MINISTRY OF HEALTH PROTECTION OF UKRAINE

ODESSA NATIONAL MEDICAL UNIVERSITY

Faculty: medical
Department of propaedeutics of internal diseases and therapy

CONFIRMED by

Rector for scientific and pedagogical work

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METHODOLOGICAL DEVELOPMENT
TO THE LECTURE ON EDUCATIONAL DISCIPLINE

Faculty, course: medical, 3
Educational discipline: Propedeutics of internal medicine

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Lecture No. 1 "Propaedeutics of internal medicine as an introduction to the internal medicine clinic. Basic methods of examination of patients"

Actuality of theme. Propedeutics is the science of the basics of diagnosing internal diseases. During his studies at our department, the applicant must learn to independently examine a patient using all the simplest methods, must learn to independently identify the most important symptoms of diseases, compile them into syndromes and clinical diseases - form a diagnosis. This is the subject of propaedeutics or diagnostics of internal diseases.

The purpose of the lecture (goals):
To acquaint the applicant with the tasks of internal medicine as a scientific discipline:

1. Recognition of diseases of internal organs (diagnosis).
2. Study of etiology and pathogenesis of diseases of internal organs.
3. Development of issues of prevention and treatment of diseases of internal organs.

Determine the main tasks for applicants at the department of propaedeutics of internal diseases, which are:

1) method of clinical examination of patients;

2) symptomatology of diseases;

3) basics of laboratory and instrumental diagnostic studies for diseases of internal organs;

4) when acquainting applicants with the main nosological units (diseases) and syndromes, teach the ability to use, for example, the data obtained during the examination of the patient for the diagnosis of specific diseases.

Basic concepts: patient examination, patient complaints, propaedeutics, palpation, percussion, auscultation,

Plan and organizational structure of the lecture.
1. Determination of the educational goal.
2. Providing positive motivation.
3. Presentation of the lecture material according to the plan:
4. Summary of the lecture. General conclusions.
5. The lecturer's answer to possible questions.
6. Tasks for self-training.

Content of the lecture material

Propedeutics is the science of the basics of diagnosing internal diseases. During his studies at our department, the applicant must learn to independently examine a patient using all the simplest methods, must learn to independently identify the most important symptoms of diseases, compile them into syndromes and clinical diseases - form a diagnosis. This is the subject of propaedeutics or diagnostics of internal diseases.

The teaching about methods of recognizing diseases is called**diagnostics (ability to recognize). Diagnostics as a science studies** anatomical and physiological features of a person and his connections with the environment.

Diagnosis (recognition) - this short medical conclusion about the essence of the disease and the condition of the patient in the terms of modern medical science. The diagnosis is formed on the basis of specific symptoms or signs of the disease. The science of semiology deals with the study of the diagnostic value of symptoms.

Syndrome is called a set of symptoms that reflect pathogenetically related changes on the part of organs or systems of the body.

Principles of diagnosis formation.

When forming a diagnosis, one should observe certain features characteristic of medical science.

1. At**making a diagnosis** indicate:

a) the main disease at the time of communication with patients;

b) complications of this disease;

c) concomitant diseases, which at the time of communication with the patient may not require special treatment, but may or should be taken into account in the treatment of the main disease.

2. It is taken into account that there are the following**types of diagnosis:**

a) direct or similar;

b) differential diagnosis;

c) synthetic or complete diagnosis;

d) diagnosis by observation of patients;

e) diagnosis according to the therapeutic effect (ex juvantibus);

3. By the time of detection diseases are distinguished:

a) early diagnosis;

b) late diagnosis;

c) retrospective diagnosis;

d) postmortem diagnosis.

It is necessary to remember that the disease is a moving process (S.P. Botkin) and the diagnosis may change during the period of observation of the patient, his treatment.

4. To the extent of reasonableness distinguish:

a) preliminary (hypothetical) diagnosis;

b) final (justified) diagnosis;

c) diagnosis under a (doubtful) question mark.

5. There are**four sides** diagnosis:

a) morphological;

b) functional;

c) pathogenetic and pathophysiological;

d) etiological.

Methodology of diagnostics.

Establishing a diagnosis is the first task of a doctor when working with a patient. It is based on the following methods:

1. physical methods;
2. laboratory methods;
3. instrumental diagnostic methods;

4. surgical methods;
5. treatment;
6. observations.

When working with the patient, the doctor uses physical methods of diagnosis or diagnosis using the senses:

1. questioning the patient and his relatives;
2. general and local review;
3. palpation (feeling);
4. percussion (tapping);
5. auscultation (listening).

The conditions for the effectiveness of these methods are:

1. mastering the technique of their implementation;
2. absolutely objective application of these methods. It is impossible to give in to the expectation of the result from the first communication with the patient. For example, if when questioning the patient it turns out that he has a cough, then it is simply wrong to expect that he will have wheezing.

Questioning.

According to the virtuoso questioning of Professor G.A. Zakharyina, questioning is a "slow and difficult path". Meanwhile, the patient's subjective feelings are a guiding thread for the doctor in his work with the patient (S.P. Botkin). Questioning the patient, which is one of the methods of direct examination of the patient, plays an extremely important role in the diagnosis of many diseases of internal organs. The complaints that the patient presents during questioning, the characteristic stages of the development of the disease, some features of the patient's life history often make it possible to make a correct assumption about the diagnosis of the disease from the very beginning, which is later confirmed by other objective research methods.

Questioning the patient acquires such an important meaning only when it is carried out methodically competently, completely and in detail. If these conditions are not met, the patient can lead the doctor away from the correct diagnosis.

The most common shortcoming when questioning a patient is haste in conducting the questioning. The great Russian therapist Professor G.A. Zakharyn interrogated the patient sometimes for several hours. Unfortunately, in the first days of work in the department with patients, recruiters return from the ward 5-10 minutes after a conversation with the patient and sincerely believe that they have already asked the patient about everything. The quality of such questioning, as a rule, turns out to be extremely low, a lot of anamnestic information that is extremely important in terms of diagnosis is overlooked.

Complaints There are main and additional complaints, main and less significant ones. The patient is asked the questions "What are you complaining about?", "What is bothering you?". It is quite appropriate at the beginning of the conversation to give the patient the opportunity to express himself, and then, taking into account what he heard, ask the necessary additional questions. Thus, questioning continues even with the most active participation of the doctor. It must be remembered that the questioner at this moment is not interested in previous diagnoses, which the patient may begin to list, but in his subjective feelings of the disease. Brevity in this case is not the "sister of talent", but results in a diagnostic defect.

When questioning the patient, clarifying his complaints, first of all, they find out the presence or absence*pain syndrome*. When describing it, set:

1. Localization of pain
2. Irradiation, or spread, of pain
3. Duration of pain
4. Intensity of pain
5. Nature of pain (dull, burning, pressing, stabbing, squeezing, distending, drilling, pains)
6. Causes of pain
7. Factors increase the pain
8. Factors that relieve pain (medicines, physical factors - heat or cold, posture)
9. Associated symptoms of pain (nausea, dizziness, etc.)
10. Patterns of pain (if any).

All complaints made by patients are recorded in an edited form, preferably systematically. The words "editing of complaints" mean recording the patient's complaints in literary correct and understandable language. In addition, the patient may present complaints chaotically. The task of the doctor is to "sort" them syndromically or systematically, so that a certain picture of the disease emerges, to write them down in detail, briefly and clearly.

History of the disease.

When clarifying the history of the real disease, they will find out when, where and under what circumstances he got sick for the first time in his life. Reasons that caused the disease (according to the patient). Conditions preceding the disease (hypothermia, neuropsychological overfatigue, etc. They ask how, acutely or gradually, the disease began and how it first manifested itself. Then, in chronological order, the entire dynamics of symptoms and the appearance of new signs of the disease are described. The reasons for remission, their duration, as well as the causes and frequency of exacerbations of the disease.

They find out in detail when and to which medical institutions the patient applied. What types of examinations were performed on him and their results. At the same time, it is possible to use not only oral information from the patient, but also all the medical documents available to him (extracts, examination results, etc.). They will definitely find out what and with what effect the patient was treated. This information is valuable not only in terms of diagnosis, but also in choosing further treatment tactics. They find out how the patient's ability to work changed during the illness, the number of days of incapacity for work over the past year. The reasons for the actual hospitalization are specified (worsening of the course of the disease, examination to clarify the diagnosis, examination, etc.). And again, do not focus on the previous diagnosis, which was given to the patient in advance, because it may turn out to be incorrect or incomplete. If the patient is already in the hospital and comes under the observation of another doctor, the dynamics of the patient's condition in the hospital from the moment of curation to the day of the patient's examination are reflected. When collecting a medically competent anamnesis of the disease, it is unacceptable to allow "failures" filled with nothing, which sometimes reach several years.

The history of the patient's life.

When finding out the history of the patient's life, they start by finding out how childhood, childhood and youth passed. The place of birth of the patient and the age of his parents at the birth of the child are specified. They are interested in the nature of feeding the patient during infancy (natural breast or artificial). They find out the age when the patient began to speak, walk, study at school. They pay attention to the material wealth of the family, the nature of the patient's nutrition, the patient's lag behind his peers in physical and mental development. Find out living conditions, the nature of nutrition. When collecting the labor anamnesis, special attention should be paid not

only to the type of profession during the patient's life, but also to the nature of work and conditions at the workplace. Having found out the bad habits that the patient has, it is necessary to find out from what age and how many tobacco products the patient smokes per day, as well as the types of tobacco products used (cigarettes, cigarettes, pipes, etc.). When asking about the use of alcoholic beverages, it is necessary to find out in detail their types (vodka, wine, beer, surrogates, and so on), as well as to find out precisely from what age, how often and in what doses they are consumed by patients. Phrases like "Drinks on holidays", "Drinks like everything else" do not carry any useful information and can sometimes look funny. Be sure to be interested in the peculiarities of the sexual history, remembering that "any woman is suspicious of pregnancy." Therefore, you need complete information about previous pregnancies, their results (birth, miscarriage, medical abortion). It is necessary to know the regularity of menstruation and the date of the last menstruation. Then they find out what diseases the patient suffered from before. They are interested in detail in the allergy history. The questioning of the patient is completed by finding out the family anamnesis, the patient's heredity.
At the next lecture, the issues of general and local examination and construction of a clinical diagnosis will be considered.

General material and mass-methodological support
lectures:
1. work program of the academic discipline
2. synopsis (plan-summary) of the lecture
3. multimedia presentation of the lecture

Questions for self-control:

1. Describe the tasks of the department of propaedeutics of internal diseases.
2. Name the main sections of the ictopia of the disease.
3. What is the significance of general examination in general clinical diagnosis?
4. Plan for general inspection. Conditions and technique of general inspection.
5. The patient's condition and criteria for its assessment.
6. Consciousness, criteria for assessing consciousness.
7. Types of impaired consciousness: blackout, stupor, sopor, coma; their reasons.
8. Name the types of the patient's position.
9. What type of unconsciousness does fainting belong to?
10. For which diseases is facial blushing characteristic: a) unilateral, b) bilateral?
11. What type of cyanosis is observed with mitral stenosis
12. Explain the concepts: the symptom of "drum sticks", "Hippocratic nails", "vascular asterisks", "carotid dance".

References:
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2. Methods of objective examination in the clinic of internal diseases: training. manual / O.O. Yakymenko, O.E. Kravchuk, V.V. Klochko et al. - Odesa, 2013. - 154 p.

3. Diagnostic methods in the clinic of internal medicine: study guide / A.S. Svintsitskyi. -K.: VSV "Medicine", 2019. – 1008 pp. + 80 pp. color. incl.
4. Methods of examination of a therapeutic patient: teaching. manual / S.M. Andreychyn, N.A. Bilkevich, T.Yu. Chernets. – Ternopil: TDMU, 2016. – 260 p.
5. Questioning and physical examination of a patient with a therapeutic profile: Training. manual for students III-IV courses of medicine. of universities / V. E. Neiko, I. V. Tymkiv, M. V. Blyznyuk [and others]. – Iv.-Frankivsk: IFNMU, 2016. – 142 p.

Electronic information resources:

1. http://moz.gov.ua - Ministry of Health of Ukraine
2. www.ama-assn.org – American Medical Association /American Medical Association
3. www.who.int - World Health Organization
4. www.dec.gov.ua/mtd/home/ - State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk – British Medical Association
6. www.gmc-uk.org - *General Medical Council* (*GMC*)
7. www.bundesaerztekammer.de – German Medical Association
8. https://onmedu.edu.ua/
9. http://www.kolos2401.net/load/
10. https://onmedu.edu.ua/kafedra/propedevtiki-vnutrishnih-hvorob-ta-terapii/

Lecture No. 2 "Symptoms in diseases of the respiratory organs based on questioning the patient, palpation, percussion of the chest and auscultation of the lungs"

1. Relevance of the topic.

Questioning of patients with respiratory diseases, examination and palpation of the chest, vocal tremor are among the main methods of research, with which the examination of the patient practically begins. Research methods are simple, do not require any equipment, are available in any conditions, and the information obtained after these studies often helps to diagnose or suspect a particular disease.

Topographic percussion of the lungs is one of the main methods of objective examination of the patient, its use is available under expensive circumstances, and does not require any special equipment. To date, topographic percussion provides important diagnostic data and cannot be eliminated by other methods.
Auscultation of the lungs, as well as their percussion, is one of the basic methods of objective research, they are available in any circumstances and do not require any complex equipment.
To this day, auscultation of the lungs provides the most important diagnostic information and cannot be replaced by other, more complex, auxiliary research methods

2. Purpose of the lecture:

Acquaint applicants with modern concepts of questioning of patients with lung diseases, determination of pathological types of breathing, examination and palpation of the chest, determination of voice tremors, types of shortness of breath and suffocation, auscultation of the lungs.
the acquirer must master:
- definition of the main and pathological types of breathing;
- chest palpation, diagnostic value of palpation;
- method of voice tremor;
- know the types of shortness of breath and suffocation.
- types, methods and techniques of lung auscultation.
- the mechanism of vesicular breathing and the place where it is best listened to.
- causes of physiological and pathological weakening and strengthening of vesicle respiration.
- causes of pathological bronchial breathing.
- to be able to perform auscultation of the lungs.
- to be able to determine the breathing of a vesicle.
- to be able to define bronchial breathing.
- types of secondary respiratory noises.
- mechanism and places of occurrence of dry and wet rales, crepitation and pleural friction noise.
- definition of bronchophonia, diagnostic values of its strengthening and weakening.
- features of auscultation of the lungs in the case of syndromes of focal compaction of the lungs, formation of a cavity in the lungs, accumulation of air in the pleural cavities.

Master:
- methods and techniques of questioning patients with respiratory diseases;
- the ability to identify pathological types of breathing;
- by the method of examination and palpation of the chest;
- by the method of vocal tremor;
- the ability to identify types of shortness of breath and suffocation;
- To master the method of differential diagnosis of secondary respiratory noises.
- Master the method of conducting bronchophony.
- Master the method of differential diagnosis of clinical syndromes in lung diseases.
3. Basic concepts: palpation, percussion, organs of the respiratory system, examination of the patient
4. Plan and organizational structure of the lecture.
1. Determination of the educational goal.
2. Providing positive motivation.
3. Presentation of the lecture material according to the plan:
4. Summary of the lecture. General conclusions.
5. The lecturer's answer to possible questions.
6. Tasks for self-training.

Content of the lecture material
Study scheme of patients with respiratory diseases
1 stage. Subjective research (interrogation of the patient):

1. Complaints.
2. Anamnesis of the disease.
3. Anamnesis of life.
2 stage. Objective research:
1. General overview.
2. Examination of the chest.
3. Palpation of the chest.
4. Percussion of the chest.
5. Auscultation of the lungs.
6. Research of other organs and systems (clinical).
7. Laboratory research methods.
8. Instrumental research methods (spirography, radiography).

Complaints

A. *Shortness of breath(dyspnoe)* — by origin can be:
- due to impaired function of the respiratory system;
- due to pathology of the cardiovascular system;
- due to violation of the transport function of blood;
- due to the pathology of enzymes of the Krebs respiratory cycle of organs and tissues;
- craniocerebral injury (central genesis).

Shortness of breath when the function of the respiratory system is impaired can be the result of the following reasons:
- on the part of the respiratory tract — an obstacle to the passage of air;
- on the part of the lung tissue — a decrease in the area of the respiratory surface of the lungs, a decrease in the elasticity of the lung tissue;
- from the side of the pleura — fluid accumulation in the pleural cavity, adhesions between the parietal and visceral leaves of the pleura;
- on the part of respiratory muscles — weakness, paresis or spasm;
- from the side of the chest - ossification of cartilage, decrease in its mobility, fracture of ribs.

The appearance of shortness of breath in all cases is associated with hypoxia and hypercapnia and the accumulation of underoxidized metabolic products in the blood with the development of acidosis.

According to its nature, pulmonary dyspnea can be:
- inspiratory, in which breathing is mainly complicated; characteristic of mechanical obstruction in the upper respiratory tract (nose, pharynx, larynx, trachea). At the same time, breathing is slowed down, and with pronounced narrowing of the airways, breathing becomes loud (stridorous breathing).
- expiratory shortness of breath — with difficult exhalation, observed with a decrease in the elasticity of the lung tissue (emphysema of the lungs) and with narrowing of the small bronchi (bronchiolitis, bronchial asthma).
- mixed shortness of breath - both phases of respiratory movements are complicated, the reason is a decrease in the area of the respiratory surface (in case of inflammation of the lung, pulmonary edema, compression of the lung from the outside - hydrothorax, pneumothorax).

Very strong shortness of breath bordering on asphyxia is called suffocation. Suffocation that occurs in attacks is called asthma. (For example, bronchial asthma, cardiac asthma). When

conducting the survey, it is necessary to remember that shortness of breath can be subjective (feeling of difficulty breathing in the form of chest tightness, inability to fully straighten the chest during inhalation or release the chest during exhalation, feeling of lack of air). It is subjective shortness of breath that is revealed when questioning the patient. Objective shortness of breath is determined by objective methods of research: change in the frequency, rhythm and depth of breathing, participation in the act of breathing of the auxiliary muscles of the upper shoulder girdle. In addition, shortness of breath can be physiological (during physical exertion or emotional stress) and pathological.

B.*Cough (tussis)* — is a complex reflex act that occurs as a protective reaction when secretions, sputum, blood accumulate in the larynx, trachea, bronchi, or when foreign bodies get into them.

Coughing is an important protective mechanism for cleaning the bronchial tubes from excessively accumulated secretions. It occurs when the receptors of the vagus nerve are irritated in the cough reflexogenic zones of the mucous membrane of the larynx, vocal cords, bifurcation of the trachea and areas of division of large and medium bronchi. There are no cough reflex receptors in the small bronchi.

The mechanism of a cough impulse is reduced to a deep breath followed by a sudden and intensified exhalation, and the beginning of exhalation occurs when the glottis is closed. A cough impulse is "a shot of air through a narrowed glottis."

When evaluating a cough, it is necessary to pay attention to its rhythm, timbre and character, to the time of its appearance and the circumstances under which it appears.

1. Rhythm:

1.1 cough in the form of individual cough impulses, otherwise coughing. It is observed in laryngitis, tracheobronchitis, often in smokers;

1.2 cough in the form of a series of successive cough impulses, repeated with some intervals (pulmonary-bronchial cough);

1.3 paroxysmal cough — when a foreign body enters the respiratory tract, with whooping cough, bronchial asthma, with pulmonary cavities, with damage to the bronchial lymph nodes;

2. By timbre:

2.1 a short and cautious cough, which is usually accompanied by a painful grimace (dry pleurisy, the beginning of croupous pneumonia);

2.2 barking cough (swelling of the false vocal cords, compression of the trachea by a tumor or goiter, damage to the larynx, hysteria);

2.3 whooping cough (inflammation of the vocal cords);

2.4 silent cough (ulcer of the vocal cords, swelling of the vocal cords, sharp general weakness);

3. By nature:

3.1 dry cough (without sputum);

3.2 wet cough (with sputum);

4. By time of appearance:

4.1 morning cough (chronic inflammation of the upper respiratory tract) — cough "while washing";

4.2 evening cough (bronchitis, pneumonia);

4.3 nocturnal cough — associated with a nocturnal increase in the tone of the vagus nerve (pulmonary tuberculosis, an increase in bronchopulmonary lymph nodes);

5. According to the conditions under which it occurs or according to the phenomena accompanied by:

5.1 cough that occurs when the position of the body changes — is observed in the presence of a cavity in the lung (bronchoectatic disease, abscess, gangrene of the lung, cavernous tuberculosis) — the contents of the cavity in a certain position are released into the bronchus and coughed up (paying attention to the position in which there is a cough, it is possible to establish the localization of the cavity);
5.2 cough that occurs when eating — is observed when the esophagus is connected to the trachea or bronchus (esophageal cancer covered with ulcers and broke into the bronchus);
5.3 cough, accompanied by discharge of large amounts of sputum ("full mouth") — characteristic of emptying the cavity;
5.4 cough accompanied by vomiting - with whooping cough in children, with pharyngitis (due to irritation of the sensitive mucous membrane of the pharynx by viscous sputum);
IN.*Sputum discharge.* Phlegm (sputum) is the secretion from the respiratory tract that is thrown out when coughing. Sputum is always a pathological phenomenon. The mechanism of sputum secretion includes three points: action of the ciliated epithelium by the mucous membrane of the bronchi; contraction of bronchial muscles; coughing fit.
To evaluate sputum as a sign (symptom) of respiratory diseases, when questioning the patient and during direct examination of sputum, it is necessary to take into account: amount, consistency, nature, color, smell, impurities.
1. The amount varies greatly from insignificant (traces) to 1-2 liters per day. Usually, when questioning the patient, they focus on the volume of the faceted glass. A large amount of sputum indicates the presence of a cavity in the lung.
2. Sputum consistency — the more mucus, the more viscous the sputum and vice versa.
3. Character of sputum:
3.1 mucous membrane — viscous, colorless and transparent or whitish (at the onset of acute pneumonia, bronchitis);
3.2 serous — liquid transparent or opalescent (resembles a soapy solution) — characteristic of pulmonary edema;
3.3 purulent — greenish creamy consistency (when an abscess breaks through a bronchus);
3.4 muco-purulent — the most frequent type — typical for most inflammatory processes of the bronchi and lungs;
3.5 serous-purulent — when standing, it is divided into 3 layers: upper (foamy), middle (liquid), and lower (lumpy-pulp purulent). Such 3-layer sputum can be observed with lung gangrene.
3.6 bloody — contains an admixture of blood. Blood can come from different parts of the respiratory tract or mix with sputum in the oral cavity. Distinguish: sputum with blood in the form of veins or blood clots (tuberculosis, bronchiectasis, tumors), pink sputum (lung edema), rusty sputum (croupous pneumonia), crimson color (lung tumor), black color (lung infarction) .
4. Color of sputum: can be very diverse. For example, greenish with purulent sputum, red with an admixture of blood.
5. The smell of sputum: absent in most cases, but with gangrene of the lungs with a foul putrid smell.
6. Impurities: food in the presence of a fistula between the trachea and esophagus, tumor particles in a lung tumor, blood in hemoptysis.
D.*Haemoptoe*. It is observed in pulmonary tuberculosis, viral pneumonia, lung tumor, abscess and lung gangrene. In diseases of the cardiovascular system, hemoptysis occurs in the case of blood

stagnation in the small circle of blood circulation (mitral stenosis), thrombosis or embolism of the vessels of the pulmonary artery (PE) and the development of lung infarction.
Hemoptysis may be in the form of streaks of blood in the sputum or blood may diffusely color the sputum. The blood released when coughing can be fresh (bright red) or changed. Bright red blood in sputum occurs in pulmonary tuberculosis, bronchogenic lung cancer.
With croup pneumonia, sputum of a rusty color is released due to the breakdown of erythrocytes and the formation of hemosiderin pigment. In cases where the amount of blood secreted with sputum exceeds 50 ml, it is said to be pulmonary bleeding. Pulmonary bleeding is observed in pulmonary tuberculosis, lung cancer, bronchiectasis, and lung abscess.
Pulmonary bleeding must be distinguished from gastrointestinal bleeding: with gastrointestinal bleeding, the blood is dark in color with food impurities, the reaction is acidic, and with pulmonary bleeding, the blood is bright red, foamy, with air bubbles, alkaline reaction, accompanied by a strong cough.
D.*pain*. Pain associated with a disease of the respiratory system is caused mainly by damage to the pleura, since damage to the lung tissue does not cause pain. The pain is usually localized in the chest, especially in its lateral parts ("pain in the side"). When the diaphragmatic pleura is damaged, the pain is localized in the upper part of the abdomen. A characteristic sign of pleural pain is their intensification during inhalation, deep breathing, and coughing. Most often, pleural pains are observed with dry pleurisy due to friction of the pleural leaves against each other.
If the pleurisy is exudative (exudative), then the pain will bother you only in the initial period of the disease, while with a small amount of exudation (exudate), the pleural leaves are still in contact with each other. To reduce pain, patients try to breathe shallowly, delay coughing impulses, lie on the side of the pleural lesion (to reduce chest excursion). Pleural pain intensifies when the body leans in the healthy direction.
Among lung diseases, pain is accompanied by those in which the pleura is involved in the pathological process (croupous pneumonia, lung infarction, pulmonary tuberculosis). Very severe pain occurs when the pleura is affected by a tumor process. Pain in the chest of pleural origin must be distinguished from pain of other genesis:

- with shingles (herpes zoster);
- with defeat of intercostal muscles (myositis);
- when the ribs are damaged (fractures);
- with damage to the intercostal nerves;
- pains of cardiovascular origin;
- pains in case of damage to the liver and biliary tract (reflected pains).

All these pains have their distinctive features.
In addition to the 5 main complaints, patients with respiratory diseases may have general complaints: headache, fever, chills, sweating, weakness, deterioration of health, decreased appetite, etc.

Anamnesis of the disease

An anamnesis of the disease in the case of damage to the respiratory organs significantly helps to recognize the disease. Thus, a sudden onset with chills and a rise in temperature to high numbers, pain in the side and a cough with rusty sputum make the diagnosis of croup pneumonia almost unquestionable. Gradual onset of the disease, periods of exacerbation (in spring and autumn) and remission, increasing shortness of breath, cough with sputum are characteristic of progressive chronic bronchitis.

Anamnesis of life

Life anamnesis allows identifying risk factors for bronchopulmonary pathology: living in environmentally unfavorable areas with dusty and polluted atmosphere, occupational hazards, smoking (including passive), alcohol consumption, socio-economic factors, contact with a tuberculosis patient, hereditary predisposition, allergic heavy anamnesis.

Present state

The overview provides a lot of valuable information. They pay attention to whether there is a forced position. For example, during an attack of bronchial asthma, the patient sits, leaning on her hands (hanging on her hands).

In some patients, bluish staining of the lips, skin of the hands, face (cyanosis) can be noted - occurs in inflammation of the lungs, obstructive bronchitis, pulmonary tuberculosis. The cause of cyanosis is a violation of gas exchange in the lungs with hypoxia and hypercapnia of the blood. With croupous pneumonia, it is possible to detect hyperemia (blush) of the face on one side, which corresponds to the affected lung due to irritation of the corresponding sympathetic nerve. In some patients, the symptom of "drumsticks" (thickening of the terminal phalanges of the fingers) and the symptom of "hour glasses" (bulging of the nail plates) can be detected. These symptoms are characteristic of long-term and debilitating diseases (pulmonary suppuration, bronchiectasis).

Examination of the neck may reveal enlarged cervical lymph nodes in patients with pulmonary tuberculosis.

A.*Examination of the chest*. It is better to perform it with the patient standing or sitting with the body bare to the waist in uniform daylight and a temperature of about 20-25 C. The chest is examined sequentially from top to bottom, front to back, right to left (or from the healthy side to the patient):

1. We determine the shape of the chest, taking into account: the ratio of front-back and side dimensions, the prominence of the supraclavicular and subclavian fossae, Louis's angle, the size of the epigastric angle, the course of the ribs in the lateral sections, the location of the shoulder blades. The shape of the chest can be: physiological, pathological. The physiological chest is distinguished by the symmetry and harmony of all its lines (some predominance of the more developed right half is allowed). There are: normosthenic, asthenic, hypersthenic forms of the chest. Variants of a pathological chest:

1.1 Emphysematous - shortened, sharply expanded, barrel-shaped, as if in the position of maximum inspiration with horizontally located ribs, high raised shoulders, short neck - this is an inspiratory form of the chest with the most sharply expressed features of the chest of a hypersthenic. This form of the chest occurs during an attack of bronchial asthma and emphysema of the lungs.

1.2 Paralytic chest — sharply elongated, flattened, as if lowered and in the position of maximum exhalation. The ribs are strongly inclined downward, the clavicles protrude sharply, the supraclavicular and subclavian fossae sink, the shoulder blades lag behind the chest (scapulae alatae) - this is the expiratory form of the chest. It occurs in severely emaciated persons of asthenic physique with tuberculosis, lung cancer.

1.3 Funnel-shaped chest - cobbler's chest - the chest has an anomaly in the form of a depression corresponding to the lower part of the sternum.

1.4 Boat-shaped ribcage — an indentation, similar in shape to the indentation of a boat, is located in the upper and middle part of the sternum (in contrast to the funnel-shaped ribcage).

1.5 A rachitic thorax — has two characteristic features: a sharply protruding breastbone in the form of a vertical ("chicken breast") or horizontal protrusion, chetsuobraznye thickenings at the place of transition of costal cartilages into the bone — "rachitic rosaries".
2. We determine the deformation of the chest: bulging parts of the chest, depression of parts of the chest, location of clavicles, location of shoulder blades, change of the spine.
2.1 With curvature of the spine: to the side (scoliosis), back (kyphosis) with the formation of a hump (gibbus), forward (lordosis), combined curvature of the spine to the side and back (kyphoscoliosis). These deformations of the chest lead to severe disorders of the function of the respiratory system.
2.2 Unilateral deformations of the chest:

2.2.1. An increase in the size of one half of the chest: accumulation of fluid in the pleural cavity (hydrothorax), accumulation of air in the pleural cavity (pneumothorax), and simultaneously fluid and air in the pleural cavity (hydropneumothorax).

