral valve create a three-membered “quail rhythm” at the top of the heart. Above the pulmonary artery // the tone is increased, often bifurcated. An important diagnostic sign of mitral stenosis is a diastolic murmur at the apex of the heart, usually with presystolic amplification. The timbre of the noise is rough, it is better heard after exercise in the position on the left side with holding the breath in the exhalation phase. Without physical activity, the noise may not be heard. With the development of atrial fibrillation and a decrease in the contractile function of the atria, the presystolic murmur usually disappears.

The absence of a mitral stenosis melody can also occur with severe fibrosis and calcification of the mitral valve cusps with limited mobility. Above the apex of the heart in most patients, a systolic murmur is heard, which is a manifestation of simultaneously existing mitral insufficiency or a consequence of calcification of the valve leaflets. In conditions of significant pulmonary hypertension over the pulmonary artery, a diastolic Still's murmur is heard - the result of relative insufficiency of the semilunar valves.

On the ECG - signs of hypertrophy of the left atrium (bimodal P wave in leads I, aVL with a duration of more than OD s) and the right ventricle, often blockade of the right leg of the bundle of His, extrasystole and atrial fibrillation. X-ray shows

smoothing of the waist of the heart due to hypertrophy of the left atrium. The contrasted esophagus is displaced to the right and backward by an enlarged left atrium along an arc of small radius (no more than 6 cm).

There may be a displacement of the heart to the left due to significant hypertrophy and dilatation of the right ventricle. Important radiological signs of mitral stenosis include venous pulmonary hypertension, manifested by the expansion of the roots with blurred boundaries. With arterial pulmonary hypertension, the shadows of the roots expand with clear contours. FCG reveals an increase in the amplitude of the I tone, a prolongation of the Q-I tone interval up to 0.08-0.12 s, an increase in the II tone or its bifurcation in the pulmonary artery, a click of the mitral valve opening 0.06-0.12 s after the I- tone (interval II-OS). As the stenosis progresses, the II-OS interval shortens. FCG also registers a diastolic murmur that begins immediately after the mitral valve opening tone or after a certain period of time after this tone. A typical presystolic murmur is often present. The value of FKG increases with atrial fibrillation.

Mitral stenosis can lead to the development of complications: hemoptysis, pulmonary edema, atrial fibrillation and flutter, thromboembolism in the systemic circulation. Thromboembolism is also possible in the vessels of the small circle. The source of emboli can be varicose veins of the lower extremities.

According to the severity, several degrees of mitral stenosis differ.

Defect I degree (slightly pronounced): a short presystolic murmur is heard, a slight increase in the left atrium is noted. The area of the mitral orifice, according to echocardiography, exceeds 3.0 cm2. Defect of the II degree (moderate): a diastolic murmur of moderate intensity is heard, a distinct increase in the left atrium is observed. The area of the mitral orifice is 3.0-2.0 cm2. Defect III degree (pronounced): a continuous diastolic murmur is heard, accompanied by a “cat's purr”, there are clear signs of right ventricular hypertrophy. The area of the mitral orifice is less than 2.0 cm2. When the area of the mitral orifice is less than 1 cm2, it is said about a sharp mitral stenosis. Severe and severe mitral stenosis should be considered an indication for surgical treatment of the defect, regardless of the stage of circulatory failure. Severe and severe mitral stenosis inevitably leads to the development of heart failure and complications, in particular, atrial fibrillation, pulmonary embolism, etc. An important role is played by an objective assessment of the outcome of a primary rheumatic attack. Signs of emerging mitral stenosis are detected no earlier than 2-3 months from the onset of the disease, and the period of complete formation of mitral stenosis is 6-12 months. Clinical and instrumental signs of mitral valve valvulitis occurring with the formation of mitral stenosis are: progressive "tightness" of the palms of the mitral leaflet in diastole with simultaneous registration on FCG of an inconstant mitral valve opening tone and mesodiastolic murmur; the appearance of a dome-shaped diastolic bend of the anterior mitral valve, its marginal thickening (the most important sign!); transformation of laminar diastolic flow into turbulent, according to Doppler echocardiography, and an increase in the transmitral diastolic pressure gradient. In

some patients, after suffering an attack of rheumatism, all signs of mitral valvulitis may disappear completely. Only residual changes in the valve leaflets remain.

Mitral valve insufficiency (mitral insufficiency, insufficient valvule mitralis) in isolated form is rarely observed (up to 5% of cases), often combined with mitral stenosis and aortic defects. There is also relative mitral insufficiency. It may be due to diffuse damage to the myocardium of the left ventricle and expansion of its cavity or changes in the tone of the papillary muscles. Anatomical damage to the mitral valve itself with this type of mitral insufficiency is not observed. Due to incomplete closure of the mitral valve cusps, part of the blood during systole is thrown back into the left atrium. An increased amount of blood enters the left ventricle, which gradually causes its moderate hypertrophy and more pronounced dilatation. The defect is compensated for by a powerful left ventricle for a long time. The weakening of the contractile function of the left ventricle leads to the development of congestion in the lungs, hypertrophy of the right ventricle and, subsequently, to the appearance of signs of decompensation in the systemic circulation. Clinical symptoms in mitral insufficiency appear only when mitral regurgitation is more than 15-20% of the stroke volume of the heart. In a state of defect compensation, patients feel well. In the future, shortness of breath on exertion and palpitations appear. Rarely, coughing can disturb, hemoptysis is extremely rare. Stitching or aching pains in the region of the heart are possible. On external examination, no noticeable changes are observed. An important acoustic sign of mitral occlusion is the weakening / tone at the apex. The second pulmonary artery tone is amplified or split. Often at the apex of the heart, a third tone is heard. The most characteristic sign of a defect is a systolic murmur, the intensity of which depends on the severity of the valvular defect. The timbre of the noise is soft, blowing. The murmur is best heard at the apex of the heart with the patient on the left side. The more and longer the systolic murmur, the more severe the mitral insufficiency. The ECG may show signs of left atrial and left ventricular hypertrophy. In the process of radiography, an increase in the left ventricle and left atrium is determined. The contrasted esophagus deviates along an arc of a large radius (more than 6 cm). In the lungs, there is an expansion of the roots with fuzzy contours. On FCG, the amplitude / tone is usually reduced, the Q-I tone interval may be increased, III tone is recorded 0.12-0.18 s after the Eaton. Systolic noise begins right after / tone, occupies the most part of a systole, often has the decreasing character. With mitral insufficiency, atrial fibrillation may also develop due to overload of the left atrium. There are three degrees of severity of mitral insufficiency: Defect I degree: low intensity systolic murmur, mild increase in the left ventricle, slight increase in the left atrium. Defect II degree: systolic murmur of moderate intensity, moderate III tone, a distinct increase in the left ventricle and left atrium. Defect III degree: intense systolic murmur, merging with / and // tones, splitting II tone with increased pulmonary component, large amplitude III tone marked enlargement of the left side of the heart. Signs of emerging mitral valve insufficiency after a rheumatic attack include: the appearance of intense systolic murmur at the apex of the heart; marginal thickening of the anterior mitral leaflet (with a domed bend in diastole in a number of patients); registration of a gradually

increasing turbulent systolic flow in the cavity of the left atrium according to Doppler echocardiography.

