MINISTRY OF HEALTH PROTECTION OF UKRAINE

ODESSA NATIONAL MEDICAL UNIVERSITY

Faculty: medical
Department of propaedeutics of internal diseases and therapy

CONFIRMED by

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METHODICAL DEVELOPMENT FOR PRACTICAL LESSONS FROM EDUCATIONAL DISCIPLINE

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

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Practical lesson No. 1
Subject: Tasks of the department of propaedeutics of internal diseases. Patient examination plan. Scheme of medical history. Questioning the patient. General examination of the patient, individual parts of the body. The diagnostic value of the symptoms detected during the examination of the patient.

Goal: To know about propaedeutics of internal diseases as a science, its purpose and tasks, the role of domestic scientists in the development of therapy. Acquiring knowledge and mastering professional competences during the examination of the patient. The ability to conduct a survey of the patient (collection of complaints with their details, medical history, life history) and physical examination of the patient (general examination and individual parts of the body). Know the main sections of the medical history and their significance in the diagnostic process. To develop skills, to cultivate attention during the collection of anamnesis, compilation of the scheme of the medical history and the general plan of the examination of the patient.

Basic concepts: In clinical practice, the study of internal diseases has a complex nature, due to the need to constantly apply knowledge of normal and pathological anatomy, normal and pathological physiology, biochemistry, pharmacology and related clinical disciplines.
The first subject that introduces students to the clinic of internal medicine is propaedeutics (fundamentals of diagnosis) of internal medicine. The term "propaedeutics" comes from the Greek.*propaideuo*, which means "teaching beforehand".
The purpose of discipline - to give the basics of diagnosis and evaluation of the main manifestations of diseases of internal organs, to form the professional skills of patient examination, the basics of clinical thinking, as well as medical ethics and deontology. Thus, the propaedeutics of internal diseases is an integral fundamental part of the clinical training of a young specialist, without which it is difficult to count on a sufficiently high-quality training of a doctor of any specialty in senior courses.
The main sections of the discipline:
- research methods: subjective (questioning), physical (examination, palpation, percussion, auscultation), laboratory, instrumental;
– symptoms and syndromes of diseases of internal organs;
- a general presentation of the main diseases of internal organs.
Tasks of the discipline:
- be able to conduct interrogation and physical examination of the patient;
– evaluate the detected symptoms, be able to explain the causes and mechanism of their occurrence;
- draw up a plan for additional laboratory and instrumental examination of the patient;
- evaluate the results of laboratory (general clinical tests of blood, urine, feces, sputum, pleural effusion, gastric and duodenal contents, biochemical blood analysis, etc.) and instrumental (electrocardiography - ECG, echocardiography, spirography and other) research methods;
- independently diagnose the main clinical syndromes;

- be able to present the results of the patient's examination in the form of a medical history with justification of the previous diagnosis and drawing up a plan for the patient's further examination;
- provide emergency care for the most common pathological conditions.

Among all research methods, subjective and objective methods are conventionally distinguished. To*subjective methods* include the examination of the patient - passport part, complaints, examination of organs and systems, medical history and life history. To*objective research methods* include: basic physical methods (examination, palpation, percussion, auscultation) and additional laboratory-instrumental methods. For additional examination methods, the concept of invasiveness is important. Invasiveness of the research method (lat.*invade, invade* –attack, invade) – the degree of violation of the integrity of the barrier between the environment and body tissues during the study. To*non-invasive*research methods include ECG, ultrasound, echocardiography, X-ray examination and conditionally endoscopy; to*invasive*research methods - angiography, biopsy and others. In view of the certain risk for the patient during invasive research, it is necessary to obtain his consent (informed consent of the patient to conduct research).

During the examination of the patient, the doctor receives certain clinical manifestations of the disease -*symptoms* (from the Greek "coincidence"), a sign of the disease (for example, cough, shortness of breath, pain, leukocytosis, hematuria, myocardial hypertrophy, etc.).*Pathognomonic symptom* – a symptom characteristic exclusively for a specific disease (crepitus in the case of lung tissue damage).

The term "syndrome" was introduced to systematize symptoms.*Syndrome* ("joint running") - a set of symptoms mutually determined by a common pathogenesis (leukocytosis, neutrophilia, a shift of the leukocyte formula to the left and an accelerated rate of erythrocyte sedimentation - a hematological inflammatory syndrome). Identifying a syndrome is an important step on the way to a correct diagnosis, since a number of syndromes are characteristic of diseases of various systems and organs.

Thus, the diagnostic process consists of three stages:
- detection of symptoms;
– selection of syndromes (syndromal diagnosis);
- self-diagnosis based on a characteristic combination of syndromes.

Methods of examination of the patient. All examination methods are conditionally divided into general clinical and special.***General clinical*** examination methods have the most important importance in the diagnosis of internal diseases, which is carried out for any disease with the aim of obtaining a holistic view of the patient and establishing or excluding the connection between damage to certain organs (for example, the organs of vision, hearing) and the pathology of other body systems.***Special***examination - methods used for the diagnosis of certain diseases, which belong to the competence of some medical specialists (ophthalmologist, urologist, dentist, etc.), which do not have diagnostic value when examining patients with other diseases. General clinical examination of the patient involves obtaining generalized information about the state of health of the examinee based on the data of the research of the nervous, endocrine, respiratory, cardiovascular, digestive, sexual, excretory and other systems.

All research methods are divided into***basic and additional***.

To**basic** historically early methods of diagnosis are included in the methods:***inquiry*** (collection of anamnesis), ***examination, palpation, percussion*** and ***auscultation.***
Scheme of medical history. The medical history is a medical and legal document that reflects all the data about the patient's illness. For the first time, the scheme of the history of the disease was proposed by the domestic clinician M.Ya. Mudrov for the systematization and complete examination of the patient according to a single plan, as well as the registration of all research results and observations of the patient during the entire period of illness. Later, the scheme of the medical history and the questioning method were improved by domestic clinicians G.A. Zakhar'in and S.P. Botkin.
They begin the questioning with the question: "what bothers you?" and give the patient the opportunity to express himself for a few minutes. Carefully listening to the patient, the doctor distinguishes the main and secondary complaints; analyzes the nature of complaints and compares them with the general condition of the patient; assesses the mental state of the patient, his consciousness, emotional state, memory; this time is also necessary for mutual study. Then, at a convenient moment of the questioning, the doctor tactfully interrupts the patient's monologue in order to clarify (detail) the identified complaints. Every complaint of the patient*is detailed*(to be clarified) for the purpose of making a preliminary diagnosis; selection of the affected conducting system (for example, pain in the chest can be a symptom of damage to the respiratory, cardiovascular, nervous, digestive, musculoskeletal or other systems); determining the sequence of inquiry by system.
Questioning about general well-being is separated into an independent section of questioning, since many serious diseases, such as tuberculosis, oncological, endocrine diseases, blood pathology and others can begin with complaints of a general nature: general weakness, fatigue, fever, skin itching, skin rash, sweating, change in body weight.
Examination of organs and systems is carried out in order to identify concomitant pathology from other systems, which is important for the diagnosis of the main disease, its complications and treatment.
The main sections of the medical history
I. Passport part.Full Name. Age. Place of residence. Place of work (name of the enterprise or institution). Position. Date of admission to the hospital. From where and how he was brought to the hospital.
II. Complaints of the patient.Identify the main and secondary complaints, and then detail each of them.
III. Questioning about general well-being.General weakness. Fatigue. Increase in temperature. Itchy skin. Rash on the skin. Sweating Change in body weight.
IV. Inquiry by organs and systems
*Central nervous system.*General performance, mood, memory, attention, sleep, headaches, dizziness, skin sensitivity, feeling of numbness, "tingles", coordination of movements, convulsions, paresis. Sight, hearing, sense of smell.
*Respiratory organs.*Runny nose, voice change. Pain in the chest connection with the phases of breathing. Dyspnea. Violent attacks. Cough. Sputum. Hemoptysis.
*Cardiovascular system.*Painful sensations in the area of the heart or behind the sternum. Dyspnea. Violent attacks. Palpitation. Interruptions Swelling
*Digestive organs.*State of appetite. Thirst. Drooling, taste in the mouth. Taste sensations, bad breath. Bleeding gums. Painful sensations in the tongue. Dysphagic phenomena. Dyspeptic

phenomena (heartburn, belching, nausea, vomiting, causes). Stomach ache. Defecation. Fasten Diarrhea. Excretion of members of helminths.
*Urinary system.*pains Urination. Urine.
*Locomotor system.*Pain in joints, bones, muscles. Movement disorders in the joints.
Notes: 1. Questioning by systems begins with the system to which the patient presents the main complaints.
2. The above-mentioned complaints are not specified during the system inquiry.
3. If new complaints are discovered during the system inquiry, they are subjected to similar detailing.

V. History of the disease

1. *The onset of the disease and its features*. When he got sick (date), the causes of the disease according to the patient, the first symptoms. Behavior of the patient at the beginning of the disease: a) did not consult a doctor (reason); b) engaged in self-medication (what treatment, dose, effectiveness); c) consulted a doctor for the first time (date). Next, indicate the place (outpatient, inpatient) and the results of the examination, the first established diagnosis (which one), the treatment carried out (which treatment, its effectiveness).
2. *Dynamics of the disease*. Step by step describe the treatment and its effectiveness, including diet and spa treatment. The appearance of new symptoms and a change or addition to the diagnosis. For persons with arterial hypertension, indicate the numbers of blood pressure: established for the first time; maximum; for patients with lesions of the gastrointestinal tract: adherence to a diet, sanatorium-resort treatment, frequency of exacerbations of the disease.
3. *Justification of hospitalization*. Indicate the date and reason for the last worsening of the patient's condition, what it was (symptoms). Have you consulted a doctor? Purpose of hospitalization (examination, treatment, establishment or change of disability group).

VI. History of life. 1. *Biographical data*: place of birth, living conditions in childhood, education, beginning of work, profession, time and place of military service, change of residence.
2. Transferred diseases: a) in childhood; b) adults; c) in wartime; d) venereal; e) gynecological; f) operations, injuries.
3. *Chronic intoxication* (smoking, alcohol, drug addiction).
4. *Family history*: marital status, family composition.
5. *Obstetric history*: menstruation, their regularity, correctness, duration, blood loss, cessation of menstruation. The number of pregnancies, births, abortions, miscarriages.
6. *Genealogical history*: the collection of anamnestic data found in the genealogy is carried out according to the scheme: the study of the proband - the person through whom the entire family is registered, most often it is the patient or the carrier of the disease being analyzed; - study of relatives (I, II, III degrees of consanguinity).
7. *Insurance history*: does he have a disability group (from which disease); frequency of use of sick leave; since what time does he have a sick leave at this time.
8. *Social and household anamnesis*: working conditions at this time; does he use vacation regularly; living conditions (number of rooms, floor, heating); characteristics of nutrition (regularity, quality of food).

9. *Allergological history*: allergic diseases in the past; reaction to blood transfusions, administration of serums, vaccines, medications; the influence of various food substances, cosmetics, and odors on the course of the disease.

VII. Objective examination of the patient.

General overview. Assessment of the patient's general condition. Position of the patient. Consciousness. Face expression. Physique, height, constitution. Skin and visible mucous membranes, elasticity, turgor, skin moisture. Subcutaneous adipose tissue. Pastiness, edema. Lymph nodes. Muscular system: development of muscles, their tone, presence of atrophy, soreness, muscle strength. Bone system – condition of limbs and spine, pain, deformation, presence of contractures. Joints: configuration, swelling, skin color over the joints, active and passive movements, soreness. Examination of the neck. Thyroid gland. Pulsation of the vessels of the neck.

Respiratory organs

Chest examination: a) form: physiological, pathological - what; b) symmetry; c) participation in the act of breathing; d) frequency, depth and rhythm of breathing; e) type of breathing; f) shortness of breath.

Palpation of the chest: a) resistance; b) soreness; c) vocal tremor.

Lung percussion. Comparative*:* a) characteristics of percussion sound; b) local changes in percussion sound (indicate the area). Topographic: a) borders of the lower edges of the lungs, b) standing height of the tops of the lungs, c) excursion of the lower edges of the lungs.

Auscultation of the lungs: basic respiratory noise; additional breathing noises.

Organs of blood circulation

Examination and palpation of the atrial region: the presence of a cardiac hump; visible apical thrust; the presence of pathological pulsation (negative apical impulse, cardiac impulse, pulsations in the II intercostal space to the right and left of the edge of the sternum, in the III and/or IV intercostal space to the left of the sternum); the presence of remote pulsation (in the epigastric region, in the right hypochondrium, jugular fossa, on the neck); characteristics of the apical shock (localization, force, height, area); atrial flutter - "cat's purr" - localization, in which phase of cardiac activity is determined.

Percussion of the heart: limits of relative dullness of the heart (right, upper, left); heart diameter; the width of the vascular bundle; configuration of the heart.

Auscultation of the heart: rhythm of cardiac activity; heart tones (loudness, timbre, bifurcation); heart murmurs (relation to phases of cardiac activity, place of maximum listening, conduct).

Pulse: symmetry on both radial arteries; rhythm; frequency; lack of pulse; filling; voltage, magnitude, speed, shape.

Blood pressure: systolic; diastolic; pulse

Digestive organs

Examination of the oral cavity (condition of teeth, tongue).

Abdominal examination: value; form; symmetry; participation in the act of breathing; prominence of subcutaneous veins; navel position; scars; traces of using a heating pad; rashes, rashes; visible peristalsis.

Abdominal palpation. Superficial palpation of the abdomen: state of muscle tone of the abdominal wall; pain; separation of rectus abdominis muscles; determination of free fluid in the abdominal cavity. determination of pain points.

Methodical deep sliding palpation of the abdomen according to Obraztsov-Strazhesko (sigmoid, blind, ascending and descending parts of the colon, transverse colon; greater curvature and portal of the stomach; liver; spleen); percussion of the liver.
Kidneys and urinary system:palpation of the kidney, Pasternacki's symptom.
Identification of syndromes and substantiation of the preliminary diagnosis.
VIII. Patient examination plan
Obligatory studies for patients with any pathology: clinical blood and urine analysis, blood and urine analysis for sugar, stool analysis for worm eggs, fluorography of chest organs, ECG.
Examination plan for patients with various pathologies internal bodies (cardiovascular, digestive, respiratory, urinary systems).
IX. Results of additional studies
1. Laboratory (name, date, results, conclusion).
2. Instrumental (name, date, results, conclusion).
3. Specialist consultations (date, conclusion).
X. Justification of the clinical diagnosis.
On the basis of clinical and laboratory-instrumental data, identify leading syndromes, formulate a clinical diagnosis according to the following scheme:*main diagnosis; complication; accompanying diseases*.
XI. Treatment plan.
XII. Observation diary.
XIII. Epicris

Review (i*nspection)* is the first and one of the main methods of objective examination of a patient for a doctor of any specialty.
Examination of the patient is the simplest and most natural method of research. With its help, you can objectively assess the actual condition of the patient (***status present objectivus*****)**. Sudden changes in the patient's appearance are often caused by pathological processes of internal organs. Pathological signs detected by the doctor during the first examination provide significant help in asking questions during questioning and sometimes make it possible to establish the correct diagnosis "at first glance" (*diagnosis to the eyes*) (for example, changes in facial features in acromegaly, thyrotoxicosis, "vascular stars" in liver cirrhosis).

General condition of the patient (status aegroti) is a collective concept that is formed in the doctor during the examination from the moment of their first meeting, taking an anamnesis and during the entire observation. The doctor makes the final conclusion about the general condition of the patient based on the data of an objective examination. The following conditions of the patient are distinguished: satisfactory, moderate, severe, extremely severe. The following indicators serve as criteria for assessing the patient's condition: consciousness; position; posture; pace; face expression; body weight; mental status.
Good condition characterized by a clear consciousness, an active position, a straight posture, a confident gait, a meaningful facial expression, a normal body weight, an adequate reaction to others and one's condition. Occurs in healthy people.sh

Satisfactory condition is characterized by clear consciousness, active or active with restriction of position, straight posture, confident or partially disturbed (specific) gait, meaningful facial expression, with normal or excess body weight, adequate mental reaction to others and one's condition. It is observed during recovery, during the period of remission in long-term chronic diseases.
Moderate condition characterized by clear consciousness, change in facial expression and position (often forced), unsteady gait, partial disturbance of mental state, especially in relation to one's condition (exaggeration or underestimation of complaints, symptoms of the disease). It is observed in the period of exacerbation of chronic or acute diseases, in case of injuries, poisoning.
Serious condition characterized by a disturbance of consciousness (dazzled, stupor, sopor), a change in facial expression (suffering, fear, indifference), patients take a passive or forced position, there is a change in body weight (weight loss or obesity), the psyche changes (inadequate reactions to the environment, medical personnel, relatives). Patients in serious condition are usually confined to bed and require external care. This condition is typical for decompensated patients with diseases of the heart, kidneys, endocrine glands and nervous system, oncological and infectious patients, as well as after operations, injuries, wounds.
*Extremely serious condition*characterized by a sharp disturbance of consciousness (sopor, coma), a passive position, an expression of indifference or suffering on the face (Hippocrates face), partial or complete lack of contact with others. It is observed in comatose states of various etiologies, shock states, agony.
Consciousness- a higher form of reflection of reality, peculiar only to man, which represents a set of psychological processes that allow orientation in the surrounding world, time, assessment of one's own personality and ensures consistency, unity and diversity of human behavior.
A clear and disturbed consciousness is distinguished.
Criteria for assessing the patient's state of consciousness:
– orientation in the surrounding environment and adequacy of behavior;
- perception of the world (nature of answers to questions, adequacy of answers, timeliness of answers);
- state of reflexes (sensory, tendon, pain) and reaction of the pupils to light (lively, sluggish, absent).
In the clinic of internal diseases, depression of consciousness has the greatest diagnostic value. Depending on the expression of suppression processes, the following degrees of impaired consciousness are distinguished: clouded consciousness, stupor, sopor, coma.
Clouded consciousness (*mental clouding* –darkened, unclear consciousness, from lat.*ob* - before*clouds* - cloud) - a state of stupor, in which the patient does not orient himself well enough in the environment, is indifferent to his condition, answers questions adequately, but with a delay. It is observed in infectious diseases, endo- and exo-intoxications.
Stupor (*stupor*, from Latin*plummet* - numbness, immobility, freezing) - a state of immobility, stupor, disorientation in the surrounding environment. The patient answers questions late or does not answer all questions, reflexes are preserved, but slightly slowed down. It is observed in cases of contusions, intoxications, poisoning with alcohol or narcotic substances, concussions, mental illnesses.

Sopor (*soap*r, from lat.*sopire* - put to sleep, stupefy, hibernation, amnesia) - a state of deep sleep, from which the patient can be brought out only for a short time by a strong stimulus (loud appeal to him, a pinch), receiving in response an unequivocal sound, movement, look, the patient "falls asleep" again ", all reflexes are preserved, but lethargic. It is observed in inflammation of the brain and meninges, severe infectious diseases (typhoid fever), severe poisoning (alcohol, narcotic substances, hypnotics), craniocerebral injuries, as well as in the initial stage of uremia and severe liver failure (hepatargia). Sopor often precedes the development of coma, so it can be considered a precomatous state.
Coma (coma, from the Greek. coma - deep sleep) - an unconscious state, deep sleep, characterized by unconsciousness and a complete loss of reactions to external stimuli and reflexes, as well as disorders of the respiratory and cardiovascular systems.
Disturbances of consciousness with a predominance of excitation processes include:**twilight consciousness, delirium (delusion), amnesia, oneiroid, delusions, hallucinations, amnesia.**
Twilight consciousness (twilight clouded consciousness) – sudden clouding of consciousness followed by amnesia – a condition in which the patient is disoriented in the environment, does not recognize relatives, doctors, sometimes excited; in the presence of threatening hallucinations (images that do not reflect reality) is capable of aggressive actions; characteristic of epilepsy and other pathological conditions.
Delirium or delirium (*delirium* - madness, delirium) - a state of disorientation of the patient in the environment in combination with vivid visual and auditory hallucinations.
Hallucinations – false sensations (visual, auditory), which arise without a corresponding external stimulus and are perceived by the patient as something real; observed in mental illnesses (schizophrenia), white fever and some infectious diseases.
Amnesia - memory impairment in case of craniocerebral injuries, atherosclerosis of cerebral vessels, poisoning.

Position of the patient

Position of the patient indicates the severity of the disease, and may also be due to the specificity of the disease. Active, passive and forced position of the patient are distinguished.
Active position is a position that the patient can change arbitrarily (walk, sit, lie down, stand), although at the same time he may experience unpleasant sensations. It is especially important to detect the ability to actively move in a lying patient, which indicates the preservation of his consciousness. However, the active position may not always be a criterion for the severity of the disease (for example, the initial stages of severe and incurable diseases).
Passive position is a position when the patient lies motionless due to sudden weakness or loss of consciousness and cannot change the position on his own, which is often extremely uncomfortable. In most cases, the passive position indicates a serious, often unconscious condition of the patient. The passive position is occupied by patients with severe infections, intoxications with damage to the central nervous system (rash, typhoid fever, typhoid fever, miliary tuberculosis, comatose states, patients in agony).
Forced position - the position taken by the patient to reduce or stop unpleasant sensations (pain, cough, shortness of breath), thereby alleviating his condition. Sometimes these conditions are so specific and characteristic that at first glance you can identify the disease (meningitis, tetanus, bronchial asthma attack).

The following types of forced position of the patient are distinguished: 1) standing; 2) sitting: with the rest of the hands; without resting hands; with the body leaning forward; squatting; 3) lying down: on the back; on the stomach; on the side (healthy or sick); 4) knee-elbow; 5) excited.

I'm walking *(entry)* - a set of features of posture and movements when walking. Individual features of the gait consist of the size of the step, the speed of walking, the position of the trunk and head, joint movements of the hands and depends on the state of the musculoskeletal system, the nervous system, as well as on the constitutional type, temperament, education, profession (the gait of soldiers, sailors, dancers). The gait of a healthy person is firm, confident, straight, and does not cause any particular tension. Depending on the state of the musculoskeletal system and its innervation, local pain sensations, damage to internal organs, a number of specific courses are observed that have symptomatic and diagnostic value.
Spastic gait *(spastic entry)* –characterized by small steps with difficulty bending the legs in the knee joints and clinging to the floor with the toes; caused by an increase in muscle tone in the case of damage to the pyramidal pathways, central lower paraparesis.
Hemiplegic gait (ingressus hemiplegicus) the mower's gait (from the Latin term - to describe a circle) is characterized by a significant deviation of the affected leg to the side, without taking the heel off the ground, as a result of which it describes a semicircle with each step, which is due to the increased tone of the extensors of the leg and the plantar flexors of the foot (leg as if lengthening); at the same time, the corresponding arm is bent at the elbow and brought to the body; observed in patients with central hemiparesis as a result of a stroke.
Paretic gait *(wall entry)* or lethargic - characterized by slow movement with difficult lifting and dragging of atonic legs; occurs as a result of paraparesis of the lower extremities.
"Rooster Walk" *(chicken entrée)* (steppage, perineal) is characterized by high lifting of the leg, throwing it forward and sharply lowering it with a slap on the floor; caused by paresis or paralysis of the muscles that extend the foot (dropped foot), and is observed when the fibular nerve is damaged.
Tactic move *(tactical entry)* (stamping, sealing, from the Greek ataktos - messy) is characterized by excessively high lifting of the legs when walking, throwing them forward; after reaching the floor, the leg continues to look for support, in order to maintain balance, patients spread their legs wide and walk with their head down, constantly monitoring the ground under their feet with their eyes. This gait is caused by a disorder of coordination and damage to the leading pathways of deep sensitivity (posterior columns of the spinal cord, peripheral nerves) and is observed in spinal tuberculosis, polyneuritis, sensitive ataxia.
Cerebellar gait *(cerebral entry)* or drunk - a type of ataxic gait, characterized by a wide spread of the legs when walking (as if spreading them), swaying of the body, balancing with raised arms; due to a violation of the coordination of the cerebellum and its connections with the cortex and subcortical structures, as well as damage to the vestibular apparatus; it is observed in the case of damage to cerebral circulation, multiple sclerosis, Meniere's syndrome, alcohol intoxication, massive blood loss, nervous stress, severe fevers.
"Puppet Walk" *(ingressus pupae), acheirokinesis (acheirokinesis s.Parkinsoni)*(similar to a mannequin, automatic doll) is characterized by quick small sliding steps without synchronous movements of the hands, which are often half bent in the elbow sockets, a frozen position of

the trunk and head (often leaning forward) with the phenomena of difficulty in the first step (propulsion) and stopping or turning (retropulsion); caused by damage to the extrapyramidal pathways, when general muscle stiffness is formed in the case of past epidemic encephalitis, parkinsonism, cerebral atherosclerosis.
Dancing gait characterized by turns or tilts of the body and head to the side, jolt-like vertical movement of the body, bizarre poses; caused by deforming muscle dystonia, slow tonic hyperkinesis of muscles of the trunk and limbs in torsion dystonia, hepatocerebral dystrophy, encephalitis and other neuroinfections, intoxications, postpartum trauma, atherosclerosis.
He made the move*(incoming entry)* (wobbly) is characterized by slow, uncertain small steps "uphill", difficult lifting of the legs, which is compensated by tilting the trunk in the opposite direction (toward the fixed leg on the ground); caused by hypotonia or atrophy of the muscles of the pelvic girdle in myopathy, subluxation of the hip joint, Recklinghausen's osteodystrophy, osteodystrophy of the hip joint, residual poliomyelitis.
I walk proudly characterized by the deviation of the upper part of the body back when walking to maintain balance during pregnancy, ascites, a massive tumor of the abdominal cavity.
Careful walking characterized by slow uncertain steps with a still body; caused by local pain in lumbago, spondylosis, arthralgias, ankylosing arthrosis.
Old, senile **the move** *(elderly entry)*characterized by small shuffling steps with uncertain and uncoordinated hand movements in patients with severe cerebral atherosclerosis.

The patient's face(*facies*, from Latin*Do* - say) provides the doctor with very valuable diagnostic and prognostic data, reveals the patient's experiences. The facial expression often reflects the mental and physical state of the patient.
Specific facial changes can be a manifestation of various diseases. For example, the discrepancy between biological and passport age: patients with rheumatism, congenital heart defects, hyperfunction of the thyroid gland may look younger, and patients with peptic ulcer disease, oncological diseases, hypofunction of the thyroid gland, on the contrary, are older than the passport age.
Inconsistency of sexual characteristics (feminine facial features in men, and vice versa) indicates endocrine disorders.
– ***face with pneumonia****(facies pneumonica)* it is characterized by a unilateral blush on the side of the inflammation (due to reflex dilation of blood vessels), a slight puffiness of the face with an elderly expression, with a running grimace when coughing (due to pain), with the “play” of the wings of the nose (due to shortness of breath), often with herpes blisters on the lips ;
– ***face with pulmonary tuberculosis****(febrile face, facies febrilis)* thin, pale, with a bright blush on the cheeks, with wide-open shiny ("burning") eyes, often with a bluish tint to the sclera, with a half-open mouth, dry lips and an excited expression;
– ***the face of an asthmatic****(facies asthmatica)* - pale with a cyanotic shade of the lips, tip of the nose and auricles; expression of general tension and desire to inhale more air; observed during an attack of bronchial asthma;
– ***"aortic face****” (aortic facies)* characterized by paleness of the skin (as a result of relative ischemia in the large circle of blood circulation - "aortic pallor"); observed in aortic defects, more often in stenosis;

– ***"mitral face"*** *(facies mitrale)* - young, bloated, with a stagnant bilateral blush of a characteristic dark red with a bluish tint (cherry color) in the form of a "mitral butterfly" and pronounced acrocyanosis; characteristic of decompensated mitral heart defects, especially with mitral stenosis.
– ***Corvisar's heart face*** *(face Corvisara, hearty face)* – the features are dull, the eyes seem to constantly water, the gaze is dull and sleepy, the complexion is a combination of yellowish pallor and bluishness, the mouth is constantly half-open, the lips are slightly protruding; characteristic of severe heart failure.
– ***plethoric****(bloated face)*a hyperemic face caused by an increase in blood mass on the periphery in hypertensive crisis patients.
– ***acromegalic face****(facies acromegalica)* characterized by the sharp development of the eyebrow arches, the disproportionately large size of the nose, lips, ears, and chin, excessive enlargement of the lower jaw, which causes the teeth to diverge; observed in acromegaly, which develops against the background of eosinophilic adenoma of the anterior lobe of the pituitary gland;
– ***base face*** *(facies basedovica)* – restless, mobile, rich in facial expressions, which quickly reddens, wet with staring, bulging (exophthalmos), unblinking, shiny eyes, which give the face an expression of frozen fright, fear, anger; characteristic of diffuse toxic goiter.
– ***myxedematous face*** *(facies myxoedemica)* – bloated, amemic, with dry pale yellow skin, devoid of the outer half of the eyebrows, with swollen eyelids, narrow eye slits. Sometimes a blush appears on a bloated and immobile face (resembles a doll's face); characteristic of a severe form of hypothyroidism (myxedema);
– ***moon-shaped cushingoid face*** *(facies selenica)* – round, moon-shaped, with a dark red blush on the cheeks, shiny skin and signs of hypertrichosis (mustache, beard in women); characteristic of Cushing's disease or Itsenko-Cushing's syndrome, as well as long-term use of glucocorticoid drugs;
– ***lupus face*** characterized by erythema in the form of a "lupus butterfly", the body of which is located on the back of the nose, and the wings are on the cheeks; specific for systemic lupus erythematosus;
– ***scleroderma face*** *(facies sclerodermica)* a mask-like, amimic face with a narrow mouth gap, with wrinkles located around it in the form of a purse; characteristic of systemic scleroderma;
– ***the face of Hippocrates*** *(peritoneal face, facies Hyppocratica)* - pale gray with a bluish, earthy shade, with sharply sharpened features and deeply sunken eyes, in which the expression of suffering froze, large drops of cold sweat on the forehead; characteristic of collaptoid conditions caused by severe purulent peritonitis, severe enterocolitis, vascular paralysis in dying patients, and is a prognostically unfavorable sign, because it often indicates imminent death.
– ***nephrotic face*** *(facies nephritica)* – bloated, pale gray in color, with swollen eyelids and narrow eye slits, disfigured beyond recognition; characteristic of nephritis, nephrosis with nephrotic syndrome.

Stature*(habitus)* (appearance, appearance, physique) - a set of external features characterizing the structure, appearance of a person (features of the structure, shape, size and

proportional ratio of individual parts of the body). The peculiarities of these external signs enable the doctor to assume the development of one or another disease.
They distinguish between correct and incorrect physique.
Correct physique characterized by the proportional ratio of individual parts of the body: trunk, head, limbs (in the absence of distortions).
Incorrect physique characterized by various distortions, deformations, disproportionate ratio of individual parts of the body, for example:
– dysplastic (habitus dysplastic) or eunuchoid physique: tall stature, long limbs, short body and small head, underdeveloped genitals and lack of secondary sexual characteristics; with features of eunuchism in men and masculinization in women;
– cushingoid physique: disproportionate, with predominant deposition of fat in the trunk, neck and relatively thin limbs ("buffalo-like physique");
– physique with chondrodystrophy: short stature, long torso, short and crooked limbs, relatively large head and massive facial features;
– physique with Marfan syndrome: tall stature, long limbs, long "spider-like" fingers, small head, heart defects and visual disturbances are characteristic of the clinical symptoms.
Human height (*the stature of a man*) depends on the size of the bone system. Height is measured using a height meter in centimeters. The normal height of a man (statura hominis normalis) at the age of 25-35 years (on average) is 168-178 centimeters, women - 157-167 centimeters. Height above 195 centimeters is considered as*gigantism (gygantismus),* and may be due to hyperfunction of the anterior lobe of the pituitary gland (secreting somatotropic hormone) or hypofunction of the gonads (eunuchoidism, hypogonadism). Height below 130 centimeters is considered as*dwarfism (nanismus)*, which may be caused by hypofunction of the anterior lobe of the pituitary gland (pituitary dwarfism) with the preservation of childlike proportions and underdevelopment of the genitals or hypofunction of the thyroid gland (hypothyroidism, myxedema).
Short stature can be the result of rickets suffered in early childhood. Very often, growth anomalies are associated with a disproportion of the trunk and limbs (chondrodystrophy). A decrease in height can be observed with tuberculosis of the spine, in the form of the formation of a hump and forward bending of the upper half of the body. Body height often decreases in old age due to skeletal atrophy.
Body weight. Body weight is measured using medical scales in kilograms.
The body mass index (BMI), which is calculated according to the formula, has a practical value in the clinic:

$$\text{BMI} = \frac{\textit{маса тіла}\ (\textit{кг})}{[\textit{зріст}\ (\textit{м})]^2}$$

Based on this indicator, the working group of WHO experts (1997) developed a classification of excess body weight and obesity in adults, according to which BMI in the range of 18.5-24.9 corresponds to normal body weight, in the range of 25.0-29.9 - excessive body weight With obesity, this indicator increases. A BMI of less than 18.5 is considered underweight. When estimating BMI, age and gender are not taken into account. BMI is not reliable for children with an unfinished period of growth, people older than 65 years, athletes and people with very developed muscles, pregnant women.

An increase in body weight can be caused by overeating, endocrine disorders (obesity), the appearance of swelling or accumulation of fluid in the cavities, recovery after an infection.

Constitutional type *(*from lat.*constitution* –establishment, organization) – a set of stable morphological and functional features of an organism, formed on the basis of hereditary and acquired factors.

Based on the morphological and functional features of the body, three constitutional types are distinguished: normosthenic, hypersthenic, asthenic, and hyper- and asthenic types are opposite in their characteristics. "Pure" constitutional type is rare, mixed is more often observed, with a predominance of hypersthenic or asthenic

Normosthenic type *(normosthenic constitution)*characterized by the proportionality of the anterior-posterior and transverse dimensions of the chest (the ratio of their lengths is 0.65-0.75), a straight epigastric angle (≈ 90°), a moderately oblique direction of the ribs, not sharply expressed intercostal spaces, supra- and subclavian fossae, shoulder blades are moderately adjacent to the chest, the sternal angle is moderately pronounced. BMI = 18.5-24.9. This constitutional type occupies an intermediate position between hypersthenic and asthenic types.

Hypersthenic type*(hypersthenic constitution)* characterized by an increase in the transverse dimensions of the chest (the ratio of anterior-posterior and transverse dimensions >0.75), epigastric angle (>90°), horizontal direction of the ribs, narrow, weakly expressed (smoothed) supraclavicular and subclavian fossae; the scapulae fit tightly to the chest, the sternal angle is well defined. These are persons of medium height, overweight, stocky with well-developed musculature. BMI >25.0.

Among the features of the internal organs, it should be noted the relative hypofunction of the thyroid gland and increased function of the gonads and adrenal glands, hence a decrease in metabolism and an increase in blood pressure, an increase in internal organs, a horizontal position of the heart, a high position of the diaphragm, increased erythropoiesis, and hypercholesterolemia.

Asthenic type *(asthenic constitution)*it is characterized by a decrease in the transverse dimensions of the chest (the ratio of anterior-posterior and transverse dimensions <0.65), the epigastric angle (<90°), a more vertical position of the ribs, wide intercostal spaces, pronounced supraclavicular and subclavian fossae, the shoulder blades stand back from the back (wing-shaped –*winged shoulder*), the sternal angle is smoothed; sometimes the X rib has a free front end (*the tenth rib fluctuating*). These individuals are distinguished by their slenderness and lightness of body structure, they are above average height, skinny with poorly developed muscular system. BMI <18.5.

Among the features of the internal organs, the relative hypofunction of the gonads and adrenal glands in combination with the hyperfunction of the thyroid gland and pituitary gland is noted, hence the increase in metabolism, a tendency to hypotension, the reduction of internal organs, the vertical position of the heart, the low position of the diaphragm.

Diagnostic value of constitutional types:

- assessment of objective research data (borders of lungs and heart, number of erythrocytes and hemoglobin), etc.;

● diagnosis of pathological processes (hypersthenics are characterized by disturbances of metabolism, reactivity, vegetative reactions; for asthenics – insufficient development of connective tissue, disorders of the digestive system).
Clinical significance:
- in hypersthenics - metabolic diseases (obesity, gout), cardiovascular diseases (atherosclerosis, ischemic heart disease, hypertension), liver diseases (gallstone disease, cholecystitis) and kidney disease (urolithiasis);
- in asthenics - diseases of the respiratory system (tuberculosis, chronic bronchitis), digestive system (ulcer disease of the stomach and duodenum), visceroptosis (prolapse of organs).
Knowledge of the main pathological processes of each constitutional type enables the doctor to prevent the development of these diseases with the help of preventive measures, to increase the body's adaptive reactions to external adverse factors.

Skin*(skin)* - an organ that is the outer covering of the body, which performs the functions of body protection, metabolism, and thermoregulation.
The main methods of skin examination are inspection and palpation. Skin examination is carried out in daylight, which provides more opportunities to detect changes in skin color. When examining the skin, you should pay attention to its features: color; the presence of skin elements (including hemorrhages, scars, ulcers); turgor and elasticity (palpation); moisture (visually and palpating), condition of derivatives (hair, nails), presence of edema.

Skin color.The color of the skin is determined by the presence of pigment and depends on the degree of development of the vascular system of the skin; filling of vessels with blood; chemical and morphological composition of blood; skin thickness; state of its innervation.
In pathological conditions, the skin can change its color, namely:
– paleness and hyperemia of the skin depends on its thickness, blood supply, innervation and can be transient in physiological conditions (fear, high or low ambient temperature);
– Jaundice, cyanosis, bronze color, gray-earthy and aspid color are usually caused by a change in the chemical composition of the blood (increase in carbon dioxide and/or bilirubin) and occur only in pathology, with the exception of physiological jaundice of infants (in the first days).
Among*causes of qualitative change* the composition of the blood is allocated:
1) blood diseases (anemia, leukemia, Werlhof's disease, etc.);
2) acute and chronic infections accompanied by hemolysis of erythrocytes (malaria, sepsis, infectious endocarditis);
3) chronic intoxications (malignant neoplasms, chronic poisoning);
Bruise or cyanosis (*cutis cyanotica, s. cyanosis*) is a bluish-purple color of the skin and mucous membranes, due to a change in the quality of the blood (excess content of carbon dioxide and reduced hemoglobin) or venous stasis.
Depending on the prevalence, cyanosis is distinguished:
– central (general, diffuse);
– peripheral (acrocyanosis);
– local.
Diffuse (general) cyanosis *(cyanosis diffusa)* can be observed in the following conditions:

- violation of gas exchange in the lungs in chronic diseases of the respiratory organs (bronchiolitis, severe pneumonia, emphysema of the lungs, pneumosclerosis, pulmonary edema, stenosis of the pulmonary artery, atelectasis of the lung, attack of bronchial asthma, thromboembolism of the pulmonary artery and its branches, foreign bodies and tumors in the bronchial tree) ;
– poisoning with hemolytic poisons (Bertolet salt, nitrobenzene) with the formation of methemoglobin;
- mixing of blood in congenital heart defects (non-union of the interventricular and interatrial membranes).

Peripheral cyanosis, acrocyanosis*(acrocyanosis)* observed with venous stasis and accumulation of reconstituted hemoglobin in the blood of patients with heart failure.
Local cyanosis (cyanosis localis) observed with compression of blood vessels or paresis of vascular nerves in patients with thrombophlebitis.
Jaundice or icterus(*jaundiced skin, s. jaundice*) of the skin and mucous membranes due to an increase in the content of bilirubin in the blood (bilirubinemia). The color of the skin depends on the degree of bilirubinemia (from light-lemon to olive-green, saffron, blackish-yellow shades). Jaundice of the skin is better detected in daylight.
Initial and slight jaundice of mucous membranes and sclera is called subicteric (*subject*), are observed in patients with heart failure, as a result of compression of the bile ducts in congested liver, as well as in severe cachexia, chronic alcoholism.
Bronze coloris an intense coloring of the skin in a dark brown color. A distinction is made between physiological and pathological (general and local) bronze coloration of the skin
Pathological bronze coloring observed in patients with bronze disease (Addison's disease), hemochromatosis - "bronze diabetes". In Addison's disease, the bronze coloration is caused by damage to the adrenal glands and the development of chronic adrenal insufficiency as a result of previous tuberculosis, leptomeningitis, and other neuroinfections.
Aspid color - the skin acquires a dark gray or smoky gray color with long-term use of silver preparations (argyrosis) or arsenic preparations, in patients with chronic malaria (melanosis).
Depigmentation (*depigmentatio*) occurs in the form of vitiligo (*vitiligo*) – symmetrical large white spots on the skin of the face, limbs, trunk, develop as a result of disorders of the function of the endocrine glands (thyroid, adrenal); leucoderma (*leukoderma*) – small white spots after boils, rashes in syphilis; albinism (*albinism*) - complete absence of pigment in the skin and its appendages.

Leather elements

In infectious diseases, allergic conditions, pathology of internal organs, specific skin elements may appear on the skin: roseola, erythema, urticaria, sweating, herpes, hemorrhages, telangiectasias, ulcers, scars, bedsores, varicose veins.
Roseoli (*roseolae*) – pale pink large spots with a diameter of 2-3 mm (the size of a pinhead, hemp seed or lentil), which rise slightly above the skin and disappear when pressed; usually caused by local inflammation and expansion of small vessels, observed:
– with typhoid fever (single on pale skin appear for 7-10 days on the lateral surface of the abdomen and the lower part of the chest);
– with typhoid fever (small, in large numbers on the hyperemic skin of the trunk and limbs, appear for 4-5 days);

– with syphilis (a large number of rashes, even on the skin of the palms and soles, which differ in paleness, in combination with other skin elements - blisters, papules; a characteristic sign of the second period of the disease);

Erythema (*erythema*) - a sharply limited large red spot that rises above the skin level, due to the expansion of blood vessels and their full blood in the case of allergic or infectious effects on the vessels, urticaria (erythema grows quickly and has clear borders, the edges rise), after consumption of certain food products (eggs, strawberries), as well as after taking some drugs (quinine, iodine, bromine) and ultraviolet radiation. In patients with systemic lupus erythematosus, erythema of the facial skin in the form of a butterfly is noted. Special forms of erythema are distinguished, in particular nodular, annular, exudative polymorphic.

Hives or hives (*urticaria*) - well-defined pale pink with a pearly hue, very itchy blisters (like a nettle burn), which rise above the skin level; due to the action of histamine, bradykinin, serotonin in allergic reactions, helminthiasis, liver diseases, diabetes.

Sweat *(crystal miles)*- small matte-white blisters made of poppy seeds, resembling dew drops; due to the delay in the secretion of sweat glands and the formation of small cysts (more often observed on the skin of the abdomen).

Blisters *(bladder)* - semicircular transparent elevations on the surface of the skin with a diameter of 0.5-1 mm (with a pinhead) with transparent or bloody contents, located more often along the course of the nerve fibers of the trigeminal nerve (on the lips - lip rash,*herpes labiallis*; wings of the nose - nasal rash,*nasal herpes*; on the wings of the nose - nasal rash, segmentally along the course of the intercostal nerves - shingles,*herpes zoster*), exist for a short time, when they crack, scabs form in their place, which then fall off; arise as a result of viral damage to the nervous system in pneumonia, influenza, malaria, paratyphoid, cerebral meningitis.

Hemorrhages or hemorrhagic purpura, hemorrhages, ecchymosis (*ecchymosis)* can be observed in the form of petechiae, bruises.

Petechiae *(petechiae)* - very small hemorrhages in the form of red dots.

Bruises *(hemophorma)* - red spots of different sizes, shapes, localization, which persist when pressed and change color in the process of evolution from red-violet to yellow-green and pale.

The main causes of hemorrhages:

– mechanical damage to skin vessels (injuries, bruises, bites);
– blood diseases (hemophilia, Werlhof's disease, acute leukemias, B-12 deficiency anemia, capillarotoxicosis, coagulopathy);
– liver disease (cirrhosis, echinococcosis);
– hypo- and vitamin deficiency (vitamins C and K);
– infectious diseases accompanied by capillarotoxicosis (typhoid, botulism, infectious endocarditis, meningitis, toxic and allergic lesions of small vessels - vasculitis).

Telangiectasias (*teleangioectasia*, from the Greek*telos* - edge or*a lot* - far,*angio* - vessel,*ektasis* - expansion) or "vascular stars" (*astra vascularia incutes*) are dark red spots with a diameter of 2-10 mm on the skin and mucous membrane, disappear when pressed, due to the expansion of small vessels under the influence of excess estrogen due to damage to the liver parenchyma (cirrhosis).

Bed-sore *(decubitus)* – necrosis of soft tissues (initially reddening of the skin, later the formation of a deep ulcer), which occurs as a result of impaired blood circulation and

trophism of the skin during long-term mechanical compression of tissues; observed in seriously ill patients who have been in bed for a long time.
Skin scars (cicatrix) - dense formations consisting of connective tissue rich in collagen fibers, which arose as a result of reparative regeneration during the inflammatory process, which testify to previous injuries, burns, operations, infections (smallpox, tuberculosis, syphilis).
Desquamation *(desquamation)*- peeling of the skin, separation of pink scales of the epidermis from the surface of the skin.
Node *(node)*- the primary morphological element of a skin rash, which is an infiltrate that develops in the deep layers of the dermis and subcutaneous tissue.
Livedo(*livedo* - bruises) - a pathological condition of the skin of a bluish-purple or bluish color due to a mesh or tree-like pattern of vessels that shine through the skin, with passive hyperemia. The following stages of livedo are distinguished: "marble skin" (*marbled skin*), mesh livedo (*livedo reticularis*), "tree-like livedo" (*livedo racemosa*). The most common cause of livedo is collagenoses (systemic lupus erythematosus, nodular periarteritis, scleroderma, dermatomyositis) and infectious diseases (tuberculosis, malaria, dysentery). The expansion of the subcutaneous veins on the anterior abdominal wall forms a peculiar pattern, which received the name*"jellyfish head"*(*Medusa's head*); due to the formation of cava-caval anastamoses with increased pressure and stagnant phenomena in the portal vein (*current gate*) when it is blocked by a thrombus or compressed from the outside; observed in patients with portal cirrhosis of the liver, heart failure (blood stagnation in the large circulatory system).

Skin turgor (*turgor*) is the degree of tissue tension, due to the state of innervation, blood supply and metabolism. Elasticity is the flexibility of the skin.
Normal - the skin is elastic, turgor is preserved, i.e. the skin fold is elastic, dense, quickly straightens
Methods of determining turgor and elasticity:
- visual: the roundness of contours and rounded body shapes, especially facial features, is visually assessed;
- palpatory: two fingers collect the skin in a fold, usually on the back surface of the hand or forearm, slightly lift and release.
Normally, the skin fold is elastic, dense, and straightens quickly - the skin is elastic with preserved turgor, with a decrease in elasticity, the skin fold straightens weakly and slowly.
Skin moisture. Normally, the skin has a certain degree of moisture, due to the release of small droplets of sweat, the secretion of sebaceous and sweat glands.
Increased humidity (sweating) or dryness indicates a violation of thermoregulation and secretion of sweat glands, as a result of innervation disorders or intoxication.

Swelling (*edema*, from Latin*howl* - tumor) - excessive accumulation of fluid in body tissues.
Swelling can occur due to:
1) increased permeability of the vascular wall (allergic, inflammatory, toxic);
2) increased intravascular pressure as a result of stagnation and retention of fluid in the body (congestive, lymphatic, renal);
3) reduction of oncotic pressure (cachectic, renal);

4) a combination of the three causes listed above (traumatic, cachectic);
5) hypothyroidism (myxedematous).
Depending on the etiology, the following types of edema are distinguished:
– stagnant (*congestive edema*) – swelling caused by stagnation of venous blood and lymph; observed in heart failure, compression of blood and lymphatic vessels by a tumor, enlarged lymph nodes, scars, blood clots.
– inflammatory - swelling caused by increased vascular permeability under the influence of inflammatory mediators; observed in rheumatism, rheumatoid arthritis, pyelonephritis, abscesses, etc.;
- renal (*renal edema*) – swelling caused by a decrease in oncotic pressure due to proteinuria and excess water retention in the body; observed in kidney diseases (acute nephritis, glomerulonephritis);
– cachectic, proteinaceous (*oedema cachecticum*) – swelling caused by a decrease in oncotic pressure, a violation of the permeability of the vascular wall; observed in extremely exhausted cancer patients;
– angioneurotic (*angioneurotic edema*), Quincke's edema - edema caused by allergic reactions; local manifestation of allergic edema can be observed with insect bites;
– toxic (oedema toxicum) – swelling caused by the influence of toxic substances on the body that increase the permeability of the vascular wall; observed in poisoning;
– traumatic (*oedema thraumaticum)* - edema caused by stagnation of blood and lymph, increased permeability of blood vessels and impaired metabolism in places of mechanical tissue damage;
– hypothyroid (*hypothyroid edema*) – swellings caused by excessive accumulation of mucin-like substances in the subcutaneous fatty tissue with hypofunction of the thyroid gland;
– oncotic (*oncotic edema*) – swelling caused by a decrease in oncotic blood pressure in hypoproteinemia;

Evaluation of the development of subcutaneous adipose tissue (PJK) is performed by measuring the thickness of the skin fold (SFT) above Traube's space (along the left midclavicular line 2 centimeters below the left costal arch), as well as by assessing the nature of its distribution.
Adiposity (obesity) - excess deposition of fat in cells and tissues (mainly in the gastrointestinal tract), caused by metabolic disorders.
There are general (generalized) obesity - alimentary obesity; and local with fat deposits in specific places and tissues - Itsenko-Cushing's disease ("buffalo" type of obesity), adiposogenital obesity.
As one of the WHO criteria, the body mass index (BMI) was proposed, which in the range of 18.5-24.9 corresponds to normal body weight, with obesity, this indicator increases accordingly: 25.0-29.9 (obesity or excessive body weight), 30.0-34.9 (obesity I degree), 35.0-39.9 (obesity II degree), >40 (obesity III degree). A BMI < 18.5 is considered to be underweight.
Emaciation*(skinny)*- a decrease in body weight due to a sharp decrease in dietary fiber, which can be due to the following reasons:

1) exogenous: starvation, improper nutrition, chronic diseases of the gastrointestinal tract, dehydration; sharp weight loss up to cachexia (severe infections, intoxications, tuberculosis, cancer, mental illnesses);
2) endogenous: damage to the pituitary gland (Simmonds' disease), thyroid gland (Based's disease), pancreas (diabetes mellitus), adrenal glands (Addison's disease).

Lymphatic system (*the lymphatic system*)
Assessment of the state of lymph nodes is carried out according to the following criteria: localization; size (from a pea to an apple, a fist); density (soft and elastic; dense and hard); soreness (painful or painless); surface (smooth or uneven); symmetry (symmetric and asymmetric); adhesion between themselves, with subordinate tissues and skin (mobile and immobile); changes in the skin over the lymph nodes (rash, blisters, ulcers, fistulas, hyperemia); prevalence (local and generalized).
Diagnostic criteria for pathologically altered lymph nodes:
1) inflammatory – lymph nodes of different sizes, soft, elastic, painful with a smooth surface, their symmetry is variable. The nodes are not connected to the skin (mobile), the skin above them may be hyperemic, they appear acutely and suddenly. Suppuration is possible with the involvement of the surrounding tissues in the inflammatory process (periadenitis). Regional lymphadenitis, caused by inflammation in the lower part of the limb, reveals a strip of hyperemia on the skin, going from the site of inflammation to the node in the projection of the inflammatory lymphatic vessel (lymphangoitis);
2) with blood diseases – a generalized significant increase in lymph nodes, they are elastic, painless, smooth, symmetrical, not joined to each other and the skin, mobile, without changes in the skin above them;
3) metastatic – hard, dense, painless, bumpy, asymmetrical, fused to each other and/or with unchanged skin above them, increasing gradually;
EXAMINATION OF CERTAIN PARTS OF THE BODY. Conducting a general examination of the patient allows the doctor to form a general idea of the severity of the patient's condition, his psyche, physique, properties of the skin, lymphatic, bone and muscle systems. Continuing the general examination, pay attention to individual parts of the body, namely the head, neck and limbs.
Head*(head).* When examining the head, pay attention to its size, shape, position, movement; and also on the eyes, nose, mouth, tongue, teeth, tonsils. Changes in the shape and size of the head are important for the diagnosis of some diseases:
Changing the size of the head:
- head enlargement (macrocephaly - macrocephalia) is observed with hydrocephalus (hydrocephaly - hydrocephlia);
- uneven enlargement of individual parts of the head (lower jaw, zygomatic and superbrow arches) is characteristic of acromegaly;
- the increase of the skull can be due to the thickening of the cranial bones in deforming osteitis, fibrinous osteodystrophy;
- a decrease in the size of the head (microcephaly) is a symptom of degeneration and is associated with congenital idiocy.
eyes Eye examination is available to every doctor and is necessary in the diagnostic practice of a therapist. Subject to examination: eyelids, eyeball, cornea, pupils.

Eyelids:
- swelling and pigmentation of the eyelids occurs in dermatomyositis;
- puffiness and swelling of the eyelids is observed in glomerulonephritis, trichinellosis, alimentary dystrophy, Based's disease, as well as in some healthy people with overtiredness;
- dark eyelids - with Addison's disease and thyrotoxicosis, tuberculosis;
- swelling of the lower eyelids ("bags under the eyes") is observed in glomerulonephritis, anemia, insomnia, paroxysmal cough;
- drooping of the eyelids (ptosis) is observed in hemiparesis, syphilis, botulism;
- unilateral ptosis in combination with sunken eyeballs (enophthalmia), narrowing of the pupils (miosis) - Claude-Bernard-Horner syndrome - is observed when the cervical part of the sympathetic trunk is affected;
- local deposition of cholesterol on the eyelids in the form of yellow spots (xanthoma) is observed in atherosclerosis, gallstone disease, and liver diseases.

The width of the eye slits due to the position of the eyeballs and the condition of the eyelids:
a) narrowing of the eye slits can be observed in glomerulonephritis, Quincke's edema, myxedema, peritonitis;
b) widening of the eye slits - in case of Based's disease, retrobulbar abscess (unilateral).
c) asymmetry of the eye slits can be a consequence of unilateral ptosis, eno- or exophthalmos with the corresponding pathology.

The movements of the eyeballs are normally synchronous, arbitrarily performed in all directions (horizontal, vertical, circular) and are carried out by the coordinated work of 6 pairs of eye muscles.

Violation of the function of one of the pairs of eye muscles, more often as a result of paresis or paralysis, leads to the development of strabismus (*strabismus*) – deviation of the visual line of one eye from the common fixation point.

Another type of eye movement disorder is nystagmus (*nystagmus*, from the Greek*nystagmus* - dozing) - involuntary rhythmic biphasic (fast and slow) movements of the eyeballs. There is a distinction between congenital nystagmus (a sex-linked recessive trait) and acquired nystagmus caused by damage to the labyrinth apparatus and the development of general muscle weakness; observed in multiple sclerosis and in exhausted patients.

Glare of the eyes can often be observed in patients with fever, with Based's disease, in an excited state. Dullness of the eyes is noted in alimentary dystrophy and avitaminosis A.

Coloration of the conjunctiva and sclera:
- blue sclera can be observed with congenital pathology;
- icteric sclera - with jaundice;
- red "rabbit eyes" caused by damage to the conjunctiva in typhoid fever;
– hemorrhages in the conjunctiva are observed in scurvy, epilepsy, subacute infective endocarditis;
- single brown spots on the conjunctiva in Addison's disease.

Cornea normally transparent; in pathology, opacities and scars can be found in congenital syphilis, parenchymal keratitis, lipoid arc in elderly people. Senile arc (*old bow*) - a whitish-gray ring 1-2 mm wide around the circumference of the cornea - one of the typical symptoms of aging.

Pupils: size, shape, pulsation, accommodation, reaction to light are evaluated.

Myosis (myosis) - narrowing of the pupils caused by irritation of the IV pair of cranial nerves or paresis of sympathetic nerves; observed in hemorrhagic stroke, uremia, intracranial bleeding, tumors and inflammatory processes of the brain, typhus, spinal tuberculosis, chronic poisoning (nicotine, chloroform), narcosis, "point pupil" in morphinists.
Mydriasis (mydriasis) - dilation of pupils caused by paresis of IV pair of cranial nerves and irritation of sympathetic nerves; observed in comatose states (with the exception of uremic and apoplectic comas); unilateral mydriasis in syphilis, sometimes in aortic aneurysm, anemia, helminthiasis.
Anisocoria - uneven expansion of the pupils; observed in Horner's syndrome, syphilis, migraine.
Pupil pulsation (pupillae pulsante), which occurs synchronously with the work of the heart - Landolfi's symptom, characteristic of severe aortic insufficiency, can sometimes accompany pathological Cheyne-Stokes breathing.
*Reaction of pupils to light.*The patient's eyes are covered with a hand for a few seconds, and then, taking the hand away, they monitor the reaction of the pupil. If the pupil narrows when light enters the eye, the reaction of the pupil to light is preserved, and vice versa. The reaction of the pupils to light is used to diagnose damage to the nervous system: lack of reaction of the pupils to light is a sign of syphilis of the nervous system, comatose states, poisoning with morphine, chloroform, atropine.
*Reaction of pupils to accommodation.*The patient is offered to fix his gaze on the tip of the finger, which is alternately brought closer and further away from his eyes. With a preserved reaction to accommodation, when the finger approaches, the pupil narrows, and when it is removed, it expands. Absence of pupil response to accommodation (accommodation paralysis) is observed in atropine poisoning, some eye and brain diseases.
Spinal tuberculosis (neurosyphilis) is characterized by a lack of reaction to light with a preserved reaction of the pupil to accommodation (Argil-Robertson symptom).
Nose*(the nose).* When examining the nose, you can get valuable diagnostic data:
– pointed nose – with peritonitis (Hippocrates face);
– a large, thick, “fleshy” nose is an initial sign of acromegaly, less often – myxedema;
– saddle-shaped nose (*saddle-shaped nose*), due to the deformation of the external nose, with the presence of a depression in the middle part of the back as a result of underdevelopment or destruction of the cartilages of the nose and nasal bones, is observed in injuries, Wegener's granulomatosis, and is a sign of tertiary syphilis;
– cone-shaped nose (*rhinophyma*) observed in chronic inflammation of the skin of the nose and cheeks with the development of infiltration with red nodules and telangiectasias; observed in cutaneous tuberculosis, systemic lupus erythematosus, leprosy;
– a red nose can be a sign of reduced tone of skin vessels, observed in chronic alcoholism and neurasthenia;
– cyanosis of the tip of the nose (*asrosuanos*) with heart failure;
– herpetic rash (*nasal herpes*) observed in flu, pneumonia;
– nosebleeds can be observed with arterial hypertension, uremia, acute leukemia;
– the participation of the wings of the nose in the act of breathing is an objective sign of severe shortness of breath;
- cleft nose (the presence of a gap along the line of the back) "Dogue nose" - a congenital anomaly of development.

Mouth. When examining the mouth, pay attention to its position, dimensions, symmetry of the corners, evaluate the shape of the lips, rashes, changes in the mucous membranes of the oral cavity. They also evaluate the condition of the gums, teeth, and tonsils, pay special attention to the examination of the tongue, and evaluate the smell of the mouth.
The oral cavity is normally limited by the lips, its corners are symmetrical, the dimensions correspond to the distance between the pupils.
An increase in the size of the mouth (macrostomia -*macrostomia*) is more often observed in congenital pathology, and a decrease (microstomia -*microstomia*) can be a manifestation of both hereditary and acquired pathology ("pouch mouth" in scleroderma and hypoparathyroidism).
Asymmetry of the corners of the mouth is observed in local manifestations (inflammatory process), as well as in paresis or paralysis of the facial and/or trigeminal nerves, stroke (with unilateral smoothing of the nasolabial fold).
A constantly half-open mouth and the inability to close it completely occurs with adenoid growth in children (due to difficulty in nasal breathing), paresis of the facial nerve, constant shortness of breath, acromegaly, Down's disease, congenital hypothyroidism (due to a sharp increase in the tongue). On the contrary, tight lips, convulsively closed mouth are observed in chorea, tetanus, eclampsia, epilepsy, acute poisoning, rabies, hysteria. "Sardonic smile" is observed in tetanus, hypoparathyroidism and some mental diseases.
Lips (labium). When examining the lips, pay attention to their size, color, rashes, scars. Enlargement of the lips can be observed with local inflammatory processes, acromegaly, and hypothyroidism. Lip color change: acrocyanosis (*acrosyanosis*) of the lips is observed in heart failure, hyperemia (redness) - in inflammation, fevers, pallor - in oncological diseases, anemia.
Rash on the lips (*cold sores*) is often observed with flu, pneumonia. The presence of ulcers*(ulcus*) and scars (cicatrix) indicate previous syphilis or lip cancer. Ulcers in the corners of the mouth (sores) are observed with vitamin B deficiency$_{2.}$
With a congenital pathology, lip deformation can be observed: "hare lip" (*the hare's mouth s.lips split*), "wolf's mouth".

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:
1. **Organizational measures** (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. **Control of the reference level of knowledge** (checking workbooks, communication with the patient in order to master the method of collecting complaints and history, general and local examination, conducting test control, solving a clinical problem, written solution of Step-2 type problems (10 problems), face-to-face interview, discussion, role play with lesson topics
2.1. Requirements for the theoretical readiness of applicants to perform practical classes: the applicant must know the purpose and tasks of the subject, the methods of examining the patient and the scheme of the medical history,

have an idea of the role of domestic scientists in the development of these methods; to know the general method of questioning patients;
to know the main sections of the medical history and their significance in the diagnostic process;
to know the main elements of a general examination of the patient;
know the purpose and master the method of local examination of the patient.
List of didactic units:
- collect in detail complaints with their details and anamnesis of illness and life, as well as conduct a survey of the patient's organ systems;
- conduct a physical examination of the patient (assess the patient's general condition, consciousness, position, constitution, condition of the skin and mucous membranes, the condition of the lymph nodes, the presence of edema), identify and evaluate the changes that were found.

2.2. **Questions to check basic knowledge on the topic of the lesson:**
question:
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".

A problem of the STEP-2 type.
1. The patient is 50 years old. He has been suffering from bronchial asthma for 8 years. He came to the clinic with complaints of attacks of dyspnoea with expiratory shortness of breath, which are repeated 2 times a day, headache, chest pain, sweating, and rapid fatigue. Select a secondary complaint.
A. Violent attacks
B. Expiratory shortness of breath
C. Cough
D. Chest pain
E. **Sweating**

3. Formation of professional skills and abilities:

Mastering communication skills (collecting complaints, detailing complaints, collecting medical and life anamnesis, interviewing organ systems, evaluating the results of the interview with the assumption of the localization of the pathological process)
Formation of the ability to conduct a general examination of the patient (the applicant must be able to conduct a general examination with an assessment of the patient's general condition, state of consciousness, position and constitution of the patient, assess the condition of the skin and mucous membranes, lymph nodes),
Formation of the ability to give a clinical assessment of the data obtained during the collection of anamnesis and general examination; carry out a clinical interpretation of the identified symptoms and syndromes.