2.2.2. Reduction in the size of one half or part of the chest: removal of part or the whole lung (lobectomy, pulmonectomy) in tuberculosis, lung cancer, shrinkage of part of the lung in the growth of connective tissue after pulmonary tuberculosis, pneumonia, collapse of part of the lung (atelectasis), development of pleural adhesions. A decrease or increase in the volume of one of the halves of the chest leads to the fact that the chest becomes asymmetrical.
3. Symmetrical participation of both halves of the chest in the act of breathing. Lagging of one of the halves of the chest in the act of breathing can be observed when the volume of one of the halves of the chest decreases or increases, as already mentioned above.
In addition, the lag of one half of the chest in the act of breathing can be observed even with a physiological chest without deformations in cases of croupous pneumonia, lung abscess, etc. pathological processes leading to a one-sided decrease in the area of the respiratory surface. Increases the lag of the affected half of the chest in the act of breathing, involving the pleura in the pathological process.
4. Determination of the type of breathing, rhythm and frequency of breathing per minute.
4.1 Type of breathing. Physiological variants: thoracic (costal), abdominal (diaphragmatic), mixed.
If breathing movements are performed due to the contraction of the intercostal muscles, then they speak of the thoracic type (characteristic of women). With the abdominal type of breathing, respiratory movements are performed due to the contraction of the diaphragm (more often found in men). With a mixed type of breathing, respiratory movements occur due to the contraction of the intercostal muscles and the diaphragm (occurs in the elderly). In the conditions of pathology, the type of breathing, usual for this person, can deviate. For example, the abdominal type of breathing turns into chest breathing in ascites, appendicitis, hepatomegaly. The thoracic type of breathing turns into abdominal breathing in the case of lung pathology (tuberculosis, pneumonia) or pleurisy (pleurisy), as well as chest pain (intercostal neuralgia, dry pleurisy). Abdominal or thoracic breathing becomes mixed during an attack of bronchial asthma.
4.2 Breathing rate. The norm for an adult at rest is 16-20 per minute, for a newborn 40-45 per minute. With age, the frequency of respiratory movements decreases. Physiological reduction (bradipnoe) in the frequency of respiratory movements is observed in sleep (12-14 per minute). An increase (tachipnoe) of the frequency of respiratory movements is normally observed during physical and neuropsychological overstrain. In pathological conditions, the reduction of the CKD is observed with narrowing of the larynx or trachea (slowing of inhalation), and often with

narrowing of small bronchi during an attack of bronchial asthma (slowing of exhalation). The frequency of CKD in pathological conditions is observed with fever, with most diseases of the respiratory organs, gases that hinder exchange in the lungs (pneumonia, emphysema, atelectasis, hydro- and pneumothorax, and the like), as well as with diseases of the circulatory system.
The rhythm of breathing should be correct under physiological conditions. In a number of pathological conditions, the breathing rhythm is disturbed. If a violation of the breathing rhythm is repeated in a certain sequence, then such breathing is called periodic.
4.3 Variants of periodic breathing: Cheyne-Stokes breathing, Biot's breathing, Grock's breathing - (wave-like breathing). The rhythm is disturbed, in addition to pauses, due to changes in the depth of breathing (Kussmaul breathing), the duration of inhalation (inspiratory dyspnea) and the duration of exhalation (expiratory dyspnea).
B. Palpation of the chest. Task: clarification of examination data, determination of chest tenderness, determination of chest elasticity (resistance), determination of voice tremor. By palpation, we clarify the shape of the chest, the symmetry of the participation of both halves of the chest in the act of breathing. Palpation can reveal the localization of pain in the chest, its prevalence in pathology of the ribs, muscles, inflammation of the intercostal muscles — intercostal neuralgia.
Pleural pains are not palpable. Pain in the chest of pleural origin intensifies when the trunk is tilted in the healthy direction. Pleural pain is reduced if the chest is immunodeficient by squeezing it from the sides with the hands (Yanovsky's symptom).
In a healthy person, the chest is elastic (elasticity is determined by pressing on the chest in the anterior-posterior direction (sternum - spine) and in the lateral sections). The elasticity of the chest decreases with emphysema of the lungs (increased air filling of the lungs), with hydrothorax (fluid in the pleural cavity), atelectasis (collapse of the lungs), large inflammation of the lungs, with tumors of the pleura and lungs, as well as in old age.
Vocal tremor (cremitus vocalis) is a palpable determination of the conduction of the voice to the chest. Voice tremor is determined by placing the palms or end phalanges of the fingers on symmetrical places of the chest. In a healthy person, voice tremors are felt approximately the same in symmetrical areas, somewhat louder in the upper areas, weaker in the lower areas. In pathological conditions of the respiratory organs, vocal tremor can be increased, weakened or not determined at all.
Increased vocal tremor - when a large area of lung tissue or the entire lung becomes dense, airless, provided that the patency of the bronchus is preserved (croupous pneumonia, tuberculosis, compression atelectasis). Alleviation of vocal tremor - when fluid or air accumulates in the pleural cavity, when a large bronchus is blocked by a foreign body or a tumor. With emphysema of the lungs, there is a uniform, symmetrical weakening of the voice tremor. Under physiological conditions, weakening of voice tremor is observed in obesity. Absence of voice tremor is observed with a large exudate in the pleural cavity.
V. Percussion. Percussion (percussio) - (Latin literally "through the skin") is a research method based on tapping on the surface of the body with a simultaneous evaluation of the received sounds. Percussion was known to ancient doctors, but the birth date of the percussion method should be considered the year 1761, when the Viennese doctor Auenbrugger published his work on direct percussion (tapping with the ends of folded and half-bent fingers of the right hand directly on the chest). In 1827, Piorri proposed indirect percussion instead of direct percussion, making blows with a finger on a plesymeter applied to the body, while receiving clearer and more

expressive blows. In 1841, Wintrich proposed intermediate percussion using a hammer and a plesymeter (instrumental stage of percussion).
Currently, the finger-toe method of percussion is used. Advantages of this method: the doctor is independent of the instrument, the finger plesymeter is convenient and easily adapted to any surface of the body, the assessment of percussion is made on the basis of both acoustic and tactile sensations.
When tapping on the human body, oscillatory movements of organs and tissues located deep from the place of percussion occur. The nature of these blows (amplitude, frequency, duration) are determined by the structure of the relevant organs, the state and properties of the tissues, as well as the force of the percussive blow. Percussion of the chest gives all three main types of percussion tone: clear (pulmonary), blunt, tympanic.
A clear lung sound is obtained when percussing those areas of the chest where unchanged lung tissue lies directly behind the chest. Pulmonary sound is long, low, loud, non-tympanic.
Dull or muffled sound - comes out on the chest wherever dense parenchymal organs are adjacent to it - heart, liver. In pathological conditions, a dulled percussion sound is observed when the lightness of the lung tissue decreases or when the pleural cavity is filled with liquid. The sound is high, quiet, short, non-tympanic.
A tympanic sound is produced by percussion of the chest wherever cavities containing air are adjacent to the chest. The sound is loud, long, low or high. Normally, a tympanic sound occurs when percussing the abdomen over the intestines and stomach.
Tasks of lung percussion: detection of pathological foci (processes) in the lungs and pleural cavities, determination of lung boundaries, mobility of the lower edge of the lungs, boundaries and dimensions of the pathological foci. In this regard, a distinction is made: comparative percussion, topographical percussion. First, comparative percussion is performed.
Percussion is best performed in a vertical or sitting position of the patient; during percussion standing, the patient's hands should be down, while sitting - hands on the knees. Stripping the patient to the waist. Percussion is performed with calm, steady breathing.
When performing comparative percussion, the following rules should be followed: percussion should be strictly on symmetrical places, the force of the percussion blow should be the same - medium, percussion should be started from the right side or from the healthy side to the patient. Comparative percussion is performed in a certain sequence — from top to bottom, front to back.
Percussion in the following symmetrical points:
1. Above the collarbones (top of the lungs);
2. On the clavicles (clavicle — plesymeter);
3. Under the collarbones (1 intercostal space);
4. 2nd intercostal space along the mid-clavicular line;
5. 4th intercostal space 1 cm lateral to the mid-clavicular line;
6. 6 intercostal space along the middle axillary line;
7. Above the shoulder blades;
8-9. In the interscapular region 2 times: at the level of the upper corner and the lower corner of the shoulder blades, the plesimeter finger is placed vertically, the shoulder blades are maximally moved to the sides, for which the patient crosses his arms on his chest;
10. Under the shoulder blades (6th intercostal space) — the patient's hands are down.

In a healthy person, the sound may not be exactly the same during comparative percussion at symmetrical points. So, above the right apex, the sound is somewhat quieter and shorter in the right axillary region compared to the left.
With pathological processes, the percussion sound can change either towards dull or tympanic. In the direction of dullness, the following are distinguished: shortening, dulling, dull sound, absolutely dull sound (femoral dullness).
These changes appear in cases when the lightness in the lungs decreases or the lightness is lost completely, the tissue becomes denser (in the early stages of croupous pneumonia); blunting - with focal pneumonia, dull, - the lung is airless - granular inflammation in the stage of "burning" (the lung becomes dense like the liver); dull sound in atelectasis. An absolutely dull sound is determined by the accumulation of fluid in the pleural cavities above the projection of this fluid.
Tympanic shade or tympanic sound - when the lungs contain a lot of air (for example, in emphysema - a box sound, a type of tympanic sound).
Local appearance of tympanic sound - in the presence of a cavity in the lung (abscess, tuberculous cavity), if it is emptied. A tympanic sound with a metallic tone is observed with an open pneumothorax.
Topographic percussion. The main rules of topographic percussion: the force of the percussive blow is quiet, the plesymeter finger is located parallel to the determined boundary, the movement of the plesymeter finger by a width of no more than 1 finger or 1 cm. Adherence to the sequence: determining the location of the vertices in front, determining the location of the vertices in the back, determining the lower borders of the lungs along vertical lines, determining the excursion of the lower lung border along vertical lines, determining the borders and sizes of the pathological center.
The upper borders of the lungs in front are located in physiological conditions 3.5-4 cm higher than the collarbones. The right apex may be lower than the left one by 0.5-1 cm Behind - at the level of the spinous process of the VII cervical vertebra. In pathological conditions, the vertices are located higher in emphysema of the lungs. The lower limits of the lungs can be lowered during pregnancy, ascites (accumulation of fluid in the abdominal cavity), flatulence (accumulation of air), large tumors in the abdominal cavity, respiratory diseases: exudative pleurisy, hydrothorax in general and pneumothorax. The lower borders of the lungs can be lowered only with emphysema of the lungs. The mobility of the lower lung edge is determined by the axillary or line (more often along the back axillary line) of the scapula on one side or the other. In a healthy adult, it is 6-8 cm.
Auscultation is a method based on listening to sounds over individual parts of the human body and analyzing the results. Auscultation can be done directly, without the use of special tools, by applying the ear to the part of the body of interest, as well as using special tools - stethoscopes and stethoscopes. Organ activity is usually accompanied by sound phenomena. Sound is a wave, the oscillation of air masses, it is carried to the surface of the body, from where sound phenomena are heard by those who investigate. When using a phonendoscope or stethoscope, sound vibrations are not dispersed, but, on the contrary, are captured and transmitted through the tubes of the phonendoscope directly to the hearing aid. The main condition for conducting auscultation is to create the maximum possible silence in the room where the research is conducted. During auscultation, the patient can lie or sit. The stethoscope is placed tightly against the skin for the best listening experience.
Usually listening is done over the lungs: during inhalation or exhalation, respiratory noises are created during the passage of air through the respiratory tract (physiological and pathological -

wheezing, crepitation, pleural friction noise). Sound phenomena in the heart that occur during the movement of blood during heart contractions (physiological - tones, pathological - noises) are no less often heard. Some noises are heard in the position of lying on the left side or after physical exertion. To better listen to the patient's heart, you can ask him to hold his breath. Sometimes the patient is asked to cough: after coughing, the pleural friction noise persists, and wheezing may disappear.

Auscultation of the lungs and heart is of great importance in the diagnosis of diseases of the bronchopulmonary and cardiovascular systems. Data obtained during auscultation of the heart correspond to phonocardiogram data registered graphically. Auscultation is also used to listen to intestinal sounds and the noise of friction of the peritoneum, noises over pathologically changed vessels (with stenosis and aneurysm of arteries).

Theoretical knowledge and experience are extremely important when conducting auscultation. When correctly applied, this method in combination with other clinical research methods allows you to correctly make a preliminary diagnosis in the vast majority of cases, without resorting to instrumental research methods. The use of laboratory and instrumental methods only confirms and clarifies the diagnosis. Auscultation is indispensable when heart pathology is detected during preventive examinations. This method should be perfectly mastered by general practitioners, as they are the ones who first come across patients with rheumatic heart disease, which do not have clinical manifestations, and patients with lung diseases.

Physiological and pathological respiratory noises

1. Vesicle respiration: mechanism, physiological and pathological options. Bronchial breathing, its characteristics, varieties, mechanism of formation

Noises arising during breathing are divided into physiological (or basic) and pathological (or additional).

The main sounds include the breathing of the vesicles, which is heard over the entire surface of the lung tissue, and bronchial breathing, which is heard over the projection of the upper respiratory tract (larynx, trachea, large bronchi) onto the surface of the front chest wall.

Additional noises include crepitation, wheezing, pleural friction noise.

In addition, with various diseases, the main respiratory noises can change their qualities, intensify, weaken, and then they are called pathological.

Breath vesicles heard when the patient breathes through the nose. It is a soft, quiet, blowing sound.

Its strengthening or weakening under normal conditions may depend on the thickness of the chest wall and physical work. Pathological increase in vesicle breathing in the expiratory phase indicates bronchospasm, and in both phases of breathing - the presence of hard breathing.

Saccade breathing - increased breathing, in which due to the contraction of the respiratory muscles (for example, when trembling), the breath becomes intermittent. Sometimes a pathological weakening of the breathing of the vesicle is heard. Since the mechanism of vesicle breathing is related to the oscillation of the walls of the pulmonary alveoli (the sound effect occurs when air enters the alveoli), its weakening is due to a violation of the oscillation of the alveolar walls or a violation of the transmission of sound effects to the front chest wall. The first situation

may be associated with impregnation of the walls with inflammatory exudate or rigidity of the walls of the alveoli. The second occurs in the presence of fluid in the pleural cavity - it dampens sound vibrations (with hydrothorax, hemothorax or pleural empyema) or air (with pneumothorax).
Weakening of the breathing of the vesicle may be associated with mechanical reasons: violation of the passage of air through the respiratory tract (partial obturation) or restriction of respiratory movements, for example, with intercostal neuralgia, when the act of inhalation is accompanied by sharp pain.
Normal**bronchial breathing** is heard over the projection of the larynx, trachea and its bifurcation. Pathological bronchial breathing is heard in certain cases above the surface of the lungs, where bronchial breathing is normally detected.
The reason for its appearance is the lack of breathing of the vesicle over the area where the lung is compacted. In this area, the oscillations of the alveolar walls are insignificant. This occurs as a result of certain reasons (for example, when squeezing (atelectasis) of the lung).
There are pathological types of bronchial breathing**stenotic** or**amphoric** breath. The latter occurs with a cavernous or large emptied abscess that connects to a large bronchus.

2. Incidental breath sounds. Dry wheezing. Wet wheezing. Crepitation. Pleural friction noise

Wheezing, crepitation and the noise of pleural friction are secondary respiratory noises, they are never heard in a healthy person.
Wheezing is formed in the bronchi and is heard above their projection on the chest wall.
If there is a narrowing of the bronchus in the air path, the air, passing through the narrowing, causes the appearance of a sound that resembles a whistle. These are dry rales. If liquid secretion accumulates in the bronchi, wet wheezing occurs when air passes through it. In relation to the phases of breathing, wheezing (both dry and wet) is heard in both phases of breathing. Dry wheezing causes sounds of different pitch (depending on the diameter of the bronchi), at the level of which there is a narrowing. Small bronchi are the source of high-pitched wheezing, whistling wheezing, and large - low, humming.
Wet wheezing, depending on the diameter of the bronchi, in which liquid content accumulates, is divided into small-vesicular (occurring in small bronchi and bronchioles), medium-vesicular (occurring in medium bronchi) and large-vesicular (occurring in large bronchi). The reason for the appearance of wet wheezing is the passage of air through the liquid contained in the bronchi.
Loudness of wheezing depends on the effect of sound resonance. It is known that dense fabric conducts sound better. If the liquid content in the bronchi is surrounded by compacted lung tissue, resonance occurs and the sound is amplified. If the bronchus, containing liquid contents, is surrounded by air tissue of the lung, soundless wheezing occurs.
To distinguish wheezing from other breathing noises, after listening to them, the patient is asked to cough. After coughing, sputum - the cause of wheezing - is removed from the bronchi, and the wheezing itself disappears. Wheezing can be distinguished from crepitus by the ratio of respiratory noise to breathing phases. Wheezing is heard both during exhalation and during inhalation, and crepitus - only during inhalation. According to the nature of the sound, wet rales resemble gurgling or crackling, and crepitation is a dry crackling.
Crepitation occurs if the pulmonary alveoli contain a small amount of exudate. The exudate contained in the alveoli disintegrates the alveoli that were asleep and stuck together during exhalation. Since air enters the alveoli at the same time, the sound effects of the bursting alveoli

are superimposed on each other, and a crepitation effect occurs. Crepitation is heard in the lower parts of the lungs during inhalation.

Pleural friction noise most often occurs when inflammatory exudate is deposited in the pleural cavity. The leaves of the pleural cavity touch each other when moving, and this is perceived as a characteristic noise. Pleural friction noise occurs in both phases of breathing. In addition, even when imitating respiratory movements with a closed mouth and a closed nose (ie, when air does not enter the respiratory tract), the pleural friction noise is determined. This is a characteristic feature when differentiating pleural friction noise from wheezing and crepitation. Depending on the nature of the process, the noise can be rough (with massive deposits of fibrin on the pleural walls) or soft - at the beginning of the process. An increase in pleural friction noise can occur when the chest is pressed with a stethoscope, while the sheets of the pleura come closer together and their mutual friction increases.

General material and mass-methodological support
lectures:
work program of the academic discipline
synopsis (plan-summary) of the lecture
multimedia presentation of the lecture

Questions for self-control:

1. Complaints of patients with respiratory diseases.
2. Shortness of breath in diseases of the respiratory organs: definition of the concept, types, causes and mechanism of occurrence.
3. Cough in diseases of the respiratory organs: definition of the concept of "cough", types, causes and mechanism of its occurrence.
4. Hemoptysis in diseases of the respiratory organs and pulmonary bleeding: definition of concepts, causes and mechanisms of their occurrence.
5. Pulmonary tissue compaction syndrome. Diagnosis of the syndrome based on the results of the survey and physical examination (palpation, percussion, auscultation). Causes of occurrence. Additional research methods.
6. Syndrome of bronchial obstruction of the lungs. Diagnosis of the syndrome based on the results of the survey and physical examination (palpation, percussion, auscultation). Causes of occurrence. Additional research methods.
7. Syndrome of fluid accumulation in the pleural cavity. Diagnosis of the syndrome based on the results of the survey and physical examination (palpation, percussion, auscultation). Causes of occurrence. Additional research methods.
8. Syndrome of accumulation of air in the pleural cavity. Diagnosis of the syndrome based on the results of the survey and physical examination (palpation, percussion, auscultation). Causes of occurrence. Additional research methods.
9. Syndrome of the presence of a cavity in the lungs. Diagnosis of the syndrome based on the results of the survey and physical examination (palpation, percussion, auscultation). Causes of occurrence. Additional research methods.
10. Syndrome of increased airiness of the lungs. Diagnosis of the syndrome based on the results of the survey and physical examination (palpation, percussion, auscultation). Causes of occurrence.

Additional research methods.
11. Respiratory failure syndrome: definition of the concept, types, causes and mechanisms of occurrence. Types of ventilation disorders, causes of their occurrence.

Lecture No. 3 "Symptoms and syndromes in diseases of the circulatory system based on questioning the patient, palpation and percussion, and auscultation. Properties of the pulse and blood pressure."

Actuality of theme.

Examination of the arterial and venous pulse, measurements of blood pressure by the Korotkov method are important components of the patient's examination.

The concept of arterial hypotension and hypertension, venous pressure is the main component of the hemodynamic support of a person and his functional state. Instrumental examinations, such as oscillography, sphygmography, capillaroscopy, are now also widely used for examination. All this gives more or less complete information about the state of the cardiovascular system.
Auscultation of the heart is one of the important and difficult moments in the examination of patients, which require deep knowledge and sufficient experience from the doctor. Correctly conducted auscultation allows in most cases to determine the nature of pathological changes in the heart

Purpose of the lecture: Acquaintance of applicants with the theoretical foundations of measuring blood pressure, pulse, palpation, percussion and auscultation of the heart. Study of the main properties of the pulse.
Study of the main symptoms and syndromes in diseases of the circulatory system.
Basic concepts: Pulse, blood pressure, Korotkov tones, cardiovascular system.

Plan and organizational structure of the lecture.
1. Determination of the educational goal.
2. Providing positive motivation.
3. Presentation of the lecture material according to the plan:
4. Summary of the lecture. General conclusions.
5. The lecturer's answer to possible questions.
6. Tasks for self-training.

Basic concepts: examination of the patient, palpation, percussion, auscultation, organs of the cardiovascular system, arterial.

Content of lecture material (lecture text)

Symptoms and syndromes in diseases of the circulatory system based on the patient's questioning, palpation and percussion, and auscultation. Properties of pulse and blood pressure.

Pulse are oscillations of the walls of arterial vessels associated with the entry of a large volume of blood into the vascular bed during systole. The pulse is most often determined on the radial arteries, but it can also be determined on the carotid, popliteal, femoral, and rear foot arteries.

To study the pulse on the radial arteries, palpation is done simultaneously on both hands. The doctor sits opposite the patient and examines the pulse on the right hand with the fingers of his left hand, and vice versa, the doctor determines the pulse on the patient's left hand with his right hand. The doctor takes the patient's wrists in his hands like a guitar neck, i.e. he presses the radial arteries on the front surface of the forearm with his fingers to the bone base.
The wall of the artery is squeezed until the blood flow stops and the properties of the vessel wall are evaluated above compression - normally it is soft and elastic. Atherosclerosis changes the qualities of the artery wall: they lose elasticity, become compacted, twisted.

Pulse properties

During the study, the pulse is first determined on both hands for comparison. Normally, on both hands, pulse waves are determined in the same quantity and with the same qualities.
Frequency of pulse oscillations of the vascular wall normally ranges from 60 to 80 times per minute. The pulse rate of a healthy person should correspond to the frequency of heart contractions. If it is noted that the frequency of the pulse exceeds the frequency of heart contractions, this phenomenon is called**pulse deficiency** . It occurs in conditions when the amount of blood entering the vascular bed during the systole of the left ventricle is so small that it is not able to cause oscillation of the vascular wall and is not defined as a pulse wave. This is characteristic of extrasystole. An increase in the frequency of heart contractions (and, accordingly, the pulse) more than 80 times per minute is called tachycardia (rapid pulse); less than 60 times per minute is called bradycardia (rapid pulse). In addition to extrasystole, there are other heart rhythm disorders. In such conditions, the pulse becomes irregular, characterized by a change in the frequency of pulse waves. If the pulse is irregular, to determine its exact frequency, it is necessary to measure the number of pulse waves
continuously for 1 min.
Filling the pulse. The pulse is considered complete if a sufficient volume of blood enters the bloodstream during systole, and empty if the filling of the bloodstream is insufficient (for example, in shock).
Pulse voltage depends on the level of systolic blood pressure. The pulse can be hard (with high blood pressure), moderate tension (with normal blood pressure) and soft (with low blood pressure).
Pulse rate. A high, or large, pulse occurs when a large volume of blood enters the bloodstream and a significant increase in pressure, and then when the pressure in the vascular bed decreases.

Accordingly, a small pulse occurs when there is little blood entering the vascular bed and a slight fluctuation in pressure.

Pulse shape. A fast pulse occurs with a rapid increase in pressure and an equally rapid drop in it, a slow pulse occurs with a slow increase and drop in pressure.

Arterial pressure, the method of its measurement. Blood pressure in normal and pathological conditions

Arterial pressure is determined by the volume of blood entering the bloodstream during systole and the total peripheral resistance of vessels.

Blood pressure measurement technique

A mercury manometer, i.e. a Riva-Rocchi apparatus, is used to measure pressure. To measure blood pressure on the brachial artery, a cuff from the device is placed on the forearm so that the built-in tube is in the center of the elbow fossa, and the cuff itself is 2 cm above the elbow bend. The cuff is put on quite tightly, but leave a gap between the cuff and the hand, equal to about 1 cm. After that, close the valve and start pumping air into the cuff, increasing the pressure in it. The hand should be placed calmly and relaxed on a comfortable surface. The phonendoscope is applied to the elbow bend in the area of pulsation of the radial artery. As long as the pressure in the cuff does not exceed the pressure in the brachial artery, the heartbeat continues to be heard. At the moment when the pressure in the cuff is compared and begins to exceed the pressure in the circulatory system, continue to pump air up to 30-40 mm Hg. Art. After that, open the valve a little and gradually, carefully and slowly deflate the cuff, noting the appearance of a heartbeat. As soon as the level of pressure in the cuff, determined on the scale of the manometer, is compared with the level of blood pressure in the brachial artery, heart sounds will begin to be heard. This value corresponds to systolic blood pressure. The last heartbeat heard and the readings corresponding to it on the manometer scale correspond to the diastolic pressure.

To measure the pressure, it is necessary to observe a number of conditions: the subject must be in a calm state; if the measurement was preceded by physical exertion, you must first calm down. It is absolutely necessary to measure blood pressure three times, taking into account the smallest number.

A systolic pressure of 120 mm Hg is considered normal. Art. and diastole - 80 mm Hg. Art. An increase in blood pressure above these values is called arterial hypertension, a decrease below them is called arterial hypotension.

Physiological, essential and symptomatic hypertension are distinguished. Physiological pressure increase occurs during physical exertion, overeating, excitement, smoking, drinking strong tea, coffee. Essential hypertension is an independent disease that occurs as a result of a violation of the blood pressure regulation system. Symptomatic hypertension is a symptom of the main disease (endocrine, renal, cardiovascular or central nervous system).

A decrease in blood pressure can occur with poisoning, blood loss, shock states, chronic infections, tuberculosis. Young people with an asthenic physique may experience a drop in blood pressure after taking a number of medications, and orthostatic hypotension may occur when moving from a horizontal position to a vertical position.

Today we will focus on**intricacies of blood pressure self-control**. Many people have measuring devices for this at home, but you need to know how to use them correctly in order to get accurate

measurement results. We remind you that in patients with diabetes, the HELL value should not exceed 130/85 mm of mercury, otherwise the development of late complications of diabetes accelerates.

The blood pressure inside the blood vessels ensures the movement of blood along them. An increase in systolic pressure above 140 mm Hg. Art., and diastole is above 90 mm Hg. Art. is called**hypertension A decrease in systolic pressure below 110 mm Hg. Art. and diastole below 66 mm Hg. Art. called hypotension**.

Changes in blood pressure occur in many diseases. Its short-term increase can be observed with heavy physical exertion, especially in untrained persons, with mental excitement, consumption of alcohol, strong tea, coffee, with excessive smoking and severe pain attacks. A long-term increase in blood pressure is observed in hypertension, many kidney diseases (nephritis, vascular nephrosclerosis), a number of diseases of the endocrine system, some heart defects, etc.

Sometimes only systolic pressure increases, while diastole remains normal or decreases, which leads to**significant increase in pulse pressure**. This is observed in aortic valve insufficiency, thyrotoxicosis, to a lesser extent — in anemia, atherosclerosis.

A decrease in blood pressure can be noted as a constitutional feature in persons with an asthenic physique, especially in an upright position — the so-called**orthostatic hypotension**. As a pathological symptom, hypotension can be observed in many acute and chronic infections, tuberculosis, Addison's disease, etc. A sharp drop in blood pressure occurs with heavy blood loss, shock, collapse, myocardial infarction. Sometimes only systolic pressure decreases, while diastole remains normal or even increases, which leads to a decrease in pulse pressure and is observed in myocarditis, exudative and adhesive pericarditis, when cardiac output decreases sharply and systolic pressure drops accordingly. A decrease in pulse pressure is observed at the narrowing of the aorta.

Over the years, many people begin to experience more and more frequent symptoms of changes in blood pressure. Moreover, this symptomatology can be absolutely the same both with an increase and with a decrease of HELL. In order to understand the reasons, they rush to measure blood pressure.

Underlying**at the heart of this diagnostic method**? The pressure value in the arterial system fluctuates rhythmically, reaching the highest level during systole, and decreases at the moment of diastole (systole — contraction, compression of the heart; diastole — its stretching, expansion).

The value of blood pressure is expressed**in millimeters of mercury**. Normal systolic (maximum) pressure varies between 100-140 mm Hg. Art., diastolic pressure (minimum) — within 60-90 mm Hg. Art. The difference between systolic and diastolic pressure is called pulse pressure; normally it is equal to 40-50 mm Hg. Art.

Blood pressure can be measured**directly and indirectly**. In direct measurement, a needle or cannula connected by a tube to a manometer is inserted directly into the artery. This method is not widely used and is mainly used in cardiac surgery.

There is an indirect way to measure blood pressure**three methods: auscultatory, palpatory and oscillographic**. In everyday practice, the most common auscultatory method. It allows you to measure both systolic and diastolic blood pressure. The measurement is made using a sphygmomanometer device.

There are mercury (Riva-Rocchi devices) and membrane or spring sphygmomanometers, which are often called tonometers. The latter consists of a mercury or spring manometer, which is connected by rubber tubes to a cuff and a rubber balloon for pumping air. There is a special valve in the balloon (pear) near the outlet of the tube, which allows you to regulate the entry of air into the manometer and the cuff and maintain the air pressure in them at the desired level. A more accurate mercury manometer (Riva-Rocci apparatus), it is a vessel with mercury into which a thin glass tube is lowered, attached to a scale with millimeter divisions from 0 to 300.

To measure the pressure, choose a room that is free from noise, so that you can distinctly and clearly hear the beating of the pulse. Usually the pressure is measured**on the brachial artery**. The left hand is placed on the table. At the same time, it is necessary to ensure that the elbow and the sphygmomanometer are located approximately at the level of the heart, if possible. A cuff is placed on the examinee's bare shoulder and fixed on it. The cuff should fit loosely so that only one finger passes between it and the skin. The edge of the cuff, into which the rubber tube is inserted, should be turned down and located 2-3 cm higher than the elbow pit. After fastening the cuff, the examinee comfortably places his hand with the palm facing up; the hand muscles should be relaxed.

Next, the brachial artery is found by pulsation in the elbow bend, a stethoscope is attached to it, the valve of the sphygmomanometer is closed and air is pumped into the cuff and the manometer. The height of the air pressure in the cuff, which compresses the artery, corresponds to the level of mercury on the scale of the Riva-Rocci apparatus or a certain indicator of the spring sphygmomanometer. Air is pumped into the cuff using a pear with the right hand until the pressure reading is at least 180-200 mm Hg. Art. If pulsation is heard at these pressure indicators, then you should continue pumping air into the cuff until the pulsation noise completely disappears and additionally increase the air pressure by 20-30 mm Hg. Art.

After that, the valve screw is slowly turned counterclockwise with the fingers of the right hand. Opening the valve in this way, begin to slowly release air from the cuff: the pressure drop in the latter should occur at a rate of 2-4 mm Hg. Art. per second At the same time, the brachial artery is listened to with a phonendoscope and the reading of the manometer scale is monitored. When the pressure in the cuff becomes a little lower than the systolic one, sounds-tones synchronous with the activity of the heart begin to be heard clearly and distinctly. Sound**of the first beat of the arterial pulse and is an indicator of systolic pressure.** The moment at which the pulse is no longer audible is recorded on the manometer as an indicator of diastolic pressure.

When measuring blood pressure, it is necessary to take into account that**its evidence changes depending on the position of the body** person at the time of measurement (lying, standing, sitting), time of day, physical exercises, eating, drinking and many other factors. In addition, the cuff may be worn incorrectly, as a result of which it is difficult to hear both the first and last pulse. The manometer needle should return to the zero, initial position. Otherwise, incorrect pressure readings may be obtained.

For extremely accurate blood pressure readings**air should be pumped into the cuff only with the right hand**. Deflation of the cuff must be done slowly, as accelerated deflation of the cuff leads to an inaccurate HELL measurement. In order to exclude incorrect readings, it is necessary to make sure that the ear pads of the stethoscope are clean and the tubes are not damaged. The manometer should be stored in a clean and dry place, inaccessible to children, not to be dropped

and not to be handled carelessly. In this case, the manometer will reliably serve you for many years.

Only systolic pressure is determined by the palpation method. When measuring pressure by this method, the radial artery is palpated during the slow release of air from the cuff of the sphygmomanometer. As soon as the pressure in the cuff becomes slightly lower than the systolic pressure, the first weak pulses will appear.

Arterial oscillography used in medical and preventive institutions, it is more informative and allows doctors to get a more complete picture of the state of the cardiovascular system.

Determination of blood pressure by any of the indirect methods**may be accompanied by some increase in the level of systolic pressure** compared to the true value, because when the vessel is compressed, it is necessary to overcome the resistance of the vessel wall itself and the tissues surrounding it. In addition, the level of systolic pressure can be affected by the hydraulic shock that occurs at the blind end of the vessel when a pulse wave collides with an artery narrowed by a cuff.

Any diagnostic method, including blood pressure measurement, is nothing more than a source of information for making the right decision. At the beginning of the article, I listed many reasons why HELL could be abnormal, that's why**the doctor must determine the specific cause and prescribe treatment**. In recent years, many people have acquired a personal tonometer and monitor their own blood pressure. This is good, especially if a person is prone to frequent fluctuations of HELL. But it is impossible to allow the medical device to turn into "monkey glasses" so that you, without finding out the reasons for increased and decreased pressure, immediately swallow pills.

Self-control should serve**by helping the doctor to find out the dynamics of your well-being and for the correct, accurate appointment of treatment. Today, the most popular devices for measuring blood pressure and pulse among the population and medical workers are a wide range of performance: from traditional arrow devices with cuffs on the forearm to digital electronic ones with memory** and printing devices, with graphic displays and memory, that take readings on the wrist or fingers. The accuracy of such devices is +/- 2%, and compactness, modern design, convenience, multifunctionality and ease of use make them very attractive for people who constantly monitor their blood pressure.

Tones of the heart. Mechanism of formation of heart tones (I, II, III, IV). Factors determining the strength of heart tones

This is a very important method of diagnosing heart diseases. Knowledge of the auscultatory pattern is especially important for detecting congenital and acquired heart defects.

During contractions of the heart, sound effects occur, which are heard by the method of auscultation and are called heart tones. Their appearance is associated with the oscillation of the walls of blood vessels, heart valves, the movement of blood flow during heart contractions, with oscillations of the walls of the myocardium. I and II heart tones are normally heard.

The first sound of the heart (systolic) consists of several components. Based on this, the tone is called valvular-muscular-vascular. The fourth component of the atrial tone. The atrial component is associated with the oscillations of the walls of the atrium during their systole, when blood is pushed into the ventricles. This component is the first component of the first tone, it merges with the following components. The valvular component of the tone is associated with the sound

effects that occur during the movement of the atrioventricular valves in ventricular systole. During systole, the pressure in the ventricles increases, and the atrioventricular valves close. The muscular component is associated with the sound effects resulting from the oscillation of the walls of the ventricles during their contraction. Ventricular systole is aimed at pushing out the volume of blood contained in them into the aorta (left ventricle) and the pulmonary trunk (right ventricle). The movement of blood under high pressure causes oscillations of the walls of large vessels (aorta and pulmonary trunk) and is accompanied by sound effects, which also constitute the first tone.
II tone is two-component. It consists of valvular and vascular components. This tone is heard during diastole (diastole). During ventricular diastole, the valves of the aorta and pulmonary trunk close, and sound effects occur when these valves oscillate.
The movement of blood into the vessels is also accompanied by the sound component of the II tone.
The III tone is not mandatory and is heard in persons of young age, as well as those who have insufficient nutrition. It occurs as a result of the oscillation of the walls of the ventricles in their diastole during their filling with blood.
IV tone occurs immediately before the first tone. The reason for its appearance is the oscillation of the walls of the ventricles during their filling during diastole.
The strength of the heart sounds is determined by the proximity of the heart valves relative to the front chest wall (therefore, weakening of the heart sounds may be associated with an increase in the thickness of the front chest wall due to subcutaneous fat). In addition, the weakening of heart tones may be associated with other reasons that cause disturbances in the conduction of sound vibrations to the chest wall. This is an increase in the lightness of the lungs with emphysema, intensive development of the muscles of the front chest wall, pneumothorax, hemothorax, hydrothorax. In young thin people with anemia, the sonority of tones increases. This is also possible due to the phenomenon of resonance when the lung cavity appears.