Associated mitral heart disease is manifested by a combination of sound symptoms of stenosis and insufficiency. For the predominance of mitral stenosis, the most characteristic are: presystolic murmur at the apex of the heart, an increase in the left atrium and right ventricle, and a less distinct systolic murmur. For a defect with a predominance of mitral insufficiency, the most typical are: systolic murmur at the apex, conducted to the left axillary region, an increase in the left ventricle, less pronounced diastolic murmur. Atrial fibrillation is more common with a combination of mitral stenosis and significant mitral insufficiency. Finding out the predominant type of defect is of particular importance when deciding on surgical treatment (mitral commissurotomy or mitral valve replacement).

Narrowing of the mouth of the aorta (aortic stenosis, stenosis ostii aortac). Approximately 3 times more common in men. Due to the obstruction of blood flow, severe hypertrophy of the left ventricle develops. The volume of its cavity does not increase. Severe hemodynamic disorders occur when the aortic orifice narrows by 75 percent or more. The defect can remain compensated for a long time. In conditions of a decrease in the contractile function of the left ventricle, its dilatation is observed. Only with severe stenosis, patients have complaints caused by the lack of an adequate increase in minute volume during exercise, increased fatigue, dizziness, fainting, compressive pain in the region of the heart and behind the sternum. The reason for the latter is a decrease in coronary circulation due to a reduced volume of blood flow to the coronary arteries and myocardial hypertrophy. Shortness of breath is inherent in the later stages of the defect. The appearance of shortness of breath and asthma attacks indicates a decrease in the contractile function of the myocardium. On examination, pallor of the skin is noted, which is associated with spasm of the skin vessels. This is the body's response to low cardiac output. In cases of severe stenosis, an increased apex beat can be observed. It shifts to the sixth - seventh intercostal space to the anterior axillary line. On palpation in the expiratory phase in the second intercostal space to the right of the sternum, systolic trembling can be determined. Signs of heart failure appear first in a small, and then a large circle of blood circulation. In the compensated stage of the defect, there can be only slight hypertrophy of the left ventricle. With the appearance of dilatation of the heart, its borders are significantly shifted to the left. The pulse is small, slowly rising. Systolic blood pressure is moderately reduced. Often there is bradycardia. The first tone at the apex of the heart is preserved or weakened, it may be splitting. The second tone on the aorta is often weakened or not determined due to stiffness of the aortic valve cusps and a decrease in pressure in the aorta. A rough, scraping, cutting or vibrating systolic murmur is heard. The epicenter of the noise is the second intercostal space to the right of the sternum or Botkin's point. Noise is well conducted to the vessels of the neck, to the jugular fossa and the interscapular region, it is better heard in the exhalation phase in the position on the right side, sometimes it is also carried out to the apex of the heart, which can serve as a cause of

misdiagnosis of mitral insufficiency. The intensity of systolic murmur may decrease in severe emphysema, concomitant mitral stenosis, tachycardia, heart failure.
Radiologically, during the defect compensation period, the dimensions of the left ventricle changed slightly. With the development of decompensation, the left ventricle expands, and then the left atrium. The heart acquires a typical aortic configuration. With severe stenosis on the ECG, there are signs of left ventricular hypertrophy, a complete blockade of the left leg of the His bundle may be recorded. Atrial fibrillation is rare. On FCG, a typical diamond-shaped systolic murmur, weakening or disappearance of // sounds on the aorta, reduced amplitudes / sounds at the apex of the heart. With subaortic (subvalvular) congenital stenosis, a large-amplitude systolic murmur is recorded not only on the aorta, but also on the apex. The amplitude // of the tone on the aorta is preserved.
A feature of stenosis of the aortic mouth is a long period of compensation. Heart failure occurs with attacks of cardiac asthma. The period of defect decompensation, as a rule, lasts a relatively short time (1-2 years). Patients may also die from coronary insufficiency, which develops as a result of inadequate blood supply to the coronary artery system due to reduced cardiac output and a mismatch between the network of coronary vessels and severe left ventricular hypertrophy.
There are 3 degrees of severity of aortic stenosis:
Defect I degree: a typical auscultatory picture in combination with slightly pronounced signs of enlargement of the left ventricle, an increase in the thickness of the wall of the left ventricle up to 1.2 cm.
Defect II degree: a typical systolic murmur (diamond-shaped in shape on FCG) is heard, which is carried out to the vessels of the neck, a distinct weakening // tone, a distinct left ventricular hypertrophy is determined, an increase in the thickness of the left ventricular wall up to 1.5 cm (according to echocardiography).
Defect III degree: severe subjective symptoms with dilatation of the left ventricle and pronounced changes in the ECG, the wall thickness of the left ventricle is more than 1.5 cm.
Insufficiency of the aortic valve (aortic insufficiency, insufficienfia valvule aortae). In about half of the cases, this defect occurs in combination with aortic stenosis. It happens more often in men. Significant reverse flow of blood from the aorta to the left ventricle as a result of incomplete closure of the valve leaflets during diastole leads to expansion of the left ventricle. As a compensatory mechanism, systolic blood output increases, which contributes to the development of left ventricular hypertrophy, and not just its expansion. The resistance of peripheral vessels on the periphery decreases. With a significant expansion of the cavity of the left ventricle, relative mitral insufficiency can develop - mitralization of the defect. In the stage of defect compensation, patients remain able to work for a long time and rarely complain. There may be a feeling of increased pulsation of the carotid arteries, palpitations. As with stenosis of the aortic mouth, chest pains of the angina pectoris type are characteristic, aggravated by physical exertion, dizziness, and a tendency to faint with a quick change in position. In the case of sudden movements, loss of

consciousness is possible. Shortness of breath appears with a decrease in the contractile function of the left ventricle.