3.1. Control materials for the final stage of the lesson:
Situational tasks:
Task 1. Disturbance of consciousness, characterized by difficult contact with the patient, unambiguous answers only after a strong stimulus, partial inhibition of reflexes, is called:
A. Stupor
B. **Sopor**
C. Coma
D. Delirium
E. Dusk

Task 2. What heart disease is characterized by pulsation of the carotid arteries ("carotid dance"):
A. Mitral insufficiency
B. **Aortic insufficiency**
C. Insufficiency of the tricuspid valve
D. Aortic stenosis
E. Mitral stenosis

Answers:
1. Such disturbances of consciousness are characteristic of sopor.
2. Pulsation of the carotid arteries (the so-called "carotid dance") is a pathognomonic symptom of aortic valve insufficiency.

Tasks of the STEP-2 type
1. What does the examination of the patient begin with:
+A. From questioning.
B. From an objective examination.
V. From instrumental examination.
G. From a laboratory examination.
D. From any of the listed items.

2. Procedure for questioning the patient:
A. Passport data, patient's complaints, current medical history, patient's life history.

+B. Complaints the patient anamnesis life the patient anamnesis current illness.
V. Complaints the patient anamnesis life the patient anamnesis current illness, passport data.
D. Passport data, results of objective examinations, complaints of the patient, anamnesis of the current illness, anamnesis of the patient's life.
D. The order of questioning does not matter.

Life anamnesis is collected according to the plan:
A. General biographical data, conditions of growth and development of the patient.
B. Living and working conditions, nutrition, transferred in the past disease.
B. Harmful habits, marital status.
D. Hereditary and allergic history.
+D. All of the above is listed.

The presence of which diseases in the patient must be clarified?
A. Tuberculosis.
B. Venereal diseases.
B. Viral hepatitis.
G. AIDS.
+D. All of the above is listed.

Active position of the patient in bed:
+A. The patient's position in bed, which he can easily change if necessary or at his own will;
B. Position in bed, which the patient cannot change;
S. The position of sitting on the bed with the legs down;
D. Knee-elbow position of the patient in bed.

Passive position of the patient in bed:
A. Position in bed on the left side;
B. The position of sitting on the bed with the legs down;
+S. The position in bed, which the patient cannot change himself;
D. The position in bed, which the patient acquired himself, to ease his condition.

3.2. Requirements for work results, including before registration: substantiation of the syndromic diagnosis based on complaints, medical history and life data, clinical examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the subject of the lesson, answering 10 tests (*if necessary*).

4. Summary, announcement of assessment results, announcement of the topic of the

next lesson.

List of recommended literature

Basic:

1. Propedeutics of internal medicine: textbook / Yu.I. Decyk, O.H. Yavorskyi, E.M. Neiko and others; under the editorship O.H. Yavorsky – 6th ed., corr. and added - K.: VSV "Medicine", 2020. - 552 p. + 12 p. color incl.
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.

Additional:

1. Methodology of examination of a therapeutic patient: teaching. manual / S.M. Andreychyn, N.A. Bilkevich, T.Yu. Chernets. – Ternopil: TDMU, 2016. – 260 p.
2. 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/

Practical lesson No. 2

Subject: Complaints of patients with respiratory diseases. Examination of the chest. Palpation of the chest.

Goal: Acquisitionacquirer knowledge and mastery of professional competencies during questioning and physical examination of patients with respiratory system pathology (questioning, collection of medical and life anamnesis, static and dynamic examination of the

chest, palpation of the chest).

Basic concepts: The method of questioning and detailing the complaints of a patient with a pathology of the respiratory system. Peculiarities of collecting medical and life anamnesis. Changes in the appearance of the patient with various pathologies of the respiratory system. Sequence of chest examination (shape, size, symmetry, condition of skin and mucous membranes, determination of respiratory rate). The concept of topographic zones on the surface of the chest. Purpose and method of chest palpation. The method of assessment of voice tremor.

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.
Main complaints patients with a pulmonological profile: shortness of breath, wheezing, cough, chest pain, expectoration, symptoms of intoxication.
Shortness of breath is the most frequent complaint in patients suffering from diseases of the respiratory system, as well as in patients with diseases of the cardiovascular system. It is manifested by a change in the frequency, rhythm and depth of breathing, a subjective feeling of dissatisfaction with inhalation or difficult exhalation, a feeling of lack of air or excessive filling of the lungs with it. With shortness of breath, xvopi may experience a predominant difficulty in breathing in (inspiratory shortness of breath, Latin inspiratio — breath) or exhaling (expiratory shortness of breath, in Latin expiratio — exhalation). In patients with chronic diseases of the bronchopulmonary apparatus, shortness of breath is more often expiratory in nature, that is, it is manifested mostly by difficult exhalation. In patients with a pulmonological (from Latin pulmon — lung and Greek logos — teaching) profile, it can be periodic, constant, or manifest as bouts of dyspnea.
Quite often, respiratory diseases cause a cough. Cough is a protective reflex act, which is a shock-like forced exhalation when the glottis is closed. When coughing, the respiratory tract is freed from extraneous impurities of sputum, dust, exfoliated epithelium of the respiratory tract, etc. Cough can be dry (without sputum production) and wet (with sputum production). Sometimes the sputum contains streaks of blood (hemoptysis) or is completely stained with blood (with pulmonary bleeding). In case of severe violations of pulmonary blood circulation (cardiac asthma, pulmonary edema), wheezing becomes bloody and foamy. Depending on the features of the pathological process, serous, purulent, mucous or glassy sputum may be released during coughing. It can expectorate easily or with difficulty, accompanied by pain or a burning sensation behind the sternum.
Chest pain in patients with respiratory diseases often worsens when coughing and breathing. For the most part, they are caused by involvement in the pathological process of the parietal and especially the visceral pleura, on the surface of which there are a large number of pain receptors. Pain can occur as a result of inflammation of the intercostal nerves, as well as when irritated by toxic or toxic-allergic factors.
For additional or general complaints patients include: fever, sweating, general weakness, increased fatigue, irritability, decreased appetite, and others. These complaints do not allow to localize the pathological process (that is why they are general), but they significantly complement the picture of lung disease and characterize the severity of the patient's condition, reflecting infectious-inflammatory and toxic processes in the body, and also limit

work capacity.
Medical history. When examining patients with pathology or suspicion of damage to the bronchopulmonary system, special attention should be paid to the anamnesis of the disease. Important information about the causes and provoking factors of the occurrence and dynamics of the disease, features of the onset, course and relapses. So, in the presence of acute lung diseases, such general symptoms as malaise, chills, fever can appear a few days before pulmonary symptoms (viral pneumonia) or almost simultaneously with them (bacterial pneumonia), and acute shortness of breath is a very important sign of bronchial asthma , acute respiratory failure and pneumothorax. A sudden onset with chills and a rise in temperature to high numbers (40 °C), pain in the side of the chest during coughing with the release of "rusty sputum" is characteristic of pneumonia. A gradual onset with a moderate increase in temperature and increasing shortness of breath indicate the possibility of effusion pleurisy. It is important to find out the circumstances that prevent or accompany the onset of the disease: contact with a sick flu (viral pneumonia), severe hypothermia (pneumonia), contact with a patient with an open form of tuberculosis (early forms of tuberculosis). Information on the implementation of medical and health and preventive measures (medicinal, surgical, physiotherapeutic, sanitary and spa treatment) and their effectiveness. These data play an important role in diagnosis based on the results of treatment (diagnosis exjuvantibus), determination of individual complex therapy with the aim of previously used means and methods.
History of life. Life anamnesis, or as it is called the "medical biography" of the patient, is extremely important not only for the diagnosis of the disease, but also for identifying the individual characteristics (acquired and hereditary) of this patient for the detailed clarification of the effect on the body of various conditions of the external environment (work , everyday life), as well as receiving information about previously suffered diseases, the presence of harmful habits. Thus, adverse conditions of development in childhood, early start of work (including in hazardous production conditions), frequent colds (acute respiratory infection, sore throat, bronchitis, pneumonia) later lead to exhaustion of the immune system, a decrease in the reactivity of the body, and how a consequence of the development of chronic bronchitis, bronchial asthma, emphysema of the lungs, tuberculosis. Previously suffered injuries of the chest lead to its deformation, osteomyelitis, and with pronounced deformation - to pulmonary and heart failure.

During the inspection breathing control is carried out in patients with lung and bronchial diseases. Breathing is a complex biological process that ensures gas exchange between the body and the environment. The act of breathing is carried out with the help of the bronchopulmonary apparatus. Oxygen enters the body during inhalation, and carbonic acid is released during exhalation.
At rest, a middle-aged adult breathes rhythmically, of moderate depth, with a frequency of 16 to 20 in 1 minute. In patients with diseases of the upper respiratory tract and lungs, the nature of breathing may change — the frequency, rhythm, and depth of breathing are disturbed. Breathing can become more frequent with fever, in the stage of exacerbation of acute and chronic inflammatory lung diseases, heart failure. In these cases, the depth of breathing also decreases, which often becomes shallow. When the respiratory center is suppressed, the breathing rhythm may be disturbed.

Determination of the frequency of breathing is carried out imperceptibly for the patient, because fixing his attention on the act of research can reflexively affect the frequency of respiratory pyxs. Therefore, for an objective assessment of breathing, it is necessary to divert the patient's attention from the procedure. 3, the following method can be used for this purpose. With one hand, the patient's wrist is taken ostensibly to count the pulse, and the other is placed on the middle part of the chest or in the area under the sternum, and the number of respiratory movements is counted for 1 minute (or in 30 seconds) based on its movements.
In addition to the frequency, the rhythm and depth of breathing are determined. The rhythm is determined by the ratio of the intervals between individual respiratory acts. Normally, breathing is rhythmic, that is, the distance between each respiratory act (inhalation and exhalation) is the same. With some diseases, including the respiratory system, the rhythm of breathing can be disturbed. Changes in the rhythm of breathing often indicate profound violations of the central mechanisms of respiratory regulation, in particular, the respiratory center.
The depth of breathing is understood as the amplitude of respiratory movements. Breathing of a healthy adult in conditions of physiological rest is defined as moderate (average) depth. The average depth of breathing is manifested by a uniform ratio of inhalation and exhalation values, as well as a moderate amplitude of chest movements. Breathing can be deep in comatose states, some poisonings. Under physiological conditions, deep breathing can also occur during physical and emotional stress, when there is insufficient oxygen in the surrounding air (hypoxia), and when there is excessive accumulation of carbon dioxide in the venous blood (hypercapnia). A decrease in the depth of breathing appears with obstructive and spastic processes in the bronchi, inflammatory diseases of the lungs, a decrease in the elasticity of the pulmonary alveoli, with sharp pains in the chest, air penetration into the pleural cavity (pneumothorax), or its filling with stagnation (hydrothorax) and inflammation (exudate) liquid. A significant decrease in the depth and appearance of shallow breathing is indicated by a decrease in the amplitude of the respiratory pyxis of the chest. When the central nervous system is damaged, breathing becomes arrhythmic: individual breathing movements of different depths occur more often, then less often. Sometimes arrhythmic breathing, due to a certain number of respiratory movements, is accompanied by a lengthening of the pause or a short-term delay in breathing (apnea). Such breathing is called periodic. Arrhythmic types of breathing include: Cheyne-Stokes breathing, Biot's breathing, Grocco's breathing.

The examination of the chest is also carried out in order to assess its shape, size, symmetry and the nature of respiratory movements.
The research is carried out in the position of the patient lying down or sitting with the torso exposed to the waist under uniform (direct and lateral) lighting, in two states: with calm breathing (static examination) and with deep breathing (dynamic examination).
The chest is examined in a strictly defined sequence with an assessment of the following indicators:
- shape and size of the chest;
- its symmetry;
- participation of auxiliary muscles in the act of breathing;

- assessment of external breathing (type of breathing, frequency, depth, rhythm).
Pathological forms of the chest. The formation of pathological forms of the chest can be due to two groups of reasons: damage to the lungs and pleura; pathology of the development of the chest (congenital pathology of the development of the skeleton, including the spine).
1. Pathological forms of the chest caused by damage to the lungs and pleura: emphysematous and paralytic.
2. Pathological forms of the chest, due to changes in the skeleton of the chest: rachitic, funnel-shaped, boat-shaped (pathology of the sternum, ribs); kyphotic, lordotic, kyphoscoliotic, scoliotic (spine pathologies).
A dynamic examination of the chest with the use of deep breathing makes it possible to assess the participation of the chest in the act of breathing. Deformation of one half of the chest is accompanied by a violation of the act of breathing: it either lags behind in the act of breathing, or does not participate in the act of breathing at all.

Palpation chest is carried out in order to determine its elasticity, assess vocal tremor, detect soreness, deformation, pleural friction noise, subcutaneous emphysema in the following sequence:
determination of chest elasticity;
detection of chest pain;
evaluation of voice tremor.

The elasticity (resistance) of the chest is determined by compressing the chest in symmetrical areas in the anteroposterior and lateral directions. In a healthy person, the chest is elastic and pliable.
Voice tremor(fremitus vocalis s.peceoralis) – a tremor of the chest caused by the transmission of low-frequency vibrations of the vocal cords to its surface at the moment of pronouncing words containing the letter "p", which causes the greatest vibration. Voice tremor is determined by palpation in symmetrical areas of the chest. The physical basis of voice tremor lies in the ability of tissues of different densities to conduct sound vibrations in different ways. The nature of voice tremors (power of sound vibrations) makes it possible to analyze changes in the state of lung tissue as a result of various pathological processes.
The purpose of determining voice tremor: to assess the strength and symmetry of sound vibrations on the surface of the chest to detect pathological processes, with a possible assessment of the nature of the process, its localization, dynamics and course.
With pathology of the chest organs, including the respiratory organs, vocal tremor can be increased, weakened, or sometimes absent.
The main causes of increased voice tremors:
- compaction of lung tissue (due to better sound conduction);
- the presence of a cavity in the lung connected to a large bronchus (amplification is due to the resonance of vibrations in the cavity and better conduction of sound through the dense tissue that surrounds the cavity).
The main causes of weakening of the voice tremor:
a) violation of the vibration of the vocal cords and damage to the vocal cords;
- weakening of the respiratory excursion of the chest, because the sound is formed during exhalation (massive pleural adhesions, myositis, neuralgia, rib fractures);

- serious condition of patients;
b) violation of sound conduction:
- increased air permeability of the lungs (pulmonary emphysema);
– a moderate amount of fluid or air in the pleural cavity (hydro-, pneumothorax);
- thickening of the chest (due to muscles, subcutaneous fat, edema);
– obturational atelectasis (local).
The main reasons for the absence of voice tremor (Table 2.18):
a) the presence of a large amount of liquid or air in the pleural cavity (hydro-, pneumothorax);
b) obturation of the main bronchus (by a foreign body, a tumor or its compression by enlarged lymph nodes of the mediastinum).

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:

1. **Organizational measures** (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge (checking workbooks, communicating with a patient with respiratory pathology for the purpose of collecting complaints and anamnesis, general and local examination, chest palpation, assessment of vocal tremor, assessment of examination data), conducting test control, solving clinical problems, written solution of Step-type problems -2 (10 tasks), face-to-face survey, discussion, role play on the subject of the lesson.

2.1. Requirements for theoretical readinessacquirers before practical classes:
the applicant must know the methods and scheme of examination of a patient with diseases of the respiratory system,
have an idea of the role of domestic scientists in the development of these methods; to know the general methodology of questioning patients with respiratory diseases;
know the characteristics and mechanisms of the main complaints (shortness of breath, cough, chest pain); the meaning and purpose of a general and local examination of a patient with respiratory pathology;
know the topographic areas of the chest;
to know the purpose, tasks and methods of chest palpation.

List of didactic units:

- collect in detail the complaints and medical history of a patient with respiratory pathology;
- perform a physical examination of the patient (general examination, chest examination, palpation of the chest and assess voice tremors), identify and evaluate the changes that have been found.

2.2. **Questions to check basic knowledge on the topic of the lesson:**

1. Name the main complaints of patients with pathology of the respiratory system.

2. What causes shortness of breath in patients with respiratory system pathology?
3. What types of shortness of breath can be found in the pathology of the bronchopulmonary system?
4. What types of cough can be found in the pathology of the bronchopulmonary system?
5. What lung diseases can cause hemoptysis?
6. Which includes examination of patients.
7. What are the pathological forms of the chest? Their diagnostic value.
8. What are pathological types of breathing? The reasons for their appearance.
9. Palpation of the chest: purpose, technique and diagnostic value.
10. Voice tremor: physical basis of the method, causes of change and diagnostic value of strengthening, weakening or absence of voice tremor

A problem of the STEP-2 type.

1. An increase in the volume of one half of the chest is observed with:

A. Pneumonia
B. Exudative pleurisy+
C. Lung emphysema
D. Pneumosclerosis
E. Lung atelectasis

2. Which form of the chest is characterized by: obtuse epigastric angle, horizontal location of the ribs, expansion of the upper half of the chest?

A. Asthenic
B. Hypersthenic
C. Emphysematous+
D. Paralytic
E. Rickets

3. Weakening of voice tremor is observed in:

A. Lung infarctions
B. Pneumonia
C. Pneumothorax+
D. Bronchitis
E. Lung abscesses

3. Formation of professional skills and abilities:

mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the conducted interrogation with the assumption of the localization of the pathological process)
formation of the ability to perform a clinical examination of a patient with a pathology of the respiratory organs (the applicant must be able to perform a general and local examination of a patient with diseases of the respiratory organs; examine and palpate the chest),
formation of the ability to give a clinical assessment obtained during examination and palpation; carry out a clinical interpretation of the main symptoms and syndromes in diseases of the respiratory organs.

3.1. Control materials for the final stage of the lesson:

Situational tasks:
In patient K., 35 years old, who complains of cough and high body temperature, the left half of the chest lags behind in the act of breathing, vocal tremor is significantly increased. Breathing is shallow. The number of respiratory movements is 33 per minute. Your diagnostic guess?

A. Pneumonia
B. Pneumothorax
C. Acute bronchitis
D. Lung abscess
E. Bronchial asthma

Answer. Such complaints and the results of examination and palpation of the chest are characteristic of pneumonia.

Tasks of the STEP-2 type.

1. When determining voice tremors, they use:

A. By deep sliding palpation;
B. Penetrating palpation;
+S. Bimanual palpation;
D. By the fluctuation method.

2. Voice tremor is determined by:

A. When holding the breath while lying down (inhalation);
B. When holding the breath during exhalation (exhalation);
C. After physical exertion;
D. After sleep;
+E. With normal breathing.

3. To determine voice tremor, the patient needs:

A. Be silent;
+V. Pronounce words that contain the letter "r";
S. Say words that contain the letter "x";
D. Pronounce words that contain the letter "f".

4. What will the voice tremble with exudative pleurisy:

A. Not changed;
+V. Weakened;
S. Reinforced;
D. Various.

5. What will be a voice tremor with pneumonia:

A. Not changed;
B. Weakened;
+S. Reinforced;
D. Various.

6. An increase in voice tremor indicates:
+A. Compaction of lung tissue;
B. Presence of hydrothorax;
C. Presence of pneumothorax;
D. Presence of hydro-pneumothorax;
E. Presence of hemothorax.

7. Weakened or absent vocal tremor is observed in:
A. Pneumonia;
B. Tuberculosis;
S. Bronchitis;
+D. Exudative pleurisy;
E. Bronchiectatic disease.

3.2. Requirements for work results, including before registration: substantiation of the syndromic diagnosis based on complaints, medical history and life data, clinical examination.
3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (if necessary).

4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 3
Subject: Lung percussion. Methodology and technique of conducting comparative lung percussion.
Goal: Acquiring knowledge and mastering professional competences in performing lung percussion, namely:
1. Know the rules and techniques of percussion.
2. Be able to distinguish types of percussion sound, know their properties, give an explanation.
3. Be able to determine the lower and upper limits of the lungs, know the norm and their changes in pathology.
4. Be able to determine the excursion of the lower border of the lungs, know the norm and its changes in pathology.
5. Determination of the standing height of the tops of the lungs from the front and back, the width of the Krenig fields.
6. Be able to perform comparative lung percussion, give a clinical interpretation.
Basic concepts: Percussion of the lungs, along with auscultation, is considered a very important method of examination of patients with pathology of the respiratory organs. According to the performance technique, direct and indirect percussion are distinguished. Among the methods of direct percussion, the methods of F.G. deserve the most attention. Yanovsky (tapping on the surface of the chest with the finger of the terminal phalanx of the middle finger of the right hand) and V.P. Obraztsova (tapping on the surface of the chest with

the finger of the terminal phalanx of the index finger, which slides over the surface of the third finger). But indirect percussion is more widely used. A methodically correctly performed percussion, with sufficient awareness of the doctor about the diagnostic significance of the detected signs, provides grounds for establishing a preliminary diagnosis.
Lung percussion - a method of objective research of the broncho-pulmonary system by striking the tissues and organs with evaluation of the sounds obtained in order to detect pathological processes in the lung tissue and lung cavity.
There are 2 types of percussion: comparative and topographic, which have their own technical and diagnostic features.
Comparative percussion - percussion performed with the aim of identifying the pathological process, determining its nature, localization and dynamics. Based on the specified purpose of percussion, the comparative percussion technique has its own characteristics:
- percussion is performed on strictly symmetrical areas of the chest in a specified sequence;
– percussion is performed strictly along the intercostal space (because due to the better conduction of sound through dense tissue, when percussing a rib, the rib's own sound is added to the percussive sound);
– blows are loud, intermittent, always with the same force, on the middle phalanx of the plesymeter finger, with the exception of the supraclavicular pits, where the blow is applied to the nail phalanx (in order to ensure close contact of the plesymeter finger with the body surface in the area of the pits);
- alternate use of strong and then weak percussion to avoid possible mistakes; with a strong percussion, you may not find surface changes, with a quiet one – deeply localized ones.
*Methodology of comparative percussion.*Comparative percussion is performed taking into account its features in the following sequence: in supraclavicular areas ("plesimeter" is located above and parallel to the clavicle); the front surface of the chest, except for the area of the heart; side areas (left and right); back surface of the chest (supra-scapular, inter-scapular, sub-scapular areas)
Normally, during comparative percussion, a clear lung sound is determined on symmetrical parts of the lungs. However, given the fact that the lungs are surrounded by tissues of different mass and density, and conduct sound differently, when evaluating the results of comparative percussion, it is worth considering the places of physiological changes in the percussion sound:
- the dulling of the percussion sound in the right supraclavicular area is caused by the lower position of the apex of the right lung in comparison with the apex of the left lung and more significant development of the muscles of the right shoulder girdle;
- dulling of the percussion sound in the II-III intercostal space to the left of the sternum is due to the close location of the heart;
- dulling of the percussion sound in the lower part of the right lateral area is due to the close location of the liver;
- the tympanic tone of the percussion sound on the left (Traube's space) is caused by the proximity of the stomach filled with air.

Peculiarities of percussive sounds in the pathology of respiratory organs
Pathological processes of the respiratory organs can be accompanied by the following changes:

a) violation of the drainage function of the trachea and bronchi;
b) change in airiness and elasticity of lung tissue;
c) compaction of lung tissue;
d) formation of cavity formations in the lungs;
e) accumulation of fluid or air in the pleural cavities.
Depending on the features of the pathological process and the nature of tissue damage above the lungs, the following changes in the percussion sound can be determined: weakening of the clear lung sound, its dulling (dulled sound, dulled-tympanic, dull sound), or strengthening (tympanic, box, metallic, "cracked sound" pot").
In the case of pathological processes of small sizes, diffusely located in unchanged lung tissue or those that lie deep from the surface of the chest, a clear lung sound is determined. A decrease in the airiness of the lung tissue, its compaction, partial or complete disruption of sound conduction leads to a weakening of the lung sound. Depending on the size, nature and localization of the pathological process, the following are distinguished: muffled lung sound, muffled tympanic or dull sound.
Dull lung sound (silent, high, quiet) due to a decrease in the air content in the alveoli (focal pneumonia, lobar pneumonia in the initial and final stages, tuberculosis, atelectasis, a small tumor), thickening or fusion in a limited area of the pleura (tumor, metastases), accumulation of a moderate amount fluid in the pleural cavity (hydrothorax, pyothorax, hemothorax).
Dulled-tympanic sound (quiet, high, with a tympanic tone) due to a decrease in the airiness of the lung tissue in combination with compaction and a decrease in the elasticity of the alveolar walls due to inflammatory infiltration. Dull-tympanic sound can be determined by:
– the initial stage of pneumonia (tension of the infiltrated lung tissue);
- bronchopneumonia (alternation of foci of compaction with areas of an intact lung);
- tuberculosis (tension infiltration of lung tissue, mainly in the upper lobes);
– compression atelectasis (compression of the lung by a tumor, liquid or air in the pleural cavity);
– partial obturation atelectasis (disruption of the drainage function of the bronchi and the entry of air into the alveoli);
- pulmonary edema (the presence of air and transudate in the alveoli at the same time).
Dull sound (quiet, high, short) due to compaction of lung tissue due to filling of alveoli with transudate, exudate or blood (pneumonia in the acute stage, lung infarction); a massive tumor or lung abscess (above pus), located near the surface of the chest; accumulation of more than 6 centimeters of fluid in the pleural cavity (exudative pleurisy or hydrothorax).
Box sound (loud, tympanic with low tonality) due to an increase in the airiness of the lung tissue as a result of a decrease in its elasticity and a violation of the drainage function of the small bronchi due to spasm. It is observed in emphysema of the lungs, bronchial asthma, chronic obstructive bronchitis.
Tympanic sound(loud, low with a tympanic tone) due to the presence of a large cavity with elastic walls, which contains air under pressure and is connected to the bronchus (abscess after the discharge of pus, tubercular cavity, tumor in the stage of decay, lung cyst, echinococcosis, closed pneumothorax).
Metallic sound (a type of tympanic sound with reduced tonality, similar to the sound of hitting a metal vessel) is found in the presence of a superficially located large cavity (at least

6 cm in diameter), with smooth walls, which contains air, and is connected to a narrow bronchus (abscess lungs, pneumothorax).
The sound of a cracked pot» (silent, high, with an intermittent tone) due to the presence of a superficially located large cavity connected by a narrow gap to the bronchus or to the external environment (open pneumothorax).

Topographic percussion of the lungsbe carried out in order to determine the limits and mobility of the lower edge of the lungs, the height of their tops and the width of the Krenig fields.
Topographic percussion technique:
- position of the patient: lying or sitting, the exception is severe patients (myocardial infarction, impaired cerebral circulation), in whom percussion is performed in a lying position, percussion is limited to the front and side surfaces of the chest. The muscles of the shoulder girdle should be relaxed;
- position of the doctor: to the right of the patient;
– position of the plesimeter: parallel to the expected border: when determining the lower limits, the finger-plesimeter is located along the intercostals parallel to the ribs, when determining the upper limits: in front – above and parallel to the clavicle, behind – above and parallel to the spine of the shoulder blades;
– impact force: medium force, closer to quiet;
- the direction of percussion: from a clear lung sound to a dull one, the limit is determined by the edge of the plesimeter finger facing the clear lung sound.
Topographic percussion of the lungs be carried out in the following sequence: determination of the standing height of the tops of the lungs (front and back) and Krenig fields, determination of the limits of the lower edge of the lungs, determination of the mobility of the lower edge of the lungs (during inhalation and exhalation).
Determination of the standing height of the tops of the lungs. The height of the tops of the lungs is determined from the front above the clavicles, and from the back above the spine of the shoulder blades. From the front, start percussing from the middle of the clavicle upwards and towards the spine until dullness appears (the plesymeter finger is placed parallel to the clavicle and percussed along the nail phalanx). Normally, the standing height of the top of the right lung is 3-4 cm from the front, and the left one is 4.0-4.5 cm above the middle of the clavicle. Behind, to determine the standing height of the tops, percussion is started from the spine of the scapula in the direction of the spinous process of the VII cervical vertebra until dullness appears. Normally, the standing height of the tops of both lungs is located at the level of the spinous process of the VII cervical vertebra.
*Determination of the width of the vertices (Krenig fields)***.** Krenig's field is a band of clear pulmonary sound 5-6 cm wide, extending from the clavicle to the scapular spine. To determine the width of Krenig's fields, the plesimeter finger is placed in the middle of the apex along the front edge of the trapezius muscle and tapped outward (down) and in the middle (up) until a dull or dull sound appears. . Normally, the width of the Krenig field is 5-6 cm, with fluctuations in the range from 3.5 to 8 cm. It should be remembered that the standing height of the apex of the right lung is normally 1 cm higher, and the width of the Krenig field is 1-1, 5 cm wider than the left lung, which is due to the anatomical mutual

location of the heart and the left lung. Narrowing of Krenig's fields (less than 4 cm) is observed in the presence of apical shrinkage (tuberculosis).
*Determination of the lower limits of the lungs***.** Determination of the lower limits of the lungs begins with the right lung (pulmonary-hepatic border), tapping from above from the subclavian fossa down to bluntness along the peristernal, midclavicular, anterior, middle and posterior axillary, scapular and paravertebral lines. Percussion is carried out along the intercostals, the plesimeter finger is placed parallel to the expected border, a mark is placed on the edge of the plesimeter from the side of the clear lung sound.
Then the lower border of the left lung is determined, starting percussion from the front axillary line, because the border of relative dullness of the heart passes along the peristernal and midclavicular lines. Further, the percussion is continued similarly to the right lung along the topographic lines (three axillary, scapular, vertebral).
The lower border of both lungs has a horizontal direction, symmetrical, except for the heart notch on the left.
*Determination of the mobility of the lower edge of the lungs (excursion of the lung edge)***.**
Mobility of the lung edge*(breathing excursion)* is the distance between the positions of the edge of the lung during maximum inhalation and maximum exhalation. The degree of respiratory excursion of the lung edge depends on the elasticity of the lung tissue, i.e. its ability to expand (inhalation) and decrease (on exhalation), the depth of the pleural sinus, and also depends on the position of the subject's body. Hence, two types of mobility of the lower edge of the lungs are distinguished: active, caused by the breathing phase, and passive, caused by a change in body position.
Determination of the mobility of the lower edge of the lungs is carried out in places of the greatest respiratory activity along the middle and posterior axillary lines.
The method of determining the mobility of the lung edge:
- with quiet percussion, determine the position of the lower edge of the lung during calm breathing and make a mark;
- then the patient is offered to take a deep breath and hold his breath at his height, and at this time percussion is performed downward until dullness appears, the position of the lower edge of the lung is determined again and a mark is made;
- then the patient is asked to take a deep breath and hold his breath at its height, and at this time percussion is performed upwards until the appearance of a lung sound, the position of the edge of the lungs at maximum exhalation is again determined and a mark is made;
- the distance between the second and third marks, measured in centimeters, is the size of the respiratory excursion of the lung edge.
Normally, the lower edge of the lungs during a deep inhalation descends 3-4 cm from the lower border of the lungs, during a deep exhalation it rises 3-4 cm from the lower border of the lungs. Thus, in total, the respiratory excursion of the lungs along the middle and posterior axillary lines is 6-8 cm, along the midclavicular line - 4 cm. In women, the size of the respiratory excursion of the lower lung edge is lower due to a smaller share of the participation of the diaphragm in the act of breathing.
Limitation or complete lack of mobility of the lung edge can be caused either by a change in the properties of the lung tissue (reduced elasticity), or by obstruction of inhalation and exhalation from the sides of the pleural cavity and diaphragm. Thus, a change in the properties of lung tissue is observed in emphysema of the lungs (reduced elasticity and

development of pneumosclerosis); lobar pneumonia (inflammatory infiltration of the lungs); pulmonary edema (due to interstitial edema of the alveolar walls); lung tumor (growth of the edges of the lungs by tumor tissue and their shrinkage).
Obstruction from the side of the pleura and the pleural cavity can be due to the fusion of the pleural sheets in the area of the sinus; or by an adhesive process after the transferred pleurisy, more often purulent; accumulation of fluid (exudate, transudate, pus, blood) or air in the pleural cavity (pneumothorax); high position of the diaphragm due to increased intra-abdominal pressure (ascites, flatulence, massive tumor); paralysis of the diaphragm.

Plan:

1. **Organizational measures** (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge (checking workbooks, conducting comparative percussion in a patient with bronchopulmonary pathology, conducting topographic percussion, namely - determination of the lower limits of the lungs on the left and right, determination of the standing height of the tops of the lungs and Krenig's fields, determination of the mobility of the lower edge of the lungs and evaluation of the obtained examination data*),* conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for the theoretical readiness of applicants to perform practical classes:

- the applicant must know the purpose of comparative and topographic percussion;
- have an idea of the role of domestic scientists in the development of percussion methods;
- to know the rules and techniques of comparative and topographical percussion, to be able to demonstrate them.
- the applicant must know the basic percussion sounds in normal and pathological conditions, be able to distinguish them;
- to know the main reasons for changes during topographic and comparative percussion.

List of didactic units:

- perform comparative percussion in a patient with lung pathology;
- carry out topographic percussion to determine the height of the tops of the lungs
- perform topographic percussion with determination of the lower border of the lungs on the right and left;
- perform topographic percussion with determination of the width of Krenig's fields;
- perform topographic percussion to determine the mobility of the lower lung edge.

2.2. **Questions to check basic knowledge on the topic of the lesson:**

1. Name the basic rules of percussion;
2. What is the normal nature of the percussion sound?
3. Mechanism of occurrence and significance of dull, dulled, tympanic, box, metal percussion sounds?
4. What is the algorithm for comparative lung percussion?

5. What is the algorithm for topographic percussion of the lungs?
6. Where are the upper and lower borders of the lungs when their displacement is observed?
7. What is the norm of the width of the Krenig fields?
8. How is the Traube space limited?
9. What is the active mobility of the lower edges of the lungs, their size is normal, the pathological conditions under which it changes?

A problem of the STEP-2 type.
A dull percussion sound is detected above the chest on the left on the back surface from the VII rib downwards. A diagnostic assumption?
A. Pneumothorax
B. Lung abscess
C. Compression atelectasis
D. Lung cavity
+E. Hydrothorax

3. Formation of professional skills and abilities:
- formation of the ability to perform a clinical examination of a patient with respiratory pathology (the applicant must be able to demonstrate the technique of comparative and topographic percussion of the lungs in patients with bronchopulmonary pathology),
- formation of the ability to give a clinical assessment of the results obtained during the comparative and topographical percussion of the lungs, to carry out a clinical interpretation of the main symptoms and syndromes in diseases of the respiratory organs.

3.1. Control materials for the final stage of the lesson:
Situational tasks:
1. When percussing the lungs over the entire surface, more on the front surface, a box sound is determined. Your diagnostic assumption.
A. Cavern in the lung
B. Pneumothorax
C. Emphysema of the lungs +
D. Obturational atelectasis of the lungs
E. Exudative pleurisy
F.
2. Percussion of the chest reveals the sound of a "cracked pot" in the following case:
A. Pneumonia
B. Bronchitis
C. Pneumosclerosis
D. Open pneumothorax+
E. Atelectasis of the lungs

3. How does the upper border of the lungs change with emphysema?
A. Does not change;
B. Decreases;
C. Increases+

D. Not defined;
E. Incorrect question

4. When does a dull sound appear over the lungs?
A. With pneumothorax
B. With emphysema of the lungs;
C. With exudative pleurisy over liquid+
D. During physical exertion;

5. What will you find by percussion over the abscess when its cavity is freed from pus?
A. Dulling of percussion sound.
V. A dull percussion sound.
C. Dull tympanitis+
D. Tympanitis.
E. Box sound.

6. From which section do comparative percussion begin?
A. Apex of the lungs from behind.
B. Apex of the lungs in front+
C. Subclavian areas.
D. Axillary areas.
E. Subscapular areas.

7. When there is a dull sound over the lungs*(quiet, short, high, femoral dullness):*

A. A significant amount of fluid in the pleura +
C. Cavern.
S. Pneumothorax.
D. Emphysema.
E. Bronchitis.

8. There are several percussive signs of emphysema. Find one of the following characteristics that is not characteristic of her:

A. Box sound
B. Lowering of the lower edges of the lungs.
C. Narrowing of Krenig + fields
D. Expansion of the Krenig fields.
E. Reducing the limits of absolute dullness of the heart.

9. The percussive sound of a "cracked pot" is characteristic of:

A. Bronchitis.
B. Pneumonia.

S. Atelectasis.
D. Caverns connected to the bronchus+
E. Pneumoconiosis.

3.2. Requirements for work results, including before registration: substantiation of the syndromic diagnosis based on complaints, medical history and life data, clinical examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the subject of the lesson, answering 10 tests (*if necessary*).

4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 4
Subject: Lung percussion. Methodology and technique of conducting topographic percussion of the lungs.
Goal: Acquiring knowledge and mastering professional competences in performing lung percussion, namely:
1. Know the rules and techniques of topographic percussion.
2. Be able to distinguish types of percussion sound, know their properties, give an explanation.
3. Be able to determine the lower and upper limits of the lungs, know the norm and their changes in pathology.
4. Be able to determine the excursion of the lower border of the lungs, know the norm and its changes in pathology.
5. Determination of the standing height of the tops of the lungs from the front and back, the width of the Krenig fields.
6. Be able to perform comparative lung percussion, give a clinical interpretation.

Basic concepts: Percussion of the lungs, along with auscultation, is considered a very important method of examination of patients with pathology of the respiratory organs. According to the performance technique, direct and indirect percussion are distinguished. Among the methods of direct percussion, the methods of F.G. deserve the most attention. Yanovsky (tapping on the surface of the chest with the finger of the terminal phalanx of the middle finger of the right hand) and V.P. Obraztsova (tapping on the surface of the chest with the finger of the terminal phalanx of the index finger, which slides over the surface of the third finger). But indirect percussion is more widely used. Correctly performed percussion, from a methodical point of view, if the doctor is sufficiently informed about the diagnostic significance of the detected signs, provides grounds for establishing a preliminary diagnosis.
Lung percussion - a method of objective research of the broncho-pulmonary system by striking the tissues and organs with the evaluation of the sounds obtained in order to identify pathological processes in the lung tissue and lung cavity.
There are 2 types of percussion: comparative and topographic, which have their own technical and diagnostic features.

Topographic percussion of the lungsbe carried out in order to determine the limits and mobility of the lower edge of the lungs, the height of their tops and the width of the Krenig fields.
Topographic percussion technique:
- position of the patient: lying or sitting, the exception is severe patients (myocardial infarction, impaired cerebral circulation), in whom percussion is performed in a lying position, percussion is limited to the front and side surfaces of the chest. The muscles of the shoulder girdle should be relaxed;
- position of the doctor: to the right of the patient;
– position of the plesymeter: parallel to the expected border: when determining the lower limits, the finger-plesymeter is located along the intercostal space parallel to the ribs, when determining the upper limits: in front – above and parallel to the clavicle, behind – above and parallel to the spine of the shoulder blades;
– impact force: medium force, closer to quiet;
- the direction of percussion: from a clear lung sound to a dull one, the limit is determined by the edge of the plesimeter finger facing the clear lung sound.
Topographic percussion of the lungs be carried out in the following sequence: determination of the standing height of the tops of the lungs (front and back) and Krenig fields, determination of the limits of the lower edge of the lungs, determination of the mobility of the lower edge of the lungs (during inhalation and exhalation).
Determination of the standing height of the tops of the lungs. The height of the tops of the lungs is determined from the front above the clavicles, and from the back above the spine of the shoulder blades. From the front, start percussing from the middle of the clavicle upwards and towards the spine until dullness appears (the plesymeter finger is placed parallel to the clavicle and percussed along the nail phalanx). Normally, the standing height of the top of the right lung is 3-4 cm from the front, and the left one is 4.0-4.5 cm above the middle of the clavicle. Behind, to determine the standing height of the tops, percussion is started from the spine of the scapula in the direction of the spinous process of the VII cervical vertebra until dullness appears. Normally, the standing height of the tops of both lungs is located at the level of the spinous process of the VII cervical vertebra.
*Determination of the width of the vertices (Krenig fields)***.** Krenig's field is a band of clear pulmonary sound 5-6 cm wide, extending from the clavicle to the scapular spine. To determine the width of Krenig's fields, the plesimeter finger is placed in the middle of the apex along the front edge of the trapezius muscle and tapped outward (down) and in the middle (up) until a dull or dull sound appears. . Normally, the width of the Krenig field is 5-6 cm, with fluctuations in the range from 3.5 to 8 cm. It should be remembered that the standing height of the apex of the right lung is normally 1 cm higher, and the width of the Krenig field is 1-1, 5 cm wider than the left lung, which is due to the anatomical mutual location of the heart and the left lung. Narrowing of Krenig's fields (less than 4 cm) is observed in the presence of apical shrinkage (tuberculosis).
*Determination of the lower limits of the lungs***.** Determination of the lower limits of the lungs begins with the right lung (pulmonary-hepatic border), tapping from above from the subclavian fossa down to bluntness along the peristernal, midclavicular, anterior, middle and posterior axillary, scapular and paravertebral lines. Percussion is carried out along the

intercostal space, the plesimeter finger is placed parallel to the expected border, a mark is placed on the edge of the plesimeter from the side of the clear lung sound.
Then the lower border of the left lung is determined, starting percussion from the front axillary line, because the border of relative dullness of the heart passes along the peristernal and midclavicular lines. Further, percussion is continued similarly to the right lung along the topographic lines (three axillary, scapular, paravertebral).
The lower border of both lungs has a horizontal direction, symmetrical, except for the heart notch on the left.
*Determination of the mobility of the lower edge of the lungs (excursion of the lung edge)***.**
Mobility of the lung edge*(breathing excursion)* is the distance between the positions of the edge of the lung during maximum inhalation and maximum exhalation. The degree of respiratory excursion of the lung edge depends on the elasticity of the lung tissue, i.e. its ability to expand (on inhalation) and compress (on exhalation), the depth of the pleural sinus, and also depends on the position of the subject's body. Hence, two types of mobility of the lower edge of the lungs are distinguished: active, caused by the breathing phase, and passive, caused by a change in body position.
Determination of the mobility of the lower edge of the lungs is carried out in places of the greatest respiratory activity along the middle and posterior axillary lines.
The method of determining the mobility of the lung edge:
- with quiet percussion, determine the position of the lower edge of the lung during calm breathing and make a mark;
- then the patient is offered to take a deep breath and hold his breath at his height, and at this time percussion is performed downward until dullness appears, the position of the lower edge of the lung is determined again and a mark is made;
- then the patient is asked to take a deep breath and hold his breath at its height, and at this time percussion is performed upwards until the appearance of a lung sound, the position of the edge of the lungs at maximum exhalation is again determined and a mark is made;
- the distance between the second and third marks, measured in centimeters, is the size of the respiratory excursion of the lung edge.
Normally, the lower edge of the lungs during a deep inhalation descends 3-4 cm from the lower border of the lungs, during a deep exhalation it rises 3-4 cm from the lower border of the lungs. Thus, in total, the respiratory excursion of the lungs along the middle and back-axillary lines is 6-8 cm, along the middle-clavicular line - 4 cm. In women, the size of the respiratory excursion of the lower lung edge is lower due to the lower participation of the diaphragm in the act of breathing.
Limitation or complete lack of mobility of the lung edge can be caused either by a change in the properties of the lung tissue (reduced elasticity), or by obstruction of inhalation and exhalation from the sides of the pleural cavity and diaphragm. Thus, a change in the properties of lung tissue is observed in emphysema of the lungs (reduced elasticity and development of pneumosclerosis); lobar pneumonia (inflammatory infiltration of the lungs); pulmonary edema (due to interstitial edema of the alveolar walls); lung tumor (growth of the edges of the lungs by tumor tissue and their shrinkage).
Obstruction from the side of the pleura and the pleural cavity can be due to the fusion of the pleural sheets in the area of the sinus; or by an adhesive process after the transferred pleurisy, more often purulent; accumulation of fluid (exudate, transudate, pus, blood) or air in the

pleural cavity (pneumothorax); high position of the diaphragm due to increased intra-abdominal pressure (ascites, flatulence, massive tumor); paralysis of the diaphragm.

Plan:

1. Organizational measures (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge (checking workbooks, conducting comparative percussion in a patient with bronchopulmonary pathology, conducting topographic percussion, namely - determination of the lower limits of the lungs on the left and right, determination of the standing height of the tops of the lungs and Krenig fields, determination of the mobility of the lower edge of the lungs and evaluation of the obtained examination data*),* conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for the theoretical readiness of applicants to perform practical classes:

- the applicant must know the purpose of comparative and topographic percussion;
- have an idea of the role of domestic scientists in the development of percussion methods;
- to know the rules and technique of comparative and topographical percussion, to be able to demonstrate them.
- the applicant must know the basic percussion sounds in normal and pathological conditions, be able to distinguish them;
- to know the main reasons for changes during topographic and comparative percussion.

List of didactic units:

- perform comparative percussion in a patient with lung pathology;
- carry out topographic percussion to determine the height of the tops of the lungs
- perform topographic percussion with determination of the lower border of the lungs on the right and left;
- perform topographic percussion with determination of the width of Krenig's fields;
- perform topographic percussion to determine the mobility of the lower lung edge.

2.2. **Questions to check basic knowledge on the topic of the lesson:**

1. Name the basic rules of percussion;
2. What is the normal nature of the percussion sound?
3. Mechanism of occurrence and significance of dull, dulled, tympanic, box, metal percussion sounds?
4. What is the algorithm for comparative lung percussion?
5. What is the algorithm for topographic percussion of the lungs?
6. Where are the upper and lower borders of the lungs when their displacement is observed?
7. What is the norm of the width of the Krenig fields?
8. How is the Traube space limited?
9. What is the active mobility of the lower edges of the lungs, their size is normal, the pathological conditions under which it changes?

A problem of the STEP-2 type.

A dull percussion sound is detected above the chest on the left on the back surface from the VII rib downwards. A diagnostic assumption?
A. Pneumothorax
B. Lung abscess
C. Compression atelectasis
D. Lung cavity
+E. Hydrothorax

3. Formation of professional skills and abilities:
- formation of the ability to perform a clinical examination of a patient with respiratory pathology (the applicant must be able to demonstrate the technique of comparative and topographic percussion of the lungs in patients with bronchopulmonary pathology),
- formation of the ability to give a clinical assessment of the results obtained during the comparative and topographical percussion of the lungs, to carry out a clinical interpretation of the main symptoms and syndromes in diseases of the respiratory organs.

3.1. Control materials for the final stage of the lesson:
Situational tasks:
1. When percussing the lungs over the entire surface, more on the front surface, a box sound is determined. Your diagnostic assumption.
A. Cavern in the lung
B. Pneumothorax
C. Emphysema of the lungs +
D. Obturational atelectasis of the lungs
E. Exudative pleurisy

2. Percussion of the chest reveals the sound of a "cracked pot" in the following case:
A. Pneumonia
B. Bronchitis
C. Pneumosclerosis
D. Open pneumothorax+
E. Atelectasis of the lungs

3. How does the upper border of the lungs change with emphysema?
A. Does not change;
B. Decreases;
C. Increases+
D. Not defined;
E. Incorrect question

4. When does a dull sound appear over the lungs?
A. With pneumothorax
B. With emphysema of the lungs;
C. With exudative pleurisy over liquid+
D. During physical exertion;

5. What will you find by percussion over the abscess when its cavity is freed from pus?
A. Dulling of percussion sound.
V. A dull percussion sound.
C. Dull tympanitis+
D. Tympanitis.
E. Box sound.

6. The lower limits of the lungs begin to be determined from the topographic line:
A) of the front axillary right.
B) parasternal case.
B) scapular line.
D) paravertebral line.

7. When there is a dull sound over the lungs*(quiet, short, high, femoral dullness):*

A. A significant amount of fluid in the pleura +
C. Cavern.
S. Pneumothorax.
D. Emphysema.
E. Bronchitis.

8. There are several percussive signs of emphysema. Find one of the following characteristics that is not characteristic of her:

A. Box sound
B. Lowering of the lower edges of the lungs.
C. Narrowing of Krenig + fields
D. Expansion of the Krenig fields.
E. Reducing the limits of absolute dullness of the heart.

9. The percussive sound of a "cracked pot" is characteristic of:

A. Bronchitis.
B. Pneumonia.
S. Atelectasis.
D. Caverns connected to the bronchus+
E. Pneumoconiosis.

3.2. Requirements for work results, including before registration: substantiation of the syndromic diagnosis based on complaints, medical history and life data, clinical examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the subject of the lesson, answering 10 tests (*if necessary*).

4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 5
Subject: Auscultation of the lungs: basic respiratory sounds (vesicular and bronchial breathing)

Goal: Acquiring knowledge and mastering professional competences in lung auscultation, namely:
- to know the purpose and tasks of lung auscultation;
- to know the procedure and basic rules for lung auscultation;
- content to perform auscultation of the lungs in a patient with pathology of the bronchopulmonary system;
- to know the mechanism of formation of basic respiratory sounds and their change in pathological conditions.

Basic concepts:auscultation of the lungs is the main physical method of examining the patient, which is included in the general mandatory examination methods and is carried out regardless of the existing pathology and during preventive examinations. It was introduced into medical practice at the beginning of the 19th century, and remains an indispensable diagnostic method for researching respiratory organs. Auscultation of the lungs allows you to determine the nature and characteristics of breathing types, compare them with the appropriate norm and, on this basis, evaluate the morphological structure and functional capacity of the organs of the respiratory system. In addition, auscultation allows, based on the detection of sound phenomena, to determine both the localization and nature of the respiratory system lesion, as well as to quickly monitor the course of the disease. The use of lung auscultation in combination with the main and additional examination methods used in pulmonology practice makes it possible to establish the leading syndrome of damage to the respiratory organs, on the basis of which a nosological diagnosis is determined and a treatment plan is prescribed in accordance with state and international standards for providing medical care to patients with therapeutic (in particular , pulmonary) profile.

Auscultation - an objective research method based on listening to sound phenomena that occur in the body as a result of its vital activity.

Auscultation rules:
- the position of the patient should be convenient for the work of the respiratory organs, therefore, auscultation of the lungs is carried out in standing, sitting positions, in seriously ill patients - lying down;
- the chest should be completely exposed, so the side sounds caused by the friction of clothes create side sounds. For the same reason, you should not hold the tube of the stethoscope or phonendoscope with your hand;
- the room should be quiet and warm to exclude sound interference and muscle tension;
– the stethoscope or phonendoscope is applied perpendicular to the surface of the patient's body, tightly, but without pressure (so as not to cause pain and not to limit the oscillations of the chest in the listening area);
– it is necessary to use the same stethoscope or stethoscope.

Auscultation is performed on symmetrical areas, in the same sequence and places as comparative percussion: the tops of the lungs in the supraclavicular areas, the front surface of the chest, except for the atrial area, axillary, supra-intermediate and subscapular areas.
Analysis of auscultatory data is carried out in the following order:
– assessment of basic respiratory sounds;
– analysis of changes in basic respiratory sounds;
- the presence of side breathing noises.

Basic breath sounds.
The main respiratory sounds include bronchial (laryngo-tracheal) and vesicular breathing.
Bronchial (laryngeal-tracheal) breathing - breathing noises, which are produced when air passes through a narrowed glottis, areas of bifurcation of the trachea and large bronchi. The mechanism of bronchial breathing is based on the turbulent movement of air during the passage of a narrowed section of the respiratory tract: the glottis, the bifurcation of the trachea and large bronchi. Bronchial breathing is a loud, coarser respiratory noise, with a musical tone reminiscent of the pronunciation of the letter "x", heard in both phases of breathing, and on exhalation these noises are louder and longer. This is explained by the fact that during exhalation the glottis is more narrowed than during inhalation, which means that the speed of air and its turbulent movement are greater, therefore the respiratory noise on exhalation is louder and longer.
Normally, bronchial breathing is heard above the larynx (in front - on the front surface of the neck, behind - at the level of the VII cervical vertebra), at the level of the bifurcation of the trachea (in front - near the handle of the sternum, behind - in the interscapular area at the level of the III-IV thoracic vertebrae). Above the other surface of the chest (lung tissue), bronchial breathing cannot be heard.
Vesicular (alveolar) respiration - respiratory noises that are produced when the alveoli are filled with air. The basis of the mechanism of vesicular breathing is the oscillation of the tense elastic walls of the alveoli at the moment of their filling with air and the expansion of the lungs during inhalation. The noises of successively occurring oscillations of a huge number of alveoli are combined, perceived as a gentle prolonged noise, which is auscultatively regarded as vesicular breathing.
Vesicular breathing is the sound of the lungs expanding, it is a quiet, gentle noise, reminiscent of the pronunciation of the letters "f", heard during inhalation and the first third of exhalation. The strengthening of vesicular breathing at the height of inhalation is explained by the summation of the vibrations of the walls of all alveoli, which are straightened, and its listening during the first third of exhalation is due to a rapid decrease in the tension of the alveolar walls due to their decline and reduced ability to vibrate.
Normally, vesicular breathing is heard over the entire surface of the lungs: the apex, front surface of the chest, axillary and subscapular areas.
Change in basic respiratory sounds
Pathological processes both in the lungs themselves and in the surrounding tissues can lead to a change in the main respiratory sounds, therefore, in clinical practice, pulmonary and extrapulmonary causes of changes in the main and the appearance of secondary respiratory sounds are conditionally distinguished.

Changes in bronchial breathing.Pathological variants of bronchial breathing include:*pathological bronchial breathing, amphoric, metallic and stenotic breathing, as well as mixed (broncho-vesicular) breathing.*

Pathological bronchial breathing - bronchial breathing heard above the surface of the lungs, except for those places where it is normally heard.
The occurrence of pathological bronchial breathing is due to the absence of vesicular breathing in this area and good conduction of bronchial breathing to it. Such conditions for the occurrence of pathological bronchial breathing are: compaction of the lung tissue and/or the presence of a lung cavity connected to the bronchus. Pathological bronchial breathing, like physiological breathing, is heard in both phases of breathing with a more pronounced and prolonged exhalation, but it differs in places of listening, and it can also have unequal strength and timbre, which is due to compaction of lung tissue and localization of the pathological focus in the lungs.
Amphoric breathing (from Greek*amphora* - a jug or a vessel with a narrow neck) is a variant of pathological bronchial breathing. It is a soft, low sound, with additional high overtones, an "empty sound" similar to the sound of blowing hard over the neck of an empty vessel. It occurs in the presence of a large smooth-walled cavity connecting to a large bronchus (lung abscess, tuberculous cavity).
Metallic breath – a variant of pathological bronchial breathing that occurs over a large smooth-walled cavity filled with air and connected to the external environment. Unlike amphoric breathing, metallic breathing is characterized by a loud, high-pitched, ringing (metallic) tone and is pathognomonic for open pneumothorax. The conditions for the occurrence of amphoric and metallic breathing can arise due to the proximity of large smooth-walled air cavities. For example, during infiltration of the lower lobe of the left lung near the stomach and significant tension of its walls, bronchial breathing can take on an amphoric or even metallic hue.
Variants of pathological bronchial breathing include***stenotic breathing*,** which is increased bronchial breathing caused by a sharp narrowing of the trachea or large bronchus; can be heard with bronchogenic cancer, massive enlargement of mediastinal lymph nodes.
Mixed (bronchovesicular) breathing – breathing, during which the inhaling phase is vesicular breathing, and the exhaling phase is bronchial. Normally, it is sometimes heard above the apex of the right lung. In pathological conditions, mixed breathing is caused either by the presence of small foci of compaction surrounded by intact lung tissue; or deeply located massive seals. In both cases, two types of breathing are simultaneously heard: bronchial (compaction of lung tissue) and vesicular (intact lung tissue), which are simultaneously perceived as mixed breathing. Mixed breathing can be heard in bronchopneumonia, pulmonary tuberculosis, pneumosclerosis.

Change in vesicular respiration
The nature of changes in vesicular breathing depends on the number and quality of the alveoli that participate in breathing, the speed and volume of air entering them, and the properties of the surrounding tissues.

Changes in vesicular respiration can be quantitative and qualitative. Quantitative changes include:*strengthening, weakening and complete absence of vesicular breathing; to quality - hard, vesicular with prolonged exhalation and intermittent*("saccade" breathing).
Weakening of vesicular breathing characterized by quieter and shorter breathing with preservation of the ratio of inhalation/exhalation phases (3/1), can be both physiological and pathological.
Physiological weakening of vesicular breathing observed in the case of thickening of the chest due to excessive muscle development (in men, athletes) or increased deposition of fat in the subcutaneous adipose tissue (in hypersthenics), in addition, the weakening of vesicular breathing is due to the peculiarities of the respiratory organs themselves (smaller volume of lung tissue above the tops of the lungs in compared to the lower departments).
Pathological weakening of vesicular respiration may be due to the following reasons:
- difficulty in getting air into the lungs due to:
  – compression of the airways (trachea, bronchi) by a tumor, enlarged intrathoracic lymph nodes, scar tissue;
  – violation of the drainage function of the bronchi, caused by inflammatory infiltration or significant swelling of their mucous membrane, the presence of viscous sputum in them, or the ingress of a foreign body;
- insufficient expansion of the alveoli during inhalation, due to a decrease in the elasticity of the lung tissue due to:
  – inflammatory infiltration (partial pneumonia, initial and final stages of lobar pneumonia);
  – interstitial edema (initial stage of pulmonary edema);
  – destruction of alveoli (pulmonary emphysema);
  – growth of connective tissue and its replacement of pulmonary tissue (pneumosclerosis).
- reflex decrease in the respiratory excursion of the chest, caused by a pain syndrome during breathing (damage to the pleura, ribs, intercostal muscles and nerves).

Absence of vesicular respiration due to a violation of its formation or an obstacle to its implementation. In the first case, the lack of breathing is caused by a complete blockage of the bronchi by a foreign body or a tumor, with the development of lung collapse (obturational atelectasis); in the second case, the accumulation of a large amount of fluid or air in the pleural cavity leads to compression of lung tissue (compression atelectasis) and at the same time is an obstacle to the conduction of respiratory sounds to the surface of the chest.
Increased vesicular breathing is louder and longer respiratory noises with preservation of the ratio of inhalation and exhalation (3:1) and is due to the rapid expansion of the alveoli during inhalation. Physiological and pathological enhancement of vesicular respiration are distinguished.
Physiological (forced) enhanced vesicular breathing due to good conduction of sound to the surface of the chest; observed during physical exertion in persons with a thin chest (asthenics). A variant of physiological enhanced vesicular breathing is*puerile breathing* (*few* - boy) - increased vesicular breathing with well-defined exhalation. Puerile breathing is characteristic of children under the age of 12-14 and is due to a thinner elastic chest and a relatively narrow diameter of the bronchi.

Pathological strengthening of vesicular breathing observed during the development of diabetic coma and other pathological conditions accompanied by the development of acidosis.

Hard breathing - increased, coarser, rattling vesicular breathing, characterized by an increase in both phases (both inhalation and exhalation), resembles puerile breathing, but differs from the latter in the mechanism of development.

The main reasons for the development of hard breathing are:

- uneven narrowing of the bronchi due to edema of the mucous membrane or wall accumulation of viscous exudate, which creates conditions for joining the vesicular breathing with the additional sound of air passing through the area of the narrowed bronchus (bronchitis, tuberculosis);
- foci of densification of lung tissue, alternating with undamaged (intact) tissue, due to improvement of sound conduction through an area of dense tissue (bronchopneumonia).

In addition, hard breathing is heard during accelerated breathing and increased ventilation of the lungs due to an increase in body temperature (fever).

Intermittent or saccade breathing - vesicular breathing with intermittent inhalation in the form of individual short breaths interrupted by the same pauses, while exhalation remains continuous. The main cause of jerky breathing is uneven contraction of the respiratory muscles (in children during crying; fatigue; muscle tremors in the cold; fever with a cold; neurasthenia, myopathy). If saccade breathing is heard in a strictly limited area, then it indicates the narrowing of small bronchi due to inflammation or their compression (tuberculosis, bronchitis).

Plan:

1. **Organizational measures** (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. **Control of the reference level of knowledge** (checking workbooks, performing lung auscultation in patients with respiratory system pathology and evaluating the obtained examination data), conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for the theoretical readiness of applicants to perform practical classes:

- the applicant must know the main purpose and tasks of lung auscultation;
- to know the mechanism of formation of the main respiratory sounds (bronchial and vesicular breathing), as well as where they are normally heard;
- to know the changes of the main respiratory sounds in various physiological and pathological conditions;
- know the technique and be able to perform lung auscultation;

List of didactic units:

– perform auscultation of the lungs in a healthy person;
– give an assessment of the main respiratory sounds in a healthy person;
– demonstrate the places of auscultation of laryngo-tracheal breathing in a healthy

person and give an assessment of noises;
– perform auscultation in a patient with lung pathology and characterize the main respiratory sounds.

2.2. **Questions to check basic knowledge on the topic of the lesson:**

1. Name the basic rules of auscultation.
2. Specify the points when auscultating the lungs.
3. What is the mechanism of formation of vesicular respiration?
4. What auscultatory criteria does vesicular breathing meet?
5. What is the mechanism of bronchial breathing?
6. What auscultatory criteria does bronchial breathing meet?
7. How can vesicular respiration change?
8. What types of pathological vesicular breathing do you know?
9. How can bronchial breathing change?
10. What types of pathological bronchial breathing do you know?

3. Formation of professional skills and abilities:

- formation of the ability to perform a clinical examination of a patient with respiratory pathology (the applicant must be able to demonstrate the technique of lung auscultation in patients with bronchopulmonary pathology),
- formation of the ability to give a clinical assessment of the results obtained during auscultation of the lungs, to carry out a clinical interpretation of the detected symptoms and syndromes in diseases of the respiratory organs.