Pathological changes in heart sounds. Mechanism. Diagnostic value
The sonority of the tones increases in the presence of a rounded cavity formation in the chest cavity, resonating sound effects, for example, caverns in pulmonary tuberculosis. Weakening of tones can be caused by the presence of fluid and air in the pleural cavity, with thickening of the front wall of the chest. Cardiac causes of decreased heart sounds include myocarditis and myocardial dystrophy. An increase in tones is observed with hyperthyroidism, excitement, drinking a large amount of coffee. Weakening of the I tone at the apex indicates insufficiency of the mitral and aortic valves. This is due to the absence of the valvular tone component in case of organic destruction of the valves. Narrowing of the mouth of the aorta can also be the cause of weakening of this tone.
Strengthening of the first tone is observed with mitral stenosis (at the apex), stenosis of the right atrioventricular opening (near the base of the xiphoid process of the sternum). Amplification of the I tone is found in tachycardia.
Weakening of the II tone above the aorta is observed in aortic insufficiency, as the valvular component of the II tone falls out, blood pressure decreases, pressures in the small circle of blood circulation.
The accent of the II tone over the aorta happens with hypertension, physical exertion.

The emphasis of the II tone over the pulmonary trunk is an indicator of mitral stenosis, mitral insufficiency, lung diseases accompanied by pulmonary hypertension.

Heart tones (characteristics of I, II tones, places of their listening). Rules of auscultation. Projection of the heart valves on the chest wall. Points of auscultation of heart valves

Tones are heard according to the projection of the valves involved in their formation. Thus, the mitral (left atrioventricular) valve is heard in the region of the apex of the heart, according to the apical impulse, normally in region V of the intercostal space along the left midclavicular region. The tricuspid (right atrioventricular) valve is heard at the point where the right ventricle adjoins the anterior chest wall, preferably at the xiphoid process of the sternum.

The mitral valve of the pulmonary artery is heard according to its projection on the area of the front chest wall - in the II intercostal space, to the left of the sternum. The aortic valve can also be heard in the II intercostal space, to the right of the sternum. The aortic valve can be heard at the point of Botkin-Erb at the point of attachment of the III-IV ribs to the left in relation to the sternum.

Places of projection of heart valves on the front chest wall.

The mitral valve is projected at the point of attachment of the III rib to the left of the sternum, the tricuspid valve - in the middle of the line running from the left of the attachment of the cartilage of the III rib to the sternum to the right, to the cartilage of the V rib. The aortic valve is projected in the middle of the distance along the line drawn along the attachment of the cartilages of the III ribs on the left and right, on the sternum. The valve of the pulmonary artery is heard in the place of its projection, namely to the left of the sternum in the II intercostal space.

Rules of auscultation

The order of auscultation is clearly defined according to the principle of primary listening to those valves that are most often affected by a pathological process. First, the mitral valve in the area of the V intercostal space along the left midclavicular line is listened to, then the aortic valve to the right of the sternum in the II intercostal space, the valve of the pulmonary artery to the left of the sternum in the II intercostal space, and the tricuspid valve in the area of the xiphoid process of the sternum. The latter is performed auscultation of the aortic valve at the point of Botkin-Erb at the level of attachment of the III - IV ribs. Thus, during auscultation, a figure of eight is performed while moving the phonendoscope.

Places for listening to heart sounds

The first sound is heard in the region of the apex of the heart and in the region of the xiphoid process of the sternum, since the valves directly involved in its formation are projected there. In addition, hearing this tone coincides with an apical impulse. It is low, long. The II sound is heard in the second intercostal space, to the right and left of the sternum, because the valves of the aorta and the pulmonary trunk are projected there, forming the valvular component of the II sound. The II tone is higher and slightly longer compared to the I tone. To better listen to the patient's tones, you can ask him to sit down. Tones can be heard when the patient is standing or lying down (for example, the mitral valve is best listened to when lying on the left side). Respiration helps distinguish respiratory from cardiac, which also facilitates diagnosis.

Auscultation of the heart is preferably performed with a specialized cardiac highly sensitive phonendoscope.

Additional heart sounds: mitral valve opening sound, ejection sound, systolic click, bifurcation and split heart sounds

Bifurcation and splitting of heart tones

In a normal state, the left and right halves of the heart work synchronously. Therefore, the heart tones formed by the valvular components of both the right and left half of the heart are heard simultaneously as a single tone. But pathological conditions accompanied by non-simultaneous closing of the valves are also accompanied by a change in the auscultatory picture. So, if the atrioventricular valves do not close at the same time, there is a bifurcation of the I tone. If the cause lies in the valves of the aorta and the pulmonary trunk, then the II tone is bifurcated. Splitting is said when the difference in valve closing time is so small that it is not perceived as an independent sound phenomenon, but is heard as tone splitting. Bifurcation of tones can be a physiological, reversible phenomenon, or it can be a pathological, irreversible sign of some disease.

Physiological splitting of the first tone is associated with deep exhalation, during which blood under high pressure enters the left atrium, as a result of which the left atrioventricular valve is delayed in closing, the tone splits. The physiological bifurcation of the II tone is also related to the phases of breathing. At the moment of inspiration, due to different blood filling of the vessels, the aortic valve closes earlier compared to the valve of the pulmonary trunk.

Pathological bifurcation of the first tone occurs with non-simultaneous excitation of the ventricles. This is possible in the case of cardiac conduction disturbances (blockage of the legs of the bundle of His). Pathological bifurcation of the II tone occurs when the valves of the aorta and the pulmonary trunk are not simultaneously closed in hypertension, aortic stenosis, mitral stenosis, and bundle branch block.

Mitral valve opening tone

This tone is heard in mitral stenosis and is associated with changes in the structure of this valve, when it is opened, it creates an additional tone. The valves, when they are defeated, are so changed that they create an obstacle when the blood moves, and when the blood hits them, they cause the appearance of sound effects. It occurs almost immediately after the II tone, so it must be distinguished from the bifurcation of the II tone.

Pericardial tone

Pericardial tone occurs after transferred pericarditis, which ended with the formation of adhesions and adhesions in the pericardial cavity. The reason for its appearance is pericardial oscillations in diastole. Also appears after the II tone. The tone, the nature of which is also related to pericardial adhesions, but which appears between I and II tones, is called a systolic click. It is loud and short.

The rhythm of quail and gallop

The rhythm of the quail is the clapping I tone, the opening tone of the mitral valve, and the II tone. The rhythm of the gallop is so named because it is similar to the tramp of a horse and occurs when listening to III or IV heart tones. An increase in the III tone causes a protodiastolic gallop rhythm, an increase in the IV heart tone - a presystolic gallop rhythm.

If both tones are heard, the gallop rhythm is called mesodiastolic.

A healthy person does not make noises during the heart's work. The movement of blood through the cavities of a healthy heart, its physiological openings occurs with the formation of only tones, which were discussed in the previous lecture.

The noises that occur during the work of the heart have a strict classification.

First, they are divided into intracardiac and extracardiac.

Secondly, they are divided into systolic and diastolic murmurs. Diastolic noises are divided into 3 types: 1) protodiastolic noise that occurs at the beginning of diastole, immediately after the 2nd

tone, 2) mesodiastolic noise that is heard in the middle of diastole, 3) presystolic noise that appears at the end of diastole before 1st tone .
Thirdly, noises are divided into 1) organic, valvular and muscular, 2) functional, 3) intermediate or noises of relative insufficiency of valves.
Noises during the operation of the heart occur most often in connection with disturbances in the functioning or structure of the valve apparatus of the heart or in connection with organic or functional disturbances on the part of the heart muscle. These are the so-called intracardiac**or intracardiac murmurs. Intracardiac murmurs can be divided into 3 large groups: 1) murmurs associated with anatomical disorders are called organic** *murmurs, 2) inorganic or functional murmurs that are not associated with anatomical disorders, and 3) murmurs of relative valvular insufficiency or intermediate* forests.
In addition, noises can be caused by extracardiac pathology. This is called**extracardiac murmurs. Extracardiac murmurs are associated with the pathology of large vessels near the heart, with changes in the pericardium (***pericardial friction noise), with changes tangent to the pericardium of the pleura (pleuropericardial noise) or lungs (cardiopulmonary noise*).
Organic noises arise as a result of organic changes in the valves or holes closed by them, as well as in connection with anatomical disorders of the structure of the heart.
Their appearance can be explained as follows. If the tube through which the liquid flows is compressed in some place so that the liquid continues to flow, then a vortex circulation (turbulence) of the liquid is formed above and below the judged section of the tube. Under their influence, the walls of the tube will begin to oscillate. Oscillations will have an irregular character (with an incorrect alternation of oscillating movements). Noise will be registered when listening to this handset. The same noises occur in the heart with defects in the valves through which blood flows. Oscillations will occur in front of and behind the valve defect, involving the valve leaflets, the chordae attached to them, and the myocardium. Auscultatively, the oscillations that occurred during the narrowing of the valve opening are perceived as noise. These noises are called*noises of exile.* They occur when the forward movement of blood is obstructed.
Noises will occur not only when the valve opening is narrowed. If the pathological process leads to the deformation of the valve leaflets, their shortening and partial destruction, then such leaflets will not be able to completely close the valve opening and will leave a more or less wide gap. Through this narrow gap, the backflow of blood will begin, generating the noise. Such noises are called*regurgitation noises.* They occur when the blood moves backwards, back against the natural flow of blood.
In addition to the presence of a narrow opening for the appearance of noise, another necessary condition is the speed of the flow of blood flowing through this opening. This explains why not all valve changes are accompanied by noise, although the degree of narrowing remains constant, and the noise may even disappear, appearing later again. The strength of the noise, thus, depends on the degree of narrowing of the opening and, to a greater extent, on the speed of blood movement through it. The faster the blood flow, the louder the noise. The smaller the narrowing, the faster the blood flow must be for the murmur to be heard. The noise will weaken and even disappear during the period of weakening of cardiac activity and slowing down of blood flow. Accordingly, it will increase when heart activity increases and blood flow accelerates. The formation of heart murmurs is facilitated by the roughness of the inner surface of the valves (endocardium) and the intima of the arteries. This roughness can be the cause of noise even with a slight narrowing of the hole. Finally, noises become louder if the edges of the hole become tight and thick. During the

postponement of extermination in valves, walls of blood vessels, noises become audible very loudly.

Organic noises are very diverse in nature: blowing, scraping, sawing, and buzzing noises. They can be sonorous, or high, musical noises. These noises appear when a small hole is formed in the valve leaflet or when a thin, tightly stretched thread from part of the valve or from the tendon chord oscillates in the lumen of the hole. However, the pitch of the sound has less diagnostic value than its strength. Of practical importance is only the fact that the appearance of a rough musical noise, scratching, high, indicates an organic, and not a functional reason for its appearance. The noise of diastole in stenosis of the mitral orifice is usually lower than other musical noises.

Intracardiac murmurs are strictly related to the phases of cardiac activity, that is, to systole and diastole. This is of great importance for recognizing various heart defects.

Noises heard during ventricular systole are called systolic.*Noises heard during diastole are called diastole.*

Systolic murmurs occur with narrowing of the mouth of the aorta, narrowing of the mouth of the pulmonary artery. In both cases, the noise is caused by the flow of blood that is expelled from the ventricles of the heart and goes in the usual direction - forward, into the large vessels (expulsion noises). These noises are heard at the points of auscultation of the aorta and pulmonary artery. In addition, the systolic murmur appears in the insufficiency of the left atrioventricular valve (mitral valve) and in the insufficiency of the right atrioventricular valve (tricuspid valve). However, the noise is caused by the flow of blood during ventricular systole not in the usual direction - from the heart to the large vessels, but in the opposite direction - from the ventricles of the heart to the atrium due to insufficiently closed valve openings (regurgitation noises).

Systolic murmurs are most intense at the very beginning of systole. Then they gradually weaken - decreasing noises (decrescendo). This is explained by the fact that blood flow through the narrowed opening is fastest at the very beginning of systole. As blood passes from the ventricle and fills the aorta, pulmonary artery, or atrium with blood, the speed of blood flow gradually decreases due to equalization of pressure on both sides of the opening. In some patients with mitral stenosis, the systolic murmur has an ascending-decreasing character and has a diamond shape.

Systolic murmurs appear with insufficiency of the mitral valve, insufficiency of the tricuspid valve, with stenosis of the mouth of the aorta and with stenosis of the mouth of the pulmonary artery. They can also appear with some types of congenital heart defects.

Diastolic murmurs are determined in those cases when blood enters the ventricles during diastole through narrowed valve openings. This happens, first of all, with stenosis (narrowing) of the left or right atrioventricular openings. In both of these cases, the noise is generated by the flow of blood flowing during diastole through narrowed openings in the usual direction - forward.

Diastolic murmurs are also heard with insufficiency of the aortic valves or insufficiency of the valves of the pulmonary artery. In these cases, the noise is caused by the flow of blood moving during diastole in the reverse direction - from the aorta or pulmonary artery through insufficiently closed valve openings back into the ventricles of the heart (regurgitation noises). Diastolic murmurs are most often intense at the beginning of their occurrence, and then weaken, that is, they have a decreasing character. However, they can have a different character. Thus, with stenosis of the mitral orifice, the diastolic murmur can have 5 variants. 1. Noise can appear at the beginning of diastole immediately after the 2nd tone (protodiastolic noise) and be such that it decreases. 2. Noise can appear at the end of diastole, when atrial systole begins. Such a noise is

called presystolic, has a rising character (crescendo) and merges with 1 tone. 3. With the same heart defect, at the beginning of diastole, when the ventricle is rapidly filled with blood, the noise may be decreasing, however, at the end of diastole, when the atrial systole begins and the rest of the blood is ejected from the atrium into the ventricle, there is a presystolic increase in the noise. 4. In some patients, the decreasing protodiastolic murmur with this defect quickly subsides, disappears, and is separated from the presystolic increase by a short pause. 5. In some patients with mitral stenosis, a uniform, ribbon-like murmur is heard, occupying the entire diastole. This noise is called mesodiastolic.

Localization of noise is of great importance in the diagnosis of heart defects. A murmur is usually heard at the same points where heart sounds are heard. Organic heart sounds are heard not only at the standard points of auscultation of heart sounds, but also over the entire heart area and even beyond it.

Usually organic noises are good*are held* by blood flow. Thus, with stenosis of the mouth of the aorta, the noise spreads along the blood flow in the aorta and can be heard in the interscapular zone at the point where the aorta approaches the spine. It can also spread to the vessels of the neck and other areas. With insufficiency of the aortic valves, the murmur is carried with the returning blood into the left ventricle and is heard in the 3rd intercostal space at the left edge of the sternum at the Botkin-Erb point, along the left contour of the heart.

In case of insufficiency of the mitral valves, the blood returns to the left atrium and can be conducted or even generally heard above the auricle of the left atrium - in 2-3 intercostal spaces along the left contour of cardiac dullness near the left edge of the sternum. Often this noise is heard at the level of the apex of the heart in the axillary region ("zero point").

In contrast to the listed noises, the noise in stenosis of the mitral orifice does not go anywhere, because in this heart defect, the point of auscultation of the mitral valve and the point of radiation of the noise coincide. It is to this point in the region of the top of the heart that the blood entering through the narrowed mitral orifice is directed.

If, when listening to one valve, noises are detected, both in the systolic and diastolic phases, then a contract should be made for a double,*combined, valve failure* - narrowing of the opening and insufficiency of the valves covering it.

If, for example, a systolic noise is heard above one hole, and a diastole noise is heard above the other, then a conclusion is drawn about the presence of two defects or*combined defeat of two valves*.

So, in practice, for each organic noise, it is necessary to determine:

1. Which phase of cardiac activity does it correspond to - systole or diastole.
2. Does it start from the first moment of systole or diastole, or a little later, what part of them does it occupy.
3. What is the nature of the noise - increasing, decreasing, ribbon-like, diamond-like or other.
4. At what point above the heart is the maximum of its power.
5. In which direction is the noise made?

Functional noises occur with anatomically unchanged heart valves, in the absence of organic changes in the valve openings and without anatomical violations of the heart structure. Their appearance is associated with functional disorders on the part of the heart muscle, with a change in the rheological properties of blood, its composition and the nature of blood flow. These noises appear, first of all, with a relative increase in the speed of blood flow. Thus, functional noises appear in patients with thyrotoxicosis, in patients with fever. In these conditions, the speed of

blood flow through the physiological openings of the heart increases, turbulent eddies appear around them, creating noise. In patients with anemia, when the number of erythrocytes decreases, blood viscosity decreases, turbulent eddies also occur during the passage of blood through the physiological openings of the heart.

Functional noises differ from organic noises in a number of ways.

1. Functional noises differ from organic noises in their high variability. They can be heard and then disappear in a short time, especially when changing the position of the body, when breathing, and the like. Organic noises do not change over a long period of time. They can change only with a change in the degree of heart disease or with a change in the contractility of the heart muscle.
2. Functional noises in timbre are usually soft, blowing and never rough, such as scraping or growling. Organic noises can be any timbre. More often they are rude and loud.
3. Functional noises are usually short, decreasing. Organic noises are usually long-lasting and can be of any shape - decreasing, increasing, diamond-shaped, and so on
4. Functional murmurs are almost always systolic, except for the rare Austin, Flint, and Graham-Steele murmurs. The Austin-Flint murmur occurs in patients with aortic valve insufficiency, but is heard at the apex of the heart, at the point of auscultation of the mitral valve. The appearance of this noise is explained by the fact that with aortic valve insufficiency, the retrograde flow of blood from the aorta to the left ventricle enters the leaflet or chord of the opening mitral valve and prevents its normal opening. The blood entering at this time from the atrium encounters an obstacle in the form of a mitral valve leaflet that has not opened and generates a diastole murmur heard at the point of auscultation of the mitral valve. It is possible that the noise can be caused by the confluence of two blood flows - normal from the atrium into the ventricle and retrograde - from the aorta into the ventricle. A Graham-Styla murmur is heard over the pulmonary artery in patients with high pulmonary hypertension and isolated right ventricular hypertrophy. At the same time, there is a relative insufficiency of the valve of the pulmonary artery and a diastole noise appears above it. Organic noises can occur in any phase of the cardiac cycle - both in systole and diastole.
5. Functional murmurs are usually heard against the background of unchanged heart tones, although they may appear in patients with previously altered heart tones. Organic noises are always heard with changed tones, sometimes replacing the heart tone.
6. Functional sounds are always heard only at one point of auscultation, mainly at the point of auscultation of the mitral valve, and are not conducted along the blood stream. Organic murmurs can be heard at any point of the heart and are widely carried along the blood stream.
7. Functional murmurs are not accompanied by a change in the volume of the heart chambers, although they may appear in patients with a changed volume of the heart chambers. For example, a hypertensive patient with an enlarged left ventricle may develop anemia and a functional murmur. Organic noises are always accompanied by a change in the volume of the heart chambers.
8. Functional murmurs are usually heard over the mouth of the pulmonary artery and over the apex of the heart. Organic noises can be heard over the entire surface of the heart, at any point of its auscultation.

Intermittent noises occur with anatomically unchanged heart valves, with normal valve openings and in the absence of changes in blood rheological properties. Their appearance is associated with the expansion of the valve ring. In patients with pronounced hypertrophy of the chambers of the heart and dilatation of the myocardium due to its weakness, there is an increase in the diameter of

the openings of the leaflet valves - mitral or tricuspid. At the same time, the flaps of the valves themselves do not change their shape or deform. However, their size becomes insufficient to completely close the increased diameter of the valve opening in the phase of ventricular systole. There is a relative insufficiency of the valve. At the same time, part of the blood begins to return to the atrial cavity and causes the appearance of noise. Such persistent stretching of the valve ring most often occurs in the left atrioventricular opening. It is almost impossible to distinguish it from valvular mitral insufficiency by ear. Much less often, the right atrioventricular opening can change in this way. With sclerotic expansion of the aorta, in some cases, the mouth of the aorta can also expand, which leads to an auscultatory picture of aortic insufficiency. At the same time, semilunar aortic valves can be completely unchanged.

Extracardiac murmurs. *Pericardial friction noise* occurs in patients with dry pericarditis, when the amount of fluid that moistens the pericardial leaves decreases, fibrin is deposited on them, and the sliding of the pericardial leaves becomes audible during the work of the heart. Accumulation of fluid in the pericardial cavity does not eliminate this noise, since the fluid in the pericardial cavity is usually located behind the heart, to the right and to the left of it. Only with a significant accumulation of fluid in the pericardial cavity, when it also fills the space in front of the heart, the friction noise of the pericardium is eliminated. Much less often, it appears with pericardial adhesions, with the formation of tubercles on it and the like. Sometimes this noise is formed with severe dehydration of the body, for example, with cholera, when there is only dryness of the leaves of the pericardium without inflammatory changes in it.

Friction noise of the pericardium differs from intracardial noises by the following features:

1. in contrast to intracardiac murmurs, pericardial friction noise has the character of scratching uneven surfaces ("crr", "crr")
2. during auscultation, pericardial friction noise is felt close to the ear
3. sometimes the pericardial friction noise can be felt with the hand
4. pericardial friction noise does not correspond to any specific phase of cardiac activity (systole or diastole), which is characteristic of intracardiac noises, but is heard in both phases, in systole and in diastole (systolic-diastolic noise), or is heard continuously, increasing during systole (this increase is explained by a more active displacement of the heart in systole)
5. pericardial friction noise is variable, both in terms of localization and duration of sound
6. pericardial friction noise is almost not carried out from the place of its origin, which is not characteristic of intracardial noises
7. pericardial friction noise intensifies when pressed with a phonendoscope in the area of absolute dullness of the heart and when the patient's body is tilted forward, which is not characteristic of intracardial noises
8. pericardial friction noise is heard over the entire cardiac region, but most of all - in the 3rd and 4th intercostal spaces to the left of the sternum and within the absolute dullness of the heart.

*Pleuropericardial murmur (*or pseudopericardial) occurs as a result of inflammatory changes in the pleura lining the costo-mediastinal sinus, that is, in that part of the pleura that, together with the lungs, covers the heart from above and to the left. When the heart contracts and when its volume decreases during systole, the edge of the lung straightens, and the pleural sheets move along with it. If they have inflammatory changes, then each of these movements is accompanied by the noise of friction of the pleura, which is heard synchronously with heart contractions. This murmur is very similar to pericardial friction murmur. It can be distinguished from the true pericardial friction noise by the following features:

1. the pleuropericardial murmur is heard along the left contour of the heart, while the pericardial friction murmur is best heard anteriorly, in the area of absolute dullness of the heart
2. pleuropericardial murmur depends to a greater extent on breathing, appearing during inspiration
3. at the same time as the pleuropericardial noise, the usual friction noise of the pleura is heard in the corresponding place.

Cardiopulmonary murmur heard along the front edge of the lung lobes - where they border the heart. It arises in this way. During the systole of the heart, its volume decreases. At the same time, negative pressure appears in the immediate vicinity of the heart and in some space from it. This space is filled with lungs. The air entering the alveoli of the lungs from the bronchi makes a noise synchronous with the systole of the heart. The murmur intensifies during inspiration, which can be used to distinguish cardiopulmonary murmurs from functional and intracardiac murmurs, which decrease during inspiration.

Cardiopulmonary murmurs sometimes occur when the pleural sheets fuse along the edge of the lung bordering the heart.

Cardiopulmonary murmurs can be heard not only during systole, but also during diastole of the heart. Such diastole noises can be heard in the area of large vessels - the aorta and pulmonary artery. Their appearance is explained by the fact that during diastole of the heart, the diameter of these large vessels decreases. The lung tissue located in this area expands and sucks air from the bronchi into the alveoli, which creates noise in the area of large vessels during heart diastole.

General material and mass-methodological support

lectures:

work program of the academic discipline
synopsis (plan-summary) of the lecture
multimedia presentation of the lecture

Questions for self-control:

1. The main complaints in diseases of the circulatory system and their diagnostic value.
2. Coponogenic and non-coponogenic pains in the area of the heart: causes and mechanism of their occurrence, details and their diagnostic value. Differences between coronary and non-coronary pains.
3. Shortness of breath in diseases of the cardiovascular system: definition of the concept of "shortness of breath", causes and mechanism of shortness of breath. Suffocation attack.
4. Hypertensive disease and symptomatic arterial hypertension. Clinical picture. Classification. Laboratory and instrumental methods of diagnosis. Principles of treatment.
5. CHD: syndrome of acute and chronic coronary insufficiency: definition. Main complaints and examination results of patients with angina pectoris. Instrumental and laboratory methods of diagnosis in angina pectoris syndrome. Principles of treatment.
6. CHD: myocardial infarction. Clinical picture. Classification. Diagnostic methods. Principles of treatment.
7. Chronic heart failure syndrome. Definition, clinical picture, classification by stages and functional classes, diagnostic methods. Principles of treatment.
8. Clinical and electrocardiographic diagnosis of atrial fibrillation and atrial flutter.

Lecture No. 4 "Question and examination of patients with pathology of the gastrointestinal tract. Palpation and percussion of the abdomen. Main symptoms and syndromes. Main symptoms and syndromes in patients with pathology of the liver and biliary tract. Portal hypertension syndrome"

Actuality of theme.

Physical examination of the organs of the abdominal cavity includes inspection, percussion, palpation and auscultation. The diagnostic value of the listed methods of gastrointestinal tract research is not the same. Palpation of the abdomen is the leading method that allows obtaining the most information about the condition of the abdominal organs. Palpation of the abdomen received recognition relatively recently - at the end of the 19th and the beginning of the 20th century.
A great role in the development of this method was played by the works of Glenar, V.P. Obraztsova, O.O. Hausman, M.D. Strazhesko.

Purpose of the lecture:

Master the methods of interviewing and examining patients with gastrointestinal tract pathology. To learn the technique of percussion of the abdomen, as well as surface palpation and deep methodical sliding palpation of the organs of the abdominal cavity according to the method of V.P. Obraztsova and N.D. Strazhesko. Learn to evaluate the data obtained during the examination.
the acquirer must master:

1. Be able to conduct a survey and examination of patients with gastrointestinal tract pathology.
2. Anatomy of abdominal organs.
3. Scheme of conditional division of the abdomen into regions.
4. Targets and diagnostic value are superficial
5. palpation of the abdomen.
6. Basic rules and techniques of conducting surface
7. approximate palpation of the abdomen.
8. Rules of methodical deep sliding palpation by method
9. V.P. Obraztsova and M.D. Guardian.
10. Techniques of deep palpation, percussive palpation
11. Auscultative percussion (africation) of the stomach and their diagnostic
12. value.
13. Techniques for deep palpation of the intestines (sigmoid
14. intestine, cecum with appendage, terminal part of ileum
15. ascending and descending colon
16. transverse colon, splenic and hepatic curvature
17. colon) and its diagnostic significance.

Basic concepts: organs of the gastrointestinal tract, patient examination, inflammation, palpation, percussion, auscultation.
Plan and organizational structure of the lecture.

1. Determination of the educational goal.
2. Providing positive motivation.
3. Presentation of the lecture material according to the plan:

4. Summary of the lecture. General conclusions.
5. The lecturer's answer to possible questions.
6. Tasks for self-training.

Content of the lecture material

When examining patients with diseases of the digestive organs, the doctor has the greatest difficulties when taking an anamnesis, as well as during a physical examination of the abdomen.
Examination of the patient, as always, begins with questioning, which includes clarification of complaints, collection of anamnesis of the disease and the patient's life.
When clarifying complaints, first of all, they pay attention to*the nature of the pain syndrome.* Correctly assessed pain syndrome in patients with gastroenterological pathology is a cornerstone in understanding the disease. Before characterizing the pain in patients with gastroenterological pathology, the mechanism of the appearance of pain in the pathology of hollow organs should be mentioned. In almost all situations, when the peritoneum is not involved in the pathological process, the pain occurs due to the fact that during the spastic contraction of the smooth muscles of the wall of the hollow organ, there are disorders of the blood supply to the contracting muscles. The blood pH in this zone decreases, which is perceived by the chemoreceptors of the intimate vessels in the hypoxia zone as pain.

1. **Localization of pain.** When the doctor surveys the patient, in all cases it is advisable not just to verbally find out the area of localization of pain, but, clarifying the survey data, to ask the patient to show the place of pain with his finger. After that, it is necessary to present which organ is located in the area of pain. Thus, with cholecystitis, the pain is localized in the right hypochondrium. With gastritis and peptic ulcer - under the xiphoid process. With colitis - in the lower abdomen or in the right or left iliac regions. Pancreatitis is characterized by pain in the left hypochondrium or girdling pain in the mesogastric area.

Depending on the nature of the disease, the pain can be localized or in a certain point, or have a diffuse character. Thus, with peptic ulcer disease, the patient more often shows a specific point of localization of pain, and with gastritis, the patient usually points to the area of localization of pain.

2. **Irradiation of pain.** Pain in diseases of the digestive organs may or may not have certain irradiation zones. With gastritis, the pain does not radiate. With peptic ulcer disease, the pain often radiates to the spine. With cholecystitis - in the right shoulder, hand, shoulder blade. With pancreatitis - in the left subscapular region and in the lumbar region.
3. **By character** pains can have a pronounced emotional color. According to the patient's sensations, they can be sharp, cutting, spasm-like, burning, dull, aching or short-lived, such as stinging when distending the intestines with gases - aching. Burning pains are characteristic of inflammation of the solar plexus (solaritis). With peptic ulcer, the pain is cutting, drilling in nature.
4. **By duration** pain can be short-term, for example, with intestinal ring, gastritis, dyskinesia of the biliary tract, or long-term, permanent, for example, with sunburn.
5. High**intensity of pain** in diseases of the digestive organs, it often speaks about the severity of the disease. Yes, the stabbing pain is quite typical for perforation of the genital organ in the abdominal cavity. However, quite intense pain occurs in patients with fairly harmless dyskinesia of the biliary tract or intestines, and in gastric ulcer and cancer, the pain may not be of great intensity.

6. **Persistence and periodicity** pain Short-term stabbing pains are a common symptom of functional diseases of the stomach and intestines. Gallstone disease is characterized by paroxysmal pain. True biliary colic can last up to several days. With peptic ulcer, the pain is also paroxysmal, but less intense and associated with food. It is also characterized by seasonality of appearance. Usually, peptic ulcer patients are characterized by exacerbations in the spring and autumn, although there may be individual seasonality associated with the peculiarities of the patient's lifestyle or work. With chronic colitis, cholecystitis, the pain is constant throughout the day and night. They are constant, usually whiney, dull.

7. **Frequency of occurrence** pain is different and can be several times a day, a month or less often. With gallstone disease, pain in general can bother the patient several times in several years. With peptic ulcer pain can occur many times a day before or after eating.

8. **Duration** pain is very variable. It can last seconds, hours and even days. Short-term stabbing pains can last only 1-2 seconds. With peptic ulcer, gastritis, the pain lasts 1-2 hours or more.

9. **Reasons contributing to the increase** pain can be different. At the same time, the doctor must establish the relationship between pain and food, its quantity and quality. With gastritis, pain occurs immediately after eating. With peptic ulcer of the stomach with localization of the ulcer in the cardiac part of the stomach, pain occurs 10-15 minutes after eating. With an ulcer in the body of the stomach - 30-45 minutes after eating. With an ulcer in the pyloric part of the stomach, there are so-called late pains - 1.5 - 2 hours after eating. With an ulcer in the duodenum, pain occurs 2-3 hours after eating, "hungry" night pains.

The nature of food defiant pain often helps to establish the nature of the disease. So the appearance of pain after eating spicy, salty, fried food is more typical for gastritis, peptic ulcer disease. Consuming fatty food, eggs, beer, and carbonated water more often causes pain in patients with gall bladder pathology. Dairy food, cabbage, black bread often provoke the appearance of pain in patients with diseases of the large intestine.

10. **Factors that relieve pain**. Taking some medicines, for example, antispasmodics, drugs helps to eliminate pain of a spastic nature, for example, with gallstone disease, tumors of the digestive organs. Often, the pain is eliminated after eating, for example, with peptic ulcer disease of the duodenum. Taking soda is a common, favorite remedy for patients with peptic ulcer pain. These same patients often induce vomiting to relieve pain.

11. It is important to discover**accompanying pain symptoms**. An increase in body temperature, vomiting, jaundice, diarrhea, vegetative crisis phenomena are often accompanied by abdominal pain. In patients with pain in the abdominal cavity, accompanying vegetative crisis can be weakness, profuse sweating, fainting, lowering of blood pressure, numbness in the hands and feet, cold extremities, hot flushes to the head and upper half of the body, retention of urine with subsequent profuse separation. Such a vegetative crisis is often accompanied by pain in peptic ulcer disease, gallstones, pancreatitis. An increase in body temperature, jaundice can occur during an attack of pain in the pathology of the gallbladder, for example, in calculous cholecystitis, a malignant formation. In intestinal diseases, for example, in intestinal dyskinesia with accelerated release, diarrhea occurs after an attack of pain.

In addition to pain, patients with pathology of digestive organs present complaints indicating the connection of their disease with digestive disorders.

Dyspepsia, or indigestion, possibly: 1. gastric, 2. intestinal, 3. hepatic.

Gastric dyspepsia.

The following types of dyspepsia are distinguished:

1. Absence (anorexia) or decreased appetite in diseases of the stomach is explained by the fact that a powerful flow of pain impulses in the central nervous system causes inhibition of the digestive center and decreased appetite. There may be a psychogenic decrease in appetite.
2. Sometimes patients indicate an increase in appetite ("wolf's appetite"), which can occur with peptic ulcer disease, and with endocrine pathology - with diabetes.
3. Aversion to food, especially meat, often occurs in patients with stomach cancer and is purely psychogenic in nature.
4. Perversion of taste suggests the use of inedible products - chalk, clay, coal, sand - occurs in gastritis, especially with secretory insufficiency.
5. A feeling of rapid satiety occurs with hypotonia or when the stomach is lowered (gastroptosis in asthenics).
6. The appearance of an unpleasant taste in the mouth, a metallic, bitter or sour taste, is not uncommon in patients with pathology of the digestive organs. Bitterness in the mouth, especially in the morning, is characteristic of diseases of the biliary tract and liver. With gastritis, a sour taste may appear in the mouth.
7. Bad breath appears with inflammation of the gums, tooth decay, chronic inflammation of the tonsils (tonsillitis), with chronic gastritis, especially with reduced secretory function.