When examining a patient, attention is drawn to the pallor of the skin, the pulsation of large vessels, especially the carotid arteries (“dance of the carotid”). Rhythmic shaking of the head (Musset symptom), pulsation of the precordial region can be detected. On the nail bed, a “capillary pulse” is sometimes found - a change in the color intensity of the nail bed synchronous with the pulse. The lifting apex beat is clearly visible, shifted to the left and down. The aortic configuration of the heart is characteristic.

For aortic insufficiency, an increase in systolic and a decrease in diastolic pressure is typical (the amplitude of the pulse pressure increases). In some patients, diastolic blood pressure drops to zero. The pulse is high and fast, which is associated with a rapid rise and decrease in blood pressure. Over large vessels, a double Durozier noise can be determined, less often a double Traube tone. Auscultation of the heart area reveals a decrease in the intensity of the I tone, a weakening or absence // of the tone on the aorta. The degree of weakening of the latter is proportional to the severity of the valvular defect. A diastolic murmur is heard with an epicenter in the second intercostal space to the right of the sternum or in the third to fourth intercostal space to the left of the sternum. The noise is soft, blowing, of varying duration, occurs immediately after Eton, is better heard in the expiratory phase in the patient's sitting position with the torso tilted forward, usually weakens with tachycardia, heart failure, atrial fibrillation. With aortic insufficiency, a systolic murmur (accompanying murmur) may also be heard, due to a swirl of blood flow due to deformation of the aortic valve cusps. At the apex of the heart, a systolic murmur may appear associated with the development of relative mitral insufficiency, as well as meso- or presystolic murmur (Flint's noise) due to the development of relative stenosis of the mitral orifice. X-ray revealed an increase in the left ventricle and expansion of the ascending aorta. The waist of the heart is pronounced. Even with "mitralization" of the aortic defect, there is no significant hypertrophy of the left atrium. ECG shows signs of left ventricular hypertrophy. In contrast to stenosis of the aortic orifice, in aortic insufficiency, high pointed T waves may appear in leads V.-V6. On FCG, a weakening / tone at the apex is determined, with a pronounced defect, there may be /// a tone. The second tone at the base of the heart is weakened. A diastolic murmur is recorded, which begins immediately after Eaton and has a decreasing character. The noise is high-frequency, in connection with which it is sometimes better heard by the ear than recorded on the FCG. In the second intercostal space to the right of the sternum, an accompanying systolic murmur may be detected. It, as a rule, does not have a specific shape and takes no more than half of the systole. With aortic insufficiency, patients are in a compensated state for a long time. However, in cases of the appearance of signs of heart failure, their condition rapidly and progressively worsens. Heart failure in this case proceeds according to the left ventricular type with attacks of cardiac asthma. In the future, it is possible to develop stagnation in the systemic circulation. Aortic insufficiency can be of three degrees of severity: Defect I degree: a short protodiastolic murmur is

heard (usually at the Botkin point), usually not recorded on FCG, a slight increase in the left ventricle. Defect II degree: diastolic murmur is more intense, II tone at the base of the heart is weakened, peripheral vascular signs are clearly expressed, an increase in the left ventricle is clearly detected. Defect III degree: continuous diastolic murmur in combination with the absence or a sharp weakening // tone, a significant increase in the left ventricle, pronounced vascular peripheral signs.

Signs of aortic valve valvulitis are:

the appearance of uneven bright echo signals from the semilunar valves along the line of their closure; registration of turbulent diastolic flow in the outflow tract of the left ventricle with Doppler echocardiography and simultaneous detection of protodiastolic murmur.

Combined aortic heart disease is clinically manifested by signs characteristic of each of its constituent lesions of the valvular apparatus of the heart. To resolve the issue of the predominance of the defect, it is necessary to carefully analyze the clinical data and the results of all paraclinical research methods. Determination of the predominant type of lesions is essential, since the indications for surgery and the nature of the surgical intervention depend on it. Invasive research methods are sometimes used to clarify the nature of the predominant lesion.

Tricuspid valve insufficiency (insufficientia valvulae tricuspidalis) is relatively common. Organic and relative insufficiency differs. The latter is much more common. Usually the defect occurs in combination with mitral or aortic defects. Relative insufficiency of the tricuspid valve is observed with a significant expansion of the right ventricle and an increase in its cavity. During the systole of the right ventricle, part of the blood from its cavity enters back into the right atrium. Stagnation of blood in the cavity of the right atrium is transmitted to the system of hollow veins. When examining patients, acrocyanosis, swelling of the cervical veins and their systolic pulsation are revealed - a positive venous pulse, slight icterus of the sclera and skin due to functional insufficiency of the congestive liver. There is a pulsation in the epigastric region due to dilatation of the right ventricle. Sometimes there is a pulsation of the liver.

Auscultation reveals a systolic murmur with an epicenter at the base of the xiphoid process. This noise increases at the height of inspiration (Rivero-Corvallo symptom), which distinguishes it from noise with mitral valve insufficiency.

During inhalation, blood flow through the right heart is accelerated and the volume of regurgitation increases. ECG shows signs of right ventricular hypertrophy, FCG shows a distinct systolic murmur at the base of the xiphoid process, starting immediately after / tone. At the height of inspiration, the amplitude of the noise increases. When making a diagnosis, differential diagnosis with adhesive pericarditis is carried out.

Stenosis of the right atrioventricular orifice (tricuspid stenosis, stenosis ostii atrioventricularis dcxrri, stenosis tricuspidalis) almost never occurs in its pure form. Usually associated with mitral heart disease. The clinical picture of the disease in most cases is determined by the defects combined with it. On examination, cyanosis, significantly dilated and pulsating jugular veins, stagnation

in the systemic circulation, hypertrophy of the right atrium are revealed. During auscultation, a decrease in the intensity and tone is found due to the absence of stagnation in the pulmonary circulation. At the base of the xiphoid process, a diastolic murmur is heard, which is also recorded on FCG in the form of a presystolic rhomboid murmur. Noise amplifies at the height of inspiration, especially in the position of the patient on the right side. Typically, there is no noticeable stagnation in the lungs, which is confirmed by x-ray. ECG shows signs of right atrial hypertrophy.

CONGENITAL HEART DEFECTS. In most patients, congenital heart defects are recognized in childhood, as in this case there is a distinct sound symptomatology and hemodynamic disturbances appear early. In some cases, due to the small severity of the defect, it proceeds hidden for a long time. And only in adulthood, the first signs of decompensation may occur, which makes the patient consult a doctor. As a result of a comprehensive examination, the noise, previously regarded as inorganic, is associated with the presence of congenital heart disease.