3.1. Control materials for the final stage of the lesson:

Situational tasks:

1. What is the main respiratory noise normally heard over healthy lungs?
A. Amphoric breathing
B. Vesicular respiration*
B. Bronchial breathing
G. Puerile breathing
D. Hard breathing

2. Where is vesicular respiration formed:
A. In the larynx
B. In the trachea
V. In the bronchi
G. In the bronchioles
D. In the alveoli*

3. Pathological weakening of vesicular breathing can be heard at:
A Fatal pneumonia at the 1st stage*
B. Fateful pneumonia at the 2nd stage
B. If there is a large cavity in the lung
D. Exudative pleurisy in the zone of fluid projection
D. With complete lung atelectasis

4. What does the intensity of vesicular respiration depend on?
A. From the number of functioning alveoli
B. From the elasticity of the alveolar wall
A. From the power of the air flow during inhalation
D. From the thickness of the conductive medium
D. From all the listed factors*
5. Bronchial breathing.
AND. Has a certain musical pitch*
B. Does not have a certain musical pitch
B. Has a certain musical pitch under certain conditions
D. Does not have a certain musical pitch under certain conditions
D. No answer is correct

6. Where is physiological bronchial breathing heard?
A. In the suprascapular region
B. In the subscapular region
A. Over healthy lung tissue
G. On the neck in the place of projection of the thyroid cartilage*
D. Above the place of accumulation of air in the pleural cavity

7. Pathological weakening of vesicular breathing can be heard at:
A. Fateful pneumonia at the 2nd stage
B. Partial narrowing of the respiratory tract by a tumor or lymph node*
B. If there is a large cavity in the lung
D. Exudative pleurisy in the zone of fluid projection
D. With complete lung atelectasis

8. What are the reasons that can lead to weakening of vesicular breathing in pathology?
A. Emphysema of the lungs
B. Focal pneumonia
B. Lobar pneumonia of the 1st and 3rd stages of the clinical course
D. The initial stage of pulmonary edema
D. All answers are correct*

9. What is the name of increased physiological vesicular breathing in children?
A. Amphoric
B. Puerile*
V. Instead
G. Vikarne
D. Hypertrophic

10. Name the reasons for the formation of rigid vesicular breathing?
AND. Uneven swelling of the mucous membrane of the respiratory tract*
B. Bronchospasm
B. A decrease in the elasticity of the alveolar wall

D. Partial narrowing of the trachea by a tumor or lymph node
3.2. Requirements for work results, including before registration: substantiation of the syndromic diagnosis based on complaints, medical history and life data, clinical examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the subject of the lesson, answering 10 tests (*if necessary*).

4. Summary, announcement of assessment results, announcement of the topic of the next lesson.
Practical lesson No. 6

Subject: Auscultation of the lungs: additional respiratory noises (crepitation, wheezing, pleural friction noise)

Goal: Acquiring knowledge and mastering professional competences in lung auscultation, namely:
- to know the procedure and basic rules for lung auscultation;
- know the mechanism of formation of additional breathing noises,
- to learn to recognize additional breathing noises according to the relevant characteristics and to distinguish between them according to the appropriate criteria;
- learn to give them a clinical and diagnostic assessment based on the definition of additional respiratory sounds.

Basic concepts: Auscultation of the lungs is the main physical method of examining the patient, which is included in the general mandatory examination methods and is carried out regardless of the existing pathology and during preventive examinations. It was introduced into medical practice at the beginning of the 19th century, and remains an indispensable diagnostic method for researching the respiratory system. Auscultation of the lungs allows you to determine the nature and features of breathing types, compare them with the appropriate norm and, on this basis, evaluate the morphological structure and functional capacity of the organs of the respiratory system. In addition, auscultation allows, based on the detection of sound phenomena, to determine both the localization and nature of the respiratory system lesion, as well as to quickly monitor the course of the disease. The use of lung auscultation in combination with the main and additional examination methods used in pulmonology practice makes it possible to establish the leading syndrome of damage to the respiratory organs, on the basis of which a nosological diagnosis is determined and a treatment plan is prescribed in accordance with state and international standards for providing medical care to patients with therapeutic (in particular , pulmonary) profile.
At the class, the winners solve a specific problem: they learn the technique of auscultation of the lungs. Most of the questions discussed in the class are of a problematic nature. Thus, the analysis of changes in the main breathing noises, the appearance of additional breathing noises makes it possible to think about changes in both the airways and the lung parenchyma, the pleural cavity. Synthesizing the data obtained during auscultation of the lungs and using knowledge about the morphofunctional changes of the respiratory organs in pathology, taking into account the data of the survey, examination, palpation and percussion of the lungs,

students learn to differentiate the reasons that lead to a change in the normal auscultatory picture over the lungs.
Auscultation of the lungs is divided into direct and indirect (using a stethoscope or stethoscope).

Lung auscultation rules:

- The chest is bare. The room is warm. There is silence in the room.
- The position of the patient depends on the condition. standing Sitting lying down
- The location of the doctor and the patient should be such that ensures parallel air flows.
- The patient breathes with a half-open mouth, takes 3-4 inhalations, exhalations, then 10-20 seconds. Resting
- During auscultation, suggest taking several deep breaths and coughing.
- The stethophone endoscope fits tightly to the patient's body. Auscultation is performed on symmetrical sections of the chest, along topographic lines, intercostals, starting from the front surface.
- In front, bypassing the projection of the heart, in the back with the shoulder blades as wide as possible.
- During auscultation, the presence of noises is assessed both inward and outward; the duration of inhalation and exhalation is estimated.
- Describing the auscultatory picture, first characterize the main and then additional respiratory sounds.

In pathological conditions, against the background of the main respiratory noises, other sound phenomena are heard, which are called additional respiratory noises and are associated with various pathological processes occurring in the tracheobronchial system.

Additional breathing noises include:

- ***Wheezing***
- ***Crepitation***
- ***Pleural friction noise***

They occur in the trachea, bronchi, alveoli, lung cavities (under the conditions of the formation of abscesses, caverns and large bronchiectasis) in the pleural cavity, and are associated with the accumulation of pathological masses - mucus, pus, exudate, transudate, blood, sputum of various consistencies. These pathological accumulations of masses during the passage of air through the respiratory tract on inhalation and exhalation fluctuate taf
Wheezing – (ronchi) additional respiratory noises arising in the trachea, bronchi, and cavities connected to them due to the movement and oscillation of sputum, exudate, transudate, blood, as well as due to swelling of the mucous membrane and spasm of smooth muscles of the bronchi.
Classification of wheezing
The mechanism of formation of dry rales

The main mechanism is the narrowing of the lumen of the bronchi. It occurs due to:

- swelling of the mucous membrane of the bronchi
- spasm of bronchial smooth muscles
- accumulation of viscous secretion in the lumen of the bronchi

The mechanism of formation of wet wheezing

- Accumulation of foreign masses of liquid or semi-liquid consistency in the lumen of the bronchi.
- Shallow, bubbly sounding wet wheezing occurs when the lung tissue is compacted around the bronchi.

Crepitation- an additional respiratory noise that occurs in the valveoli at the moment of their disintegration at the height of inspiration when there is liquid on their surface or their impregnation with a pathological liquid. 100% a sign of inflammation of the lung tissue.

Crepitation occurs when:

1. Inflammation of the lungs.
2. Pulmonary edema.
3. Lung infarctions.

Pleural friction noise

- Pathological states of the pleura lead to changes in the physical properties of the pleural sheets. There is roughness, unevenness of the surface of the pleura.
- Pleural friction noise occurs in both phases of the respiratory cycle.

Characteristic signs of pleural friction noise

- It is heard during inhalation and exhalation.
- Does not disappear during coughing.
- Intensifies when pressing on the chest.
- It is heard during diaphragmatic breathing.

Pleural friction noise can be a sign of:

- The beginning of exudative pleurisy
- Pleural adhesions
- Dry pleurisy
- Lobe pneumonia
- Tuberculosis of the pleura
- Metastatic lesion of the pleura
- Exycosis due to prolonged vomiting or diarrhea

Differential diagnostic differences of additional respiratory noises

	ezing	**on**	**noise**

to exhalation	halation and n	eight	halation and n
to cough	nge	disappear, does not	disappear, does ge
to the diaphragmatic maneuver	ppear	rs	disappear

Plan:

1. **Organizational measures** (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge (checking workbooks, performing lung auscultation in patients with respiratory system pathology and evaluating the obtained examination data), conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for the theoretical readiness of applicants to perform practical classes:

- the applicant must know the main purpose, tasks and technique of lung auscultation;
- to know the mechanism of formation of additional respiratory noises (wheezing, crepitation and pleural friction noise);
- be able to recognize additional breathing noises;
- to be able to distinguish between additional breathing noises;
- to learn to give a comprehensive assessment of the auscultatory picture over the lungs, and to summarize the data obtained during the survey, examination, palpation of the chest and carrying out comparative and topographical percussion of the lungs, as well as auscultation of the lungs.

List of didactic units:

– perform lung auscultation in patients with lung pathology;
– detect additional respiratory sounds and characterize them;
– carry out a differential characterization of additional respiratory sounds.

2.2. **Questions to check basic knowledge on the topic of the lesson:**

1. Name the basic rules of auscultation.
2. How are additional breath sounds classified?
3. What is the mechanism of dry wheezing?
4. What is the mechanism of formation of wet rales
5. What is the similarity and what is the difference between wet loud and quiet wheezing?
6. What is crepitation, what is the mechanism of its occurrence?
7. Diagnostic value of crepitation
8. How to distinguish moist rales from crepitations during lung auscultation?

9. What is pleural friction noise, what are the mechanisms of its occurrence?
10. Diagnostic value of pleural friction noise.
11. How to distinguish pleural friction noise from crepitation and wheezing?

A problem of the STEP-2 type.
1. In patient N., 26 years old, during an objective examination, the right half of the chest lags behind in the act of breathing, from the lower third of the right shoulder blade - sharply weakened vesicular breathing. What part of the right lung is affected by these changes?
A. secondary
B. upper
C. middle and upper
D. lower
E. middle and lower
2. The patient heard a noise over the lungs, reminiscent of the pronunciation of the letter "f", during the entire inhalation and at the beginning of exhalation. What breath noise was heard?
A. bronchial breathing
B. vesicular respiration
C. amphoric breathing
D. saccadic breathing
E. puerile breathing

Standard answer to problem 1.- D if the patient's objective examination of the right half of the chest lags behind in the act of breathing, from the lower third of the right scapula - sharply weakened vesicular breathing, this indicates the defeat of which lower lobe of the right lung

The standard of the answer to problem 2. - Exactly vesicular breathing resembles the pronunciation of the letter "f", during the entire inhalation and at the beginning of exhalation.

3. Formation of professional skills and abilities:
- formation of the ability to perform a clinical examination of a patient with respiratory pathology (the applicant must be able to demonstrate the technique of lung auscultation in patients with bronchopulmonary pathology),
- formation of the ability to give a clinical assessment of the results obtained during auscultation of the lungs, to carry out a clinical interpretation of the detected symptoms and syndromes in diseases of the respiratory organs.

3.1. Control materials for the final stage of the lesson:
Situational tasks:
1. The patient's objective examination revealed: the chest is symmetrical, the right half of it lags behind during breathing, dulling of the percussion sound to the right from the corner of the shoulder blade to the bottom, bronchial breathing. What can be assumed in the patient?
A. Dry pleurisy
B. Echinococcus lung

C. Inflammatory infiltration of lung parenchyma
D. Pneumothorax
E. Lung abscess after its breakthrough into the bronchus

2. The patient's objective examination revealed: the chest is symmetrical, it participates evenly in the act of breathing, the lower borders of the lungs are lowered, the respiratory excursion is 1.5-2 cm, a percussion sound with a tympanic tone, breathing is vesicular, sharply weakened. What pathology can be assumed in the patient?
A. Pneumothorax
B. Infiltrative tuberculosis
C. Hydrothorax
D. Lung abscess
E. Emphysema of the lungs
F.
3. Weakening of vesicular breathing was found in the patient. What extrapulmonary factor led to this**?**
A. Accumulation of fluid in the pleural cavity
B. Accumulation of air in the pleural cavity
C. Fusion of pleural sheets
D. Adiposity
E. Splanchnoptosis

4. The patient has croup pneumonia in stage 3. What objective research data can be expected?
A. Dulling of percussion sound and bronchial breathing
B. Dulling of percussion sound and vesicular breathing
C. Dull-tympanic percussion sound and lack of breathing
D. Dull tympanic percussion sound and weakened vesicular breathing
E. Dull-tympanic percussion sound and amphoric breathing

5. The patient has right-sided exudative pleurisy (the amount of fluid is 1.5 liters). What kind of breathing would be determined on the healthy side?
A. hard
B. Bronchial
C. Vesicular hard with prolonged exhalation
D. Weakened vesicular
E. Enhanced vesicular

6. The additional respiratory noise heard in the patient only at the height of inhalation did not change after coughing. In the place of its listening, an increase in vocal tremor and dulling of the percussive sound is determined. Is this most likely?
A. A. Moist wheezing
B. B. Dry rales
C. B. Crepitation
D. G. Pleural friction noise

E. D. Noise of a cracked pot

7. What is the basis of crepitation?
A. A. Stenosis of the lumen of the bronchi
B. B. The presence of a cavity containing liquid and air
C. B. Deglutition of stuck alveoli on inhalation, on the walls of which fibrin has been deposited
D. G. The presence of bronchiectasis filled with pus
E. D. Friction of inflamed pleural sheets

8. Name additional respiratory sounds?
A. A. Wheezing
B. B. Crepitation
C. V. Pleural friction noise
D. D. All answers are correct
E. D. All answers are incorrect

9. Where are dry rales formed?
A. A. In the pleural cavity
B. B. In cavity formations of the lungs
C. V. In the bronchi
D. G. In the alveoli
E. D. In the larynx

10. Where does crepitation occur?
A. A. In the pleural cavity
B. B. In cavity formations of the lungs
C. V. In the bronchi
D. G. In the alveoli
E. D. In the larynx
Answer standard for tasks: 1-C, 2-E, 3-D, 4-D, 5-C, 6-C, 7-C, 8-D, 9-C, 10-D,
3.2. Requirements for work results, including before registration: substantiation of the syndromic diagnosis based on complaints, medical history and life data, clinical examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the subject of the lesson, answering 10 tests (*if necessary*).

4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 7

Subject: Questioning and general examination of patients with pathology of the cardiovascular system. Treatment of patients. Examination of the pulse and measurement of blood pressure.

Goal: Acquiring knowledge and mastering professional competences in the examination of patients with pathology of the cardiovascular system, namely:
- to know the main signs and to be able to interpret them during the examination of patients with pathology of the cardiovascular system;
- learn the methodology and technique of interviewing patients with cardiovascular system pathology;
- learn the sequence and technique of a general examination of a patient with a pathology of the cardiovascular system;
- be able to determine the pulse, know its characteristics, interpret pathological changes;
- be able to measure blood pressure, know the norm.

Basic concepts: Cardiovascular diseases are the most frequent cause of death of the population of Ukraine and most countries of the world. Arterial hypertension and ischemic heart disease rank first among diseases of the cardiovascular system and play a leading role in the formation of causes of cardiovascular death. In particular, at least 11 million residents of Ukraine have elevated blood pressure, so a deep study of risk factors for cardiovascular diseases, basic and additional methods of examining patients, symptomatology of diseases of the heart and blood vessels is an important task for the training of future doctors.

Complaintswith the pathology of the cardiovascular system, they can have coronary and non-coronarogenic origin and can be associated with arterial insufficiency (ischemia) and venous insufficiency (stasis).

Pain in the region of the heart

They distinguish:
— ischemic pain (angina, myocardial infarction);
— cardialgias associated with non-ischemic heart changes (pericarditis, myocarditis);
— pain in the area of the heart, not related to heart pathology (for example, dissecting aortic aneurysm, PE).

Direct causes of pain:
— insufficient coronary blood circulation, anginal pain in case of coronary blood flow disorders (angina, myocardial infarction), low diastolic pressure in the aorta (aortic insufficiency), coronary artery disease
— hemic hypoxemia and myocardial hypoxia (anemia);
— irritation of interoreceptors in the pericardium, aorta;
— reflex effects on coronary blood flow (cholecystitis, stomach ulcer).

Non-cardiogenic pain in the area of the heart can be caused by:
— pleurisy (increases with coughing, deep inhalation),
— cervical osteochondrosis (provoked by movements in the spine, aggravated by palpation of the vertebrae),
— damage to muscles, ribs (long-lasting, aggravated by palpation of the chest)

Palpitations and interruptions in the work of the heart

Palpitation is unusual for patients in terms of the intensity of the feeling of contractions (beating) of their heart. It occurs in organic (heart defects, myocarditis, cardiosclerosis, etc.) and functional (cardioneurosis) heart diseases. During the survey, they find out how the patient feels the heartbeat constantly or periodically (in the form of attacks), establish their connection with physical exertion, excitement, food intake, etc.:

— constant heartbeat is characteristic of organic lesions of the cardiovascular system and is often one of the frequent signs of endocarditis, myocarditis or severe myocardial infarction.

— periodic palpitations associated with physical exertion (running, walking) in most cases indicate a weakening of the heart muscle and can be caused by both organic and functional diseases.

— sudden attacks of palpitations, accompanied by rapidly developing signs of heart failure (shortness of breath, cyanosis, liver enlargement, edema), are characteristic of paroxysmal tachycardia.

Interruptions in the work of the heart - impulses of "contraction" in the area of the heart. They are usually found in heart rhythm disorders (more often in extrasystole, less often in other forms of arrhythmias), their nature can be accurately recognized with the help of an ECG. They find out whether the interruptions are constant or occur in the form of attacks, the frequency of their occurrence, duration and reasons for their occurrence (physical work, excitement, excessive smoking, intake of alcohol, strong coffee, tea, etc.).

The most common causes of palpitations and interruptions in the work of the heart:

— extrasystole;

— tachyarrhythmias (atrial flutter and fibrillation);

— bradyarrhythmias (heart blocks).

The sensation of pulsation in some parts of the body (neck, head, limbs) occurs, for example, with:

— aortic valve insufficiency,

— thyrotoxicosis,

— hypertensive disease.

Dyspnea

Shortness of breath is a frequent and important symptom of diseases of the cardiovascular system, indicating heart failure (HF). Characterizes tolerance to physical exertion. It is one of the criteria on the basis of which the stage and functional class of chronic heart failure is determined. In the early stages of heart failure, shortness of breath occurs only during physical exertion (fast walking, climbing uphill), and as the weakness of the heart muscle increases - during light physical exertion (getting out of bed, slowly walking around the room) or constantly bothers the patient, getting worse with the slightest movements. Patients with dyspnea of cardiac origin occupy a forced orthopneic position. Cardiac asthma is an attack of suffocation that occurs mainly at night. Shortness of breath, especially in the form of a suffocation attack, can be the equivalent of a pain syndrome in angina pectoris and myocardial infarction.

Cough and hemoptysis

Cough in heart disease can be:

— dry, with scanty sputum (blood stagnation in a small circle of blood circulation);

— dry barking cough (irritation of the branches of the vagus nerve due to enlargement of

the left atrium, aneurysm of the aorta);
— wet, with pink foamy sputum (with pulmonary edema)
Causes of hemoptysis:
— diapedesis of erythrocytes (blood stagnation in the small circle of blood circulation (MCC) due to left ventricular failure, mitral heart defects);
— rupture of small bronchial vessels (pronounced blood stagnation in the MCC due to left ventricular failure, mitral heart defects);
— TELA (heart attack-pneumonia);
— rupture of an aortic aneurysm into the respiratory tract (profuse pulmonary hemorrhage)
Swellingof cardiac origin is a symptom of right ventricular heart failure.
Signs of cardiac edema:
— first appear on the lower limbs;
— increase (or appear) by the end of the day;
— with increasing decompensation, they spread higher (thighs, lower back, etc.);
— dense;
— the skin above them is cold, the color is cyanotic
With severe heart failure, ascites develops.
Unlike edema in right ventricular failure, edema in venous insufficiency (thrombophlebitis, phlebothrombosis, varicose veins, extravasal compression, congenital vein pathology) is characterized by:
— are localized below the site of vein occlusion or in the area of varicose veins;
— may be asymmetrical;
— soft, warm skin;
— are accompanied by a feeling of heaviness and / or distending pains, significantly relieved when the affected limb is in an elevated position;
With chronic venous insufficiency, pigmentation and induration of the skin and subcutaneous tissue appear.
Swellings in lymphatic insufficiency (lymphostasis in congenital vascular pathology, after operations and injuries, blockage of lymphatic drainage by parasites or metastases) are characterized by:
— often asymmetric, soft (testy) consistency;
— painless;
— the skin is of normal color, the venous pattern is not clearly expressed;— the skin fold is thickened, but mobile (easily shifts and gathers into a fold);
— they can increase (in the warm season, after physical exertion) and decrease until they completely disappear (autumn-winter period, after a long rest).
Syncope (fainting)
Fainting begins with a feeling of nausea, ringing in the ears, then loss of consciousness follows. The leading factor in the genesis of fainting is a transient disturbance of blood supply to the brain.
There are various mechanisms of fainting:
— decrease in peripheral vascular resistance → drop in blood pressure (orthostatic hypotension, psychogenic fainting caused by hyperactivity of the vagus nerve);
— heart rhythm and conduction disturbances (Morganhi-Edems-Stokes syndrome,

paroxysmal rhythm disturbances);
— small cardiac output (aortic stenosis, rarely - aortic insufficiency);
— hemic hypoxia (cardiopulmonary diseases);
— disorders of cerebral blood circulation (hypertensive crisis, arterial hypotension, including drug-induced genesis).

Other complaints in patients with cardiovascular pathology

Digestive system:
— dull pain and heaviness in the right hypochondrium and epigastrium (stretching of the Glisson capsule of the liver with right ventricular failure);
— dyspeptic phenomena: decreased appetite, nausea, vomiting, flatulence, constipation (venous stasis in the stomach, intestines).

Urinary system:
— oliguria;
— nocturia

Central nervous system:
— faint;
— headaches, emotional lability, sleep disturbances, depression, indifference, agitation, tearfulness (manifestations of impaired blood supply to the brain due to ischemia, venous congestion, arterial hypertension).

If the patient has both hypertension and coronary artery disease, the history of this disease begins with a description of the disease that appeared earlier (hypertension and coronary artery disease are mutually risk factors). It is necessary to reflect the connection of the disease with transferred infectious (angina, scarlet fever) and other diseases (tuberculosis, acute respiratory viral infection, thrombophlebitis), physical and nervous overstrain (high blood pressure, coronary artery disease); describe the development of symptoms during the course of the disease. The presence of modified and unmodified risk factors, hereditary predisposition, bad habits, concomitant diseases that occur with damage to the cardiovascular system (diabetes mellitus, thyrotoxicosis) are found out.

At**general overview**the study pays special attention to:

Position of the patient in bed:
— orthopnea (cardiac asthma, pulmonary edema);
— forced horizontal position (unconsciousness, collapse, shock);
— sitting with an inclination forward and to the side (exudative pericarditis).

Skin covers:
— acrocyanosis (right ventricular heart failure);
— diffuse cyanosis ("blue" congenital heart defects, total heart failure);
— "mitral face";
— pallor (aortic heart disease);
— jaundice (severe heart failure with the formation of cardiogenic liver fibrosis);
— "coffee with milk" color (infective endocarditis);
— hyperemia, especially on the face (hypertensive crisis);
— nails in the form of "watch glasses" (congenital heart defects, infectious endocarditis).

Subcutaneous tissue:
— expressiveness of the venous pattern;

— the presence of edema

Presence of lymphadenopathy (infective endocarditis, acute rheumatic fever).

Musculoskeletal apparatus:

— fingers in the form of "drumsticks" (congenital heart defects, infectious endocarditis);
— joint damage (rheumatoid arthritis, systemic lupus erythematosus).
— Musset's symptom - aortic insufficiency.

Study of the pulse. The main characteristics of the pulse on the radial artery.

Determination of symmetry of the pulse on the radial arteries: the doctor covers the patient's left hand above the radiocarpal joint with his right hand, and the right hand with his left hand, so that the tips of the II-IV fingers of the examinee are located on the front surface of the examinee's radius bone between its outer edge and the tendon of the thumb and the palms were located on the back of the forearm. At the same time, one should strive to ensure that the position of the hands is comfortable for both the doctor and the patient. Focusing on the sensations in the fingertips, the doctor sets them in the position in which the pulse is detected and determines the synchronicity of the occurrence of pulse waves on both arteries (that is, the simultaneous occurrence of pulse waves on the left and right hand) and their uniformity. In a healthy person, the pulse on both radial arteries is synchronous and the same. In patients with severe stenosis of the left atrioventricular opening due to expansion of the left atrium and compression of the left subclavian artery, the pulse wave on the left radial artery (when compared with the right) is smaller and delayed. In Takayasu syndrome (obliterating arteritis of the branches of the aortic arch), a pulse may be absent in one of the arteries. An unequal and non-synchronous pulse is called pulsus differens. If the pulse is synchronous and the same, the remaining properties of the pulse are determined by palpating one hand.

Rhythm and pulse rate. They determine whether pulse waves occur at equal (rhythmic pulse) or unequal time intervals (arrhythmic pulse). The appearance of separate pulse waves, smaller in size and occurring earlier than the usual time, followed by a longer (compensatory) pause, indicates extrasystole. In atrial fibrillation, pulse waves occur at irregular intervals and are limited in size. If the pulse is rhythmic, it is considered within 20 or 30 seconds. Then determine the pulse rate for 1 minute by multiplying the obtained value by 3 or 2, respectively. If the pulse is irregular, it is read for at least 1 minute.

Voltage and filling of the pulse

The doctor's hand is set in a typical position. The proximal finger gradually presses the artery to the radius. With a finger located distally, the moment of cessation of arterial pulsation is detected. The pulse voltage is judged by the minimal effort that had to be exerted to completely compress the artery with a proximally located finger. At the same time, with a finger located distally, it is necessary to catch the moment when the pulsation stops. The voltage of the pulse depends on the systolic blood pressure: the higher it is, the more intense the pulse. At high arterial systolic pressure, the pulse is hard, at low pressure - soft. Pulse voltage also depends on the elastic properties of the artery wall. When the artery wall is thickened, the pulse will be hard.

When examining the filling of the pulse, the examinee places his hand in a position typical for examining the pulse. At the first stage, with a finger located proximally on the hand, the artery is completely compressed until the pulsation stops. The moment of cessation of pulsation is caught with a finger located distally. At the second stage, the finger is raised to

the level when the pad of the palpating finger will barely feel a pulsation. The filling is judged by the distance to which the pressing finger needs to be raised to restore the original amplitude of the pulse wave. This corresponds to complete straightening of the artery. The filling of the pulse, thus, is determined by the diameter of the artery at the moment of the pulse wave. It depends on the stroke volume of the heart. If the stroke volume is high, the pulse is full, if it is low, it is empty.

Size and shape of the pulse.

The researcher places the right hand in a typical research position. Then, with the middle (with 3 palpable) fingers, he presses the artery to the radius bone until it is completely squeezed (he checks this with the distally located finger) and, focusing on the sensation in the proximally located finger, determines the strength of pulse impulses. The size of the pulse is greater, the greater the tension and filling of the pulse, and vice versa. A full solid pulse is large, empty and soft - small. Having placed the right hand in a position typical for palpation of the pulse and focusing on the feeling in the palpating fingertips, the researcher should determine the rate of rise and fall of the pulse wave. The shape of the pulse depends on the tone of the arteries and the speed of their systolic filling: with a decrease in the tone of the vessels and insufficiency of the aortic valves, the pulse becomes fast, with an increase in the tone of the vessels or their compaction - slow.

Determination of pulse deficiency.

The researcher determines the pulse rate, which the assistant at the same time auscultatively counts the number of heart contractions in 1 minute. If the heart rate is greater than the pulse rate, there is a pulse deficit. The size of the deficit is equal to the difference of these 2 values. Deficiency of the pulse is detected with an arrhythmic pulse (for example, with atrial fibrillation).

Vascular research is completed by sequential palpation of other arteries: carotid, temporal, brachial, ulnar, femoral, popliteal, posterior tibial, posterior foot arteries.

Blood pressure measurement

Blood pressure is the pressure of blood in the large arteries of a person. There are two indicators of blood pressure:

- Systolic (upper) arterial pressure is the level of blood pressure at the moment of maximum contraction of the heart.
- Diastolic (lower) arterial pressure is the level of blood pressure at the moment of maximum relaxation of the heart.

Blood pressure is measured using a special device - a sphygmomanometer, or, as it is also called, a tonometer. It consists directly of a sphygmomanometer, which is used to compress the brachial artery and register the pressure level, and a phonendoscope, which is used to listen to the pulsation tones of the artery. In order to measure blood pressure, it is necessary to wrap the tonometer cuff around the patient's shoulder (that is, above the elbow by a couple of centimeters). Next, the head of the phonendoscope is applied to the area of the ulnar fossa, a little to the inside. After that, air is pumped into the cuff with a pear. Thus, the brachial artery heals. Usually it is enough to bring the pressure in the cuff to 160 - 180 mm Hg, but sometimes it is necessary to raise the pressure level even higher, if the pressure is measured in a patient suffering from arterial hypertension. Having reached the BP level, the air from

the cuff begins to be gradually released with the help of a valve. At the same time, the pulsation tones of the brachial artery are listened to. As soon as the pulsation beats of the artery appear in the phonendoscope, this level of blood pressure is considered upper (systolic blood pressure). Then the air continues to be released, and the tones gradually weaken. As soon as the pulsation stopped being heard, this blood pressure level is considered lower (diastolic). In addition, it is possible to measure the pressure without a phonendoscope. Instead, the blood pressure level is determined by the appearance and disappearance of the pulse on the wrist. Today, there are electronic devices for measuring blood pressure. Sometimes it is necessary to measure blood pressure on both arms, as it can be different. Pressure measurement should be carried out in a calm environment, the patient should sit quietly. Optimal BP - SBP (systolic blood pressure) < 120/ DBP (diastolic blood pressure) < 80 mm Hg.

- Normal AT SBP 120-129/DBP 80-84 mm Hg.
- High-normal AT SBP 130-139/DBP 85-89 mm Hg.
- 1st degree of hypertension - SAD 140-159/DAD 90-99.
- 2nd degree AG - SAD 160-179/DAD 100-109.
- 3rd degree of hypertension - SAD 180 and above/DAD 110 and above.
- Isolated systolic hypertension - SBP greater than or equal to 140/DBP below 90.

Plan:

1. **Organizational measures** (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. **Control of the reference level of knowledge** (checking of workbooks, surveying and examination of patients with pathology of the circulatory system and evaluation of the obtained examination data), conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for theoretical readiness of applicants and performance of practical classes:

- the applicant must know the main complaints of patients with pathology of the heart and blood vessels,
- the main differences between coronary pain and non-coronary pain,
- to know the general examination plan of patients with cardiovascular pathology,
- to know the main characteristics of the pulse on the radial artery,
- to know the method of measuring blood pressure and the degree of gradation of BP in normal and pathological conditions.

List of didactic units:

- conduct a survey of a patient with a pathology of the cardiovascular system, establish the main complaints, give them an assessment.
- conduct a general examination of a patient with pathology of the heart and blood vessels,
- evaluate pulse parameters on the radial artery,
- measure blood pressure and evaluate the results.

2.2. **Questions to check basic knowledge on the topic of the lesson:**
1. List the main complaints of patients with heart disease.
2. Describe the pains of a coronary nature and their difference from non-coronary pains.
3. What is the mechanism of shortness of breath in patients with heart failure?
4. What are the possible causes of hemoptysis in patients with pathology of the cardiovascular system?
5. What are the causes of syncope (fainting) in diseases of the cardiovascular system?
6. What is included in the general examination of patients with heart pathology?
7. What characteristics of the pulse are studied?
8. What is pulse filling and what does it depend on?
9. What is pulse deficiency and in what pathology does it occur?
10. Describe the conditions and methods of blood pressure measurement.
11. Which blood pressure values are normal, and which indicate pathology?

A problem of the STEP-2 type.
1. A 52-year-old patient periodically notes a short-term feeling of tightness behind the sternum in the morning while walking to work. During an attack, the patient slows down or stops. For the first time, the feeling of compression arose about a month ago. What pathology can be thought of based on the patient's complaints?
+A. Angina
B. Myocardial infarction
S. Myocarditis
D. Pericarditis
E. Mitral stenosis
2. A 48-year-old patient complains of an attack of pain behind the sternum, which does not stop for more than 30 minutes, pain of a squeezing and burning nature, accompanied by radiation to the left shoulder, arm, under the left shoulder blade. What pathology can be thought of based on the patient's complaints?
A. Angina
+V. Myocardial infarction
S. Myocarditis
D. Cardiac asthma attack
E. Thromboembolism of the pulmonary artery

Standard answer to problem 1. -A. If the patient complains of squeezing pains behind the sternum during physical exertion for a month, one can think about angina pectoris.
The benchmark for the answer to problem 2. –B . Pains behind the sternum that appeared suddenly, of a squeezing and burning nature lasting more than 30 minutes with radiation to the left shoulder, arm, under the left scapula are characteristic of a myocardial infarction.

3. Formation of professional skills and abilities:
- formation of the ability to perform a clinical examination of a patient with a pathology of the cardiovascular system (the candidate must be able to demonstrate the methodology of

surveying and general examination of patients with heart pathology, as well as blood pressure measurement and evaluation of the pulse on the radial artery),
- formation of the ability to give a clinical assessment of the results obtained during the survey and general examination, to carry out a clinical interpretation of the detected symptoms and syndromes in diseases of the cardiovascular system.

3.1. Control materials for the final stage of the lesson:

Situational tasks:

1. Diseases of the cardiovascular system are characterized by a forced position in bed:

A. Sitting, resting his hands on the edge of the bed

B. Sitting with the legs down

S. Lying on the left side

D. Sitting in bed, leaning forward, pressing the front wall of the abdomen with your hands

E. Lying on the back

2. Diseases of the cardiovascular system are characterized by the shade of the skin:

A. Zhovtyanichnyi

V. Bronze

S. Voskovidniy

D. Cyanotic

E. Rozhevy

3. Cardiac edema is initially localized:

A. On the face

A. On the lower limbs

S. In arms

D. On the eyelids

Well, I was losing

4. Pain behind the sternum, relieved by nitroglycerin, is characteristic of:

A. Arterial hypertension

B. Acquired heart defects

S. Myocarditis

D. Angina

E. Pericarditu

6. Pulsation of the carotid arteries is most characteristic of:

A. Mitral insufficiency

B. Pericarditis

S. Mitral stenosis

D. Aortic insufficiency

E. Arterial hypertension

7. Pale skin is a sign of:

A. Deficiencies of the tricuspid valve

V. Mitral stenosis

S. Aortic insufficiency

D Aortic stenosis

E. Mitral insufficiency

8. A 52-year-old patient periodically notes a short-term feeling of tightness behind the sternum in the morning while walking to work. During an attack, the patient slows down

or stops. For the first time, the feeling of compression arose about a month ago. What pathology can be thought of based on the patient's complaints?
A. Angina
B. Myocardial infarction
S. Myocarditis
D. Pericarditis
E. Mitral stenosis
9. A 48-year-old patient complains of an attack of pain behind the sternum, which does not stop for more than 30 minutes, pain of a squeezing and burning nature, accompanied by radiation to the left shoulder, arm, under the left shoulder blade. What pathology can be thought of based on the patient's complaints?
A. Angina
B. Myocardial infarction
S. Myocarditis
D. Cardiac asthma attack
E. Thromboembolism of the pulmonary artery
10. A 46-year-old woman suffering from thrombophlebitis, while in the surgical department, on the 7th day after an operation for acute cholecystitis, when she tried to sit down, suddenly felt nausea. What pathology can be thought of based on the patient's complaints?
A. Angina was detected for the first time
B. Myocardial infarction
S. Myocarditis
D. Cardiac asthma attack
E. Thromboembolism of the pulmonary artery

3.2. Requirements for work results, including before registration: substantiation of the syndromic diagnosis based on complaints, medical history and life data, clinical examination.
3.3. Control materials for the final stage of the lesson: solving two clinical problems on the subject of the lesson, answering 10 tests (*if necessary*).

4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 8

Subject: Inspection and palpation and percussion of the atrial area and large vessels.

Goal: Acquiring knowledge and mastering professional competences in the examination of patients with pathology of the cardiovascular system, namely:
- to know and be able to detect characteristic signs during the examination of the atrial region in patients with heart and vascular pathology,
- to know the technique of palpation of the atrial region, and the main characteristics of the apical and cardiac impulse,

- to know the reasons for changes in the apical and cardiac impulse in patients with heart pathology,
- to know the technique of conducting percussion in order to determine the limits of the relative dullness of the heart,
- to know the technique of conducting percussion in order to determine the limits of relative absolute dullness of the heart,
- to know the reasons for changes in the displacement of the limits of relative and absolute dullness of the heart.

Basic concepts: Cardiovascular diseases are the most frequent cause of death of the population of Ukraine and most countries of the world. Examination, palpation of the atrial region with determination of the properties of the apical and heart impulse, determination of the limits of relative and absolute dullness of the heart with the help of percussion are important methods of physical examination of patients with cardiovascular pathology.

Inspection and palpation of the atrial region

Examination of the heart area includes the following steps:

- detection and presence of chest deformation in the atrial region: a) cardiac hump, b) exudative pericarditis;
– the presence of an apical shock and determination of its localization;
- the presence of pathological pulsation in the atrial region;
- presence of epigastric pulsation.

An examination of the area of the heart allows you to identify some symptoms characteristic of heart disease. These include: cardiac hump, visible pulsation in various parts, dilatation of skin veins in the heart. Pay attention to the features of the chest skeleton. A cardiac hump is a protrusion of the chest in the heart, associated with a significant increase in its size. A significantly increased apical impulse can be determined visually, and its confusion to the left gives very important information, which is supported by palpation and percussion research. A protrusion in the area of the aorta due to its aneurysm can have diagnostic value. Increased pulsation in the area of the pulmonary artery is determined in case of high pulmonary arterial hypertension. Epigastric pulsation is determined in healthy people in a clinostatic position and is caused by pulsation of the abdominal aorta. With a deep breath, it either weakens or changes. With a deep inspiration, the pulsation of the right ventricle increases because the diaphragm descends and the right ventricle is closer to the epigastric angle.

Palpation of the atrial region.

Research plan:
- evaluation of the localization and properties of the apical shock;
- the presence of the "cat's purring" symptom;
- palpation of the pulsating liver and abdominal aorta.

Technique of palpation of the atrial region: the palm of the right hand must be placed horizontally on the left half of the chest in the projection of the top of the heart, while the fingers are directed to the armpit between the IV and VI ribs. (approximate palpation). If the impulse is not detected when the patient is lying down, palpate when the body is bent forward, during deep exhalation, or when lying on the left side. After the doctor feels the impulse of the apex of the heart, the position of the palm is changed to vertical and palpation

continues with the tips of three bent fingers in order to assess the characteristics of the apex impulse in detail.
Palpation of the atrial region allows to more clearly determine the localization of the apical impulse, to give an assessment of its properties: prevalence, height, strength (resistance). The height of the apical shock is the amplitude of the oscillation of the part of the chest in the area of the projection of the apex of the heart. The strength or resistance of the apical impulse is determined by the resistance that the apex of the heart makes to the palpating fingers during systole.
Palpation also provides an opportunity to find the phenomenon of atrial fibrillation - "*cat purring*»
The apical impulse is normally determined in the fifth intercostal space 1-1.5 cm medially from the left mid-clavicular line.
With a change in the size of the heart and its cavities, different variants of displacement of the apical impulse are possible. With hypertrophy of the left ventricle, the apical impulse shifts outward, with dilatation of the left ventricular cavity, the apical impulse shifts downward, with a combination of hypertrophy and dilatation, the apical impulse shifts outward and downward.
Properties *apical thrust:*prevalence, height, strength.
Prevalence apical thrust is normally 2 cm^2. In various physiological and pathological conditions, the apical impulse can be diffuse and limited.
Height apical impulse can be normal, high and low.
Strength (Resistance) horse push:*strong*or a resistant apical push - a sign of left ventricular hypertrophy in aortic defects, arterial hypertension, mitral insufficiency;*attenuated*apical impulse is determined in emphysema of the lungs, obesity, left-sided exudative pleurisy, exudative pericarditis (with a small amount of fluid).
Domed the apical impulse is localized in the VI-VII intercostal space along the left front, middle axillary line, diffuse, high, strong, is determined in the case of insufficiency of the aortic valves.
Symptom of atrial fibrillation – it's a tactile sensation that resembles stroking a purring cat, hence the name "cat purr."
The method of determining "cat's purr": the palm of the right hand is placed flat on all points of auscultation of the heart, without pressing firmly. If chest wall tremor is detected, determine the relationship with the apical impulse: it precedes it (diastolic "cat's purr") or occurs simultaneously (systolic "cat's purr").
This phenomenon has great diagnostic value and is due to the presence of low-frequency intracardiac murmurs in organic heart defects.
Epigastric pulsationIn order to determine the epigastric pulsation, place the palm of the hand under the xiphoid process, press on the front abdominal wall, establish whether there is pulsation, the relationship to the phases of breathing. Pay attention to whether the fingers diverge during the pulsation, check the synchrony of the pulsation with the apical impulse and the pulse on the jugular veins. Check for Plesh's symptom - swelling of the neck veins when pressing on the liver. Then you need to move your palm lower and check for pulsation. Epigastric pulsation can be caused by pulsation of the heart, liver, and aorta. Epigastric pulsation appears with significant hypertrophy and dilatation of the right ventricle in patients with mitral stenosis, tricuspid valve insufficiency, chronic pulmonary heart disease.

Percussion. Percussion allows you to determine the position, size, configuration of the heart and vascular bundle. Percussion can be performed in a horizontal and vertical position of the patient. At the same time, it is necessary to take into account the fact that the dimensions of cardiac dullness in the vertical position are smaller than in the horizontal position. This is due to the mobility of the heart and the displacement of the diaphragm when the body position changes.

The right contour of the dullness of the heart and the vascular bundle is formed in the direction from top to bottom by the superior vena cava to the upper edge of the III rib, downwards by the right atrium. The left contour is formed from above by the left part of the arch of the aorta, then by the pulmonary trunk, at the level of the III rib by the left atrium, and downwards by the narrow strip of the left ventricle. The front surface is formed by the right ventricle, the back - by the left ventricle. The heart, as an airless organ, gives a dull sound when percussion. On the sides, it is partially covered by the lungs, due to which relative or absolute dullness is determined. The relative dullness of the heart corresponds to its real limits and is a projection of the front surface of the heart onto the chest, while absolute dullness defines only the front surface of the heart, which is not covered by the lungs.

Determination of the relative dullness of the heart. When determining the limits of relative cardiac dullness, it is necessary to percuss along the intercostals to avoid lateral propagation of vibrations along the ribs. Percussion of the heart should be performed by applying a percussive blow in the direction from the clear
percussion sound to a duller one, that is, from the lungs to the heart. Marking the border of the heart is carried out along the edge of the plesimeter finger, turned to the organ that gives a louder percussion sound. First, the right, then the left and upper limits of the relative dullness of the heart are percussed.

When determining the right border of the relative dullness of the heart, the height of the diaphragm is first determined. To determine the standing height, the diaphragm is percussed with blows of medium force along the right mid-clavicular line from top to bottom along the intercostal space, starting from the second, until a muffled sound appears - normally in the fifth intercostal space. Then the plesimeter finger is placed on one intercostal space above, in the III-IU intercostal space, parallel to the right border of the heart, and, moving towards the heart, percussion blows of medium force are applied until the percussion sound changes, that is, the transition of a clear the sound is muffled. In a healthy person, this border is located 1-1.5 cm outward from the right edge of the sternum and is formed by the right atrium.

Determining the left limit of relative cardiac dullness begins with finding the apical impulse that coincides with this limit. If the apical impulse is not found, then percussion is performed in the intercostal space, starting from the middle axillary line. The plesimeter finger is placed parallel to the left border that is being sought and, going in the direction of the heart, percussive blows of medium force are applied until a clear percussive sound turns into a dull one. In a healthy person, the left border of the relative dullness of the heart is located 1-1.5 cm medially from the mid-clavicular line and is formed by the left ventricle.

When determining the upper limit of the relative dullness of the heart, the finger-plesimeter is placed near the left edge of the sternum parallel to the ribs and, starting from the second intercostal space, is lowered down, applying blows of medium force. When the dulling of the percussion sound appears, marks are made on the upper edge of the finger. In healthy people,

the upper limit of relative cardiac dullness is located along the upper edge of the III rib and is formed by the pulmonary artery and the left atrial appendage.
With many pathologies of the cardiovascular system, there are shifts in the limits of the relative dullness of the heart. Thus, with aortic defects, hypertrophy of the left ventricle is observed (the so-called aortic configuration of the heart). With mitral defects of the heart, the waist of the heart is flattened due to the enlargement of the left atrium and right ventricle (the so-called mitral configuration of the heart).
Having determined the limits of the relative dullness of the heart, measure the diameter of the heart with a centimeter tape, for which the distance from the extreme points of the limits of the relative dullness of the heart to the front midline is determined. Normally, the distance from the right border of the relative dullness, which is located in the IU intercostal space, to the front midline is 3-4 cm, and the distance from the left border of the relative dullness of the heart, located in the U intercostal space, to the same line is 8- 9 cm. The sum of these values is defined as the diameter of the relative dullness of the heart and is normally equal to 11-13 cm.
In order to have an idea about***configuration of the heart,*** perform percussion in the higher intercostal spaces on the left and right, that is, in II, III, IU intercostals on the right and in U, IU, III, II on the left. The points obtained during percussion connect with each other and, thus, determine the configuration of the heart.
The width of the vascular bundle is determined in the II intercostal space on the right and left in the direction from the mid-clavicular line to the sternum, using soft percussion. When dulling of the percussion sound appears, a mark is made on the edge of the finger, turned to a clear lung sound. In healthy people, it is 5-6 cm.
In healthy people, there is an obtuse angle along the left heart contour between the vascular bundle and the left ventricle. In such cases, they speak of a normal configuration of the heart. In pathological conditions, with the expansion of the heart, mitral and aortic configurations are distinguished. The mitral configuration of the heart is characterized by expansion, first of all, of the right ventricle of the auricle of the left atrium, the pulmonary trunk of the left pulmonary artery; it is found in mitral heart defects. With aortic defects - the heart acquires an aortic configuration, in which isolated enlargement of the left ventricle is noted.
Determination of absolute dullness of the heart. Absolute heart dullness corresponds to the area of the front wall of the heart that is not covered
lungs, which is formed by the right ventricle. When percussion of this area of the heart, a dull sound is noted. Soft percussion is used to determine the limits of absolute dullness of the heart. First, the right border of the absolute dullness of the heart is determined, then the left, and after that - the upper. When determining the right border, the plesimeter finger is placed on the right border of the relative dullness of the heart parallel to the right edge of the sternum and, applying soft percussion blows, move it inside until a completely dull sound appears. The limit is marked on the outer edge of the finger, turned to a clear sound. In healthy people, the right border is found along the left edge of the sternum. When determining the left border of the absolute dullness of the heart, the finger-plesimeter is placed parallel to the left border of the relative dullness, retreating slightly outward from it, soft percussion blows are applied until a dull sound appears. Normally, the left border is 1.5-2 cm inward from the left border of relative dullness of the heart. The upper limit of absolute cardiac dullness is normally placed on the IV rib. The plesimeter finger is placed to

the previously found upper limit of relative cardiac dullness and quietly percussed, moving the finger down until a dull sound appears.

Changes in the limits of cardiac dullness.

Changes in the limits of relative and absolute dullness of the heart depend on the height of the diaphragm, heart enlargement and changes in the lungs. The displacement of the limits of the relative dullness of the heart is caused, first of all, by the expansion (dilatation) of the heart cavities and, to a lesser extent, by hypertrophy of the myocardium. In severe heart diseases (dilated cardiomyopathy, cardiosclerosis, heart defects), when circulatory insufficiency develops, the relative dullness limits shift in all directions. With a sharp increase in all parts of the heart, one speaks of a "bull's heart". Expansion of the borders to the left indicates hypertrophy or expansion of the left ventricle; up - about the expansion of the left atrium or the bulging of the pulmonary artery arch; to the right - about hypertrophy or expansion of the right ventricle. Expansion of the borders to the left is found in aortic valve insufficiency or stenosis of its mouth, mitral valve insufficiency, as well as in other acquired and congenital heart defects and hypertension. Expansion of the borders to the right indicates stenosis of the left venous opening, narrowing of the pulmonary artery, pulmonary heart. Upward expansion often occurs with stenosis of the mitral orifice.

A decrease in the area of absolute dullness of the heart is noted in emphysema of the lungs, when the front edges of the expanded lungs cover a larger than normal part of the front surface of the heart. At the same time, the absolute dullness shifts downward, which depends on the lowering of the diaphragm, on the left dome of which the heart is located. As a result, the upper limit of the absolute dullness of the heart can pass along the V and even the VI ribs with emphysema of the lungs. A decrease in the area of absolute dullness also occurs during an attack of bronchial asthma due to the acute spread of the lungs.

With left-sided pneumothorax, the gas-distended left anterior pleural sinus covers the front surface of the heart on the left side and thus contributes to reducing the absolute dullness of the heart. With right-sided pneumothorax, the right anterior pleural sinus shifts to the left, and therefore the area of absolute dullness of the heart is reduced on the right side.

A decrease in absolute dullness of the heart is accompanied by an accumulation of air in the pericardium. At the same time, the heart is pushed back, and at the place of dullness, a tympanic sound is heard on percussion. A decrease in the absolute dullness of the heart is observed: with subcutaneous emphysema in the area of the heart, when due to the accumulation of air in the subcutaneous tissue, percussion in the area of the heart produces a tympanic sound instead of a dull one; with a low position of the diaphragm, due to enteroptosis, when the intrathoracic pressure decreases, the lungs are spread out and their front edges are pushed against the heart.

An increase in the area of the absolute dullness of the heart occurs with: 1) shrinkage of the front edges of the lungs, as this exposes a larger than normal part of the front surface of the heart; 2) inflammatory compaction of the front edges of the lungs; 3) left-sided exudative pleurisy; 4) large tumors of the posterior mediastinum due to close adherence of the heart to the chest wall due to the pressure of the tumor, as well as increased intrathoracic pressure when the front edges of the lungs move away from the surface of the heart.

Plan:

1. Organizational measures (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge (checking workbooks, performing lung auscultation in patients with circulatory system pathology and evaluating the obtained examination data), conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for theoretical readiness of applicants and performance of practical classes:

- the applicant must know the main changes that can be observed during the examination of patients with heart pathology,
- know the goals and methods of palpation of the heart,
- to know the characteristics and properties of apical and heart impulses in normal and pathological conditions,
- to know the goals and methods of determining the limits of relative and absolute dullness of the heart,
- to know the causes and nature of the displacement of the borders of the heart in various pathological conditions.

List of didactic units:

- conduct an examination of a patient with a pathology of the cardiovascular system, establish the main changes, give them an assessment,
- palpate the atrial region in a patient with a pathology of the cardiovascular system, give an assessment of the apical and heart impulses,
- perform percussion and determine the limits of the relative dullness of the heart, give an assessment in the presence of changes,
- perform percussion and determine the limits of absolute heart dullness, give an assessment if there are changes.

2.2. **Questions to check basic knowledge on the topic of the lesson:**

1. What is "carotid dance" and in what pathology does it occur?
2. In what diseases is "heart hump" found and what does it characterize?
3. In which heart diseases is "Facies mitralis" found?
4. Describe the algorithm for palpation of the atrial region.
5. Name the main characteristics of an apical thrust.
6. How does the apical impulse change with aortic heart defects?
7. How does the apical impulse change with mitral heart defects?
9. What is a heartbeat, describe it.
10. What is the "cat's purring" symptom, in which heart diseases does it occur.
11. Describe the algorithm for determining the limits of relative and absolute dullness of the heart.
12. Name the reasons for shifting the limits of the relative dullness of the heart to the left.
13 Name the reasons for shifting the limits of the relative dullness of the heart to the right.

14. What causes can lead to a shift in the limits of absolute dullness of the heart?

A problem of the STEP-2 type.

1. The apical impulse is shifted to the left by 2 cm, diffuse, high, strong, resistant. The left border of relative cardiac dullness is shifted to the left. The reason for these changes.
A. Angina
B. Mitral stenosis
S. Insufficiency of the tricuspid valve
D. Pericarditis
+E. Aortic insufficiency
2. When palpating the atrial area above the aorta during systole, a "cat's purr" is determined. The apical impulse is high, spilled, resistant. The left border of the relative dullness of the heart is shifted to the left. Diagnostic assumption.
+A. Aortic stenosis
B. Mitral insufficiency
S. Mitral stenosis
D. Aortic insufficiency
E. Aortic aneurysm

Standard answer to problem 1.*+E. Aortic insufficiency.* Displacement of the apical impulse to the left by 2 cm, while it is diffuse, resistant, as well as displacement of the left border of the relative dullness of the heart to the left is characteristic of aortic insufficiency.
Standard answer to problem 2.*+A. Aortic stenosis.*The "cat's purr" symptom during systole is pathognomonic for aortic stenosis.

3. Formation of professional skills and abilities:
- formation of the ability to perform a clinical examination of a patient with a pathology of the cardiovascular system (the candidate must be able to demonstrate the technique of examining the atrial region in patients with a pathology of the heart, as well as conducting palpation and percussion to determine the limits of relative and absolute dullness of the heart),
- formation of the ability to give a clinical assessment of the results obtained during the examination, palpation of the atrial region and percussion of the heart borders, to carry out a clinical interpretation of the detected symptoms and syndromes in diseases of the cardiovascular system.
3.1. Control materials for the final stage of the lesson:
Situational tasks:

1. In the patient, during the examination of the atrial region, a pulsation to the right of the handle of the sternum in the I-II intercostal space is determined. Your diagnostic assumption.
+A. Aneurysm of the ascending aorta
B. Pulsation of the liver

C. Pulsation of the abdominal aorta
D. Aneurysm of the heart
E. Pulmonary hypertension

2. The apical impulse is shifted to the left by 2 cm, diffuse, high, strong, resistant. The left border of relative cardiac dullness is shifted to the left. The reason for these changes.

A. Mitral insufficiency
B. Mitral stenosis
S. Aortic stenosis
D. Pericarditis
+E. Aortic insufficiency

3. Aortic pulsation is visible in the second intercostal space on the right. The vascular bundle is 16 cm. The reason for these changes.

A. Aortic stenosis
B. Aortic insufficiency
S. Mitral stenosis
+D. Aortic aneurysm
E. Mitral insufficiency

4. Percussion of the limits of relative dullness of the heart: right - 3.5 cm outward from the right edge of the sternum; upper - the upper edge of the II rib along the left parasternal line; left 1 cm inward from the left middle clavicle line. What parts of the heart are changed?

A. Dilatation of the left ventricle
B. Dilatation of the left and right ventricle
S. Dilatation of the left atrium and left ventricle
+D. Dilatation of the left atrium and right ventricle
E. Dilatation of the right atrium and right ventricle

***5. With mitral stenosis, the following limits of relative dullness of the heart shift outward**:*

A. Right and left
B. Left and upper
+S. Upper and right
D. Right, top, left
E. Liva

***6. With stenosis of the aorta, the relative dullness of the heart moves outward**:*

A. Right and left
B. Right and upper
+S. Left
D. Rights
E. Right, left and upper

***7. With insufficiency of aortic valves, the limit of relative dullness of the heart shifts**:*

A. Right and up
A. Right and left
S. Up and to the left
+D. Left
E. To the right

8. When palpating the heart, a chest tremor is detected at the apex, which does not coincide with the pulsation on the carotid artery. This is typical of;
+1) Mitral stenosis
2) Mitral insufficiency
3) Aortic stenosis
4) Aortic insufficiency
5) Shortcomings of the tricuspid valve.

9. When palpating the heart, a chest tremor is detected at the apex, which coincides with the pulsation of the carotid artery. This is typical for;
1) Mitral stenosis
2) Mitral insufficiency
+3) Aortic stenosis
4) Aortic insufficiency
5) Shortcomings of the tricuspid valve.

10. Absolute cardiac dullness is formed
1) Left atrium
2) Left ventricle
+3) Right ventricle
4) Right atrium
5) Left atrium and right ventricle.

3.2. Requirements for work results, including before registration: substantiation of the syndromic diagnosis based on complaints, medical history and life data, clinical examination.
3.3. Control materials for the final stage of the lesson: solving two clinical problems on the subject of the lesson, answering 10 tests (*if necessary*).

4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

1.
Practical lesson No. 9

Subject: Auscultation of the heart: the main heart sounds and their changes. Additional heart tones.

Goal: Acquiring knowledge and mastering professional competences in the examination of patients with pathology of the cardiovascular system, namely:
- to know the mechanisms of the formation of heart tones,
- to know the main components that form heart tones,
- to know the main reasons that lead to changes in the volume of heart tones, their splitting and bifurcation,
- to know additional heart tones and their causes
- to know the method of auscultation of the heart.

Basic concepts: Cardiovascular diseases are the most frequent cause of death of the population of Ukraine and most countries of the world.
Auscultation, as well as palpation and percussion, belong to the main methods of clinical examination of the patient. The auscultatory picture of the heart is an important link in the diagnostic search during damage to the cardiovascular system.
Mechanism of formation of heart tones. In order to understand the mechanisms of the formation of heart tones, it is necessary to clearly distinguish the phases of the cardiac cycle, which will make it possible to explain the mechanisms of the formation of tones.
During systole phases of the ventricles are distinguished:
1) asynchronous contraction, when not all areas of the myocardium are covered by contraction, and intraventricular pressure does not increase;
2) isometric contraction, when the main part of the myocardium of the ventricles contracts, the atrioventricular valves close, the intraventricular pressure increases significantly;
3) expulsion, when the intraventricular pressure reaches the level of pressure in the main vessels, the semilunar valves open.
During diastole, the ventricles relax:
1. The closing phase of the semilunar valves;
2. Phase of isometric relaxation — relaxation of the ventricles in the case of closed atrioventricular and semilunar valves until the pressure in the cavity of the ventricle becomes lower than in the atria.
3. Phase fast i slow filling ventricles openatrioventricular valves — i blood is coming from the atria to the ventricles.
4. Atrial systole and the heart cycle repeats again.

Sound phenomena that occur during the work of the heart are called heart tones. During the work of the heart, 4 tones arise: I, II, III IV. However, during auscultation of the heart, we can hear only I and II tones. III, IV tones are low, quiet, rarely heard, but clearly detected during recording of the phonocardiogram. Two tones are heard in healthy people: I — systolic (during systole), II — diastolic (during diastole).
Components of the first tone: The 1st tone (systolic) consists of 4 pairs or 8 components (knowledge of each is necessary to analyze the reasons for changing the sonority of heart tones).
1. Atrial component:
a) myocardial tension of the right atrium during contraction;
b) left atrial myocardial tension during contraction.
2. Valve component:
a) oscillations of the two-leaf valve during its closing; b) oscillations of the tricuspid valve during its closure.
3. Muscle component:
a) isometric tension and contraction of the myocardium and right ventricle;
b) isometric tension and contraction of the myocardium and left ventricle.
4. Vascular component:
a) fluctuations of the initial sections of the pulmonary artery during the period of expulsion

b) oscillations of the initial sections of the aorta during the period of blood expulsion.

Components of the second tone: II tone(diastolic) consists of 2 components.

1. Valve component:

a) fluctuations of the semilunar valves of the pulmonary artery during the ïx closing period;
b) oscillations of semilunar aortic valves during the ïx closing period.

2. Vascular component:

a) oscillations of the walls of the pulmonary artery; b) oscillations of the aorta walls.

III tonecaused by fluctuations during the rapid filling of the ventricles with blood from the atria during diastole, occurs 0.12-0.15 s after the second tone. It is not normally listened to

IV the tone at the end of diastole is due to the rapid filling of the ventricles with blood due to the contraction of the atria. It is not normally listened to.

The perception of tones depends not only on the proximity of the projection of the valves, but also on the conduction of oscillations along the blood stream. The places of projection of the valves on the front chest wall are very close, and if you listen to the points of projection of the valves, it is very difficult to decide which valve is involved in the pathological process. As a result of clinical practice, the points of the best auscultation of the valves, which do not coincide with the projection points, were established. An exception is the valve of the pulmonary artery, in which the auscultation point and the projection coincide.

The sequence of auscultation points and the projection of the heart valves onto the chest are shown in the table.

			y trunk		rba point
h	nt of the the n the left	lle of the t the level rib	the left 0.5 rd from the	lle of the f attachment to m III left and V tilages	the work of the
on	e heart	the right	the left	of the e e	ttachment h the left

Factors determining the sonority of the first tone:state of structures of atrioventricular valves, ïx position during systole; contractile function of the ventricles; degree of filling of the ventricles with blood; the rate of contraction of the ventricles.

Factors determining the sonority of the second tone:conditions of the structures of

semilunar valves of the aorta and pulmonary trunk; the level of pressure in the large and small circles of blood circulation; elastic properties of the aorta and pulmonary trunk.
The first stage of listening should always be analytical, with the division of auscultatory symptoms into fragments. First, it is necessary to focus attention on the tones of the heart (rhythmicity, number, volume), then on the pauses between the tones. On the basis of the received data, a comprehensive assessment of the melody of the heart is provided.

Auscultation of the heart is performed according to the plan:
— first determine the rhythm of cardiac activity;
— assess heart rate;
— characterize the tones of the heart (their sonority, timbre);
— evaluate the presence of bifurcations and additional tones;
— evaluate the presence of heart murmurs.

Rhythm of heart activity
First of all, during auscultation, it is necessary to determine the rhythm of cardiac activity. The further tactics of the doctor depend on this, in particular the calculation of heart rate. It is necessary to determine whether the activity of the heart is correct (rhythmic) or incorrect (arrhythmic). For this, it is necessary to estimate the volume ratio of I and II heart sounds at all points of auscultation. Normally, constant time intervals between I and II, II and I tones are heard, constancy in the volume ratio of the tones at different points of listening, that is, the activity of the heart is correct or rhythmic. In case of violation of this regularity, the activity of the heart is arrhythmic.
Heart rate(heart rate) is calculated during auscultation at the top of 15 s with subsequent multiplication by four for the correct rhythm of cardiac activity. At the same time, it should be emphasized that only the first tone of the heart should be counted. In the presence of an irregular heart rhythm, the heart rate must be counted for at least one minute. Normally, an adult's resting heart rate is 60-80 beats per minute. A heart rate greater than 90 beats per minute is defined as tachycardia, less than 60 beats per minute is defined as bradycardia.
Analysis of heart soundsconsists in determining sonority and timbre. The sonority of tones depends on a number of factors. At the top of the heart (1st point of auscultation) and at the base of the sternum (4th point of auscultation), the first tone is louder than the second heart tone. Since the accent falls on the I tone, the rhythm of the tones is heard as a chorus, which in the case of phonation of syllables can be reproduced as ta-ta, ta-ta, ta-ta.
Bigger the loudness of the I tone at the first point of auscultation is explained by the fact that the sound phenomena that form the I tone are best conducted precisely at the apex of the heart, while the II tone arises far from the apex and is worse conducted to this area. Therefore, only the I tone is evaluated and analyzed at the apex at the base of the sternum. Based on the heart: in the 2nd and 3rd points of auscultation, the second heart sound is louder than the first sound. At these points of auscultation, the accent falls on the II tone, so the melody of cardiac activity resembles an iambic, which in the case of phonation of syllables can be reproduced as i-th, i-th, i-th. Since the sound phenomena forming the II tone arise on the basis of the heart, then I analyze the II tone precisely on the basis of the heart. During the evaluation of the II heart tone, attention should be paid not only to the fact that its volume should be greater than the volume of the I tone, but also to the comparison of the volume at the second point of auscultation (above the aorta) and at the third point of auscultation (above

the pulmonary artery). The loudness of the II tone over the aorta and pulmonary artery is normally the same.
A change in heart tones can be manifested by: strengthening/weakening of the sonority of one or both tones, the appearance of bifurcation or splitting of tones, the appearance of additional tones (triple rhythms).
Strengtheningof both tones is associated with such conditions as a thin chest wall, significant physical exertion, a high position of the diaphragm, a large lung cavity next to the heart, a large gas bladder of the stomach, thyrotoxicosis, psychoemotional excitement.
Weakeningof both tones is associated with such conditions as excessive development of subcutaneous fat and muscle mass, chest wall edema, subcutaneous emphysema, pulmonary emphysema, left-sided exudative pleurisy/hemothorax/pneumothorax, cardiosclerosis, myocarditis, anemia, collapse, exudative pericarditis, dystrophic myocardial changes.
Amplification of the I tone
Amplification of the I tone at the apex. In the case of insufficient blood filling, the left ventricle (LV) contracts more actively and strongly (to ensure geodynamic ejection), with a half-empty LV, the prerequisites are also created for better listening to the closing of the mitral valve valve (strengthening of the valve component). This is observed in the case of mitral stenosis, tachycardia (physiological and pathological), extrasystole, atrial fibrillation, complete AV blockade (coincidence of contraction of the atria and ventricles — cannon Strazhesco).
Strengthening I tone on the basis of the xiphoid process - in similar conditions during stenosis of the tricuspid valve.
Weakening of the I tone
Weakening tone at the apex is due to the fact that during ventricular systole the leaflets of the mitral valve do not close and the tone weakens due to the loss of the valve component — mitral insufficiency.
Weakening of the tone at the apex may be caused by the loss of the muscle component, which occurs in the case of myocarditis, heart attack, cardiosclerosis, metabolic cardiodystrophies, cardiomyopathies.
Weakening tons on the basis of the xiphoid process occurs, respectively, in case of insufficiency of the tricuspid valve.