Disturbance of swallowing or difficulty in passing food through the esophagus is called dysphagia. Dysphagia can be caused by both organic and functional disorders, which can be differentiated by careful questioning of the patient. With functional spasms of the esophagus, patients note that only solid food passes freely, and liquid food does not. With tumors of the esophagus, first there are difficulties in passing only solid, and then, when the size of the tumor increases, and liquid food.

Belching is due to the fact that the stomach contracts when the cardiac sphincter is open. The term belching usually refers to two similar phenomena. First, it can be a loud release of air through the mouth from the esophagus and stomach (eructatio). Secondly, when particles of eaten food enter the oral cavity from the stomach together with air (regurgitation with food or regurgitation).

Air belching is often associated with patient swallowing atmospheric air. It is often heard at a distance and is often found in patients with neuroses. In this case, belching of odorless gas occurs. But the gas released during belching often has an odor that indicates one or another disease.

When food stagnates in the stomach with the appearance of putrefactive processes in it, for example, in patients with pyloric stenosis, belching with the smell of rotten eggs can be noted, which indicates the breakdown of sulfur-containing proteins. If these same patients are dominated by fermentation processes, then belching may have the smell of rancid oil due to organic acids that appear in the stomach cavity during fermentation processes.

In patients with increased secretion of gastric juice, belching may acquire a sour taste. Bitter belching occurs with duodenal-gastric reflux, when contents containing bile are thrown from the duodenum into the stomach.

Fecal belching appears in those cases when there is a connection between the stomach and the intestines, for example, when an ulcer penetrates the intestines, during retroperistalsis in patients with acute intestinal obstruction.

Heartburn (pyrosis) - burning along the esophagus or in the area under the breast. It appears when gastric contents are thrown or regurgitated into the esophagus. It is often believed that the appearance of heartburn indicates increased gastric secretion, but this is incorrect. The appearance

of heartburn only indicates the insufficiency of the cardiac sphincter. Stomach contents almost always have an acidic pH, so a feeling of heartburn will always occur when gastric contents are neglected in the esophagus.

Nausea (nausea) is a reflex act associated with the excitation of the vagus nerve, and characterized by a difficult-to-determine feeling of pressure in the epigastric region. Nausea is probably the initial manifestation of irritation of the vomiting center located in the medulla oblongata, in the lower part of the bottom of the sixth ventricle. Sometimes nausea precedes vomiting. During nausea, gastric antiperistalsis is recorded when the cardiac and pyloric sphincters are closed. Nausea is quite characteristic for patients with stomach pathology (gastritis, peptic ulcer disease, stomach cancer, etc.), as well as for irritations of the central nervous system (intoxication, brain injuries, cerebral edema, hypertensive states, etc.). Nausea associated with stomach pathology occurs more often after eating, especially after eating spicy, stomach-irritating food.

Vomiting (emesis, vomitus) is associated with irritation of the vomiting center of the medulla oblongata. At the same time, gastric contents are thrown out through the esophagus, pharynx, mouth, and sometimes through the nose. Depending on the causes of those that caused vomiting, the following are distinguished: 1. vomiting of central origin (pathology of the central nervous system), 2. peripheral or reflex vomiting (in diseases of the digestive organs - gastritis, peptic ulcer disease of the stomach and duodenum, etc.): often relief brings peripheral vomiting and 3. hematogenous-toxic vomiting. Vomiting of central origin is distinguished by the fact that it usually does not bring relief to the patient. Peripheral vomiting associated with irritation of the receptors of the gastric mucosa often brings relief. However, with appendicitis, gallstone disease, the resulting reflex vomiting does not bring relief to the patient.

Vomiting is a very important symptom in various diseases. Therefore, when questioning the patient, they find out the time of vomiting, its connection with food, and the onset of pain syndrome. Be sure to specify the amount of vomitus that has been released, the nature of vomitus (eaten food, gastric juice, rotten masses, mixed with bile, fecal vomit). If the vomited masses contain food eaten - 1-2 days before vomiting, rotten masses, then this rather indicates stenosis of the vakhter with stagnation of food in the stomach. Vomiting of pure gastric juice is more common in peptic ulcer disease. It usually stops the emerging pain syndrome. Vomiting with an admixture of bile indicates duodeno-gastric reflux, for example, in patients with duodenitis, cholecystitis, gallstone disease, and so on. If the nature of the vomiting mass becomes fecal, then this may indicate the appearance of a fecal fistula between the stomach and the colon, for example, with a penetrating stomach ulcer. Fecal vomiting may also appear during retroperistalsis in patients with intestinal obstruction.

A very important characteristic is the connection between the appearance of vomiting and food. If vomiting occurs in the first 10-15 minutes after eating, then this may indicate a pathology in the cardia of the stomach (ulcer or cancer of the cardia of the stomach, pronounced acute gastritis). The appearance of vomiting at the height of gastric digestion 2-3 hours after eating indicates the same pathology in the body of the stomach. With pathology of the duodenum, late vomiting usually occurs - 3-4-8 hours after eating.

The reaction of emetic masses can be different - from acidic to neutral and even alkaline (with vakhter's stenosis, duodeno-gastric reflux, etc.).

The smell of vomitus is usually sour, but, as indicated above, it can be putrid, with the smell of rancid oil, sometimes fecal.

Always carefully assess the nature of impurities in emetic masses. Special attention is paid to blood impurities (gaematemesis). It can be bright red blood when bleeding from the veins of the esophagus. If the vomitus is dark brown in color and has the appearance of coffee grounds, then this is bleeding from the vessels of the stomach in peptic ulcer disease or in the rupture of the gastric mucosa in Mallory-Weiss syndrome.

Other complaints presented by patients with pathology of the digestive organs are:

1. Feeling of rolling after eating in the area under the breast, heaviness, pressure in the epigastrium.
2. A feeling of bloating in the stomach, often associated with increased gas formation in the intestines (flatulence).
3. Rumbling and overflow in the stomach indicate the appearance of intense intestinal peristalsis in combination with increased gas formation.
4. A rumbling noise in the stomach occurs when the motor function of the intestines is disturbed
5. Drooling or salivation may appear with hypersecretion in the stomach, in the presence of worm infestations.
6. Hiccups are convulsive contractions of the diaphragm that occur reflexively during esophageal peristalsis disorders or reflexively, for example, during flatulence. The appearance of hiccups can also be due to neurological pathology, for example inflammation, irritation of the phrenic nerve.
7. obstipacio - a rare release of the intestines, which is carried out less often than 1 time in 48 hours. The appearance of a lock is usually associated with a slowdown in intestinal peristalsis. Severe persistent locks may indicate a congenital abnormality of the development of the large intestine, for example, an increase in the length of the large intestine (dolichosigma) or an increase in the large intestine, both in length and in diameter - megacolon.
8. Diarrhea (diarrhoea) - frequent release of the intestines, which is carried out mostly with the release of liquid fecal masses. The appearance of diarrhea indicates an increase in the activity of intestinal peristalsis and an acceleration of the movement of chyme through the intestines. At the same time, a normal fecal lump does not have time to form. The appearance of diarrhea does not always indicate intestinal pathology. Even the frequent occurrence of diarrhea has a neurogenic nature, for example, with vegetoneurosis.

Peculiarities of anamnesis collection in gastroenterological patients.

When communicating with a patient, the doctor is not a passive listener, but plays an active role.

First of all, you should find out how the disease began: acutely or gradually. Then the nature of the course of the disease is clarified - monotonous, gradual or relapsing course. Thus, peptic ulcer disease usually has a recurrent course, and relapses of the disease can occur quite rarely - once every 2, 3, 4 years. For many functional diseases of the digestive organs, for example, dyskinesia of the biliary tract, irritable bowel syndrome, a monotonous flow is more characteristic.

When questioning the patient, it is important to find out the cause of the exacerbation. These may be violations of the rhythm of nutrition, the recommended diet. It is important to identify the connection of exacerbation with the nature of nutrition, quality and quantity of food, with neuropsychological factors. Be sure to find out which medical institutions the patient went to and when before the actual examination. It is necessary to find out what methods of diagnostic examination were performed on the patient, and what are the results of these studies. It is necessary to find out the effectiveness of the previously used therapeutic measures.

For patients with pathology of the digestive organs, it is very important to collect a detailed dietary history. At the same time, they find out the nature of the food usually consumed by the patient. They are interested in whether the patient's diet is varied or his nutrition is one-sided. It should be remembered that carbohydrate food contributes to the development of fermentative dyspepsia, and protein - to gastritis and other diseases. They clarify the regularity of food. With proper questioning of the patient, an erudite doctor can already establish a presumptive diagnosis of the disease during the questioning of the patient.

Objective methods examination of the abdomen. They include inspection, palpation, percussion, auscultation. Here I would like to dwell only on the most difficult questions for applicants to understand.

Palpation of the organs of the abdominal cavity is an extremely important research method from a diagnostic point of view, which requires a high level of artistry in the work of a doctor. The founder of the method of palpation of abdominal organs is the French doctor of the end of the 19th century, Glenard. In later years, at the beginning of the 20th century, Glenard's method was developed by domestic doctors Zrazkiv and Hausman. It was Hausman who managed to perfect the method of abdominal palpation. It so happened that the developed technique was described by Dr. Hausman in a book published in German and only later - in Russian. Perhaps that is why deep, sliding, topographical, methodical palpation of the abdomen received the name of Obraztsova and Strazhesko.

An extremely important issue in gastroenterology is the order of physical examination of the organs of the abdominal cavity. This procedure must be strictly followed by the doctor who conducts the examination and wishes to obtain the maximum necessary information. The meaning of building this order is simple - when examining a patient, we move from the easiest technique in terms of effect on the stomach to the next one, which has a more intense effect on the organs of the abdominal cavity, which means that it can be more difficult for the patient.

PROCEDURE FOR PHYSICAL EXAMINATION OF ORGANS OF THE ABDOMINAL CAVITY:

1. Examination of the abdomen in a vertical and horizontal position.
2. Superficial or approximate palpation of the abdomen.
3. Identification of zones of skin hyperesthesia Zakharyin-Ged.
4. Abdominal percussion (search for free fluid in the abdominal cavity).
5. Abdominal auscultation.
6. Deep, sliding, topographic, methodical palpation of the abdomen according to Obraztsov - Strazhesko.
7. Determination of pain points and pain symptoms.

Abdominal examination is carried out in the vertical and horizontal position of the patient. The shape of the abdomen is evaluated - the correct shape, retracted abdomen, bulging, lowered (protrusion of the abdomen below the navel).

Determine whether there are hernial protrusions in the area of the white line of the abdomen, in the area of the umbilical ring, in the inguinal areas. When examining a patient in a horizontal position, the "frog's belly" in ascites is better revealed, it is easier to see a tumor in the abdominal cavity, the formation of a cyst, and an increase in organs.

Superficial palpation.

When it is carried out, the patient must take a certain position: lying on his back, legs and arms extended along the body, the doctor sits on a chair next to the patient's bed to the right of the

patient (the doctor's pelvis is at the level of the patient's pelvis). Palpation is carried out by lightly pressing the abdomen in a clockwise direction, starting from the area where there is no pain, slowly and smoothly.
Tasks: 1. Approximate detection of morbidity; 2. Detection of muscle tension of the abdominal wall (defense); 3. Detection of hernial protrusions.
Tension - peritonitis - Shchetkin-Blumberg symptom - pain appears when the hand is removed from the abdomen.
Identification of areas of skin hyperesthesia.
There are 2 methods of determining the areas of skin hyperesthesia: 1. with the help of a needle - kneeling with the same force is carried out with a needle on symmetrical areas of the abdomen; 2. skin roller method - the skin fold is rolled between the doctor's fingers.
This study is based on the fact that impulses from internal organs go to the segments of the spinal cord and return to the skin along the neuron. Therefore, the appearance of skin tenderness indicates the pathology of an internal organ innervated by the same neuron as the skin area.
Thus, with chronic cholecystitis, skin hyperesthesia is detected in the right hypochondrium and in the area of the right scapula. With chronic colitis - in the lower part of the abdomen in the iliac regions (with typhlitis - on the right, and with sigmoiditis - on the left).
In pancreatitis, areas of skin hyperesthesia are found in the left hypochondrium or ring-shaped at the level of the navel.
In such diseases as gastritis, peptic ulcer, enteritis, the area of skin hyperesthesia is not determined.
Abdominal percussion allows you to identify the areas of his pain. Light percussion and palpation reveal point tenderness. In a patient with peptic ulcer disease, point pain in the epigastrium, in the area of the projection of the stomach onto the anterior abdominal wall (Mendel's symptom) is determined. The nature of the sound obtained during percussion in the stomach region is normally tympanic.
Percussion also determines the size of the stomach, liver, and spleen.
It should be noted that if when percussing the area of the liver, its dullness is not determined, and when percussion produces a tympanic sound, this indicates perforation of the stomach or intestines. In patients with percussive ascites, it is very difficult to determine the size of the organs located in the abdominal cavity.
Auscultation abdomen allows to detect intestinal peristalsis. In healthy people, there is usually rumbling, overflow in the intestines, and with peritonitis, intestinal obstruction, complete silence is often found. With peritonitis, it is rarely possible to determine the noise of friction of the peritoneum over the liver and spleen.
Deep, sliding, topographic, methodical palpation of the abdomen according to Obraztsov - Strazhesko.
The meaning of the terms in the name of the method should be explained. "Deep" - the doctor's hand penetrates the stomach to the back abdominal wall. What "slides" - during palpation, the doctor's hand slides in a transverse direction through the intestine. "Topographic" - palpation is performed according to the topographic location of the palpated organ. "Methodical" - palpation of internal organs is carried out in a strict order and sequentially.
The order of deep, sliding, topographic, methodical palpation of the abdomen according to Obraztsov - Strazhesko:
1. sigmoid colon

2. descending
3. blind
4. ascending
5. transverse rim
6. terminal segment of the ileum
7. appendix
8. stomach
9. liver and gallbladder area
10. spleen.

Palpation of the organs of the abdominal cavity is carried out in certain ways*rules*, which ensure the successful work of the doctor. The patient should be in a comfortable lying position. The doctor sits next to the patient at the level of his pelvis. Palpation is usually performed with one right hand. However, it is often necessary to resort to palpation with a "double hand" (bimanual palpation), when the left hand is placed on the right hand to increase the pressure on the tissue. At the same time, the left hand is often used to relax the muscles of the anterior abdominal wall in the area of palpation. It can be located in the lumbar region to bring the palpated organ closer to the right hand or to palpate the organ between two hands.

Palpation of the organs of the abdominal cavity includes*4 moments*: 1. placement of the doctor's hands perpendicular to the axis of the palpated organ or to its edge; 2. displacement of the skin and the formation of a skin fold for the subsequent free movement of the palpating hand; 3. careful immersion of the hand deep into the abdomen during exhalation of the patient to the back abdominal wall or to the palpated organ; 4. sliding with the tips of the fingers in the direction of the transverse axis of the palpated organ. At the same time, they roll through the palpated intestine or slip out of the great curvature of the stomach.

After palpating the intestine, determine its location, diameter, displacement or mobility (easily displaced or fused with surrounding tissues), tenderness, density, surface condition (smooth or bumpy), presence or absence of grumbling upon palpation. The listed criteria allow the doctor to make a conclusion about the presence or absence of a pathological process in the examined organ.

General material and bulk-methodological support of the lecture:

Questions for self-control

1. The main complaints of patients with diseases of the gastrointestinal tract, their characteristics and semiological significance. Examination data of patients with pathology of the gastrointestinal tract and their semiological significance.
2. Examination of the oral cavity, abdomen, their clinical significance.
3. Abdominal palpation. Objectives and tasks during superficial and deep palpation of the abdomen. Division of the abdomen into anatomical regions.
4. Acute and chronic gastritis: classification, main symptoms and syndromes, clinical, laboratory and instrumental methods of diagnosis.
5. The main syndromes and symptoms of peptic ulcer disease of the stomach and duodenum. Diagnostic methods. Complication.
6. The main symptoms and syndromes in diseases of the liver and biliary tract. Hepatitis. Clinical picture, main syndromes. Diagnostic methods.
7. Jaundice syndrome, pathogenetic classification, detection methods. Laboratory diagnostics.

8. Differential diagnosis of jaundice.
9. Cirrhosis. Clinical, laboratory and instrumental methods of diagnosis.
10. Portal hypertension syndrome.

Lecture No. 5
"Main symptoms and syndromes in kidney diseases. Symptomatology of acute and chronic glomerulonephritis and pyelonephritis"

Actuality of theme.

Kidneys, like few other organs, are also the target organ in the case of pathological conditions of other organs and systems, which determines the importance of nephrology as a kind of integrating medical specialty. These lesions can be of different etiology, can have the character of a complication, a secondary lesion. Often, kidney damage against the background of pathological conditions of other organs is caused by the toxic effect of drugs used in the treatment of the primary (main) disease.

The complexity of diagnosis and differential diagnosis of kidney diseases is due to the small and asymptomatic nature of their course. Therefore, their detection requires programmed activity on the part of the doctor, proper nephrological literacy of doctors of various specialties.

Purpose of the lecture:

1. be able to detect symptoms of diseases of the kidneys and urinary system;
2. to know the mechanisms of edematous, nephrotic, hypertensive and urinary syndromes in patients with kidney pathology;
3. to know the main signs during the objective examination of patients with pathology of the urinary system;
4. know additional methods for examining patients with suspected kidney pathology;
5. interpret functional kidney tests;
6. to know the main syndromes in kidney pathology;

Basic concepts:organs of the urinary system, inflammation, pyelonephritis, glomerulonephritis, patient examination, palpation, percussion.

Plan and organizational structure of the lecture.
1. Determination of the educational goal.
2. Providing positive motivation.
3. Presentation of the lecture material according to the plan:
4. Summary of the lecture. General conclusions.
5. The lecturer's answer to possible questions.
6. Tasks for self-training.

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2. www.ama-assn.org – American Medical Association /American Medical Association
3. www.who.int - World Health Organization
4. www.dec.gov.ua/mtd/home/ - State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk – British Medical Association
6. www.gmc-uk.org - *General Medical Council* (*GMC*)
7. www.bundesaerztekammer.de – German Medical Association
8. https://onmedu.edu.ua/
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The main symptoms and syndromes in kidney diseases. Symptomatology of acute and chronic glomerulonephritis and pyelonephritis

Brief anatomical and physiological data

The urinary system consists of kidneys and urinary tract. The buds are located transabdominally in the adipose tissue near the spine at the level between the 11th and 12th thoracic and 2nd and 3rd lumbar vertebrae (right one vertebra below the left) and are covered by a capsule. Inside the buds are the renal pelvises, which pass into the ureters, which flow into the bladder. From the urinary bladder, located in the front part of the small pelvis (behind the pubis), the urethra comes out. Buds have cortical and medullary layers. Kidneys consist of two main systems - glomeruli and tubules. The glomerulus consists of capillary loops and the Bowman's capsule covering them. Bowman's capsule has two leaves lined with epithelium and separated from each other by a gap. The glomerulus with Bowman's capsule forms a Malpighian body. An arteriole that brings blood (vas afferens) approaches the Malpighian corpuscle. A tubule loop emerges from Bowman's capsule. The canal consists of the main, transitional, middle and connecting sections, as well as a system of collecting tubes. The glomerulus-tubule system makes up the nephron (a functional unit). There are more than 3 million nephrons in the buds.

Features of blood circulation in buds are: 1) a large capillary network; 2) high pressure in the renal artery; 3) high intensity of blood circulation (20 times more than in other organs). products of protein metabolism, including nitrogenous slags, water, salts, and some other substances are released from the body through the buds. Normally, the kidneys secrete about 1.5 liters of urine

per day. The mechanism of urine formation is connected with filtration-reabsorption and secretory processes in the nephron. In the glomeruli, blood plasma is filtered (except for proteins) with the formation of "provisional urine". In the tubules, liquid and dense substances are absorbed (reabsorption) and the so-called definitive urine is formed. Dense substances that pass through the buds are divided into threshold, completely reabsorbed in the tubules (for example, sugar), partially threshold (urea) and non-threshold, those that are not reabsorbed in the tubules (creatinine). Glomerular filtration is about 100 liters per day. thus, all the plasma is processed by the buds about 60 times a day. In the buds, there is also a process of secretion by the epithelium of the renal tubules. Some substances that are mainly introduced into the body from the outside, such as drugs, enter the tubules not only through the capillaries of the glomeruli, but also through the blood vessels that depart from the renal artery and feed the tubules, and are excreted in the urine by the secretion of the epithelium of the renal tubules.

KIDNEY RESEARCH METHODS

Questioning. Patients with kidney disease complain of general weakness, headaches, shortness of breath, swelling, nausea, impaired vision, pain in the lumbar region, urinary disorders (dysuric complaints), and a change in the type of urine.

Some additional complaints of patients are caused by kidney disease itself, a violation of their functions. These are complaints about deterioration of health, swelling, skin itching, nausea, vomiting.

Swelling in patients with kidney diseases, they are associated with the fact that the removal of liquid from the body is sharply reduced. These swellings are characterized by a low protein content in the swollen fluid. Because of this, the swellings are soft to the touch, mobile. They appear, first of all, in the area of the eyelids, then they spread to the whole person, and later - to the whole body with the development of cavitary edema and anasarca. Since swellings are mobile, there are often so-called hidden swellings, which are very difficult to detect by conventional methods. Therefore, to establish the presence of edema in patients with kidney pathology, it is advisable to carry out the McClure-Aldridge blister test. When performing this test, 0.2 milliliters of physiological solution is injected intradermally into the palm of the forearm. The time of resorption of the "lemon peel" (blister) that has formed is noted. The control time for blister resorption is 40 minutes. In case of edema, the hydrophilicity of tissues increases and the blister dissolves in less than 30 minutes.

Pathogenesis of the development of "renal" edema.

1. The developing hypoproteinemia leads to a decrease in the oncotic pressure of the blood plasma.
2. Capillary permeability increases.
3. The electrolyte composition of the blood changes.
4. Blood pressure increases.

Other complaints - headaches, visual disturbances, shortness of breath - are explained by increased blood pressure, accompanying kidney disease. Pain in the lower back and urinary disorders (dysuria) are more often associated with urological diseases.

pains In patients with pathology of the kidneys, the usual localization of pain is in the lumbar region.

Patients with nephritis note small, unexpressed pains in the lumbar region, associated with swelling of the buds and stretching from the capsule. Pain in patients with nephritis is noted on both sides and is symmetrical in strength.

With pyelonephritis, the pain is often asymmetric in strength, since in this disease one bud is more often affected. Such pain usually radiates along the course of the ureter to the lower abdomen, to the inguinal region, to the perineum, to the region of the inner surface of the thigh.
With urolithiasis, the pain is very intense, attack-like. During an attack of pain, the patient rushes, excited. At this time, he may vomit. Such pain occurs due to the movement of a stone in the renal pelvis and is called renal colic. An attack of such pain is usually quite long in time, but usually has a clear moment of the beginning and end of the attack.
In patients with cystitis, the pain is localized above the pubis and occurs at the end of the act of urination. With urethritis, pain occurs directly during urination.
Increase***arterial pressure*** in patients with pathology of the kidneys, it differs in that with increased retention of fluid in the patient's body, an edematous syndrome develops, including edema of the intima of the vessel, and the degree of increase in diastolic pressure usually exceeds the degree of increase in systolic pressure.
Dyspeptic disorders not infrequently occur in patients with kidney pathology. Their appearance can be caused by swelling of the intestinal tube and secondary digestive dysfunction. In addition, with renal failure with the development of uremia, slags, ammonia compounds, urea begin to be released through the mucous membrane of the stomach and uremic gastritis develops. It can be accompanied by the development of erosions and ulcers, the appearance of bloody vomiting, diarrhea with blood.
Uremic bronchitis develops when urea is secreted on the mucous membrane of the bronchi. Uremic pericarditis, uremic peritonitis, and uremic pleurisy develop when urea and other slags are released into the pericardial cavity, pleura, and abdominal cavity.
At***anamnesis collection*** it is necessary to pay attention to diseases transferred in the past: sore throat, scarlet fever, malaria, tuberculosis and other infections. It is important to identify chronic purulent-inflammatory diseases (chronic tonsillitis, chronic otitis, abscesses, fistulas), which can be a source of kidney damage. You should also pay attention to occupational hazards: working with lead, mercury and other chemicals that affect buds. Frequent and prolonged cooling are also important.
Review. During the examination, edema is often observed in kidney patients, first on the face, and then on the body, limbs and in the cavities - ascites. Pallor of the skin is often noted, which depends on the spasm and compression of the blood vessels of the skin by the swollen fluid, and in chronic kidney diseases also on the developing anemia. With a purulent disease or with a tumor of the kidneys, there may be swelling on the affected side in the abdominal area and swelling at the back in the lumbar region.
Palpation the bud is made bimanually, as well as by the "voting" method in the position of lying on the back and standing, and sometimes in the position on the side. Normally, the kidneys are not palpable. You can feel the bud when it is enlarged by one and a half to two times or when it is displaced.
There are three measures of kidney displacement: 1 measure - one third - half of the bud can be felt; 2 measure - the entire bud is felt, but on its side (ren mobilis; 3 measure - the entire bud is felt in the other half of the abdomen (ren migrans, or wandering bud). More often, the right bud is displaced.
During palpation with the patient lying on his back, the left palm is placed under the lumbar region, the subcostal edge to the right or left of the spine. The right hand is placed (with slightly bent fingers) on the corresponding side outside of the rectus abdominis below the costal arch.

During deep breathing, the patient's hands are brought together as much as possible, almost until the fingers touch. When changing the location or size of the bud, the lower pole of the bud or the entire bud "slips" between the fingers during inhalation. Buds are also palpated when the patient is standing.

Percussion. Due to the deep location of the buds, percussive determination of their borders is impossible. The method of beating the area of the buds from the side of the waist with the edge of the palm or the fist on the back of the hand is used. Such beating can be painful on the side of the affected bud (Pasternacki's symptom). But the result is better if you ask the patient to stand up on his toes and drop sharply on his heels. At the same time, there is pain in the lumbar region on the side of the affected bud.

Research of other organs and systems. When examining patients with kidney diseases, special attention should be paid to the state of the cardiovascular system, which is often involved in the pathological process in various kidney diseases. One of the frequent symptoms of kidney disease is arterial hypertension. Therefore, it is necessary to measure blood pressure in every patient with kidney disease. An increase in blood pressure can cause hypertrophy and expansion of the left ventricle of the heart and an increase in the second sound on the aorta, which is detected by percussion and auscultation of the heart. In such cases, the electrocardiogram shows signs of hypertrophy of the left ventricle of the heart and sometimes changes in the S-T interval and Tonna's wave, myocardial nutrition, which indicate a violation.

When examining the fundus, changes in retinal vessels associated with arterial hypertension (narrowing of the arteries) may be detected. With severe kidney damage, swelling of the retina and hemorrhage into it may occur.

With amyloidosis, a protein substance - amyloid is deposited in the kidneys, as well as in other organs - the liver, spleen, intestines. In such cases, an enlarged, dense liver and spleen are palpated.

A blood test often reveals a decrease in the number of hemoglobin and erythrocytes, which is especially pronounced in chronic kidney diseases, which are accompanied by a delay in the body of toxic products of protein metabolism. In such cases, leukocytosis and acceleration of ROE can also be observed. With chronic kidney damage, disorders of protein and fat metabolism can occur, resulting in a decrease in the amount of protein in the blood serum (hypoproteinemia) and an increase in cholesterol (hypercholesterolemia).

Urine examination. Examination of urine is very important for the diagnosis of kidney diseases.

Clinical examination of urine includes analysis of physical and chemical properties of urine and microscopic examination of urine sediment. The morning fresh portion of urine, as the most concentrated, is subject to examination.

The study of the physical properties of urine begins with examining it in a glass cylinder. At the same time, the color of urine is noted: straw-yellow or yellow (normal), red, brown-red (due to blood admixture, the effect of some drugs - pyramidone, antipyrine, santonin), green or brown (due to the presence of bile pigments), bright yellow (when treated with nitrofurans, for example, furazolidone), orange (with jaundice due to the content of a large amount of bilirubin), milky white (with phosphaturia and lipiduria), black (with alkaptonuria, hemoglobinuria), blue (secretion of significant amount of methylene blue).

The transparency of urine is determined: transparent (normal urine), cloudy, cloudy (admixture of salts, mucus, cellular elements, fat).

The specific gravity of urine is determined using a urometer - a small-sized hydrometer for a specific gravity of 1000 to 1050.
Under normal conditions, the specific gravity of the morning portion is 1015-1020, at different times of the day it can range from 1001 to 1028.
The reaction of urine is usually determined using a universal indicator paper. In an acidic environment, the paper retains its yellow color, and in an alkaline environment, it becomes green. If the pH of the urine is acidic, and there is a yellowish turbidity in the urine, which disappears when the urine is heated, then, most likely, this turbidity is formed by urate salts. If the urine has an alkaline color and contains a white turbidity that disappears when acetic acid is added to the urine, then this turbidity is formed by phosphates
Chemical examination of urine includes determination of the content of protein, bilirubin, urobilin, sugar and ketone bodies.
There are several chemical reactions to determine the protein content in urine. All of them are based on protein folding or precipitation with special reagents. When glomerular capillaries are damaged, first finely dispersed (albumin) and then coarsely dispersed blood plasma proteins penetrate into the filtrate. With changes in the tubular apparatus, this protein is not reabsorbed and is excreted in the urine. Albuminuria can also appear with functional disorders of the renal filter: orthostatic albuminuria - after physical exertion, with hypothermia, with fever, with starvation, after increased palpation of the kidneys, known stagnant albuminuria and with other diseases.
Determination of bilirubin in urine is done using qualitative tests based on the transformation of bilirubin under the influence of some oxidant, such as copper sulfate and xchloroform, into green biliverdin. You can use the spectroscopic method of determining urobilin. Urobilin gives a characteristic absorption band between the blue and green part of the spectrum, between the Fraunhofer lines E and F.
For the qualitative determination of sugar in urine, the Gaines test is used, based on the ability of glucose to reduce salts of heavy metals. Normally, urine does not contain sugar.
The presence of ketone bodies is also determined in the urine using nitroprusside samples. Ketone bodies can appear in the urine of patients with diabetes.
For microscopic examination, urine is placed in a centrifuge for 5-10 minutes at 1000-1500 revolutions per minute. Centrifuged urine is drained from the sediment by a one-time tilt of the test tube. A drop of the sediment is taken with a pipette, transferred to a glass slide, covered with a cover glass and microscoped with the illuminator lowered, first under low magnification (8x10), and then under high magnification (40x10). 15-20 fields of view are viewed and the average content of certain cells is determined and recorded, for example: squamous epithelium in a small amount, leukocytes 3-7 in the field of view, erythrocytes unchanged or lysed, 10-25 in the field of view, renal epithelial cells single in every field of vision.
When counting leukocytes, the number of so-called active leukocytes, or Sternheimer-Malbin cells, is usually determined. These are large leukocytes that are stained with gentian violet. Their appearance in urine indicates pyelonephritis or prostatitis in men.
Depending on the number of erythrocytes contained in the urine, a distinction is made between macrohematuria (cloudy and reddish when examined) and microhematuria (erythrocytes are detected in an increased amount only during sediment microscopy).
In addition to cells, the urine may contain bodies called cylinders, which are protein casts of the tubules of the loop of Henle, formed during the concentration of primary urine. Since the number of cylinders is often small, they are counted in the entire preparation. If there are a lot of cylinders,

they are counted in the same way as the counting of cells, according to the field of view (the field of view of high magnification is always meant) with the determination of the nature of the cylinders as hyaline, granular, waxy, as well as lipid, prothrombin, epithelial, erythrocyte and leukocyte cylinders.

In addition to cells, the presence of mucus, bacteria and salt crystals is noted. Acidic urine may contain uric acid, oxalates, uric acid ammonium, oxalic lime, alkaline urine - triple phosphates, amorphous phosphates. In some cases, crystals of cholesterol, bilirubin, cysteine, leucine, tyrosine, etc. can be found in urine deposits.

To determine the source of the entry of erythrocytes and leukocytes into the urine, they are used***three-glass sample.*** For this, the patient is given three signed containers, into which he urinates continuously, filling them with urine in approximately the same amount. If during urine microscopy the number of leukocytes or erythrocytes decreases from the first glass to the last glass, then a pathological process (inflammation or trauma) is present in the urethra. If during urine microscopy the number of leukocytes or erythrocytes increases from the first glass to the last glass, then the pathological process is localized in the bladder, from the walls of which, during urination, shaped elements are squeezed out. If leukocytes or erythrocytes are evenly contained in all three glasses, then the source of the formed elements is located above the bladder. If the examined urine does not contain cylinders, then the source of erythrocytes is below the glomerular apparatus of the buds. It can be, for example, a calculus of the renal pelvis, ureter, a disintegrating tumor, etc.

To detect hidden leukocyturia there are a number of tests.

1. *Yako's trial - Addis.* For this, the patient collects urine during the day and the amount of formed elements is determined in the daily sample. Normally, the number of formed elements per 1 liter is: leukocytes - no more than 2 million, erythrocytes - no more than 2 million, cylinders - no more than 1 thousand.
2. *Rehearsal of Amburgh.* With this test, the number of formed elements is calculated for 1 minute of diuresis.

Kakovsky-Addis and Amburghe tests are now rarely used. In practice, they have been supplanted by the three-glass punch and its simplified version - the Nechiporenko punch.