Classification of congenital malformations of the heart and blood vessels

1. Malformations with shunting of blood from right to left: Triad of Fallot, Tetralogy of Fallot, Pentade of Fallot, Departure of the aorta and pulmonary artery from the right ventricle, Atresia of the tricuspid valve (with the usual discharge of large vessels), Malformations of the right ventricle (hypoplasia, muscle defects), Transposition large vessels, Atresia of the aortic arch, Malformations of the left half of the heart (atresia or hypoplasia of the aortic orifice, left ventricular diverticulum), Common arterial trunk (true and false), Common ventricle, Arteriovenous aneurysms (systemic, pulmonary),

2. Defects with initial discharge of blood from left to right. Open ductus arteriosus, Aortopulmonary fistula, Primary and secondary atrial septal defect Common atrium Lutembashe syndrome Triatrial heart Common atrioventricular canal Ventricular septal defect Aneurysm of the sinus of Valsalva with a breakthrough in the pulmonary circulation

3. Defects of the right half of the heart. Pulmonary artery stenosis (valvular, infundibular, supravalvular) Ebstein's disease Primary pulmonary hypertension

4. Defects of the left half of the heart. Aortic arch anomalies, Aortic coarctation, Aortic orifice stenosis, Coronary artery anomalies, Mitral valve stenosis

5. Other anomalies

Anomalies in the position of the heart and its individual chambers (dextrocardia, dextroversion with a reverse arrangement of internal organs, etc.)

Congenital malformations with right-to-left shunting are also called “blue type” defects, and those with left-to-right shunting are called “pale type” defects. According to the syndromic principle, congenital heart defects can be divided into pure gateways (aortic stenosis, coarctation of the aorta, pulmonary stenosis), pure shunts (open ductus arteriosus, atrial and ventricular septal defects), a combination of shunts and a gateway (Fallot's triad and tetrad, transposition of the main vessels).

Tetralogy of Fallot. The classic tetralogy of Fallot consists of stenosis or atresia of the outlet pulmonary artery, ventricular septal defect, aortic dextroposition (shift to

the right), and right ventricular hypertrophy. In the triad of Fallot, there is no dextroposition of the aorta. Hemodynamics in tetralogy of Fallot primarily depends on the degree of narrowing of the pulmonary artery. The discharge of blood occurs from right to left. Shortness of breath, cyanosis, fingers in the form of "drumsticks" are determined. During auscultation, the 1st tone, as a rule, is not changed, the 2nd tone on the pulmonary artery is weakened or not heard.

On the left edge of the sternum, a sharp systolic murmur is heard with the greatest intensity in the second - third intercostal space. Noise is well conducted on the vessels of the neck and less in the interscapular region. On the ECG, the deviation of the electrical axis of the heart to the right. X-ray reveals depletion of the lung pattern due to insufficient blood flow. The aorta is dilated and displaced to the right.

Patent ductus arteriosus is more common in women. The clinical picture is determined by the amount of blood shedding through the shunt. The discharge of blood occurs from the aorta into the pulmonary artery. With a small reset, patients remain able to work for many years, the defect is often detected during a random examination. In conditions of significant discharge, shortness of breath, fatigue occur early, and physical development is disturbed. Auscultation and FKG revealed systole-diastolic murmur in the second intercostal space to the left of the sternum.

The murmur is related to the movement of blood from the aorta to the pulmonary artery during both systole and diastole. This noise is figuratively compared with the noise of “the roar of a train in a tunnel”, “machine noise”. It increases on inspiration, as at this time the pressure in the pulmonary artery decreases. ECG shows signs of left heart hypertrophy. X-ray in the lungs are determined by the strengthening of the pulmonary pattern due to the overflow of the arterial bed, the expansion and pulsation of the roots of the lungs.

Atrial septal defect is more commonly diagnosed in adults (10% of congenital heart defects). In most patients, the long-term course is relatively favorable. The discharge of blood occurs from left to right. Patients complain of shortness of breath, fatigue, stabbing pains in the region of the heart, palpitations. On auscultation, a moderate intensity systolic murmur is heard in the second - third intercostal space to the left of the sternum. With right ventricular failure, the intensity of the noise decreases sharply. The strengthening of Eaton on the pulmonary artery is determined. X-ray reveals an increase in the heart to the left due to displacement by an enlarged right ventricle and bulging of the pulmonary artery, an increase in the pulmonary pattern. ECG signs of partial or complete blockade of the right leg of the His bundle, right ventricular hypertrophy.

Lutembashe's syndrome is a combination of atrial septal defect and mitral stenosis. The latter is more often of rheumatic etiology, but may also be congenital. With Lutembashe's syndrome, there is an increase in blood flow from left to right, and therefore hemodynamic disturbances appear earlier. Diagnosis is based on identifying symptoms of atrial septal defect and mitral stenosis.

Ventricular septal defect (Tolochinov-Roger disease) may be isolated or combined with other cardiac anomalies. It is more common in the upper third of the septum, the size is 1-2 mm or the septum is completely absent. The discharge of blood occurs

from left to right. The symptomatology of the defect depends on the size of the defect. With the natural course of the defect, the condition of patients usually begins to progressively worsen after 20-25 years. Rapid fatigue, palpitations, shortness of breath appear. Can be detected heart hump, systolic trembling in the third - fourth intercostal space to the left of the sternum. On auscultation, a loud systolic murmur ("much ado about nothing") is heard with a maximum sound in the third or fourth intercostal space to the left of the sternum.

X-ray revealed bulging of the arch of the pulmonary artery, an increase in the left ventricle. On FCG, systolic murmur in the form of an oval or rhombus, amplification and splitting // tone on the pulmonary artery.

The common atrioventricular canal is a large septal defect that involves the lower part of the atrial septum and the adjacent part of the interventricular septum, and a split common atrioventricular valve overhanging the ventricular component of the septal defect.

Isolated pulmonary stenosis has a relatively benign course, often associated with other defects in the development of the heart. The decrease in right ventricular ejection leads to a decrease in pulmonary blood flow. Shortness of breath, rapid fatigue develop. During auscultation and on FCG in the second intercostal space to the left of the sternum, a rough systolic murmur in the form of a rhombus is determined. The second tone on the pulmonary artery is sharply weakened and bifurcated.

Ebstein's disease is characterized by displacement of the tricuspid valve into the right ventricle due to abnormal attachment of its cusps. The abnormally located valve opening divides the right ventricle into a proximal "atrialized" part and a distal functional small ventricular chamber. Hemodynamic disturbances occur due to inefficient functioning of the small right ventricle.