Amplification of the II tone
Due to the compaction of the wall of the main vessel (aorta and pulmonary trunk), the prerequisites are created for better conduction of sound (it is best conducted in the compacted area), and the increase in pressure in the small and large circles of blood circulation contributes to the strengthening of the vascular component of the II tone.
Accent II tone over the aortaassociated with such diseases as primary arterial hypertension, secondary hypertension, atherosclerosis of vessels, ischemic heart disease, syphilitic mesoaortitis.
Accent II tone over the pulmonary trunkobserved in the case of mitral stenosis, non-union of the Botal duct, sclerosis of the pulmonary artery, emphysema of the lungs, pneumosclerosis, chronic obstructive pulmonary disease, bronchial asthma, bronchiectasis.
Weakening of the II tone
It is associated with a violation of the period of closed semilunar valves. The valve

component weakens. It occurs, respectively, in case of insufficiency of the valves of the aorta and the pulmonary trunk. Also, the weakening of the second tone is possible due to the weakening of the vascular component of the II tone during hypotension in the large or small circles of blood circulation (respectively, stenosis of the mouth of the aorta and the pulmonary trunk).

Weakening of the II tone over the aorta. Aortic valve insufficiency (valvular component falls out), stenosis of the aortic orifice — a decrease in pressure in the aorta (hypotension in the aorta).

Weakening of the II tone over the pulmonary trunk. Insufficiency of the valve of the pulmonary trunk (falls out of the valve component), stenosis of the pulmonary trunk (reduced pressure in the pulmonary trunk).

Bifurcation and splitting of heart tones.

During auscultation, it is possible to detect a change in the number of heart tones due to splitting or bifurcation of the I or II tone. In some states, additional tones appear. Splitting and bifurcation of tones appear as a result of asynchronous occurrence of components involved in the formation of heart tones. Non-simultaneous closure of the atrioventricular valves will lead to splitting and bifurcation of the first tone, non-simultaneous closing of the semilunar valves - to splitting and bifurcation of the second heart tone.

Under physiological conditions, the first component of the first tone corresponds to the closure of the left atrioventricular (mitral) valve, the second component to the closure of the right atrioventricular (tricuspid) valve, which is determined by the physiological delay of the systolic right ventricle. The interval between these components is 0.01-0.03 s, recorded using phonocardiography. An increase in the interval between the components of the first tone is perceived auscultatively as splitting or bifurcation.

Under physiological conditions, the first component of the II tone corresponds to the closing of the semilunar valves of the aorta, the second component — the closing of the semilunar valves of the pulmonary artery, which is explained by the physiological delay in the end of the systole of the right ventricle. The interval between the aortic and pulmonary components is 0.02-0.04 s and is recorded using phonocardiography. An increase in the interval between the components of the II tone is perceived auscultatively as splitting or bifurcation.

Split tone— two short sounds, occurring alternately and appearing instead of a tone. The bifurcation of the I tone can be compared to an anapest (ta-ta-tam), and the bifurcation of the II tone resembles a dactyl (tam-ta-ta).

Split tone— both parts of the split tone are separated from each other by a small time interval, so they are not heard as independent sounds,however create an auscultatory the melody of heterogeneity a ton That is, under cleavage time a ton the impression is created of two sounds coming out one after the other without a noticeable pause tra-ta or ta-tra.

Splitting and bifurcation of heart tones can be due to physiological and pathological reasons.

Physiological splitting and bifurcationheart tones is related to breathing phases, body position, muscle tension, is characterized by lability (applies mainly to the II tone). Physiological splitting and bifurcation of the I tone is determined in a standing position during deep exhalation. Physiological splitting and bifurcation of the II tone is determined during muscle tension as a result of increased pressure in the aorta and the arrival of a large

mass of blood. The systole of the left ventricle is lengthened, the diastolic is delayed, the pulmonary component of the II sound is heard earlier.

Pathological splitting of the first tone associated with asynchronous closing of the mitral and tricuspid valves and is observed during the blockade of the legs of the bundle of His (splitting by the valvular component) and stenoses of the AV holes (of the mitral and tricuspid valves) (unequal blood pressure in the ventricles and, as a result, splitting by the valvular component).

Pathological splitting of the II tone associated with asynchronous closing of the semilunar valves of the aorta and pulmonary trunk and is observed in the case of mitral stenosis (different pressure in the large and small circles of blood circulation leads to the fact that the systole of the right ventricle ends later than the systole of the left ventricle, due to which split tone).

Additional heart tones.

Heart tones due to the appearance of additional tones are always pathological, normally they cannot be heard in a healthy person. These include: systolic click, quail rhythm, gallop rhythms, pericardial tone, embryocardia.

Quail rhythm— three-part rhythm, heard at the top of the heart in case of mitral stenosis. This rhythm consists of an increased clapping of the first tone, a normal second tone, and an additional pathological sound that occurs in protodiastole 0.07-0.13 s after the second tone. An additional mitral tone is called OS (opening snap — a click from the opening of the mitral valve).

Gallop rhythms. Tripartite rhythms, which are heard at the apex of the heart and consist of three separate sounds occurring at approximately equal intervals of time and reminiscent of the tramp of a galloping horse, are best heard by direct auscultation. They occur during severe heart lesions (dystrophic, inflammatory, necrotic). The systolic, presystolic, and protodiastolic gallop rhythms are distinguished by the time of appearance of the additional tone. A gallop systolic rhythm is observed in case of blockade of the legs of the bundle of His (I tone is split, II tone is unchanged).

The protodiastolic gallop rhythm is observed during myocardial infarction, dilated cardiomyopathy, severe myocarditis (the I tone is weakened due to the loss of the muscle component; the II tone is unchanged; the III tone is pathologically increased, (it is formed in the phase of rapid blood filling of the ventricles, the muscles of which are straightened faster than normal due to loss of ii elasticity.) Presystolic gallop rhythm

— pathological strengthening of the IV tone (the IV tone at the end of diastole is caused by the contraction of the hypertrophied left atrium during the simultaneous loss of ventricular muscle tone due to inflammatory and degenerative processes), the I tone is unchanged, the II tone is unchanged.

Systolic click— an auscultatory phenomenon, heard in case of prolapse of the mitral valve at the top of the heart, in the III-IV intercostal space on the left near the edge of the sternum. It is caused by the dysfunction of the papillary muscle, which leads to sagging of one or both leaflets of the mitral valve into the cavity of the left atrium during the systole of the left ventricle. An isolated mesosystolic click mesosystolic click or multiple systolic clicks may occur. After the click, there is a late systolic murmur of a rising character. A characteristic feature is the variability of auscultatory data depending on the position of the body and loading tests. When the patient rises sharply, clicking and noise are heard more distinctly, in

the lying position they may disappear.
Pericardial toneis observed in the case of constrictive pericarditis, after the second sound, occurs as a result of the vibration of the fused pericardium during the sudden expansion of the ventricle at the beginning of diastole, is registered after 0.01-0.06 s. The place of best listening is in the region of the top of the heart or more medially in the direction of the xiphoid process. It is often heard over the entire area of the heart and main vessels.
Embryocardia— a pendulum-like rhythm reminiscent of fetal heart tones. These rhythms are usually found in the case of severe myocardial damage (cardiosclerosis, myocarditis, cardiomyopathies).

Plan:
1. **Organizational measures** (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. **Control of the reference level of knowledge** (checking of workbooks, auscultation of the heart of patients with circulatory system pathology and evaluation of the obtained data), test control, solving a clinical problem, written solution of Step-2 type problems (8 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for theoretical readiness of applicants and performance of practical classes:
- the applicant must know the mechanisms of the formation of heart tones,
- the main components in the formation of the first and second tones of the heart,
- mechanisms of strengthening and weakening of heart tones,
- mechanisms of bifurcation and splitting of heart tones,
- to know pathological tones and the mechanism of their formation.

List of didactic units:
- perform auscultation of the heart guided by the methodology and technique,
- perform auscultation of a patient with a pathology of the cardiovascular system, describe the first tone at the apex.
- perform auscultation of a patient with a pathology of the cardiovascular system, describe the II tone,
- identify and evaluate changes in the volume of both heart sounds in normal and pathological conditions,
- to interpret the diagnostic value of the influence of physiological conditions (breathing, physical exertion) on changes in the volume of heart sounds.

2.2. **Questions to check basic knowledge on the topic of the lesson:**

1. Describe the mechanism of origin of heart tones (I, II, III, IV).
2. Projections of the heart valves on the front chest wall, places of auscultation, sequence of auscultation.
3. Name the characteristic features for I and II tones.
4. Name the reasons for the change in volume (amplification, attenuation) simultaneously for both tones

5. Changing the volume of tones (increasing, weakening) in isolation of one of them, the mechanism of changes, acoustic properties.
6. Changes in timbre of tones, mechanism of changes, acoustic properties.
7. The diagnostic value of the influence of physiological conditions on the loudness of tones.
8. Diagnostic value of changes in volume and timbre of heart sounds in pathology.
9. The sequence of activity of the valvular apparatus of the heart.
10. What is the physiological and pathological bifurcation and splitting of the I and II tones of the heart.
11. Reasons for bifurcation and splitting of both tones.
12. Diagnostic value of bifurcation and splitting of tones.
13. Classification, mechanism of occurrence of additional heart sounds in diastole and systole.
14. Diagnostic value of additional heart sounds.

A problem of the STEP-2 type.
1. In patient L., 52 years old, the border of the heart is shifted to the left by 1.5 cm outside the left midclavicular line, blood pressure is 170/110 mm Hg. Two tones are heard on the aorta, and the louder one comes at the end of a short pause, here it is much louder than the corresponding tone at the end of a short pause on the pulmonary artery, and a quieter tone comes at the end of a long pause.
Which of them is I and P tone on the aorta? Evaluate changes in tone volume.

A. The louder second tone is the accent of the second tone.
A. A louder II tone is the norm.
S. A louder I tone is the norm.
D. A quieter second tone is a pathology (weakening).

2. Conscript D., 18 years old, complains of stabbing pains in the region of the heart, palpitations, weakness, shortness of breath when excited (periodically desires to take deep breaths 2-3 times). The physique is asthenic. Excited, the thyroid gland is not enlarged. Pulse 100 in 1 min., rhythmic, blood pressure 110/80 mm Hg. Art. The heart is not enlarged. At the top of the I tone is louder than the II tone, systolic noise at the beginning of systole, in protodiastole - an additional low-pitched tone, is not carried anywhere, is unstable, disappears in a horizontal position; on the pulmonary artery, the accent of the II tone. ECG is normal. Lungs and organs of the abdominal cavity without features.
Assess for extra tone in protodiastole.

A. III tone, physiological.
B. III tone, pathological.
P. IV tone, physiological.
D. IV tone, pathological.

Standard answer to problem 1. -A. The louder II tone over the aorta after a short pause is characterized as an accent of the second tone over the aorta.

Standard answer to problem 2.- -A. The presence of an additional (III tone) irregular tone in protodiastole in young people is considered a physiological norm.

3. Formation of professional skills and abilities:
- formation of the ability to perform a clinical examination of a patient with a pathology of the cardiovascular system (the applicant must be able to perform auscultation of the heart, detect the first and second sounds, give them a characteristic),

- formation of the ability to give a clinical assessment of the results obtained during auscultation of patients with pathology of the heart and blood vessels, to give a clinical interpretation of the detected symptoms and syndromes in diseases of the cardiovascular system.

3.1. Control materials for the final stage of the lesson:
Situational tasks:
1. Patient F., 47 years old, normosthenic, was admitted to the clinic with complaints of shortness of breath at rest 32 in 1 minute, pain in the left half of the chest, weakness. When examining the patient, there is a dull sound over the left lung percussing up to the 2nd intercostal space, auscultation over the dullness of breathing is almost impossible to hear, the borders of the heart and the heartbeat cannot be determined, auscultation - sharply weakened tones. What causes weakening of heart tones.

+A. Exudative pericarditis.
B. Left-sided exudative pleurisy.
S. Obesity.
D. Emphysema of the lungs.

2. Conscript D., 17 years old, complains of stabbing pains in the region of the heart, palpitations, weakness, shortness of breath when excited (periodically desires to take deep breaths 2-3 times). The physique is asthenic. Excited, the thyroid gland is not enlarged. Pulse 100 in 1 min., rhythmic, blood pressure 110/80 mm Hg. Art. The heart is not enlarged. At the top of the I tone is louder than the II tone, systolic noise at the beginning of systole, in protodiastole - an additional low-pitched tone, is not carried anywhere, is unstable, disappears in a horizontal position; on the pulmonary artery, the accent of the II tone. Assess for extra tone in protodiastole.

+A. III tone, physiological.
B. III tone, pathological.
P. IV tone, physiological.
D. IV tone, pathological

3. The volume of both tones increases in case of:
A. mitral stenosis;
B. mitral insufficiency;
C. tricuspid stenosis;

D. + thyrotoxicosis.

4. The first tone at the top of the heart increases in case of:
A. + mitral stenosis;
B. mitral insufficiency;
C. tricuspid stenosis;
D. hypothyroidism;

5. II tone on the pulmonary artery increases in case of:
A. tricuspid insufficiency;
B. pulmonary artery stenosis;
C. +pulmonary hypertension;
D. arterial hypertension;

6. Patient D. is 48 years old, a three-part rhythm is heard at the top of the heart, the loudest tone coincides with the heart beat, and an additional tone appears before it, what is this rhythm:

A. + quail rhythm
IN. presystolic gallop rhythm
S. protodiastolic gallop rhythm
D. embryonic rhythm

7. In what pathology is there an increase in the first sound at the top of the heart:
A. Mitral insufficiency
B. Aortic stenosis
S. Hypertensive disease
D.+ Mitral stenosis

8. Name the main component of the first tone:
A. Tension and oscillations of aortic valves
B. Contraction of the atrium
C. Pulmonary valve tension
D. + Tension and oscillations of the mitral valve

3.2. Requirements for work results, including before registration: substantiation of the syndromic diagnosis based on complaints, medical history and life data, clinical examination.
3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 8 tests (if necessary).

4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 10

Subject: Auscultation of the heart: organic and functional heart sounds.

Goal: Acquiring knowledge and mastering professional competences in the examination of patients with pathology of the cardiovascular system, namely:
- to know the mechanism of formation of heart murmurs,
- to know the classification and characteristics of heart murmurs,
- to know the differences between functional and organic noises,
- to know the main extracardiac murmurs, their origin and their difference from intracardiac murmurs,
- to be able to detect the presence of heart murmurs in patients with cardiovascular pathology,
- be able to differentiate noises depending on the phases of cardiac activity (systolic, diastolic),
- to be able to differentiate noises depending on their origin (organic, functional).

Basic concepts: Despite the active introduction into clinical practice of modern instrumental methods of examination, auscultation remains one of the main physical methods of heart research, because when using a minimum of equipment, it gives an idea of the state of the heart muscle and the processes that take place in its cavities. Normally, blood flows through the valve openings of the heart (to the heart and from the heart to the aorta and pulmonary trunk) silently. In the presence of deformations of valve structures, blood flows become turbulent, change speed, creating sound phenomena called noises. Auscultation of the heart is the first and most accessible method of detecting these changes. The correct recognition of changes in the heart depends on the professional skill of the doctor. To understand auscultatory phenomena, it is necessary to have a good command of the technique of listening to the heart, to be able to clearly interpret the heard sound phenomena. Therefore, auscultatory detection and assessment of the sound of noises are of great importance for timely diagnosis and the appointment of adequate treatment, which in some cases will contribute to the preservation of patients' lives.

During auscultation of the heart, not only tones are determined, but there are often noises as well. Heart murmurs are sound phenomena that occur in the heart, in addition to tones.

It should be noted that heart tones are not pure sounds, but are short low-frequency sounds. Therefore, in a physical sense, tones and noises are close to each other. And yet these sound phenomena have differences: tones are perceived as short sounds, and noises as longer.

Murmurs are divided into intracardiac and extracardiac. Intracardial, in turn, are divided into functional (disruption of the function of unchanged valves) and organic (existing anatomical changes in the structure of the valve).

Functional murmurs arise in an intact heart due to acceleration of blood flow, decrease in its density, rapid growth during childhood and adolescence, anemia.

Organic murmurs occur in the presence of anatomical changes in the heart (change in the structure of valves or openings in the form of stenosis or insufficiency, or their combination) or in the vessels departing from the heart (in the aorta or pulmonary artery).

Systolic and diastolic noises are distinguished by the time of noise appearance during systole or diastole. Systolic murmur occurs when during systole blood, moving from one part of the heart to another or from the heart to large vessels, encounters an obstacle on its way. A systolic murmur is heard in stenosis of the mouth of the aorta or pulmonary trunk, because in these defects, during the expulsion of blood from the ventricles, a narrowing of the vessel occurs in the path of blood flow (systolic expulsion murmur).
A systolic murmur can also be heard with mitral and tricuspid valve insufficiency. Its occurrence is explained by the fact that during ventricular systole, blood flows not only into the aorta and pulmonary trunk, but also back into the atrium through a not tightly closed atrioventricular opening, i.e. through a narrow gap (systolic regurgitation noise - return against the physiological flow (direction)).
Diastolic noise appears in those cases when the left or right atrioventricular opening is narrowed, because with these defects during diastole there is a narrowing of the path of blood flow from the atria to the ventricles. A diastolic murmur also occurs with insufficiency of the valves of the aorta and pulmonary trunk - due to the reverse flow (regurgitation noise) of blood from the vessel into the ventricles through the gap that is formed when the leaflets of the changed valve are not fully closed.
Depending on the causes, organic murmurs are divided into acquired (systolic and diastolic) and congenital. Functional - on systolic and diastolic (Flint's murmur, Graham-Steele's murmur, Coombs' murmur).
Extracardiac (extracardial) murmurs – pericardial, cardiopulmonary, and pleuropericardial.
During auscultation, determine:
- ratio of noise to systole or diastole
- nature of noise, power, timbre, sonority
- localization of noise, the place of its best listening (noise epicenter)
- the direction of the noise is radiation, the place of its propagation

The relation of the noise to the phases of cardiac activity is distinguished by the same signs as the differences of 1 and 2 tones.
Systolic murmur appears together with 1 tone, or immediately after it, during a short pause of the heart, it coincides with the apical push and pulse on the carotid artery.
A diastolic murmur occurs after 2 tones during a long cardiac pause. There are 3 types of diastolic noise:
- protodiastolic - occurs at the beginning of diastole, immediately after the 2nd tone;
- mesodiastolic, occurring in the middle of diastole;
- presystolic, appearing at the end of diastole, closer to 1 tone.

Classification of heart murmurs:
- Extracardial: pericardial friction noise, pleuro-pericardial noise.
- Endocardial: organic, functional.
- Organic: valvular, muscular.
- Valves: systolic, diastolic

Noise properties. By timbre, noises are divided into blowing, scraping, sawing, rough, and soft. The noise can be compared to the "whistle of a young rooster" (V.F. Zelenin), "train", rubbing sandpaper on wood, etc. But the assessment of noise timbre has a certain diagnostic value.

For example, a diastolic murmur in mitral stenosis often differs in timbre from a diastolic murmur in aortic valve insufficiency. The first has a lower tone, often resembles a rumble, and the second is more often gentle, blowing.
Noises with an increasing character - crescendo, and fading ones - decrescendo are distinguished by their form.
With a graphic image (phonocardiogram), they can be rhomboid, ribbon, spindle-shaped. The best places for listening to noises - the epicenter - are the points of auscultation of the valves in which they were formed. The noises that are generated on the bicuspid valve are louder in the area of the apical thrust. Noises from the mouth of the pulmonary artery are louder in the 2nd intercostal space on the left. Noises from the mouth of the aorta are better heard in the 2nd intercostal space on the right.
Irradiation (conduction) of noise depends on the direction of blood flow. The noise is produced along the blood flow, due to which it can be heard not only at the point of auscultation of this valve, but also at a certain distance from it. Noises caused by pathological changes in the bicuspid valve are carried to the axillary region, to the middle and even the posterior axillary line on the left, sometimes under the scapula and occasionally to the spine. The systolic murmur in case of mitral valve insufficiency can be carried both upwards and to the point of Botkin-Erb (point 5). Noises caused by the pathology of the tricuspid valve can lead to the right subclavian area, sometimes to the jugular fossa, often to the vessels of the neck. A similar noise with stenosis of the pulmonary artery is made in the left subclavian cavity. Diastolic murmur in the case of aortic valve insufficiency is produced by blood flow to the Botkin-Erb point. As the noise moves away from the place of its origin, the volume of the noise gradually decreases. For example, with the insufficiency of the bicuspid valve, the noise weakens when approaching the posterior axillary line. But in the subscapular area, it can increase again. The systolic murmur in stenosis of the mouth of the aorta weakens towards the lower edge of the sternum, but in the supra-abdominal area above the abdominal aorta it sometimes becomes distinct again.

Clinical techniques for diagnosing heart murmurs

Organic noises are better heard at the end of exhalation and weaker - during inhalation, when the lungs are filled with air and cover the heart. Functional noises are heard at the end of inspiration. During inhalation, the volume of blood in the left parts of the heart decreases, and in the right - increases due to the suction action of the chest. Therefore, all sound phenomena are amplified over the valves of the right half of the heart, and they weaken over the left half.
All noises are best heard when the patient is lying on his back. Systolic murmurs often increase when the patient is lying down.
Diastolic murmurs are affected relatively little by the position of the patient. The murmur of aortic valve insufficiency is sometimes better listened to in a standing position, and mitral murmurs – lying on the left side. The systolic murmur in aortic stenosis, aortitis, aortic sclerosis becomes louder when examining the patient using the Kupovarov-Syrotynin method (the patient moves his head slightly back while standing, hands are thrown behind the neck), or when the Udintsev method is used, the trunk is tilted forward. Then the accent and systolic noise intensifies. The inclination of the trunk also sometimes helps to better listen to the diastolic murmur in aortic valve insufficiency.
The most popular classification of noise by loudness is the classification proposed by

Freeman and Levine (Freeman and Levine):

- noise of the 1st degree is the weakest noise that can be heard only with maximum concentration of hearing, with great effort and not immediately.
- noise of the 2nd degree - loud enough for the ear to catch it immediately.
- 3rd degree noise - easy to hear.
- grade 4 noise – the noise is relatively loud and is accompanied by a palpable tremor.
- A grade 5 murmur is a noise so loud that it can be heard even when only the edge of the phonendoscope membrane is applied to the chest wall.
- a grade 6 murmur is heard even when the stethoscope does not touch the chest wall - it is heard even when the membrane is over the chest wall.

Despite some subjectivity, this classification has an important practical meaning: noises of degrees 4-6 almost always have an organic origin, and noises of 1-2 degrees are almost always functional.

Functional noises

Functional murmurs are murmurs that are produced in the heart when the valvular apparatus is intact.

They occur in the following cases:

- with relative insufficiency of the valves due to stretching of one of the ventricles;
- with insufficiency of valves due to weakening of the tone of the papillary muscles;
- with significant acceleration of blood flow in nervous, excited persons;
- fever;
- thyrotoxicosis;
- anemia;
- decrease in blood density.

Characteristic features of functional noise are its variability, softness of timbre, impermanence. Unlike organic murmurs, functional murmurs do not go anywhere, they are mainly heard at the apex of the heart and pulmonary artery.

Functional murmurs in most cases are systolic. But in clinical practice, there are three types of diastolic murmurs: Flint's, Graham-Steel's, and Coombs' murmur.

Graham-Steele's murmur occurs with severe mitral stenosis, with some chronic lung lesions accompanied by hypertension of the small circle, with some congenital heart defects. The genesis of this noise is explained by the relative insufficiency of the valve of the pulmonary artery with stretching of its valve ring. It is best to listen to the Graham-Steele murmur in 2-3 intercostal spaces to the left of the sternum.

Flint's murmur occurs with pronounced aortic insufficiency and significant expansion of the left ventricle. A presystolic murmur is heard over the apex of the heart due to the vibration of the mitral valve caused by the reverse flow of blood (from the aorta to the left ventricle) during diastole.

A Coombs murmur is an early diastolic functional murmur that is best heard in the area of absolute cardiac dullness, near the apex. The noise is short, soft, appears immediately after the 2nd tone and, as a rule, is heard only in the presence of the 3rd tone, which indicates an increase in the filling of the left ventricle. This noise occurs when the left ventricle is significantly enlarged and the mitral orifice is unchanged in size. A relative insufficiency of the mitral valve is formed, which leads to the return of part of the blood from the ventricle to the left atrium. Thus, the left atrium is filled with blood, for which the unchanged mitral

orifice becomes relatively narrow. The second factor that leads to the appearance of this noise is an increase in the speed of blood flow from the atrium to the left ventricle, the tone of which is reduced, so it does not offer any resistance to the blood entering it.
Mitral valve prolapse is the bending of one or two leaflets of the mitral valve into the atrial cavity, it occurs more often in people with an asthenic body type and a flat chest.
It occurs with coronary artery disease, rheumatism, cardiomyopathies, myocarditis, can develop with damage to the papillary muscles with their dysfunction, with myxomatous changes in the valve leaflets or degenerative changes in the chordal collagen.
The most characteristic auscultatory signs of the syndrome are an additional tone in the middle of systole and a systolic noise that occurs after it, rising and falling to the aortic component of 2 tones. These auscultatory manifestations are best determined near the left edge of the lower third of the sternum. Prolapse of the mitral valve is diagnosed with an echocardiogram.

Pericardial friction noise belongs to extracardial. It occurs in the case of friction of the visceral and parietal leaves of the pericardium in case of their inflammation.

- Pericardial friction noise is heard both in systole and diastole.
- Pericardial friction noise does not have a typical localization, but most often it is found in the area of absolute heart dullness.
- The pericardial friction noise intensifies when the body is bent forward, or if the stethoscope is pressed harder against the chest wall.
- Perceived as sound that occurs closer to the ear.
- Does not radiate.
- It can be quiet or loud, reminiscent of rustling, grinding, crunching.
- The frequency of pericardial friction noise coincides with the pulse rate.
- When breathing is delayed, the pericardial friction noise continues to be heard.

Pleuro-pericardial murmur occurs in case of inflammation of the area of the pleura directly adjacent to the heart, in particular, the pleura lining the left costo-diaphragmatic sinus.

- A pleuro-pericardial murmur is heard in the area of the left border of relative dullness of the heart.
- Intensifies during deep inhalation.
- During exhalation or breath hold, the pleuro-pericardial friction noise sharply weakens or even disappears.
- The frequency of pleural friction noise coincides with the frequency of breathing.

Algorithm for differential diagnosis of heart murmurs
For the purpose of differential diagnosis of heart murmurs, first of all, it is necessary to establish - is the murmur intracardiac or extracardiac?
In what phase of the cardiac cycle does the murmur occur? If the noise occurs in diastole, then such noises are always organic. If the epicenter of the murmur is above the apex, it is a murmur with mitral stenosis. And if the epicenter of the noise is in the II intercostal space to the right of the sternum - in aortic insufficiency.
The main signs of a murmur in the heart with mitral stenosis:

- diastolic;

- epicenter: above the top of the heart;
- holding: not held;
- volume: low-frequency noise (rumble) up to 4 degrees of volume, with presystolic amplification;
- changes: intensifies in the position on the left side, after physical exertion;
- associated signs: increased (caressing) 1st tone over the apex, mitral valve opening tone ("quail rhythm"), emphasis of the 2nd tone over the pulmonary artery, diastolic "cat's purr".

The main signs of a heart murmur in aortic insufficiency:
- diastolic murmur;
- epicenter: II intercostal space to the right of the sternum;
- holding: not held;
- volume: soft;
- changes: it increases in a standing or sitting position with the body bent forward, in a supine position;
- associated signs: weakened I tone over the apex and II tone over the aorta, diastolic murmur over the apex (Austin Flint's murmur).

If the murmur is systolic, it is necessary to determine whether it is organic or functional?

The main signs of a functional heart murmur:
- as a rule - systolic murmur;
- epicenter: II intercostal space near the left edge of the sternum, at the top;
- holding: not held;
- duration: short;
- changes: increases during physical or psycho-emotional stress, decreases or disappears during the Valsalva test.
- associated signs: it is not accompanied by changes in I and II tones, there are no additional tones, the limits of cardiac dullness have not changed.

If you have determined that it is an organic systolic murmur, then if the epicenter is at the apex, then this murmur is due to mitral valve insufficiency, if it is in the II intercostal space to the right of the sternum, it is due to aortic stenosis.

The main signs of heart murmur in mitral valve insufficiency:
- systolic murmur;
- epicenter: apex of the heart;
- carrying out: it is carried out in the left axillary fossa;
- volume: descending;
- changes: the noise intensifies when the patient is lying on the left side, when lying down, and after physical activity. load.
- associated signs: weakening of the 1st tone at the apex, emphasis of the 2nd tone over the pulmonary artery.

The main signs of heart murmur in aortic stenosis:

- systolic murmur;
- epicenter: II intercostal space near the right edge of the sternum;
- loudness: loud, ascending-descending (diamond-shaped);
- timbre: rough, scraping;
- conduction: vessels of the neck (carotid arteries);
- changes: intensifies in a standing position, lying on the back;
- associated signs: systolic "cat's purr", weakening of the second tone on the aorta, and the first tone on the apex.

Plan:

1. Organizational measures (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge (checking of workbooks, auscultation of the heart of patients with circulatory system pathology and evaluation of the obtained data), conducting a test control, solving a clinical problem, written solving of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for theoretical readiness of applicants and performance of practical classes:

- the applicant must know the mechanisms of heart murmur formation,
- the applicant must know the classification and characteristics of heart murmurs,
- the main differences between organic and functional noises,
- causes of the appearance of functional and extracardiac murmurs,

List of didactic units:

- perform auscultation of the heart guided by the methodology and technique,
- to be able to detect noise in patients with heart pathology and characterize it according to the algorithm,
- to identify the noise epicenter and the irradiation zone,
- be able to distinguish organic noise from functional,
- be able to apply techniques that help differentiate organic noise from functional noise.

2.2. **Questions to check basic knowledge on the topic of the lesson:**

1. What are the mechanisms of formation of heart murmurs?
2. By what criteria are heart murmurs characterized?
3. What is an organic heart murmur?
4. When do functional noises occur?
5. What is the difference between organic noise and functional noise?
6. When a systolic murmur is heard at the apex of the heart when which valves are affected?
7. When a diastolic murmur is heard at the apex of the heart, which valves are damaged?
8. What are the causes of functional noises?
9. How to determine the epicenter of noise and where it can be conducted?
10. What are extracardiac murmurs?

A problem of the STEP-2 type.
1. A 30-year-old patient complains of shortness of breath during moderate physical exertion, palpitations, and leg swelling. Objectively: pulsation in the III-V intercostal space on the left, in the epigastric area; diastolic tremor above the apex. Auscultation: strengthening of the I tone at the apex, the II tone is split, presystolic rising noise. What pathology can you think of?
A. Aortic insufficiency
B. Mitral insufficiency
C. Aortic stenosis of the pulmonary artery
D. Stenosis of the aortic valve
E. Mitral stenosis

2. A 44-year-old patient complains of weakness, pain in the area of the heart during exercise, dizziness. Objectively: pallor of the integuments, the I tone at the top is weakened, a rough systolic murmur in the II intercostal space on the right, which is performed on the carotid artery. II tone on the aorta is weakened. Blood pressure 100/70 mm Hg. What pathology can you think of?
A. Insufficiency of aortic valves
B. Insufficiency of the mitral valve
S. Stenosis of the pulmonary artery
D. Aortic stenosis
E. Complex mitral defect with the advantage of insufficiency

Standard answer to problem 1. -E. The presence of a diastolic tremor during palpation, as well as an increase in the I tone at the apex and an increasing presystolic murmur are characteristic of mitral stenosis.
Standard answer to problem 2. –D. Pallor of the skin, weakening of the I tone at the apex of the heart, the II tone on the aorta, and a coarse systolic murmur in the II intercostal space on the right, which is applied to the vessels of the neck, are characteristic of aortic stenosis.

3. Formation of professional skills and abilities:
- formation of the ability to perform a clinical examination of a patient with a pathology of the cardiovascular system (the applicant must be able to perform auscultation of the heart, establish the presence of heart murmurs and give their characteristics),

- formation of the ability to give a clinical assessment of the results obtained during auscultation of patients with pathology of the heart and blood vessels, to give a clinical interpretation of the detected symptoms and syndromes in diseases of the cardiovascular system.
3.1. Control materials for the final stage of the lesson:
Situational tasks:
1. A 30-year-old patient complains of shortness of breath when walking, heart pain and palpitations. Objective state of moderate severity, acrocyanosis. Auscultatively: I tone at the

apex is weakened, rough systolic noise at the apex, emphasis of the II tone on a. pulmonalis. What pathology can you think of?
A. Insufficiency of aortic valves
+V. Insufficiency of the mitral valve
C. Stenosis of aortic valves
D. Mitral stenosis
E. Insufficiency of the tricuspid valve

2. A 40-year-old patient complains of palpitations, pain in the heart area. Objectively: the skin is pale. Increased pulsation of the carotid arteries. Auscultation: I tone on the apex is weakened, II tone on the aorta is weakened, diastolic murmur, which is carried to Botkin's point. What pathology can you think of?
+A. Aortic insufficiency
B. Mitral insufficiency
S. Aortic stenosis
D. Mitral stenosis
E. Insufficiency of the tricuspid valve

3. A 32-year-old patient complains of shortness of breath during moderate physical exertion, cough with light-colored sputum with blood streaks, swelling of the lower extremities. Objective condition of moderate severity, acrocyanosis. BH 26 per minute. Auscultation: the I tone at the apex is amplified, protodiastolic and short systolic noise at the apex, emphasis and bifurcation of the II tone at a. pulmonalis. Swelling of the lower extremities. What pathology can you think of?
A. Aortic insufficiency
B. Mitral insufficiency
S. Aortic stenosis
D. Mitral stenosis
+E. Combined mitral valve disease with predominance of stenosis

4. The patient is 38 years old, complains of pain in the right hypochondrium, palpitations, swelling of the lower legs. During a general examination: swelling of the face, acrocyanosis. Pulse 82 beats per minute. Blood pressure - 130/80 mm Hg. Art. When examining the atrial region: heartbeat. On palpation: an apical thrust in the V intercostal space 1.5 cm medially from the left midclavicular line. Percussion: shift of the right border of the relative dullness of the heart to the right. An increase in the area of absolute dullness of the heart. On auscultation: weakening of the first tone and systolic murmur at the base of the xiphoid process. What disease can be thought of based on these data?
A. Mitral stenosis.
+V. Insufficiency of the tricuspid valve.
S. Aneurysm of the ascending aorta.
D. Stenosis of the mouth of the pulmonary artery.
E. Aortic stenosis.
5. A 52-year-old patient complains of shortness of breath, heart pain, palpitations, and dizziness. During the examination: pallor of the skin, marked pulsation of the carotid arteries,

rhythmic shaking of the head. Pulse 90 beats per minute, fast, high, large (p. Celer, altus et magnus). Blood pressure - 170/30 mm Hg. Art. During palpation of the atrial region: an apical impulse in the VI intercostal space 1 cm outward from the left midclavicular line, dome-shaped. On percussion: the left border of relative cardiac dullness is shifted to the left. During auscultation: weakening of the I tone at the top of the heart, weakening of the II tone over the aorta. Diastolic murmur in the II intercostal space to the right of the sternum and at the point of Botkin-Erb. What disease can be thought of based on these data?
A. Mitral stenosis.
B. Aortic stenosis.
C. Insufficiency of the tricuspid valve.
D. Stenosis of the mouth of the pulmonary artery.
+E. Aortic insufficiency.
6. A 43-year-old patient complains of shortness of breath, attacks of dyspnea, cough, hemoptysis, palpitations. On examination: acrocyanosis. The pulse is irregular, the frequency is 76 beats. Heart rate 90 bpm, pulse deficit 14 bpm. Blood pressure - 130/80 mm Hg. Art. On palpation: an apical thrust in the V intercostal space 1 cm medially from the left midclavicular line. Symptom of diastolic "cat's purr" at the top of the heart. On percussion: the right and upper border of relative cardiac dullness are shifted outward. During auscultation: increased, clapping I tone at the apex, emphasis of the II tone over the pulmonary artery, quail rhythm at the apex of the heart, diastolic murmur. What disease can be thought of based on these data?
A. Mitral stenosis.
B. Aortic stenosis.
C. Insufficiency of the tricuspid valve.
D. Stenosis of pulmonary artery valves.
E. Aortic insufficiency.

7. What noise is heard at the point of auscultation in aortic insufficiency?
A. Diastolic murmur at the top of the heart.
B. Systolic murmur at the base of the xiphoid process.
S. Systolic murmur in the II intercostal space to the right of the sternum.
+D. Diastolic noise in the II intercostal space to the right of the sternum.
E. Systolic-diastolic murmur in the II intercostal space to the right of the sternum.
8. What kind of noise is heard at the point of auscultation with mitral stenosis?
+A. Diastolic murmur at the apex of the heart.
B. Diastolic murmur at the base of the xiphoid process.
S. Systolic murmur in the II intercostal space to the right of the sternum.
D. Diastolic murmur in the II intercostal space to the right of the sternum.
E. Systolic-diastolic murmur in the II intercostal space to the right of the sternum.
9. Functional noises are characteristic of:
A. Myocardial infarction.
B. Acquired heart defects.
S. Angina.
D. Congenital heart defects.
+E. Anemia

10. What kind of noise is heard at the point of auscultation in aortic stenosis?
A. Systolic murmur at the top of the heart.
B. Systolic murmur at the base of the xiphoid process.
+S. Systolic murmur in the II intercostal space to the right of the sternum.
D. Diastolic murmur in the II intercostal space to the right of the sternum.
E. Systolic-diastolic murmur in the II intercostal space to the right of the sternum

3.2. Requirements for work results, including before registration: substantiation of the syndromic diagnosis based on complaints, medical history and life data, clinical examination.
3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (if necessary).

4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 11

Subject: Methodology of registration and analysis of ECG. ECG signs of ventricular and atrial hypertrophy.

Goal: Acquiring knowledge and mastering the professional competences of ECG registration and analysis by the learner, namely:
- know the main components of the ECG, their origin and normal characteristics,
- be able to correctly apply electrodes and perform ECG registration,
- be able to evaluate the technical qualities of the registered ECG (voltage, speed of registration, presence of artifacts),
- know the scheme (algorithm) of ECG analysis,
- be able to determine the pacemaker and calculate the frequency of contractions on the ECG,
- be able to determine the electrical axis of the heart on the ECG,
- know the main changes on the ECG in left and right atrial hypertrophy,
- be able to detect signs of atrial hypertrophy on the ECG,
- know the main changes on the ECG in hypertrophy of the left and right ventricles of the heart,
- to be able to detect signs of hypertrophy of the ventricles of the heart on an ECG.

Basic concepts: Although electrocardiography is an additional method of examination of the patient, it is included in the general mandatory ones, which are carried out regardless of the existing pathology and during preventive examinations. ECG allows to determine changes in the functions of the heart, which are not manifested by clinical signs (leg blocks, silent ischemia, atrial fibrillation, etc.), but are manifestations of cardiac pathology; to confirm target organ damage in a hypertensive patient (left ventricular hypertrophy), detect ECG signs of chronic pulmonary heart disease, differentiate coronary syndrome, etc. With the help

of an ECG, fatal arrhythmias are detected, which is a frequent cause of death of the patient, which can be prevented by applying defibrillation. Signs of complete atrio-ventricular blockade are also determined, which is the cause of syncopal conditions, and in the III degree, the death of the patient, who can be saved by timely identifying this conduction disorder and installing a pacemaker.
Electrocardiography (ECG) is a method of recording biopotentials of the heart during its operation. ECG allows to analyze the excitability, conduction, automatism, depolarization and repolarization of both the myocardium in general and its individual parts. As a rule, the ECG does not make it possible to establish a nosological diagnosis, because various pathological processes in the heart (inflammatory, dystrophic, ischemic, hypertrophic) lead to the same type of ECG changes. This is an additional method of examining the patient. Because of this, the diagnosis is established only on the basis of subjective data and meticulous clinical physical examination of the patient.
The principle of operation of the electrocardiograph consists in the graphic registration of the difference in the potentials of the myocardium over time. The myocardium consists of a large number of fibers that have their own electric field with a certain direction and strength, graphically displayed as a vector arrow. The summation of these vectors makes it possible to obtain a general integral vector of the heart, the position of which coincides with the anatomical position of the heart, that is, it is directed from right to left, from top to bottom, from back to front.
The electric field of the heart is projected onto the entire surface of the human body. The optimal location of electrodes on the body was proposed by the German physiologist Einthoven. These leads are called standard. And lead - the electrodes are placed on the left and right hands. II lead - right arm and left leg. III lead - left arm and left leg.
There are also unipolar Wilson leads, which are taken from 6 points in the chest: V1 - 4 intercostals on the right near the sternal border.
V2 – 4th intercostal space on the left near the edge of the sternum. V3 is in the middle of the line connecting V2 and V4.
V4 – 5th intercostal space 1.5 cm inwards from the mid-clavicular line. V5 – 5th intercostal space along the anterior axillary line.
V6 – 5 intercostals along the mid-axillary line.
There are also increased unipolar Holzberger leads from the left (AVL), right (AVR) arms, and left leg (AVF).
Let's name the areas of the myocardium that are predominantly reflected in different leads.
And, AVL, V1, V2 - the front wall of the left ventricle
I, AVL, V3 - septum of the heart
And, AVL, V4 - apex
I, AVL, V5, V6 – the lateral wall of the left ventricle
III, AVF - the back wall of the left ventricle.

Formation of ECG waves and intervals.
The sinus node is excited first and the wave of excitement covers the atria. An R wave appears on the ECG. Further, the excitation spreads through the atrio-ventricular node, the common trunk of the bundle of His, the legs of the bundle of His, and Purkinje fibers. This corresponds to the interval PQ. Depolarization of the ventricles begins - a QRS complex

appears on the ECG. The first wave, which is directed downwards, is the Q wave. The next one, which is directed upward, is the R wave. The next wave, after R, which is directed downwards, is the S wave. The complete coverage of the heart by excitation corresponds to the QT interval. Repolarization - T wave. Q wave reflects
excitation of the heart membrane, R – the main mass of the left ventricle, S – excitation of the posterior-basal sections of the myocardium.

ECG decoding scheme

1. *Assessment of automaticity and excitability function:*
— The rhythm is sinus or non-sinus.
— The rhythm is correct or incorrect.
— Heart rate.
2. *Estimation of contractility function:*
— The amplitude of the teeth.
— The position of the electrical axis of the heart.
— The duration of the QRST interval - the electrical systole of the heart.
3. *Evaluation of the conducting function:*
— The duration of the PQ interval, the QRS complex (duration and deformation).
4. *Assessment of the presence of ischemic changes:*
— Pathological Q wave.
— The position of the ST segment relative to the isoelectric line.
— The shape and amplitude of the T wave.

Hypertrophy of the right atrium

*Reasons:*chronic obstructive pulmonary disease, bronchial asthma, pulmonary emphysema, primary pulmonary hypertension, acquired and congenital heart defects.
ECG signs:(P-pulmonale) – the amplitude of the P wave increases in II, III, AVF, the apex of the P wave is pointed. There is no general prolongation of the duration of the P wave.
*Clinical signs:*displacement of the right border of relative heart dullness to the right, an increase in the size of the right atrium due to the thickness of the myocardium or an increase in the cavity during ultrasound diagnosis.

Left atrial hypertrophy

*Reasons:*acquired mitral and aortic heart defects, relative mitral insufficiency
ECG signs:(P-mitral) – in I, II, AVR, AVL the P wave is two-humped, and in leads III, AVF it can be biphasic (+ -). The duration of R may be increased.
*Clinical signs:*different pulse on both hands (pulsus diferens), pulsation in the II and III intercostal spaces to the left of the sternum, displacement of the upper limit of the relative cardiac dullness upwards, an increase in the size of the left atrium due to the thickness of the myocardium or an increase in the cavity during ultrasound diagnosis.

Hypertrophy of the right ventricle

*Reasons:*chronic obstructive pulmonary disease, bronchial asthma, pulmonary emphysema, primary pulmonary hypertension, acquired and congenital heart defects.
*ECG signs:*spelling (RIII>SI)
RIII>15 mm, SI >15 mm RIII + SI>25 mm,
RV1 > RV2 > RV3 > RV4, RV1 > 7mm,

SV1< 2mm; RV1 + SV5,6≥35 mm,
QRS>10 mm, deformed III, AVF, V1, V2 ST below the isoline in III, AVF, V1, V2 transition zone in V4.
*Clinical signs:*pulsation in the epigastric area under the xiphoid process, a negative heart impulse in the area of absolute cardiac dullness, or an apical impulse shifted to the left, displacement of the right and then the left borders of relative cardiac dullness, an increase in the diameter of the heart; an increase in the size of the right ventricle due to the thickness of the myocardium, or an increase in the cavity during ultrasound diagnosis.
Hypertrophy of the left ventricle
*Reasons:*acquired mitral insufficiency, relative mitral insufficiency, aortic heart defects, congenital heart defects, coarctation of the aorta
ECG signs: leftogram (RI> RIII);
RI + SIII>25 mm; RV5,6 > RV4
R V5,6 + SV1 ≥35 mm,
QRS>10 mm, deformed I, II, AVL, V5, V6; ST below the isoline in leads I, II, AVL, V5, V6; transition zone in V2.
*Clinical signs:*shifted to the left and down, spilled, high and resistant apical impulse, displacement of the left border of relative cardiac dullness; increase in the diameter of the heart; an increase in the size of the left ventricle due to the thickness of the back wall or membrane of the myocardium, or an increase in the cavity during ultrasound diagnosis.

Plan:
1. **Organizational measures** (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. **Control of the reference level of knowledge** (checking workbooks, performing ECG analysis in normal patients and in patients with signs of hypertrophy of heart chambers), conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for theoretical readiness of applicants and performance of practical classes:
- the applicant must know the mechanisms of formation of the main components of the ECG and their normal characteristics,
- the applicant must know the technique of ECG registration,
- the applicant must know the algorithm (sequence) of ECG analysis,
- the applicant must know the changes on the ECG in case of atrial hypertrophy
- the acquirer must know the changes on the ECG in the case of ventricular hypertrophy

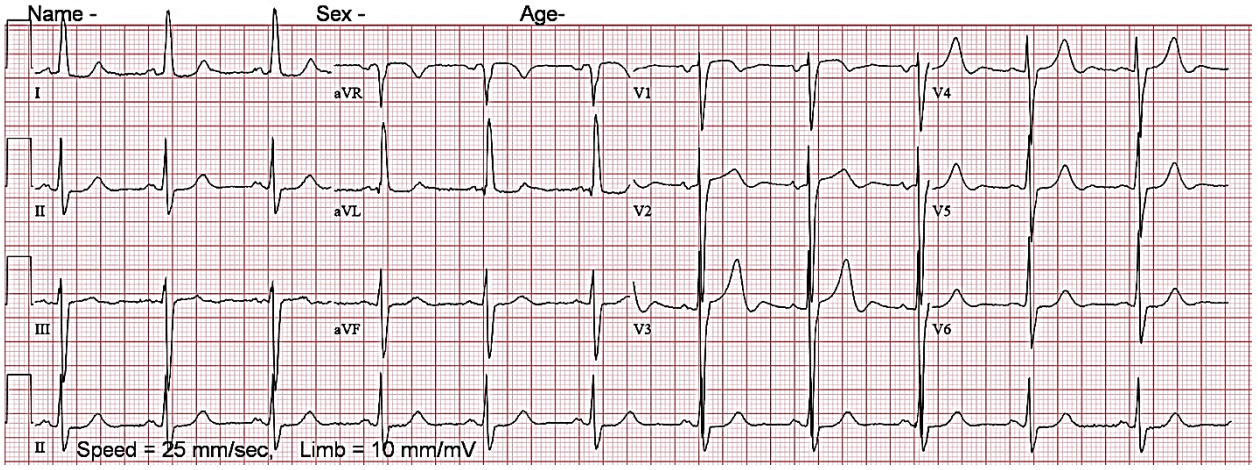
List of didactic units:
- register an ECG, following the necessary rules,
- be able to evaluate the technical characteristics of the registered ECG (voltage, speed, presence of artifacts),
- set the main pacemaker and calculate the heart rate on the ECG,
- be able to evaluate the electrical axis of the heart,
- to be able to detect signs of atrial hypertrophy on the ECG

- to be able to recognize the signs of hypertrophy of the ventricles of the heart on the ECG.

2.2. **Questions to check basic knowledge on the topic of the lesson:**

What functions of the heart can be evaluated with the help of an ECG?
What are depolarization and repolarization and how do they appear on the ECG?
1. What main waves and complexes form the normal ECG, describe them.
2. What does the P wave on the ECG show and what is its normal characteristic?
3. What does the P-Q segment show on the ECG and what is its normal characteristic?
4. What does the QRS complex on the ECG reflect and what is its normal characteristic?
5. What does the S-T segment show on the ECG and what is its normal characteristic?
6. What does the P wave on the ECG show and what is its normal characteristic?
7. What is the electrical axis of the heart, its characteristics in normal and pathological conditions.
8. How to evaluate the main rhythm driver on the ECG and count the number of heart contractions?
9. What are the signs of atrial hypertrophy on the ECG?
10. What are the signs of ventricular hypertrophy on the ECG?

A problem of the STEP-2 type
Patient K, 65 years old, came to the family doctor with complaints of shortness of breath upon light exertion and pain in the heart. He has been suffering from hypertension for more than 10 years. Blood pressure - 170/110 mm Hg. An ECG was registered in the reception room. Perform an ECG analysis.



Answer standard. Against the background of a sinus rhythm with a heart rate of 68 per 1 min, there is a deviation of the EOS to the left ((RI>RII>RII), signs of left atrial hypertrophy (in leads I, II, a double-humped P wave, 0.13s wide) and signs of left ventricular hypertrophy (R AVL=12 mV, RV5.6 > RV4, R V5.6 + SV1≥35 mm).

3. Formation of professional skills and abilities:
- formation of the ability to register and evaluate the electrocardiogram of a patient with a pathology of the cardiovascular system (the applicant must be able to register an ECG,

conduct an ECG analysis in a patient with hypertrophy of the heart chambers),

- formation of the ability to give a clinical assessment of the results obtained during ECG registration in patients with pathology of the heart and blood vessels, to give a clinical interpretation of the detected changes.

3.1. Control materials for the final stage of the lesson:

Situational tasks:

1. In what place of the conduction system of the heart does an impulse normally occur?

+A. In the sinus node

B. In the atrioventricular node

S. In the left leg of the bundle of His

D. In the right leg of the bundle of His

E. In Purkinje fibers

2. What ECG interval is used to determine the heart rate?

A. P-Q

B. QRS

C. QRST

+D. R-R

E. P-P

3. What bioelectrical process does the T wave reflect?

A. Repolarization of the left ventricle

B. Repolarization of the left atrium

S. Repolarization of both atria

D. Depolarization of both atria

+E. Repolarization of both ventricles

4. Is the Q wave characteristic normal?

A. > 1/4 R, 0.04 sec

V. < 1/4 R, 0.04 sec

+S. < 1/4 R, 0.03 sec

D. > 1/4 R, 0.03 sec

E. = 1/4 R, 0.02 sec

5. ECG signs of left atrial hypertrophy appear in leads:

+A. I, II, AVL, V5-6

B. II, III, AVF, V1-2

V. II, III, V5

G. II, AVL, V4

D. V1-3

6. ECG signs of right atrial hypertrophy appear in leads:

A. I, II, AVL, V5-6

+B. II, III, AVF, V1-2

V. II, III, V5

G. II, AVL, V4

D. V1-3

7. With right atrial hypertrophy, the P wave is:

A. Positively
B. High
B. Biphasic with a more pronounced positive phase in lead V1
G. Gostrokintsev
+D. All of the above is listed

8. The cause of left atrial hypertrophy is:

+A. Mitral stenosis
B. Insufficiency of the tricuspid valve
B. Essential hypertension
G. Bronchial asthma
E. Peptic ulcer

9. The cause of right atrial hypertrophy is:

A. Chronic obstructive pulmonary disease
B. Lung emphysema
B. Primary pulmonary hypertension
G. Bronchial asthma
+D. All of the above is listed

10. With left atrial hypertrophy, the P wave is:

A. Positively
B. Low
V. Dvohorbym
G. Extended
+D. All of the above is listed

3.2. Requirements for work results, including before registration: demonstration of the skill of recording an ECG and justification of changes in the analysis of an electrocardiogram in a patient with a pathology of the cardiovascular system.
3.3. Control materials for the final stage of the lesson: solving two clinical problems on the subject of the lesson, answering 10 tests (*if necessary*).

4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

List of recommended literature
Basic:

1. Propedeutics of internal medicine: textbook / Yu.I. Decyk, O.H. Yavorskyi, E.M. Neiko and others; under the editorship O.H. Yavorsky – 6th ed., corr. and added - K.: VSV "Medicine", 2020. - 552 p. + 12 p. color incl.
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.

Additional:

1. Methodology of examination of a therapeutic patient: teaching. manual / S.M. Andreychyn, N.A. Bilkevich, T.Yu. Chernets. – Ternopil: TDMU, 2016. – 260 p.
2. 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.
3. Yepishyn A.V. Propaedeutics of internal diseases with care of therapeutic patients / AV. Yepishyn// K. - 2015. 768p.
4. Kovalova OM. Propedeutics of internal medicine/ OM. Kovalova, NA Safargalina-Kornilova// K.: Medicine 2010 - 750p.
5.Macleod's Clinical Examination / Ed. G.Douglas, F.Nicol, C.Robertson.– 13th ed.– Elsevier. 2013. – 471 p.
6.Bates' Guide to Physical Examination and History Taking /Ed. Lynn S. Bickley, Peter G. Szilagyi. – Wolters Kluwer, 2017. – 1066 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. https://onmedu.edu.ua/kafedra/propedevtiki-vnutrishnih-hvorob-ta-terapii/
10. http://www.kolos2401.net/load/
11. http://pvb.odessa.ua/index.html

Practical lesson No. 12

Subject: ECG signs of violations of automaticity and excitability.

Goal: Acquiring knowledge and mastering the professional competencies of ECG analysis in patients with rhythm disorders, namely:
- to know the classification of heart rhythm disorders,
- know and be able to detect electrocardiographic signs of sinus and ectopic rhythms of the heart,
- to know and be able to detect electrocardiographic signs of heart rhythm disturbances in extrasystolic arrhythmia,
- know the ECG signs of extrasystolic arrhythmias of different origins,
- know and be able to detect electrocardiographic signs of paroxysmal tachycardia,
- to know and be able to detect electrocardiographic signs in flickering and fluttering of the atria and ventricles.

Basic concepts:heart rhythm disorders, which are currently a very widespread pathology, are in their vast majority life-threatening. The high prevalence of rhythm disorders among the population, including the population of Ukraine, determines the great importance of this problem for practical health care. According to WHO experts, every third patient with cardiovascular diseases suffers from various heart rhythm disorders. The tragic point of this problem is that recently there is a clear trend towards an increase in the percentage of arrhythmias in the population. For example, ventricular fibrillation is currently considered one of the main causes of sudden cardiac death. Diagnosis of many heart rhythm disorders is possible only with the help of an electrocardiographic study. Thus, the timely provision of emergency medical care largely depends on a deep knowledge of the basics of electrocardiography**.**

Classification of cardiac arrhythmias

I. Violation of the formation of impulses

1. Violation of sinus node automatism (nomotopic arrhythmias):

– sinus tachycardia;
– sinus bradycardia;
– sinus (respiratory) arrhythmia;
– sinus node weakness syndrome.

2. Increasing the automaticity of ectopic pacemakers:

– atrial rhythm;
– nodal;
– idioventricular (ventricular);
– migration of the rhythm driver;

3. Ectopic (heterotopic) arrhythmias caused by increased myocardial excitability:

– extrasystole;
– paroxysmal tachycardia;
- fluttering and flickering of the atria;
- fluttering and flickering (fibrillation) of the ventricles.

II. Conduction disorders

1. Sinoauricular blockade.

2. Intraatrial blockade.

3. Atrioventricular block (I, II, III degrees).
4. Intraventricular blockade (blockage of the legs of the bundle of His):
– blockade of the right leg of the bundle of His;
- blockade of the left leg of the bundle of His.
III. Syndrome of premature excitation of the ventricles
1. Wolf-Parkinson-White syndrome;
2. Clark-Levy-Critescu syndrome.

Sinus tachycardia - an increase in heart rate due to an increase in the automaticity of the sinus node while maintaining the correct sinus rhythm.
ECG signs of sinus tachycardia:
– heart rate = 90-160 (180) per minute;
- the presence of a P wave before the QRS complex,
- maintenance of correct sinus rhythm.
Additional features:
– shortening of the TP interval, sometimes according to the "P to T" type;
- an increase in the amplitude of the P wave in leads II, III, aVL ($P_{III} > P_{II} > P_I$);
– increasing the amplitude of the T wave;
– oblique-ascending ST depression (2 mm);
– shortening of the PQ interval (< 0.12 s for Wolf-Parkinson-White, Clark-Levy-Critescu syndromes).

Sinus bradycardia - a decrease in heart rate due to a decrease in the automaticity of the sinus node while maintaining the correct sinus rhythm.
ECG signs of sinus bradycardia:
– heart rate < 60 (59-40) per minute;
- the presence of a P wave before the QRS complex,
- maintenance of correct sinus rhythm.
Additional features:
– lengthening of the T-P interval;
- an increase in the amplitude of the P wave in leads I, aVL ($P_I > P_{II} > P_{III}$);
– smoothed T wave in lead II, negative T wave in lead III, increased T wave in lead V_1-IN_2;
– S-T segment above the isoline;
– an increase in the PQ interval > 0.2-0.01 sec.

Sinus (respiratory) arrhythmia - irregular sinus rhythm, characterized by acceleration and deceleration of the rhythm, caused by fluctuations in the tone of the vagus nerve in connection with the phases of breathing (heart rate increases during inhalation, slows down during exhalation).
ECG signs of sinus arrhythmia:
- fluctuations in the duration of the RR interval due to the phases of breathing: the interval decreases during inhalation, and increases during exhalation;
- maintenance of sinus rhythm.
Sinus node weakness syndrome – a syndrome manifested by one of the following signs or their combination:

– sustained marked sinus bradycardia (HR<50 per minute);
- the presence of ectopic rhythms against the background of sudden periodic disappearance of sinus rhythm (sinus node arrest);
- periodic occurrence of sinoauricular blockade in combination with a single ectopic complex;
- persistent pronounced bradysystolic form of atrial fibrillation);
– tachycardia-bradycardia syndrome – alternation of liquid sinus and frequent ectopic rhythms (fibrillation or fluttering).

Increasing automaticity of ectopic pacemakers
Atrial rhythm- a rhythm in which the atrial regions become the pacemaker.
General signs of atrial rhythm:
– change in polarity and deformation of the P wave (smoothed, negative, biphasic) in leads II, III, aVF, V_1-IN_6 and a positive P wave in lead aVR;
– heart rate = 60-90 per minute;
- the QRS complex is unchanged.
There are right atrial, left atrial and inferior atrial (coronary sinus rhythm).
ECG signs of right atrial rhythm:
– negative P wave in leads II, III, aVF or in leads V_1-IN_6 or in leads II, III, aVF, V_1-IN_6;
– P-Q interval > 0.12 sec;
– positive P wave in lead aVR.
ECG signs of left atrial rhythm (Fig. 3.47 b):
– negative P wave in leads II, III, aVF, V_3-IN_6, possible negative wave P in leads I, aVL, V_4-IN_6;
– P-Q interval ≥ 0.12 sec;
– positive wave P in lead aVR;
– wave P is biphasic (±, "shield-on-sword") in lead V_1.
Coronary sinus rhythm (inferior atrial rhythm) is a rhythm caused by the activation of a group of cells located in the area of the coronary sinus.
ECG signs of coronary sinus rhythm:
– negative P wave in leads II, III, aVF;
– positive wave P in lead aVR;
– two-phase or smoothed P wave in leads V_1-IN_6;
– interval PQ < 0.12 sec.

Nodal rhythm – a rhythm in which the source of excitation becomes the pacemaker of the II order (atrioventricular connection)
General ECG signs of nodal rhythm:
– HR = 30-60 per minute (for passive rhythms), HR > 60 per minute (for active rhythms);
– change in the positivity and polarity of the P wave.
ECG signs of nodal rhythm with premature atrial excitation (inferior atrial rhythm):
– negative P wave in leads II, III, aVF, V_1-IN_3;
– positive wave P in lead aVR;
– smoothed P wave in leads I, V_4-IN_6;
– interval PQ < 0.12 sec;

– Heart rate = 50-60 per minute.
ECG signs of nodal rhythm with simultaneous excitation of the atria and ventricles:
– wave P is absent in all leads because it merges with the QRS complex;
– Heart rate = 50-60 per minute.
ECG signs of nodal rhythm with premature excitation of the ventricles:
– a negative P wave occurs after an unchanged QPS complex or after a T wave in leads II, III, aVF;
– interval QP < 0.2 s;
– Heart rate = 30-60 per minute.