Nechyporenko's trial. When performing this test, the patient begins to urinate in the toilet, collects an average portion of urine in a container according to sensations, and the number of formed elements in 1 liter of urine is calculated. Normally, the content of leukocytes is no more than 4 million per 1 liter, erythrocytes - no more than 1 million, cylinders no more than 250,000 per 1 milliliter.

To detect hidden leukocyturia, it is possible to conduct*provocation test with prednisone.* When setting a prednisolone test, 30 milligrams of prednisone is administered parenterally to the patient, and after 2-3 hours urine is collected for microscopy. Urine is collected again after a day. If the number of leukocytes in the urine increases more than 2 times after a day, the test is considered positive.

Functional studies of kidneys. In patients with kidney diseases, it is important not only to make a diagnosis, but also to determine the functional state of the kidneys. Functional research methods make it possible to determine the extent to which the bud is able to concentrate and remove the end products of metabolism from the body. There are three types of research to determine the functional ability of the kidneys: 1) determination of the concentration and excretory function of the kidneys by measuring the amount and specific gravity of urine; 2) quantitative determination

of the content of end products of protein metabolism in the blood; 3) haemorenal tests, which allow judging the ability of the kidneys to cleanse the blood of protein impurities.

Volgard offered two samples - for dilution and for concentration. In the first sample, for dilution, after weighing, the patient drinks one and a half liters of water on an empty stomach and collects urine every half hour for four hours. The volume and specific gravity of each portion is measured. Normally, urine (one and a half liters) is excreted in 4 hours, one of the portions, more often the third, should be at least 300.0 cm3 with a drop in the specific gravity in it to 1001-1002, after 4 hours the weighing is repeated. This test makes it possible to judge the water-excreting function of the kidneys, although a number of extrarenal factors also affect water excretion.

With the second, concentration sample, dry eating is carried out. After the water load, the patient is not given liquid until the next morning. Urine is collected every two hours. Normally, in each portion of urine, a little is released - 20.0-60.0 cm3, the specific gravity by the end of the day reaches 1030 and sometimes it is higher. In cases of functional insufficiency of the kidneys during the Folgard test, the specific gravity of urine remains monotonous, low, with large fluctuations in the volume of portions - isohypostenuria.

Folgard's tests are non-physiological, as they place buds in artificial conditions of water stress or dehydration. In addition, loading with a large amount of water can be harmful for a patient with a tendency to edema, and dry food - harmful for a patient with a delay in the release of nitrogenous wastes. Therefore, these tests are now rarely used in the clinic and are replaced by a simpler, less harmful and physiological test proposed by S.S. Zimnytskyi

Zimnytsky's trial is carried out in this way.

The patient with his usual eating and drinking regimen collects urine every 3 hours during the day (8 portions). The amount of urine and specific gravity are determined in each portion. Normally, the amount of urine and specific gravity fluctuate within wide limits, daytime diuresis is greater than nighttime. With a decrease in the concentration function of the kidneys, the specific gravity in all proportions of urine is low - isohypostenuria. If the water excretory function of the kidneys is also impaired, urine is scarce in all proportions, and its specific gravity remains low. Thus, Zimnytsky's test allows us to judge the state of the concentration and excretory functions of the kidneys. Normally, the specific gravity of urine during the day ranges from 1003 to 1030. At night, the concentration function of the kidneys is higher, and the amount of urine excreted is lower than during the day. The usual ratio of daytime and nighttime diuresis is 4:1. The total volume of daily urine is 1-2 liters. When evaluating Zimnytsky's test, the following terms are used: polyuria - a large volume of excreted urine, oliguria - a small amount of urine, anuria - the absence of urine excretion, isosthenuria - fluctuations in the daily specific gravity of urine are insignificant, hyposthenuria - the specific gravity of urine is small.

It is known, although not so often used in practice, functional*Reiselman test*. When performing this test, the patient collects each portion of urine that is released into separate containers, not according to time, but according to desire. The assessment of Raiselman's test is carried out in the same way as the assessment of Zimnytskyi's test.

The ability of the kidneys to release the end products of protein metabolism from the body can be judged by the results of a biochemical blood test. Most often, the content of the so-called residual nitrogen in the blood, which remains in the blood after the complete precipitation of protein metabolism - urea, uric acid, creatinine, indican - is determined. Normally, the residual nitrogen content indicates a violation of the release of nitrogenous slags by the buds. With various kidney diseases, the content of residual nitrogen can be 100-200 mg% or more. Kidney dysfunction can

also be judged by examining the content of individual fractions of residual nitrogen in the blood. Normally, the content of urea in the blood is 20-40 mg%, uric acid - 2-4 mg%, creatinine - 0.5-1.5 mg%, indican - 0.05-0.1 mg%.

The third group of functional methods of kidney research is based on the comparative determination of the amount of nitrogenous and some other substances in the blood and urine - these are the so-called haemorenal tests. They provide more accurate definitions of the excretory function of the kidneys, and also allow determining the functional functions of the kidneys - the amount of glomerular filtration, tubular reabsorption and the amount of blood flowing per unit of time through the kidneys (the amount of effective renal blood flow).

Such studies include*deporation test(*purification) of blood from urea. The content of urea in the blood and in the urine collected over a certain period of time is studied. Comparison of the obtained data allows judging the ability of the kidneys to purify the blood from urea per unit of time (Van-Slyke test).

According to the same principle, a study is conducted to clean the blood from creatinine (*Rehberg's test in the modification of E.N. Tareeva*). Creatinine belongs to non-threshold substances: passing through the filter of the kidney glomeruli, it is not reabsorbed in the tubules, nor is it secreted by the epithelium of the kidney tubules. Therefore, by the amount of blood cleared of creatinine per minute, it is possible to judge the amount of glomerular filtration per minute. Knowing the amount of glomerular filtration and the volume of diuresis per minute (an hourly portion of urine divided by 60), it is possible to calculate the amount of tubular reabsorption, that is, to determine what percentage of the liquid that passes through the glomeruli is reabsorbed in the tubules. Normally, the amount of glomerular filtration reaches 80-120 ml per minute. The amount of tubular reabsorption is normally 97-99% of the entire liquid part of urine filtered in the glomeruli. Thus, Rehberg's test makes it possible to determine the state of partial functions of the kidneys - glomerular filtration and tubular reabsorption.

X-ray examination includes an overview picture of the kidneys and pictures during the previous filling of the renal pelvis with a contrast agent (bromine or iodine salt solutions) through the urethra or intravenously - pyelography, as well as an X-ray of the kidneys against the background of injected intraperitoneal oxygen - pneumoren.

Cystoscopy - endoscopic method of examination of the mucous membrane of the urinary bladder using a special device - a cystoscope. Cystoscopy is widely used in urological practice. During cystoscopy, its variant - chromocystoscopy - is often used. During chromocystoscopy, the patient is intravenously injected with a solution of methylene blue, which quickly begins to be secreted by buds. During the examination, the doctor sees how blue urine begins to flow from the ureters. If any bud does not function, is absent, or the ureter is impassable, then colored urine does not flow from the ureter on the side of the lesion.

Ultrasound examination of urinary organs extremely widely used in medical practice. This technique allows you to detect the position of the buds, their size, shape, and the presence of concretions.

Radioisotope examination of the kidneys involves radioisotope scanning of the kidneys and secretory urography.

When scanning the buds, the patient is intravenously injected with a solution of a substance marked with a radioactive isotope, for example, a solution of neohydrin containing radioactive mercury Hg197, and then the accumulation of the injected drug in the buds is determined with the help of a scanner. This allows you to determine the size, shape and size of the buds. According to

the intensity of the obtained shadow of the buds, focal defects of the buds are revealed, which indicate a tumor, tubercular damage to the kidneys, and other destructive processes.
When conducting isotopic secretory nephrography, the patient is intravenously injected with solutions of diotrast or hippuran, marked with a radioactive isotope of iodine (I131

MAIN SYNDROMES IN KIDNEY DISEASES

Urinary disorders. Normally, a person excretes about 100% of the liquid he drinks per day, which is about 1.5 liters of urine. The excretion of urine for a known period of time, for example, for a day, is called *diuresis An increase in the amount of urine excreted by more than 2 liters per day is called polyuria*. It can have both renal and extrarenal origin. Polyuria occurs in diabetes mellitus and in non-diabetic (pituitary) enuresis, in the progression of edema, simply in the case of generous liquid consumption, as well as in the case of a decrease in the concentration capacity of the kidneys, when the excretion of nitrogenous wastes occurs due to an increase in the amount of urine - compensatory or forced polyuria.
In case of violation of the formation of urine in the glomeruli or increased reabsorption in the tubules, it occurs*oliguria - a decrease in the amount of urine. Anury* - complete cessation of urination. Secretory urine is not formed in the glomeruli, excretory urine cannot enter the bladder and be excreted. In addition to impaired kidney function, anuria can be due to mechanical reasons - blockage by a stone, spasm of the urinary tract (reflex anuria).
Nocturia - the predominant discharge of urine at night, when the rhythm of urination is disturbed, is often observed in heart diseases. Frequent urination is called pollakiuria. A disorder of the adaptive function of the kidneys is indicated by isuria - excretion of urine in portions of the same volume throughout the day.
Hyposthenuria - the release of urine of low specific gravity. Isosthenuria - constant, long-term excretion of urine all the time of the same low specific gravity without fluctuations. Dysuria is called a urinary disorder, for example, painful, difficult urination or frequent urination, accompanied by pain and cuts in the urethra. Dysuria is usually observed in diseases of the urinary tract - bladder, urethra.
Swelling - a frequent syndrome in kidney disease. The pathogenesis of edema in kidney patients is different and can be explained by the following factors:
a) violation of the processes of filtration and reabsorption, which leads to the retention of sodium chloride and water in the tissues;
b) increased permeability of capillaries;
c) a decrease in the amount of protein in the blood - hypoproteinemia, especially due to shallowly dispersed proteins - albumin, which lead to a decrease in the oncotic pressure of the blood and to the exit of the liquid part of the blood from the blood vessels in the tissue.
Edemas in kidney patients are often associated with albuminuria and protein metabolism disorders. The excretion of large amounts of protein in the urine over a long period of time leads to a decrease in the content of protein in the blood - hypoproteinemia. (Mainly the finely dispersed fraction of proteins - albumin) decreases. This leads to a decrease in oncotic blood pressure and the formation of edema. The so-called edematous-albuminuric syndrome develops, which is characteristic of some chronic diseases of the kidneys, mainly for dystrophic lesions of the kidneys - nephroz, therefore the syndrome is also called nephrotic.
Renal edema, unlike cardiac edema, occurs quickly. Swelling begins in the tissues of the paraorbital region, the eyelids, then spreads to the face, since it is in these places that the most

vascularized loose fiber is found. Then the edema spreads throughout the body and can be very significant in the serous cavities and the substance of the brain. Since the swollen liquid during the development of renal edema contains little protein, the edema is watery, soft and mobile. They are not accompanied by liver enlargement, tachycardia, or cyanosis.

Arterial hypertension - is a frequent symptom of kidney diseases. The pathogenesis of renal hypertension is complex. An increase in blood pressure is associated with the pathology of the capillary network of the kidney glomeruli and with a violation of the humoral function of the kidneys. When the cells of the juxta-glomerular apparatus of the renal glomerulus are irritated, renin is released, which, combining with alpha-globulin of the blood, forms angiotensin, which has a pronounced pressor effect. Persistent arterial hypertension leads to the development of hypertensive syndrome, characteristic of a number of kidney diseases.

Renal hypertension proceeds with the same changes in the internal organs as essential hypertension: hypertrophy and expansion of the left ventricle of the heart develops with corresponding changes on the X-ray of the heart and the electrocardiogram. With pronounced changes in the heart, symptoms of acute left ventricular failure can occur in the form of cardiac asthma attacks. During the examination of the fundus, changes in retinal vessels are revealed (retinal angiopathy of the fundus), and in more severe cases - phenomena of retinopathy: swelling of the papillae of the optic nerves, hemorrhages in the retina. Changes in the fundus in patients with renal hypertension, although similar to changes in patients with essential hypertension, are still somewhat different. The fact is that changes in the fundus in patients with kidney pathology are explained not only by spasm of blood vessels, but also by increased permeability of capillaries. In the first period of renal retinopathy, or renal neuroretinitis, there is some narrowing of the arteries and arterioles of the retina and flattening of the venules under the arteries crossing them. Venules before this intersection have a small ampoule-like expansion. This is called the Hann-Salus I symptom.

In the later stages of the disease, as a result of prolonged spasm of arterioles and their hyalinosis, arterioles become narrowed, the arteries preceding them twist. Veins are squeezed by arteries that cross them. In front of the place where they cross, the ampoule-like expansion of the veins is more pronounced. This is a symptom of Salus II. In the final stages of the disease, sclerosed arteries and arterioles resemble silver wire. Venules also become sclerosed and, before crossing them with arteries, are pressed into the depth of the retina with the illusion of a break. This is a symptom of Salus III.

With high hypertension, which is combined with large edemas, cerebral edema with attacks of renal eclampsia may occur.

Renal eclampsia syndrome (from the Greek word eclampsis - convulsion) develops in patients with edematous syndrome. Most often, eclampsia develops in patients with acute glomerulonephritis, although it can also occur with exacerbations of chronic glomerulonephritis, nephropathy of pregnant women. The cause of renal eclampsia is spasm of cerebral vessels with impaired permeability, increased intracranial pressure and swelling of the brain substance. The development of eclampsia is provoked by a large intake of liquids and the consumption of salty food.

Eclampsia develops against the background of high blood pressure and large edema. Its first signs are unusual for the patient weakness, lethargy, drowsiness. Then a severe headache, vomiting, speech disorders, fleeting paralysis, clouded consciousness appear. At this time, the patient's blood

pressure rises very much. Convulsions appear unexpectedly, sometimes they are preceded by a short scream or a deep noisy sigh of the patient. In the first 30-90 seconds of a convulsive attack, strong tonic muscle contractions are noted, which are then replaced by clonic convulsions or twitching of individual muscle groups, involuntary urination, defecation, and speech disorders. The patient's face turns blue, the eyes roll back or slant to the side. It should be noted that during an eclampsia attack, the pupils remain wide.

Eclampsia attacks last several minutes, sometimes longer. Usually, after 2-3 attacks, the patient calms down and enters a deep sopor or coma. After the patient regains consciousness for some time, he may have amaurosis (blindness of central origin) and aphasia (loss of speech). Erased attacks of eclampsia are possible. It should be remembered that renal eclampsia is a life-threatening condition that requires immediate medical attention.

Another, extremely difficult syndrome in patients with renal failure is***uremia (from the Greek words uron - urinating and haima - blood). A distinction is made between uremia that occurred in acute and chronic cases. Pathogenesis*** *uremia* complex. The main role is played by the retention of products of nitrogenous metabolism in the body - urea, uric acid and creatinine. There are changes in mineral metabolism (increased potassium, sodium and phosphorus content), the development of acidosis. The specific gravity of urine falls, and the level of residual nitrogen in the blood increases, sometimes to significant levels. Acidic products of intermediate metabolism accumulate in the blood, acidosis occurs.

In the pathogenesis of acute renal failure and acute uremia, the main importance is attributed to shock and accompanying hemodynamic disturbances, primarily in the kidneys.

In cases where acute renal failure develops due to intoxication or severe infection, the pathogenesis of uremia is due to the direct effect of toxins on the renal parenchyma.

A patient with uremia complains of weakness, apathy, headaches, and loss of appetite. The skin is pale, with an earthy gray or waxy tint. Swelling in this stage of the disease almost passes. The puffiness of the face remains, and pastiness on the lower legs. Dry, flaky skin. Skin itching and traces of scratching often occur. Sometimes the skin is as if sprinkled with powder or frost, which is associated with the release of urea crystals by the sweat glands. The tongue and mucous membranes of the oral cavity are dry. There is a smell of urine from the mouth - foetor ex orae and from the patient's body. A whole range of symptoms of uremia is associated with the compensatory release of nitrogenous slags by various glands - sweat, gastric, intestinal. Typical nausea and vomiting are signs of secretory uremic gastritis. Uremic colitis develops, sometimes ulcerative, which is accompanied by diarrhea, often hemorrhagic. All this leads to dehydration of the body, thirst, dryness of the skin and mucous membranes, exhaustion. Deposition of urea crystals sometimes occurs on serous membranes - pleura, pericardium and causes the development of aseptic dry pleurisy and pericarditis. The noise of friction of the pericardium, which is heard at the same time, is usually observed in the terminal stage of the disease and, according to the figurative expression of French clinicians, is called the "Funeral bell". The body temperature drops a little. Urine is released very little, up to the development of complete anuria.

One of the signs of intoxication of the body in uremia is suppression of the hematopoietic function of the red bud. The number of erythrocytes and hemoglobin decreases significantly, severe hypochromic anemia develops. The number of blood leukocytes is usually increased. The ability of blood to clot is disturbed and hemorrhagic diathesis phenomena develop. Examination of the patient reveals skin hemorrhages. There is a tendency to bleeding from the nose, gastrointestinal tract, urinary tract, uterus. Patients can develop hemorrhages in any internal organs.

Marked signs of intoxication of the nervous system: lethargy, drowsiness, stiffness, disorder of consciousness, sluggish reaction of the pupils to light, small twitches of individual muscle groups. As a rule, with uremia, there is chemical irritation of the receptors of the serous membranes of the cavities of the peritoneum, pleura, pericardium, and joints. Therefore, patients experience severe pain throughout the body, both at rest and when moving. A high concentration of ammonia in the blood stimulates the brain and disrupts the normal alternation of periods of sleep and vigilance. A comatose state gradually develops - a uremic coma, which is the final stage of the disease. At this time, patients lose consciousness, Kussmaul's noisy deep breathing appears, as a manifestation of severe acidosis. In the final stage of renal failure, the patient is in a deep coma. Sometimes he has individual muscle twitches, after some time death comes.

Changes in urine kidney disease is characterized by the appearance of protein (albuminuria) and formed elements of blood in the sedimentation of urine.
Violation of the excretory function of the kidneys leads to a delay in the body of the end products of protein metabolism, which have a toxic effect. The content of residual nitrogen and its fractions increases in the blood - azotemia. Clinically, this is manifested by uremia syndrome, which is the result of intoxication of the body with products of protein metabolism. Uremia can develop with various kidney diseases: with chronic nephritis, amyloidosis, arteriolosclerosis of the kidneys, with necrotic nephrosis.

General material and mass-methodological support
lectures:
work program of the academic discipline
synopsis (plan-summary) of the lecture
multimedia presentation of the lecture

Questions for self-control:

1. The main complaints of patients with urinary diseases, the mechanism of their development and semiological significance.
2. Characteristic signs during examination of patients with pathology of the urinary system. Results of palpation and percussion of the kidneys. Edema syndrome in kidney disease. Mechanism of occurrence, features of renal and cardiac edema.
3. Mechanism of development of arterial hypertension syndrome in kidney diseases. Peculiarities of blood pressure profile in kidney pathology. Complication of renal arterial hypertension.
4. Urinary syndrome with kidney disease. Clinical and laboratory signs of urinary syndrome.
5. Nephrotic syndrome in kidney pathology. Causes, symptomatology and clinical and laboratory diagnostics.
6. Syndrome of chronic renal failure. Etiology, clinical signs. Diagnosis of CKD syndrome.
7. Chronic glomerulonephritis: etiology, symptomatology, laboratory data, principles of treatment.
8. Chronic pyelonephritis: etiology, symptomatology, laboratory data, principles of treatment.

Lecture No. 6 «**The main symptoms and syndromes in diseases of the hematopoietic system. General blood test. Symptomatology and diagnosis of anemia, leukemia. Hemorrhagic syndrome (hemorrhagic diatheses)."**

Actuality of theme.
Diseases of hematopoietic organs can be encountered in the practice of a doctor of any specialty, especially in the practice of a doctor. This especially applies to anemias of various origins.
The ability to collect and classify complaints of patients with blood diseases, their anamnesis, to conduct an objective examination of such patients; correctly evaluating laboratory data makes it possible to immediately correctly and timely diagnose a blood disease, which means, in turn, timely and correct treatment. Formation of the future doctor's skills and abilities in the examination of such patients is necessary for their future successful medical activity.
Blood testing is one of the more important and necessary methods of additional testing of patients. In terms of its value, blood research is no less important than the main research methods. In any case, a general blood test is mandatory for the examination of an expensive patient, both outpatient and inpatient, and therefore it can be classified as one of the main research methods. Thus, blood research is a necessary element of the training of a clinician, and without a perfect mastery of it, the training of a doctor is impossible.

Purpose of the lecture:

1. the applicant must know the general classification of diseases of the kpovy
2. the applicant must know the method of survey, objective examination of patients with blood diseases
3. the applicant must know the semiotics and symptomatology of blood diseases
4. the applicant must know the standards of basic blood laboratory tests
5. the applicant must be able to collect and evaluate complaints, anamnesis of patients with blood diseases
6. the applicant must be able to conduct an objective examination of patients with blood diseases
7. the applicant must be able to draw up a plan for additional examination of these patients
8. the applicant must be able to give a clinical assessment of the data of a general blood test

Basic concepts: hematopoietic organs, general blood analysis, formed blood elements, patient examination, palpation, percussion.

Plan and organizational structure of the lecture.
1. Determination of the educational goal.
2. Providing positive motivation.
3. Presentation of the lecture material according to the plan:
4. Summary of the lecture. General conclusions.
5. The lecturer's answer to possible questions.
6. Tasks for self-training.

Content of the lecture material

Brief anatomical and physiological data

Blood is a special tissue in which peculiar cells (blood corpuscles) are freely suspended in a liquid medium. When we talk about blood diseases, we usually mean disorders in that part of the blood that is formed in hematopoietic organs, namely, disorders in its cellular composition.

The amount of blood in a person under normal conditions is from 1/13 to 1/20 of the body weight. The color of blood depends on the content of oxyhemoglobin in it. The specific gravity of blood is 1060 in men and 1056 in women. The blood reaction is slightly alkaline and is 7.35 (at 38ºC).

The liquid part of blood, plasma, consists of 90% water. Its dense substances are represented by proteins (albumin, globulins, fibrinogen), sugar, cholesterol, nitrogenous products and the like. Plasma deprived of fibrinogen is called serum.

The ratio between plasma and formed elements is subject to fluctuations, but in general hematocrit is 40-48% in men and 36-42% in women.

The hematopoietic system includes organs in which the processes of hematopoiesis and blood destruction take place, and the blood itself with all its cellular elements, chemical substances and plasma. Hematopoietic organs include bone marrow, lymph nodes and spleen.

Cellular, or formed, elements of blood are products of vital activity of hematopoietic organs, from where they enter the blood and perform certain

functions in the body. In recent decades, the unitary theory of hematopoiesis, according to which all blood cells originate from the reticular cell, has been revised. It is believed that at the base of the genealogical tree of all cellular elements of the blood lies a stem cell, morphologically similar to a lymphocyte. This cell can develop in different directions, as a result of which red blood cells - erythrocytes, white blood cells - leukocytes and blood platelets - thrombocytes are formed. Each of these cells passes through intermediate stages of development, reaching the stage of a mature cell. This process of blood cell development takes place in hematopoietic organs. The blood of a healthy person contains only mature cells. In diseases of the hematopoietic system, immature (intermediate) cells may appear in the blood. In the bone marrow, erythrocytes, platelets and a part of leukocytes belonging to granular cells - granulocytes are produced. Granulocytes, in turn, are divided into neutrophils, eosinophils and basophils. Bone marrow tissue that produces blood cells is called myeloid tissue. It consists of a red hematopoietic germ, which produces erythrocytes, a white hematopoietic germ, which produces granulocytes (granular leukocytes), and a platelet hematopoietic germ, which produces platelets.

In the lymph nodes, lymphocytes are produced - cells belonging to agranular leukocytes (agranulocytes).

Research methods

Questioning of patients with blood diseases. Complaints of patients with diseases of the hematopoietic system are diverse. In most cases, patients present complaints of a general nature: weakness, fatigue, decreased appetite, headaches, dizziness, fainting, palpitations, shortness of breath. With some diseases, patients complain of bleeding gums, nosebleeds, bloody vomiting and bloody stools, uterine bleeding, and the appearance of various sized hemorrhages on the skin. In some cases, patients complain of skin itching, profuse sweating, and fever. A peculiar complaint can be a burning sensation at the tip of the tongue. Sometimes a perversion of taste is noted - the patient has a need to eat chalk, clay, coal.

When taking an anamnesis, it is necessary to ask the patient about past diseases, especially infectious ones, which may be the cause of changes in the hematopoietic system. Infections such

as malaria, tuberculosis, and syphilis are of particular importance. It is also important to identify chronic foci of infections: tonsillitis, sinusitis, otitis, cholecystitis, and others. Worm infestations can be of great importance in the origin of diseases of the hematopoietic system.

It is also necessary to ask the patient about the presence of bleeding in the past (nasal, uterine, from the gastrointestinal tract), which may be the cause of the development of anemia. It is also necessary to take into account that long-term use of some drugs, for example, pyramidone, sulfonamide drugs, methylthiouracil, can lead to suppression of bone marrow function and a decrease in the number of white and red blood cells in the blood.

It is recommended to pay attention to the nature of the patient's nutrition (lack of proteins and vitamins in food). Collecting a professional history is important. Because working with some chemicals (benzene, arsenic, phosphorus, lead), as well as with X-rays, radioactive isotopes can in some cases cause damage to the hematopoietic system.

Review. When examining the patient, first of all, you should pay attention to the color of the skin and visible mucous membranes. When the number of erythrocytes and hemoglobin in the blood decreases (anemia), the skin and mucous membranes become pale. In some forms of anemia, pallor of the skin is combined with jaundice. With an increase in the amount of hemoglobin and erythrocytes in the blood (polycythemia and erythrema), the skin acquires a dark red color, sometimes with a bluish tint.

With a number of diseases of the hematopoietic system, hemorrhages of various sizes and locations may appear on the skin. There may be small point hemorrhages - petechiae and larger hemorrhages in the skin in the form of blood spots, which later turn into bruises. Hemorrhages can also be detected on the mucous membrane of the oral cavity, gums and conjunctiva of the eyes. When examining the oral cavity, one should pay attention to the condition of the gums (swelling, loose, bleeding), tongue (redness of the tongue, cracks, aphthous rashes, sometimes smooth shiny tongue with atrophied papillae), mucous membrane of the oral cavity, tonsils. With severe lesions of the hematopoietic system (leukemia, agranulocytosis), necrotic changes develop in the oral cavity and tonsils.

Palpation. A number of diseases of the hematopoietic system are accompanied by a multiple increase in lymph nodes as a result of hyperplasia of lymphoid tissue. During palpation, an increase in cervical, supraclavicular, axillary, inguinal and other lymph nodes can be determined. Nodes are hard or soft elastic consistency, mobile during palpation. Sometimes they grow together and with the surrounding tissues, forming dense conglomerates.

Palpation of the abdominal cavity often reveals enlargement of the liver and spleen. In some diseases (chronic leukemia, hemolytic anemia), the spleen is huge, becomes dense, with a smooth surface and a rounded edge.

Change of other organs and systems. Tachycardia, increased sonority of heart sounds and the appearance of a functional systolic murmur at the apex of the heart are often observed with anemia.

When examining the stomach contents, achillea is sometimes found, which plays a decisive role in the development of some forms of anemia. Urinalysis may reveal hematuria, usually with other bleeding.

Blood tests are the main method of diagnosing diseases of the hematopoietic system, as well as an important diagnostic method for a wide variety of diseases. All the numerous methods of blood research used in the clinic are divided into morphological, biochemical, bacteriological and serological. These include: calculation of the number of erythrocytes and leukocytes, a qualitative

study of erythrocytes and leukocytes with the study of the leukocyte formula, determination of hemoglobin, calculation of the blood color index and erythrocyte sedimentation rate (ESR). All these studies are called a general clinical blood test. Normally, the rate of sedimentation of erythrocytes in men is 1-10 mm per hour, in women 2-15 mm per hour. The level of hemoglobin in men is 130-160 g/l, in women 120-140 g/l. To count the number of erythrocytes and leukocytes, blood is diluted in special mixers. The normal content of erythrocytes in men is 4 x 1012 /l - 5.1 x 1012 /l, in women it is 3.7 x 1012 /l - 4.7 x 1012 /l. The number of leukocytes - 4 x 109 /l - 8.8 x 109 /l.
The color indicator of blood expresses the ratio of the amount of hemoglobin to the number of erythrocytes. Normally, the color index of blood is 0.86-1.05. The percentage ratio of individual types of leukocytes is expressed by the leukocyte formula - leukogram.

Normal content of leukocytes in adults
Types of leukocytes Content %
Basophils 0-1
Eosinophils 0-5
Neutrophils are rod-shaped
segmento-nuclear 1-6
45-70
Lymphocytes 18-40
Monocytes 2-9

Research on hemorrhagic syndrome. In cases of propensity to bleeding, special laboratory methods of blood coagulation research are used. They include mandatory classic tests. What determines the activity (blood clotting ability, number of platelets, duration of bleeding, blood clot retraction, capillary resistance) and differentiated tests that determine the activity of individual phases and components of the blood coagulation system.
Blood clotting time. Of all the methods for determining the spontaneous coagulation of whole blood, the Lee-White method, which gives a normal coagulation equal to 4-9 minutes, received wide recognition.
Bleeding time. When a fingertip or earlobe is pricked to a depth of 3 mm (Dick's test), the duration of bleeding normally should not exceed 3 minutes. Bleeding time is dramatically extended in thrombocytopenic conditions, liver diseases, phosphorus and chlorine poisoning.
Thrombocytes (blood platelets). The number of platelets is counted in a stained blood smear (dry method) or in a chamber (wet method). Under normal conditions, the number of platelets is 180-320 x 109 /l of blood, and it is subject to significant fluctuations during the day. At the beginning of the menstrual cycle, their number can decrease by 30-50% from the initial level. A sharp decrease in platelets is observed in thrombocytopenic purpura (Werlhoff's disease).
Clot retraction. Retraction of a blood clot means its ability to contract and separate serum. A simple method can be used to determine retraction. 0.5-1.0 ml of venous blood is injected into a small test tube and monitored. How long does it take for the first drop of serum to come out? Normal time is from 1 to 5 hours. The degree of retraction depends on the number and quality of platelets.
Capillary resistance. 1. Symptom of the harness, or symptom of M.P. Konchalovsky - Rumpel - Leede, it is considered positive if petechiae appear no later than 3 minutes after applying the

tourniquet, or if after 15 minutes of applying the tonometer cuff at a pressure of 50 mm Hg, at least one petechiae per 1 cm2 appears skin 2. A symptom of an injection - the appearance of skin hemorrhages after needle injections. 3. Pinch symptom (Moser's symptom) - a hemorrhagic spot quickly appears at the place of the pinch.
Positive test results are observed in various vascular hemorrhagic diathesis (hemorrhagic vasculitis, scurvy, etc.), with thrombocytopenia and thrombocytopenia (Werlhoff's disease, hemorrhagic thrombocytopenia and Glazmann's disease, etc.).
Prombinovy index according to Kwik. The prothrombin index (PI) reflects the total activity of the so-called prothrombin complex (factors II, V, VII, X), as well as the activity of heparin-like substances. The normal value of PI is 80-100%.
Fibrinogen. The content of fibrinogen normally ranges from 2 to 4 g/l.
General material and mass-methodological support
lectures:
work program of the academic discipline
synopsis (plan-summary) of the lecture
multimedia presentation of the lecture

Questions for self-control:
1. Anemia syndrome. Clinical and laboratory signs of anemia.
2. Classification of anemias
3. Iron deficiency anemia. Clinical picture. Principles of treatment.
4. B12 deficiency anemia. Clinical picture. Principles of treatment.
5. Posthemorrhagic anemia. Clinical picture. Principles of treatment.

Lecture No. 7 "Main symptoms and syndromes in diseases of the endocrine system. Diabetes. Diffuse toxic goiter."

Actuality of theme.

Diseases of internal secretion glands are well differentiated by the clinical picture. Some of them have such a characteristic clinic (for example, Bazedov's disease) that the diagnosis does not present difficulties. Other forms of diseases (for example, "erased forms" of the same underlying disease) require more careful differentiation with the involvement of laboratory studies (for example, determination of the main metabolism). Diagnosis of diseases such as diabetes requires a complex laboratory examination of the patient. In a word, when studying endocrine disorders, they use all the methods of examining patients, outlined in the course of propaedeutics and a large number of complex laboratory biochemical analyses.
Purpose of the lecture:

1. - the applicant must know the mechanisms of action of the hormones of internal secretion glands in normal and pathological conditions;

2. - must know the main symptoms characteristic of the defeat of the glands of internal secretion;
3. - master the main complaints characteristic of diseases of internal secretion organs;
4. - must be able to examine patients with endocrine pathology;
5. - main symptoms during physical examination of patients with pathology of the endocrine system;
6. - to know the main additional research methods used for the diagnosis of endocrine pathology; **Plan and organizational structure of the lecture.**

1. Determination of the educational goal.
2. Providing positive motivation.
3. Presentation of the lecture material according to the plan:
4. Summary of the lecture. General conclusions.
5. The lecturer's answer to possible questions.
6. Tasks for self-training.

Basic concepts: endocrine system, hormone, patient examination, diabetes, hyperthyroidism, hypothyroidism**.**

"Main symptoms and syndromes in diseases of the endocrine system. Diabetes. Diffuse toxic goiter."

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Complaints patients, taking into account the versatile influence of the glands of internal secretion on various functions of the body, are very diverse. Patients may complain of increased mental excitability, irritability, intermittent and shallow sleep, decreased memory, sweating, palpitations, chills, tinnitus, rush of blood to the head, weight loss. When questioning patients, the patient's neuropsychological appearance, characteristic of some endocrine disorders, is revealed - fussiness, apathy, lethargy, and others. Delays in mental development may be detected (with critinism).