Coarctation of the aorta is a significant narrowing of some part of the aorta, often in the region of the ductus arteriosus, sometimes in the lower thoracic or abdominal aorta. Some patients are treated for hypertension for a long time before the diagnosis is clarified. Coarctation of the aorta leads to the development of hypertension and collateral circulation, bypassing the site of narrowing. Blood pressure rises due to a mechanical obstruction to blood flow and the inclusion of the endocrine mechanism (renin - angiotensin - aldosterone). Patients complain of headache, fatigue, pain in the ankle joints. When examining blood pressure in the arms, a systolic murmur is heard. On the legs, blood pressure is reduced or not detected at all, and the pulse is also weakened. In such patients, the upper shoulder girdle is usually well developed compared to the lower extremities. The pulsation of the carotid arteries is increased. At the base of the heart (in the second intercostal space on the left or on the right, in the third intercostal space on the left), a systolic murmur is heard. It is well carried out on the vessels of the neck and in the interscapular region. ECG shows signs of left ventricular hypertrophy. An x-ray showed enlargement of the left ventricle. Usuration of the III-VII pairs of ribs is characteristic due to a sharp increase in the diameter of the intercostal arteries and their tortuous course. Aortography helps to establish a reliable diagnosis.

Aortic stenosis obstructs the flow of blood from the left ventricle. More common is aortic valve stenosis - congenital deformation of the valve leaflets or narrowing of the valve ring, less often - subvalvular (fibrous or muscular) aortic stenosis, and even less often - supravalvular stenosis. The clinical symptoms of this malformation mainly resemble the manifestations of acquired stenosis of the aortic orifice.

Treatment of heart defects in most cases is performed surgically. The main type of operation is valve prosthetics. If it is impossible to carry out surgical correction of heart disease, treatment is carried out with the help of medicines. Treatment of decompensated heart disease is carried out according to the same principles as the treatment of chronic heart failure of a different origin.

Primary prevention

1. Prevention aimed at preventing diseases in which heart defects develop
2. Prevention of the occurrence of CHD: medical genetic counseling and explanatory work among the contingent of an increased risk of the disease
3. Fighting consanguineous marriages
4. Careful observation and study of women who have had contact with the rubella virus or have comorbidities that can lead to the development of congenital heart defects.

Secondary prevention

1. Prevention of the adverse development of heart disease: timely detection of the defect, provision of proper care and determination of the optimal method for the correction of the defect (surgical for congenital heart disease)

Tertiary prevention

1. Surgical interventions (acquired heart defects)
2. Prevention of complications of congenital heart defects (bacterial endocarditis)

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LECTURE TOPIC: FEVER SYNDROME. FEVER OF UNKNOWN ORIGIN. PREVENTION.

TRAINING TECHNOLOGY

<table>
<tr><td>Number of students -</td><td>Time - 2 hours</td></tr>
<tr><td>Form of the lesson</td><td>Lecture – show and demonstrate</td></tr>
<tr><td>Lecture plan</td><td>• Explain the febrile syndrome
• The main causes of febrile syndrome
• Pathogenesis of febrile syndrome
• Clinical signs of febrile syndrome
• Characteristic signs of febrile syndrome
• Classification of febrile syndrome
• Characteristic features of febrile syndrome variants
• Changes in internal organs in febrile syndrome
• Complications of febrile syndrome
• Factors leading to death as a result of a disease accompanied by a febrile syndrome
• Treatment of febrile syndrome</td></tr>
<tr><td colspan="2">The purpose of the training session: To provide students with information about the febrile syndrome, the classification of the febrile syndrome, the main diagnostic criteria for diseases associated with the febrile syndrome, complications, diagnosis and training in prevention and emergency care.</td></tr>
<tr><td>Pedagogical tasks:
1. Provide complete information to students about the classification, clinical signs and factors leading to febrile syndrome. To deepen their knowledge of diagnosis and emergency care for febrile syndrome.
2. Explain the principles of differential diagnosis of diseases accompanied by febrile syndrome
3. To achieve independent mastery of practical skills in febrile syndrome and diseases accompanied by febrile syndrome
4. Explain to students the principles of prevention</td><td>Learning outcomes:
The GP needs to know:
1. Be able to diagnose febrile syndrome
2. Clinical signs of febrile syndrome
3. To acquire knowledge of the main causes leading to a febrile syndrome
4. Be able to determine the diagnostic criteria for diseases accompanied by nephrotic syndrome
5. Be able to identify risk factors leading to sudden death in febrile syndrome
6. Complications of febrile syndrome
7. Principles of diagnosis and treatment of diseases accompanied by nephrotic syndrome
8. Prevention and rehabilitation of diseases accompanied by nephrotic syndrome</td></tr>
</table>

Teaching Methods	The text of the lecture, videos, brainstorming, solving situational problems, competitions between groups, “yes-no” technique
Training uniform	Laser projector, visual materials, communication with specialized equipment
Training aids	Team
learning tool	Audience

TECHNOLOGICAL CARD LECTURES

Stages, time	**Activity**	
	Teacher	**students**
Stage 1 Login(5min)	1. Tell the topic, goals and expected plans	1. Listen
2nd stage Improve knowledge (20 minutes)	2.1. Ask questions to students to improve their knowledge 1. Feverish syndrome, what do you mean? 2. What are the main criteria for diagnosing febrile syndrome? 3. List the group of diseases leading to febrile syndrome? 4. Causes leading to the development of sudden death in febrile syndrome? 5. Emergency medical care when spontaneous complications develop in febrile syndrome? Conducts a quick survey 2.2. Offers to get acquainted with the purpose of the lecture by showing on the screen. Gives explanations of the information presented at the lecture Slide 1, 2,...	2.1. Answer questions 2.2. No. 1 Familiarize yourself with the slides 2.3. No. 2 get acquainted with the slides
Stage 3 Main stage (informative) (55 minutes)	2.1. Ask questions to students to improve their knowledge 1. Feverish syndrome, what do you mean? 2. What are the main criteria for diagnosing febrile syndrome? 3. List the group of diseases leading to febrile syndrome? 4. Causes leading to the development of sudden death in febrile syndrome?	3.1. Asking questions and discuss the given materials

	5. Emergency medical care when spontaneous complications develop in febrile syndrome? Conducts a quick survey 2.2. Offers to get acquainted with the purpose of the lecture by showing on the screen. Gives explanations of the information presented at the lecture Slide 1, 2,...	Write down the main points
Stage 4 Final (10 minutes)	4.1. The question is asked: 1. Explain the febrile syndrome 2. Variants of febrile syndrome 3. The main causes of febrile syndrome 4. Complications in diseases accompanied by febrile syndrome 5. Complications of febrile syndrome and emergency medical care 4.2. Give tasks for independent work: Fever of unknown origin	4.1. answer questions 4.2. listening, recording

FEVER is an increase in body temperature relative to normal daily values, due to changes in the thermoregulatory center

Hyperthermia - This is an increase in body temperature without the participation of the hypothalamus, usually due to insufficient heat transfer (for example, during exercise, taking drugs that reduce sweating, and high ambient temperature)

Pyrogens:

 Pyrogens are called endogenous or exogenous substances that cause fever.