Idioventricular (ventricular)*rhythm* - a rhythm in which the source of excitation is the third-order rhythm driver, that is, the legs of the bundle of His.
ECG signs of idioventricular rhythm:
– HR 40 (30-40, 15-30) per minute;
– expansion (> 0.12 s) and deformation of the QRS complex;
– there is no regular relationship between the P wave and the QRS complex;
- the appearance of the P wave more often than the QRS complex;
– the R-R interval is less than the P-P interval (R-R < P-P).

Migration of the rhythm driver - arrhythmia caused by constant movement of the rhythm source from the sinus node to the atrioventricular junction and back.
ECG signs of pacemaker migration:
– alternation of different shape, amplitude and polarity of P wave;
– change in the duration of the P-Q interval;
– vague fluctuations of the R-R interval;
– the QPS complex has not been changed.

Ectopic (heterotopic) arrhythmias caused by increased myocardial excitability
Extrasystole - premature out-of-order excitation of the heart, caused by increased excitability of the myocardium, occurs against the background of the main rhythm:
1. Premature excitation (QRS complex).
2. Shortening of the preextrasystolic interval (P-P' or R-R' coupling interval)
3. Lengthening of the post-systolic interval (compensatory R'-R pause).
Extrasystoles are characterized by localization (sinus, atrial, nodal, ventricular, monotopic, polytopic), time of appearance (early, late) and regularity (single, paired, group, alorhythms).
ECG signs of sinus extrasystole:
1. Premature excitation (QRS complex).
2. Shortening of the P-P' interval.
3. The QRS complex is not changed.
4. The compensatory pause is absent or incomplete.
ECG signs of atrial (inferior atrial) extrasystole:
1. Premature excitation (the QRS complex is not changed).
2. Shortening of the P-P' interval.
3. Change in polarity and localization of the P wave.
4. Incomplete compensatory pause (2R-R').

ECG signs of nodular extrasystole:
1. Premature excitation (QRS complex).
2. The QRS complex is not changed.
3. Prong P is changed:
- negative in leads II, III, aVF - inferior atrial extrasystole;
- absent - midnodal extrasystole;
- negative, which appears after the QRS complex - infranodal extrasystole.
4. Compensatory pause:
– incomplete (<2R-R);
– full (=2R-R – for trunk extrasystole).
ECG signs of ventricular extrasystole:
1. Premature excitation (QRS complex).
2. The R wave is missing.
3. Deformation and expansion of the QRS complex (>0.12c).
4. Compensatory pause – complete (= 2R-R).
5. Discordance of the ST segment and the T wave.
Monotopic extrasystole (from one source of excitation) - all extrasystolic complexes are the same in shape (Fig. 91).
*Polytopic*extrasystole - extrasystoles occur simultaneously from different areas of the myocardium: sinus node, atria, ventricles, etc.).
*Early*extrasystole - extrasystole that appears in the first third of diastole.
It's late extrasystole - extrasystole that appears in the second half of diastole.
Allorithms - rhythms with a certain regularity of extrasystoles: bigeminy (every second), trigeminy (every third), quadrigeminy (every fourth).

Paroxysmal tachycardia - arrhythmia, characterized by the sudden onset and sudden end of an attack of increased heart rate (from 140 to 250 per minute) while maintaining a regular rhythm.
ECG signs*paroxysmal*tachycardia:
– heart rate = 140-220 per minute;
– correct rhythm (R-R = R-R);
- sudden beginning and end.
ECG signs*supraventricular* paroxysmal tachycardia:
– heart rate = 140-250 per minute or more;
– a change in the polarity of the P wave or its absence;
– the QRS complex is not changed;
– R-R interval (0.04 c).
ECG signs *ventricular* paroxysmal tachycardia:
– heart rate = 140-220 per minute;
- the P wave is absent;
– deformation of the QRS complex;
– ST segment discordance and T wave.

Atrial flutter - arrhythmia characterized by a violation of the excitability, automaticity and contractility of the atrial myocardium with an atrial excitation frequency of 250-400 per minute.
ECG signs of atrial flutter:
- the disappearance of the P wave;
- appearance of "sawtooth" f-waves in leads III, aVF, V_1-IN_2 with a frequency of 250-400 per minute;
– multiplicity of F-waves of the QRS complex (2:1, 3:1, 4:1, etc.);
– unchanged QRS complex;
– with the correct form of flutter, the R-R intervals are the same;
- with an irregular form of flutter, the R-R intervals are different.
Atrial fibrillation (atrial fibrillation) - an arrhythmia characterized by a violation of automaticity, excitability and contractile function of the atrial myocardium, in which there is frequent (350-700 per minute), disorderly (chaotic) excitation of individual muscle fibers of the atria, each of which is a kind of ectopic focus excitation.
ECG signs of atrial fibrillation:
- absence of P wave;
- the presence of irregular F-waves in leads III, av, V_1 with a frequency of 350-700 per minute (duration of the F-wave from 0.04 s to 0.12 s);
– the rhythm is incorrect (different R-R intervals).
– different amplitude of QRS complexes.
They distinguish:
a) long-wave (amplitudeF- waves >5 mm with a frequency of 350-450 per minute);
b) medium-wave (amplitudeF-waves = 2 mm with a frequency of 450-550 per minute);
c) small-wave (amplitudeF- waves of 1-2 mm with a frequency of 550-700 per minute).
Fluttering of the ventricles - arrhythmia caused by impaired excitability, automaticity and contraction of the myocardium of the ventricles, often develops against the background of ventricular paroxysmal tachycardia.
ECG signs of ventricular flutter:
– ECG has the form of a sinusoid;
– deformation of the ventricular QRS complex, fusion of its initial and final parts;
– absence of isoline (TR);
- lack of ST segment and T and P waves;
– Heart rate = 200-300 per minute.
Flickering of the ventricles (ventricular fibrillation) is a rhythm disorder characterized by frequent (200-500 per minute) disordered irregular contractions of individual muscle fibers of the ventricles. This terminal disorder is caused by asynchronous electrical activity of individual muscle fibers.
ECG signs of ventricular fibrillation:
- there is no differentiation of all the waves, ECG in the form of a small-wave and large-wave line.
– Heart rate is about 200-500 per minute.

Plan:

1. **Organizational measures** (greetings, verification of those present, announcement of

the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2. Control of the reference level of knowledge (checking workbooks, carrying out ECG analysis in patients with signs of heart rhythm disorders), conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for theoretical readiness of applicants and performance of practical classes:

- the applicant must know the main types of heart rhythm disorders,
- the applicant must know the main types of arrhythmias associated with impaired automaticity and excitability,
- the applicant must know ECG signs of sinus tachy- and bradyarrhythmia, nodular arrhythmia,
- the applicant must know the ECG signs of paroxysmal tachycardia (supraventricular and ventricular),
- the applicant must know the ECG signs of ventricular and atrial flutter and flutter.

List of didactic units:

– to be able to detect signs of sinus rhythm of the heart and its variants (sinus bradycardia, sinus tachycardia, sinus arrhythmia) on the ECG,
– to be able to detect signs of LV dysfunction (weakness of the sinus node) on the ECG,
– be able to detect signs of ectopic heart rhythms (extrasystoles, paroxysmal tachycardia) on the ECG,
– to be able to distinguish the ECG in ventricular and supraventricular paroxysmal tachycardia,
– to be able to detect electrocardiographic signs of heart rhythm disturbances in atrial flutter and flutter, electrocardiographic signs of heart rhythm disturbances in ventricular flutter and flutter.

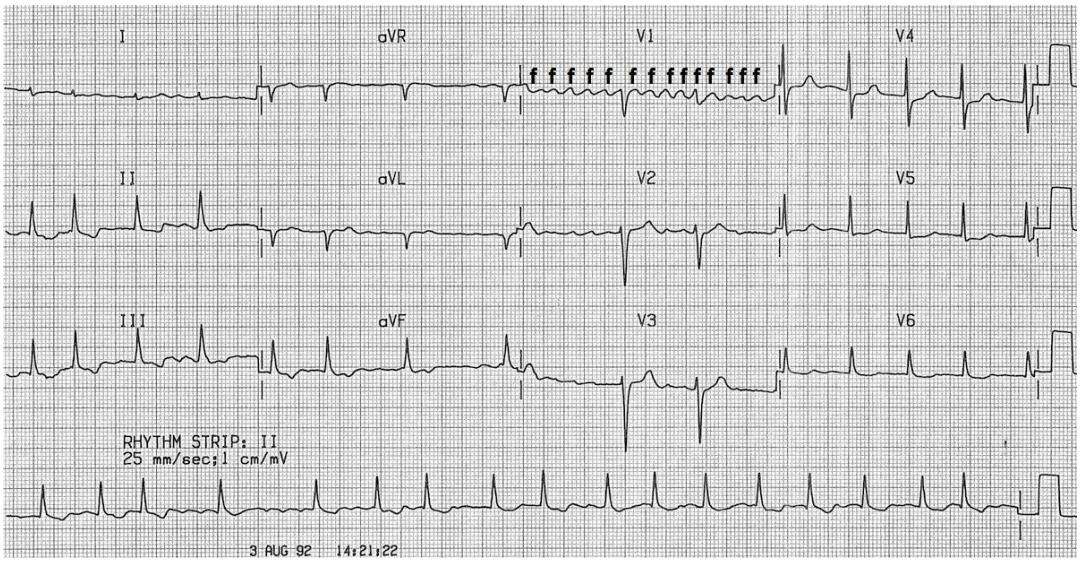
2.2. **Questions to check basic knowledge on the topic of the lesson:**

1. What rhythm disorders are classified as automaticity disorders?
2. What rhythm disorders are classified as excitability disorders?
3. What are the ECG signs of sinus tachycardia and bradycardia?
4. What are the ECG signs of supraventricular extrasystoles?
5. Name the ECG signs of ventricular extrasystoles.
6. What is alorhythmia?
7. Name the ECG signs of supraventricular tachycardia.
8. Name the ECG signs of ventricular tachycardia.
9. What are the ECG signs of atrial flutter?
10. What are the ECG signs of atrial fibrillation?
11. Name the ECG signs of ventricular flutter and fibrillation.

A problem of the STEP-2 type

Patient K., 70 years old, came to the doctor with complaints of palpitations and irregular pulse. During the examination, an arrhythmic pulse with a frequency of 78 per minute was

detected, auscultatory heart rate = 98 per minute (pulse deficit = 20). Registered ECG. Give an analysis.



Answer standard. Since during the examination of the patient, a pulse deficiency was found, and the P wave was absent on the ECG (f waves were recorded instead), the interval between the QRS complexes was different, these characteristics are pathognomonic for atrial fibrillation.

3. Formation of professional skills and abilities:

- formation of the ability to register and evaluate the electrocardiogram of a patient with a pathology of the cardiovascular system (the applicant must be able to register an ECG, conduct an ECG analysis in a patient with a heart rhythm disorder),

- formation of the ability to give a clinical assessment of the results obtained during ECG registration in patients with pathology of the heart and blood vessels, to give a clinical interpretation of the detected changes.

3.1. Control materials for the final stage of the lesson:

Situational tasks:

1. Which type of arrhythmia does not belong to a violation of myocardial excitability?

+A. Extrasystole

B. Sinus arrhythmia

S. Sinus bradycardia

D. His leg block

E. Atrial fibrillation

2. What is the ECG sign of ventricular extrasystole?

A. There is no regular relationship between the P wave and the QRS complex

+B. Premature appearance of a wide QRS complex (> 0.12sec)

C. Deformation and expansion of the QRS complex (> 0.12sec)

D. Shortening of the P-P' interval

E. The presence of the R wave

3. What is the ECG sign of ventricular flutter?

A. Tachycardia-bradycardia syndrome

B. Alternation of different shape, amplitude and polarity of the P wave
C. There is no regular relationship between the P wave and the QRS complex
D. Disappearance of the R wave
+E. The ECG has the form of a sinusoid

4. A negative R wave is registered on the ECG. What is your ECG conclusion?

A. Sinus rhythm
+V. Atrial rhythm
S. Idioventricular rhythm
D. Sinus arrhythmia
E. Sinus arrhythmia

5. In which case is there no differentiation of all the waves on the ECG?

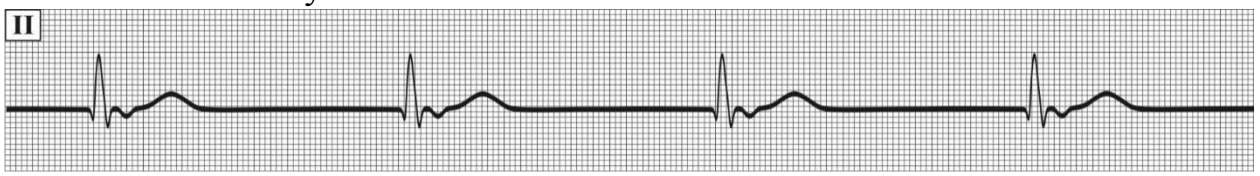
A. Atrial fibrillation
B. Atrial fibrillation
S. Fluttering of the ventricles
+D. Ventricular fibrillation
E. Artificial pacemaker

6. In a patient with ischemic heart disease, an ECG is recorded in the form of a small- and large-wave line. Name a possible rhythm disorder.

A. Atrial fibrillation
+V. Ventricular fibrillation
C. Atrial flutter
D. Ventricular flutter
E. Artificial pacemaker

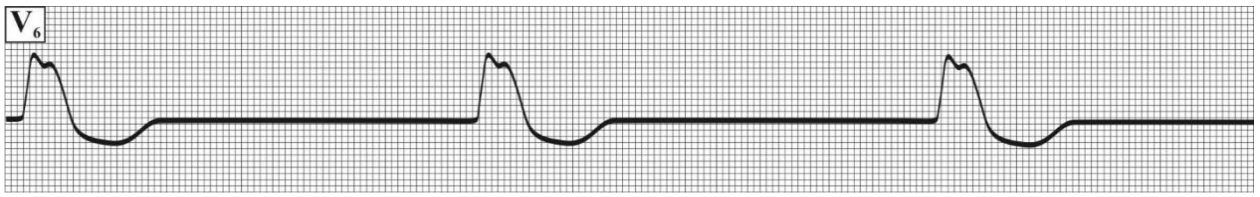
7. Describe the following ECG:

A. Sinus rhythm.
B. Rhythm with AV coupling with simultaneous excitation of the atria and ventricles.
+S. Rhythm with AV coupling with preliminary excitation of the ventricles.
D. Rhythm with AV coupling with prior excitation of the atria.
E. Idioventricular rhythm.



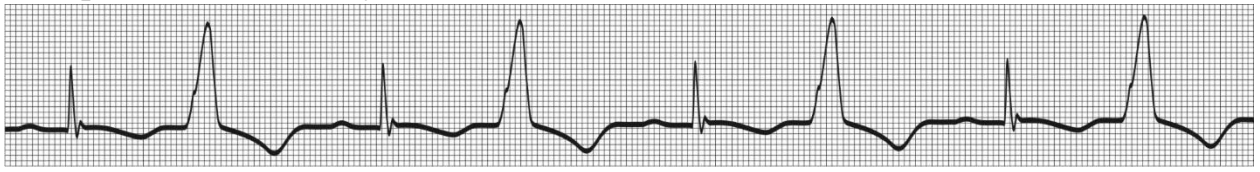
8. Describe the following ECG:

A. Sinus rhythm.
B. Sinus bradycardia.
S. Sinus arrhythmia
D. Rhythm with AV coupling with simultaneous excitation of the atria and ventricles.
+E. Idioventricular rhythm



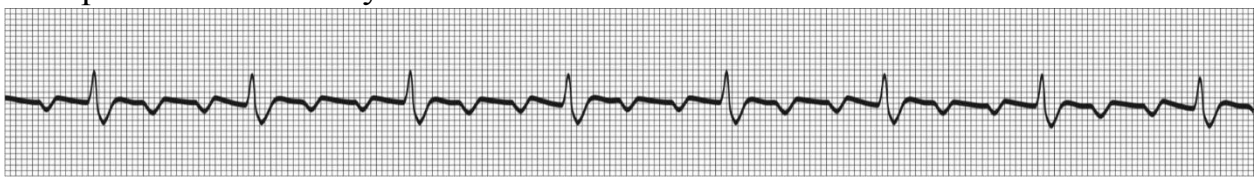
9. Describe the following ECG:

A. Atrial extrasystoles.
+V. Ventricular extrasystoles.
S. Atrial fibrillation.
D. Atrial flutter.
E. Supraventricular tachycardia



10. Describe the following ECG:

A. Atrial extrasystoles.
V. Ventricular extrasystoles.
S. Atrial fibrillation.
+D. Atrial flutter.
E. Supraventricular tachycardia



3.2. Requirements for work results, including before registration: demonstration of the skill of recording an ECG and justification of changes in the analysis of an electrocardiogram in a patient with a pathology of the cardiovascular system.
3.3. Control materials for the final stage of the lesson: solving two clinical problems on the subject of the lesson, answering 10 tests (*if necessary*).

4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 13

Subject: ECG signs of conduction disorders.

Goal: Acquiring knowledge and mastering the professional competencies of ECG analysis in patients with conduction disorders, namely:
- know the location of the main drivers of the rhythm of the conducting system of the heart,
- to know the hierarchy of the main rhythm drivers and the speed of impulse conduction in different parts of the conduction system,

- know and be able to detect electrocardiographic signs of sinoatrial blockades,
- know and be able to detect electrocardiographic signs of atrioventricular block,
- know and be able to identify electrocardiographic signs of intraventricular blockade (blockage of the legs of the bundle of His and their branches).

Basic concepts: Despite the significant progress of instrumental research methods, electrocardiography remains one of the most common and informative examination methods in clinical practice. This method is a priority in the diagnosis of heart rhythm and conduction disorders, acute myocardial infarction, heart defects, hypertensive heart and other diseases. As for conduction function disorders, electrocardiography is the only diagnostic method, thanks to which a timely diagnosis of the type of disorders will ensure the patient's life. Conduction of the excitation pulse can be slowed down or interrupted in this section of the conducting system. Depending on the place where it occurs, sinoauricular, intraatrial, atrioventricular and intraventricular blockades are distinguished.

Sinoatrial block

Sinoatrial block is a slowing or termination of the conduction of the sinoatrial (CA) connection, as a result of which the conduction of some impulses from the CA is slowed down or they cannot reach the atria at all and the heart does not contract. There are three degrees of CA blockade: I and II - incomplete and full III degree. With complete CA blockade (III degree), all sinus impulses are blocked and none of them can reach the atria. Thus, complete disappearance of P waves and their corresponding QRS complexes occurs.
*Hemodynamics:*CA blockade can cause or worsen arterial hypotension, heart failure, myocardial and brain ischemia. Prolonged complete SA blockade leads to cardiac asystole, which can be fatal.
*Clinic.*Irregular heart activity and the feeling of "as if the heart has stopped" are caused by pauses in CA blockade. Asystole of the heart, which lasts a few seconds, causes the appearance of various symptoms from the brain, up to the developed picture of MES syndrome.
Establishing a diagnosis of CA blockade is possible only on the basis of ECG studies.

Intraatrial blockade

With intraatrial blockade, the passage of the sinus impulse through one or more internodal conduction paths of the atria is slowed down or interrupted.
ECG signs:

— change in shape, amplitude and polarity of P;
— P> 0.12 s.

Atrioventricular block (AV block)

AV block is a violation of the conduction of excitation impulses from the atria to the ventricles, which can be slowed down or interrupted due to a pathological prolongation of the refractory period of the atria, AV node and bundle of His (or) of both legs of Tavar.
Classification of AV blockade:

- ❖ Incomplete (partial)
- I degree (PQ interval more than 0.22 sec)
- II degree: type I – Samoilova-Wenkebach, Mobitz type I;

type II – Mobitz type II or with a constant PQ interval.
- ❖ III degree (full).

First-degree AV block characterized by lengthening of the interval between the excitation of the atria and ventricles PQ (in the absence of the Q wave, we estimate the PR interval) above the upper normal limit, taking into account age and heart rate. For adults, PR>0.20 s, for children >0.18 s is considered first-degree AV block. With first-degree AV block, conduction of impulses through the AV system is slowed down, but all sinus or ectopic impulses reach the ventricles, the rhythm remains correct.
ECG criteria:
1. Prolonged PQ interval>0.21 s, but not more than 0.45-0.5 s.
2. All atrial impulses are conducted into the ventricles and therefore a QRS complex appears after each P wave.

Incomplete AV blockade of the first degree can turn into complete AV blockade. Clinically, it manifests itself as MES syndrome.

Atrioventricular block II degree.
- ❖ type I – Samoilova-Wenkebach, Mobitz type I;
- ❖ type II – Mobitz type II or with a constant PQ interval.

Type I - in the case of second-degree AV blockade, a gradual slowing of conduction through the AV node is observed, from one complex to the next, up to the complete delay of one (rarely two or three) electrical impulses.
At the same time, a gradual lengthening of the PQ interval with subsequent loss of the QRST complex can be observed on the ECG. After a prolonged pause - a period of failure of ventricular contraction - conduction through the AV node is restored and a normal or slightly prolonged PQ interval is again recorded on the ECG, after which the entire cycle is repeated again. And type II AV blockade is most often observed with proximal (nodal) conduction disturbances and therefore, of course, is not accompanied by deformation of the ventricular complexes. It often turns into complete AV block.
At***Type II*** In II-degree AV blocks, failure of ventricular contractions is not accompanied by a gradual lengthening of the PQ interval, which remains constant (normal or prolonged). Loss of ventricular complexes can be regular or irregular. This type of blockade is more often observed in distal atrioventricular conduction disorders at the level of the bundle of His. Therefore, with this type of AV block, QRS complexes can be widened and deformed.
Hemodynamics: is determined by the frequency of ventricular contractions, the leading heart disease and the contractility of the heart muscle.
Clinical picture: subjective symptomatology depends on the duration and frequency of heart contractions of the ventricles. With longer pauses of the ventricles, especially in the presence of atherosclerosis of the brain, symptoms of cerebral ischemia, dizziness, weakness almost to the point of an MES attack may appear.

Incomplete AV blockade of the 3rd degree
With this version of the blockade, either every second, or two (2:1) or more ventricular complexes in a row appear on the ECG (blockade 3:1, 4:1). This leads to sharp bradycardia,

against the background of which dizziness and loss of consciousness may occur. QRS complexes can be either unchanged in proximal blockade or widened and deformed in distal blockade.

Complete AV block

With complete AV block, none of the atrial impulses (sinus or ectopic) are conducted to the ventricles due to complete interruption of the conduction of the AV node, bundle of His or both of its legs. As a result, a complete AV dissociation occurs, that is, contractions of the atria and ventricles occur independently of each other. Complete AV block may result from interruption of conduction in the AV node, bundle of His, or both bundles. In nodal complete AV block, the replacement ectopic center is most often located in the lower part of the AV node or in the bundle of His below the site of blockade, while in complete AV block due to bilateral blockade of the bundle of His, the replacement center is located somewhere in the legs, below places of blockade.

Complete AV block is the result of lengthening of the absolute refractory period, thus the latter occupies the entire heart contraction cycle and the phase of the relative refractory period is absent.

Conductive***features of hemodynamics:***

– increased systolic volume
– arterial systolic hypertension with a large pulse amplitude;
– increased systolic pressure in the pulmonary artery and in the cavities of the right heart;
– normal or reduced cardiac output at rest
– decreased minute volume during exercise.

Clinical objective symptoms:

— Bradycardia of the correct rhythm, HR<40 per minute.
— Heart rate is usually constant
— Intermittent accentuation of the 1st tone ("cannon" tone Strazhesco)
— Dull atrial sounds during long diastole ("echoes" symptom)
— Weak pulsations of the jugular veins during diastole, corresponding to atrial systole
— Arterial pulse is slowed, satisfactory filling
— MES syndrome

Atrial waves do not have a constant connection with the ventricular complexes, as a result of which the PQ interval continuously changes in length, and the P waves fall into different places and are superimposed on other elements of the ECG.

The frequency of ventricular complexes (QRS) and their width depends on the localization of the center of the replacing impulses.

N.B. attacks of MES syndrome must be distinguished from other diseases that occur with transient loss of consciousness and convulsions (epilepsy). Changes in the color of the skin are of great importance for the differential diagnosis of epilepsy. In the first seconds of an MES attack, the patient's face is markedly pale. At the moment of spasms, it becomes bluish and reddens even at the first heartbeats. During an epileptic seizure, the face is plethoric and bluish, and after the seizure stops, the face becomes pale.

Violation of intraventricular conduction

(blockage of the legs of the bundle of His)
Complete blockade of the legs of the bundle of His refers to blockades with a pronounced asynchrony of the excitation of the ventricles and an increase in the duration of the QRS > 0.12 c. These forms of bundle branch block are caused either by the complete cessation of conduction in the corresponding bundle or by a sharp slowing of conduction, as a result of which the excitation wave reaches the blocked ventricles only by a circuitous route from the other ventricle.

Blockade of the right leg of the bundle of His
Clinic. Significant splitting of the 1st and 2nd tones. Delayed activation of the right ventricle causes asynchrony of the contractions of both ventricles and non-simultaneous closing of the bicuspid and tricuspid valves with significant splitting of the first sound, as well as non-simultaneous closing of the aortic and pulmonary valves with splitting of the second sound. Cleavage increases during inhalation, when, due to the filling of the ventricles with blood, there is a physiological slowing down of the closing of the pulmonary valve.
ECG:
1. Extended QRS complex>0.12 s;
2. Split QRS complex in the form of the letter "M", rSR, rsR, RSR (V_1, V_2, V_3, avR)
3. Deep, wide and jagged S with a duration of more than 0.04 s in leads V_4-V_6, sometimes in II and avL
4. Downshifted ST segment and negative T wave in leads: III, avF, V_1, V_2, V_3

Blockade of the left leg of the bundle of His
In most cases, complete blockade of the left leg of the bundle of His is associated with a high degree of hypertrophy and damage to the myocardium of the left ventricle.
Clinic. Blockade of the left leg causes bifurcation of the second sound in a paradoxical type, that is, the pulmonary component precedes the aortic component of the second sound. Splitting is more pronounced during expiration and yawns or decreases during inspiration, when closure of the pulmonary valves is physiologically delayed, in which case the pulmonary and aortic components of the second sound approach each other.
ECG criteria:
1. The QRS complex is extended, duration >0.12 s
2. Wide and split R wave in leads I, avL, V_5, V_6
3. Widened and jagged S wave or complex QS and leads V_1, V_2, III, avF
4. ST segment shifted down with a negative asymmetric T wave in leads I, avL, V_5, V_6.

Plan:
1. Organizational measures (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking workbooks, carrying out ECG analysis in patients with signs of heart rhythm disorders), conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for theoretical readiness of applicants and performance of practical

classes:
- to know the hierarchy of the main rhythm drivers and the speed of impulse conduction in different parts of the conduction system,
- know the electrocardiographic signs of sinoatrial blocks,
- know electrocardiographic signs of atrioventricular block,
- to know the electrocardiographic signs of intraventricular blocks (blockage of the legs of the bundle of His and their branches).

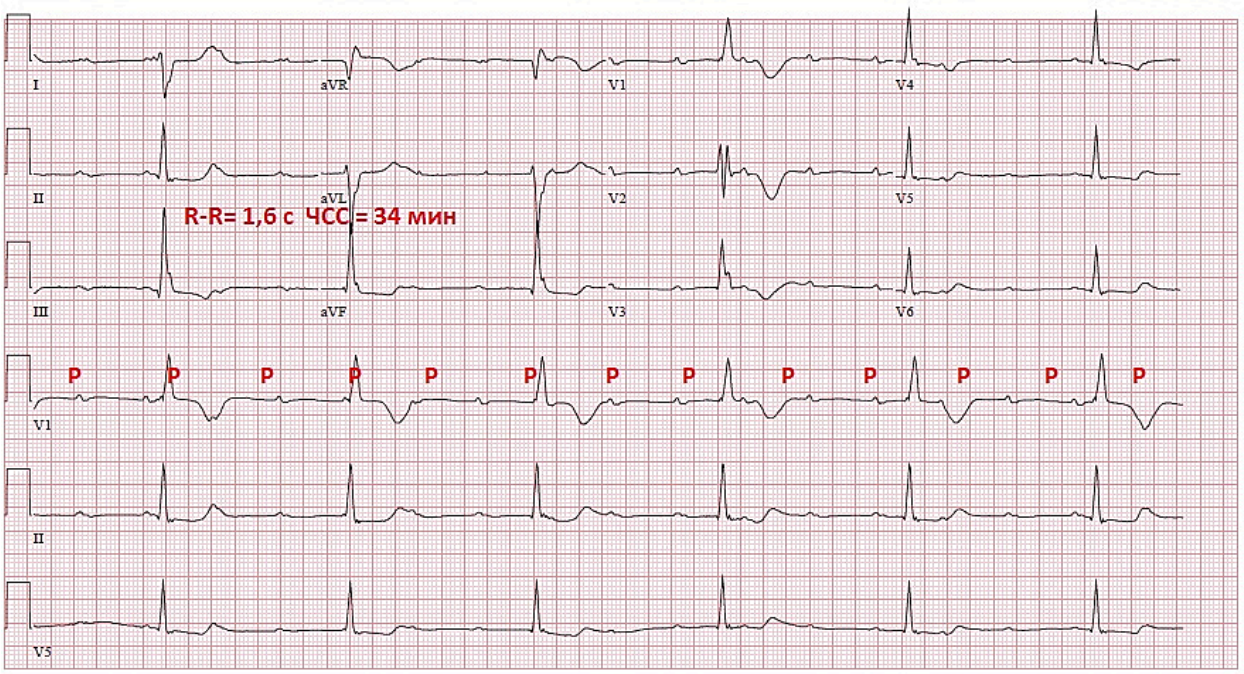
List of didactic units:
- be able to detect sinoatrial blockade on the ECG,
- to be able to detect atrioventricular blockade of various degrees on the ECG,
- to be able to detect blockade of the right and left branches of the bundle of His on the ECG.

2.2. **Questions to check basic knowledge on the topic of the lesson:**

1. What are the first, second and third order rhythm drivers?
2. What is the hierarchy of pacemakers in the conduction system of the heart?
3. What are the normative indicators of the PQ interval on the ECG?
4. Is atrioventricular block of the first degree accompanied by hemodynamic disturbances?
5. What hemodynamic disturbances are possible with blockades of the II degree?
6. What hemodynamic disturbances are possible with blockades of the III degree?
7. What signs on the ECG are characteristic of the blockade of the 1st degree?
8. What signs on the ECG are characteristic of the blockade of the II degree?
9. What signs on the ECG are characteristic of the blockade of the III degree?
10. What signs on the ECG are characteristic of blockade of the right branch of the bundle of His?
11. What signs on the ECG are characteristic of blockade of the left branch of the bundle of His?

A problem of the STEP-2 type

Patient K., 78 years old, turned to the doctor with complaints of weakness, shortness of breath, episodes of dizziness. During the examination, blood pressure = 160/60 mmHg, heart rate - 34 per minute. An ECG was taken. Give an analysis of rhythm disturbances on the ECG.



Answer standard. The frequency of ventricular contractions (R-R interval = 1.6 seconds) on the ECG is 34 per 1 minute, the frequency of atrial contractions (R-R interval) is 100 per 1 minute. The atria contract in their own rhythm (with a frequency of 100 per 1 min), and the ventricles in their own with a frequency of = 34 per 1 min. Such changes are characteristic of complete atrioventricular blockade.

3. Formation of professional skills and abilities:

- formation of the ability to register and evaluate the electrocardiogram of a patient with a pathology of the cardiovascular system (the applicant must be able to register an ECG, conduct an ECG analysis in a patient with a conduction disorder),

- formation of the ability to give a clinical assessment of the results obtained during ECG registration in patients with pathology of the heart and blood vessels, to give a clinical interpretation of the detected changes.

3.1. Control materials for the final stage of the lesson:

Situational tasks:

1. The P-Q interval is:

a. The time of passage of the impulse through the atria.
b. Atrioventricular delay time.
c. The time of passage of the impulse from the sinus node to the atrium.
d. Time of passage of the pulse through the His system.
e. ***Time of passage of the impulse through the atria, atrioventricular node, His system to the working myocardium.***

2. Normally, the P-Q interval is equal to:

a. 0.05-0.06 s.
b. 0.08-0.09 s.
c. 0.10-0.12 s.
d. 0.07-0.14 s.
e. ***0.12-0.12 s.***

3. What ECG interval is used to determine the frequency of heart impulses?

a. P-Q
b. QRS
c. QRST
d. ***R-R***
e. P-P

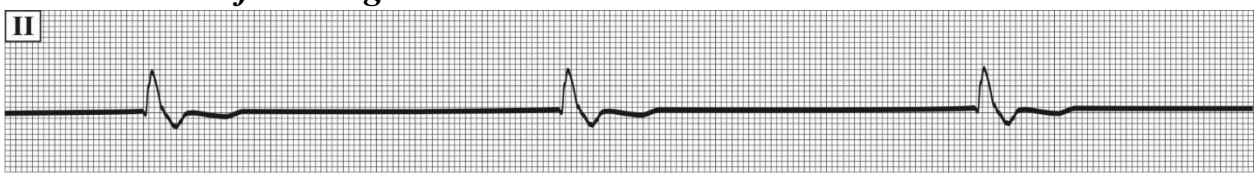
4. What element of the ECG shows the conduction of the impulse through the AV junction?

a. ***Segment P-Q***
b. R-T interval
c. Zubets R
d. Zubets T
e. QRS complex

5. Which element of the ECG reflects the conduction of the impulse along the legs of the bundle of His?

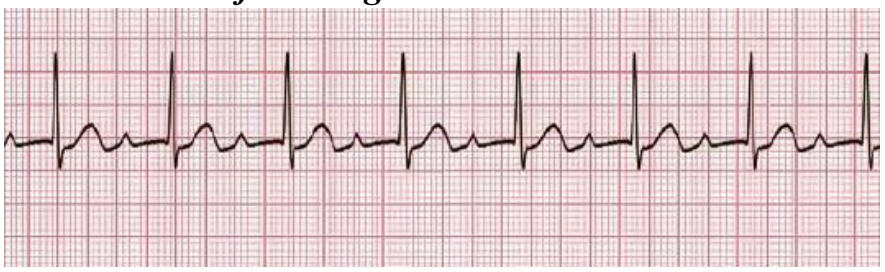
a. Segment P-Q
b. P-Q interval
c. Zubets R
d. Zubets T
e. ***QRS complex***

6. Describe the following ECG:



a. Migration of the supraventricular pacemaker
b. Rhythm with AV coupling with simultaneous excitation of the atria and ventricles.
c. Rhythm with AV coupling with preliminary excitation of the ventricles.
d. Rhythm with AV coupling with prior excitation of the atria.
e. ***Idioventricular rhythm.***

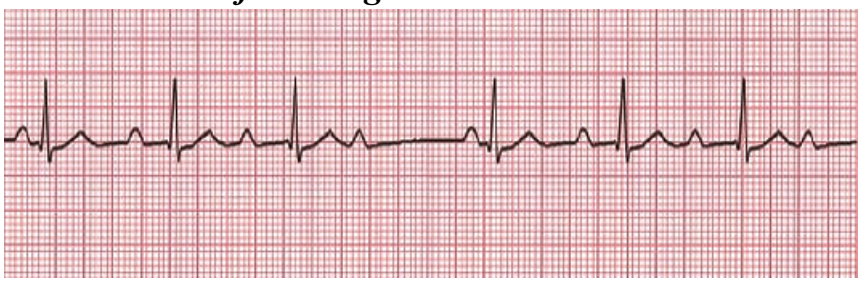
7. Describe the following ECG:



a. Sinoatrial block,
b. ***Atrioventricular blockade of the first degree,***

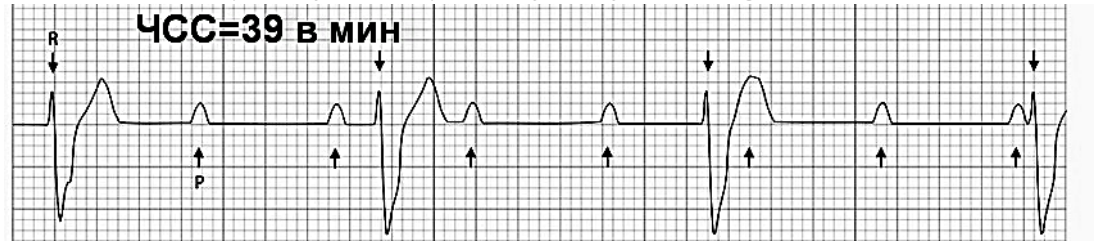
c. Atrioventricular block II degree,
d. Atrioventricular blockade of the III degree,

8. Describe the following ECG:



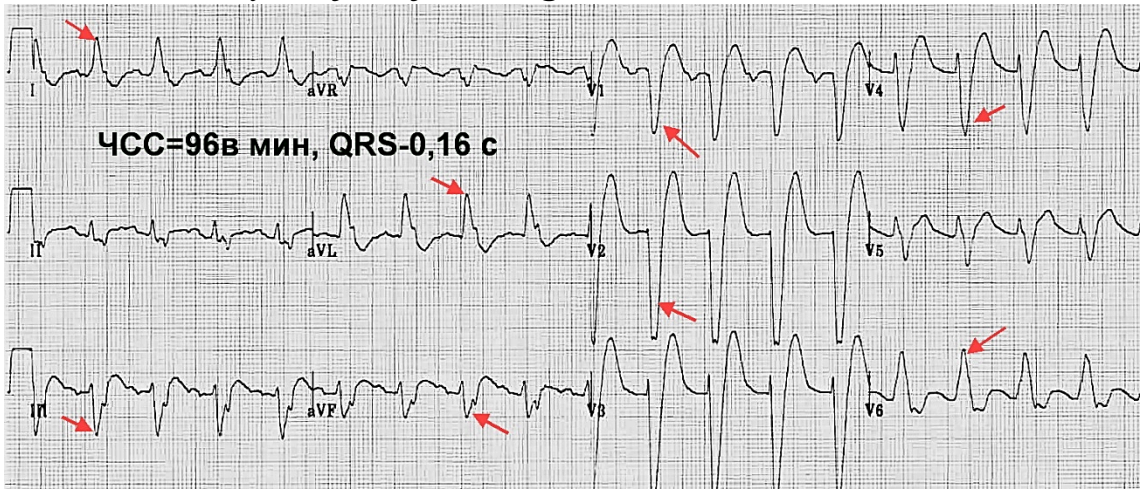
a. Sinoatrial block,
b. Atrioventricular blockade of the first degree,
c. ***Atrioventricular block II degree,***
d. Atrioventricular blockade of the III degree,

9. Give an analysis of the rhythm of the following ECG



a. Sinoatrial block,
b. Atrioventricular blockade of the first degree,
c. Atrioventricular block II degree,
d. ***Atrioventricular blockade of the III degree,***

10. Give an analysis of the following ECG



a. On the ECG, signs of left ventricular hypertrophy,
b. On the ECG, signs of left ventricular hypertrophy,
c. ***On the ECG, there are signs of blockade of the left leg of the branch of His,***
d. On the ECG, there are signs of blockade of the right leg of the branch of His,

3.2. Requirements for work results, including before registration: demonstration of the skill of recording an ECG and justification of changes in the analysis of an electrocardiogram in a patient with a pathology of the cardiovascular system.
3.3. Control materials for the final stage of the lesson: solving two clinical problems on the subject of the lesson, answering 10 tests (*if necessary*).

4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

List of recommended literature

Basic:

1. Propedeutics of internal medicine: textbook / Yu.I. Decyk, O.H. Yavorskyi, E.M. Neiko and others; under the editorship O.H. Yavorsky – 6th ed., corr. and added - K.: VSV "Medicine", 2020. - 552 p. + 12 p. color incl.
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.

Additional:

1. Methodology of examination of a therapeutic patient: teaching. manual / S.M. Andreychyn, N.A. Bilkevich, T.Yu. Chernets. – Ternopil: TDMU, 2016. – 260 p.
2. 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.
3. Yepishyn A.V. Propaedeutics of internal diseases with care of therapeutic patients / AV. Yepishyn// K. - 2015. 768p.
4. Kovalova OM. Propedeutics of internal medicine/ OM. Kovalova, NA Safargalina-Kornilova// K.: Medicine 2010 - 750p.
5.Macleod's Clinical Examination / Ed. G.Douglas, F.Nicol, C.Robertson.– 13th ed.– Elsevier. 2013. – 471 p.
6.Bates' Guide to Physical Examination and History Taking /Ed. Lynn S. Bickley, Peter G. Szilagyi. – Wolters Kluwer, 2017. – 1066 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. https://onmedu.edu.ua/kafedra/propedevtiki-vnutrishnih-hvorob-ta-terapii/
10. http://www.kolos2401.net/load/
11. http://pvb.odessa.ua/index.html

Practical lesson No. 14

Subject: Questioning and examination of patients with diseases of the digestive organs. Examination and superficial palpation of the abdomen.

Goal: Acquiring knowledge and mastering professional competences during questioning and

physical examination of the gastrointestinal tract (examination, superficial and deep palpation of the abdomen).

Basic concepts: The method of questioning and detailing the complaints of a patient with a pathology of the gastrointestinal tract. Peculiarities of collecting medical and life anamnesis. Changes in the appearance of the patient with various pathologies of the gastrointestinal tract. The sequence of the examination of the abdomen (shape, dimensions, symmetry, condition of the skin and navel, fatness, condition of subcutaneous vessels, pattern of hair). The concept of topographic zones and topographic lines on the surface of the abdomen. Tasks and methods of conducting surface palpation of the abdomen (methodology and technique of palpation, checking for symptoms of peritoneal irritation, detection of separation of rectus abdominis muscles, presence of umbilical hernias and hernias of the white line of the abdomen). Methods of detecting ascites (survey, percussion, fluctuations). The sequence of conducting deep sliding methodical palpation of the intestinal sections according to the Obraztsov-Strazheska method: normal parameters of the sigmoid, cecum, terminal ileum, ascending, descending and transverse colon. Methods of determining the lower border of the stomach (percussion, palpation, stetoacoustic, by the sound of a splash). Rules of palpation of the goalkeeper.

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, attention is paid to the nature of the pain syndrome. Correctly assessed pain syndrome in patients with gastroenterological pathology is key to 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 colic, gastritis, dyskinesias 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 abdominal cavity 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.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:
1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking workbooks, communication with a patient with pathology of the digestive organs in order to collect complaints and history, general and local examination, superficial and deep palpation of the abdomen, assessment of examination data), conducting test control, solving a clinical problem, written solving tasks of the Step-2 type (10 tasks), face-to-face survey, discussion, role-playing on the topic of the lesson.
2.1. Requirements for the theoretical readiness of applicants to perform practical classes:
the applicant must know the methods and scheme of examination of a patient with diseases of the digestive organs,
have an idea of the role of domestic scientists in the development of these methods; to know the general methodology of questioning patients with diseases of the digestive organs;
know the characteristics and mechanisms of the main complaints (pain, dyspeptic complaints); the meaning and purpose of a general and local examination of a patient with a pathology of the digestive organs;
know the topographic lines and areas of the abdomen;
to know the purpose, tasks and methods of superficial and deep palpation of the abdomen.
List of didactic units:
- collect in detail the complaints and history of a patient with a pathology of the digestive organs;
- conduct a physical examination of the patient (general examination, examination of the abdomen, superficial and deep palpation of the abdomen), identify and evaluate the changes that have been found.

2.2. Questions to check basic knowledge on the topic of the lesson:
question:

1. Complaints of patients with stomach diseases.
2. What are the mechanisms of pain in stomach diseases?
3. Types of pain by the time of its occurrence from the moment of eating, seasonality in the nature of pain.
4. Types of vomiting according to the mechanism of its occurrence.
5. Signs of gastric bleeding.
6. What is heartburn? Causes and mechanism of heartburn.
7. Types of appetite disorders.
8. Complaints of patients with intestinal diseases.
9. What is diarrhea? Causes and mechanisms of diarrhea.
10. What is intestinal dyspepsia?
11. What is constipation? Causes and mechanisms of constipation.
12. What are the signs of upper intestinal bleeding?
13. What are the signs of bleeding from the lower intestines?
14. What should you pay attention to during an examination of the oral cavity?
15. What topographic areas is the stomach divided into?
16. What changes can be detected during an examination of the abdomen?
17. What is the purpose of superficial indicative palpation of the abdomen?
18. Methodology and technique of superficial abdominal palpation.

A problem of the STEP-2 type. Patient M., 30 years old, complains of acid belching, heartburn, pain in the epigastric region, which occur on an empty stomach, 1.5-2 hours after eating, at night, constipation, weakness. He has been sick for about 5 years. Deterioration of well-being is noted after spicy food, in spring and autumn. Objectively: the tongue is coated with a white coating. Abdomen on palpation is soft, painful in the epigastric region.
What disease can be assumed in the patient?
A - gastric ulcer
+B - duodenal ulcer
C - gastritis
D - gastroduodenitis
E - cholecystitis

3. Formation of professional skills and abilities:
mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the conducted interrogation with the assumption of the localization of the pathological process)
formation of the ability to perform a clinical examination of a patient with a pathology of the digestive organs (the candidate must be able to perform a general and local examination of a patient with diseases of the digestive organs; perform superficial and deep palpation of the abdomen),
formation of the ability to give a clinical assessment of the data obtained during palpation of the abdomen; carry out a clinical interpretation of the main symptoms and syndromes in diseases of the digestive organs.

3.1. Control materials for the final stage of the lesson:
Situational tasks:
Task 1. The patient's examination revealed a red "lacquered" tongue with smoothed papillae. For what pathological conditions is this characteristic?
Task 2. The patient turned to the doctor with complaints of pain in the subspiramental area, heartburn, and acid belching. What do these complaints indicate? What is the plan for their detailing?
Answers:
1. Such a change in the tongue is characteristic of atrophic gastritis, stomach cancer, V_{12}-deficiency anemia.
2. About the defeat of the stomach. Irradiation, periodicity, connection with food intake, conditions of appearance and disappearance.

Tasks of the STEP-2 type

1. Patient A., 27 years old, suffers from peptic ulcer disease. Periodically there is black stool. A decrease in the number of erythrocytes and hemoglobin is noted in the blood.
What disease could the doctor think of?
+A - bleeding
U - gastritis
C - cholecystitis
D - hepatitis
E - pancreatitis

2. Patient G., 42 years old, brought to the surgical department in serious condition. Complaints of sudden "dagger" pain in the stomach. He has a history of gastric ulcer. About: pulse 120 beats in 1 minute, rhythmic blood pressure 80/60 mm. mercury Art. The abdomen participates in the act of breathing, it is board-like, sharply painful.
What disease should the doctor assume?
+A - gastric ulcer, perforation
In - gatekeeper stenosis
C - malignancy
D - penetration
E - bleeding

3. Patient M., 50 years old, complains of a feeling of weight in the epigastric region, belching of a "rotten" egg, profuse vomiting of food eaten the day before. For many years he has been suffering from peptic ulcer disease with frequent exacerbations. About: Medium weight condition. The abdomen is soft, during palpation there is diffuse tenderness in the epigastric region, "a splashing noise" at the level of the navel.
What disease can be assumed in the patient?
+A - gatekeeper stenosis
In - peptic ulcer of the stomach

C - peptic ulcer disease of the DPK
D - cholecystitis
E - hepatitis

4. Patient K., 45 years old, has been suffering from rheumatoid arthritis for 5 years. For the past 3 years, he has been taking steroid hormones - prednisolone. He periodically feels pain in the epigastric region, which often occurs after 30-60 minutes. after eating, heartburn, nausea. Overall, the condition is satisfactory, the tongue is coated with white plaque. The abdomen is soft, painful in the epigastrium.
What preliminary diagnosis can be given to the patient?
+A - medicated steroid stomach ulcer
In - peptic ulcer disease of the DPK
C - cholecystitis
D - hepatitis
E - gastritis

3.2. Requirements for work results, including before registration: substantiation of the syndromic diagnosis based on complaints, medical history and life data, clinical examination.

3.3. Control materials for the final stage of the class: solving two clinical problems on the subject of the class, answering 10 tests (if necessary).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 15

Subject: Deep sliding methodical palpation of the abdomen.

Goal: Acquiring knowledge and mastering professional competences during deep palpation of the abdomen).

Basic concepts: The technique and sequence of deep sliding methodical palpation of the intestinal sections according to the Obraztsov-Strazheska method: normal parameters of the sigmoid, cecum, terminal ileum, ascending, descending and transverse colon. Methods of determining the lower border of the stomach (percussion, palpation, stetoacoustic, by the sound of a splash). Rules for palpation of the pyloric part of the stomach.

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 according to certain rules that 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 points: 1. placing 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 the 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.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:

1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking workbooks, communication with a

patient with pathology of the digestive organs in order to collect complaints and history, general and local examination, superficial and deep palpation of the abdomen, assessment of examination data), conducting test control, solving a clinical problem, written solving tasks of the Step-2 type (10 tasks), face-to-face survey, discussion, role-playing on the topic of the lesson.

2.1. Requirements for the theoretical readiness of applicants to perform practical classes:
the acquirer must know:
a) topographic lines and areas of the abdomen
b) the purpose, tasks and methods of superficial and deep palpation of the abdomen
c) properties of palpable organs in normal and pathological conditions
the applicant must be able to:
a) make a deep palpation of the abdomen
b) give a clinical assessment of the data obtained during palpation of the abdomen
List of didactic units:

- Recognize subjective signs of symptoms of diseases of the digestive organs (Chapter of Normal Physiology, Chapter of Pathophysiology).
- Use topographic landmarks for physical examination - examination, palpation, percussion of the abdomen, know the topography and anatomy of the abdominal organs (normal anatomy department, top anatomy department).
- To know the mechanisms of pain syndrome, dyspeptic phenomena (pathophysiology department, path anatomy department).

Apply Latin terminology (Latin language section).

2.2. Questions to check basic knowledge on the topic of the lesson:
question:

1. Complaints of patients with stomach diseases.
2. What are the mechanisms of pain in stomach diseases?
3. Types of pain by the time of its occurrence from the moment of eating, seasonality in the nature of pain.
4. Types of vomiting according to the mechanism of its occurrence.
5. Signs of gastric bleeding.
6. What is heartburn? Causes and mechanism of heartburn.
7. Types of appetite disorders.
8. Complaints of patients with intestinal diseases.
9. What is diarrhea? Causes and mechanisms of diarrhea.
10. What is intestinal dyspepsia?
11. What is constipation? Causes and mechanisms of constipation.
12. What are the signs of upper intestinal bleeding?
13. What are the signs of bleeding from the lower intestines?
14. What should you pay attention to during an examination of the oral cavity?
15. What topographic areas is the stomach divided into?
16. What changes can be detected during an examination of the abdomen?
17. What is the purpose of superficial indicative palpation of the abdomen?
18. Methodology and technique of superficial abdominal palpation.
19. Methodology and technique of deep abdominal palpation.

A problem of the STEP-2 type. Patient M., 30 years old, complains of acid belching, heartburn, pain in the epigastric region, which occur on an empty stomach, 1.5-2 hours after eating, at night, constipation, weakness. He has been sick for about 5 years. Deterioration of well-being is noted after spicy food, in spring and autumn. Objectively: the tongue is coated with a white coating. Abdomen on palpation is soft, painful in the epigastric region.
What disease can be assumed in the patient?
A - gastric ulcer
+B - duodenal ulcer
C - gastritis
D - gastroduodenitis
E - cholecystitis

3. Formation of professional skills and abilities:
mastering the skills of deep palpation of the abdomen),
formation of the ability to give a clinical assessment of the data obtained during palpation of the abdomen; carry out a clinical interpretation of the main symptoms and syndromes in diseases of the digestive organs.

3.1. Control materials for the final stage of the lesson:
Situational tasks:
Task No. 1
During the analysis of the gastric contents of the 35-year-old patient K., the following was found: the amount of gastric contents in the first phase was 150 mol., the output hour of free NSI was 220 mg., after the administration of histamine, the "hourly voltage" was 200 mol., the output hour of free NSI was 350 mg.
What additional methods should be performed to clarify the diagnosis?
+A - X-ray of the stomach
In - ultrasound of the abdominal cavity
S - FGDS
D - X-ray of the lungs
E - ECG

Task No. 2
The patient, a 50-year-old woman, complains of constant pain in the epigastric region, nausea, weakness. He has been sick for 3 years. About: Abdomen is soft, painless. In the epigastric region, a dense immobile formation is determined. The liver and spleen are not palpable.
Analysis of gastric contents: free hydrochloric acid is absent, lactic acid is abundant.
What disease can be assumed in the patient?
+A - stomach tumor
In - peptic ulcer of the stomach
C - Ulcerative disease of the DPK
D - Cholecystitis
E - Gastritis

Task No. 3
An x-ray examination of the stomach of patient N., 47 years old, revealed a filling defect from the corner of the stomach to the prepyloric region. The folds of the mucous membrane in this area are broken, peristalsis is absent.
What additional instrumental methods of research need to be carried out to clarify the diagnosis?
A - ultrasound of abdominal organs
In - FGDS
+Z - targeted biopsy of the stomach
D - ECG
E - X-ray of the lungs

Task No. 4
Patient D., 45 years old, came to the clinic with complaints of belching air, sometimes a rotten egg, nausea, heaviness in the epigastric region. About: pain in the epigastric region.
What disease could the doctor think of?
+A - atrophic gastritis
In - peptic ulcer of the stomach
C - duodenal ulcer
D - cholecystitis
E - hepatitis

Task No. 5
Patient G., 42 years old, was brought to the surgical department in serious condition. Complaints of sudden "dagger" pain in the stomach. He has a history of gastric ulcer. About: pulse 120 beats in 1 minute, rhythmic, blood pressure 80/60 mm. mercury Art. The abdomen does not participate in the act of breathing, it is board-like, sharply painful.
What disease should the doctor assume?
+A - gastric ulcer, perforation
In - gatekeeper stenosis
C - malignancy
D - penetration
Well – bleeding

3.2. Requirements for work results, including before registration: substantiation of the syndromic diagnosis based on complaints, medical history and life data, clinical examination.

3.3. Control materials for the final stage of the class: solving two clinical problems on the subject of the class, answering 10 tests (if necessary).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

List of recommended literature
Basic:
1. Propedeutics of internal medicine: textbook / Yu.I. Decyk, O.H. Yavorskyi, E.M. Neiko and others; under the editorship O.H. Yavorsky – 6th ed., corr. and added - K.: VSV "Medicine", 2020. - 552 p. + 12 p. color incl.
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.

Additional:
1. Methodology of examination of a therapeutic patient: teaching. manual / S.M. Andreychyn, N.A. Bilkevich, T.Yu. Chernets. – Ternopil: TDMU, 2016. – 260 p.
2. 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.
3. Yepishyn A.V. Propaedeutics of internal diseases with care of therapeutic patients / AV. Yepishyn// K. - 2015. 768p.
4. Kovalova OM. Propedeutics of internal medicine/ OM. Kovalova, NA Safargalina-Kornilova// K.: Medicine 2010 - 750p.
5.Macleod's Clinical Examination / Ed. G.Douglas, F.Nicol, C.Robertson.– 13th ed.– Elsevier. 2013. – 471 p.
6.Bates' Guide to Physical Examination and History Taking /Ed. Lynn S. Bickley, Peter G. Szilagyi. – Wolters Kluwer, 2017. – 1066 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/ - Odesa National Medical University
9. https://onmedu.edu.ua/kafedra/propedevtiki-vnutrishnih-hvorob-ta-terapii/ - Department of propaedeutics of internal diseases and therapy Odessa National Medical University

Practical lesson No. 16
Subject: Examination of patients with diseases of the liver and biliary tract. Review. Percussion. Palpation of the liver and spleen, determination of their size by the Kurlov method.
Goal: Acquiring knowledge and mastering professional competences during questioning and physical examination of a patient with liver pathology (examination, percussive

determination of the limits and sizes of the liver according to Obraztsov and Kurlov, palpation of the lower edge of the liver, percussive determination of the size of the spleen according to Kurlov, palpation of the spleen).

Basic concepts: Methods of questioning a patient with liver pathology. Main complaints. Abdominal examination. Determining the size and limits of the liver according to the methods of Obraztsov and Kurlov. Reasons for increasing and decreasing the size of the liver. The technique of deep sliding palpation of the liver. Characterization of the normal palpation picture and possible changes of the lower edge of the liver in pathology. The technique of percussive determination of the size of the spleen, the main reasons for its increase. Rules for palpation of the spleen.
Definition of the main syndromes in liver pathology. Syndromes of portal hypertension, liver failure and hepatoileal syndrome with liver damage. The main manifestations of jaundice syndrome and cholestasis syndrome, their laboratory signs.

Liver percussion
Palpation is the main method of physical examination of the liver. However, it is impossible to determine the size of the liver by palpation, so this action must be preceded by its percussion. The purpose of liver percussion is:
determination of liver boundaries (upper and lower);
determining the size of the liver.

Technique of liver percussion according to the method of M. G. Kurlov
The patient lies on his back, the doctor sits on a chair to the right of the patient. First, the upper border is determined along the right mid-clavicular line (l. medioclavicularis dextra). For this purpose, the plesimeter finger is at their disposal parallel to the expected upper limit and they carry out soft percussion from top to bottom along the intercostals until a dull sound appears (point 1).
Normally, the upper limit of the absolute dullness of the liver is at the level of the VI rib. It is marked with the upper edge of the plessimeter finger. Then along the same line (l. medioclavicularis dextra) determine the lower limit of the absolute dullness of the liver. To do this, the finger-plesimeter is placed parallel to the expected border at the level of the navel or below, so that the tympanic sound is determined when a blow is applied. Conducting quiet percussion, gradually moving the finger-plesimeter upwards, reaching the limit of transition of the tympanic sound completely dull. The border of the liver is marked on the lower edge of the plesimeter finger (2nd point).
Normally, the lower border of the liver by l. medioclavicularis dextra passes the lower edge of the costal arch. The upper border of the liver along the front median line is conventionally determined by drawing a perpendicular from the point obtained by percussion of the upper border along the mid-clavicular line to its intersection with the front median line (l. mediana anterior) (3rd point).
After that, the lower border of the liver is determined along the same line (l. mediana anterior). The finger-plesimeter is set parallel to the expected border at the level of the navel and, conducting quiet percussion, is gradually directed upwards until a dull sound appears, marking the lower edge of the finger-plesimeter (4th point).

Normally, the lower edge of the liver along the front midline is located on the border between the upper and middle third of the distance between the xiphoid process and the navel. Then the border of the liver is determined by the left costal arch. To do this, the plesimeter finger is placed perpendicular to the left costal arch, approximately at the level of the IX rib, and, while conducting a soft percussion, is advanced to the sternum. After receiving a change in the percussion sound, put a mark on the outer edge of the finger (fifth point).
Normally, the lower edge of the liver is here at the level of the VII-VIII rib on the l. parasternalis sinistra. The dimensions of the liver according to Kurlov are normal: – the first line (between points 1 and 2) – 9 cm (±1–2 cm); – the second straight line (between the 3rd and 4th points) – 8 cm (±1–2 cm); – oblique (between the 3rd and 5th points) – 7 cm (±1–2 cm).
Palpation of the liver
Purposes of palpation:
- clarification of the lower border of the liver;
- determination of the properties of the liver: consistency, tenderness, shape of the edge, nature of the surface (with an increase in the liver, the presence of pathological formations.

Method of palpation of the lower edge of the liver according to Obraztsov-Strazhesko.
The patient lies horizontally on his back with his head slightly raised on a low pillow, with his arms bent to the point of longing with his arms crossed on his chest. The doctor sits on a chair to the right, facing the patient
Palpation of the lower edge of the liver consists of four moments:
1. Position of the doctor's hands The doctor places the palm and the remaining four fingers of the left hand on the lumbar region and partially on the last two ribs, with the thumb of the left hand, compresses the costal arch from the front, compressing the right lumbar region with the left hand exposes the back abdominal wall forward, compressing the rib edge with the thumb prevents expansion of the chest during inhalation. bent so that the tips of the fingers lie on the same line) on the stomach below the costal arch along the right mid-clavicular line perpendicular to the edge of the liver (costal arch);
2. Displacement of the skin. During inhalation, the doctor's fingers move the skin slightly downward (in the direction of the navel).
3. Penetration. During exhalation, the patient's fingers gradually (not roughly) penetrate deep into the right hypochondrium;
4. Probing. Without letting go of the hand that is in the abdominal cavity, the patient is asked to take a deep breath, during which the lower edge of the liver falls down, approaches the doctor's fingers and falls into an artificial pocket formed by pressing the abdominal wall with the fingers of the right hand. Then, during the contraction of the diaphragm, it slips out of the pocket, goes around the fingers and slips down under them. The researcher's hand remains motionless throughout, and the procedure is repeated several times.
When sliding the edge of the liver under the fingertips, it is possible to determine its localization, shape (sharp or rounded), consistency, nature (smooth or bumpy), sensitivity to pressure.
Liver properties are normal
The liver is not palpable or is palpable on l. axillaris anterior dextra and l. medioclavicularis dextra along the edge of the costal arch, along l. mediana anterior at 1/3 of the distance

between the xiphoid process and the navel; elastic, with a smooth surface, even sharp or slightly rounded edge, painless.
Properties of the liver in pathological conditions
Size: • increased: inflammatory diseases of the parenchyma (acute and chronic hepatitis), stasis of venous blood in the liver (due to impaired blood circulation), stasis of bile (stone or cancer of the hepatic or common bile duct), hypertrophic cirrhosis of the liver, and (In the initial stage), liver tumors, parasitic lesions, syphilis, blood diseases, peptic ulcer disease, croup pneumonia, gout, diabetes, etc.;
• reduced: acute liver dystrophy (severe variant of Botkin's disease), atrophic cirrhosis, as well as in the case of turning the liver upside down; • uneven increase: tumors, echinococcosis.
Surface: • smooth: acute and chronic hepatitis, diseases of the intrahepatic bile ducts (cholangiohepatitis), congestion;
• hilly: liver cancer, its metastatic lesion, echinococcosis.
Edge of the liver: • acute: cirrhosis of the liver, its parasitic lesions (echinococcus), its fatty degeneration;
• rounded: hepatitis, cirrhosis, congestion;
• uneven liver cancer, cirrhosis.
Consistency (density): elastic: hepatitis, congestion; • moderately mild: septic processes, purulent angiocholiths, an attack of gallstone disease, the development of fatty infiltration at the beginning of liver dystrophy;
• hard (liver cirrhosis), "woody" or "stony" (cancer).
Pain: • painless: normal, cirrhosis, cancer (initial stage), amyloidosis, fatty degeneration;
• painful (hepatitis, end-stage liver cancer, liver congestion);
sharply painful: the appearance of rapid stretching of the capsule (cardiac decompensation), the transition of the inflammatory process to the serous coating of the liver (perihepatitis). In the presence of ascites, pronounced flatulence, when the liver is pushed up, it is advisable to palpate the edge in the vertical position of the patient.