Anamnesis. When taking an anamnesis, you should find out the causes preceding the onset of the disease - mental trauma, the period of puberty, childbirth, the climacteric period. It is imperative to find out the patient's heredity, severe diseases previously suffered by the patient, for example, tuberculosis, which can contribute to the development of adrenal gland insufficiency, syphilis, which can play a role in the development of insufficiency of the function of the thyroid gland and pituitary gland.

Review. An examination of a patient with endocrine gland pathology is a very valuable diagnostic method. Often, the diagnosis can be suspected already at a simple examination of the patient, based on his general appearance. This applies to such diseases as diffuse toxic goiter (hyperfunction of the thyroid gland), myxedema (hypofunction of the thyroid gland), acromegaly

and gigantism (hyperfunction of the pituitary gland), dwarfism (dwarf growth with hypofunction of the pituitary gland), pituitary cachexia and some other diseases of the endocrine system.
Patient's facial expression. With hyperfunction of the thyroid gland, attention is drawn to wide-open, staring, rarely blinking shiny eyes, which give the person an expression of frozen fear.
With hypofunction of the thyroid gland (myxedema), the face is wide, round, without wrinkles, with frozen eyes, it looks dull and indifferent.
With hyperfunction of the pituitary gland (acromegaly), disproportionately developed eyebrow arches, nose, lips, chin and tongue attract attention.
Hypofunction of the pituitary gland, accompanied by obesity, gives a man's face a feminine appearance.
Growth. Giant, more than 195 cm, the height of the patient is often the result of hyperfunction of the anterior lobe of the pituitary gland (acromegalic gigantism) or the result of hypofunction of the gonads (eunuchoid gigantism).
Dwarf height (less than 135 cm) is usually the result of hypofunction of the anterior lobe of the pituitary gland with the preservation of childlike proportions of the body, underdevelopment of the genitals, and the absence of secondary sexual characteristics. Similar symptoms can be the result of hypofunction of the thyroid gland with the addition of myxedema and sharp mental underdevelopment up to complete idiocy.
Skin. Paleness of the face with a yellowish tint of the skin is characteristic of myxedema (hypofunction of the thyroid gland).
In patients with hyperfunction of the anterior lobe of the pituitary gland (Itsenko-Cushing's disease), the face is hyperemic.
In patients with insufficient function of the adrenal cortex (Addison's disease - Birmer's disease, bronze disease), the skin and mucous membranes acquire a bronze color. This coloring is especially noticeable in the area of skin folds.
Dry, scaly skin is noted in patients with hypofunction of the thyroid and parathyroid glands. At the same time, in patients with hypothyroidism, the skin becomes cold to the touch and swollen due to impregnation with mucin. Increased brittleness of nails is also found in these patients.
In patients with hyperthyroidism, the skin is hot and moist.
In patients with acromegaly, the skin is thickened due to hypertrophy of its papillary layer.
Abdominal skin atrophy is found in patients with Itsenko-Cushing syndrome. At the same time, stretch marks appear on the skin in the form of reddish-purple stripes.
In patients with diabetes, it is often possible to see scratching and furunculosis on the skin, cholesterol deposits on the skin of the eyelids (xanthoma spots).
Hair cover. The female type of hair loss in men is characteristic of eunuchoidism, or hypofunction of the gonads. Enhanced growth of male-type hair, which is found even in women, is characteristic of acromegaly and Itsenko-Cushing syndrome. Loss of hair on the head, eyebrows, eyelashes, mustache and beard is characteristic of myxedema.
Subcutaneous fat layer. Even excessive distribution of subcutaneous fat is characteristic of hypofunction of the thyroid gland. Obesity is predominant in the area of the lower abdomen, pelvis, thighs, characteristic of pituitary or sexual obesity. Excessive obesity of the face and trunk is one of the symptoms of Itsenko-Cushing syndrome. Severe weight loss can be a symptom of diabetes, thyrotoxic goiter, pituitary cachexia.

Bone system. In patients with eunuchoidism, delayed ossification of the epiphyses of long tubular bones is noted, and in acromegaly there is a sharp thickening of the bones and their disproportionate development.
Muscular system. with hypofunction of the parathyroid glands, hypocalcemia develops and a tendency to tonic convulsions, involving mainly muscle groups - flexors. The palm of the hand acquires a characteristic shape, which is called the "midwife's hand". With spasms of the facial muscles in these patients, the impression of a forced smile is created.
In patients with acromegaly, strong muscle development is noted.
Palpation. Of all the glands of internal secretion, only the thyroid gland and testicles are palpable. When palpating the thyroid gland, it is easy to assess its size, density, surface character, and the presence of nodes. Palpation of the thyroid gland is carried out with two hands. At the same time, four fingers of each hand are placed behind the back edges of the sternoclavicular-mastoid muscle, and the thumbs are placed on the front edges of these muscles, near the jugular notch. Then the patient is asked to take a sip. At the same time, the thyroid gland, in particular its lobes, moves together with the larynx between the doctor's thumbs, which palpate it. The isthmus of the thyroid gland is examined with sliding movements of the fingers from top to bottom, in the direction of the jugular notch.
Percussion. Percussion is used to determine the retrosternal goiter.
Auscultation. In patients with thyrotoxicosis (hyperfunction of the thyroid gland), auscultation allows you to listen to tones and noises caused by increased blood flow in the area of the gland.
BAZEDOVA'S DISEASE (MORBUS BASEDOWI), or diffuse toxic goiter.
In 1840, the German doctor Basedovy described the main triad of symptoms of this disease: goiter, strangulation, and tachycardia. At the heart of the disease are disorders of the functions of three systems: the thyroid gland itself, the sympathetic nervous system, and the pituitary gland (its thyroid-stimulating hormone). Undoubtedly, the vast majority of cases of base disease develop after previous acute mental traumas, as well as severe infections. Young and middle-aged people (from 16 to 40 years old) get sick most often, and women get sick 5-6 times more often than men.
In addition to the underlying disease with a complex neuro-endocrine substrate, there are diseases of the thyroid gland with the same symptoms, but with a different pathogenesis. They are associated either with an acute infection (infectious thyroiditis), or with the development of a malignant tumor in the thyroid gland (most often an adenoma), or with the phenomena of autoimmune thyroiditis.
Clinic base disease is a complex picture of symptoms. On the one hand, there are symptoms associated with the increased function of the thyroid gland, and on the other - with increased activity of the sympathetic nervous system and a number of disorders from the side of higher nervous activity.
The main and leading symptom of basal disease associated with hyperfunction of the thyroid gland is an increase in basic metabolism. Even at rest, seriously ill patients absorb 75-80% more oxygen than healthy people. In order to satisfy this huge need of tissues for oxygen, external respiration increases. Cardiac activity increases (tachycardia). The mass of circulating blood increases. The combustion process in the body is so great that patients lose weight. In connection with the increase in the basic exchange, they are continuously feverish. Their skin is hot to the touch, increased sweating is noted. Patients constantly experience a feeling of heat, cannot dress according to the season.

An increase in the size of the thyroid gland (goiter) is diverse and not always proportional to the disorder of its functions. As a result of a disorder of the functions of the sympathetic nervous system, patients have constant tachycardia (not only as an adaptive reaction).
There are a number of eye symptoms characteristic of Bazedov's disease:
1) exophthalmos, or exophthalmos;
2) lag of the upper eyelid from the pupil when the eyeball is lowered down. This light strip between the upper eyelid and the pupil often remains even with a fixed eyeball (Grafe's symptom);
3) lag of the lower eyelid from the lower edge of the iris during horizontal gaze fixation (Dalrymple's symptom);
4) tremor of the eyelids (Rosenbach's symptom);
5) a convergence disorder develops, why when trying to fix vision on one point close to the face - the tip of the nose, one of the eyes at a certain distance from the fixed point moves to the side (Möbius symptom);
6) the need to moisten the eyeball by blinking disappears or is sharply reduced. The patient's gaze becomes strained and as if motionless (Shtelvag's symptom).
All these disorders of the functions of the oculomotor muscles create a special expression on the face of a patient with Based's disease - a "mask of frozen horror." From the side of higher nervous activity, painful manifestations are also observed: instability of mood, tendency to affect, to acute psychosis, general excitement, peculiar fussiness, small tremor of outstretched fingers (Mara's symptom).
Thyrotoxicosis causes myocardial dystrophy (brown atrophy, fatty degeneration), often with the development of atrial fibrillation and heart failure. The pulse takes the character of celer et altus with a high pulse pressure (for example, 140/60 mm Hg), which depends on the increased systolic volume. The liver is often affected (fatty dystrophy). There is a tendency to diarrhea (gastric achilles, functional bowel disorders), sometimes the pancreas is also affected.
With a pronounced picture of the disease, hypofunction of the gonads develops, up to the development of amenorrhea in women, suppression of the function of the adrenal cortex (hypocorticism). Diabetes may join.
Blood tests reveal hypochromic anemia, leukopenia, lymphocytosis, hypercholesterolemia, and hyperglycemia.
Clinically, Bazedov's disease can be of varying degrees of severity. Light erased forms are often observed.
Diagnosis is based on a complex of clinical symptoms, on laboratory research of the main metabolism. On the study of absorption of radioactive iodine by the thyroid gland.

MYXOEDEMA

Myxedema is a disease associated with insufficient function of the thyroid gland. It suddenly appears as an independent disease. It is more common after goitre removal surgery. Another thing is erased forms of myxedema, which are often found, especially in women in the climacteric period.
The clinic of myxedema is directly opposite to the clinic of basal disease. In connection with a decrease in the function of the thyroid gland, the basic metabolism is significantly reduced (by 20-40% below the norm); therefore, patients suffer from hypothermia (even acute infectious diseases occur in them with hypothermia). The tissue's need for oxygen in these patients is

reduced. The number of breaths is 8-12 per minute, the pulse is about 50 beats per minute. Heart sounds are muffled, the electrocardiogram shows a decrease in voltage.
The characteristic appearance of the patients: the skin is dry, pale, the face is mask-like, puffy. A kind of swelling develops, after which the disease got its name - myxedema, or mucous swelling. Due to swelling, the skin cannot be folded. When pressed on the skin in the area of edema, a characteristic pit is not formed. A number of dystrophic processes develop in the epithelial formations: the hair is dull and falls out. Characteristic baldness of the outer parts of the eyebrows. Nails crumble, break. Mucous membranes thicken, deafness develops, voice becomes raspy.
Patients complain of rapidly developing fatigue, lower back and back pain. They are lethargic, sleepy, forgetful, their speech is slowed down.
Complaints about various false sensations in the skin ("creeping ants" and so on), tinnitus, headaches, neurology and so on are noted. In connection with the reduction of oxidative processes, patients are prone to weight gain and increased cholesterol levels in the blood. The latter circumstance contributes to the early and significant development of vascular atheromatosis.
The diagnosis is confirmed by determining the content of iodine in the blood, which is significantly reduced in myxedema, as well as by decreasing the absorption of radioactive iodine by the thyroid gland.

DIABETES MELLITUS

The name of the disease comes from the Greek word diabaion (pass through) and the Latin mel (honey, sweet). Diabetes is a deep and complex metabolic disorder. Carbohydrate, protein, fat, water and other types of metabolism in the body are disturbed.
It has been established that the basis of the development of diabetes lies in neuroendocrine disorders: a decrease in the function of the islet apparatus of the pancreas to secrete the hormone insulin into the blood; disorders of central nervous regulation (after mental trauma).
These disorders are combined with a number of other metabolic disorders (obesity, gout), movement and nutrition disorders, and tumors of the brain, pituitary gland, or adrenal glands.
A disorder of carbohydrate metabolism is manifested in the fact that the synthesis of glycogen in the liver and muscles decreases, and the burning of glucose in tissues decreases. At the same time, the formation of glucose in the liver is enhanced. As a result of these disorders, the content of glucose in the blood increases. Glucose appears in the urine (glucosuria).
It should be emphasized that the appearance of glucose in the urine can sometimes occur even with normal carbohydrate metabolism - temporary glucosuria. At the same time, the glucose content in the fasting blood is normal. An example of such a condition can be renal glucosuria, associated with a decrease in the reabsorption of sugar by the renal tubules (during pregnancy, nephrosis).
As a result of hyperglycemia, the tissues become dehydrated, the patients feel thirsty (polydipsia), they drink a lot (sometimes up to 10 liters per day), but water is not retained in the tissues and the patients emit a lot of urine (polyuria). Urine has a high specific gravity (1030-1050), contains a lot of glucose, both in percentage terms and in absolute numbers, the total amount of glucose released per day sometimes reaches several hundred grams. Since there is very little burning of it in the body, there is an increased burning of proteins and fats, patients lose weight sharply, their muscle strength decreases, they experience a feeling of increased appetite (polyphagia), a feeling of "wolf hunger" (bulimia).

A disorder of fat metabolism leads to flooding of the blood with fats. The content of cholesterol in the blood increases. The complete burning of fatty acids due to the lack of burning of carbohydrates stops at the stage of ketone bodies (acetone, acetoacetic acid, β-oxybutyric acid).
From the very beginning of the disease, patients complain of muscle weakness, loss of working capacity, increased appetite, thirst. Some patients develop itching of the skin, especially in women, in the genital area. Sexual weakness appears in men. Patients lose weight quickly due to loss of fat and protein and severe dehydration of the body.
To diagnose diabetic metabolic disorders, in addition to determining fasting blood glucose, the so-called glycemic curve is determined, that is, after giving the patient 100 g of glucose solution, its blood level is determined every half hour. With normal metabolism, the carbohydrate load causes an appropriate reaction of the islet apparatus of the pancreas, and short-term moderate hyperglycemia by the end of the second hour is replaced by a decrease in the sugar level even below the initial level (α). In hidden forms of diabetes, hyperglycemia is higher and its level in the blood decreases more slowly, not reaching the initial numbers by the end of the second hour. In severe forms of the disease, the glycemic curve does not return to normal even after 3-4 hours (b).
Clinical forms of diabetes are divided into two groups: "skinny" diabetes. Which is more severe and occurs mainly in young people, and "fatty" diabetes, which is easier, with shallow disorders of carbohydrate metabolism; occurs in obese people, often in combination with other metabolic disorders.
The course of diabetes can be mild or severe, which determines medical measures regarding the regulation of the patient's nutritional balance: only diet or the need to use hormone (insulin) replacement therapy.
The most dangerous complication of diabetes is diabetic coma (coma diabeticum). This condition is caused by a sharp decrease in the burning of carbohydrates and a significant accumulation of ketone bodies in the blood. pH The reaction of the blood shifts to the acidic side and blood acidosis develops - acid poisoning of the body, mainly of the nervous system. The patient loses consciousness, he has deep, large Kussmaul breathing.
When examining the patient, the tongue is dry. The eyeballs are soft (vitreous dehydration), the pupils are narrow. Blood pressure falls (collapse). The pulse is small and frequent. Hypothermia, oliguria up to anuria are observed.
There is sharp hyperglycemia in the urine. Leukocytosis can reach 50 x 109/l with a neutrophil shift to the left. Exhaled air smells of acetone ("apples"). Glucosuria and ketone bodies appear in the urine.
An absolutely opposite picture develops in patients with hypoglycemia, for example, when a diabetic patient overdoses on a therapeutic dose of insulin.
Hypoglycemic coma. It develops quickly, sometimes within a few minutes. The onset of coma is preceded by a sudden feeling of hunger, sharp weakness, trembling of the whole body, mental and motor excitement, profuse sweating.
Patients in a coma are pale. Their skin is wet. Increased tendon reflexes and muscle tone. Convulsions are possible. The eyeballs are hard to the touch, the pupils are dilated. Blood pressure is elevated, pulse is arrhythmic.
The level of glucose in the blood is sharply reduced.

General material and mass-methodological support

lectures:

work program of the academic discipline

synopsis (plan-summary) of the lecture
multimedia presentation of the lecture

Questions for self-control:

1. Diabetes mellitus: etiology, symptomatology,
2. Classification of diabetes mellitus, data of additional studies.
3. Complications of diabetes and principles of treatment.
4. Types of coma in diabetes.
5. Diagnosis of hyper- and hypoglycemic coma. Treatment.
6. Hyperthyroidism: etiology, clinical picture, diagnosis, complications, principles of treatment.
7. Hypothyroidism: etiology, clinical picture, diagnosis, complications, principles of treatment.

Lecture no8 «**Clinical picture of pneumonia, bronchial asthma, pleurisy. Auxiliary research methods.**»

Actuality of theme.

The incidence of acute pneumonia has remained stable for 30 years and is 14 per 1,000 population in European countries, among non-specific lung diseases it accounts for up to 40% of cases. The disease is characterized by a pronounced pathomorphosis - the etiology and symptoms of acute pneumonia have changed, views on some key issues of diagnosis and treatment of the disease have changed. Among patients with non-acute pneumonia, men predominate - 55%. The incidence of acute pneumonia increases with age, 55% of patients belong to the age group of 40-59 years, 34% of patients are over 60 years old.

Pleurisy always occurs as a secondary process, syndrome or complication of a number of diseases of internal organs (pneumonia, tuberculosis, rheumatism, diffuse connective tissue diseases, hypothyroidism, hemoblastosis, tumors). Pleural effusion is one of the common pathological syndromes that occurs in 5-10% of patients with a therapeutic profile.

Goal:

1. Master the basics of identifying characteristic symptoms and syndromes in patients with pneumonia and pleurisy.
2. Get acquainted with modern research methods, as well as with changes in indicators of laboratory and instrumental research methods for these diseases.
3. Get acquainted with the general principles of treatment of this category of patients.

Basic concepts:

organs of the respiratory system, examination of the patient, palpation, percussion, auscultation, inflammation.

Plan and organizational structure of the lecture.

1. Determination of the educational goal.
2. Providing positive motivation.
3. Presentation of the lecture material according to the plan:
4. Summary of the lecture. General conclusions.
5. The lecturer's answer to possible questions.
6. Tasks for self-training.

Content of the lecture material

Pneumonia -polyetiological focal infectious-inflammatory lung disease with the obligatory presence of intra-alveolar inflammatory exudation.
Distinguish**:**

1) non-hospital, widespread, ambulatory, home pneumonia

(NP).

2) intra-hospital, hospital, nosocomial pneumonia (VP);

3) aspiration pneumonia (AP);

4) pneumonia in persons with severe immune defects (IDP).

The occurrence of each of these types of pneumonia is associated with a rather limited list of microorganisms, which allows for successful empiric therapy.
In a group**non-hospital** pneumoniae are typical pathogens and "atypical". The former most often belong to the first*Strertososusrneumoniae*(15-34%),*Naemorhilus influenzae*(5-13%). They are atypical *Garbage lasma*

pneumoniae, Legionella spp., Chlamidophila pneumoniae and Chlamidia psitatti(5-20%).
Special attention is paid to pneumonia that occurred in the elderly, as the disease progresses very rapidly in them.

Depending on the degree of severity, pneumonia with a mild and severe course is distinguished.

Pneumonia with a severe courseis a special form of a different disease
etiology, which is manifested by a pronounced intoxication syndrome, hemodynamic changes, severe respiratory failure and/or signs of severe sepsis or septic shock, is characterized by an unfavorable prognosis and requires intensive therapy.

It is recommended to distinguish "small" and "large" criteria for the severe course of pneumonia.

«*Small" criteria for a severe course of pneumonia:*

- respiratory rate 30 per minute;

- disturbance of consciousness;

- $Star_2$<90% (according to pulse oximetry), PaO_2<60 mmHg Art.;
- systolic blood pressure (SBP) <90 mm Hg. art.;

- bilateral or multipart lung damage, presence of decay cavities, pleural effusion.

"Big" criteria for a severe course of pneumonia:

- the need for artificial lung ventilation;

- rapid progression of focal infiltrative changes in the lungs - an increase in the size of infiltration by more than 50% within 2 days;

- septic shock or the need to administer vasopressor drugs;

- acute renal failure (the amount of urine < 80 ml in 4 h or the level of creatinine in the blood serum > 0.18 mmol/l, or the concentration of urea nitrogen > 7 mmol/l (urea nitrogen = urea (mmol/l/2.14) by absence of chronic kidney failure).

No-hospital pneumonia.

NP should be understood as an acute disease that occurred in an outpatient setting and is accompanied by symptoms of a lower respiratory tract infection (fever; cough; sputum discharge, possibly purulent in nature; chest pain and shortness of breath) and radiological signs of focal infiltrative changes in the lungs in the absence of obvious diagnostic alternatives
Allocatefour ways of infection, which with varying frequency lead to the development of pneumonia:

aspiration of oropharyngeal contents; inhalation of an aerosol containing microorganisms;

hematogenous spread of microorganisms from an extrapulmonary focus of infection (endocarditis with damage to the tricuspid valve, septic thrombophlebitis of the pelvis);

direct spread of infection from affected tissues of neighboring organs (for example, liver abscess) or as a result of infection during permeable
chest injuries.

Aspiration of the contents of the oropharynx is the main way of infection of the respiratory tract of the lungs with NP. Under normal conditions, a number of microorganisms, such as S. pneumoniae, can colonize the oropharynx, but the lower respiratory tract remains sterile.

Microaspiration of oropharyngeal contents is a physiological phenomenon that occurs in 40-70% of healthy individuals during sleep. However, the cough reflex, regulated mechanism of mucociliary clearance, antibacterial activity of alveolar macrophages and secretory immunoglobulins ensure the elimination of infected secretions from the lower respiratory tract and their sterility.

In case of violation of these mechanisms of "self-cleaning" of the tracheobronchial tree, for example, with a respiratory viral infection, in which the function of the cilia of the bronchial epithelium is disturbed and the phagocytic activity of alveolar macrophages decreases, favorable

conditions are created for the development of pneumonia.

In some cases, an independent pathogenetic factor can be the massiveness of the infectious dose of microorganisms or the penetration of even single highly virulent microorganisms resistant to the action of the protective mechanisms of the macroorganism into the respiratory tract of the lungs.

Inhalation of an aerosol containing microorganisms is a less common mechanism for the development of NP, which plays a major role during infection with obligate microorganisms, such as Legionella spp.
Hematogenous (for example, Staphylococcus spp.) and direct spread of the pathogen from the focus of infection are even less important (in terms of frequency of detection).

Taking into account the above features of the pathogenesis of NP, it is obvious that its etiology is related to the microflora of the upper respiratory tract. Among the numerous types of microorganisms that colonize the upper respiratory tract, only some (with increased virulence) are able to cause the development of an inflammatory reaction even with minimal violations of protective mechanisms in case of penetration into the respiratory department of the lungs.
The dependence of the species composition of the microflora of the upper respiratory tract on the nature of the environment in which the individual is, his age and general state of health makes it possible to predict the etiology of NP. It should be emphasized that mixed infection is often noted in adult patients with NP. For example, almost 50% of patients with a pneumococcal etiology of the disease simultaneously show serological signs of an active mycoplasma or chlamydial infection.
Groups of patients with community-acquired pneumonia**.**

For the purpose of standard empiric antibacterial treatment of community-acquired pneumonia, 4 groups of patients are distinguished, taking into account the age of the patient, the severity of the course of pneumonia, and the presence of concomitant pathology.

I group. Group I includes patients with NP with a mild course who do not require hospitalization, without concomitant pathology and other modifying factors.
II group. Group II includes patients with PN with a mild course who do not require hospitalization, with concomitant pathology (chronic obstructive pulmonary disease, kidney and heart failure, cerebrovascular disease, tumor, diabetes, chronic liver disease of various etiologies, mental disorder, alcoholism) and/or other modifying factors.

III group. Patients with NP with a mild course who require hospitalization in a therapeutic department for medical reasons (presence of adverse prognostic factors) belong to the III group.
IV group. Patients with NP with a severe course who require hospitalization in the intensive care unit belong to the IV group.
Community-acquired pneumonia is mostly treated at home, primarily due to economic reasons. When deciding on hospitalization, you should be guided by the following criteria:

1. Age over 65 years.

2. The presence of concomitant diseases (chronic obstructive pulmonary diseases, diabetes, chronic renal failure, chronic heart failure, chronic liver diseases, previous hospitalizations in the current year for pneumonia, suspected aspiration, alcoholism. Condition after splenectomy, impaired mental status).

3. Physical data requiring hospitalization (BP > 30/min., systolic pressure < 90 mm Hg, body temperature > 38.8°C, leukocytes < $4x10^9$/l or > $30x10^9$/l, hematocrit < 30% or hemoglobin < 90 g/l, decrease in the number of platelets).

4. Changes in other bodies and systems. Septic arthritis, meningitis, signs of sepsis, metabolic acidosis.

The diagnosis of NP is based on the detection of general (general weakness, adynamia, decreased appetite, fever) and local respiratory (cough, sputum discharge, shortness of breath, chest pain) symptoms, as well as physical data (dull or dull percussion sound, weakened or stiff bronchial breathing , a focus of sonorous fine-vesicular rales and/or crepitations).
The expression of these signs depends on the patient's condition at the beginning
the disease, the degree of severity of the course of the disease, the volume and localization of the lesion of the lung parenchyma, age, the presence of concomitant diseases. This symptom complex is not specific for NP, but it is sufficient to establish a preliminary clinical diagnosis.

However, in about 20% of patients, the objective signs of NP may differ from typical ones or be absent. In persons of older age groups and/or with an inadequate immune response in the clinical picture of the disease, the main symptoms may be confusion, exacerbation/decompensation of concomitant diseases, absence of fever.

The most important diagnostic study in patients with NP is radiography of the chest cavity, which must be performed in two projections (posterior-anterior and lateral) in order to increase the informativeness of this examination method. The diagnosis of pneumonia almost always involves the detection of focal infiltrative changes in the lungs in combination with the corresponding clinical manifestations of lower respiratory tract infection.

X-ray picture in pneumonia.

Croupous pneumonia (pleuropneumonia)

In the inflow stage, an increase in the pulmonary pattern in the damaged part due to hyperemia is revealed. The transparency of the lung field remains normal or slightly reduced. The root of the lung on the damaged side is barely expanded, its structure is unclear.

The transition of the influx stage to the stage of red hepatization is characterized by increased exudation in the alveoli, compaction of the lung lobe, and an increase in its volume, which is radiologically manifested by a homogeneous shadow of medium intensity with a clear convex border.

After 1-3 days, the stage of red hepatification passes into the stage of gray hepatification. In this case, the X-ray picture remains the same as in the previous stage, but gentle foci of darkening appear, mainly in the basal zone, which quickly increase in size, merge with each other and create a picture of intense darkening of the damaged part. At the same time, one of the contours of the shadow takes the form of a clear line located in one of the interparticle slits.

The resolution stage takes place unevenly over the entire damaged part, usually lasting 7-10 days. In this stage, the intensity and size of the homogeneous darkening gradually decrease, and a slow restoration of the transparency of the lung tissue begins. The shadow of the root of the lung remains expanded and structureless for a long time. The pulmonary pattern at the site of the former liver infection remains enhanced for 2-3 weeks after clinical recovery, and the pleura remains unchanged even longer.
Focal pneumonia (bronchopneumonia).

Focal pneumonia usually begins with damage to the wall of the bronchus, which is why it is called bronchopneumonia.

The X-ray picture of focal pneumonia is a reflection of morphological changes in the damaged part of the lung and can be diverse.

Focal shadows can be small (acinus pneumonia) and larger (lobular pneumonia) with unclear contours of an irregular shape, located along the course of the bronchi or in the form of separate clusters in different parts of the lungs.

The transparency of the damaged area is reduced. The predominant localization of bronchopneumonic shadows is the posterior lower segments, but there may also be segments of the upper lobes of the lungs.
Carrying out additional X-ray examinations (X-ray tomography, computed tomography - CT) is appropriate for differential diagnosis in cases of lesions of the upper lobes of the lungs, lymph nodes, mediastinal organs, in case of a decrease in the volume of a lung lobe, assumption of abscessation, as well as in case of ineffectiveness of previous antibacterial therapy.

*Microbiological research*with NP aimed at identifying the causative agent of the disease in the material obtained from the source of infection. The material for research must be taken before the start of antibacterial therapy. However, despite the difficulties of conducting a microbiological study in full, you should not delay the appointment of an antibiotic.

Standard research methods are bacterioscopy of Gram-stained smears of sputum and microbiological examination of sputum obtained during deep expectoration. Conducting these studies is mandatory during the treatment of patients with a severe course of NP and optional in the case of a mild course of the disease. The material obtained during bronchoalveolar lavage (BAL) and bronchoscopy has a high diagnostic value only if "protected" brushes are used.

The material of transtracheal aspirate, smears from intubation tubes, pharynx and tracheostomy

have a low diagnostic value.

The following rules should be followed when collecting and examining sputum:

- sputum must be collected before starting antibacterial therapy, preferably in the morning before meals, after thoroughly rinsing the oral cavity with boiled water;
- the patient should be instructed on the need to obtain for examination the contents of the lower respiratory tract, but not the oropharynx or nasopharynx;
- sputum must be collected in sterile containers, the storage period of which should not exceed 1-2 hours at room temperature.

Before starting a microbiological examination of sputum, it is necessary to carry out bacterioscopy of smears stained according to Gram. If present in smears

< 25 leukocytes and > 10 epithelial cells in the field of view (during the study, at least 8-10 fields of view at low magnification), further research is not appropriate, because in this case it can be asserted with high probability that the material under study is the contents of the oral cavity.
Detection of a significant number of gram-negative or gram-positive microorganisms with a typical morphology in smears (gram-positive lanceolate diplococci - S. pneumoniae, clusters of gram-positive cocci in the form of clusters - S. aureus, gram-negative coccobacilli - H. influenzae) can be a guide in the selection of drugs for the appointment of empiric antibiotic therapy.

In the case of a severe course of the disease and the impossibility of obtaining sputum samples suitable for research, in case of suspicion of pulmonary tuberculosis and the absence of a productive cough, in the presence of obstructive pneumonia on the background of bronchogenic carcinoma, in case of aspiration of a foreign body into the bronchi, etc., invasive diagnostic methods should be used. The use of invasive research methods is appropriate in patients who are on artificial lung ventilation.

It should be noted that, according to the majority of experts, fibrobronchoscopy is not a routine diagnostic study in patients with NP and the need for its performance is determined by clinical expediency - to rule out local bronchial obstruction and obtain material from the lower respiratory tract.

Clinical blood analysis data do not allow to determine the potential causative agent of pneumonia. Biochemical blood tests (functional liver and kidney tests, glycemia, etc.) do not provide specific information, however, in the presence of deviations from normal indicators, they indicate damage to certain organs/systems, which has a certain clinical and prognostic significance.

In patients with signs of respiratory failure caused by widespread pneumonic infiltration, massive pleural effusion, the development of pneumonia against the background of chronic obstructive pulmonary disease, it is necessary to determine blood oxygen saturation or arterial blood gases.
Criteria for the diagnosis of nosocomial pneumonia.

The diagnosis of NP is determined if the patient has X-ray confirmed focal infiltration of lung tissue and 2 clinical signs:

- acute onset of the disease with an increase in body temperature > 38°C;
- cough with phlegm;

- physical signs (dull or dull percussion sound, weakened
or hard bronchial breathing, a focus of sonorous fine-vesicular rales and/or crepitations);
- leukocytosis (>10*10^9/l) and/or rod nuclear shift (>10%).

In the absence or impossibility of obtaining radiological confirmation of the presence of focal infiltrations in the lungs, the diagnosis of NP is imprecise/indeterminate. At the same time, the diagnosis of the disease is established taking into account the data of the epidemiological history, the patient's complaints and the corresponding physical signs found in the patient. It should be noted that in such a situation, the diagnosis of NP is confirmed radiologically in only 22% of cases.

The assumption of the presence of NP is unlikely in patients with fever, complaints of cough, shortness of breath, sputum discharge and/or chest pain in the absence of physical signs and the impossibility of conducting an X-ray examination of the chest cavity.

Treatment of nosocomial pneumonia.

Antibacterial therapy of patients with NP in outpatient settings

Group patients	**Possible exciter**	**antibiotic**		**Antibiotic**
		eparation of choice	**Alternative th drug**	**second row**
rse of ut ying and ns	niae, oniae, oniae tae	n de	olone	de or ne for y cillin enici- olone erations tive- load
rse of y ying	niae, tae, oniae, oniae alis,	n/ a e	olone or e	or py

s ers ns	*teriac*		n)*	olone erations

Note: * -parenteral administration of ceftriaxone is prescribed when oral administration of drugs of choice is impossible.

Antibacterial therapy of patients with NP in hospital conditions

Group	**Possible**	**antibiotic**		**ntibiotic**
		Preparation	**Alternative**	
patients	**exciter**	**of choice**	**preparation**	**cond row**
ed st lepartment se of	*niae,* *zae,* ,	n n): cillin, + (*per us*) - rations le	n: olone erations	n: olone ms m
ed IT with se of	*niae,* *zae,* o- tive e- - ., bial ns	n: cillin or orin ion +	n: olone III– ion + β-	n: m + olone ms m +
		suspicion of*P.* *a* sly on: orin erations	plication orin erations,	n: m + oside

		; in p ent oside + cin cin)	p ent oside +	cin-

The effectiveness of empiric therapy is evaluated 72 hours after the start of treatment. The main criteria are: tendency to normalization of body temperature, reduction of signs of intoxication syndrome.
In the absence of positive changes, antibiotics are prescribed according to the results of a microbiological study or drugs of an alternative (different) series are recommended: cephalosporins of the IV generation, monobactams, carbopenems or fluoroquinolones.

Nosocomial pneumonia.

Nosocomial pneumonia- an acute infectious disease of the lower respiratory tract, confirmed x-ray, which occurred 48 hours after the patient was admitted to the hospital.