 Endogenous pyrogens are produced in the body in response to various stimuli; most often it is an infection or inflammation.

 Exogenous pyrogens are mainly represented by microorganisms, their toxins and waste products

An increase in body temperature is one of the most frequent and characteristic manifestations of many infectious and non-infectious diseases. Often, practical doctors, having revealed an elevated body temperature in a patient, already assume

that he has an infectious disease. However, the prevalence of fever, which can occur in almost all infectious diseases, makes it difficult to differentiate this syndrome, especially since an increase in body temperature is one of the earliest signs when there are no other clinical manifestations of the disease, including many fever parameters that have a differential diagnostic value (duration, nature of the temperature curve, etc.).

It must be borne in mind that not every increase in body temperature is a fever, but it is characteristic of infectious diseases. Fever is understood as a thermoregulatory increase in body temperature, which is an organized and coordinated response of the body to a disease, i.e. the body itself raises the body temperature above normal [.

An increase in body temperature can be caused not only by regulatory mechanisms, but can also occur as a result of an imbalance between heat production and heat loss, which leads to an increase in body temperature despite the attempts of the body to maintain normal temperature. Such an increase in body temperature is called hyperthermia (this term should not be considered as a synonym for fever, which is sometimes found in the literature).

Hyperthermia is observed in the so-called thermal diseases (heat stroke, hyperthyroidism, atropine poisoning, etc.). Finally, an increase in body temperature may be due to normal activity or physiological processes. A slight increase in body temperature may be associated with circadian rhythms (daily fluctuations). The body temperature in a healthy person usually reaches a maximum level by 18 o'clock and a minimum occurs at 3 o'clock in the morning. A slight increase in body temperature can occur after a heavy meal and a more significant increase after heavy and prolonged physical exertion. Thus, we can talk about different mechanisms for increasing body temperature.

A number of classifications of fever have been proposed.

1. Depending on the cause of occurrence, infectious and non-infectious fever are distinguished.

2. According to the degree of body temperature increase: subfebrile (37-37.9 °C), febrile (38-38.9 °C), pyretic or high (39-40.9 °C) and hyperpyretic or excessive (41 °C and higher).

3. According to the duration of fever: acute - up to 15 days, subacute - 16-45 days, chronic - over 45 days.

4. According to the change in body temperature over time, the following types of fever are distinguished:

1. Constant - body temperature is usually high (about 39 ° C), lasts for several days with daily fluctuations within 1 ° C (with lobar pneumonia, typhus, etc.).

2.Laxative - with daily fluctuations from 1 to 2 ° C, but not reaching the normal level (for purulent diseases).

3. Intermittent - alternation after 1-3 days of a normal and hyperthermic state (characteristic of malaria).
4. Hectic - significant (over 3 ° C) daily or at intervals of several hours temperature fluctuations with a sharp drop and rise (in septic conditions).
5. Return - with periods of temperature rise up to 39-40 ° C and periods of normal or subfebrile temperature (with relapsing fever).
6. Wavy - with a gradual increase from day to day and the same gradual decrease (with Hodgkin's disease, brucellosis, etc.).
7. Irregular fever - without a definite pattern in the daily fluctuation (with rheumatism, pneumonia, influenza, oncological diseases).
8. Perverted fever - the morning temperature is higher than the evening one (with tuberculosis, viral diseases, sepsis).

5. In combination with other symptoms of the disease, the following forms of fever are distinguished:

1. Fever - as if a significant manifestation of the disease or its combination with such non-specific symptoms as weakness, sweating, irritability in the absence of inflammatory acute phase shifts in the blood and local signs of the disease. In such cases, it is necessary to make sure that there is no simulation of fever, for which it is necessary, observing tact, to measure the temperature in the presence of medical workers simultaneously in both axillary fossae and even in the rectum

2. Fever is combined with non-specific, sometimes very pronounced acute phase reactions (increased ESR, fibrinogen content, changes in the structure of globulin fractions, etc.) in the absence of local pathology detected clinically and even with instrumental examination (fluoroscopy, endoscopy, ultrasound, ECG, etc. .). The results of laboratory studies exclude data in favor of any acute specific infection. In a word, the patient, as it were, “burns out” for an unknown reason.

3. Fever is combined with both severe nonspecific acute phase reactions and organ changes of unknown nature (abdominal pain, hepatomegaly, arthralgia, etc.). Options for combining organ changes can be very different, while not always associated with a single mechanism of development. In these cases, to establish the nature of the pathological process, one should resort to more informative laboratory, functional-morphological and instrumental research methods.

The scheme for the initial examination of a patient with fever includes such generally accepted methods of laboratory and instrumental diagnostics as a complete blood count, urinalysis, chest X-ray, ECG and echocardiography. With their low information content and depending on the clinical manifestations of the disease, more complex methods of laboratory diagnostics are used (microbiological, serological, endoscopic with biopsy, CT, arteriography, etc.). By the way, in the structure of fever of unknown origin, 5-7% falls on the so-called medicinal fever. Therefore, if there are no obvious signs of an acute abdomen, bacterial sepsis or endocarditis, then for the period of the examination it is advisable to refrain from using antibacterial and other drugs that tend to cause a pyrogenic reaction.

Differential Diagnosis

The variety of nosological forms, manifested by hyperthermia for a long time, makes it difficult to formulate reliable principles of differential diagnosis. Taking into account the prevalence of diseases with severe fever, it is recommended to focus the differential diagnostic search primarily on three groups of diseases: infections, neoplasms and diffuse connective tissue diseases, which account for 90% of all cases of fever of unknown origin..

Fever in diseases caused by infection

The most common cause of fever for which patients consult a general practitioner are:

1. infectious and inflammatory diseases of internal organs (heart, lungs, kidneys, liver, intestines, etc.);

2. classic infectious diseases with severe acute specific fever.

Infectious and inflammatory diseases of the internal organs. All infectious and inflammatory diseases of the internal organs and nonspecific purulent-septic processes (subdiaphragmatic abscess, abscesses of the liver and kidneys, cholangitis, etc.) occur with fever of varying degrees.