Percussion of the spleen
Percussion technique. Percussion can only be an approximate method of determining the size of the spleen. Because the spleen adjoins the upper pole of the stomach and intestines, which produce a tympanic sound and resonance on percussion, soft percussion should be used. Percussion is performed with the patient on his right side.
1. The plesymeter finger is placed near the edge of the left costal arch perpendicular to the X rib. Percussion is performed from the edge of the costal arch directly along the X rib. At the place where the muffled sound is detected, a mark (the first point) is placed on the edge of the finger facing the tympanic sound.
2. The plesymeter finger is placed perpendicular to the X rib along the back axillary line. Percussion is performed in the direction of the first point. At the place where the dulling of the percussive sound is detected, a mark is made on the edge of the finger facing the clear sound (second point). The segment connecting the first and second points characterizes the length of the spleen, which is normally 6–8 cm.
3. To determine the width of the spleen, its length is divided into 2 parts and percussion is performed from a certain point perpendicular to the X rib. First, they move up from a dull

sound to a clear one (third point). Then they move down, as well as from a dull to a clear sound (fourth point) or from a clear percussive sound to the middle of the spleen. By connecting the third and fourth points, a segment is obtained that characterizes the width of the spleen, which is normally equal to 4-6 cm.
In cases where the spleen is enlarged so much that it protrudes beyond the edge of the costal arch, the method described above is not used, because at the first moment of percussion on the X rib, a dull sound will be obtained. In this case, percussion should be started over the abdominal cavity from the navel towards the costal arch to the place where the X rib is attached to it.

Palpation of the spleen Palpation of the spleen is the main method of examination of this organ, while percussion is almost never used.
The purpose of palpation of the spleen: determination of the lower edge, localization, consistency, shape, tenderness, surface character. Method of palpation of the spleen according to Obraztsov-Strazhesko. The principle of the method is the same as when palpating the liver, that is, to obtain tactile sensations with palpating fingers, the movement of the spleen is used together with the respiratory movements of the diaphragm through the fingers, which stand still or make slight movements towards the organ. It is better to palpate the spleen in the position of the patient on the right side and with hands placed under the head. The doctor sits on a chair to the right of the patient, facing him.
1. Position of the doctor's hands. The researcher places the four fingers of the left hand flat on the left half of the patient's chest, the thumb on the rib cage to fix the chest. This method achieves limitation of chest excursion during breathing and a compensatory increase in respiratory movements of the left diaphragm, to which the spleen belongs.
2. The palm of the right hand with slightly bent fingers is placed flat in the left hypochondrium, perpendicular to the costal arch (edge of the spleen). The fingertips should be in the corner between the X and XI ribs. During inhalation of the patient, the doctor's fingers move the skin slightly downward.
3. During exhalation of the patient, the fingertips penetrate deep into the left hypochondrium, making a pocket from the abdominal wall.
4. The patient is asked to take a deep breath, keeping the fingers still, during which the spleen descends to meet the palpating fingers and touches them.
Thus, the lower edge of the spleen is palpated. At the same time, it is necessary to make a reliable representation of its localization, edges (smooth, jagged), consistency (dense, soft), nature of the surface (smooth, bumpy), mobility (mobile, immobile) and sensitivity (painful, painless).
Normal: the spleen is not accessible for palpation (its lower pole is 3-4 cm higher than the costal arch). If it was possible to feel the edge of the spleen near the edge of the costal arch, it is considered that it has been enlarged by approximately 1.5 times.
With pathology, the spleen becomes dense, its edge often retains a rounded shape (portal hypertension syndrome) or becomes acute (cirrhosis of the liver). If the spleen is significantly enlarged, a physiological cut can be felt along the front edge. This feature distinguishes the spleen from the left kidney. The surface of the spleen is usually smooth. Pain is characteristic in the case of acute blood stagnation in the spleen. Standards of answers

The generally accepted classification of jaundice is the classification of A.F. Blugera: Jaundices are distinguished:
I. Suprahepatic. II. Parenchymatous. III. Subhepatic
I. Suprahepatic jaundice is caused by increased breakdown of erythrocytes or their immature precursors. The consequence of this is an increase in the formation of bilirubin, which the liver is completely unable to remove. The main cause of suprahepatic jaundice is hereditary and acquired hemolytic anemia. In addition, it can be with significant hematomas. In hemolytic anemias (suprahepatic type of jaundice), the skin color is lemon-yellow, the color intensity increases during crises. In the anamnesis, there may be indications of the presence of similar diseases in relatives, the appearance of jaundice for the first time in childhood, its intensification during exposure to the cold. When examining patients, splenomegaly and sometimes hepatomegaly are observed. In the blood: anemia, reticulocytosis, a decrease in the osmotic resistance of erythrocytes, an increase in the content of indirect bilirubin not linked to glucuronic acid. The level of bile acids is not increased; bilirubin is absent in the urine. Mesobilinogen (urobilinogen) will appear in the urine, which will be oxidized to mesobilin (urobilin) and will be considered as a result of hepatocyte overload with indirect bilirubin. Hepatocytes "do not have enough" to capture mesobilinogen (urobilinogen) and process the latter into di- and tripyrroles. Urine will have a dark color. The stool is dark, the reaction to stercobilin is sharply positive. From other studies, a positive Coombs reaction is often found, heat and cold antibodies are detected in the blood serum, and an increased level of serum iron. At the same time, the type of hemolysis is taken into account - intra- and extravascular.
II.1. Parenchymal (intrahepatic) jaundice of the I type, benign, enzyme-pathic, are: – Gilbert's syndrome, which develops as a result of a genetically determined decrease in uredin-diphosphate-glucoronyltransferase (inherited in an autosomal dominant type) or as a result of a defect in the process of binding bilirubin to legantins (transport proteins x, y and z) and transporting it from the space of Disse into the hepatocyte. The amount of indirect bilirubin in the blood increases slightly without signs of hemolysis - up to 70 μmol/l, rarely up to 140 μmol/l. Boys are mostly sick. Jaundice periodically occurs during physical exertion, during the course of infectious and other serious diseases, hypothermia, and starvation. There is no liver failure. The forecast is good.
II.2. Parenchymatous cytolytic jaundice occurs as a result of organic damage to hepatocytes. The contents of hepatocytes enter the blood. Increased direct indirect bilirubin in the blood. The activity of protoplasmic enzymes increases (ALT, AST, glutamate dehydrogenase, fructose diphosphate aldolase, arginase, ketosomonophosphate aldolase). There may be bound bilirubin in the urine, little stercobilin in the feces. Cytolytic jaundice is one of the most frequent syndromes of acute and chronic liver damage. Occurs in acute and chronic hepatitis, infectious mononucleosis, leptospirosis, alcoholic liver damage, cirrhosis, hepatocellular cancer, toxic medicinal hepatitis (isoniazil, indomethacin, paracetamol and other 8 nonsteroidal anti-inflammatory drugs), shock liver, acute heart failure, chronic heart failure, etc.
II.3. Parenchymatous cytolytic-cholestatic jaundice - severe jaundice with high hyperbilirubinemia and conjugated and unconjugated bilirubin and phenomena of intrahepatic cholestasis. Occur with severe course of viral hepatitis B, C, delta, F, etc., acute alcoholic hepatitis, especially against the background of cirrhosis of the liver, chronic active

viral hepatitis, chronic autoimmune hepatitis and drug-induced hepatitis (Aimalin, tubazid, aminazine, rifampicin, β-mercantopurine , cimetidine, piperazine, etc.), primary biliary cirrhosis of the liver. Increased activity of cytolytic and membrane-dependent enzymes.
II.4. Cholestatic jaundice occurs in acute and chronic viral hepatitis, acute medicinal hepatitis (testosterone, anabolic steroids, contraceptives, etc.), in pregnant women in the last trimester of pregnancy, in alcoholic hepatitis. If the cause is not clarified, then it is idiopathic benign recurrent cholestasis. With cholestatic jaundice, conjugated bilirubin in the blood predominates. The clinic resembles mechanical jaundice, but there are no mechanical obstacles. Complicated transfer of bilirubin from the hepatocyte to the bile duct, both independently and as part of a micelle. Thanks to the latter - itchy skin. The number of membrane-dependent enzymes increases in the blood. Dark-colored urine indicates the release of conjugated bilirubin. There is not much stercobilin in feces.
III Subhepatic jaundice (mechanical) develops when the flow of bile from the bile ducts to the duodenum is obstructed. Etiological factors: obstruction by calculi, tumors, parasites, enlarged lymph nodes, postoperative narrowing of the common bile duct, biliary atresia, etc. Increased bilirubin in the blood is mainly due to bound, acholia feces. There will be neither stercobilin nor mesobilin (urobilin) in the urine, bilirubin may be bound. Along with the analysis of the clinical course of diseases, great attention is paid to the results of ultrasound, biochemical indicators of bilirubin metabolism: cytolytic and cholestatic syndromes, data from FGDS, laparoscopy, biopsy, contrast and isotopic research methods. Differential diagnosis should be carried out primarily between types and variants of jaundice, taking into account the etiological factor and variants of the course of the disease in each specific patient. The doctor's tactics depend on the variant (type) of jaundice, the degree of its damage, etiology, the presence of complications and main (combined) diseases, the functional state of the liver and the severity of hepatocellular insufficiency. The main methods of treatment can be both conservative and surgical. The approach is dictated by the etiological factor of jaundice, the degree of compensation of impaired functions, the general condition of the patient, etc.
Clinic and symptoms. The clinical picture of obstructive jaundice is based on the symptoms of impaired bile outflow. - a pain syndrome characteristic of obstructive jaundice due to choledocholithiasis. A clinic of hepatic colic is observed - intense, paroxysmal pain in the right hypochondrium, with radiation to the right shoulder, scapula, supraclavicular fossa. However, the pain syndrome can often be absent when the obturation has arisen due to a choledochal stricture or pancreatic head cancer; - jaundice of the skin, sclera and visible mucous membranes. The rate of its growth and intensity depend on the degree of obstruction of the biliary tract, i.e. on the extent to which the passage of bile into the duodenum is preserved or absent. In the case of obturation with calculi, jaundice occurs on the second day after an attack of hepatic colic, in the case of "valve stone" choledochal jaundice it has an intermittent nature, in the case of cancer of the head of the pancreas it increases gradually (within a week) and is very persistent, in the case of acute pancreatitis it increases gradually and is slightly pronounced and disappears with effective treatment of pancreatitis; - itching of the skin - occurs as a result of the toxic effect of bile acids.
With tumor genesis of obturation, itching often precedes the appearance of jaundice, and with obturation with calculi, it accompanies jaundice; - darkening of urine and discoloration of feces. This symptom occurs as a result of disorders of the biochemical exchange of bilirubin.

Urine with obstructive jaundice has a dark brown color and is excessively foamy ("beer-colored urine"). Feces are acholic, white or slightly grayish (“white clay”); - increase in body temperature, fever. This feature indicates concomitant 10 cholangitis. As a rule, the body temperature during the day is normal or subfebrile, but in the afternoon, patients have an episodic sharp increase to 39-40C (hectic temperature). If the body temperature is high (38-39C) during the day, this may be a sign of metastasis of a tumor of the head of the pancreas, choledoch, etc.; - Courvoisier's syndrome is usually found in thin patients, in whom an enlarged, elastic, smooth and painless gall bladder can be palpated against the background of yellowness of the skin and sclera. This syndrome is often a sign of pancreatic head cancer. . Hepatic coma - acute dystrophy of the liver, portosystemic encephalopathy - a disturbance of consciousness, which is associated with a deep suppression of liver function.

- I degree - yellowness of the skin and sclera, emotional and mental imbalance, feeling of discomfort, fatigue, memory loss, sleep disturbance, headache, tachycardia (up to 100 beats per minute), oliguria (700-800 ml per day), hyperbilirubinemia (200 μmol/l), dysproteinemia, moderate activity of cytolytic enzymes in the blood.
- II degree - manifestations of toxic encephalopathy appear: retardation, slowed reaction to stimuli, tremors of the hands, eyelids, sharp weakening of memory and sleep disturbances. The skin and sclera are icteric, severe skin itching is observed, appetite worsens, nausea and vomiting occur, the heart rate increases to 100-120 beats per minute, blood pressure decreases, the volume of daily urine is mixed to 500-600 ml, the amount of total bilirubin in the blood 200-350 μmol/l, hyperglycemia, blood urea rises to 10-20 mmol/l. The activity of AsT, AlT, LF increases, which indicates damage to the intracellular bioenergetic systems of hepatocytes - mitochondria.
- III degree - symptoms of severe cerebral dysfunction prevail: confused consciousness, adynamia, periodic psychomotor excitement, euphoria or depression, dysarthria. In addition, pronounced jaundice, severe general condition, sometimes fever, muscle pain, tachycardia above 120 beats per minute, hypotension, vomiting, oliguria (300-400 ml per day), hyperbilirubinemia (350 μmol/ l and above), hyperglycemia, blood urea rises by 15 to 20 mmol/l and above, creatinine – above 300 μMol/l. The activity of cytolytic enzymes increases sharply. The level of total bilirubin in the blood does not always correspond to the severity of acute liver dysfunction.

An important factor in its development is the influence of natural factors in the anamnesis: transferred infectious hepatitis, chronic liver diseases, long-term medication, harmful working conditions (exposure to toxic compounds), chronic poisoning, alcohol abuse, etc.

Cholangitis

Acute cholangitis is a frequent companion of obstructive jaundice. This inflammation of the bile ducts can be both a consequence and a cause of cholestasis. Course: - - - acute; chronic; obliterating-sclerotic. According to the nature of morphological changes: - - - - catarrhal; fibrinous; fibrinous-ulcerative; purulent. According to the clinical course; - jaundiced form - with predominant signs of jaundice and pronounced intoxication. Prolonged jaundice with this form of cholangitis quickly leads to acute liver and kidney dysfunction and death of the patient; - septic form - against the background of obstructive jaundice, an inflammatory process occurs in the extrahepatic and intrahepatic passages. The clinical signs of this form of cholangitis are a violent onset with an increase in body temperature up to 40C, a hectic nature of temperature (decline and rise in temperature accompanied by sweating), severe pain

in the right hypochondrium, yellowness of the skin and sclera, pain and muscle tension are observed when palpating the abdomen in the right subcostal area, positive symptoms of Ortner, Murphy, Mussi, signs of purulent inflammation in the general blood test; 16 - pancreatic form - against the background of clinical signs of acute cholecystitis and cholangitis, signs of acute pancreatitis are added. Pronounced vomiting occurs, which does not bring relief, jaundice, hectic body temperature, collapse, drop in blood pressure, point hemorrhages on the front abdominal wall, high activity of diastasis of blood and urine. When cholangitis and obstructive jaundice are combined, the complex of medical measures includes not only unloading of the biliary tract and drainage of the choledochal, but also powerful antibacterial and detoxification therapy.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:

1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with a patient with liver pathology in order to collect complaints and history, general and local examination of the abdomen, percussive determination of the limits and sizes of the liver according to Obraztsov and Kurlov, palpation of the lower edge of the liver, percussive determination of the size of the spleen according to Kurlov, palpation of the spleen, assessment of examination data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for the theoretical readiness of applicants to perform practical classes:
the applicant must know the methods and scheme of examination of a patient with liver diseases,
have an idea of the role of domestic scientists in the development of these methods; to know the general method of questioning patients with liver diseases;
know the characteristics and mechanisms of the main complaints (pain, dyspeptic complaints); the meaning and purpose of the general and local examination of a patient with liver pathology;
to know the purpose, tasks and methods of percussive determination of liver boundaries and dimensions according to Obraztsov and Kurlov, palpation of the lower edge of the liver, percussive determination of spleen dimensions according to Kurlov, palpation of the spleen
know the definition of jaundice; the mechanism of bilirubin formation, the role of the liver in the formation of bilirubin; the main types of jaundice and mechanisms of their development;
to know the characteristics and mechanisms of the main complaints of patients with various types of jaundice; general examination data for various types of jaundice; results of laboratory tests of blood, feces and urine in various types of jaundice
to know the mechanism of development of the syndrome of functional insufficiency of liver cells, the importance of subjective and objective research in its detection;
to know the concept and mechanism of development of portal hypertension syndrome

List of didactic units:
- collect complaints and medical history of a patient with liver pathology in detail;
- conduct a physical examination of the patient (general examination, abdominal examination, percussive determination of liver boundaries and sizes according to Obraztsov and Kurlov, palpation of the lower edge of the liver, percussive determination of the size of the spleen according to Kurlov, palpation of the spleen).
- detect and evaluate the changes that have been found.

2.2. Questions to check basic knowledge on the topic of the lesson:
1. What is jaundice?
2. What is the mechanism of bilirubin formation?
3. The main types of jaundice.
4. What is the mechanism of development of hemolytic jaundice?
5. What is the mechanism of development of parenchymal jaundice?
6. What is the mechanism of development of mechanical jaundice?
7. What complaints can patients with various types of jaundice present?
8. What are the general examination data for different types of jaundice?
9. What are the results of blood, urine, and stool tests for different types of jaundice?
10. Describe the syndrome of functional insufficiency of liver cells.
11. What is the mechanism of portal hypertension?
12. Name the clinical manifestations of portal hypertension.
13. Describe the clinic of hepatic coma.

Situational problems.

Task 1. Patient T., 47 years old, notes yellow staining of the sclera and skin, discolored stools and the appearance of beer-colored urine after an attack of severe pain in the right hypochondrium. General: jaundice of the sclera and skin. Palpation of the liver is painful, its edge is blunt, it protrudes from under the costal arch by 2 cm. An enlarged gall bladder is palpated. Urinalysis: bilirubin +++, no urobilin. There is no stercobilin in feces. What disease does the patient have? What type of jaundice?

Problem 2. Patient R., 41 years old, came with complaints of a feeling of weight, sometimes pain in the right hypochondrium, bitterness in the mouth, fatigue, weight loss. For 10 years, he abused alcohol and ate poorly.

About: reduced nutrition, "hepatic" palms. The abdomen is enlarged, the navel is bulging. On the front abdominal wall there are dilated subcutaneous veins diverging radially from the navel. The liver protrudes 2 cm from under the edge of the costal arch, is soft, with a smooth surface, sensitive to palpation. The spleen is not enlarged. Which syndrome is characterized by these changes? What disease does the patient have?

Problem 3. Patient V., 44 years old, was brought to the clinic in a serious condition: consciousness is dull, reflexes are reduced, clonic convulsions. Kussmaul's breath. A sweet liver smell is noted from the mouth. On the skin, petechial rash, sclera and skin jaundice. The liver is not enlarged. What syndrome is there?

Answers:
1. Probably gallstone disease. Mechanical (subhepatic) jaundice.
2. For portal hypertension. Portal cirrhosis of the liver.
3. Liver failure.

3. Formation of professional skills and abilities:
mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the conducted interrogation with the assumption of the localization of the pathological process)
formation of the ability to perform a clinical examination of a patient with liver pathology (the candidate must be able to perform a general and local examination of a patient with liver disease; carry out percussive determination of the limits and sizes of the liver according to Obraztsov and Kurlov, palpation of the lower edge of the liver, percussive determination of the size of the spleen according to Kurlov, palpation of the spleen),
formation of the ability to give a clinical assessment of the data obtained during the clinical examination of a patient with liver pathology; carry out a clinical interpretation of the main symptoms and syndromes in liver diseases (syndrome of functional insufficiency of liver cells, portal hypertension, hepatolienal syndrome).

3.1. Control materials for the final stage of the lesson:
Situational tasks:
Tasks of the STEP-2 type
1. Patient K., 35 years old, complains of poor appetite, nausea, stool disorders, weakness, aching pain in the right hypochondrium, low-grade fever, yellow color of the skin and sclera. Urine is the color of beer, and the stool is discolored. About: an increase in the liver by 4 cm. It is moderately dense, painful. In the urine, bilirubin +++, urobilin +++. In the blood, direct bilirubin is 60 mmol/l, indirect bilirubin is 42 mmol/l.
What type of jaundice does the patient have?
+A - parenchymatous
B - mechanical
C - hemolytic
D is false
2. Patient P., 56 years old, complains of weakness, low-grade fever, nausea, lack of appetite, abdominal distension, constant pain in the right hypochondrium. 12 years ago, he suffered from Botkin's disease, after which dull pains remained in the right hypochondrium. After 7 years, an increase in the liver was detected. About: jaundice, the liver protrudes 5 cm from under the costal margin, dense, painful. On the skin, individual vascular stars, single small hemorrhages.
What disease could the doctor think of?
+A - cirrhosis of the liver
B - gastric ulcer
C - pancreatitis
D - Cholecystitis
E - Gastritis

3. Patient G., 42 years old, complains of weakness, aching pain in the right hypochondrium, nausea, arthralgia, low-grade fever. The disease developed within a month after the flu. 4 years ago, he suffered viral hepatitis C. General: light yellow color of the sclera, single vascular stars. The liver protrudes from under the costal margin by 4 cm, is moderately dense and painful.
What disease should the doctor assume?
A - cirrhosis of the liver
B - gastric ulcer
C - pancreatitis
D - Cholecystitis
+E - chronic active hepatitis
4. Patient S., 50 years old, came in with complaints of a feeling of heaviness, sometimes pain in the right hypochondrium, bitterness in the mouth, unstable stool, weakness, quick fatigue. For 10 years, he ate irregularly and drank alcohol. Ob-but: "hepatic palms", the abdomen is enlarged in volume, on the skin the expansion of veins is visible. The liver protrudes 2 cm from under the costal arch, is soft, with a smooth surface, and is sensitive to palpation. The size of the spleen according to Kurlov - 12x18 cm.
What disease can be assumed in the patient?
+A- cirrhosis of the liver
B - gastric ulcer
C - pancreatitis
D - Cholecystitis
E - Gastritis
5. Patient S., 50 years old, came in with complaints of a feeling of heaviness, sometimes pain in the right hypochondrium, bitterness in the mouth, unstable stool, weakness, quick fatigue. For 10 years, he ate irregularly and drank alcohol. Ob-but: "hepatic palms", the abdomen is enlarged in volume, on the skin the expansion of veins is visible. The liver protrudes 2 cm from under the costal arch, is soft, with a smooth surface, and is sensitive to palpation. The size of the spleen according to Kurlov - 12x18 cm.
For which clinical syndrome are these changes characteristic?
A - cholestasis syndrome
+B - portal hypertension syndrome
C - cytolysis syndrome
D - jaundice
E - hepatorenal syndrome
6. The patient was delivered in serious condition. Consciousness is confused. Skin and mucous membranes are jaundiced. Kussmaul's breath. "Hepatic" smell from the mouth. The liver is not enlarged. What syndrome is there?
A. cholestasis syndrome
B - portal hypertension syndrome
C - cytolysis syndrome
+D - hepatocellular failure syndrome
E - hepatorenal syndrome
7. The patient was delivered in serious condition. Consciousness is confused. Skin and mucous membranes are jaundiced. Exhausted. Kussmaul's breath. "Hepatic" smell from the

mouth. The abdomen is enlarged, dilated veins are visible on the skin. The liver is not enlarged. Your diagnosis?
+A- cirrhosis of the liver
B - gastric ulcer
C - pancreatitis
D - Cholecystitis
E - Gastritis
8. A 60-year-old patient complains of constant intense pain in the right hypochondrium, weight loss, loss of appetite. The skin is icteric with a greenish tint. The lower edge of the liver protrudes to the level of the navel, stony density, large lump. In the urine - bilirubin ++++, they did not have it. What kind of organ can be damaged?
A- intestines
B - stomach
C - pancreas
+D - liver
E - bud
9. A 60-year-old patient complains of constant intense pain in the right hypochondrium, weight loss, loss of appetite. The skin is icteric with a greenish tint. The lower edge of the liver protrudes to the level of the navel, stony density, large nodule. In the urine - bilirubin ++++, they did not have it. What type of jaundice can you think of?
A - parenchymatous
+B - mechanical
C - hemolytic
D is false
10. A 60-year-old patient complains of constant intense pain in the right hypochondrium, weight loss, loss of appetite. The skin is icteric with a greenish tint. The lower edge of the liver protrudes to the level of the navel, stony density, large lump. In the urine - bilirubin ++++, they did not have it. What disease can you think of?
A - cirrhosis of the liver
B - gastric ulcer
C - pancreatitis
+D - liver cancer
E - gastritis

3.2. Requirements for work results, including before registration: substantiation of the syndromic diagnosis based on complaints, medical history and life data, clinical examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (*if necessary*).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 17
Subject: Differential diagnosis of jaundice.
Goal: Acquiring knowledge and mastering professional competences during questioning and physical examination of a patient with pathology of the liver and biliary tract. Differential diagnosis of jaundice.

Basic concepts:

The generally accepted classification of jaundice is the classification of A.F. Bluger: Jaundice is distinguished:
I. Suprahepatic. II. Parenchymatous. III. Subhepatic
I. Suprahepatic jaundice is caused by increased breakdown of erythrocytes or their immature precursors. The consequence of this is an increase in the formation of bilirubin, which the liver is completely unable to remove. The main cause of suprahepatic jaundice is hereditary and acquired hemolytic anemia. In addition, it can be with significant hematomas. In hemolytic anemias (suprahepatic type of jaundice), the skin color is lemon-yellow, the color intensity increases during crises. In the anamnesis, there may be indications of the presence of similar diseases in relatives, the appearance of jaundice for the first time in childhood, its intensification during exposure to the cold. When examining patients, splenomegaly and sometimes hepatomegaly are observed. In the blood: anemia, reticulocytosis, a decrease in the osmotic resistance of erythrocytes, an increase in the content of indirect bilirubin not linked to glucuronic acid. The level of bile acids is not increased; bilirubin is absent in the urine. Mesobilinogen (urobilinogen) will appear in the urine, which will be oxidized to mesobilin (urobilin) and will be considered as a result of hepatocyte overload with indirect bilirubin. Hepatocytes "do not have enough" to capture mesobilinogen (urobilinogen) and process the latter into di- and tripyrroles. Urine will have a dark color. The stool is dark, the reaction to stercobilin is sharply positive. From other studies, a positive Coombs reaction is often found, heat and cold antibodies are detected in the blood serum, and an increased level of serum iron. At the same time, the type of hemolysis is taken into account - intra- and extravascular.
II.1. Parenchymal (intrahepatic) jaundice of the I type, benign, enzyme-pathic, are: – Gilbert's syndrome, which develops as a result of a genetically determined decrease in uredin-diphosphate-glucoronyltransferase (inherited in an autosomal dominant type) or as a result of a defect in the process of binding bilirubin to legantins (transport proteins x, y and z) and transporting it from the space of Disse into the hepatocyte. The amount of indirect bilirubin in the blood increases slightly without signs of hemolysis - up to 70 μmol/l, rarely up to 140 μmol/l. Boys are mostly sick. Jaundice periodically occurs during physical exertion, during the course of infectious and other serious diseases, hypothermia, and starvation. There is no liver failure. The forecast is good.
II.2. Parenchymatous cytolytic jaundice occurs as a result of organic damage to hepatocytes. The contents of hepatocytes enter the blood. Increased direct indirect bilirubin in the blood.

The activity of protoplasmic enzymes increases (ALT, AST, glutamate dehydrogenase, fructose diphosphate aldolase, arginase, ketosomonophosphate aldolase). There may be bound bilirubin in the urine, little stercobilin in the feces. Cytolytic jaundice is one of the most frequent syndromes of acute and chronic liver damage. Occurs in acute and chronic hepatitis, infectious mononucleosis, leptospirosis, alcoholic liver damage, cirrhosis, hepatocellular cancer, toxic medicinal hepatitis (isoniazil, indomethacin, paracetamol and other 8 nonsteroidal anti-inflammatory drugs), shock liver, acute heart failure, chronic heart failure, etc.

II.3. Parenchymatous cytolytic-cholestatic jaundice - severe jaundice with high hyperbilirubinemia and conjugated and unconjugated bilirubin and phenomena of intrahepatic cholestasis. Occur with severe course of viral hepatitis B, C, delta, F, etc., acute alcoholic hepatitis, especially against the background of cirrhosis of the liver, chronic active viral hepatitis, chronic autoimmune hepatitis and drug-induced hepatitis (Aimalin, tubazid, aminazine, rifampicin, β-mercantopurine , cimetidine, piperazine, etc.), primary biliary cirrhosis of the liver. Increased activity of cytolytic and membrane-dependent enzymes.

II.4. Cholestatic jaundice occurs in acute and chronic viral hepatitis, acute medicinal hepatitis (testosterone, anabolic steroids, contraceptives, etc.), in pregnant women in the last trimester of pregnancy, in alcoholic hepatitis. If the cause is not clarified, then it is idiopathic benign recurrent cholestasis. With cholestatic jaundice, conjugated bilirubin in the blood predominates. The clinic resembles mechanical jaundice, but there are no mechanical obstacles. Complicated transfer of bilirubin from the hepatocyte to the bile duct, both independently and as part of a micelle. Thanks to the latter - itchy skin. The number of membrane-dependent enzymes increases in the blood. Dark-colored urine indicates the release of conjugated bilirubin. There is not much stercobilin in feces.

III Subhepatic jaundice (mechanical) develops when the flow of bile from the bile ducts to the duodenum is obstructed. Etiological factors: obstruction by calculi, tumors, parasites, enlarged lymph nodes, postoperative narrowing of the common bile duct, biliary atresia, etc. Increased bilirubin in the blood is mainly due to bound, acholia feces. There will be neither stercobilin nor mesobilin (urobilin) in the urine, bilirubin may be bound. Along with the analysis of the clinical course of diseases, great attention is paid to the results of ultrasound, biochemical indicators of bilirubin metabolism: cytolytic and cholestatic syndromes, data from FGDS, laparoscopy, biopsy, contrast and isotopic research methods. Differential diagnosis should be carried out primarily between types and variants of jaundice, taking into account the etiological factor and variants of the course of the disease in each specific patient. The doctor's tactics depend on the variant (type) of jaundice, the degree of its damage, etiology, the presence of complications and main (combined) diseases, the functional state of the liver and the severity of hepatocellular insufficiency. The main methods of treatment can be both conservative and surgical. The approach is dictated by the etiological factor of jaundice, the degree of compensation of impaired functions, the general condition of the patient, etc.

Clinic and symptoms. The clinical picture of obstructive jaundice is based on the symptoms of impaired bile outflow. - a pain syndrome characteristic of obstructive jaundice due to choledocholithiasis. A clinic of hepatic colic is observed - intense, paroxysmal pain in the right hypochondrium, with radiation to the right shoulder, scapula, supraclavicular fossa. However, the pain syndrome can often be absent when the obturation has arisen due to a

choledochal stricture or pancreatic head cancer; - jaundice of the skin, sclera and visible mucous membranes. The rate of its growth and intensity depend on the degree of obstruction of the biliary tract, i.e. on the extent to which the passage of bile into the duodenum is preserved or absent. In the case of obturation with calculi, jaundice occurs on the second day after an attack of hepatic colic, in the case of "valve stone" choledochal jaundice it has an intermittent nature, in the case of cancer of the head of the pancreas it increases gradually (within a week) and is very persistent, in the case of acute pancreatitis it increases gradually and is slightly pronounced and disappears with effective treatment of pancreatitis; - itching of the skin - occurs as a result of the toxic effect of bile acids.
With tumor genesis of obturation, itching often precedes the appearance of jaundice, and with obturation with calculi, it accompanies jaundice; - darkening of urine and discoloration of feces. This symptom occurs as a result of disorders of the biochemical exchange of bilirubin. Urine with obstructive jaundice has a dark brown color and is excessively foamy ("beer-colored urine"). Feces are acholic, white or slightly grayish (“white clay”); - increase in body temperature, fever. This feature indicates concomitant 10 cholangitis. As a rule, the body temperature during the day is normal or subfebrile, but in the afternoon, patients have an episodic sharp increase to 39-40C (hectic temperature). If the body temperature is high (38-39C) during the day, this may be a sign of metastasis of a tumor of the head of the pancreas, choledoch, etc.; - Courvoisier's syndrome is usually found in thin patients, in whom an enlarged, elastic, smooth and painless gall bladder can be palpated against the background of yellowness of the skin and sclera. This syndrome is often a sign of pancreatic head cancer.
. Hepatic coma - acute dystrophy of the liver, portosystemic encephalopathy - a disturbance of consciousness, which is associated with a deep suppression of liver function.
- I degree - yellowness of the skin and sclera, emotional and mental imbalance, feeling of discomfort, fatigue, memory loss, sleep disturbance, headache, tachycardia (up to 100 beats per minute), oliguria (700-800 ml per day), hyperbilirubinemia (200 μmol/l), dysproteinemia, moderate activity of cytolytic enzymes in the blood.
- II degree - manifestations of toxic encephalopathy appear: retardation, slowed reaction to stimuli, tremors of the hands, eyelids, sharp weakening of memory and sleep disturbances. The skin and sclera are icteric, severe skin itching is observed, appetite worsens, nausea and vomiting occur, the heart rate increases to 100-120 beats per minute, blood pressure decreases, the volume of daily urine is mixed to 500-600 ml, the amount of total bilirubin in the blood 200-350 μmol/l, hyperglycemia, blood urea rises to 10-20 mmol/l. The activity of AsT, AlT, LF increases, which indicates damage to the intracellular bioenergetic systems of hepatocytes - mitochondria.
- III degree - symptoms of severe cerebral dysfunction prevail: confused consciousness, adynamia, periodic psychomotor excitement, euphoria or depression, dysarthria. In addition, pronounced jaundice, severe general condition, sometimes fever, muscle pain, tachycardia above 120 beats per minute, hypotension, vomiting, oliguria (300-400 ml per day), hyperbilirubinemia (350 μmol/ l and above), hyperglycemia, blood urea rises by 15 to 20 mmol/l and above, creatinine – above 300 μMol/l. The activity of cytolytic enzymes increases sharply. The level of total bilirubin in the blood does not always correspond to the severity of acute liver dysfunction.
An important factor in its development is the influence of natural factors in the anamnesis: transferred infectious hepatitis, chronic liver diseases, long-term medication, harmful

working conditions (exposure to toxic compounds), chronic poisoning, alcohol abuse, etc.
Cholangitis
Acute cholangitis is a frequent companion of obstructive jaundice. This inflammation of the bile ducts can be both a consequence and a cause of cholestasis. Course: - - - acute; chronic; obliterating-sclerotic. According to the nature of morphological changes: - - - - catarrhal; fibrinous; fibrinous-ulcerative; purulent. According to the clinical course; - jaundiced form - with predominant signs of jaundice and pronounced intoxication. Prolonged jaundice with this form of cholangitis quickly leads to acute liver and kidney dysfunction and death of the patient; - septic form - against the background of obstructive jaundice, an inflammatory process occurs in the extrahepatic and intrahepatic passages. The clinical signs of this form of cholangitis are a violent onset with an increase in body temperature up to 40C, a hectic nature of temperature (decline and rise in temperature accompanied by sweating), severe pain in the right hypochondrium, yellowness of the skin and sclera, pain and muscle tension are observed when palpating the abdomen in the right subcostal area, positive symptoms of Ortner, Murphy, Mussi, signs of purulent inflammation in the general blood test; 16 - pancreatic form - against the background of clinical signs of acute cholecystitis and cholangitis, signs of acute pancreatitis are added. Pronounced vomiting occurs, which does not bring relief, jaundice, hectic body temperature, collapse, drop in blood pressure, point hemorrhages on the front abdominal wall, high activity of diastasis of blood and urine. When cholangitis and obstructive jaundice are combined, the complex of medical measures includes not only unloading of the biliary tract and drainage of the choledochal, but also powerful antibacterial and detoxification therapy.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:
1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with a patient with liver pathology in order to collect complaints and history, general and local examination of the abdomen, percussive determination of the limits and sizes of the liver according to Obraztsov and Kurlov, palpation of the lower edge of the liver, percussive determination of the size of the spleen according to Kurlov, palpation of the spleen, assessment of examination data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.
2.1. Requirements for the theoretical readiness of applicants to perform practical classes:
- the applicant must know the methods and scheme of examination of a patient with liver diseases,
- have an idea of the role of domestic scientists in the development of these methods; to know the general method of questioning patients with liver diseases;

- know the characteristics and mechanisms of the main complaints (pain, dyspeptic complaints); the meaning and purpose of the general and local examination of a patient with liver pathology;
- to know the purpose, tasks and methods of percussive determination of liver boundaries and dimensions according to Obraztsov and Kurlov, palpation of the lower edge of the liver, percussive determination of spleen dimensions according to Kurlov, palpation of the spleen
- know the definition of jaundice; the mechanism of bilirubin formation, the role of the liver in the formation of bilirubin; the main types of jaundice and mechanisms of their development;
- to know the characteristics and mechanisms of the main complaints of patients with various types of jaundice; general examination data for various types of jaundice; results of laboratory tests of blood, feces and urine in various types of jaundice
- to know the mechanism of development of the syndrome of functional insufficiency of liver cells, the importance of subjective and objective research in its detection;
- to know the concept and mechanism of development of portal hypertension syndrome

List of didactic units:
- collect complaints and medical history of a patient with liver pathology in detail;
- conduct a physical examination of the patient (general examination, abdominal examination, percussive determination of liver boundaries and sizes according to Obraztsov and Kurlov, palpation of the lower edge of the liver, percussive determination of the size of the spleen according to Kurlov, palpation of the spleen).
- detect and evaluate the changes that have been found.

2.2. Questions to check basic knowledge on the topic of the lesson:
1. What is jaundice?
2. What is the mechanism of bilirubin formation?
3. The main types of jaundice.
4. What is the mechanism of development of hemolytic jaundice?
5. What is the mechanism of development of parenchymal jaundice?
6. What is the mechanism of development of mechanical jaundice?
7. What complaints can patients with various types of jaundice present?
8. What are the general examination data for different types of jaundice?
9. What are the results of blood, urine, and stool tests for different types of jaundice?
10. Describe the syndrome of functional insufficiency of liver cells.
11. What is the mechanism of portal hypertension?
12. Name the clinical manifestations of portal hypertension.
13. Describe the clinic of hepatic coma.

Situational problems.

Task 1. Patient T., 47 years old, notes yellow staining of the sclera and skin, discolored stools and the appearance of beer-colored urine after an attack of severe pain in the right hypochondrium. General: jaundice of the sclera and skin. Palpation of the liver is painful, its edge is blunt, it protrudes from under the costal arch by 2 cm. An enlarged gall bladder is

palpated. Urinalysis: bilirubin +++, no urobilin. There is no stercobilin in feces. What disease does the patient have? What type of jaundice?

Problem 2. Patient R., 41 years old, came with complaints of a feeling of weight, sometimes pain in the right hypochondrium, bitterness in the mouth, fatigue, weight loss. For 10 years, he abused alcohol and ate poorly.
About: reduced nutrition, "hepatic" palms. The abdomen is enlarged, the navel is bulging. On the front abdominal wall there are dilated subcutaneous veins diverging radially from the navel. The liver protrudes 2 cm from under the edge of the costal arch, is soft, with a smooth surface, sensitive to palpation. The spleen is not enlarged. Which syndrome is characterized by these changes? What disease does the patient have?

Problem 3. Patient V., 44 years old, was brought to the clinic in a serious condition: consciousness is dull, reflexes are reduced, clonic convulsions. Kussmaul's breath. A sweet liver smell is noted from the mouth. On the skin, petechial rash, sclera and skin jaundice. The liver is not enlarged. What syndrome is there?

Answers:
1. Probably gallstone disease. Mechanical (subhepatic) jaundice.
2. For portal hypertension. Portal cirrhosis of the liver.
3. Liver failure.

3. Formation of professional skills and abilities:
mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the conducted interrogation with the assumption of the localization of the pathological process)
formation of the ability to perform a clinical examination of a patient with liver pathology (the candidate must be able to perform a general and local examination of a patient with liver disease; carry out percussive determination of the limits and sizes of the liver according to Obraztsov and Kurlov, palpation of the lower edge of the liver, percussive determination of the size of the spleen according to Kurlov, palpation of the spleen),
formation of the ability to give a clinical assessment of the data obtained during the clinical examination of a patient with liver pathology; carry out a clinical interpretation of the main symptoms and syndromes in liver diseases (syndrome of functional insufficiency of liver cells, portal hypertension, hepatolienal syndrome).

3.1. Control materials for the final stage of the lesson:
Situational tasks:
Tasks of the STEP-2 type
1. Patient K., 35 years old, complains of poor appetite, nausea, stool disorders, weakness, aching pain in the right hypochondrium, low-grade fever, yellow color of the skin and sclera. Urine is the color of beer, and the stool is discolored. About: an increase in the liver by 4 cm. It is moderately dense, painful. In the urine, bilirubin +++, urobilin +++. In the blood, direct bilirubin is 60 mmol/l, indirect bilirubin is 42 mmol/l.

What type of jaundice does the patient have?
+A - parenchymatous
B - mechanical
C - hemolytic
D is false
2. Patient P., 56 years old, complains of weakness, low-grade fever, nausea, lack of appetite, abdominal distension, constant pain in the right hypochondrium. 12 years ago, he suffered from Botkin's disease, after which dull pains remained in the right hypochondrium. After 7 years, an increase in the liver was detected. About: jaundice, the liver protrudes 5 cm from under the costal margin, dense, painful. On the skin, individual vascular stars, single small hemorrhages.
What disease could the doctor think of?
+A - cirrhosis of the liver
B - gastric ulcer
C - pancreatitis
D - Cholecystitis
E - Gastritis
3. Patient G., 42 years old, complains of weakness, aching pain in the right hypochondrium, nausea, arthralgia, low-grade fever. The disease developed within a month after the flu. 4 years ago, he suffered viral hepatitis C. General: light yellow color of the sclera, single vascular stars. The liver protrudes from under the costal margin by 4 cm, is moderately dense and painful.
What disease should the doctor assume?
A - cirrhosis of the liver
B - gastric ulcer
C - pancreatitis
D - Cholecystitis
+E - chronic active hepatitis
4. Patient S., 50 years old, came in with complaints of a feeling of heaviness, sometimes pain in the right hypochondrium, bitterness in the mouth, unstable stool, weakness, quick fatigue. For 10 years, he ate irregularly and drank alcohol. Ob-but: "hepatic palms", the abdomen is enlarged in volume, on the skin the expansion of veins is visible. The liver protrudes 2 cm from under the costal arch, is soft, with a smooth surface, and is sensitive to palpation. The size of the spleen according to Kurlov - 12x18 cm.
What disease can be assumed in the patient?
+A- cirrhosis of the liver
B - gastric ulcer
C - pancreatitis
D - Cholecystitis
E - Gastritis
5. Patient S., 50 years old, came in with complaints of a feeling of heaviness, sometimes pain in the right hypochondrium, bitterness in the mouth, unstable stool, weakness, quick fatigue. For 10 years, he ate irregularly and drank alcohol. Ob-but: "hepatic palms", the abdomen is enlarged in volume, on the skin the expansion of veins is visible. The liver protrudes 2 cm

from under the costal arch, is soft, with a smooth surface, and is sensitive to palpation. The size of the spleen according to Kurlov - 12x18 cm.
For which clinical syndrome are these changes characteristic?
A - cholestasis syndrome
+B - portal hypertension syndrome
C - cytolysis syndrome
D - jaundice
E - hepatorenal syndrome
6. The patient was delivered in serious condition. Consciousness is confused. Skin and mucous membranes are jaundiced. Kussmaul's breath. "Hepatic" smell from the mouth. The liver is not enlarged. What syndrome is there?
A. cholestasis syndrome
B - portal hypertension syndrome
C - cytolysis syndrome
+D - hepatocellular failure syndrome
E - hepatorenal syndrome
7. The patient was delivered in serious condition. Consciousness is confused. Skin and mucous membranes are jaundiced. Exhausted. Kussmaul's breath. "Hepatic" smell from the mouth. The abdomen is enlarged, dilated veins are visible on the skin. The liver is not enlarged. Your diagnosis?
+A- cirrhosis of the liver
B - gastric ulcer
C - pancreatitis
D - Cholecystitis
E - Gastritis
8. A 60-year-old patient complains of constant intense pain in the right hypochondrium, weight loss, loss of appetite. The skin is icteric with a greenish tint. The lower edge of the liver protrudes to the level of the navel, stony density, large lump. In the urine - bilirubin ++++, they did not have it. What kind of organ can be damaged?
A- intestines
B - stomach
C - pancreas
+D - liver
E - bud
9. A 60-year-old patient complains of constant intense pain in the right hypochondrium, weight loss, loss of appetite. The skin is icteric with a greenish tint. The lower edge of the liver protrudes to the level of the navel, stony density, large nodule. In the urine - bilirubin ++++, they did not have it. What type of jaundice can you think of?
A - parenchymatous
+B - mechanical
C - hemolytic
D is false
10. A 60-year-old patient complains of constant intense pain in the right hypochondrium, weight loss, loss of appetite. The skin is icteric with a greenish tint. The lower edge of the

liver protrudes to the level of the navel, stony density, large lump. In the urine - bilirubin ++++, they did not have it. What disease can you think of?
A - cirrhosis of the liver
B - gastric ulcer
C - pancreatitis
+D - liver cancer
E - gastritis

3.2. Requirements for work results, including before registration: substantiation of the syndromic diagnosis based on complaints, medical history and life data, clinical examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (*if necessary*).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.
List of recommended literature
Basic:
1. Propedeutics of internal medicine: textbook / Yu.I. Decyk, O.H. Yavorskyi, E.M. Neiko and others; under the editorship O.H. Yavorsky – 6th ed., corr. and added - K.: VSV "Medicine", 2020. - 552 p. + 12 p. color incl.
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.

Additional:
1. Methodology of examination of a therapeutic patient: teaching. manual / S.M. Andreychyn, N.A. Bilkevich, T.Yu. Chernets. – Ternopil: TDMU, 2016. – 260 p.
2. 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.
3. Yepishyn A.V. Propaedeutics of internal diseases with care of therapeutic patients / AV. Yepishyn// K. - 2015. 768p.
4. Kovalova OM. Propedeutics of internal medicine/ OM. Kovalova, NA Safargalina-Kornilova// K.: Medicine 2010 - 750p.
5.Macleod's Clinical Examination / Ed. G.Douglas, F.Nicol, C.Robertson.– 13th ed.– Elsevier. 2013. – 471 p.
6.Bates' Guide to Physical Examination and History Taking /Ed. Lynn S. Bickley, Peter G. Szilagyi. – Wolters Kluwer, 2017. – 1066 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/ - Odesa National Medical University
9. https://onmedu.edu.ua/kafedra/propedevtiki-vnutrishnih-hvorob-ta-terapii/ - Department of propaedeutics of internal diseases and therapyOdessa National Medical University

Practical lesson No. 18.
Subject: Syndrome of functional insufficiency of liver cells. Portal hypertension. Hepatolienal syndrome.

Goal: Acquiring knowledge and mastering professional competences during questioning and physical examination of a patient with liver pathology (examination, percussive determination of the limits and sizes of the liver according to Obraztsov and Kurlov, palpation of the lower edge of the liver, percussive determination of the size of the spleen according to Kurlov, palpation of the spleen).

Basic concepts: Methods of questioning a patient with liver pathology. Main complaints. Abdominal examination. Determining the size and limits of the liver according to the methods of Obraztsov and Kurlov. Reasons for increasing and decreasing the size of the liver. The technique of deep sliding palpation of the liver. Characterization of the normal palpation picture and possible changes of the lower edge of the liver in pathology. The technique of percussive determination of the size of the spleen, the main reasons for its increase. Rules for palpation of the spleen.
Definition of the main syndromes in liver pathology. Syndromes of portal hypertension, liver failure and hepatoileal syndrome with liver damage.

Liver percussion
Palpation is the main method of physical examination of the liver. However, it is impossible to determine the size of the liver by palpation, so this action must be preceded by its percussion. The purpose of liver percussion is:
- determination of liver boundaries (upper and lower);
- determining the size of the liver.

Technique of liver percussion according to the method of M. G. Kurlov
The patient lies on his back, the doctor sits on a chair to the right of the patient. First, the upper border is determined along the right mid-clavicular line (l. medioclavicularis dextra). For this purpose, the plesimeter finger is at their disposal parallel to the expected upper limit and they carry out soft percussion from top to bottom along the intercostals until a dull sound

appears (point 1).
Normally, the upper limit of the absolute dullness of the liver is at the level of the VI rib. It is marked with the upper edge of the plessimeter finger. Then along the same line (l. medioclavicularis dextra) determine the lower limit of the absolute dullness of the liver. To do this, the finger-plesimeter is placed parallel to the expected border at the level of the navel or below, so that the tympanic sound is determined when a blow is applied. Conducting quiet percussion, gradually moving the finger-plesimeter upwards, reaching the limit of transition of the tympanic sound completely dull. The border of the liver is marked on the lower edge of the plesimeter finger (2nd point).
Normally, the lower border of the liver by l. medioclavicularis dextra passes the lower edge of the costal arch. The upper border of the liver along the front median line is conventionally determined by drawing a perpendicular from the point obtained by percussion of the upper border along the mid-clavicular line to its intersection with the front median line (l. mediana anterior) (3rd point).
After that, the lower border of the liver is determined along the same line (l. mediana anterior). The finger-plesimeter is set parallel to the expected border at the level of the navel and, conducting quiet percussion, is gradually directed upwards until a dull sound appears, marking the lower edge of the finger-plesimeter (4th point).
Normally, the lower edge of the liver along the front midline is located on the border between the upper and middle third of the distance between the xiphoid process and the navel. Then the border of the liver is determined by the left costal arch. To do this, the plesimeter finger is placed perpendicular to the left costal arch, approximately at the level of the IX rib, and, while conducting a soft percussion, is advanced to the sternum. After receiving a change in the percussion sound, put a mark on the outer edge of the finger (fifth point).
Normally, the lower edge of the liver is here at the level of the VII-VIII rib on the l. parasternalis sinistra. The dimensions of the liver according to Kurlov are normal: – the first line (between points 1 and 2) – 9 cm (±1–2 cm); – the second straight line (between the 3rd and 4th points) – 8 cm (±1–2 cm); – oblique (between the 3rd and 5th points) – 7 cm (±1–2 cm).
Palpation of the liver
Purposes of palpation:
- clarification of the lower border of the liver;
- determination of the properties of the liver: consistency, tenderness, shape of the edge, nature of the surface (with an increase in the liver, the presence of pathological formations.

Method of palpation of the lower edge of the liver according to Obraztsov-Strazhesko.
The patient lies horizontally on his back with his head slightly raised on a low pillow, with his arms bent to the point of longing with his arms crossed on his chest. The doctor sits on a chair to the right, facing the patient
Palpation of the lower edge of the liver consists of four moments:
1. Position of the doctor's hands The doctor places the palm and the remaining four fingers of the left hand on the lumbar region and partially on the last two ribs, with the thumb of the left hand, compresses the costal arch from the front, compressing the right lumbar region with the left hand exposes the back abdominal wall forward, compressing the rib edge with the thumb prevents expansion of the chest during inhalation. bent so that the tips of the fingers lie on the

same line) on the stomach below the costal arch along the right mid-clavicular line perpendicular to the edge of the liver (costal arch);
2. Displacement of the skin. During inhalation, the doctor's fingers move the skin slightly downward (in the direction of the navel).
3. Penetration. During exhalation, the patient's fingers gradually (not roughly) penetrate deep into the right hypochondrium;
4. Probing. Without letting go of the hand that is in the abdominal cavity, the patient is asked to take a deep breath, during which the lower edge of the liver falls down, approaches the doctor's fingers and falls into an artificial pocket formed by pressing the abdominal wall with the fingers of the right hand. Then, during the contraction of the diaphragm, it slips out of the pocket, goes around the fingers and slips down under them. The researcher's hand remains motionless throughout, and the procedure is repeated several times.
When sliding the edge of the liver under the fingertips, it is possible to determine its localization, shape (sharp or rounded), consistency, nature (smooth or bumpy), sensitivity to pressure.
Liver properties are normal
The liver is not palpable or is palpable on l. axillaris anterior dextra and l. medioclavicularis dextra along the edge of the costal arch, along l. mediana anterior at 1/3 of the distance between the xiphoid process and the navel; elastic, with a smooth surface, even sharp or slightly rounded edge, painless.
Properties of the liver in pathological conditions
Size: • increased: inflammatory diseases of the parenchyma (acute and chronic hepatitis), stasis of venous blood in the liver (due to impaired blood circulation), stasis of bile (stone or cancer of the hepatic or common bile duct), hypertrophic cirrhosis of the liver, and (In the initial stage), liver tumors, parasitic lesions, syphilis, blood diseases, peptic ulcer disease, croup pneumonia, gout, diabetes, etc.;
• reduced: acute liver dystrophy (severe variant of Botkin's disease), atrophic cirrhosis, as well as in the case of turning the liver upside down; • uneven increase: tumors, echinococcosis.
Surface:
• smooth: acute and chronic hepatitis, diseases of the intrahepatic bile ducts (cholangiohepatitis), congestion;
• hilly: liver cancer, its metastatic lesion, echinococcosis.
Edge of the liver: • acute: cirrhosis of the liver, its parasitic lesions (echinococcus), its fatty degeneration;
• rounded: hepatitis, cirrhosis, congestion;
• uneven liver cancer, cirrhosis.
Consistency (density): elastic: hepatitis, congestion; • moderately mild: septic processes, purulent angiocholiths, an attack of gallstone disease, the development of fatty infiltration at the beginning of liver dystrophy;
• hard (liver cirrhosis), "woody" or "stony" (cancer).
Pain: • painless: normal, cirrhosis, cancer (initial stage), amyloidosis, fatty degeneration;
• painful (hepatitis, end-stage liver cancer, liver congestion);
sharply painful: the appearance of rapid stretching of the capsule (cardiac decompensation), the transition of the inflammatory process to the serous coating of the liver (perihepatitis). In

the presence of ascites, pronounced flatulence, when the liver is pushed up, it is advisable to palpate the edge in the vertical position of the patient.

Percussion of the spleen
Percussion technique. Percussion can only be an approximate method of determining the size of the spleen. Because the spleen adjoins the upper pole of the stomach and intestines, which produce a tympanic sound and resonance on percussion, soft percussion should be used. Percussion is performed with the patient on his right side.
1. The plesymeter finger is placed near the edge of the left costal arch perpendicular to the X rib. Percussion is performed from the edge of the costal arch directly along the X rib. At the place where the muffled sound is detected, a mark (the first point) is placed on the edge of the finger facing the tympanic sound.
2. The plesymeter finger is placed perpendicular to the X rib along the back axillary line. Percussion is performed in the direction of the first point. At the place where the dulling of the percussive sound is detected, a mark is made on the edge of the finger facing the clear sound (second point). The segment connecting the first and second points characterizes the length of the spleen, which is normally 6–8 cm.
3. To determine the width of the spleen, its length is divided into 2 parts and percussion is performed from a certain point perpendicular to the X rib. First, they move up from a dull sound to a clear one (third point). Then they move down, as well as from a dull to a clear sound (fourth point) or from a clear percussive sound to the middle of the spleen. By connecting the third and fourth points, a segment is obtained that characterizes the width of the spleen, which is normally equal to 4-6 cm.
In cases where the spleen is enlarged so much that it protrudes beyond the edge of the costal arch, the method described above is not used, because at the first moment of percussion on the X rib, a dull sound will be obtained. In this case, percussion should be started over the abdominal cavity from the navel towards the costal arch to the place where the X rib is attached to it.

Palpation of the spleen. Palpation of the spleen is the main method of examining this organ, while percussion is almost never used.
The purpose of palpation of the spleen: determination of the lower edge, localization, consistency, shape, tenderness, surface character. Method of palpation of the spleen according to Obraztsov-Strazhesko. The principle of the method is the same as when palpating the liver, that is, to obtain tactile sensations with palpating fingers, the movement of the spleen is used together with the respiratory movements of the diaphragm through the fingers, which stand still or make slight movements towards the organ. It is better to palpate the spleen in the position of the patient on the right side and with hands placed under the head. The doctor sits on a chair to the right of the patient, facing him.
1. Position of the doctor's hands. The researcher places the four fingers of the left hand flat on the left half of the patient's chest, the thumb on the rib cage to fix the chest. This method achieves limitation of chest excursion during breathing and a compensatory increase in respiratory movements of the left diaphragm, to which the spleen belongs.
2. The palm of the right hand with slightly bent fingers is placed flat in the left hypochondrium, perpendicular to the costal arch (edge of the spleen). The fingertips should

be in the corner between the X and XI ribs. During inhalation of the patient, the doctor's fingers move the skin slightly downward.
3. During exhalation of the patient, the fingertips penetrate deep into the left hypochondrium, making a pocket from the abdominal wall.
4. The patient is asked to take a deep breath, keeping the fingers still, during which the spleen descends to meet the palpating fingers and touches them.
Thus, the lower edge of the spleen is palpated. At the same time, it is necessary to make a reliable representation of its localization, edges (smooth, jagged), consistency (dense, soft), nature of the surface (smooth, bumpy), mobility (mobile, immobile) and sensitivity (painful, painless).
Normal: the spleen is not accessible for palpation (its lower pole is 3-4 cm higher than the costal arch). If it was possible to feel the edge of the spleen near the edge of the costal arch, it is considered that it has been enlarged by approximately 1.5 times.
With pathology, the spleen becomes dense, its edge often retains a rounded shape (portal hypertension syndrome) or becomes acute (cirrhosis of the liver). If the spleen is significantly enlarged, a physiological cut can be felt along the front edge. This feature distinguishes the spleen from the left kidney. The surface of the spleen is usually smooth. Pain is characteristic in the case of acute blood stagnation in the spleen. Standards of answers

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:
1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with a patient with liver pathology in order to collect complaints and history, general and local examination of the abdomen, percussive determination of the limits and sizes of the liver according to Obraztsov and Kurlov, palpation of the lower edge of the liver, percussive determination of the size of the spleen according to Kurlov, palpation of the spleen, assessment of examination data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.
2.1. Requirements for the theoretical readiness of applicants to perform practical classes:
- the applicant must know the methods and scheme of examination of a patient with liver diseases,
- have an idea of the role of domestic scientists in the development of these methods; to know the general method of questioning patients with liver diseases;
- know the characteristics and mechanisms of the main complaints (pain, dyspeptic complaints); the meaning and purpose of the general and local examination of a patient with liver pathology;
- to know the purpose, tasks and methods of percussive determination of liver boundaries and dimensions according to Obraztsov and Kurlov, palpation of the lower edge

of the liver, percussive determination of spleen dimensions according to Kurlov, palpation of the spleen
- know the definition of jaundice; the mechanism of bilirubin formation, the role of the liver in the formation of bilirubin; the main types of jaundice and mechanisms of their development;
- to know the characteristics and mechanisms of the main complaints of patients with various types of jaundice; general examination data for various types of jaundice; results of laboratory tests of blood, feces and urine in various types of jaundice
- to know the mechanism of development of the syndrome of functional insufficiency of liver cells, the importance of subjective and objective research in its detection;
- to know the concept and mechanism of development of portal hypertension syndrome

List of didactic units:
- collect complaints and medical history of a patient with liver pathology in detail;
- conduct a physical examination of the patient (general examination, abdominal examination, percussive determination of liver boundaries and sizes according to Obraztsov and Kurlov, palpation of the lower edge of the liver, percussive determination of the size of the spleen according to Kurlov, palpation of the spleen).
- detect and evaluate the changes that have been found.

2.2. Questions to check basic knowledge on the topic of the lesson:
1. What is jaundice?
2. What is the mechanism of bilirubin formation?
3. The main types of jaundice.
4. What is the mechanism of development of hemolytic jaundice?
5. What is the mechanism of development of parenchymal jaundice?
6. What is the mechanism of development of mechanical jaundice?
7. What complaints can patients with various types of jaundice present?
8. What are the general examination data for different types of jaundice?
9. What are the results of blood, urine, and stool tests for different types of jaundice?
10. Describe the syndrome of functional insufficiency of liver cells.
11. What is the mechanism of portal hypertension?
12. Name the clinical manifestations of portal hypertension.
13. Describe the clinic of hepatic coma.

Situational problems.

Task 1. Patient T., 47 years old, notes yellow staining of the sclera and skin, discolored stools and the appearance of beer-colored urine after an attack of severe pain in the right hypochondrium. General: jaundice of the sclera and skin. Palpation of the liver is painful, its edge is blunt, it protrudes from under the costal arch by 2 cm. An enlarged gall bladder is palpated. Urinalysis: bilirubin +++, no urobilin. There is no stercobilin in feces. What disease does the patient have? What type of jaundice?

Problem 2. Patient R., 41 years old, came with complaints of a feeling of weight, sometimes pain in the right hypochondrium, bitterness in the mouth, fatigue, weight loss. For 10 years, he abused alcohol and ate poorly.

About: reduced nutrition, "hepatic" palms. The abdomen is enlarged, the navel is bulging. On the front abdominal wall there are dilated subcutaneous veins diverging radially from the navel. The liver protrudes 2 cm from under the edge of the costal arch, is soft, with a smooth surface, sensitive to palpation. The spleen is not enlarged. Which syndrome is characterized by these changes? What disease does the patient have?

Problem 3. Patient V., 44 years old, was brought to the clinic in a serious condition: consciousness is dull, reflexes are reduced, clonic convulsions. Kussmaul's breath. A sweet liver smell is noted from the mouth. On the skin, petechial rash, sclera and skin jaundice. The liver is not enlarged. What syndrome is there?

Answers:
1. Probably gallstone disease. Mechanical (subhepatic) jaundice.
2. For portal hypertension. Portal cirrhosis of the liver.
3. Liver failure.

3. Formation of professional skills and abilities:
- mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the conducted interrogation with the assumption of the localization of the pathological process)
- formation of the ability to perform a clinical examination of a patient with liver pathology (the candidate must be able to perform a general and local examination of a patient with liver disease; carry out percussive determination of the limits and sizes of the liver according to Obraztsov and Kurlov, palpation of the lower edge of the liver, percussive determination of the size of the spleen according to Kurlov, palpation of the spleen),
- formation of the ability to give a clinical assessment of the data obtained during the clinical examination of a patient with liver pathology; carry out a clinical interpretation of the main symptoms and syndromes in liver diseases (syndrome of functional insufficiency of liver cells, portal hypertension, hepatolienal syndrome).