In the group of nosocomial pneumonias, there are pneumonias in people with spontaneous breathing and pneumonias in intubated patients (that is, people who developed pneumonia after anesthesia, artificial ventilation, etc.). In this category of patients, gram-negative aerobic flora are often the causative agents of the disease*Pseudomonas aeruginosa, Enterobacter spp., Klebsiellapneumoniae.*

Gram-positive bacteria also often cause these pneumonias, or are associated with gram-negative flora.

The criteria for the classification of nosocomial pneumonia are the duration of the disease, the presence or absence of risk factors for its development.

- *early GP*-occurs within the first 5 days from the moment of hospitalization and is caused by pathogens that the patient had before admission to the hospital, -
*S. pneumoniae, H. influenzae,*methicillin sensitive *S. aureus*(hereinafter - MSSA) and other representatives of the normal microflora of the oropharyngeal cavity. Most often, these pathogens are sensitive to traditionally used antimicrobial drugs, and pneumonia has a more favorable prognosis;

- late HP - develops no earlier than 6 days of hospitalization and is caused by the hospital microflora itself with a higher risk of the presence of highly virulent and polyresistant pathogens, such as*P. aeruginosa,Acinetobacter spp.,* family representatives *Enterobacteriaceae*,
methicillin resistant*S. aureus*(hereinafter referred to as MRSA). Such GP is characterized by a less favorable prognosis.
Given the severity of the course of the disease, the seriousness of the prognosis and the

peculiarities of the management of intensive care patients, they are allocated to a special form, the so-called*ventilator-associated pneumonia (VAP)*- pneumonia that arose due to

48 hours from the start of mechanical ventilation in the absence of signs of pulmonary infection to the moment of intubation.

Diagnosis of hospital pneumonia.

Despite the known limitations, clinical examination remains the "starting point" for the diagnosis of HP, and the data of other methods (including invasive ones) are only interpreted taking into account the clinical picture of HP.

HP is characterized by the appearance of new infiltrative changes on a chest x-ray in combination with such signs of an infectious disease as fever, purulent sputum and/or leukocytosis.

B connection with this*among the number of formalized diagnostic criteria of GP include*:

- the appearance of new focal and infiltrative changes on the X-ray

lungs;

- or two of the following signs:

1) body temperature above > 38.3°C;

2) bronchial hypersecretion;

3) $PaO_2/Wire_2$ ($Wire_2$ - fraction of oxygen in exhaled air, %)

less than 240;

4) cough, tachypnea, local crepitation, wet wheezing, bronchial

breath;

5) the number of leukocytes in the blood is less than $4.0*10^9$/l or more $12.0*10^9$/l, rod nuclear shift more than 10%;
6) purulent sputum / bronchial secretion (more than 25 leukocytes in the field of vision under microscopy with low magnification * 100).

Treatment of nosocomial pneumonias.

The diagnosis of HP is an absolute indication for the use of antibiotics, which are the basis of treatment in such patients. Antibacterial treatment should be started immediately after diagnosis. It is absolutely unacceptable to delay the urgent appointment of antibiotics to patients with a

severe course of the disease due to the lack of results of bacterioscopy and sputum culture, since delaying the administration of the first dose of the antibiotic for 4 hours. causes a significant increase in the risk of death of such patients.

The most important factor in increasing the survival of patients with HP is the timely appointment of adequate antibiotic therapy.

The most justified approach to empiric antibiotic therapy of patients with HP is treatment depending on the timing of the occurrence of pneumonia ("early", "late") and the presence of modifying factors of the risk of infection with polyresistant strains of microorganisms.

In patients with "*early" GP with risk factors* in the presence of polyresistant strains of pathogens and "late" HP, the most likely causative agents of the disease may be gram-negative bacteria (*P. aeruginosa, K. pneumoniae*(producers ESBL)*Acinetobacter spp. L. pneumophila*) or gram-positive cocci resistant to methicillin -*S. aureus*(MRSA). For the treatment of these patients, the following are used: a cephalosporin with anti-blue-pus activity (cefepime, ceftazidime) or a carbapenem (imipenem, meropenem), or a protected β-lactam (piperacillin/tazobactam) in combination with a fluoroquinolone with anti-blue-pus activity or an aminoglycoside (amikacin, gentamicin, tobramycin), and also with linezolid or vancomycin (in the presence of MRSA risk factors or a high frequency of nosocomial infections in this hospital).

The traditional duration of antibiotic therapy for patients with HP is, as a rule, 14-21 days. An increase in its duration can lead to superinfection with polyresistant nosocomial pathogens, in particular*P. aeruginosa* and microorganisms of the family*Enterobacteriaceae*. With VAP, a significant clinical improvement is observed already within the first 6 days of therapy, and an increase in its duration to 14 days leads to colonization by P. aeruginosa and microorganisms of the family*Enterobacteriaceae*.

General material and mass-methodological support

lectures:

work program of the academic discipline
synopsis (plan-summary) of the lecture
multimedia presentation of the lecture

Questions for self-control:

1. Physical and microscopic properties of sputum according to general sputum analysis and their diagnostic significance.
2. Pulmonary tissue compaction syndrome. Diagnosis of the syndrome based on the results of the survey and physical examination (palpation, percussion, auscultation). Causes of occurrence. Additional research methods.
3. Syndrome of bronchial obstruction of the lungs. Diagnosis of the syndrome based on the results of the survey and physical examination (palpation, percussion, auscultation). Causes of occurrence. Additional research methods.
4. Syndrome of fluid accumulation in the pleural cavity. Diagnosis of the syndrome based on the results of the survey and physical examination (palpation, percussion, auscultation). Causes of

occurrence. Additional research methods.
5. Croupous pneumonia: etiology, classification, results of clinical examination, main syndromes, complications and principles of treatment.
6. Bronchopneumonia: etiology, classification, results of clinical examination, main syndromes, complications and principles of treatment.
7. Acute and chronic bronchitis (COPD): etiology, classification, clinical data, main syndromes, complications, principles of treatment.
8. Pleurisy: etiology, results of clinical examination, main syndromes, complications, principles of treatment.

Lecture No. 9 "Acquired heart defects (mitral and aortic). Clinical picture and diagnosis."

Actuality of theme.
A persistent pathological deviation in the structure of the heart valves, which is accompanied by a violation of its function, is called a heart defect. Heart defects can be congenital or acquired. Congenital heart defects are rare, they account for 1-3% of all cases of organic heart lesions. Acquired heart defects are observed much more often. Mainly they develop as a result of transferred rheumatism (90% of all cases), less often etiological factors are infectious endocarditis, atherosclerosis, syphilis, injuries (rarely).
The basis of the development of acquired heart defects is either incomplete closure (insufficiency) of the valves due to their deformation, or narrowing (stenosis) of the valve opening. Quite often, a combination of both types of defects is observed on the same valve.
Mitral valve insufficiency is characterized by incomplete closure of the left atrioventricular opening by the valve leaflets during ventricular systole. Mitral insufficiency can be organic or functional.
Narrowing of the left atrioventricular opening (mitral stenosis) occurs after rheumatic endocarditis due to sclerosing and fusion of the valve leaflets with each other at their base and thickening of the fibrous ring to which they are attached.
The occurrence of organic failure is most often caused by destructive changes in the valve leaflets after rheumatic endocarditis.**Acquired heart defects**
Aortic valve insufficiency is a heart defect in which the semilunar valves do not completely close the aortic opening, and during diastole blood from the aorta partially returns to the left ventricle.
Stenosis of the mouth of the aorta (aortic stenosis) most often develops as a result of rheumatic endocarditis, less often the cause of its occurrence is septic (infectious) endocarditis, atherosclerosis, or it is congenital. Narrowing of the mouth of the aorta occurs as a result of the fusion of the valve leaflets and the onset of its sclerosing of the opening to which they are attached.
The occurrence of organic failure is most often caused by destructive changes in the valve leaflets after rheumatic endocarditis.
The disease of the valvular apparatus of the heart is of great importance, because it leads to disability and shortening of a person's life.
In recent years, the clinic has developed a large number of new instrumental methods for the study of the cardiovascular system, however, physical methods of examining the patient are still of primary importance in diagnostics. Mastering the skills of questioning, examination, palpation,

percussion and auscultation of the heart, the ability to evaluate the obtained data in order to determine the location of the valvular lesion is important for a doctor of any clinical specialty.

Purpose of the lecture:

Master the methods of interviewing and examining patients with acquired heart defects.
the acquirer must master:

1. To be able to conduct a survey and examination of patients with acquired heart defects.
2. Anatomy of the cardiovascular system.
3. Scheme of division of the heart region.
4. Methods of palpation and percussion of the heart.
5. Auscultation of the heart
6. Mechanism of formation of pathological heart murmurs.

Basic concepts: acquired heart defects, auscultation of the heart, stenosis of the valve, insufficiency of the valve, examination of the patient, auscultation of the heart, basic and additional heart sounds.

Plan and organizational structure of the lecture.

1. Determination of the educational goal.
2. Providing positive motivation.
3. Presentation of the lecture material according to the plan:
4. Summary of the lecture. General conclusions.
5. The lecturer's answer to possible questions.
6. Tasks for self-training.

Acquired heart defects (mitral and aortic). Clinical picture and diagnosis

Etiological factors and mechanisms of development of aortic insufficiency and aortic stenosis. Hemodynamic changes in aortic heart defects. The main complaints of patients with aortic stenosis and aortic valve insufficiency. Data of examination, palpation of the atrial region and percussion in aortic heart defects. Auscultatory picture of stenosis of the aortic orifice and aortic insufficiency. ECG and FCG signs of aortic heart defects. X-ray signs of aortic defects.

Definition and spread of heart defects, frequency of damage to heart valves. Rheumatism, modern classification and main clinical manifestations.

The main causes and mechanisms of the development of mitral insufficiency and mitral stenosis. Hemodynamic changes in mitral heart defects. The value of the Kitaev reflex. The main complaints of patients with mitral stenosis and mitral valve insufficiency. Data of examination, palpation of the atrial region and percussion in mitral heart defects. Auscultatory picture of mitral stenosis and mitral insufficiency. ECG and FCG signs of mitral heart defects. X-ray signs of mitral defects.

1. Mitral valve insufficiency syndrome.
1.1. Causes of mitral valve insufficiency.
1.2. Hemodynamic disorders in the presence of mitral valve insufficiency.

1.3. Complaints of patients with mitral valve insufficiency syndrome.
1.4. Physical examination data of a patient with mitral valve insufficiency syndrome.
1.4.1. Data of a general examination and examination of the area of the heart and large vessels.
1.4.2. Percussion data of the borders of the heart.
1.4.3. Heart auscultation data.
1.4.4. Data of laboratory-instrumental research methods.

2. Syndrome of stenosis of the left atrioventricular opening.
2.1. Causes of stenosis of the left atrioventricular opening.
2.2. Violations of hemodynamics in the presence of stenosis of the left atrioventricular opening.
2.3. Complaints of patients in the case of stenosis of the left atrioventricular opening.
2.4. Data of a physical examination of a patient with stenosis of the left atrioventricular opening.
2.4.1. Data of a general examination and examination of the area of the heart and large vessels.
2.4.2. Palpation data of the heart area.
2.4.3. Percussion data of the borders of the heart.
2.4.4. Heart auscultation data.
2.4.5. Data of laboratory-instrumental methods of examination.

1. Aortic valve insufficiency syndrome.
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1.4.3. Heart auscultation data.
1.4.4. Data of laboratory-instrumental research methods.

2. Aortic stenosis syndrome.
2.1. Causes of aortic stenosis.
2.2. Violations of hemodynamics in the presence of aortic stenosis.
2.3. Complaints of patients with aortic stenosis.
2.4. Data of a physical examination of a patient with aortic stenosis.
2.4.1. Data of a general examination and examination of the area of the heart and large vessels.
2.4.2. Palpation data of the heart area.
2.4.3. Percussion data of the borders of the heart.
2.4.4. Heart auscultation data.
2.4.5. Data of laboratory-instrumental methods of examination.

Samong organic diseases of the cardiovascular system (CCS), acquired defects make up 25% and rank 3rd after coronary artery disease and coronary artery disease.

Definition

Acquired heart defectsis a pathology due to morphological and (or) functional disorders of the valvular apparatus caused by acute and chronic diseases that disrupt the function of the valves and change intracardiac hemodynamics and general blood circulation.

The most common causes of acquired heart defects*is*rheumatic fever (rheumatism) and IE. Damage to the mitral and aortic valves occurs most often. Heart defects are based on dysfunction of the valvular apparatus, which can be manifested by defects in the valve or heart muscle, resulting in regurgitation - the movement of blood in the reverse direction (in case of insufficiency of the valves), or the appearance of obstacles in the form of stenosis on the way of blood movement.

There are simple, combined and combined defects.**Simple heart defect -**this is one "pure" defect;**combined-** the presence of stenosis and insufficiency; combined - when several valves are affected.

Classification of acquired heart defects

Etiology: rheumatic, non-rheumatic (with clarification).

Localization (valve):mitral, aortic, tricuspid, pulmonary.

Character of valve damage: stenosis, insufficiency, combined valve defect.

Stages:I, II, III, IV, V.

The main causes of acquired heart defects:rheumatic disease (in 2/3 cases the mitral valve is affected), IE, systemic connective tissue diseases, atherosclerosis and ischemic heart disease (IXO" syphilis trauma).

Mitral stenosis (MS)(Fig. 3.4) is a heart defect in which the left atrioventricular opening is narrowed, which obstructs

Fig. 3.4.*Mitral stenosis*

causes the movement of blood from the left atrium to the left ventricle in diastole.

Violation of hemodynamics. The area of the opening is normally 4-6 cm2. When it narrows to 1.5 cm2, there are pronounced signs of hemodynamic disorders. During diastole, blood does not have time to flow out of the left atrium into the left ventricle, which leads to overflowing of the atrium with blood, increasing its pressure. Initially, compensation occurs due to increased contractions of the atrium, hypertrophy of its muscles. But the muscles of the hypertrophied atrium are quite weak, so the contractile ability quickly decreases. Blood stagnation in the atrium increases. This leads to irritation of the baroreceptors of the left atrium and the activation of a compensatory mechanism - the Kitaev reflex (narrowing of the pulmonary arteries). There is stagnation in the small circle of blood circulation. This leads to an increase in the load on the right ventricle, which hypertrophies and expands. The pulmonary artery also expands. A significant increase in pressure in the pulmonary artery and the right ventricle prevents emptying of the right atrium, the cavity of which expands, the pressure in it increases, which leads to stagnation of blood in a large circle of blood circulation.

The left ventricle receives less blood during diastole, performs less work during systole, so its size decreases slightly, diastolic dysfunction of the left ventricle develops.

Classification

Stages of MS: I – compensation, II – pulmonary congestion, III – right ventricular failure, IV – dystrophic, V – terminal.

The main complaints are due to hemodynamic disorders: as a result of stagnation in the small circle of blood circulation, the patient is disturbed by shortness of breath and palpitations, first during physical exertion, and then at rest; periodically there are attacks of cardiac asthma, the cough is dry or with a small amount of mucous sputum, sometimes with blood impurities, general weakness and fatigue are expressed. With atrial fibrillation - a frequent sign of MS - there are interruptions in the activity of the heart. As a result of insufficient blood supply to the hypertrophied right ventricle, pain in the area of the heart (aching, squeezing) disturbs; stagnation in the large circle of blood circulation is accompanied by swelling, heaviness in the right hypochondrium.

General overview. If the heart defect occurred in childhood, the so-called "mitral dwarfism" is observed - the child's physical development lags behind. The position of the body is forced - orthopnea. A cyanotic blush on the cheeks, cyanosis of the lips and the tip of the nose attract attention -*facies mitralis,*swelling Examining the area of the heart, it is possible to detect a cardiac hump, which leads to the development of the defect in childhood, and Botkin's symptom - a decrease in the volume of the left half of the chest. Characteristic pulsation of the right ventricle in the epigastric area and in the III–IV intercostal space to the left of the sternum, as well as swelling of the neck veins and pulsation of the liver in functional insufficiency of the tricuspid valve. On palpation, diastolic tremors are felt in the area of the heart - a symptom of "cat's purring" and a symptom of two hammers (amplified first sound at the apex and emphasis of the second sound at the pulmonary artery). Percussion shows the displacement of the limits of the relative dullness of the heart up and to the right (due to the expansion of the left atrium and right ventricle).

Auscultative picturehas a high diagnostic value. Above the apex of the heart, an increased (flattering) tone is heard as a result of the accelerated contraction of the insufficiently filled left ventricle with blood and the rapid closing of the fibrous leaflets of the mitral valve. II tone is accentuated on the pulmonary artery and can be split. The accent is caused by a sharp increase in pressure in the pulmonary artery, and splitting depends on the non-simultaneous closing of the valves of the pulmonary artery and aorta. After the II tone, the click of the mitral valve opening is heard, which occurs as a result of the oscillation of the leaflets, which open sharply due to the arrival of blood under high pressure from the left atrium. These auscultatory phenomena together create a kind of melody - "quail rhythm".

A diastolic murmur is heard at the apex (Fig. 3.5), usually with a presystolic increase, which occurs as a result of the accelerated movement of blood through a narrow mitral opening. It is short, increasing in intensity (*crescendo*) and ends with a clapping I tone.

X-ray signs of MS:smoothing of the waist of the heart, deviation of the esophagus along the arc of a small radius, expansion of the arc of the pulmonary artery, stagnation in the small circle of blood circulation and enlargement of the right heart. On**electrocardiogram**– signs of atrial fibrillation, left atrial hypertrophy (P-mitral in I, II) and right atrium

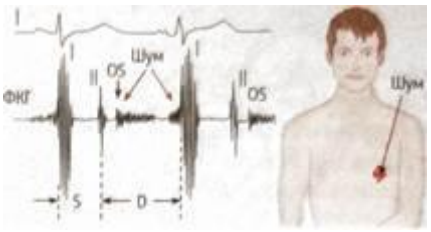

Fig. 3.5. *"Quail" rhythm and diastolic murmur in mitral stenosis*

night (EVS deviation to the right, increase in R waves in V1-V2 and S waves in V5-V6). On**FCG**diastolic noise, increased I tone, Q interval – I tone continues for more than 0.07 s (which indicates delayed closing of the mitral valve due to increased pressure in the left atrium), the opening snap of the mitral valve is recorded – OS (opening snap) .

Echocardiographymakes it possible to determine the enlargement of the left atrium, the narrowing of the venous opening and the peculiarities of the movement of the mitral valve, the enlargement of the right ventricle.

Mitral insufficiency (MI)is a heart defect when the leaflets of the left atrioventricular valve do not completely close the mitral orifice and there is regurgitation of blood from the left ventricle into the left atrium in systole (Fig. 3.6).

Violation of hemodynamics. The degree of MN depends on the intensity of regurgitation

Clinic and diagnosis

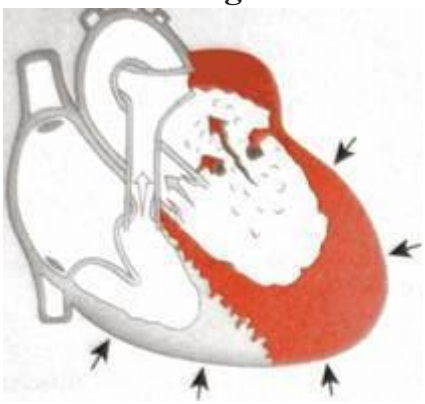

Fig. 3.6.*Mitral insufficiency*

gitation (reverse movement of blood in the heart), which can affect the state of hemodynamics. Regurgitation of up to 5 ml of blood does not change the hemodynamics of the heart, up to 10 ml is considered insignificant, more than 10 ml is significant, and 25-35 ml is severe. As a result of the reverse movement of blood from the left ventricle to the left atrium, the pressure in the latter increases. The volume of blood increases in the left ventricle. Increased pressure in the left atrium leads to the development of pulmonary hypertension (Kitaev's reflex), and congestion occurs in the veins of the small blood circulation. Over time, hypertrophy of the right ventricle develops. Decompensation of the right ventricle is characterized by relative insufficiency of the tricuspid valve, congestive changes in the central veins, liver and other internal organs. This is the last, terminal stage of heart disease.

Classification. Stages of MH: I – compensation, II – subcompensation, III – left ventricular failure, IV – dystrophic, V – terminal.

If we take into account that hemodynamic disturbances mainly "fall" on the most functionally powerful left ventricle, then MN can develop for a long time without subjective sensations and clinical manifestations and can be detected only on objective examination. Over time, during

physical exertion, shortness of breath, palpitations, a feeling of compression in the heart area, and slight cyanosis may occur. During decompensation, shortness of breath and palpitations increase, cough, hemoptysis, pain and heaviness in the right hypochondrium are added due to an increase in the liver and peripheral edema. It should be noted that cardialgias are expressed more often than in MS due to the relative insufficiency of blood supply to the hypertrophied left ventricle.
There are no changes on examination of patients in the state of compensation. With decompensation, cyanosis of the lips and mucous membranes is noticeable. During palpation in the V, sometimes VI intercostal space outward from the left midclavicular line, an increased, spilled apical impulse is determined - an important sign of mitral insufficiency. Percussion, the borders of the heart shift up and to the left. With the development of decompensation, the limit of relative cardiac dullness shifts to the right. The pulse is normal or changes insignificantly. An auscultatory sign of mitral insufficiency is characteristic and mandatory - a weakening of the first tone, which occurs due to the absence of a "period of closed valves". Another auscultatory sign is the appearance of a pathological third tone at the top, caused by the oscillations of the walls of the left ventricle during the rapid influx of an increased volume of blood from the atrium.
The most important and constant sign of MN is a "blowing" systolic noise (Fig. 3.7), caused by regurgitation of blood into the left atrium. It is better listened to at the top of the heart in a horizontal position (the pulse becomes thinner, the systolic blood volume increases). The noise is made up and to the left, in the axillary area. With the development of pulmonary hypertension, an accent of the II tone appears over the pulmonary artery, but it is less pronounced than with MO On**x-rays**expansion of the borders of the heart to the left, then to the right, a flattened waist of the heart, deviation of the esophagus along the arc of a large radius, later - congestion in the lungs are revealed.**EKG**characterized by signs of hypertrophy of the left ventricle (tall R wave in V4, deep S wave in V1) and left atrium (wide P), deviation of the electrical axis of the heart to the left. On**FCG**a weakened I tone and systolic murmur are registered.
The main methoddiagnosis of mitral insufficiency - echocardiography, which allows you to detect the expansion of the cavities of the left ventricle and atrium, as well as regurgitation (reverse circulation) to the atrium.

Aortic stenosis
Aortic stenosis (AS)(Fig. 3.8) is a heart defect in which the mouth of the aorta narrows, which makes it difficult for blood to move in systole from the left ventricle to the aorta. Aortic valve defects rank second in frequency after the mitral valve, with aortic stenosis accounting for 5 to 20% of all acquired defects.
Violation of hemodynamics. With AS, the duration of the systole of the left ventricle and the period of expulsion of blood from it increase. The work of the left ventricle increases in order of compensation in this heart defect. Over time, the left ventricle begins to empty with the rest of the blood. Diastolic pressure increases, dilatation of the left ventricle develops. The pressure in the left atrium increases due to the relative pressure

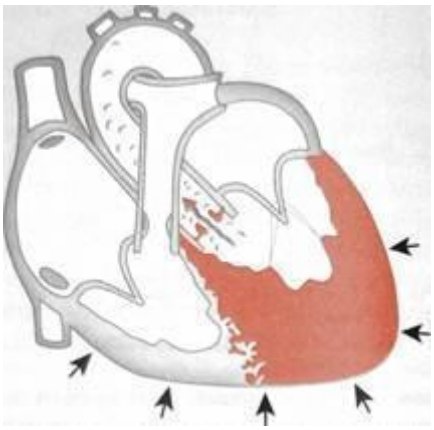

Fig. 3.8.*Aortic stenosis*

insufficiency of the bicuspid valve (moralization of the defect). In the small circle of blood circulation, the filling of venous vessels with blood increases. Next, right ventricular failure develops (stage of AC decompensation).

Classification

Stages of AC: I – complete compensation, II – hidden HF, III – relative coronary insufficiency, IV – pronounced left ventricular insufficiency, V – terminal.

Clinic

AS has an asymptomatic course for a long time due to the powerful compensatory capabilities of the left ventricle. Often, patients find out about the disease by accident, during preventive examinations.

The first*complaints*usually associated with the inadequacy of the minute volume of blood circulation to increase the load. Among them, one should highlight increased fatigue, headache, dizziness and fainting associated with insufficient cerebral blood circulation, anginal pains. As the contractile capacity of the left ventricle decreases, shortness of breath, cardiac asthma attacks, and often pulmonary edema occur. In the later stages – heaviness and pain in the right hypochondrium, peripheral edema as a result of right ventricular decompensation.

Diagnostics

Patients are pale (due to a decrease in cardiac output and spasm of skin vessels). Cyanosis occurs in case of decompensation of heart disease. On examination of the area of the heart in the V-VI intercostal space, an increased apical impulse is visualized. During palpation, an increased, resistant apical impulse is determined in the V-VI intercostal space, shifted to the left to the midclavicular line or even beyond it. When palpating the chest in the II intercostal space on the right (above the aorta), it is often possible to determine a systolic tremor (a cat's purr"), caused by the eddies of blood when passing through a narrowed aortic opening. The pulse is weak, slow, rare (*pulse small and slow)*since blood flows too slowly in the aorta, there is not enough of it. Systolic AT decreases, diastolic does not change, pulse pressure decreases. Percussion, the left border of the heart is shifted to the left and down.

During auscultation in the area of the apex of the heart, the 1st tone is weakened due to the overflow of blood in the left ventricle and its slow contraction and slight tension of the valves; II tone on the aorta is weakened or absent. A characteristic sign of AS is a systolic murmur with an epicenter on the aorta (II intercostal space to the right of the sternum) (Fig. 3.9). The systolic murmur is made on the right carotid and subclavian arteries, has a rising-falling character.

Additional research methods. On the ECG -signs of hypertrophy and systolic overload of the left ventricle, manifestations of coronary insufficiency (negative

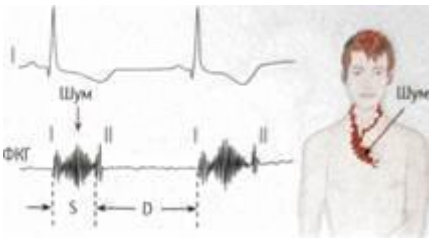

Fig. 3.9.*Systolic murmur in aortic stenosis*

waves of T in I-II and left thoracic leads).**FCG signs**: systolic murmur of a diamond-shaped nature, in aorta II tone weakens or disappears completely. And the tone is weakened or split. On**x-rays**enlargement of the left ventricle without pronounced dilatation, expansion of the ascending part of the aorta and calcification of the aortic valves are revealed; the heart takes the form of a "sitting duck".**Echocardiography**makes it possible to detect the density of calcified aortic valves, hypertrophy of the left ventricle, and assess its functional state. There is a thickening of the interventricular septum, a decrease in the degree of valve opening during systole.

Aortic insufficiency

Aortic insufficiency (AN)(Fig. 3.10) is a type of heart defect when the semilunar valves do not close the aortic opening and there is regurgitation of blood from the aorta into the left ventricle during diastole.

Hemodynamic disorders. Regurgitation of blood from the aorta into the left ventricle during diastole increases its diastolic filling. The magnitude of the reverse movement of blood depends on the area of the open part of the aortic opening, the pressure gradient between the aorta and the left ventricle during diastole. The end-diastolic pressure in the left ventricle increases, it hypertrophies. Systolic pressure in the arteries of the great circle of blood circulation increases, and diastolic pressure decreases. During the compensation period, the cardiac output, pressure in the left atrium, pulmonary capillaries correspond to normal values. With the development of myogenic dilatation of the left ventricle, relative insufficiency of the left venous opening ("mitralization" of the defect) develops, stagnation occurs in the left atrium, veins and capillaries of the small blood circulation. This increases the workload of the right ventricle and the right atrium and leads to decompensation of the heart defect - total HF.

Classification. Stages of AH: I – full compensation, II – hidden HF; III – subcompensation, IV – decompensation, V – terminal.

Clinic

AN can be asymptomatic for a long time and is often discovered accidentally during preventive examinations. The earliest and most frequent complaint is palpitations and an unpleasant feeling of beating heart. As a result of sharp oscillations of the AT, there is a feeling of "widespread pulsation" of the arteries, especially the carotid ones. Due to the deterioration of the blood supply to the brain, pulsating headaches, dizziness, and a tendency to lose consciousness occur. Typical complaints include anginal pains in the area of the heart, caused by the deterioration of coronary blood circulation. as decompensation develops and the contractility of the myocardium decreases, shortness of breath (even at rest), cardiac asthma attacks, and symptoms of right ventricular failure develop - heaviness and pain in the right hypochondrium and peripheral edema.

Diagnostics
A characteristic symptom of AN is pallor of the skin of the face and mucous membranes (small filling of arterial vessels in diastole of the heart). In advanced stages of the defect, there is visible increased pulsation of the carotid arteries - "carotid dance", as well as pulsation of other large arteries (subclavian, temporal, brachial). In patients, the head shakes synchronously with the pulse - Musset's symptom. The pupils constrict and dilate synchronously with the pulse - Landolfi's symptom. Quincke's pseudocapillary pulse is a rhythmic change in the color of the nail bed when pressing on the tip of the nail or the pulsation of a spot of hyperemia after rubbing the skin of the forehead. These symptoms are associated with a sharp fluctuation of pressure in the arterial system. Sometimes the appearance of a patient with AN is defined as*knocking man*- "pulsating man". The pulse has the character of rapid filling and falling (Corrigan's pulse) -*pulse fast and high.*AT has the most important diagnostic value and is manifested by high systolic and low (sometimes up to 0 mm Hg) diastolic pressure; pulse pressure is high. Systolic pressure is much higher in the lower extremities than in the upper extremities. During palpation of the heart area: the apical impulse in the VI–VII intercostal space is increased, diffuse, dome-shaped, shifts down and to the left. If the contractility of the left ventricle is significantly weakened, the apical impulse is defined as two impulses (bysystole according to Obraztsov). Percussion: relative cardiac dullness is shifted down and to the left due to hypertrophy and dilatation of the left ventricle. Auscultatively: over the top of the heart, the first sound is weakened, which is due to the absence of a period of closed valves, overflow of blood in the left ventricle. On the II aorta, the tone is also weakened or absent due to the change of the aortic valves and the absence of a period of closed valves, it can be completely replaced by noise. The pathognomonic sign of this defect is a loud descending diastolic murmur of a soft "blowing" nature, which occurs immediately after the II tone, is heard above the aorta and at the point of Botkin-Erb and is well conducted to the apex of the heart (Fig. 3.11). It is better heard in the patient's position standing with raised arms (Syrotinin-Kukoverov symptom).

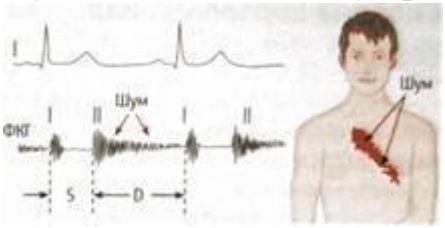

Fig. 3.11.*Diastolic murmur in aortic insufficiency*
Additional research methods. ECG signshypertrophy of the left ventricle and shift of the electrical axis to the left; with mitralization - signs of left atrial hypertrophy.**FCG**- weakening of both heart sounds is determined and a diastolic noise is recorded, which has a decreasing intensity until the end of diastole.**X-ray examination**reveals hypertrophy and expansion of the left ventricle, arch and ascending part of the aorta, which form the typical aortic configuration of the heart in the form of a "sitting duck". Echocardiography can confirm dilatation and hypertrophy of the left ventricle, increase in the amplitude of oscillation of the interventricular septum and the back wall of the left ventricle, an increase in the anteroposterior size of the left atrium, and the absence of diastolic closure of the aortic valve leaflets.

General material and mass-methodological support
lectures:

work program of the academic discipline
synopsis (plan-summary) of the lecture
multimedia presentation of the lecture

Questions for self-control:

1. Mitral stenosis: causes.
2. Mitral stenosis: mechanisms of hemodynamic compensation and decompensation;
3. Mitral stenosis: main complaints, mechanism of their occurrence, clinical characteristics, diagnostic methods.
4. Mitral valve insufficiency: causes.
5. Mitral valve insufficiency: hemodynamic compensation and decompensation mechanisms; Mitral valve insufficiency: main complaints, mechanism of their occurrence, clinical characteristics, diagnostic methods.
6. Aortic valve stenosis: causes.
7. Aortic valve stenosis: mechanisms of hemodynamic compensation and decompensation.
8. Aortic valve stenosis: complaints, mechanism of their occurrence, clinical characteristics, diagnostic methods.
9. Aortic valve insufficiency: causes.
10. Aortic valve insufficiency: mechanisms of hemodynamic compensation and decompensation.
11. Aortic valve insufficiency: main complaints, mechanism of their occurrence, clinical characteristics, diagnostic methods.

Lecture No. 10 "Ischemic heart disease: angina pectoris, myocardial infarction. Clinic, diagnosis, principles of treatment. Hypertensive disease and symptomatic hypertension. Clinical picture, classification, diagnosis. Syndrome of heart and vascular insufficiency."