This section discusses those of them that are most often encountered in the medical practice of a doctor and for a long time can manifest themselves only as a fever of unknown origin.

Endocarditis. In the practice of the therapist, a special place as a cause of fever of unknown origin is currently occupied by infective endocarditis, in which fever (chills) often far outstrips the physical manifestations of heart disease (murmurs, expansion of the boundaries of the heart, thromboembolism, etc.). In the risk group for infective endocarditis are drug addicts (drug injections) and people who have been injected parenterally with drugs for a long time. In this case, the right side of the heart is usually affected. According to a number of researchers, it is difficult to identify the causative agent of the disease: bacteremia, often intermittent, in almost 90% of patients requires 6 blood cultures. It should be borne in mind that in patients with a defect in the immune status, fungi can be the cause of endocarditis.

Treatment - antibacterial drugs after determining the sensitivity of the pathogen to them. HIV infection caused by a retrovirus that primarily damages lymphocytes, macrophages, nerve cells and intestinal epithelium manifests itself as a slowly progressive immunodeficiency, initially without pronounced clinical signs (asymptomatic stage), but then gradually there is a fever with general weakness and night sweats, lymphadenopathy, diarrhea, headache. Then severe manifestations of AIDS develop, various opportunistic infections (pneumonia, candidiasis, meningitis, herpetic infections, tuberculosis) join, and exhaustion progresses sharply. In adults, the main mode of transmission of the virus is sexual and parenteral. Risk groups are homosexuals, bisexuals, heterosexuals, drug addicts, recipients of blood and its components. Diagnosis: isolation of the virus from blood, semen, saliva, vaginal secretion, determination of antibodies to HIV infection in the blood, specific IgA in the blood and urine, etc.

Treatment should be carried out under constant supervision and with the participation of specialists. Etiotropic treatment: the appointment of reverse transcriptase inhibitors (zidovudine, zalcid, etc.) and protease inhibitors (saquinovir, indinavir). In order to prevent the development of resistance, it is recommended to prescribe at least two drugs, in particular one from each group. In complex treatment, an effect on opportunistic infection should be envisaged.

Currently, atypical pneumonia is very relevant in the world, one of the early manifestations of which may be a fever of unknown origin. The task of a general practitioner is to closely monitor the development of the situation, carefully ask patients about contacts, stay in countries with an unfavorable situation in terms of morbidity, etc.

Treatment is antibiotic therapy. The cause of a fever of unknown origin is sometimes hidden exudative pleurisy, peritonitis, organ (liver, kidney, pancreas) and interorgan abscesses of the abdominal cavity. Of course, more often we are talking about relatively small abscesses and without pronounced local symptoms. Women of childbearing age should not forget about ovarian endometriosis.

Fever in infectious diseases. In infectious diseases, clinical variants or types of fevers have long been one of the controversial symptoms in differential diagnosis. Most of them are characterized by their own type of fever, which is of great help in establishing a clinical diagnosis even when other symptoms are not expressed, and decisive laboratory data are doubtful or absent.

Clinical and laboratory examination of the patient in the presence of fever should be directed primarily to the exclusion of infectious diseases, since the initial examination of the patient and the first laboratory clinical data determine the range of anti-epidemic measures arising from the preliminary conclusion about the nature of the infectious disease.

A reasonable exclusion of an infectious disease opens the way for further examination of the patient and an earlier diagnosis of a non-infectious disease (hematological, oncological, autoimmune, immunocomplex, etc.) occurring with fever.

Treatment. With a moderate increase in body temperature (up to 38-38.5 ° C), the appointment of antipyretics is not required, since during a fever, protective and adaptive reactions and immunity are mobilized in the body. An exception is made by patients who do not tolerate febrile body temperature (38.5 ° C or more).

Fever with neoplasms

More than 1/3 of cases of fever of unknown origin is associated with difficult-to-recognize primary localization of malignant neoplasms, more often in the abdominal cavity, namely: in the kidneys (hypernephroma), colon, prostate gland, liver, stomach. Often accompanied by fever metastasized cancer of the lungs, pancreas. Rare neoplasms such as myxoma of the heart, usually of the left atrium, can be the cause of fever. Myxoma is more common in middle-aged women. Like diseases associated with chronic infection, myxoma is often accompanied not only

by fever, but also by weakness, arthralgia, and an increase in ESR. Developing heart failure and recurrent thromboembolism help establish the diagnosis.

Fever in neoplasms is associated not only with the decay of the tumor, but also with the production of endogenous pyrogenic substances already at the stage of a "small" malignant tumor with "irritation" of leukocytes and macrophage elements with the release of endogenous pyrogens. It should also be borne in mind that an early clinical manifestation of malignant tumors may be paraneoplastic syndrome, which is sometimes mistaken for a systemic disease (SLE, rheumatoid arthritis, etc.) or allergy.

Treatment, depending on the prevalence and course of the process, can be surgical, radiological, chemotherapeutic.

Pheochromocytoma is a benign or malignant tumor that develops from the chromaffin tissue of the adrenal glands, which produces catecholamines. In almost 20% of cases, chromaffin cells form a tumor outside the adrenal glands. The most pronounced, and sometimes the only manifestation of the disease is an increase in blood pressure, but often against the background of a vegetative disorder (tachycardia, increased sweating, hot flashes, headache, etc.).

Surgical treatment.

Fever in diffuse connective tissue diseases

Of the diffuse diseases of the connective tissue, fever most often develops with polyarteritis nodosa. It is usually undulating and sometimes for a certain time may be the only symptom of the disease, in addition, there may be manifestations of multiple organ pathology - coronary disease, bronchospasm, Wegener's syndrome, abdominal syndrome, kidney damage with hypertension, polyneuritis, neutrophilic leukocytosis, an increase in fibrinogen, ESR, a2- and y-globulins.

The cause of prolonged fever may be macrovascular vasculitis:

1. Horton's disease (predominantly the elderly, especially women, are affected; the temporal arteries are most often affected, less often the maxillary artery, the aorta and its branches, the vessels of the heart, brain, joints with the so-called polymyalgia rheumatica, etc.);

2. Takayasu's disease (mostly women suffer; large vessels are affected - the aorta and its branches, in the early stage of the disease, systemic manifestations characteristic of rheumatic diseases are often observed);

3. Buerger's disease (mostly men get sick; generalized vasculitis - intermittent claudication, paresthesia, phlebitis);

4. systemic rheumatoid vasculitis (men are more likely to get sick; skin integuments are often involved in the pathological process with the development of ulcers, purpura, petechiae, pyoderma gangrenosum, organ pathology - damage to the nervous system, lungs, heart, intestines, usually there is an increase in ESR, thrombocytosis, hypergammaglobulinemia; the pathological process in vasculitis, developing in the visceral large vessels or the aorta, at first may not be manifested by local symptoms, but over time pain occurs, which may not be intermittent, but even at rest, constant, the diagnosis is established using selective angiography and biopsy).