3.1. Control materials for the final stage of the lesson:
Situational tasks:
Tasks of the STEP-2 type
1. Patient P., 56 years old, complains of weakness, low-grade fever, nausea, lack of appetite, abdominal distension, constant pain in the right hypochondrium. 12 years ago, he suffered from Botkin's disease, after which dull pains remained in the right hypochondrium. After 7 years, an increase in the liver was detected. About: jaundice, the liver protrudes 5 cm from under the costal margin, dense, painful. On the skin, individual vascular stars, single small hemorrhages.
What disease could the doctor think of?
+A - cirrhosis of the liver
B - gastric ulcer
C - pancreatitis
D - Cholecystitis

E - Gastritis
2. Patient S., 50 years old, came in with complaints of a feeling of weight, sometimes pain in the right hypochondrium, bitterness in the mouth, unstable stool, weakness, quick fatigue. For 10 years, he ate irregularly and drank alcohol. Ob-but: "hepatic palms", the abdomen is enlarged in volume, on the skin the expansion of veins is visible. The liver protrudes 2 cm from under the costal arch, is soft, with a smooth surface, and is sensitive to palpation. The size of the spleen according to Kurlov - 12x18 cm.
For which clinical syndrome are these changes characteristic?
A - cholestasis syndrome
+B - portal hypertension syndrome
C - cytolysis syndrome
D - jaundice
E - hepatorenal syndrome
36. The patient was delivered in serious condition. Consciousness is confused. Skin and mucous membranes are jaundiced. Kussmaul's breath. "Hepatic" smell from the mouth. The liver is not enlarged. What syndrome is there?
A. cholestasis syndrome
B - portal hypertension syndrome
C - cytolysis syndrome
+D - hepatocellular failure syndrome
E - hepatorenal syndrome

3.2. Requirements for work results, including before registration: substantiation of the syndromic diagnosis based on complaints, medical history and life data, clinical examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (*if necessary*).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 19
Subject:**Examination of patients with diseases of the kidneys and urinary tract. Questioning Inspection, percussion, palpation. Laboratory, instrumental, X-ray, morphological examination of kidneys.**.

Goal: Acquiring knowledge and mastering professional competences during questioning, physical examination of a patient with kidney and urinary tract pathology (examination, palpation of kidneys, determination of Pasternacki's symptom), instrumental research.

Basic concepts: The main complaints of patients with kidney pathology. Review methodology. The diagnostic value of determining Pasternacki's symptom. Methods of palpation of the kidneys in standing and lying positions. Diagnostic value of instrumental methods of kidney research (survey radiograph of kidneys, excretory urography, retrograde pyelography, selective renal angiography; radioisotope radiography, kidney scan, kidney biopsy).

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).

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.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:

1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking workbooks, communicating with a patient with kidney pathology in order to collect complaints and anamnesis, general and local examination, palpation of the kidneys, determination of Pasternacki's symptom, evaluation of clinical examination data and instrumental data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for the theoretical readiness of applicants to perform practical classes:
the applicant must know the methods and scheme of examination of a patient with kidney diseases,

have an idea of the role of domestic scientists in the development of these methods; to know the general method of questioning patients with kidney diseases;

to know the characteristics and mechanisms of the main complaints (pain, dysuric complaints); the meaning and purpose of a general and local examination of a patient with kidney pathology;

to know the purpose, tasks and methods of palpation of the kidneys in the lying and standing position

to know the method of determining Pasternacki's symptom and its interpretation

to know the purpose and basic principles of conducting an examination radiograph of the kidneys, excretory urography, retrograde pyelography, selective renal angiography;
know the purpose and basic principles of radioisotope radiography, kidney scanning, kidney biopsy;
to know the main pathological signs from the side of the eye fundus in case of renal hypertension.

List of didactic units:

- collect complaints and medical history of a patient with kidney pathology in detail;
- conduct a physical examination of the patient (general examination, local examination, palpation of the kidneys, percussive determination of Pasternacki's symptom).
- identify and evaluate changes found during clinical examination
- draw up a plan for additional instrumental studies for the diagnosis of diseases of the kidneys and urinary tract
- evaluate the results of instrumental research.

2.2. Questions to check basic knowledge on the topic of the lesson:

1. Peculiarities of the examination of patients with diseases of the kidneys and urinary tract;
2. Kidney palpation technique;
3. Percussion technique - determination of Pasternacki's symptom;
4. Purpose and basic principles of conducting a survey radiograph of the kidneys, excretory urography, retrograde pyelography, selective renal angiography;
5. Purpose and basic principles of radioisotope renography, kidney scanning, kidney biopsy;
6. The main pathological signs from the side of the eye fundus in renal hypertension.

Tests with standard answers:

1. According to the amount of displacement of the kidneys, the degrees of nephroptosis are distinguished:

A) three;
B) four;
B) five.

Bip answer: A.

2. Kidney disease is characterized by the face:

A) deep-set eyes, sharp features, skin sharply pale with a bluish tint, covered with drops of cold sweat;
B) swollen, uniformly swollen, amemic, eye slits are narrowed, hair on the outer halves of the eyebrows is absent, the nose and lips are thickened, the skin is pale;
B) with swollen eyelids and narrowed eye slits, the skin is pale, and possibly dry, with scratches, the smell of ammonia.

Bip answer: V.

3. Formation of professional skills and abilities:

mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the conducted interrogation with the assumption of the localization of the pathological process)

formation of the ability to perform a clinical examination of a patient with kidney pathology (the candidate must be able to perform a general and local examination of a patient with kidney disease; perform percussion determination of Pasternacki's symptom, palpation of the kidneys),

formation of the ability to evaluate the data obtained during the clinical examination of a patient with kidney pathology; carry out a clinical interpretation of the main symptoms and syndromes in kidney diseases

formation of the ability to perform an instrumental examination of a patient with kidney pathology (the applicant must be able to prescribe an instrumental examination plan, give an assessment of the examination results)

3.1. Control materials for the final stage of the lesson:
Situational tasks:

Tasks of the STEP-2 type

1. A 19-year-old patient complains of weakness, dull pain in the lumbar region, headaches, change in the color of urine ("meat wash"). He fell ill 2 weeks after having a sore throat. About: the skin is pale, the face is swollen, the legs are swollen. Blood pressure 160/110 mm Hg. Tones of the heart are clear, accent A2. Daily diuresis 400 ml, specific gravity of urine 1027, protein - 2.5 g/l, in sediment - erythrocytes 80-100 in p/zr, mostly leached, hyaline cylinders - 4-5 in p/zr. Creatinine in blood serum - 142 μmol/l.
What syndromes can be distinguished?
A. Nabryakovy.
B. Hypertensive.
S. Sechovyi.
D. Nephrotic.
+E. Edema, hypertensive, urinary.

2. A 20-year-old patient complains of facial swelling, worse in the morning, headaches, change in the color of urine ("meat slops"). He has been sick for 10 days, after hypothermia. About: paleness of the skin, swelling of the eyelids and legs. Blood pressure 170/100 mm Hg. Tones of the heart are clear, accent A2. Daily diuresis 500 mol, specific gravity of urine - 1025, protein - 1.65 g/l, in urine sediment - erythrocytes 60-80 in p/sp., leached, hyaline cylinders 2-4 in p/sp., blood creatinine - 132 μmol/l. Your diagnosis?
A. Acute pyelonephritis.
B. Amyloidosis of the kidneys.
S. Urinary stone disease.
+D. Acute glomerulonephritis.
E. Chronic glomerulonephritis.

3. A 40-year-old patient complains of severe pain in the lumbar region on the left side, radiating to the left inguinal region, increased t (38℃), cloudy urine. The examination showed pallor of the skin, swelling of the eyelids. Pasternacki's symptom on the left sharply (+). In urine - specific gravity 1020, protein 0.99 g/l, pyuria (leukocytes 100-120 in p/zr), fresh erythrocytes 20-30 in p/zr. What is your diagnosis?
A. Chronic glomerulonephritis.
B. Chronic pyelonephritis.
C. Congestive kidney.
D. Urinary stone disease.
+ E. Urinary stone disease, secondary pyelonephritis.

4. The patient's X-ray examination showed a decrease in the size of one of the kidneys. This can be beneficial:
+A. chronic pyelonephritis
B. acute pyelonephritis
B. the presence of cysts in the kidney tissue
G. amyloidosis of the kidneys
D. acute glomerulonephritis

3. To confirm the diagnosis of kidney amyloidosis, the most informative method is:
A. urine culture
B. radioisotope examination of the kidneys
+V. kidney biopsy
D. ultrasound examination of the kidneys

3.2. Requirements for work results, including before registration: substantiation of the syndromic diagnosis based on complaints, medical history and life data, clinical and instrumental examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (*if necessary*).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

List of recommended literature
Basic:

1. Propedeutics of internal medicine: textbook / Yu.I. Decyk, O.H. Yavorskyi, E.M. Neiko and others; under the editorship O.H. Yavorsky – 6th ed., corr. and added - K.: VSV "Medicine", 2020. - 552 p. + 12 p. color incl.

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.

Additional:

1. Methodology of examination of a therapeutic patient: teaching. manual / S.M. Andreychyn, N.A. Bilkevich, T.Yu. Chernets. – Ternopil: TDMU, 2016. – 260 p.

2. 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.

3. Yepishyn A.V. Propaedeutics of internal diseases with care of therapeutic patients / AV. Yepishyn// K. - 2015. 768p.

4. Kovalova OM. Propedeutics of internal medicine/ OM. Kovalova, NA Safargalina-Kornilova// K.: Medicine 2010 - 750p.

5.Macleod's Clinical Examination / Ed. G.Douglas, F.Nicol, C.Robertson.– 13th ed.– Elsevier. 2013. – 471 p.

6.Bates' Guide to Physical Examination and History Taking /Ed. Lynn S. Bickley, Peter G. Szilagyi. – Wolters Kluwer, 2017. – 1066 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/ - Odesa National Medical University
9. https://onmedu.edu.ua/kafedra/propedevtiki-vnutrishnih-hvorob-ta-terapii/ - Department of propaedeutics of internal diseases and therapyOdessa National Medical University

Practical lesson No. 20

Topic: Urine research. Clinical interpretation of urine analysis. Analysis of data from laboratory research methods: sample of Zimnytskyi, Ambyurzhe, Nechyporenko, residual nitrogen, etc.

Goal: Acquiring knowledge and mastering professional competences during the conduct and clinical interpretation of laboratory tests of urine (general analysis of urine, functional and quantitative samples).

Basic concepts: Methodology of general urine analysis.
Clinical interpretation of general urinalysis indicators, functional (Zymnytskyi, Reberg test) and quantitative tests (Ambyurzhe, Nechyporenko test).

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.

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.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:

1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, interpretation of laboratory urine analysis data*),* conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for the theoretical readiness of applicants to perform practical classes: the applicant must know the methods and scheme of examination of a patient with kidney diseases,

have an idea of the role of domestic scientists in the development of these methods;

know the technique of macroscopic and microscopic examination of urine

know the methods of determining the chemical properties of urine
to know the diagnostic value of changes in indicators that are determined during the study of physical and chemical properties of urine, microscopic examination of urine sediment
to know the diagnostic value of changes in indicators of functional and quantitative kidney tests
to know the principles and diagnostic value of indicators of bacterioscopic and bacteriological examination of urine.

List of didactic units:

- conduct a urine sample for general clinical, functional, quantitative, bacteriological analyses
- determine indicators of general clinical analysis of urine: physical and chemical properties, microscopic examination of urine sediment
- identify and evaluate the changes that were found during the laboratory examination of a patient with kidney pathology

2.2. Questions to check basic knowledge on the topic of the lesson:
1. How the collection is carried out**urine?**Urine collection rules.
2. What is included in the general clinical analysis of urine and in what sequence is it conducted?
3. How big is the daily diuresis and what does it depend on?
4. What is meant by polyuria, oliguria, nocturia and when do they occur?
5. What determines the color of urine in the norm? What color urine can be found in pathology?
6. How is the specific gravity of urine determined?
7. What is the specific gravity of urine in the norm? What does it depend on?
8. In what cases is there a high (low) specific gravity of urine? What function does the specific gravity of urine reflect?
9. What is isosthenuria, hyposthenuria, hypoisosthenuria?
10. Zimnytsky's test (methodology, normal indicators, clinical significance.
11. What are the methods for determining protein in urine? Is it of clinical significance?
12. Under what physiological conditions does protein appear in the urine?
13. What is meant by renal and postrenal proteinuria? In what diseases do they occur?
14. What are the methods of determining sugar in urine? Their clinical significance?
15. The level of sugar in the urine of a healthy person. What is meant by glycosuria?
16. Methods of determination of ketone bodies in urine. Their clinical significance?
17. Methods of determining bile acids in urine. Their clinical significance?
18. What color is urine in the presence of bile pigments? When and what bilirubin can appear in the urine?
19. What is hematuria? What diseases does it occur in?

20. What is pyuria? What diseases does it occur in?
21. What is the essence of Nechiporenko's test?
22. The clinical value of determining "disorganized" urine sediment.
23. What are cylinders? What are their types? Diagnostic value of cylinders?

Tests with standard answers:

1. What is hyposthenuria?
A) Urine with a low specific gravity of urine.
B) Decreased urine output (less than 500 ml per day).
B) Predominant urination at night.
Bip answer: A.

2. Elements of organized urine sediment are:
A) Salt, tears.
B) Blood elements.
B) Protein, glucose in urine.
Bip answer: B.

3. Formation of professional skills and abilities:

mastering communication skills with the patient (when collecting urine for laboratory tests - general urine analysis, functional and quantitative samples)

formation of the ability to conduct a laboratory study of urine - general analysis of urine, functional and quantitative samples

formation of the ability to carry out clinical interpretation of indicators of general analysis of urine, functional and quantitative samples), to identify the main laboratory syndromes.

3.1. Control materials for the final stage of the lesson:
Situational tasks:

Tasks of the STEP-2 type

1. When examining the urine of the patient K., it was found that the urine is "meat slop", the specific gravity is increased, and the protein is 0.33 g/l. The presence of which pathology can be assumed in the patient?
A. Chronic pyelonephritis
B. Chronic glomerulonephritis
+V. Acute glomerulonephritis
M. Diabetes mellitus
D. Urinary stone disease

2. When examining the urine of patient P., it was found that the relative density of urine was 1011, and protein was 0.66 g/l. The presence of which pathology can be assumed in the patient?
+A. Chronic nephritis
B. Chronic glomerulonephritis
B. Acute nephritis
M. Diabetes mellitus
D. Urinary stone disease

3. During examination of urine according to Zimnytskyi, patient K. found: specific gravity of 1 serving - 1012, 2 servings - 1011, 3 servings - 1010, 4 servings - 1012, 6 servings - 1010, 7 servings - 1012, 8 servings - 1013. The presence of which pathological symptom can be assumed in the patient?
A. Hypersthenuria
+B. Hyposthenuria
B. Leukocyturia
M. Uraturia
D. Hematuria

3.2. Requirements for work results, including before registration: substantiation of the conclusion regarding the clinical interpretation of urine laboratory test data, definition of the leading laboratory syndromes.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (*if necessary*).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 21
Topic: Examination of patients with blood diseases. Questioning Review. Palpation. Percussion.

Goal: Acquiring knowledge and mastering professional competences during questioning, physical examination of a patient with pathology of the blood system (examination, percussive determination of the size of the liver and spleen according to Kurlov, palpation of lymph nodes, liver and spleen)

Basic concepts:
The main complaints of patients with blood diseases. Main clinical syndromes. Review data. The main types of bleeding.
Percussion data of the liver and spleen, palpation of lymph nodes, liver, spleen.

Questioning. General manifestations of many diseases of the blood system can be

non-specific complaints of general weakness, fatigue, dizziness, flickering "flies" before the eyes, tinnitus, shortness of breath at rest or during physical exertion, palpitations that gradually progress.
More specific are complaints of bone pain, increased bleeding, increased lymph nodes, increased body temperature, itchy skin

Pain in the bones (osalgia) and, especially, in the sternum (sternalgia) increases when pressing on the bone or tapping on it.
Dull pain or a feeling of heaviness and distension in the left hypochondrium is the result of an increase in the spleen and overstretching of its capsule in leukemia, erythremia, thrombosis of the splenic vein.
A significant increase in the liver can cause a feeling of heaviness and constant pain in the right hypochondrium. Sharp pain is characteristic of perisplenitis, it increases with deep breathing and coughing.
Acute leukemia is characterized by a sore throat as a result of the development of ulcerative-necrotic tonsillitis. Excruciating burning pain in the tips of the fingers due to a violation of microcirculation occurs in erythremia.
Increase in temperature. Subfebrile temperature is often observed in hemolytic and B12-deficient anemias. Pronounced fever with chills occurs with a hemolytic crisis. Lymphogranulomatosis is characterized by a wave-like temperature fluctuation: a gradual increase over 8-15 days, then a gradual decrease.
Increased bleeding. Patients with hemorrhagic diatheses, leukemia and myeloaplastic syndrome may complain of the appearance of hemorrhagic rashes and bruises on the skin and mucous membranes, bleeding from the nose, gums, which occur under the influence of minor injuries (pressure, bruise) or spontaneously. Bleeding from the gastrointestinal tract can be manifested by the darkening of feces or the appearance of fresh blood in it, from the kidneys - hematuria; characteristic metrorrhagia and prolonged menstruation in women, bleeding from the lungs. With hemophilia, hemorrhages in the joints, serous cavities, long-term external bleeding that are difficult to stop often occur.
Loss of appetite and weight loss are characteristic of many diseases of the blood system. They are most pronounced in leukemias, malignant lymphomas (lymphogranulomatosis, lymphosarcoma). With iron-deficiency anemia, especially with the so-called early and late chlorosis, there is a distortion of taste (patients eat chalk, clay, earth, coal), as well as smell (patients enjoy sniffing the vapors of ether, gasoline, other odorous substances with a sharp smell).
Dyspeptic disorders: nausea, dysphagia - observed in anemia. Skin changes. Patients with lymphogranulomatosis, erythremia, and lymphocytic leukemia may experience itching of the skin. With lymphocytic leukemia, it is especially pronounced, often becomes exhausting and can be the first sign of the disease. Anemic patients may complain of dry skin, hair loss and early graying, brittle nails. Jaundice of the skin and mucous membranes often worries patients with hemolytic anemia. With lymphogranulomatosis and chronic lymphocytic leukemia, lymphosarcoma patients often notice local protrusions of the skin, in particular in the neck, inguinal and other areas, which is a consequence of the increase in lymph nodes.
Medical history. It is necessary to ask the patient about his general condition before the appearance of the first signs of the disease, about the possible reasons with which the patient

associates its onset; to establish when certain symptoms of the disease first appeared, to investigate its dynamics. It is necessary to find out about the results of blood tests that were carried out on the patient in the past, to find out what treatment was prescribed earlier and its results.

History of life. When collecting the anamnesis of the patient's life, it should be borne in mind that often the cause of blood diseases, in particular anemia, is acute and chronic industrial intoxication with mercury salts, compounds of lead, phosphorus, arsenic, benzene, contact with chemical dyes, gasoline, bismuth, gold preparations, body irradiation (ionizing radiation, long-term X-ray exposure, contact with radioisotope materials). Long-term uncontrolled use of certain medications, in particular sulfonamides, nonsteroidal anti-inflammatory drugs, butadione, some antibiotics (levomycetin, streptomycin), cytostatics can cause hypoplastic anemia, agranulocytosis, hemorrhagic syndrome, hemolytic anemia. A number of diseases of the blood system are genetically determined, in particular hemophilia, some types of hemolytic anemia. Therefore, it is necessary to ask the patient about the state of health of relatives. Monotonous and substandard nutrition with insufficient vitamins and microelements, improper work and rest regime, insufficient exposure to fresh air often contribute to the development of iron- and B12-deficient anemia. Often, damage to the blood system is a consequence of chronic pathology of internal organs, infectious diseases (tumors, bronchiectasis, pulmonary tuberculosis, etc.). Severe anemia can occur against the background of chronic kidney disease. Chronic liver diseases are complicated by hemorrhagic syndrome: the synthesis of a number of enzymes, blood coagulation factors, in particular prothrombin and fibrinogen, is disrupted

Review. First of all, during the examination, the general condition of the patient and his state of consciousness are determined. A severe condition with loss of consciousness occurs in the terminal stages of blood diseases: leukemia, myeloid aplasia, progressive anemia. Acute posthemorrhagic anemia may be accompanied by signs of hemorrhagic shock. When examining the skin and mucous membranes, their color, trophic state, moisture, turgor, and the presence of rashes are evaluated. Pale skin can be a sign of anemia. More reliable information is provided by the assessment of the color of the visible mucous membranes; most often, the conjunctivae of the eyelids are examined: in anemia they are pale, in erythremia they are red-cyanotic. With different types of anemia, the skin tone has certain features. Thus, with juvenile chlorosis, the skin has an alabaster pallor, sometimes with a greenish tint, with B12-deficiency anemia, the skin is somewhat yellowish, waxy, and with hemolytic anemia, the skin and mucous membranes are yellow. In patients with chronic leukemia, the skin acquires an earthy-gray hue. With erythremia, it is cherry-red, "full-blooded", especially on the face, neck, and hands; visible bluish-red mucous membranes. In patients with hemorrhagic syndrome on the skin and visible mucous membranes (mouth, pharynx), hemorrhages can be detected in the form of spots of various sizes and shapes: from small dots (petechiae) to larger ones (purpura, ecchymoses). Hemorrhages should be distinguished from rashes of inflammatory origin and telangiectasias. The latter disappear for a few seconds when you press them with your finger. Rashes of an inflammatory nature (for example, with hemorrhagic vasculitis) rise above the skin level (papular-petechial type of rashes)

Examination of the oral cavity can reveal some signs of blood diseases. Thus, hemorrhagic diatheses are manifested by hemorrhages in the mucous membrane of the mouth and throat.

B12-deficient anemia is characterized by sharp atrophy of the papillae of the tongue, as a result of which it has a smooth, "varnished" surface ("Gunter's glossitis"). Atrophic glossitis, angular stomatitis (cracks in the corners of the mouth) are observed in iron deficiency anemia. Leukemias and aplastic anemia are characterized by lesions of the mucous membrane of the mouth and pharynx of an ulcer-necrotic nature. When examining the neck and trunk of patients with acute leukemia, regional protrusions can be detected due to an increase in the corresponding groups of cervical, supraclavicular, inguinal, and less often other lymph nodes. Protrusion in the left hypochondrium due to a significant increase in the spleen is found in chronic myeloid leukemia. Some blood diseases are characterized by changes in the bone and joint system. In particular, with hemophilia, there is deformation and limitation of joint mobility (especially knee joints) due to repeated hemorrhages

Palpation. Regional lymph nodes are examined by palpation: cervical, submandibular, supra- and subclavian, axillary, elbow, inguinal and popliteal. Normally, you can feel the submandibular, anterior, posterior cervical, axillary and inguinal lymph nodes in the form of bean-shaped or round formations of an elastic consistency. The size of lymph nodes does not exceed 1.5-2.0 cm. In healthy people, lymph nodes may not be palpable at all. An increase in lymph nodes is found in many blood diseases. In chronic leukemias, lymphogranulomatosis, the lymph nodes are not painful. Lymph nodes in blood diseases are usually mobile, do not fuse with the surrounding tissues, do not suppurate and do not form fistulas, the skin above them is not changed. Lymph nodes of an extremely dense consistency are found in lymphosarcoma. Damage to lymph nodes in hematological pathology has a systemic nature, it begins with some one group, and later spreads to others

Palpation of the spleen is most often carried out with the patient on the right side, the head is slightly tilted forward to the chest, the left arm is bent at the elbow, it lies freely on the front surface of the chest, the right leg is extended, the left is bent at the knee and hip joints (this is how maximum relaxation is achieved abdominal muscles, the spleen moves forward). When palpating the spleen, the following characteristics are evaluated: size, tenderness, density, consistency, shape, mobility; determine the presence of cuts on the front edge. Normally, the spleen is not palpable. It becomes available for palpation, as a rule, only when it is enlarged or lowered (extreme degree of visceroptosis). An increase in the size of the spleen, or splenomegaly (from the Greek splen - spleen, megas - large), is observed in hemolytic anemias, especially hereditary, thrombocytopenic purpura, acute and chronic leukemias, lymphoreticulosis. In most diseases, in particular hemolytic, the spleen is not painful when palpated. Pain appears with a heart attack, perisplenitis, its rapid increase in size due to stretching of the capsule (venous stasis with thrombosis of the splenic vein - often occurs with leukemia). A dense consistency of the spleen is characteristic of blood diseases. The surface of the spleen, as a rule, is smooth, the unevenness of the edge is observed in perisplenitis, old heart attacks (there is retraction), the bumpy surface occurs in the presence of cysts, syphilitic gums, echinococcosis, and damage to the organ by a tumor. The mobility of the spleen in healthy individuals is quite pronounced, but in cases of sharp enlargement and perisplenitis, it is immobile. Diseases of the blood system in most cases are accompanied by an increase in the liver - hepatomegaly. More often, it is a sign of leukemia or hemolytic anemia with intracellular hemolysis, erythremia

Percussion. Liver and spleen sizes are determined by percussion. Percussion of the spleen is performed with the patient standing or lying on the right side. They use the method of quiet

percussion. Normally, the splenic dullness is determined between the IX and XI ribs and is 4-6 cm. Along the length, the splenic dullness is percussed along the X rib. Normally, the size of dullness is 6-8 cm. It is difficult to accurately determine the borders of the spleen percussively, since it is surrounded by hollow organs that give a loud tympanic sound when percussion. The liver is percussed according to the Kurlov method or the border of its lower edge is determined along the right midclavicular line. Bone percussion is also used in the diagnosis of blood diseases. In conditions accompanied by bone marrow hyperplasia (leukemias, megaloblastic anemias, erythremia, hemolytic anemias), tapping and pressing on the flat bones (sternum) is painful.
Auscultation. A number of auscultatory signs of damage to the cardiovascular system are found in anemia. As a result of a decrease in blood viscosity and dystrophic processes in the myocardium, a functional systolic murmur is heard at the apex of the heart and at the V point of auscultation, a "spinal murmur" on the jugular veins, more often on the right. Tachycardia is characteristic, heart tones are weakened. Hypotension is found in most cases. A characteristic sign of erythremia is an increase in blood pressure. In case of perisplenitis over the area where the spleen is located, you can listen to the noise of friction of the peritoneum.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:
1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with a patient with pathology of the blood system in order to collect complaints and anamnesis, general examination, percussive determination of the size of the liver and spleen according to Kurlov, palpation of lymph nodes, liver and spleen, evaluation of clinical examination data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for the theoretical readiness of applicants to perform practical classes: the applicant must know the methods and scheme of examination of a patient with diseases of the blood system,

have an idea of the role of domestic scientists in the development of these methods; to know the general method of questioning patients with diseases of the blood system;

to know the characteristics and mechanisms of the main complaints; the meaning and purpose of a general examination of a patient with a pathology of the blood system;

to know the purpose, tasks and method of percussive determination of the size of the liver and spleen according to Kurlov

to know the method of palpation of lymph nodes, liver, spleen

to know the main clinical syndromes in pathology of the blood system

List of didactic units:

- collect in detail the complaints and medical history of a patient with blood system pathology;
- conduct a physical examination of the patient (general examination, percussive determination of the size of the liver and spleen according to Kurlov, palpation of lymph nodes, liver and spleen).
- identify and evaluate the changes that were found during the clinical examination,
- identify and evaluate the main clinical syndromes in the pathology of the blood system

2.2. Questions to check basic knowledge on the topic of the lesson:

1. Give a clinical assessment of the main complaints of patients with diseases of the hematopoietic system.

2. Explain objective changes in patients with blood diseases**,** give them a clinical assessment.
3. Describe the method of clinical examination of hematological patients.
4. Describe the signs of hemorrhagic and anemic syndromes.

Situational task.

Patient M., 32 years old, complains of weakness, increased fatigue, dizziness, subfebrile body temperature. 3 anamnesis is known to have been sick for about 3 months, the disease began gradually, worsening (increasing intensity) of symptoms**notes** 7-8 last days. On objective examination: the skin is pale, with a yellowish tinge, percussion of the sternum is painful; an increase in the liver and spleen is noted. In the blood, the content of erythrocytes and hemoglobin is reduced with a normal color index, poikilocytosis, anisocytosis, hypochromia, and microcytosis are noted.

1. Give a clinical interpretation of the described symptoms.
1. Make a plan for examining the patient, describe the expected results of additional studies.

3. Formation of professional skills and abilities:

mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the conducted interrogation with the assumption of the localization of the pathological process)

formation of the ability to perform a physical examination of a patient with a pathology of the blood system (the applicant must be able to perform a general examination of a patient with diseases of the blood system; perform percussive determination of the size of the liver and spleen according to Kurlov, palpation of lymph nodes, liver and spleen),

formation of the ability to evaluate the data obtained during the clinical examination of a patient with pathology of the blood system; carry out a clinical interpretation of the main symptoms and syndromes

3.1. Control materials for the final stage of the lesson:
Situational tasks:

Tasks of the STEP-2 type

1. A 21-year-old patient came to the clinic because of painful and prolonged menstruation, which takes the form of bleeding. General weakness, dizziness, tinnitus, shortness of breath, hair loss and brittle nails are a concern. Blood analysis: HB-56 g/l; er-2.8 t/l; CPU-0.6; leuk.-2.5 g/l; ESR-14 mm/h, platelets 139 g/l; reticulocytes-4.5%; anisocytosis with a tendency to microcytosis, hypochromia. Which of the diagnoses is possible?

+A. iron deficiency anemia;

B.B12 deficiency anemia;

S. autoimmune anemia;

D. aplastic anemia;

E. Werlhof's disease.

2. A 69-year-old patient complains of weakness, dizziness, burning tongue, decreased appetite. Gastric resection 5 years ago. Objectively: the skin is pale, the tongue is crimson, smooth; systolic murmur at the apex of the heart. In the blood analysis: er. - 2.2 t/l; HB-76 g/l; CPU-1,1; leukopenia; ESR 30 mm/h. Jolly bodies, Cabot rings. Your diagnosis?

A. stomach cancer

B. liver cirrhosis

S. Hemolytic anemia

+D. B12 deficiency anemia

E. iron deficiency anemia.

3.2. Requirements for work results, including before registration: substantiation of the syndromic diagnosis based on complaints, medical history and life data, clinical examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the

topic of the lesson, answering 10 tests (*if necessary*).

4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

List of recommended literature

Basic:

1. Propedeutics of internal medicine: textbook / Yu.I. Decyk, O.H. Yavorskyi, E.M. Neiko and others; under the editorship O.H. Yavorsky – 6th ed., corr. and added - K.: VSV "Medicine", 2020. - 552 p. + 12 p. color incl.

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.

Additional:

1. Methodology of examination of a therapeutic patient: teaching. manual / S.M. Andreychyn, N.A. Bilkevich, T.Yu. Chernets. – Ternopil: TDMU, 2016. – 260 p.

2. 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.

3. Yepishyn A.V. Propaedeutics of internal diseases with care of therapeutic patients / AV. Yepishyn// K. - 2015. 768p.

4. Kovalova OM. Propedeutics of internal medicine/ OM. Kovalova, NA Safargalina-Kornilova// K.: Medicine 2010 - 750p.

5.Macleod's Clinical Examination / Ed. G.Douglas, F.Nicol, C.Robertson.– 13th ed.– Elsevier. 2013. – 471 p.

6.Bates' Guide to Physical Examination and History Taking /Ed. Lynn S. Bickley, Peter G. Szilagyi. – Wolters Kluwer, 2017. – 1066 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/ - Odesa National Medical University

9. https://onmedu.edu.ua/kafedra/propedevtiki-vnutrishnih-hvorob-ta-terapii/ - Department of propaedeutics of internal diseases and therapyOdessa National Medical University

Practical lesson No. 22
Topic: Blood test. Clinical interpretation of blood analysis.

Goal: Acquiring knowledge and mastering professional competences during laboratory blood tests, clinical interpretation of blood tests.

Basic concepts:
Blood collection technique and methodology for general clinical analysis (hemoglobin determination, erythrocyte count, leukocyte count and leukocyte formula determination, platelet count, ESR determination). Morphological characteristics of formed blood elements. Norms of indicators of basic laboratory tests of blood. Clinical values of general blood analysis indicators. Clinical interpretation of general blood analysis.

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.

Laboratory diagnostics for lesions of hematopoietic organs In the clinic, morphological studies of blood cells are most widely used - general blood analysis, which includes the study of the quantitative and qualitative composition of the formed elements of blood (the number of erythrocytes, leukocytes and the ratio of their individual forms, hemoglobin content). In some patients, if necessary, additional studies are carried out: reticulocyte count, platelet formulas.
Determination of hemoglobin. The content of hemoglobin in the blood is determined for the diagnosis of anemia, erythremia, erythrocytosis, assessment of the degree of blood loss, the effect of ionizing radiation, blood thickening during dehydration of the body, and the effectiveness of hemotransfusion. The hemoglobin index in healthy women is 120-140, in men 130-160 g in 1 liter of blood. Knowing the number of erythrocytes in the blood and the content of hemoglobin in it, its concentration in one erythrocyte is calculated. For this, a conventional value (color index) is used. Normally, the color index is close to 1.0. A decrease in the indicator of less than 0.85 (hypochromia of erythrocytes) indicates insufficient saturation of the erythrocyte with hemoglobin, an indicator above 1.05 (hyperchromia of erythrocytes) is detected when the size of erythrocytes is larger than normal.
Erythrocyte count. The reason for the increase in the number of erythrocytes per unit volume of blood (erythrocytosis) may be the loss of fluid by the body or the excessive production of erythrocytes by the bone marrow, which is observed in erythremia, heart diseases, intoxications, dehydration, etc. A decrease in the amount of hemoglobin and erythrocytes indicates the presence of anemia; with iron-deficiency anemia, the hemoglobin content decreases to a greater extent, the color indicator decreases. With megalocytic anemia, the

color index increases due to the larger size of erythrocytes. For the diagnosis of various blood diseases, it is important to assess the shape, size, color of erythrocytes, and the presence of inclusions in them. In anemias, unequal intensity of erythrocyte coloration is observed - anisochromia, their unequal sizes - anisocytosis. Smaller cells are found - microcytes (average diameter 5.5 μm), less often larger cells - macrocytes (average diameter 8.0-8.5 μm), megalocytes (diameter greater than 12.5 μm). Iron deficiency anemia is characterized by microcytosis, for 175 B12-deficient anemia - megalocytosis. Macrocytosis occurs in liver diseases. When erythrocyte maturation is disturbed, poikilocytosis is detected - a change in the shape of cells to oval, pear-shaped, elongated, in the form of mulberry berries, plates, etc., and different erythrocytes have different shapes. Poikilocytosis is most often observed simultaneously with microcytosis. A significant increase in the number of reticulocytes (reticulocytosis) is observed in hemolytic anemia. An insignificant number of reticulocytes or their complete absence in the smear is a sign of a decrease in the regenerative activity of the bone marrow, which is observed in hypo- and areregenerative anemias

Study of white blood cells. Most often, during a clinical examination, the total number of leukocytes is counted. An increase in the number of white blood cells is leukocytosis, and their decrease is leukopenia. Leukocytosis often occurs under physiological conditions, for example, in pregnant women and during lactation, after meals, physical and mental stress. Under pathological conditions, the cause of leukocytosis can be acute infections, inflammatory and purulent-septic processes, comatose states (uraemic, diabetic, hepatic coma), various intoxications (food, carbon monoxide, arsenic, benzene derivatives), malignant neoplasms, aseptic inflammation, significant blood loss, hemolytic crises, concussion, surgical interventions, etc

Leukopenia is observed in some infections (typhoid, malaria, brucellosis, protracted septic endocarditis, viral hepatitis), collagenoses. It also occurs in blood diseases: acute leukemia, Addison-Birmer's disease, some splenopathies that occur with hypersplenism, as well as in hypoplastic hematopoietic conditions, the cause of which is often chronic benzene intoxication, radiation sickness. In many cases, leukopenia is caused by medication. Determination of the percentage ratio of individual forms of leukocytes (leukocyte formula) is of important diagnostic value. Most often, changes in neutrophils are observed in the leukocyte formula. An increase in their number (neutrophil leukocytosis) occurs with many infections, intoxications, malignant tumors, hemolysis and tissue decay. Active neutropoiesis is characterized by not only an increase in the number of neutrophils, but also a rejuvenation of their composition (or shift to the left - a nuclear shift of neutrophils). It consists in increasing the share of young forms of neutrophils in the blood formula - rod-shaped, young forms, sometimes - myelocytes. A decrease in the number of neutrophils - neutropenia - occurs when bone marrow function is suppressed by toxins of some microbes (causing agents of typhoid, brucellosis, etc.), viruses, ionizing radiation, and some medications. An increase in the total number of lymphocytes (lymphocytosis) is observed with viral lesions, during the recovery period after infectious diseases. In patients with whooping cough, infectious lymphocytosis, tuberculosis, chronic lymphocytic leukemia, up to 80% of lymphocytes are detected in the myelogram.

A decrease in the number of lymphocytes (lymphopenia) is observed in inflammatory and purulent-septic diseases, some severe infections. Absolute lymphopenia occurs with lymphogranulomatosis, lymphosarcoma, and malignant lymphoma. Sharply expressed

lymphopenia with absolute neutropenia develops with radiation sickness.
An increase in the number of eosinophils (eosinophilia) by more than 5-6% is observed in allergic reactions (bronchial asthma, dermatoses, serum sickness), helminthiasis, as well as in collagenoses (rheumatism, dermatomyositis), Dressler's syndrome, lymphogranulomatosis, chronic myeloid leukemia, malignant neoplasms, some infectious diseases (scarlet fever, tuberculosis, syphilis). In infectious and septic diseases, the appearance of eosinophils against the background of lymphocytosis and a slight shift of the formula to the right is a sign of recovery
A decrease in the number (eosinopenia) or the absence of eosinophils in the blood (aneosinophilia) is observed in the midst of acute infections with pronounced intoxication syndrome (typhoid, sepsis, severe forms of tuberculosis, etc.), in agony, bone marrow aplasia, hyperproduction of corticosteroids (acute infections, intoxication, shock , surgery, childbirth).
An increase in the number of basophils (basophilia) occurs in chronic myeloid leukemia, polycythemia, acute thrombocytopenia, as well as hypothyroidism
An increase in the number of monocytes (monocytosis) can occur in sepsis, tuberculosis, malaria, visceral leishmaniasis, syphilis, infectious mononucleosis, viral diseases (chicken pox, rubella, measles, influenza, diphtheria, typhus). A decrease in the content of monocytes (monocytopenia) is always found in severe septic processes, hypertoxic forms of typhoid fever, and other severe infectious diseases. A sensitive indicator of the presence of a pathological process is degenerative changes in leukocytes: toxic granularity of neutrophils, cytoplasmic vacuolization, the presence of various inclusions in leukocytes, degenerative changes in the nucleus - pyknosis, karyolysis
Investigation of platelets. A change in the number of platelets is observed in the form of thrombocytosis and thrombocytopenia. Thrombocytosis can occur in myeloproliferative processes (chronic myeloleukemia, polycythemia, osteomyelosclerosis), hemorrhagic thrombocythemia, malignant neoplasms (especially lung and pancreas tumors), purulent processes, lymphogranulomatosis, active forms of pulmonary tuberculosis.
Thrombocytopenia is a manifestation of bone marrow damage with suppression of thrombocytopoiesis. A decrease in the number of platelets occurs in Werlhof's disease and symptomatic thrombocytopenia, acute leukemia, hypoplastic anemia, radiation sickness, collagenoses, chronic nephritis, splenopathies, with long-term use of such medications as sulfonamides, quinine, barbiturates, streptomycin, and others
Determination of the sedimentation rate of erythrocytes. Erythrocyte sedimentation rate (ESR) is a non-specific indicator that characterizes the protein and mucopolysaccharide composition of blood and the state of erythrocytes. The value of ESR in healthy men is 2-10 mm per hour, in women - 2-15 mm per hour. An increase in the indicator is noted in most inflammatory processes, infections, malignant neoplasms, tissue decay, and to a certain extent proportional to the severity of the lesion. A decrease in ESR occurs with a decrease in the total amount of blood protein (alimentary and wound dystrophy, debilitating diseases), an increase in the amount of CO_2 in the blood (heart failure), an increase in the total number of erythrocytes (erythremia), an increase in the content of bile acids in the blood (mechanical and parenchymal jaundice), long-term use of certain medications (calcium, diuretics, phenobarbital, acetylsalicylic acid)
Study of blood coagulation system. Blood in the human body is in a liquid state due to the

dynamic balance of coagulation, anticoagulation and fibrinolytic systems. Most often in the clinic, classic coagulation tests are used, which make it possible to assess the state of the coagulation system as a whole. They include:
1. Determination of blood coagulation time: use the method of Lee and White. With DVZ-syndrome, acute purulent inflammation, croup pneumonia, diphtheria, acute rheumatic polyarthritis, etc. blood clotting time is reduced. With hemophilia A, it increases to several hours, it is significantly longer with severe liver diseases.
2. Determining the duration of bleeding according to Duque characterizes the time of spontaneous cessation of bleeding after damage to small vessels. Normally, it is 2-4 minutes. With thrombocytopenia, the bleeding time increases significantly, and with impaired capillary tone, the size of blood drops becomes larger
Capillary wall resistance test: a) tourniquet symptom: a tourniquet is placed on the examinee's forearm. Normally, petechiae appear on the skin of the forearm after 3 minutes; if they are detected earlier - the test is positive; b) pinch symptom - with increased capillary fragility, a hemorrhagic spot appears at the pinch site, which gradually increases and becomes more intense; c) hammer symptom - the appearance of bruises at the place of percussion with a neurological hammer. The listed symptoms are positive in severe infectious diseases, scurvy, Werlhoff's disease, uremia, leukemia, phosphorus poisoning, allergic reactions.
To determine the activity of the I phase of blood coagulation, the following tests are used:
1. Time of recalcification of blood plasma: is 60-70 seconds. 2. Prothrombin consumption test (prothrombin time according to Kwik): Normal prothrombin time is 12-18 s. Its lengthening indicates a deficiency of one of the factors of the prothrombin complex. With a simultaneous increase in thrombin time, one should think about hereditary hypo- or dysprothrombinemia, hypovitaminosis K, mechanical jaundice, intestinal dysbiosis, damage to the liver parenchyma
2. Assessment of the activity of the II phase of blood coagulation is carried out by determining the prothrombin index, plasma tolerance to heparin. 1. The prothrombin index is the ratio of the prothrombin time of the donor's plasma to the patient's plasma, expressed as a percentage. Normal indicators are 80-100%. 2. When studying the tolerance of plasma to heparin, the coagulation time of plasma is determined when a heparin-calcium mixture is added to it. With a tendency to thrombus formation, the tolerance of plasma to heparin increases, the clotting time of blood plasma decreases. With hemorrhagic syndrome, the changes will be the opposite. Normal indicators are 7-11 minutes.
3. Determining the quantitative content of fibrinogen makes it possible to assess the III phase of hemocoagulation. Normal indicators of plasma fibrinogen: weight method - 2-4 g/l, colorimetric - 2.5-3.0 g/l. A decrease in the level of fibrinogen in blood plasma occurs with acute DVZ syndrome, with the use of fibrinolytic therapy and treatment with defibrinating drugs, with hereditary hypo- and afibrinogenemia. Hyperfibrinogenemia is characteristic of acute and protracted inflammatory, immune and destructive processes (pneumonia, rheumatism, glomerulonephritis, etc.), occurs in CVD syndrome, systemic microthrombovasculitis, atherosclerosis, coronary heart disease

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:

1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking workbooks, interpretation of laboratory blood test data*),* conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for the theoretical readiness of applicants to perform practical classes: the applicant must know the methods and scheme of examination of a patient with diseases of the blood system,

to have an idea of the role of domestic scientists in the development of laboratory blood test methods;

to know the standards of indicators of basic laboratory blood tests and the diagnostic value of their changes

List of didactic units:

- take a blood sample for a general clinical analysis
- count the number of erythrocytes, leukocytes, and platelets
- determine the leukocyte formula, ESR
- evaluate the results of a general clinical blood test.

2.2. Questions to check basic knowledge on the topic of the lesson:

1. Describe the method of blood sampling for general analysis.
3. Describe the method of blood sampling for general analysis.
4. Describe the method of counting the number of erythrocytes.
5. Describe the method of counting the number of leukocytes.
6. Describe the method of determining the leukocyte formula.
7. Describe the method of determining ESR.
8. Describe the method of calculating the color index.
9. Describe the method of counting the number of platelets.
10. Describe the technique of staining a blood smear.
11. Describe the method of determining changes in erythrocytes and their degenerative forms.

Situational task.

Patient M., 32 years old, complains of weakness, increased fatigue, dizziness, subfebrile body temperature. 3 anamnesis is known to have been sick for about 3 months, the disease

began gradually, worsening (increasing intensity) of symptoms**notes** 7-8 last days. On objective examination: the skin is pale, with a yellowish tinge, percussion of the sternum is painful; an increase in the liver and spleen is noted. In the blood, the content of erythrocytes and hemoglobin is reduced with a normal color index, poikilocytosis, anisocytosis, hypochromia, and microcytosis are noted.

1. Make a plan for examining the patient, describe the expected results of additional studies.

3. Formation of professional skills and abilities:

mastering communication skills with the patient (during blood collection for laboratory tests)

formation of the ability to conduct a general clinical laboratory examination of blood - to draw blood for a general clinical analysis, count the number of erythrocytes, leukocytes, platelets, determine the leukocyte formula, ESR

formation of the ability to perform clinical interpretation of general blood analysis indicators, to identify basic laboratory syndromes.

3.1. Control materials for the final stage of the lesson:
Situational tasks:

Tasks of the STEP-2 type

1. When studying the results of the clinical blood analysis of patient S., the following data were obtained: erythrocytes - 4.4 T/l, hemoglobin - 138 g/l, color index - 0.9, leukocytes - 7.8 M/l, basophils - 0 %, eosinophils - 2%, young -0%, rod-nuclear - 6%, segmentonuclear - 67%, lymphocytes - 18%, monocytes - 7%. ESR - 8 mm/h. Sugar - 4.7 mmol/l. Evaluate the results obtained.
A. Hypochromic anemia
B. Leukocytosis
+C. There are no pathological changes
D. Hyperglycemia
E. Eosinophilia

2. When examining the results of the clinical blood analysis of patient N., the following data were obtained: erythrocytes - 4.38 T/l, hemoglobin - 136 g/l, color indicator - 0.9, leukocytes - 6.5 M/l, basophils - 0 %, eosinophils - 1%, young -0%, rod-nuclear - 6%, segmentonuclear - 68%, lymphocytes - 18%, monocytes - 7%. ESR - 8 mm/h. Sugar - 7.2 mmol/l. Evaluate the results obtained.
A. Hypochromic anemia
B. Leukocytosis

C. There are no pathological changes
+D. Hyperglycemia
E. Eosinophilia

3. When studying the results of the clinical blood analysis of patient L., the following data were obtained: erythrocytes - 4.3 T/l, hemoglobin - 127 g/l, color indicator - 0.9, leukocytes - 12.8 M/l, basophils - 0 %, eosinophils - 1%, young -0%, rod-nuclear - 5%, segmentonuclear - 70%, lymphocytes - 17%, monocytes - 7%. ESR - 19 mm/h. Platelets - 260 M/l. Evaluate the results obtained.
A. Thrombocytosis
+B. Leukocytosis, increased ESR
C. There are no pathological changes
D. Lymphocytosis
E. Eosinophilia

4. When examining the results of the clinical blood analysis of patient L., the following data were obtained: erythrocytes - 4.3 T/l, hemoglobin - 127 g/l, color indicator - 0.9, leukocytes - 12.8 M/l, basophils - 0 %, eosinophils - 1%, young -0%, rod-nuclear - 5%, segmentonuclear - 70%, lymphocytes - 17%, monocytes - 7%. ESR - 21 mm/h. Platelets - 260 M/l. The presence of which pathology can be assumed in the patient?
A. Hypochromic anemia
B. Aplastic anemia
C. There are no pathological changes
D. Allergic reaction
+E. Inflammatory process

5. When studying the results of the clinical blood analysis of patient F., the following data were obtained: erythrocytes - 4.4 T/l, hemoglobin - 138 g/l, color indicator - 0.9, leukocytes - 7.8 M/l, basophils - 0 %, eosinophils - 2%, young -0%, rod-nuclear - 6%, segmentonuclear - 68%, lymphocytes - 17%, monocytes - 7%. Platelets - 150 M/l. ESR - 9 mm/h. Evaluate the results obtained.
+A. Thrombocytopenia
B. Leukocytosis, increased ESR
C. There are no pathological changes
D. Lymphocytosis
E. Eosinophilia

6. When examining the results of the clinical blood analysis of patient Z., the following data were obtained: erythrocytes - 3.4 T/l, hemoglobin - 100 g/l, color indicator - 0.89, leukocytes - 8.3 M/l, basophils - 0 %, eosinophils - 1%, young - 0%, rod-nuclear - 6%, segmentonuclear - 63%, lymphocytes - 21%, monocytes - 9%. ESR - 13 mm/h. Evaluate the results obtained.
A. Hypochromic anemia
B. Leukocytosis, increased ESR
C. There are no pathological changes
D. Lymphocytosis

+E. Normochromic anemia

7. When studying the results of the clinical blood analysis of patient D., the following data were obtained: erythrocytes - 4.04 T/l, hemoglobin - 114 g/l, color indicator - 0.85, leukocytes - 2.8 M/l, basophils - 0 %, eosinophils - 1%, young -0%, rod-nuclear - 5%, segmentonuclear - 70%, lymphocytes - 17%, monocytes - 7%. ESR - 10 mm/h. Evaluate the results obtained.
A. Hypochromic anemia
B. Leukocytosis, increased ESR
C. There are no pathological changes
+D. Leukopenia
E. Normochromic anemia

3.2. Requirements for work results, including before registration: substantiation of the conclusion regarding the clinical interpretation of laboratory blood test data, definition of the leading laboratory syndromes.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (*if necessary*).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 23.
Subject: Pneumonia: Symptoms and syndromes. Clinical-instrumental and laboratory research methods. Respiratory failure syndrome in the pathology of the bronchopulmonary system.

Goal: Acquiring knowledge and mastering professional competences during the examination of a patient with pneumonia, bronchitis, bronchial asthma, pulmonary emphysema - questioning, physical examination, instrumental and laboratory research.

Basic concepts: Definition and modern classification of pneumonias (hospital, non-hospital, aspiration, pneumonia in immunocompromised persons), classification according to the nature of lung damage (pleuropneumonia, bronchopneumonia, interstitial pneumonia). The main etiological factors of pneumonia. Complaints of patients and peculiarities of these physical methods of examination of patients with pleuro- and bronchopneumonia. Criteria for a severe course of pneumonia. Possibilities of instrumental diagnosis of pulmonary tissue compaction. Laboratory signs of inflammatory syndrome in pneumonia. Pulmonary tissue compaction syndrome.
Definition and main mechanisms of development of chronic bronchitis and bronchial asthma. The main complaints and physical examination data of patients with chronic bronchitis and bronchial asthma. Syndrome of bronchial obstruction, mucociliary insufficiency and increased airiness of the lungs. Basic methods of instrumental diagnostics. Laboratory signs of bronchial asthma based on general blood analysis and sputum examination. Definition and

main clinical manifestations of bronchiectasis. The concept of chronic obstructive pulmonary disease.

Definition. Pneumonia is an acute infectious disease, mainly of bacterial etiology, which is characterized by focal lesions of the respiratory sections of the lungs and the presence of intra-alveolar exudation. Since pneumonia, by definition, is an acute infectious disease, the use of the term "acute" in the diagnosis of "pneumonia" is redundant, especially since the term "chronic pneumonia" is not used.
Etiology and pathogenesis. The reasons for the development of an inflammatory reaction in the respiratory departments of the lungs can be both a decrease in the effectiveness of the protective mechanisms of the macroorganism, and the massiveness of the dose of microorganisms and/or their increased virulence.
There are 4 ways of infection: - 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 pelvic veins); - direct spread of infection from affected tissues of nearby organs (for example, liver abscess) or as a result of infection during chest injuries.
Aspiration of the contents of the oropharynx is the main route of infection of the respiratory tract of the lungs during pneumonia. Under normal conditions, a number of microorganisms, such as Streptococcus pneumoniae, can colonize the oropharynx, but the lower respiratory tract remains sterile. Microaspiration of oropharyngeal contents is a physiological phenomenon observed 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, when 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. The massiveness of the dose and the virulence of individual microorganisms, hematogenous and direct spread of the pathogen from the focus of infection are of little importance (in terms of frequency of detection). Inhalation of an aerosol containing microorganisms is the cause of pneumonia when infected with obligate microorganisms, such as Legionella sp. An etiological diagnosis is necessary for a reasonable appointment of etiotropic therapy. However, in 50% of patients, even when using all possible research methods, it is not possible to determine the etiology of the disease (taking into account atypical, viral and anaerobic pathogens). The composition of the causative agents of pneumonia has some differences in patients depending on the severity of the course of the disease. With a mild course in 40-50% of patients, the etiology remains unverified. Streptococcus pneumoniae is detected more often (in 9-36% of cases) when sputum is cultured. However, the results of serological studies indicate the significant importance of Mycoplasma pneumoniae (13-37%), Chlamydophyla pneumoniae (17%), Haemophilus influenzae (5-10%), Legionella sp. (0.4-2.8%), gram-negative enterobacteria (0.2-1.3%), viruses (10-13%). The main causative agents of severe pneumonia, which threatens the patient's life, are Streptococcus pneumoniae (20-22%), Legionella sp. (5.5-17.8%),

Haemophilus influenzae (3.8-5.3%), Staphylococcus aureus (7-8.7%), gram-negative enterobacteria (1.6-8.6%), Mycoplasma pneumoniae (2-2.7%) and viruses (4-9.7%). If patients with severe pneumonia have specific risk factors, such as bronchiectasis, Pseudomonas aeruginosa may be among the potential pathogens. But in 50-60% of cases, the etiology of pneumonia with a severe course also remains undetermined.

Pneumonias are divided into primary and secondary. Primary pneumonia occurs in a person with healthy lungs who does not have diseases of other organs and systems that contribute to the occurrence of acute pneumonia or naturally lead to its occurrence as a complication. A mandatory, but not sufficient, condition for the development of the disease is the arrival of microflora from other parts of the respiratory tract or the environment - bronchogenically during inhalation with air; with aspiration of the contents of the nasopharynx, stomach; hematogenously or lymphogenously from a remote focus of infection.

The entry of the causative agent of acute pneumonia into the lungs can be exogenous or endogenous. The occurrence of pneumonia is often associated with a number of contributing conditions, or risk factors. 1. Viral infections of the upper respiratory tract. Inflammatory diseases of the nasopharynx and paranasal sinuses are often the underlying disease in acute pneumonia, which disrupts nasal breathing and creates conditions for infected secretions to enter the bronchi. 2. Obstruction of the bronchial tree. With chronic obstructive bronchitis, bronchial asthma, local obstruction of the bronchus by a tumor, foreign body, pneumosclerosis, peristaltic contractions of the bronchi and mucociliary transport are disturbed, which leads to mucus retention. 3. Alcohol. In patients with alcoholism, the pharyngeal reflex is impaired, which leads to periodic aspiration of the oropharyngeal flora. They also have mucociliary transport disorders and secondary immunodeficiency states. 4. Smoking and inhalation of toxic substances. At the same time, the ciliated epithelium suffers, the functional insufficiency of alveolar macrophages develops, and the formation of IgG decreases. Some hydrocarbons (gasoline, kerosene, kerosene, naphtha), fats of mineral, vegetable or animal origin, inhaled in high concentration, cause widespread burns of the mucous membrane of the bronchopulmonary apparatus, contribute to its secondary infection. Violation of the drainage function of the bronchi (increase in the amount of bronchial secretion, increase in its viscosity, stickiness, inhibition of the ciliated epithelium) contributes to the colonization of microorganisms and the bronchogenic spread of infection. A viral infection "prepares the ground" for the development of pneumonia. Viruses cause necrosis of epithelial cells of the upper respiratory tract and bronchi. Affected epithelial cells exfoliate, and the de-epithelialized surface is easily infected by bacteria. A decrease in the effectiveness of the body's protective factors is of decisive importance in the occurrence of pneumonia. According to leading pulmonologists, people do not get pneumonia, but get sick. The effectiveness of local factors of immune protection decreases - the activity of lysozyme, lactoferrin, secretory IgA, the concentration of bacterial antibodies decreases. Often, especially with a prolonged course of pneumonia, there is a decrease in the level of humoral immune factors - Ig A, M, G. Indicators of cellular immunity are also disturbed - the phagocytic activity of neutrophilic granulocytes and alveolar macrophages decreases, which contributes to intracellular parasitism of microorganisms and viruses, dissemination and progression of the inflammatory process in the lungs Pneumonia is accompanied by changes in the system of hemocoagulation and fibrinolysis. Enhancement of hemocoagulant and inhibition of fibrinolytic activity contributes to the limitation of the inflammatory zone. In the

period of the reverse development of the disease, fibrinolytic activity increases, which ensures sanogenesis of the inflammatory focus. Violation of the balance between the formation of fibrin and its destruction leads to the development of complications: the predominance of the processes of dissolution and elimination of fibrin leads to the further spread of inflammatory infiltrate, destruction of lung tissue, hemoptysis, pulmonary bleeding. The predominance of fibrinogenesis causes carnification of the lung parenchyma, the formation of pleural adhesions, and bronchial obstruction. Hyperfibrinogenemia, more often local, can lead to intravascular aggregation of platelets and the formation of platelet emboli, which cause local hemorrhagic necrosis of lung tissue. The influence of bacterial toxins, mediators of inflammation, hypoxia causes activation of endogenous phospholipases, as a result of which the oxidation of cell membrane lipids is activated. The destruction of membrane structures is accompanied by the accumulation of toxic products of hydroperoxides, fatty acids, lipid peroxidation: peroxides, lysophospholipids. Pneumonia is secondary to others, if it occurs against the background of a chronic bronchopulmonary disease - bronchiectasis, tumors, pneumoconiosis - or is a complication of diseases. The following main causes of secondary pneumonia are distinguished: circulatory disorders; aspiration, compression of the bronchi; lung or chest injury; postponed operative interventions; thermal effects; the influence of pathogenic physical factors: radiation, proton; sepsis; exacerbation of chronic obstructive bronchitis.

Classification. The classification of pneumonia, which most fully reflects the peculiarities of its course and allows prescribing etiotropic therapy to the patient, should certainly be based on the etiological principle. However, in practice, the etiological diagnosis of pneumonia in 50-70% of patients is complicated by the insufficient informativeness and considerable duration of traditional microbiological studies (the absence of a productive cough in 20-30% of patients, the impossibility of isolating intracellular pathogens when using standard diagnostic approaches, identification of the pathogen is possible only after 48- 72 hours after receiving the material, difficulties in distinguishing the "witness microbe" and the "causing microbe", the widespread practice of patients using antibacterial drugs before seeking medical help). Therefore, many countries of the world use a classification that takes into account the conditions for the occurrence of the disease, the features of lung tissue infection, as well as the state of immune reactivity of the patient's body. This makes it possible to predict the possible causative agent of the disease with a fairly high degree of probability. According to this classification, the following types of pneumonia are distinguished:

- non-hospital (outpatient, general, outpatient);
- nosocomial (hospital);
- aspiration;
- pneumonia in persons with severe immune disorders (congenital immunodeficiency, HIV infection, iatrogenic immunosuppression).

Of greatest practical importance is the division of pneumonia into non-hospital (acquired outside a medical institution) and nosocomial (acquired in a medical institution). This division is not related to the severity of the course of the disease, and the only criterion for division is the environment in which pneumonia developed. In addition, depending on the severity, pneumonia of a mild, moderate and severe course is distinguished. However, clear criteria for the distribution of mild and moderate pneumonia have not yet been developed. Since the scope of diagnostic and treatment measures for pneumonia of these degrees of

severity is almost the same, it is advisable to combine them into one group - pneumonia with a mild course. The following definition of pneumonia with a severe course should be followed - it is a special form of the disease of various etiology, which is manifested by a severe 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 single out "small" and "large" criteria for the severe course of pneumonia.
"Small" criteria for a severe course of pneumonia: - respiratory rate 30 in 1 minute. and more; - disturbance of consciousness; - SaO2 is less than 90% (according to pulse oximetry data), the partial tension of oxygen in arterial blood (Pa O2) is below 60 mm Hg. art.; - systolic blood pressure below 90 mm Hg. art.; - bilateral or multipart lung damage, decay cavity, pleural effusion.
"Big" criteria for a severe course of pneumonia: - the need for artificial lung ventilation; - rapid progression of focal and infiltrative changes in the lungs - an increase in the size of the infiltration by more than 50% during the next 2 days; - septic shock or the need to administer vasopressor drugs within 4 hours. and more; - acute renal failure (the amount of urine is less than 80 ml in 4 hours or the level of creatinine in the blood serum is higher than 0.18 mmol/l or the concentration of urea nitrogen is higher than 7 mmol/l (urea nitrogen = urea (mmol/l) / 2.14) for (absence of chronic renal failure). Severe pneumonia is indicated by the presence of at least two "small" or one "large" criteria in patients, each of which reliably increases the risk of a fatal outcome. In such cases, urgent hospitalization of patients in the anesthesiology department is recommended and intensive therapy.

Respiratory failure syndrome. The feeling of lack of air can be caused by frequent coughing, chest pain when breathing. Objective signs of respiratory insufficiency - tachypnea more than 30 respiratory movements per minute, cyanosis - occur with a severe course of pneumonia. In severe cases with pronounced intoxication and respiratory insufficiency, there is swelling of the wings of the nose, tension of the respiratory muscles.
Cough is the leading "local" symptom of pneumonia, appearing on the first day of the disease. At first, the cough is dry, painful, strong, sometimes to the point of vomiting. With the appearance of sputum, the cough softens. There may be no sputum in the first days of the disease. The nature of sputum often changes with the development of the disease: at first, the sputum is mucous, scanty, often contains streaks of blood, and is sometimes uniformly stained with blood. The appearance of "rusty" sputum with a high content of hemolyzed erythrocytes is possible. In the midst of pneumonia, sputum, as a rule, has a mucous-purulent character. At the end stage of the disease, sputum again acquires a mucous character, becomes liquid, and easily leaves. If pneumonia arose as a result of a disease of the cardiovascular system, sputum may be bloody throughout the disease. Chest pains with pneumonia can have different origins and characteristics. Parietal pains caused by intercostal myalgia or neuralgia are local, intensify during breathing and movements associated with the load on this group of muscles and upon palpation of this area. The most intense parietal pain occurs at the beginning of the disease. Parenchymatous pains are accompanied by massive compaction in the lungs, have an unclear character, are not strong, without a clear localization, but are practically constant. Pleural pains due to inflammatory lesions of the pleura, as a rule, have an intense nature, decrease when lying on the affected side, increase

with deep breathing and coughing. When the basal segments are affected, the pain may radiate to the abdominal cavity or be completely localized there. With inflammation of the lingual segments, pain may occur in the area of the heart or behind the sternum. Damage to the upper lobe is often accompanied by reflex tension of the occipital muscles. Reactive inflammation of the diaphragmatic pleura can simulate a picture of an acute abdomen - strong, sharp pains in the abdomen caused by irritation of the diaphragmatic, vagus and sympathetic nerves, sometimes incessant vomiting. When the pleura is involved in the process, breathing becomes frequent, shallow, the affected half of the chest lags behind when breathing, the patient takes care of it, often holds it with his hand.