Actuality of theme :

Hypertension is a separate independent disease, the main symptom of which is elevationAD, caused by neuro-functional disorders of the regulation of vascular tone. Hypertensive disease is followed clearly**distinguish** from the so-called symptomatic hypertension, which occurs secondary to the presence of certain diseases of the kidneys and endocrine glands, as well as organic lesionsCENTRAL NERVOUS SYSTEM or large vessels (aorta, renal, carotid).
The main etiological factor of hypertension is considered to be neuropsychological overstrain of the central nervous system, caused by short-term acute or long-term negative nervous effects (emotional stress, strong excitement, excessive mental workload, high responsibility, and the like). These factors lead to the development of primary functional disorders in the brain stem, especially in the hypothalamic centers that regulate blood pressure.
A number of factors contribute to the occurrence of hypertension, which are called risk factors. The most important among them are: heredity, professional factors, abuse of table salt, suffered craniocerebral injuries and brain diseases.
Hypertensive disease should be clearly distinguished from the so-called symptomatic hypertension, which occurs secondary to the presence of certain diseases of the kidneys and

endocrine glands, as well as organic lesions of the central nervous system or large vessels (aorta, renal, carotid).
The term "coronary heart disease" refers to a group of diseases (angina, myocardial infarction, cardiosclerosis), which are based on a mismatch between coronary blood circulation and the myocardium's need for blood supply, caused by atherosclerosis of the coronary arteries.
Ischemic heart disease is one of the most common diseases of the adult population in economically developed countries countries of the world.
A number of factors contribute to the occurrence of coronary heart disease, which are called factors risk The most important among**n**they are considered: overweight bodies, bad habits**(**smoking, overeating**),** concomitant diseases (diabetes mellitus, hypertension), weighted relative to coronary heart disease, heredity (myocardial infarction among close relatives under 50).
Ischemic heart disease (unstable angina, tension angina, myocardial infarction)**is**very important for study by the acquirers of that points of view that can be encountered in the practice of a doctor of any specialty, as well as in connection with high mortality, in the absence of treatment, a large number of complications that lead to invalidation of patients.

Purpose of the lecture:

1. the applicant must know the clinical manifestations of hypertension at different stages of the course of the disease.
2. the applicant must know the methods of diagnosing hypertension.
3. the applicant must know the classification of hypertension.
4. the applicant must know the examination plan for hypertensive patients.
5. the applicant must know the etiology of symptomatic arterial hypertension.
6. the applicant must know the examination plan for a patient with symptomatic arterial hypertension
7. the applicant must know the classification of the main groups of symptomatic arterial hypertension
8. the applicant must know the clinical manifestations of symptomatic arterial hypertension.
9. the applicant must be able to evaluate the data of instrumental and laboratory studies in patients with hypertension and symptomatic hypertension.
10. the applicant must know the etiology and mechanism of development of coronary heart disease.
11.
12. the applicant must know the classification of coronary heart disease, functional classes of angina pectoris.
13. the applicant must know the clinical picture of angina pectoris.
14. the applicant must know the basic laboratory and instrumental methods of diagnosing angina pectoris.
15. the applicant must know the risk factors for the development of CHD.
16. the applicant must know the etiology and mechanism of development of myocardial infarction.
17. the applicant must know the clinical picture of various variants of the course of myocardial infarction.

18. the applicant must know the laboratory diagnosis of myocardial infarction.
19. the applicant must know the main electrocardiographic changes in myocardial infarction.
20. the applicant must know the main complications of myocardial infarction.

Basic concepts: ischemic heart disease, angina pectoris, myocardial infarction. hypertension, heart failure, patient examination.

Plan and organizational structure of the lecture.

1. Determination of the educational goal.
2. Providing positive motivation.
3. Presentation of the lecture material according to the plan:
4. Summary of the lecture. General conclusions.
5. The lecturer's answer to possible questions.
6. Tasks for self-training.

Content of the lecture material

The leading symptom of hypertensive disease (mordus hypertonicus) is an increase in blood pressure, caused primarily by neuro-functional disturbances in the regulation of vascular tone. Both men and women suffer from hypertension equally often, mainly after 40 years.

It is necessary to carefully distinguish between hypertensive disease and so-called symptomatic hypertension, when an increase in blood pressure is a symptom of the disease, and at the same time it is far from the main one. Symptomatic hypertension is observed with coarctation of the aorta, atherosclerosis of the aorta and its large branches, with impaired function of the endocrine glands (Itsenko-Cushing's disease, pheochromocytoma, hyperthyroidism, primary aldosteronism - Kohn's syndrome), damage to the kidney parenchyma, occlusive damage to the main renal arteries, brain tumors, etc. further

Etiology and pathogenesis. The main importance in the origin of the disease is given to overstrain of the central nervous system, caused by prolonged or strong excitement, excessive mental stress, and emotional upheaval. In some cases, hypertension develops after a brain concussion (contusion-comcussion form). The importance of neurogenic factors in the development of this disease was pointed out by G.F. Lang back in 1922, and later this was confirmed by the experience of Soviet doctors during the Great Patriotic War. Thus, during the blockade of Leningrad, a massive development of hypertension in previously perfectly healthy people was noted.

Profession has a certain importance in the development of the disease. The disease most often occurs in people whose work is associated with nervous and mental overstrain: in those engaged in mental work (scientists, engineers, doctors), in drivers, telephone operators, and so on, the factors contributing to the development of the disease include a hereditary predisposition. In some families, there is a high frequency of hypertensive disease, which, without a doubt, depends on the action of the same factors of the external environment, and partly on the hereditary characteristics of the nervous system and metabolism.

In the period of its occurrence, hypertensive disease is characterized by a neuro-functional disorder of the regulation of vascular tone. Further, the process includes, as consecutive pathological links, disorders of vegetative-endocrine and renal regulation of vascular tone. Overexertion of the sphere of higher nervous activity leads to a vasopressor adrenalin reaction, as a result of which there is a narrowing of arterioles, mainly internal organs, especially kidneys. The resulting ischemia of the kidney tissue, in turn, leads to the production of renin by the juxtaglomerular cells of the kidney glomeruli, which in the blood plasma leads to the formation of angiotensin II. The latter has a pronounced pressor effect and stimulates the secretion of the "sodium-retaining hormone" - aldosterone - by the cortex of the adrenal glands. Aldosterone promotes the transition of sodium from the extracellular fluid to the intracellular fluid, thereby increasing the sodium content in the vascular wall, leading to its swollen swelling and narrowing, which in turn is reflected in an increase in blood pressure.

There is no doubt that there is a system of depressant factors in the body, the weakening of which functions undoubtedly also play a role in the pathogenesis of hypertension. So, bradykinin and angiotensinase, which have a depressant effect, were detected. It is believed that for some currently unknown reasons, a change in the depressor system occurs in hypertension.

The results of long-term observations unanimously show that elevated blood pressure is a significant and independent risk factor for the development of coronary heart disease, cerebral stroke, congestive heart failure, uremia and naturally shortens life expectancy.

In people with a diastolic blood pressure of 105 mm Hg. the probability of a stroke is 10 times higher, and coronary heart disease is 5 times higher than in people with a diastolic pressure of 75 mm Hg. Art. Long-term reduction of diastole blood pressure by 5 and 10 mm Hg. leads to a decrease in the frequency of cerebral strokes by 34 and 56%, respectively, and coronary heart disease by 21 and 37%.

In the general population, the number of persons with high PECLO is relatively small, and patients with moderate and slight elevation of BP predominate. However, in general, the contribution of hypertension with a moderate and slight increase in blood pressure to overall mortality and the development of complications is much greater. And although hypertension is an independent risk factor for premature death due to the development of complications from the cardiovascular system, this risk increases significantly if the patient has hypercholesterolemia (above 5.2 mmol/l), smoking, and diabetes. Therefore, at the same blood pressure level, the probability of developing complications will be determined by the presence of other risk factors or their combinations.

Pathological anatomy. With hypertension, a violation of the permeability of vascular walls, their protein impregnation, which in later or more severe forms of the disease leads to sclerosis or necrosis of the wall of small arteries with secondary changes in organ tissues, gradually develops. Atherosclerotic changes are usually observed in the walls of large vessels. The varying degree of damage to the vessels of different organs is characteristic, therefore there are different clinical and anatomical variants of the disease with predominant lesions of the vessels of the heart, brain or kidneys (in the latter case, "primary shriveling of the buttocks" occurs).

Clinical picture. In the early period of the disease *complaints* patients are mainly neurotic. They are concerned about general weakness, reduced work capacity, inability to concentrate on work, insomnia, fleeting headaches, heaviness in the head, dizziness, tinnitus, sometimes palpitations. Then shortness of breath appears during physical exertion - climbing stairs, running.

Contrary to the popular belief that hypertension or GB are characterized by certain complaints (headaches, dizziness, heart pain, increased irritability, poor sleep, etc.), the following should be noted. The indicated pathology is subjectively mildly symptomatic, and the presence of the above complaints most likely indicates the addition of another disease, namely, vegetative dystonia. Therefore, the study of the patient's complaints in terms of the diagnosis of hypertension is not important, and the measurement of blood pressure plays a decisive role here.

However, it should be remembered that the appearance of cardiovascular complaints in a patient with a long and persistent increase in blood pressure may be due to damage to the target organs. So, for example, headaches, dizziness, paresthesias, weakness in the limbs, and transient visual impairment will indicate damage to the brain and retina. Heart palpitations, shortness of breath during physical exertion, pain behind the sternum when walking will indicate heart failure.

Mainly*an objective sign* disease is an increase in blood pressure: both systolic (above 140-160 mm Hg) and diastolic (more than 90 mm Hg).

Diagnosis of hypertension is quite simple and is based mainly on the repeated detection of elevated BP numbers. However, in order to avoid hypo- or hyperdiagnosis when measuring blood pressure, a number of rules must be followed. Blood pressure is usually measured by a doctor or nurse on the right shoulder. A mercury or aneroid manometer is used for this. At the same time, the first should be preferred, since the second needs frequent calibration.

Before blood pressure measurement, the patient should not smoke for 20-30 minutes, spend at least 5 minutes in a warm room and relax. The study is usually carried out in a sitting position, although it is possible to measure blood pressure in a lying or standing position.

However, in all cases, the ulnar fossa should be located at the level of the heart (fourth intercostal space), the patient's clothing should not squeeze the shoulder, and the applied cuff should cover it evenly (at least 80% of the circumference). For adults, a cuff 13-15 cm wide and 30-35 cm long is used

When measuring blood pressure, the cuff is quickly inflated with air to a pressure value of 30 mm Hg where the pulse disappears, which exceeds the level. Then slowly, at a speed of 2 mm Hg. air is released from the cuff. In the course of this time, with the help of a stethoscope located above the brachial artery, Korotkov's tones are listened to.

At the appearance of tones (I phase), systolic pressure is recorded, and diastole - at the moment of disappearance of tones (V phase of Korotkov tones). It is necessary to pay attention that the diastolic pressure is determined precisely by the disappearance of tones, and not by their weakening (1V phase). BP must be determined twice with an interval of at least 2 minutes. At the same time, if two numbers differ by more than 5 mm Hg. Art., an additional measurement must be performed. Then the average of all measurements is calculated.

Since PECLO is a variable value, before diagnosing hypertension, its steady increase should be confirmed by repeated measurements. It is especially necessary to do this when so-called mild hypertension is detected (HELL within 140-159/90-99 mm Hg).

Blood pressure measurement by the patient himself or his relatives should be encouraged in every possible way. Usually, the blood pressure values obtained in this case are lower than office blood pressure values (more often in elderly people), make it possible to avoid the so-called "white coat hypertension" and allow the doctor to better orient himself in the tactics of carrying out treatment and preventive measures.

In the initial stages of the disease, blood pressure is often prone to large fluctuations, later it becomes more constant. During the examination of the heart, signs of hypertrophy of the left

ventricle are noted: increased apical impulse leading to failure, displacement of cardiac dullness to the left. An accent of the second tone is heard above the aorta. The pulse becomes hard, tense.

An X-ray examination shows the aortic configuration of the heart. The aorta is elongated, compacted and expanded.

On the ECG, the left type, displacement of the segment S - T is revealed; smoothed, negative or biphasic Tonnes wave in I - II standard and left chest leads (V5 - V6

Atherosclerosis of the coronary arteries often develops at the same time, which can lead to the development of angina attacks and myocardial infarction. In the later period of the disease, heart failure may occur due to overfatigue of the heart muscle due to increased pressure; often it manifests itself acutely in the form of cardiac asthma attacks or pulmonary edema, or chronic circulatory failure develops.

With a severe course of the disease, a decrease in vision may occur; examination of the fundus reveals its general pallor, narrowness and tortuosity of the arteries, slight dilatation of the veins, sometimes hemorrhages in the retina (angiospastic retinitis).

In cases of damage to the vessels of the brain under the influence of high pressure, a violation of cerebral blood circulation may occur, leading to paralysis, impaired sensitivity, and often to the death of the patient; it is due to spasm of blood vessels, thrombosis, hemorrhages as a result of rupture of a vessel or the release of erythrocytes per diapedesem.

Damage to the kidneys leads to a violation of their ability to concentrate urine (nocturia, isohypostenuria occurs), which can cause a delay in the body of metabolic products to be excreted in the urine, and the development of uremia.

Hypertensive disease is characterized by periodically occurring short-term increases in blood pressure - a hypertensive crisis. The appearance of such a crisis is facilitated by mental trauma, nervous tension, fluctuations in barometric pressure, etc.

Hypertensive crisis manifested by a sudden rise in blood pressure of varying duration (from several hours to several days), which is accompanied by a sharp headache, dizziness, a feeling of heat, sweating, palpitations, stabbing pains in the area of the heart, sometimes impaired vision, nausea, vomiting. In severe cases, loss of consciousness may occur during the crisis. During a crisis, patients have an excited, frightened appearance, or they are lethargic, sleepy, inhibited. There is hyperemia of the face, increased skin moisture. During auscultation of the heart, an increase in the emphasis of the second tone over the aorta, tachycardia is revealed. The pulse becomes more frequent, but may not change or become shorter, its voltage increases. Blood pressure is sharply increased: systolic up to 200 mm Hg. Art. and above

The ECG shows a decrease in the S-Ton interval, flattening of the T wave.

In the late stages of the disease, when there are already organic changes in the vessels, during a crisis, disorders of cerebral circulation, myocardial infarction, and acute left ventricular failure may occur.

Classification. Currently, several classifications of AG are proposed. All of them are based on the data of numerous epidemiological studies and prospective observations of individuals with elevated PECLO, on the results of an in-depth examination of patients with hypertension and their active treatment. Classification of hypertension is usually based on three characteristics: etiology, degree of damage to target organs, and the height of the HELL.

According to the WHO Expert Committee (1997), in more than 95% of patients with hypertension, it is not possible to establish its cause. In these cases, they talk about primary, essential hypertension or hypertensive disease (HB). Therefore, the absolute majority of patients

with hypertension when dividing them by etiology are patients with hypertension. Only a small group of people with hypertension have the opportunity to find out the specific cause of the disease. In these cases, symptomatic or secondary hypertension is diagnosed.
The WHO expert committee (1997) proposes to highlight the following causes of symptomatic hypertension.
1. *Medicines and exogenous substances* : hormonal contraceptives, corticosteroids, sympathomimetics, cocaine, food products containing thiamine or monoamine oxidase inhibitors, nonsteroidal anti-inflammatory drugs, cyclosporine, erythropoietin.
2. *Kidney disease:* acute and chronic glomerulonephritis, chronic pyelonephritis, obstructive nephropathies, polycystic kidneys, diabetic nephropathies, hydronephrosis, congenital hypoplasia of the kidneys, kidney injuries, renovascular hypertension, renin-secreting tumors, renoprivative hypertension, primary salt retention (Liddle's syndrome, Gordon's syndrome).
3. *Endocrine diseases:* acromegaly, hypothyroidism, hypercalcemia, hyperthyroidism, diseases of the adrenal glands (lesion of the cortical layer - Itsenko-Cushing syndrome, primary aldosteronism, congenital hyperplasia of the adrenal glands, lesion of the medulla (pheochromocytoma, tumor of chromaffin cells located outside the adrenal glands, cancer).
4. *Coarctation of the aorta and aortitis.*
5. *Complications of pregnancy.*
6. *Neurological diseases:* increased intracranial pressure, brain tumors, encephalitis, respiratory acidosis, sleep apnea, total limb paralysis, acute porphyria, lead poisoning, Guillain-Barré syndrome.
7. *Complications of surgical interventions:* postoperative hypertension (for example, after coronary artery bypass grafting).
The principles of such differential diagnosis are developed in detail.
At the first stage of the examination, quite simple methods are used, most of which can be performed in the clinic.
The second stage of the examination is carried out, as a rule, in a specialized clinic, where patients whose diagnosis could not be definitively established at the first stage go. Quite complex and expensive biochemical, hormonal, instrumental and morphological (bud biopsy) methods are used here, indicators for surgical and medical treatment are specified.
Already at the first examination of a patient with elevated blood pressure, a doctor can suspect the presence of symptomatic hypertension based on the history and objective data and prescribe an additional examination.
History data that may indicate symptomatic hypertension.
Diseases of the kidneys, urinary tract, hematuria, abuse of analgesics (damage of the kidney parenchyma). Kidney disease in parents (polycystic kidney disease).
Frequent use of various drugs and substances: oral contraceptives, nonsteroidal anti-inflammatory drugs, cocaine, nasal drops.
Episode of excitement with headache, sweating (phaeochromocytoma).
Episodes of muscle weakness (primary hyperaldosteronism).
Objective examination data that may indicate symptomatic hypertension.
Symptoms of Itsenko-Cushing's disease.
Symptoms of thyrotoxicosis, hypothyroidism.
Change in facial features, shoe size (acromegaly).
Neurofibromatosis of the skin (may indicate pheochromocytoma).

Enlargement of the kidneys, which is detected by palpation (polycystic kidney).
During auscultation, noise in the projection of the renal arteries (renovascular hypertension).
Auscultatory noises in the area of the heart and chest (coarctation of the aorta or aortitis).
Weakened or such that the pulse on the femoral artery is delayed and the blood pressure on the thigh is lowered (coarctation of the aorta, aortitis).
The degree of damage to the cardiovascular system in hypertension usually adjusts both with the level of blood pressure and the duration of its increase. In this regard, in 1978, the WHO expert committee proposed a classification of hypertension, primarily essential hypertension (GB), which involves dividing the disease into 3 stages depending on the degree of damage to the target organs. At the same time I*stage* characterized only by an increase in HELL (160/95 mm Hg and above), while there are no organic changes in the cardiovascular system.
GB*In stage II, in addition to high blood pressure, at least one of the signs of damage to the target organs is manifested: hypertrophy of the left ventricle, narrowing of retinal vessels, microalbuminuria, etc. and finally, at stage III* there are already clinical manifestations of target organ damage (angina, previous myocardial infarction, heart failure, stroke, hypertensive encephalopathy, retinal hemorrhages and vision loss, kidney failure, etc.). At this stage of the disease, blood pressure may decrease after heart attacks and strokes.
The given classification generally reflects the progress of the disease over time and convinces with its logic. However, during two decades of its use, shortcomings were revealed, which are mainly reduced to the following. First, the severity of damage to target organs in patients with hypertension does not always correspond to the height of blood pressure and the duration of its increase. High blood pressure can be observed even in the absence of organic changes on the part of the cardiovascular system and, on the contrary, the manifestation of decompensation of the concerned organs is often observed with moderately elevated pressure. Secondly, determining the stage of hypertension according to this classification requires the mandatory use of various diagnostic methods, sometimes quite expensive and inconvenient for the patient, which complicates the diagnosis of such a widespread pathology as hypertension. Thirdly, according to this classification, only patients with PEKLO 160/95 mm Hg are included in patients with GB. and above However, there is a rather large group of patients with PECLO in the so-called "dangerous zone", who have signs of GB and even have damage to target organs and who, according to the formal criteria of AG, cannot be recognized as sick. Fourthly, the doctor focuses not only on the degree of damage to the target organs, but also on the height and stability of blood pressure, which is practically not taken into account in this classification. The use of this classification, as it were, predetermines the course of the disease and its steady progress, while it is now indisputably proven that long-term hypotensive therapy, which is successfully carried out, can lead to the reversal of pathological changes in the target organs.
Of the currently proposed classifications of hypertension according to the level of pressure, the classification of the United States National Committee for Detection, Evaluation and Treatment of High Blood Pressure (1993) undoubtedly deserves attention.
The proposed classification is essentially the result of a careful analysis of the results of years of hard work by general practitioners and cardiologists to reduce morbidity and mortality from diseases of the cardiovascular system in the period from 1972 to 1990. mortality from coronary heart disease decreased in the US by almost 50%, and from strokes - by 57%.
In 1977, the "Sixth Report of the Joint National Committee on the Prevention, Recognition, Evaluation and Treatment of High Blood Pressure (USA) - JNC - 6* was published, which was

created on the basis of the synthesis of the latest scientific data and is essentially a guide for doctors of the "First line". In this report, the new classification of pressure, which is now international, was continued.

The most important feature of the given classification is also the introduction of the concept "**Optimum pressure", which should be *in an adult, regardless of age, it is below 120/80 mm of mercury***. This actually contradicts the widespread opinion that the pressure should supposedly increase with age and specifically shows the values that a healthy person should have.

Normal pressure is considered below 130/85 mm Hg. Art. This, without a doubt, mobilizes doctors and patients to a stricter approach to PRESSURE values. High-normal PRESSURE is highlighted, which requires close attention, since patients in this category have a higher risk of developing a stroke and a result in GB than that of "normotensives". PRESSURE 140/90 mm Hg. Art. is already considered as a manifestation of the I stage of GB, that is, in fact, the criteria of the disease, according to this classification, are significantly strengthened. This classification provides for the division of GB into four stages.

Classification of blood pressure (in mm Hg)
for adults (18 years and older)

E category	Blood pressure	
	< 80	
	< 85	
mal	39 or 85-89	
pertension		
	90-99	
	100-109	
	110	

According to the classification, the diagnosis of hypertension and special recognition of the stage of the disease is based on the repeated measurement of pressure in persons not receiving hypotensive therapy. Average values from at least two PECLO determinations obtained during two visits to the doctor should be used.

In other words, a doctor who has discovered hypertension in a patient should not immediately prescribe antihypertensive drugs. He should invite the patient to come to him for an appointment two more times. During this period of time, the required amount of research should be carried out (ECG, urinalysis, fundus, cholesterol level in the blood), identify risk factors for hypertension and conditions that aggravate the course of the disease, determine the presence and degree of damage to target organs, and only after that diagnose, prescribe treatment. This is of fundamental importance, since the established stage of GB actually determines the patient's treatment tactics for many years.

It is also important to note that when systolic and diastolic HELL fall into different categories of the HELL classification, it is necessary to take a higher criterion for individual assessment of hypertension. For example, the level of HELL is 165/90 mm Hg. Art. should be classified as stage II, and 170/110 mm Hg. art - as stage III.

When examining a patient, the doctor must take into account the presence of risk factors for diseases of the cardiovascular system. The main ones are the following:

- hereditary burdened (a case of hypertension and other diseases of the cardiovascular system in the family: in men up to 55 years old, in women up to 65 years old);
- excessive body weight;
- hypodynamia;
- increased consumption of table salt;
- alcohol;
- smoking;
- dyslipidemia (serum cholesterol 5.2 mmol/l and above);
- triglycerides - 1.5 mmol/l and above;
- diabetes in a patient with hypertension and his parents.

The first five of the indicated risk factors are the main ones, and they are directly or indirectly related to the development of GB and its further progress. Their elimination or at least a reduction of clinical and laboratory manifestations (for example, a decrease in body weight, an increase in physical performance under the influence of training) can prevent the development of the disease or delay the time of manifestation of hypertension in healthy individuals, and in patients with hypertension, reduce and minimize the dose of hypotensive agents used, improve forecast. Of course, burdened heredity cannot be eliminated (the so-called uncorrected risk factor), but its accounting is very important in terms of determining the intensity of all treatment and preventive measures for both a specific GB patient and her children, who have a fairly high probability of developing the disease. Such risk factors as smoking, dyslipidemia, and diabetes mellitus are concomitant, they do not directly participate in the pathogenesis of GB, but their presence in the patient significantly aggravates the course of the disease and contributes to the development of complications. Risk factors are not indicated in the diagnosis, but they are necessarily taken into account when developing a treatment and rehabilitation strategy for a specific patient.

Under the term "coronary heart disease" unite a group of diseases based on absolute or relative violation of coronary blood circulation of various genesis, including angina pectoris, myocardial infarction, chronic coronary insufficiency, coronary cardiosclerosis.

Nowadays, this disease is very common, especially in highly developed countries. The probability of developing CHD in Europeans over 50 years old reaches 63%. Quite often it leads to early disability and premature death. The main reason leading to the development of coronary heart disease (in 59% of cases) is an increase in plasma cholesterol of more than 5 mmol/l. Arterial hypertension of more than 140/90 mmHg is in second place among the risk factors for CHD. Art. In third place - excess body weight. In fourth place - smoking.

Angina pectoris (synonyms: chest pain, angina pectoris) is a disease whose main clinical symptom is chest pain attacks caused by acute but transient coronary blood circulation disorders. Angina is a common disease. It occurs in most cases in people over 40 years of age, and in men much more often than in women. This disease mainly affects people with mental work.

Etiology and pathogenesis. The most common cause of angina is atherosclerosis of the coronary arteries of the heart

At the heart of an angina attack is hypoxia (ischemia) of the myocardium, which develops in conditions when the amount of blood flowing through the coronary arteries to the working heart muscle becomes insufficient and the myocardium unexpectedly experiences oxygen starvation. The resulting temporary oxygen starvation leads to a reversible violation of redox processes in the myocardium. Irritation by the products of disturbed exchange of interoreceptors of the myocardium or the adventitia of vessels causes a flow of impulses along the central pathways to the cerebral cortex and causes the characteristic symptom of this disease - retrosternal pain. In addition, increased activity of the sympathoadrenal system is important in the development of angina pectoris.

Pathological anatomy. Sometimes, in people who died during an attack of angina pectoris, no organic changes are found, but more often, in 85-90% of cases, signs of atherosclerosis of coronary arteries of varying severity are found.

Clinical picture.

The disease has a chronic course. Attacks may be rare, once a week or less, may not recur for several months or even years, or may become increasingly frequent and severe. An attack of angina lasting more than ½ to 1 hour can end in a myocardial infarction. In patients who suffer from angina pectoris for a long time, cardiosclerosis develops, the heart rhythm is disturbed, and symptoms of heart failure appear.

The following forms of angina are distinguished:

- **Angina tension** - seizures are caused by an increase in the metabolic needs of the myocardium (increased blood pressure, tachycardia), mainly as a result of physical or emotional stress.
- **Spontaneous (special) angina pectoris** - attacks occur without apparent connection with factors leading to an increase in the metabolic needs of the myocardium.
- **Angina is stable** - attacks occur constantly when performing the same load.
- **Angina is progressing** - attacks of chest pain begin to occur with less than usual physical exertion.
- **Angina is unstable** - it is divided into angina that is progressing, first occurring and spontaneous or Prinzmetal's angina.

<u>Stable angina pectoris</u>

Complaints The main clinical symptom of the disease is pain localized in the center of the sternum (sternal pain), less often in the area of the heart. The nature of the pain is different; many sufferers experience squeezing, squeezing, burning, heaviness, and sometimes cutting or sharp pain. Pain sensations are extremely intense and are often accompanied by a feeling of fear of death. Irradiation of pain in angina pectoris is characteristic and very important for diagnosis: in the left shoulder, left arm, left half of the neck and head, lower jaw, interscapular space, and sometimes in the right side or in the upper part of the abdomen. Irradiation of pain in angina pectoris is caused by increased sensitivity of the skin and pain in the zones that innervate the VII cervical and I, - V thoracic segments of the spinal cord (Geda-Zakharin zones). Irritations from the heart go through these segments and pass to the centrifugal spinal nerves according to the principle of viscerosensory reflux.

Pain occurs under certain conditions: when walking, especially fast, and other physical exertion (chest pain). With physical stress, the heart muscle needs a greater supply of nutrients with the

blood, which can't be provided by the damaged arteries with atherosclerotic damage. The patient must stop, and then the pain stops after a few minutes. Especially typical for angina is the appearance of pain after the patient leaves a warm room for a cold one, which is more often observed in autumn and winter, especially when the atmospheric pressure changes. In case of excitement, pains appear even out of connection with physical tension.

The level of stress that causes an angina attack is the most important criterion in determining the severity of coronary disease. The division of patients with stable angina into functional classes is based on the tolerability of physical exertion.

1 functional class. The patient tolerates normal physical activity well. Angina attacks occur only during high-intensity exercise.

2 functional class. Slight limitation of usual physical activity. Angina attacks occur when walking on level ground at a distance of more than 500 m, when climbing more than one floor. The probability of an angina attack increases when walking in cold weather, against the wind, during emotional excitement or the first hour after waking up.

3 functional class. Expressed limitation of usual physical activity. Attacks occur when walking at a normal pace on level ground for a distance of 100 to 500 m and/or when climbing one floor.

4 functional class. Angina occurs with light physical exertion, walking on level ground at a distance of less than 100 m. The occurrence of angina attacks at rest is characteristic.

Myocardial infarction (infarctus myocardii) - a disease characterized by the formation of a necrotic focus in the heart muscle as a result of a violation of coronary blood circulation. Myocardial infarction is observed mainly in people older than 45 years, and more often in men than in women.

Etiology. One of the main reasons leading to the development of myocardial infarction (at least in 90-95% of cases) is atherosclerosis of the coronary arteries of the heart. In very rare cases, myocardial infarction occurs as a result of coronary embolism in endocarditis or septic thrombophlebitis, on the basis of inflammatory lesions of the coronary arteries - rheumatic or syphilitic coronary disease, obliterating endarteritis, nodular periarteritis. Fatigue, nervous shock, physical overexertion, overeating, alcohol intoxication, and bad smoking contribute to the occurrence of a heart attack.

Pathogenesis compiled and not fully studied. Nowadays, the view that several factors are involved in the pathogenesis of the disease is gaining more and more popularity.

Some researchers consider coronary thrombosis and stenosing coronary sclerosis to be the main ones. The development of coronary thrombosis is facilitated by local changes in the walls of the vessels, characteristic of atherosclerosis, as well as disorders in the anticoagulation system of the blood, the content of heparin in the blood, manifested by a decrease, and a decrease in its fibrinolytic activity. In the absence of thrombosis, a major role in the occurrence of myocardial infarction is attributed to the increased work of the heart in conditions of reduced blood supply to the myocardium as a result of stenotic coronary sclerosis

. ***Clinical picture.*** Clinical manifestations of the disease are diverse. In 1909, prominent Russian clinicians V.P. Zrazkiv and N.D. Strazhesco was the first in the world to describe the clinical picture of myocardial infarction and developed three variants of its course: angina, asthmatic and abdominal (gastroenterological) forms. Further studies showed that the three forms described do not cover all clinical manifestations of the disease. Currently, the number of described variants of the course of OIM has increased significantly. This is a cerebral form, an arrhythmic form, a painless form, etc.

The anginal form occurs most often and is clinically manifested by a pain syndrome. There are squeezing pains behind the sternum or in the area of the heart, as in angina pectoris. Sometimes they cover the entire breast. As a rule, the pain radiates to the left shoulder and left arm, less often to the right shoulder. Sometimes the pains are so acute that they cause the development of cardiogenic shock, which is manifested by increasing weakness and adynamia, paleness of the skin, a cold sticky afterwards and a decrease in blood pressure. In contrast to pains in angina pectoris, pains in myocardial infarction do not go away after taking nitroglycerin and are very long-lasting (from ½ hour to several hours). They are so intense that patients often rush to bed without finding a place for themselves. Prolonged pain during myocardial infarction is referred to as status anginosus.

With asthmatic form the disease begins with an attack of cardiac asthma and pulmonary edema. The pain syndrome is weakly expressed or absent. This variant of the disease is typical for large or repeated myocardial infarctions.

For abdominal shape Myocardial infarction is characterized by abdominal pain, more often in the epigastric region. Pain may be accompanied by nausea, vomiting, stool retention (gastralgic form of myocardial infarction).

For the diagnosis of myocardial infarction, it is important to determine the activity of the series***enzymes***, which are released as a result of necrotic changes in the myocardium: aspartate amine transferase (AST), creatine phosphokinase (CFC), and to a lesser extent glutamine-pyruvic transaminase (GPT). An increase in CFC is observed only in myocardial infarction and skeletal muscle diseases, and its activity increases earlier than that of other enzymes (normally, CFC activity ranges from 0.2 to 4 units; its increase during a heart attack begins already 3-4 hours after the onset of the disease) .

They are especially important***electrocardiographic*** research, because with their help it is possible not only to establish the presence of myocardial infarction, but also to clarify a number of the most important details - the localization, depth and extent of the damage to the heart muscle.

In the first hours of the development of the disease, there is a change in the S-Ton segment and the T wave. The descending knee of the R wave, not reaching the isoelectric line, passes into the S - T segment, which, rising above it, forms an arc turned by a convexity upwards and which merges directly with the wave T. A so-called monophasic curve is formed, which is sometimes called a "cat's back".

These changes usually last 3-5 days. Then the segment S - T gradually decreases to the isoelectric line, and the Tonna wave becomes negative, deep. A deep Q wave appears and the R wave becomes low or completely disappears, and then a QS wave is formed. The appearance of a QS wave is characteristic of a transmural infarction.

General material and mass-methodological support
lectures:

1. work program of the academic discipline
2. synopsis (plan-summary) of the lecture
3. multimedia presentation of the lecture

Questions for self-control:

1. CHD: syndrome of acute and chronic coronary insufficiency: definition.

2. Main complaints and examination results of patients with angina pectoris.
3. Instrumental and laboratory methods of diagnosis in angina pectoris syndrome.
4. Principles of treatment.
5. CHD: myocardial infarction. Clinical picture.
6. Classification of myocardial infarction. Diagnostic methods. Principles of treatment.
7. Chronic heart failure syndrome. Definition.
8. Chronic heart failure syndrome: clinical picture, classification by stages and functional classes, diagnostic methods. Principles of treatment.