Fever with hypereosinophilia (90%) is observed in allergic eosinophilic angiitis. Men are more often ill. In addition to fever of the wrong type, weight loss, pain in the joints and muscles, lymphadenopathy, migrating infiltrates in the lungs, heart damage (parietal endocarditis) with circulatory failure, allergic rhinitis, bronchitis, bronchial asthma and other organ damage are characteristic. Treatment is the appointment of glucocorticoids.

Hectic temperature followed by damage to the joints, heart (pericarditis with effusion), hepatolienal syndrome, often with a positive rheumatoid factor in the blood, is observed in juvenile rheumatoid arthritis, in particular in its variants such as Wissler-Fanconi syndrome and Still's syndrome. The anarthritic debut of rheumatoid arthritis and Bechterew's disease in the form of a fever of unknown origin is described in the literature, but this is rare in clinical practice.

Treatment is the same as for rheumatoid arthritis. NSAIDs - first-line drugs (naproxen, ibuprofen). Second-line drugs - gold salts, penicillamine, chloroquine, sulfasalazine, chlorbutine. With a systemic variant - pulse therapy with glucocorticoids, cytostatics, plasmapheresis.

Isolated fever may be the first manifestation of systemic lupus erythematosus, a diffuse connective tissue disease of an autoimmune nature. Typical skin lesions (erythematous dermatitis of the nose and cheekbones like a butterfly, skin manifestations of vasculitis, etc.), mucous membranes (cheilitis, erosion), joints (arthralgia, damage to ligaments and tendons), internal organs (lungs, pleura, heart, kidneys, organs of the gastrointestinal tract), CNS (focal and diffuse disorders, depression, psychosis), peripheral nervous system (neuritis, chorea), Sjögren's syndrome (dry конъюнктивит, ксеростомия, хронический атрофический гастрит, поражение мелких суставов, мышц, лимфаденопатия).

Treatment:

1. With artalgia and fever - NSAIDs.

2. With damage to the joints and skin - aminoquinoline derivatives (plaquenil, chloroquine).

3. In severe form of the disease - immunosuppressive therapy (glucocorticoids, cytostatics).

4. With refractory thrombocytopenia - immunoglobulin preparations.

5. With intercurrent bacterial and viral infections - interferons.

6. In case of impaired renal function - hemodialysis.

medicinal fever

Severe fever is one of the forms of an allergic reaction to many drugs, accompanied by headache, muscle pain, chills. It is caused by many antibacterial

drugs (antibiotics, salicylates, sulfonamides), antihistamines, barbiturates, some heart drugs (novocainamide), less often - allopurinol, captopril, hydralazine, metindol, azathioprine, isoniazid, nitrofurans, PAS. A febrile reaction is known in cocaine and amphetamine addiction. Sometimes fever is pathogenetically associated with the introduction of sera. Of course, polypharmacy and allergic predisposition are risk factors for drug fever.

Malignant hyperthermia sometimes develops on the introduction of inhalation anesthetics (halothane, cyclopropane, halothane). Some scientists believe that there is a hereditary predisposition to such a hyperthermic reaction. The first signs of malignant hyperthermia are usually muscle rigidity, tachycardia and arrhythmia, acidosis, hypotension, and cyanosis of the skin. Late manifestations of the syndrome include pulmonary edema, DIC, and acute renal failure.

Hyperthermia is a common symptom of neuroleptic malignant syndrome in patients receiving phenothiazine derivatives, butyrophenone, and thioxanthene. The most common neuroleptic malignant syndrome develops when taking haloperidol.

Treatment: withdrawal of antipsychotics, use of bromocriptine (a dopamine receptor agonist), correction of metabolic and cardiovascular disorders. Mortality in neuroleptic malignant syndrome, unfortunately, is quite high - 20%.

Fever of non-infectious origin

Periodic disease is a disease of unknown etiology. The disease has a number of synonyms: familial polyserositis, Janeway-Mosenthal paroxysmal syndrome, periodic peritonitis, Reimann disease, Sigal-Kattana-Mamu syndrome, hereditary familial amyloidosis without neuropathy. It is more common in people of Mediterranean origin. Manifestations of the disease: transient and recurrent fever with abdominal pain and signs of peritonitis. Less common are pain in the chest (pleurisy) and large joints, erythema on the legs and feet, similar to that of erysipelas. Usually the attack lasts 1-2 days, the course is recurrent. ESR, leukocytosis, C-reactive protein, and serum fibrinogen often increase during an attack. The state of health between attacks is quite satisfactory.

Treatment is colchicine. The doctor faces great diagnostic difficulties with neurogenic hyperthermia, which often and for a long time proceeds as a fever of unknown origin. First of all, this hyperthermia is associated with a psychovegetative disorder and a violation of the central mechanisms of thermoregulation. At the same time, fever can be very diverse: it can be asymptomatic for a long time or be not only brightly psycho-emotionally colored, but also be accompanied by such clinical manifestations as excessive sweating, chills, heart pain, difficulty breathing (suffocation), a perversion of the daily physiological fluctuations in body temperature (morning fever). body temperature is higher than evening), discrepancy between body temperature and heart rate, etc., which, having suddenly arisen, can

also suddenly disappear.A thorough clinical and laboratory-instrumental study of persons with neurogenic hyperthermia usually does not reveal pathological changes in the internal organs with which hyperthermia could be associated. But psycho-emotional changes, sometimes quite pronounced, are often detected.

Taking into account the importance of psychogenic factors in the development of hyperthermia, the treatment of such patients should be carried out with the participation of experienced psychoneurologists and psychopharmacologists. Exudative pleurisy, peritonitis, pericarditis are also accompanied by fever, while effusion in these cavities with cirrhosis of the liver, nephritis, heart failure usually occurs without fever.

In some cases, fever may be the result of a reflex effect on the centers of thermoregulation (for example, when a stone passes through the ureter or biliary tract). These are general and far from all possible mechanisms for the onset of fever, which are inherent in various nosological forms to varying degrees..

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Hilson AJ (July 1995). "Pel-Ebstein fever". *N. Engl. J. Med.* 333 (1): 66–7. doi:10.1056/NEJM199507063330118. PMID 7777006.. They cite Richard Asher's lecture *Making Sense* (Lancet, 1959, 2, 359)