Physical examination. Manifestations during physical examination depend on the clinicomorphological form of pneumonia. When examined, patients with croup pneumonia often show a characteristic appearance: a feverish blush on the cheeks, more intense on the affected side due to the involvement of the cervical node of the sympathetic nerve in the process. Mucous membranes can acquire a cyanotic shade. Elderly people with concomitant damage to the cardiovascular system have marked cyanosis of the lips, tips of the ears, cheeks, and distal phalanges of the fingers. 30% of patients have herpes rashes on the lips and wings of the nose. The sclera may be subicteric. The position of the patient is forced: he lies on the affected side of the chest, the head is raised. Breathing is superficial, tachypneous 30-40 per minute. The affected half lags behind in the act of breathing, the auxiliary respiratory muscles are tense, the intercostal spaces are smoothed.

During palpation, already in the first hours of the disease, physical signs of compaction of the lung tissue due to hyperemia and microbial edema are revealed - over the area of the affected segments, an increase in bronchophonia and vocal tremor is determined (in 70-90% of patients). Segments in which inflammation develops become less saturated with air than normal and better conduct sound vibrations to the chest.

Percussion in the stage of hyperemia and microbial edema over the area of the affected segments is determined by a dull dulling of the percussion sound in almost all patients due to compaction of the lung tissue. In addition, the percussion sound acquires a peculiar tympanic tone, as the elasticity of the lung tissue and the tone of the alveoli decrease, the latter stretch and expand. In the stage of lung hepatization, the percussion sound acquires a more pronounced dull character, the tympanic component disappears completely or is heard locally. Excursion of the lower edge of the lungs on the affected side is sharply reduced. In the phase of completion of pneumonia, percussive dullness with a tympanic shade changes to a clear lung sound. Auscultatively, croupous pneumonia can be manifested by various sound phenomena depending on the phase of the disease. When examining a patient with focal pneumonia, the general condition can be satisfactory, often of moderate severity. A forced position with the head raised is characteristic of elderly patients. In a third of patients, there is a lag in the act of breathing of the chest on the side of the lesion and a decrease in the mobility of the lower edge of the lungs by 2-3 cm. In upper lobe pneumonia, tension and soreness of the trapezius muscle on the side of the lesion are observed. Pronounced paleness of the skin against the background of acrocyanosis or hyperemia of the cheeks is possible. Herpetic rashes are observed in 30-40% of patients. In the area of the pneumonic focus, the tenderness of the intercostal spaces can be determined when pressing with a finger or a stethoscope. When the diaphragmatic leaf of the pleura is damaged, pain appears during deep palpation in the hypochondrium. An increase in vocal tremor is determined only in 10-15%

of patients with large focal or draining pneumonias. A more important symptom is increased bronchophonia - observed in 2/3 of cases. Percussion in small foci of pneumonia is not very informative. In case of superficial focal pneumonias, the percussion sound is shortened, large focal and draining pneumonias are characterized by a significant dulling of lung sounds over a large area. The most significant for the diagnosis of focal pneumonia are auscultatory manifestations. Small foci pneumonia is characterized by hard breathing and local small-bubble wet wheezing. With medium-focal pneumonia, harsh breathing and small-bubble moist rales are heard over a larger area. Large focal pneumonia is distinguished by bronchial or hard breathing, scattered wet wheezes. In congestive focal pneumonia, in addition to the above-mentioned symptoms, crepitation can be determined. Objective data for pneumonia depend on the prevalence, localization and phase of the inflammatory process. Pulmonary tissue compaction syndrome appears when there is a massive, somewhat superficial compaction of lung tissue. Physical signs of the syndrome are determined in places where the zone of inflammation projects onto the surface of the chest. Condensation of lung tissue can develop quickly, within a day. The earliest symptoms are increased bronchophonia and vocal tremor. The dulling of the percussion tone is determined percussively. Auscultatively - bronchial breathing, prolonged exhalation can be heard completely. Bronchitis syndrome: depending on the viscosity of the exudate that fills the bronchi, dry or moist rales are heard. When the small bronchi are affected, wheezing is dry with a squeak or whistling or moist with small bubbles. When large bronchi are involved - dry buzzing and large-bubble wet. Wheezing can disappear after expectoration of sputum or use of bronchodilators. Pleural effusion syndrome occurs with croupous pneumonia. A dulling of the percussion sound appears over the posterior basal parts of the lungs, limited by an oblique line with the highest point along the posterior axillary line. Bronchophonia and vocal tremor over the area of fluid accumulation are weakened, vesicular breathing is also sharply weakened. Atelectasis syndrome can sometimes accompany pleural effusion syndrome, rarely develops independently. There is a local dulling of the percussive tone, a local increase in vocal tremor and bronchophonia, vesicular breathing is sharply weakened or absent. Physical manifestations of pneumonia often depend on the phase of morphological changes. The phase of exudation - at the beginning of the disease, a tympanic shade of percussion tone is detected above the site of pronounced exudation in the lung tissue, due to a decrease in the elasticity of the lung tissue. The accumulation of exudate in the alveoli leads to a dulling of the percussion sound. Auscultatively, in the phase of exudation, weak breathing is heard over the affected area. In the first days of the disease, a gentle crepitation can be heard at the height of inhalation - crepitatio indux. This is a rather rare symptom, it is not observed with shallow and frequent breathing. Filling the bronchi with exudate causes the appearance of symptoms of bronchitis - scattered dry and wet wheezes appear. The consolidation phase - on the 2nd-3rd day of the disease, there is an increase in bronchophonia and vocal tremor, the severity of the dulling of the percussive sound increases, although the tympanic tone remains. The end stage of pneumonia is characterized by a mosaic percussion pattern - zones of blunting of the percussion sound are adjacent to areas that give a tympanic shade of the percussion tone. After resorption of the exudate, a clear lung sound is determined by percussion. With the restoration of alveolar aeration, bronchial breathing weakens, crepitation appears again - crepitatio redux. Breathing becomes hard, and after that - vesicular, resounding small-bubble rales often appear. Vicarious emphysematous expansion

of a healthy lung is often detected. Reactive changes in the cardiovascular system are most often manifested by tachycardia up to 100-120 beats per minute, a decrease in blood pressure. Less often, there is an expansion of the borders of the heart to the right due to acute expansion of the right ventricle and atrium, the emphasis of the II tone over the pulmonary artery is a sign of acute pulmonary hypertension. Functional disorders of the digestive organs are manifested by nausea, vomiting, anorexia, constipation. The tongue is often coated, dry, and the stomach is bloated. With a severe course of pneumonia, jaundice of the skin and sclera may appear, the liver increases in size and becomes painful. Changes on the part of the nervous system appear in weakened patients, with a severe course of the disease: increased excitability, delirium, phenomena of acute psychosis. The appearance of meningeal symptoms is possible - stiffness of the occipital muscles, Kernig's symptom, hyperesthesia of the skin, impaired consciousness, severe headache. With a mild course of pneumonia, there may be complaints of headache.

Additional research methods.

1. Hemogram. Patients with pneumonia most often have leukocytosis, often moderate (10-12x109/l), neutrophilia 80-90%, rod-nuclear shift up to 7-30%, sometimes young forms of leukocytes and myelocytes appear. The content of eosinophils, basophils, lymphocytes in the peripheral blood decreases, the level of monocytes increases. Thrombocytopenia is often observed, sometimes in combination with a hemorrhagic syndrome. ESR often and significantly increases.
2. When examining the biochemical blood analysis, signs of an immuno-inflammatory syndrome are determined - dysproteinemia (increased levels of α-1- and α-2-, γ-globulins), increased levels of reactive protein, sialic acids, seromucoids, fibrinogen, haptoglobin and other acute phase indicators .
3. When examining urine, symptoms of acute toxic kidney disease may be detected - priteinuria, cylindruria, microhematuria.
4. Sputum research and microbiological diagnosis. Establishing an etiological diagnosis is of great importance for the treatment of pneumonia - identifying the causative agent of the disease and its sensitivity to antibiotics. The sequence of microbiological diagnostics in pneumonia: - Microscopy (bacterioscopy) of smears stained according to Gram, for the differentiation of gram-positive and gram-negative microflora (oriented express method) - Sowing of material (bacteriological method) for isolation and identification of the causative agent, determination of its sensitivity to antibiotics - Microscopy of smears according to Ziel-Nielsen (mycobacterium tuberculosis bacterioscopy) - Determination of specific antibodies and antigens in blood serum by serological methods (for the verification of atypical pneumonia) - the method of paired serums - 2 blood tests in the acute period of the disease and in the period of convalescence - after a few weeks from onset of the disease The etiological role of the microorganism in the development of the disease is confirmed by the increase of antibodies to this microorganism in serum series by four or more times. For the identification of antibodies, the complement binding reaction (CRK), the hemagglutination inhibition reaction (RHGA), the neutralization reaction (PH), and the immunofluorescence reaction (IFR) are used. 5. Immunological studies. Immunological changes make it possible to detect various disorders of cellular and humoral immunity, to assess the immune reactivity of this patient. In many patients with pneumonia, there is a decrease in the number and activity of lymphocytes, the percentage of phagocytic cells, the phagocytic index, and the

amount of lysozyme in lymphocytes and monocytes. Viral and bacterial pneumonias that developed after a viral infection are characterized by an increased content of T-suppressors and a decrease in the number of T-helpers. With a prolonged course of pneumonia, immunological changes are more significant: decreased content of T- and B-lymphocytes, Ig A, M, G, activity of lysozyme, lactoferrin, concentration of antibacterial antibodies decreases.

6. Spirography. When studying spirometric indicators, a mixed type of ventilation disorders is revealed - a combination of restrictive and obstructive changes, even if there are no clinical manifestations of bronchial obstruction. The clinical equivalent of brochial obstruction at the level of large bronchi is paroxysmal cough; at the level of small bronchi - constant expiratory shortness of breath. Scattered dry rales with whistling appear when bronchial patency is disturbed at the level of medium and small bronchi.

5. X-ray studies. X-ray symptoms of pneumonia depend on the stage of the disease. In the tidal stage, the strengthening of the lung pattern and the decrease in the background transparency due to the overflow of blood in the pulmonary vessels are determined on X-rays. If the affected area is less than one fate, the diagnosis of changes is complicated. The root of the lung on the affected side is expanded, its structure is blurred. When the lower lobe segments are affected, there is a decrease in the mobility of the dome of the diaphragm. In the stage of hepatization, there are homogeneous intense darkenings, which in terms of density resemble atelectasis without displacement of the mediastinal organs in the direction of the lesion. The intensity of the shadow increases towards the periphery. In case of massive granular pneumonia with the involvement of the entire lobe of the lung, the shadow is uniform throughout. The most frequent for croupous pneumonia is damage to 2-3 segments (70% of patients). 1-3 segments are affected only in 5% of patients. Right-sided pneumonia is observed 1.7 times more often than left-sided pneumonia. Damage to the areas of the lungs along the interlobular fissures can be diagnosed only radiologically - auscultatory symptoms are not detected, since the focus is located very deep. Pericissural pneumonias in lateral projections form elongated shadows - one contour is clear, rectilinear (from the side of the interlobular pleura), the other contour is blurred (from the side of the lung parenchyma). Croupous pneumonia is often accompanied by a reaction of the interlobular and costal pleura - in a third of patients, fluid is found in the interlobular fissures, exudative pleurisy may join. In the final stage of croupous pneumonia, the intensity of the shadow decreases, its size decreases. The strengthening of the pulmonary pattern at the place of the pneumonic focus persists for 3-4 weeks after the resolution of the pneumonia. The diagnosis of pneumonia, which is not fully completed, is carried out with the help of the Valsalva test - the patient tries to exhale through the nose with closed nostrils and mouth - the lung pattern becomes paler due to the narrowing of the vessels. Muller's test is applied - the patient tries to inhale with a closed glottis - the pulmonary pattern is enhanced due to the overflow of blood vessels. If the deformation of the vascular pattern in the post-pneumonic zone is caused by pneumosclerosis, the expressiveness of the vascular pattern will not change during the tests. Post-pneumonic changes include expansion, homogenization of the corresponding laterality of the lung root pneumonia, which can be observed within 3-4 weeks. The exudate in the pleural cavity after the elimination of the inflammatory process is absorbed within 1-1.5 months.

6. Determination of arterial blood gases.

7. Study of pleural effusion.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:

1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with a patient with pneumonia, bronchitis, bronchial asthma, emphysema of the lungs for the purpose of collecting complaints and anamnesis, general and local examination, palpation of the chest, comparative and topographical percussion of the lungs, auscultation of the lungs, evaluation clinical examination data and laboratory-instrumental data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for the theoretical readiness of applicants to perform practical classes:

- the applicant must know the modern definition, etiology, pathogenesis, classifications of pneumonia, bronchitis, bronchial asthma, pulmonary emphysema, subjective and objective data in these diseases

- to know laboratory, X-ray and instrumental data for this pathology.

List of didactic units:

- to conduct a subjective examination of patients with pneumonia, bronchitis, bronchial asthma, pulmonary emphysema
- conduct an objective examination of patients with pneumonia, bronchitis, bronchial asthma, pulmonary emphysema
- evaluate X-ray changes in patients with pneumonia, bronchitis, bronchial asthma, pulmonary emphysema
- evaluate laboratory indicators in patients with pneumonia, bronchitis, bronchial asthma, emphysema of the lungs
- master the skills and abilities to assess leading clinical syndromes in patients with pneumonia, bronchitis, bronchial asthma, pulmonary emphysema

2.2. Questions to check basic knowledge on the topic of the lesson:

Questions to test basic knowledge

1. Etiology, pathogenesis, symptomatology of acute bronchitis;
2. Etiology, pathogenesis, symptomatology, methods of diagnosis of chronic bronchitis;
3. Etiology, pathogenesis, classification, symptomatology, methods of diagnosis of bronchial asthma;
4. Etiology, pathogenesis, classification, symptomatology, methods of diagnosis of pulmonary emphysema;
5. Instrumental and X-ray symptomatology of pulmonary emphysema;

6. General indicators during spirometry and their changes in obstructive and restrictive disorders of the function of external breathing.
7. Etiology, pathogenesis of fateful and focal pneumonia.
8. Clinical manifestations, clinical and morphological classification of focal and lobar (croupous) pneumonia.
9. Clinical manifestations of focal pneumonia.
10. X-ray and laboratory data in pneumonia.

Tests to check basic knowledge

1. In the patient, after examining the function of external breathing, an obstructive type of ventilation disorder was determined.
Which of the listed diseases should be considered in this patient?
A. Atelectasis
B. Pneumonia
S. Exudative pleurisy
+D. COPD
E. Pneumothorax

2. The Tiffno index of 56% is determined in the patient when examining the function of external breathing.
Which of the listed diseases should be considered in this patient?
A. Atalectasis
B. Pneumonia
S. Exudative pleurisy
+D. COPD
E. Pneumothorax

3. After examining the function of external respiration, the patient was diagnosed with a restrictive type of ventilation disorder.
Which of the specified changes were detected in him?
+A. Reduction of lung volumes
B. A decrease in the Tiffno index
S. Reduction of the volume of forced exhalation in 1 second
D. An increase in the forced vital capacity of the lungs
E. Reduction of the average volume velocity of the middle of exhalation

3. Formation of professional skills and abilities:
- mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the interview)

- formation of the ability to perform a clinical examination of a patient with (the applicant must be able to perform a physical examination of a patient with pneumonia, bronchitis, bronchial asthma, pulmonary emphysema),
- formation of the ability to evaluate the data obtained during the clinical examination of a patient with pneumonia, bronchitis, bronchial asthma, emphysema; carry out a clinical interpretation of the main symptoms and syndromes in these diseases

- formation of the ability to conduct a modern laboratory-instrumental examination of a patient with pneumonia, bronchitis, bronchial asthma, emphysema of the lungs (the applicant must be able to prescribe a plan of laboratory-instrumental examination, give an assessment of the examination results)

3.1. Control materials for the final stage of the lesson:
1. Conduct a subjective examination of a patient with pneumonia.
2. Conduct an objective examination of a patient with pneumonia,
3. Conduct a subjective examination of a patient with bronchitis and bronchial asthma.
4. Conduct an objective examination of a patient with bronchitis and bronchial asthma.
5. Conduct a subjective examination of a patient with pulmonary emphysema.
6. Conduct an objective examination of a patient with pulmonary emphysema.
7. To draw up a plan of laboratory-instrumental examination of a patient with pneumonia.
8. Draw up a plan for laboratory-instrumental examination of a patient with bronchitis and bronchial asthma.
9. To draw up a plan of laboratory-instrumental examination of a patient with emphysema of the lungs.
10. Carry out a clinical interpretation of laboratory and instrumental examination data in a patient with pneumonia.
11. Clinical interpretation of laboratory and instrumental examination data in a patient with bronchitis and bronchial asthma.

Situational tasks:
Tasks of the STEP-2 type
1. A 62-year-old patient, a smoker with a 43-year "experience", complains of a morning cough with slimy sputum, mainly in the cold season. Periods of coughing with sputum discharge, shortness of breath are noted for a total of 4-5 months a year for 10-12 years. What is the most likely diagnosis?
A. Pneumonia.
B. Bronchial asthma.
S. Acute bronchitis.
+D. COPD.
E. Lung abscess.

2. The patient, 23 years old, complains of an attack of shortness of breath with difficulty exhaling, which forced him to take a sitting position with support on his hands, the attack lasts 30 minutes, accompanied by a non-productive cough.

What cough is most likely at the end of the attack?
A. Dry
B. With the release of a large amount of purulent sputum
S. With discharge of three-layer sputum
+D. With discharge of vitreous sputum
E. The cough will stop

3. The patient has an attack of expiratory dyspnea accompanied by a nonproductive cough. Occurred after 10 minutes of being in the hayloft. An examination reveals a swollen face, acrocyanosis.
What changes will take place during the examination of the neck?
A. "Carotid Dance"
V. "Dance of the jugular veins"
C. Enlargement of the thyroid gland
D. Swelling of the carotid arteries
+E. Swelling of jugular veins

4. A 43-year-old patient has suffered from bronchial asthma for 23 years. Usually notes the daily symptoms of bronchial asthma: attacks of expiratory wheezing, dry or unproductive cough, feeling of tightness in the chest. Short-acting beta agonists are used to relieve symptoms. The last attack of dysentery lasts about a day, for several hours inhalation of a beta-agonist strengthens the dysentery.
What condition has the patient developed?
A. Cardiac asthma
+V. Asthmatic status
C. Hypercapnic coma
D. Respiratory distress syndrome
E. Prolonged attack of bronchial asthma

6. A 30-year-old patient developed an attack of expiratory wheezing accompanied by a nonproductive cough. During the examination, a swollen face, acrocyanosis, swelling of the neck veins, protrusion of the supraclavicular spaces, and smoothness of the intercostal spaces are determined. What percussion sound will be determined at
comparative percussion?
A. Yasny
V. Dull tympanitis
+S. Korobkovy
D. Tympanic
E. Stupid

7. A 35-year-old patient was admitted to the surgical department 4 days ago due to appendicitis. He was operated on on the day of admission. Complaints of cough with slimy sputum, weakness, shortness of breath. In the evening the day before, chills were noted, a moderate fever appeared. The right half of the chest lags behind when breathing, the vocal

tremor below the angle of the scapula is increased, the percussion sound is dulled, the vesicular breath is weakened, wet fine-vesicular rales.
Your previous diagnosis?
+A. Hospital (nosocomial) pneumonia
V. Community-acquired pneumonia
S. Aspiration pneumonia
D. Acute bronchitis
E. Exacerbation of chronic bronchitis

8. The patient, 17 years old, became acutely ill the day before, complaining of sharp pain in the right half of the chest when breathing and coughing, shortness of breath, dry cough. On objective examination: body temperature -39.4C, shortness of breath, 30 breaths per minute, the right half of the chest lags behind during breathing, vocal tremor below the angle of the scapula is increased, percussively there is dull tympanitis, breathing is bronchovesicular, crepitation.
What kind of crepitation does the patient have?
+A. Inducing crackling
B. Recurrent rattle
C. Hard crackling
D. A small rattle
E. A big bang

9. The patient, 19 years old, became acutely ill 3 days ago, complaining of pain in the right half of the chest when breathing and coughing, difficulty breathing, coughing with the release of "rusty" sputum. On objective examination: body temperature - 39.0 C, shortness of breath, 27 breaths per minute, the right half of the chest lags behind during breathing, vocal tremor below the angle of the scapula is increased, percussively there - dullness.
What additional research is the most appropriate to conduct for the patient?
A. Study of the function of external breathing
B. Roentgenoscopy of the chest organs
+S. X-ray of chest organs
D. Computer tomography of chest organs
E. Tomography of chest organs

10. Patient, 25 years old. Complaints of cough with slimy sputum, weakness, shortness of breath. The disease is associated with hypothermia 3 days ago. In the evening the day before, chills were noted, a moderate fever appeared. The right half of the chest lags behind when breathing, vocal tremor below the angle of the scapula is increased, dulling of the percussion sound, weakened vesicular breathing, wet fine-vesicular rales.
What changes with topographic percussion can be expected in this patient?
A. The apex of the right lung is not defined
B. Extended Krenig field on the right
C. Decreased mobility of the lower edge of the left lung
+D. The mobility of the lower edge of the right lung is reduced
E. Extended Krenig field on the left

3.2. Requirements for work results, including before registration: substantiation of the diagnosis based on complaints, medical history and life data, clinical and laboratory-instrumental examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (*if necessary*).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

List of recommended literature

Basic:
1. Propedeutics of internal medicine: textbook / Yu.I. Decyk, O.H. Yavorskyi, E.M. Neiko and others; under the editorship O.H. Yavorsky – 6th ed., corr. and added - K.: VSV "Medicine", 2020. - 552 p. + 12 p. color incl.
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.

Additional:
1. Methodology of examination of a therapeutic patient: teaching. manual / S.M. Andreychyn, N.A. Bilkevich, T.Yu. Chernets. – Ternopil: TDMU, 2016. – 260 p.
2. 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.
3. Yepishyn A.V. Propaedeutics of internal diseases with care of therapeutic patients / AV. Yepishyn// K. - 2015. 768p.
4. Kovalova OM. Propedeutics of internal medicine/ OM. Kovalova, NA Safargalina-Kornilova// K.: Medicine 2010 - 750p.
5.Macleod's Clinical Examination / Ed. G.Douglas, F.Nicol, C.Robertson.– 13th ed.– Elsevier. 2013. – 471 p.
6.Bates' Guide to Physical Examination and History Taking /Ed. Lynn S. Bickley, Peter G. Szilagyi. – Wolters Kluwer, 2017. – 1066 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/ - Odesa National Medical University
9. https://onmedu.edu.ua/kafedra/propedevtiki-vnutrishnih-hvorob-ta-terapii/ - Department of propaedeutics of internal diseases and therapyOdessa National Medical University

Practical lesson No. 24
Topic: Symptoms and syndromes in chronic obstructive pulmonary disease, bronchial asthma, bronchitis, pulmonary emphysema. Data analysis of instrumental research methods. The concept of impaired function of external breathing. Spirography
Goal: Acquaint applicants with the current state of the problem of bronchial asthma, chronic obstructive pulmonary disease, bronchitis, emphysema of the lungs. Etiology, pathogenesis, symptomatology. Instrumental and X-ray symptomatology. General indicators during spirometry and their changes in obstructive and restrictive disorders of the function of external breathing.

Basic concepts: Chronic obstructive pulmonary disease (COPD) and bronchial asthma belong to the group of chronic obstructive pulmonary diseases. The most characteristic symptoms of this group of diseases are cough, sputum discharge, signs of varying degrees of expressiveness. However, other diseases have similar clinical symptoms (for example, bronchostasis, specific lung diseases - tuberculosis, pneumoconiosis, etc.), these diseases can be combined, so the separation of these conditions is one of the tasks of the doctor.
Emphysema of the lungs is characterized by the destruction of the walls of the alveoli and a pathological increase in the air spaces beyond the terminal re-respiratory bronchioles.
The relevance of the researched topic is also determined by the prevalence of chronic obstructive pulmonary diseases, the steady growth of their morbidity and mortality from admissions associated with COPD. The social and economic consequences of COPD are very diverse, suffice it to say that 11-19% of people in the general population suffer from severe airway obstruction. According to research, about 3% of humanity suffers from bronchial asthma; in the south of Ukraine, about 5% of the population is sick with it. Chronic bronchitis accounts for 65 to 90% of COPD. Over the past decades, COPD mortality has increased every 5 years. Thus, this problem is important not only in medical, but also in social aspects.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:

1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with a patient with dry and exudative pleurisy for the purpose of collecting complaints and history, general and local examination, chest palpation, comparative and topographical percussion of the lungs, auscultation of the lungs, assessment of clinical examination data and laboratory instrumental data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.
2.1. Requirements for the theoretical readiness of applicants to perform practical classes:

2.2. Questions to check basic knowledge on the topic of the lesson:
3. Formation of professional skills and abilities:

"STEP" tasks
1. The patient, 23 years old, complains of an attack of shortness of breath with difficulty exhaling, which forced him to take a sitting position with support on his hands.
What side breath noises are most likely in this patient?
A. Pleural friction noise.
B. Moist fine-vesicular rales.
C. Crepitation.
+D. Dry wheezing
E. Wet vesicular rales.

2. A 62-year-old patient, a smoker with 43 years of "experience", complains of a morning cough with slimy sputum, mainly in the cold season. Periods of coughing with sputum discharge, shortness of breath are noted for a total of 4-5 months a year for 10-12 years.
What is the most likely diagnosis?
A. Pneumonia.
B. Bronchial asthma.
S. Acute bronchitis.
+D. Chronic bronchitis.
E. Lung abscess.

3. The patient, 23 years old, complains of an attack of shortness of breath with difficulty exhaling, which forced him to take a sitting position with support on his hands, the attack lasts 30 minutes, accompanied by a non-productive cough.
What cough is most likely at the end of the attack?
A. Dry
B. With the release of a large amount of purulent sputum
S. With discharge of three-layer sputum
+D. With discharge of vitreous sputum
E. The cough will stop

4. In a patient, an attack of expiratory dyspnea accompanied by a non-productive cough. Occurred after 10 minutes of being in the hayloft. An examination reveals a swollen face, acrocyanosis.
What changes will take place during the examination of the neck?
A. "Carotid Dance"
V. "Dance of the jugular veins"
C. Enlargement of the thyroid gland
D. Swelling of the carotid arteries
+E. Swelling of jugular veins

5. A 43-year-old patient has suffered from bronchial asthma for 23 years. Usually notes the daily symptoms of bronchial asthma: attacks of expiratory wheezing, dry or unproductive cough, feeling of tightness in the chest. Short-acting beta agonists are used to relieve symptoms. The last attack of dysentery lasts about a day, for several hours inhalation of a beta-agonist strengthens the dysentery.
What condition has the patient developed?
A. Cardiac asthma
+V. Asthmatic status
C. Hypercapnic coma
D. Respiratory distress syndrome
E. Prolonged attack of bronchial asthma
6. A 30-year-old patient developed an attack of expiratory dyspnea accompanied by a nonproductive cough. During the examination, a swollen face, acrocyanosis, swelling of the neck veins, protrusion of the supraclavicular spaces, and smoothness of the intercostal spaces are determined. What percussion sound will be determined at
comparative percussion?
A. Yasny
V. Dull tympanitis
+S. Korobkovy
D. Tympanic
E. Stupid

7. A 33-year-old patient has suffered from bronchial asthma since he was 29 years old. Usually attacks of expiratory wheezing, dry or unproductive cough, a feeling of tightness in the chest are noted less often once a week. A day after another attack of dysentery, the patient was examined by a doctor.
What is the most likely auscultatory picture of the lungs?
+A. Vesicular breath, there are no side breath sounds.
B. Bronchovesicular breath, crepitatio indux
S. Pathological bronchial breath
D. Vesicular weakened breath, moist fine-vesicular rales
E. Pathological bronchial breath, moist fine-vesicular wheezing

8. A 60-year-old patient, a smoker with 43 years of "experience", complains of a morning cough with slimy sputum, mainly in the cold season. Periods of coughing with sputum

discharge, shortness of breath are noted for a total of 4-5 months a year for 10-12 years. Over the past few years, leg swelling, hepatomegaly, and neck vein swelling have been noted.
What complication is most likely to develop in the patient?
+A. Chronic pulmonary heart
B. Emphysema of the lungs
S. Acute pulmonary heart
D. Bronchiectasis
E. Ischemic heart disease

9. A 56-year-old patient, a welder by profession, has had a morning cough with thin green sputum, shortness of breath during moderate physical exertion, and sometimes elevated body temperature for 12 years.
What is the most likely diagnosis?
+A. Chronic purulent bronchitis
B. Acute bronchitis
C. Chronic simple bronchitis
D. Chronic hemorrhagic bronchitis
E. Pneumonia

10. After an attack of expiratory dyspnea, which was accompanied by a nonproductive cough, the patient secreted a large amount of vitreous sputum, some of which was in the form of "casts" from the bronchi.
What elements are most likely to be detected in sputum during microscopic examination?
A. Tuberculosis mycobacteria
B. Uric acid crystals
C. Erythrocytes
+D. Charcot-Leyden crystals
E. Rings of Cabot

"Symptomatology of pulmonary emphysema; clinic, diagnosis"

1. When examining a 64-year-old patient, a symphony orchestra trombonist, shortness of breath with difficulty exhaling, a barrel-shaped chest is determined.
What percussion sound will be determined by comparative percussion?
A. Yasny
V. Dull tympanitis
+S. Korobkovy
D. Tympanic
E. Stupid

2. The patient is 56 years old, has been suffering from chronic bronchitis for 16 years, the patient has swelling of the supraclavicular regions, the epigastric angle is 120, the voice tremor is weakened, and a box sound is heard over the lungs.
What auscultatory phenomena are most likely in this patient?
A. Pleural friction noise.

B. Metal breath
C. Crepitation.
+D. Dry wheezing
E. Amphoric breath

3. A patient, a 25-year-old stockbroker by profession, who complains of shortness of breath during moderate physical exertion, has a barrel-shaped chest, a box-like percussion sound, with topographic percussion - an increase in the standing height of the tops of the lungs, and the width of the Krenig fields. During auscultation - weakening of breath.
Which of the etiological factors of the disease is the most likely?
+A. Antitrypsin deficiency
B. Working with a computer
C. Hypodynamia
D. Chronic emotional stress
E. Using a mobile phone

4. When examining a 45-year-old patient, a worker in the "hot" workshop of the Zaporozhstal plant, shortness of breath with difficult exhalation, a barrel-shaped chest, weakened vesicular breathing, dry wheezing is determined.
Which of the etiological factors is not important in this disease?
A. Work in a "hot" shop
V. Smoking
S. Living in an industrial area
D. Viral respiratory infections
+E. Alcohol abuse

5. The patient is 38 years old, has been suffering from chronic bronchitis for 9 years, the patient has swelling of the supraclavicular regions, the epigastric angle is 90, the voice tremor is weakened, a box sound over the lungs.
What auscultatory phenomenon will not be determined in the patient?
A. Weakened vesicular breath
V. Dry wheezing
+S. Pathological bronchial breathing
D. Moist rales
E. Lengthening of exhalation

6. The patient, 45 years old, a worker in the "hot" workshop of the Zaporozhstal plant, complains of shortness of breath with difficulty exhaling, morning cough with sputum production for 9-10 years. During the examination, a barrel-shaped chest, weakened vesicular breathing, dry wheezing is determined.
What complication is most likely to develop in the patient?
A. Chronic pulmonary heart
+V. Emphysema of the lungs
S. Acute pulmonary heart
D. Bronchial asthma

E. Ischemic heart disease

7. A 26-year-old patient, who has been suffering from chronic bronchitis for 3 years, underwent a study of the function of external breathing.
Which of the indicators will be the most informative for determining early obstructive changes?
+A. The average volume velocity of the middle of exhalation
B. Volume of forced exhalation in 1 second
S. Index Tifno
D. Peak expiratory volume velocity
E. Forced vital capacity of the lungs

8. In the patient, after the examination of the function of external breathing, an obstructive type of ventilation disorder was determined.
Which of the listed diseases should be considered in this patient?
A. Atalectasis
B. Pneumonia
S. Exudative pleurisy
+D. COPD
E. Pneumothorax

9. The Tiffno index of 56% is determined in the patient when examining the function of external breathing.
Which of the listed diseases should be considered in this patient?
A. Atalectasis
B. Pneumonia
S. Exudative pleurisy
+D. COPD
E. Pneumothorax

Practical lesson No. 25
Subject: The main symptoms and syndromes in dry and exudative pleurisy.
Goal: Acquiring knowledge and mastering professional competences during the examination of a patient for dry and exudative pleurisy, abscess and gangrene of the lungs, bronchiectatic disease, lung cancer, respiratory failure - questioning, physical examination, instrumental and laboratory tests.
Basic concepts: Causes of inflammation of the pleural sheets. Ways of formation and circulation of intrapleural fluid in normal and pathological conditions. Peculiarities of complaints of patients with dry and exudative pleurisy, differences in physical examination data (palpation, percussion, auscultation of the lungs) with different forms of pleurisy. Syndromes of fluid and air accumulation in the pleural cavity. Possibilities of instrumental diagnostics. Pleural puncture: examination of the contents of the pleural cavity. Difference between exudate and transudate according to physical and laboratory examination data. The main clinical manifestations and stages of respiratory failure syndrome in lung diseases.

Pleuritis is an inflammatory lesion of the pleura, with the formation of fibrin on its surface and/or the accumulation of effusion in the pleural cavity. Pleurisy is not an independent disease and is a pathological process that is a complication of certain diseases of the lungs, and much less often, the chest wall of the mediastinum, diaphragm or organs of the subdiaphragmatic space. Etiology and pathogenesis. Since pleurisy is a complication of some disease, primarily of the lungs, it is conventionally accepted to consider the cause that led to the appearance of the main disease as their etiology. According to etiological features, pleurisy is divided into infectious (including infectious-allergic) and aseptic. Non-infectious pleurisy can occur in rheumatoid arthritis, lung cancer, metastases, malignant lymphomas, myocardial infarction and Dressler's syndrome, as well as in benign ovarian tumors with ascites and pleurisy (Meigs syndrome), mesothelioma, fungal lesions, hypothyroidism. Fluid of a non-inflammatory nature (transudate) in the pleural cavity occurs in congestive heart failure, nephrotic syndrome and liver cirrhosis as a result of an increase in hydrostatic and/or decrease in oncotic blood pressure. With infectious-allergic pleurisy, antigens (microbes and toxins), as well as protein and high-polymer protein-polysaccharide complexes, which are formed as a result of the damaging effect of microbes on tissues and changes in tissue metabolism, are observed from the focus of infection to the subpleural zone of the lungs and pleura. As a result of this, a large number of biologically active substances appear, which causes an increase in the permeability of the vascular wall, the formation of effusion and damage to the complex non-vascular structures of the pleura - the tissue barrier. With purulent pleurisy, the direct influence of microbes prevails in the pathogenesis. Fungal pleuritis occurs mainly in people with signs of immunodeficiency. The risk group includes people who take immunosuppressants, corticosteroids for a long time, as well as patients with chronic diseases that contribute to a decrease in immunity (AIDS, diabetes, etc.). Pleurisy can have the following causes: 1) the spread of a pathological process from the lungs to the pleura (in case of pneumonia, pulmonary infarction); 2) penetration of an infectious agent or irritating substance into the pleural cavity (pancreatic pleurisy, amoebic empyema, etc.); 3) transfer to the pleura with blood or lymph of an infectious, toxic agent or tumor cells (tuberculous, uremic pleurisy, effusion in rheumatoid arthritis, systemic lupus erythematosus, carcinomatosis of the pleura); 4) injuries of the pleura, especially with rib fractures; 5) pleural effusion is rarely associated with medication. First, the pleura becomes swollen and congested, then cellular infiltration occurs, and a fibrinous exudate forms on the surface of the pleura. It can dissolve or consolidate into fibrous tissue with the formation of pleural adhesions. Some diseases can progress without noticeable exudation of fluid from the inflamed pleura, then the pleurisy remains dry (fibrinous). But more often, pleural exudate is formed as a result of the penetration of liquid containing many plasma proteins from damaged vessels. Sometimes fibrosis of the pleura and even its calcification occurs without previous acute pleurisy, for example, with asbestosis or with idiopathic calcification of the pleura.

Classification. There is currently no generally accepted classification of pleurisy. It should be emphasized once again that pleurisy, as a rule, is not an independent disease. First of all, according to the origin, pleural effusion is divided into inflammatory, tumor and congestive. Inflammatory pleurisy can be conditionally divided into: 1) infectious - bacterial, fungal, viral, etc.; 2) parasitic - amebiasis, echinococcosis, paragonimosis, etc.; 3) fermentogenic - pancreatogenic; 4) allergic and autoimmune – exogenous allergic alveolitis, Dressler's

syndrome, drug allergy; 5) in rheumatic diseases – systemic lupus erythematosus, rheumatism, rheumatoid arthritis, etc.; 6) traumatic.
Clinical manifestations. Usually, pleurisy of inflammatory origin is characterized by a sudden onset, and for tumorous or stagnant pleurisy – a slow gradual increase in symptoms. Three main syndromes are distinguished in the clinical picture of pleurisy: syndrome of dry (fibrinous) pleurisy, syndrome of exudative (with liquid exudate, non-purulent) pleurisy, syndrome of purulent pleurisy (pleural empyema). These syndromes can be observed in isolation or pass into one another. Manifestations of dry pleurisy complement the signs of the main process in the lungs (for example, pneumonia) or come to the fore. In the latter case, the general condition suffers slightly. Patients complain of pronounced stabbing pain, mostly in the lateral part of the chest on the side of the lesion, which are aggravated by breathing, coughing and bending the trunk in the opposite direction. Irritation of the back and peripheral parts of the diaphragmatic pleura can cause pain that spreads to the lower part of the chest wall or the abdomen, simulating abdominal pathology. Damage to the central part of the diaphragmatic pleura causes pain that radiates to the shoulders and neck. The cough is dry, the frequency of breathing increases. Sometimes hiccups and painful swallowing are observed.
The anamnesis is unclear, more often associated with a "cold". With exudative (purulent) pleurisy, patients note a feeling of heaviness, distension and simultaneous compression on the affected side of the chest, sometimes a dry cough against the background of general malaise. Accumulation of a large amount of liquid makes itself known by shortness of breath with certain difficulty exhaling, acceleration of the heartbeat. Occasionally, signs of acute cardiopulmonary disease may appear. When the effusion appears, the pain usually subsides.
Physical examination. Dry pleurisy: the chest on the side of the lesion lags behind in the act of breathing. There are no percussive changes in the absence of lung damage. During auscultation on the side of the lesion, vesicular breathing is slightly weakened (due to superficial breathing due to pain) and pleural friction noise. Pleural friction noise is not always heard and only in the first 24-48 hours after the onset of pain. The noise can be gentle, barely audible, imitating crepitation, or it can be loud, crackling, screeching. The noise is synchronous with breathing, heard during inhalation and exhalation. Noises heard during pleurisy in the pericardial area - pleuro-pericardial friction - can be associated with both heartbeat and breathing. Voice tremor and bronchophonia have not changed. Exudative pleurisy: the patient is in a forced position on the affected side. Cyanosis on the face appears on the background of pallor, neck veins and intercostal spaces on the affected side of the chest may swell with its asymmetrical increase over the affected area. Voice trembling is weakened or absent, breathing noises weaken or disappear. The greater the volume of effusion, the more pronounced these symptoms. Massive exudate reduces lung volume, causing or increasing shortness of breath. The patient's condition can be affected by a shift of the mediastinum to the healthy side (with para-pneumonic pleurisy) or to the affected side (with effusion combined with atelectasis or pneumofibrosis). Percussion in the lower parts of the location of the fluid is a massive blunting with a parabolic upper level, the top of which is located on the posterior axillary line. On the back, it gently descends to the spine, and in front - down to the midclavicular line. At the same time, two right-angled triangles are distinguished on the chest: Garland and Grokko-Rauchfus. The legs of the Garland triangle are the ridge line and the perpendicular dropped from the top of the liquid level to the ridge

line, and the Sokolov-Damoisot line is the hypotenuse. Dull tympanic sound is determined due to compression atelectasis. The Grokko-Rauchfus triangle is found in the presence of a large amount of fluid, and its legs are the spine line, the tender edge of the lung on the healthy side, and the extension of Damoiseau's line on the healthy side serves as the hypotenuse. Dull percussion sound - due to displacement of the mediastinum in the healthy direction. In the area of the exudate, vesicular respiration is weakened or completely absent, in the Garland triangle - with a bronchial shade, in the Grokko-Rauchfus triangle - weakened vesicular.

Data of additional research methods.

1. Hemogram. Moderate leukocytosis, ESR is slightly impaired in mild forms, and in case of cancerous pleurisy or empyema of the pleura, changes in the blood can be significant: pronounced neutrophilic leukocytosis, anemia, high ESR indicators.
2. Biochemical analysis of blood. The number of albumins decreases, and the number of alpha-1 and alpha-2 globulins increases, the indicators of fibrinogen and the activity of proteinase inhibitors are high.
3. X-ray of the chest. X-ray of chest organs does not play a significant role in the diagnosis of dry pleurisy, because pleural damage does not give any shadows, only pleural thickening can be observed. But chest radiography is an important method of detecting pleural fluid. In the absence of adhesions between the parietal and visceral pleura, the pleural fluid is located in the lower parts of the chest. Due to the elastic traction of the lungs, the upper edge of the effusion has the shape of a parabola (Damoisot line). The minimum amount of fluid that can be detected in a patient in an upright position is 300-500 ml, but upon careful examination with a change in the position of the patient's body, even a small amount of fluid can be detected: 10-15 ml. Adhesions between the parietal and visceral pleura often lead to atypical localization of pleural fluid.
4. Computed tomography is an extremely important method for assessing the state of the lung parenchyma in patients with pleural damage. A lung abscess, pneumonia, and obscuration due to bronchogenic cancer may appear at the site of the ossified pleural effusion.
5. Ultrasound examination With the help of ultrasound, not only the volume of effusion is determined, but it is possible to differentiate serous or purulent exudate based on echogenicity indicators. Ultrasound makes it possible to increase the accuracy of diagnosis of limited sinus, basal, interlobular, ossified and mantle-like effusions. The combination of x-ray methods with ultrasound makes it possible to determine not only the prevalence and localization of the effusion, but also accompanying changes in the lungs and other internal organs.
6. Pleural puncture is included in the mandatory diagnostic minimum. Pleural thoracentesis allows you to confirm the presence of fluid and determine its signs. The appearance of pleural contents has a certain diagnostic value. Hemorrhagic exudate is observed in pulmonary embolism, injuries, tumors; brown - with amebiasis; milk - with chylothorax; increased viscosity is characteristic of mesethelioma; rotten smell - for empyema.
7. Bacteriological examination of pleural contents gives the maximum information in case of infectious nature of pleurisy. In the punctate, the amount of protein is examined, Rivalt or Lucerini tests are carried out. 8. Microscopic examination of the Gram-stained sediment of the pleural fluid is important in all cases when it is purulent. At the same time, in addition to

bacteria, it is sometimes possible to detect fungi and actinomycetes. An increase in the number of neutrophils during cytological examination of the punctate can indicate suppuration, multinucleated atypical cells - its tumor nature. Pleural empyema may occur with pneumonia complicated by parapneumonic pleurisy. Empyema should be suspected even with a non-purulent-looking exudate, if the number of neutrophils in it exceeds 1×1011/l, bacteria (more often anaerobes) are found in Gram-stained smears, and pH < 7.2. Empyema also occurs with contamination of the pleural cavity when a lung abscess breaks through. The process can be complicated by a bronchopleural fistula, which sometimes occurs when an empyema breaks through the lung tissue into the bronchus. Empyema can also be the result of a penetrating wound, thoracotomy, the spread of infection from a liver or subdiaphragmatic abscess, or is the result of a rupture of an internal organ. Breakthrough of the purulent focus occurs during a strong cough. When lung destruction breaks into the free pleural cavity, a total pyopneumothorax occurs, and if the pleural cavity is partially obliterated (in the area of destruction or in other places), then it is limited. If, on the basis of physical and X-ray examination data, there is an assumption about the development of empyema, a diagnostic puncture of the pleura and aspiration of the contents should be performed.

Effusion with the properties of transudate accumulates in congestive heart failure, the criteria of which are an increase in the size of the heart, liver, ascites, and swelling of the lower extremities. Shortness of breath and atrial fibrillation are also characteristic. Accumulation of transudate can occur in cirrhosis of the liver with portal hypertension (splenomegaly, ascites, varicose veins of the subcutaneous veins of the anterior abdominal wall, veins of the esophagus, stomach, hemorrhoidal veins), as well as in nephrotic syndrome (proteinuria, hypoproteinemia, hypercholesterolemia). Transudate is also observed in patients with myxedema, alimentary dystrophy, cachexia, hypovitaminosis C, B1. Pleural effusion occurs in approximately 40% of patients with systemic lupus erythematosus or drug-induced lupus.

Complication. Complications with pleurisy are divided into local (pulmonary) and general (extrapulmonary).

Pulmonary complications include respiratory failure, atelectasis, pneumotrovex, diaphragmatic hernia. Atelectasis is a frequent complication in patients in the postoperative period (it does not matter for what reason the surgical intervention was performed). Extrapulmonary complications, which mainly arise as a result of pleural empyema, include renal amyloidosis and toxic nephrozonephritis. Pneumothorax is an acute condition characterized by the appearance of air in the pleural cavity and lung collapse (collapse, compression atelectasis). There is primary (idiomatic or spontaneous) and secondary (symptomatic) pneumothorax. Primary pneumothorax most often occurs as a result of rupture of subpleural emphysematous bubbles located mainly at the top of the lung, a limited adhesion process in the pleural cavity, lung infarction, pneumoconiosis, lung and pleural tumors. Pneumothorax also develops when a tubercular cavity, abscess or lung cyst breaks into the pleural cavity, lung is injured by a rib fragment.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:

1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with a patient with dry and exudative pleurisy for the purpose of collecting complaints and history, general and local examination, chest palpation, comparative and topographical percussion of the lungs, auscultation of the lungs, assessment of clinical examination data and laboratory instrumental data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for the theoretical readiness of applicants to perform practical classes:

- the applicant must know the etiology of pleurisy, subjective symptoms of dry and exudative pleurisy, objective symptoms of dry and exudative pleurisy
- to know the physical, biochemical, morphological characteristics of pleural exudate, the method of collecting instruments for pleural puncture and the technique of pleurocentesis, the main complications of pleurocentesis.
- to know the etiology, pathogenesis, clinical manifestations of abscess, lung gangrene, bronchiectasis disease, laboratory, X-ray and instrumental data in this pathology.

List of didactic units:

- to conduct a subjective examination of patients with pleurisy.
- conduct an objective examination of patients with pleurisy.
- to assess X-ray changes in patients with pleurisy.
- to evaluate the laboratory indicators of pleural exudate.
- collect a set of tools and prepare the patient for pleural puncture
- perform a pleural puncture
- master the skills of clinical examination of patients with abscess, lung gangrene, bronchiectasis.
- master the skills and abilities of evaluating clinical data and data of diagnostic studies in these diseases.

2.2. Questions to check basic knowledge on the topic of the lesson:

Questions to test basic knowledge

1. Definition of dry and exudative pleurisy.
2. Classification of dry and exudative pleurisy.
3. Etiology and pathogenesis of abscess of dry and exudative pleurisy.
4. Conducting subjective research:

a. Clarification of complaints.
b. Collection of analysis.

5. The value of methods of objective research of respiratory organs at diagnosis of dry and exudative pleurisy.

a. General overview.
b. Examination of the chest.

V. Palpation of the chest.
Mr. Lung percussion.
d. Auscultation of the lungs.

6. To evaluate the physical and macroscopic properties of pleural exudate.
7. Definition of lung abscess, lung gangrene, bronchiectasis.
8. Classification of lung abscess, lung gangrene, bronchiectasis.
9. Etiology and pathogenesis of lung abscess, lung gangrene, bronchiectasis.
10. Conducting subjective research:
a. Clarification of complaints.
b. Collection of analysis.
11. The value of methods of objective research of respiratory organs at diagnosis of lung abscess, lung gangrene, bronchiectasis.
a. General overview.
12. Examination of the chest.
V. Palpation of the chest.
Mr. Lung percussion.
d. Auscultation of the lungs.
b. The importance of laboratory, instrumental and X-ray methods examination for the diagnosis of purulent lung diseases.

Tests to check basic knowledge

1. The most frequent etiological factor in the development of exudative pleurisy is:
+A. Tuberculosis.
B. Diffuse connective tissue diseases.
S. Chest injury.
D. Hemophilia.
E. Diabetes mellitus.

2. Name the manifestations of pleurisy:
A. Pressing pain in the precardial region.
B. Increased vocal tremor.
+S. Lagging of the chest on the affected side.
D. Amphoric breathing.
E. Fine-bubble wet rales.

3. Pain in case of dry pleurisy intensifies:
A. In the supine position.
A. With a deep breath.
+S. When leaning in the opposite direction.
D. During the day.
E. When the fever decreases.

4. The main distinguishing physical symptom of dry pleurisy is:

A. Cough.
B. Connection of pain with breathing.
S. Shortness of breath.
+D. Pleural friction noise.
E. Soreness of the trapezius muscles.

5. What is the pleural friction noise characterized by:
A. It is heard only during inhalation.
A. It is heard only on exhalation.
S. Decreases after coughing.
D. Heard at a distance.
+E. It is heard during inhalation and exhalation

3. Formation of professional skills and abilities:
- mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the interview)
- formation of the ability to perform a clinical examination of a patient with kidney pathology (the applicant must be able to perform a physical examination of a patient for dry and exudative pleurisy, lung abscess and gangrene, bronchiectasis, lung cancer, respiratory failure),
- formation of the ability to evaluate the data obtained during the clinical examination of a patient with dry and exudative pleurisy, lung abscess and gangrene, bronchiectasis, lung cancer, respiratory failure; carry out a clinical interpretation of the main symptoms and syndromes in kidney diseases
- formation of the ability to conduct a modern laboratory-instrumental examination of a patient with dry and exudative pleurisy, lung abscess and gangrene, bronchiectasis, lung cancer, respiratory failure (the applicant must be able to prescribe a plan for laboratory-instrumental examination, give an assessment of the examination results)

3.1. Control materials for the final stage of the lesson:
1. Conduct a subjective examination of a patient with dry pleurisy.
2. Conduct an objective examination of a patient with dry pleurisy.
1. Conduct a subjective examination of a patient with exudative pleurisy.
2. Conduct an objective examination of a patient with exudative pleurisy.
3. Define the Damoiseau line.
4. Define the Garland triangle.
5. Define Rauchfuss-Grocco triangle.
6. Determine the indications for pleural puncture.
7. 3 choose instruments for pleural puncture.
10. Prepare the patient for pleural puncture.
11. Assist the doctor during pleural puncture.

Situational tasks:
Tasks of the STEP-2 type
1. After determining the lower border of the right lung, the doctor found that it is 2 cm along

all topographic lines. above the norm.
What additional research should be done?
*A - X-ray
In - Bronchophonia
C - Bronchography
D - Biopsy
E - X-ray

2. When percussing the chest, the doctor found a dulling of lung sounds on the left. The upper border of this blunting has the form of a parabolic line with the apex along the posterior axillary line. Palpatory vocal tremor is not determined.
What pathological process should the doctor think about?
*A - left-sided exudative pleurisy
In - right-sided exudative pleurisy
C - focal pneumonia
D - chronic bronchitis
E - partial pneumonia

3. When percussing the chest, the doctor found a dulling of lung sounds on the left. The upper border of this blunting has the form of a parabolic line with the apex along the posterior axillary line. Palpatory vocal tremor is not determined.
What should be the patient's percussion sound in Traube's space
*A - blunting
In - amplification
C - box
D - tympanic
E - blunt-tympanic

4. During comparative percussion, the doctor found a dull percussion sound to the right from the X rib downwards with the upper border in the form of the Damoiseau-Sokolov line, vocal tremor and bronchophonia are absent in this area.
What disease should the doctor think about?
A - left-sided exudative pleurisy
*B - right-sided exudative pleurisy
C - focal pneumonia
D - chronic bronchitis
E - partial pneumonia

5. Patient F., 27 years old, came in with complaints of pains in the left side of the chest that worsen with deep breathing, shortness of breath, dry cough, 5 days ago, after hypothermia, the body temperature rose to 38 C and pains in the left side of the chest appeared. cells The pains were very strong at first, then they became weaker, but shortness of breath increased. Ob-but: in the lungs on the left there is a shortening of the percussive down to the 4th rib. Breathing in the upper parts is bronchial, further down it is sharply weakened and cannot be heard.

What disease should the doctor think about?
*A - left-sided acute purulent pleurisy
In - right-sided exudative pleurisy
C - focal pneumonia
D - chronic bronchitis
E - partial pneumonia

List of recommended literature

Basic:

1. Propedeutics of internal medicine: textbook / Yu.I. Decyk, O.H. Yavorskyi, E.M. Neiko and others; under the editorship O.H. Yavorsky – 6th ed., corr. and added - K.: VSV "Medicine", 2020. - 552 p. + 12 p. color incl.
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.

Additional:

1. Methodology of examination of a therapeutic patient: teaching. manual / S.M. Andreychyn, N.A. Bilkevich, T.Yu. Chernets. – Ternopil: TDMU, 2016. – 260 p.
2. 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.
3. Yepishyn A.V. Propaedeutics of internal diseases with care of therapeutic patients / AV. Yepishyn// K. - 2015. 768p.
4. Kovalova OM. Propedeutics of internal medicine/ OM. Kovalova, NA Safargalina-Kornilova// K.: Medicine 2010 - 750p.
5.Macleod's Clinical Examination / Ed. G.Douglas, F.Nicol, C.Robertson.– 13th ed.– Elsevier. 2013. – 471 p.
6.Bates' Guide to Physical Examination and History Taking /Ed. Lynn S. Bickley, Peter G. Szilagyi. – Wolters Kluwer, 2017. – 1066 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/ - Odesa National Medical University
9. https://onmedu.edu.ua/kafedra/propedevtiki-vnutrishnih-hvorob-ta-terapii/ - Department of propaedeutics of internal diseases and therapyOdessa National Medical University.

Practical lesson No. 26

Topic: Main symptoms and syndromes in lung abscess, gangrene, bronchiectasis. Lung cancer.

Goal: Acquiring knowledge and mastering professional competences during the examination of a patient for dry and exudative pleurisy, abscess and gangrene of the lungs, bronchiectatic disease, lung cancer, respiratory failure - questioning, physical examination, instrumental and laboratory tests.

Basic concepts:

Lung abscess (AL) is a disease characterized by the presence of a limited focus of necrosis in the lungs (more often within a segment) with the formation of one or more destruction cavities filled with pus and surrounded by perifocal infiltration, as a result of purulent or putrefactive melting of lung tissue under the influence of infection.

Pulmonary gangrene (HL) is a purulent necrosis of a significant area of lung tissue or the entire lung with ichorous decay and rejection without a tendency to limit from viable parenchyma (without signs of demarcation), which has a tendency to spread. Lung gangrene is a serious disease that develops in people with severely impaired immunity, as a rule, it is caused by an association of microorganisms, among which anaerobic microflora is mandatory.

Etiology and pathogenesis. The reasons leading to the development of destructive lung lesions are diverse. However, the main thing is the combined interaction of the following factors: an acute infectious inflammatory process in the lung parenchyma; violation of bronchial patency; violation of blood circulation, blood supply and the formation of necrosis of lung tissue. Most often, bronchogenic infection of the lung tissue causes inflammation of the parenchyma and small bronchi. Atelectasis of the lung occurs as a result of violation of the patency of the bronchi due to spasm, edema or obstruction. Progressing tissue infiltration and swelling leads to compression of blood vessels and capillaries by the infiltrate, to a blood flow disorder that reaches stasis and thrombosis. Hematogenous or lymphogenous penetration of pathogenic microflora from an obstructed bronchus, from the upper respiratory tract, oral cavity into necrotized areas of the lung leads to purulent decomposition. With a satisfactory state of the body's defenses, low virulence of the microflora, adequate draining function of the bronchi, rational drug therapy, resorption of the infiltration, collapse and healing of the abscess cavity occurs - recovery occurs. With high pathogenicity of the microflora, sharp suppression of immune reactions, the progression of the purulent-necrotic process beyond the primary lesion and the development of widespread gangrene begins. The development of lung abscesses is due to various reasons, the main of which, in addition to the presence of purulent pathogenic microflora, an acute inflammatory process in the lung tissue, is a violation of bronchial patency and a local disorder of pulmonary blood circulation, which leads to necrosis of the lung tissue.

According to the mechanism of development, bronchopulmonary (in particular aspiration), hematogenous-embolic, post-traumatic and lymphogenic pleural abscesses are distinguished. A favorable background for the development of lung abscesses is chronic bronchopulmonary diseases - chronic obstructive bronchitis (in smokers), bronchiectasis, bronchial asthma. Lung abscesses complicate the course of pneumonia mainly in people with immunodeficiency - alcoholics, drug addicts, HIV-infected people. Infectious diseases of the lungs (for example, flu), severe trauma, blood diseases, hypovitaminosis, and diabetes mellitus sharply disrupt the general resistance of the body and contribute to the development

of lung abscesses. Acute pulmonary-pleural suppuration is mainly a polymicrobial infection caused by associations of anaerobic-aerobic microorganisms. Most often, lung abscesses are single; multiple abscesses are usually unilateral and can occur simultaneously or spread from one focus. Abscesses of aspiration origin are localized mainly in the upper segment of the lower lobe and in the posterior segment of the upper lobe. Solitary abscess caused by bronchial obstruction or infected embolus begins as necrosis of most of the affected bronchopulmonary segment. The base of the segment is usually adjacent to the chest wall, and the pleural cavity in this place is often obliterated by adhesions of inflammatory origin. Hematogenous dissemination is characterized by multiple scattered foci, it may be associated with tricuspid valve endocarditis, especially in injection drug users. In an embolic lung abscess, anaerobic bacteria or aerobes can be detected, which caused purulent thrombophlebitis. An abscess usually breaks into a bronchus, its contents are coughed out, and a cavity filled with air and fluid remains. In some cases, the abscess breaks into the pleural cavity, which causes empyema, sometimes with a bronchopleural fistula.
Breakthrough of a large abscess into a bronchus or vigorous attempts to drain it can lead to massive bronchogenic dissemination. Erosion of blood vessels can cause severe bleeding. Occasionally, septic emboli migrate through the pulmonary veins into the arterial system and cause a secondary brain abscess. Rarer late complications include bronchiectasis and amyloid.
Classification. Currently, there is no single classification of destructive lung diseases that would meet both theoretical and practical requirements. According to Boyko V.V., Florikyan A.K. (2007) the working classification of acute purulent diseases of the lungs and pleura is as follows:
1. By pathogenesis: - post-pneumonic - - - - aspiration-obturation hematogenous-embolic traumatic lymphogenic
2. According to the nature of the process: - purulent abscess - - - gangrenous abscess widespread gangrene pyopneumothorax
3. According to the localization of the process: - a segment of the lung - - a portion of the lung, the whole lung
4. According to the prevalence of the process: - single abscesses - - multiple abscesses bilateral abscesses mild
5. According to the degree of severity: - - - of medium severity, severe
6. According to the presence of complications: - uncomplicated - complicated: pulmonary bleeding, pleural empyema (limited, total), pyopneumothorax (limited, total, intense), sepsis (septicemia).
Clinical manifestations of AL. In the course of acute infectious destruction of the lungs, two periods are conventionally distinguished, which differ significantly in terms of clinical and laboratory manifestations.
1. The initial, infiltrative-necrotic period - from the moment of the abscess to its breakthrough into the draining bronchus.
2. The period of drainage of the abscess into the draining bronchus. The syndrome of general intoxication develops already at the beginning of the disease. General malaise, weakness, adynamia are increasing. The body temperature rises to 39 C or more, chills appear. Sometimes the symptoms of the initial period are mild - without malaise, cough and shortness of breath, with a subfebrile temperature. "Smeared beginning" can be caused either

by the reduced immune reactivity of the patient (diabetes mellitus, alcoholism), or by the primary anaerobic nature of the pathogen. In both cases, the prognosis of the disease is severe.
Pleural syndrome is characteristic of subpleural abscess localization: sharp, sharp pains in the chest appear, which intensify with deep inhalation. The localization of pain corresponds to the localization of suppuration. When the basal segments are affected due to the involvement of the return nerve in the process, radiation of pain in the neck appears - the phrenic phenomenon.
Cough can be expressed in different degrees - from superficial weak to severe intense. An increase in the strength of the cough is often a harbinger of the breakthrough of the abscess into the bronchus, because the cough occurs as a result of irritation with the contents of the abscess and inflammatory exudate of the receptor zones of the bronchial wall.
Shortness of breath is caused both by the exclusion of a significant amount of lung tissue from the respiratory process and by a decrease in lung ventilation - the depth of breathing is significantly reduced, as a deep breath causes pain. The disease, as a rule, occurs against the background of one- or two-sided pneumonia, most often of aspiration origin or influenza.
The clinical picture in the stage of formation of a purulent cavity in the lung is determined by the dominance of symptoms of purulent-resorptive fever, which is associated with suppuration, resorption of the products of tissue decay and microbial life, and significant protein loss. During this period, patients have a high, sometimes hectic temperature, chills, increased sweating, signs of intoxication, often bother with cough, chest pain. Sputum is purulent, unless the abscess is completely isolated from the bronchi. Sometimes there are veins of blood. Chest pain indicates pleural damage. Clinical manifestations of GL. The beginning of the disease is characterized by hectic fever, chills, profuse sweating, intoxication. There is pain when coughing, sputum with an unpleasant smell, the breath becomes smelly. A large zone of dullness and weakening of breathing is determined in the lungs. In the blood, high leukocytosis often changes to leukopenia with the appearance of young forms of leukocytes, anemia progresses. Symptoms of purulent-resorptive exhaustion, septic condition develop very quickly.
Physical examination. With AL, a small area of shortening of the percussion sound and weakened breathing, small or medium-bubble wheezes are possible. If the cavity is large, tympanitis and amphoric breathing may occur. If the abscess perforates the bronchus wall, a large amount of purulent, sometimes foul-smelling sputum is coughed up within several hours or days, often with gangrenous lung tissue. Fever, lack of appetite, weakness, inability to work are characteristic. Pronounced losses of protein and electrolytes in the acute phase of inflammation, provided their insufficient compensation, lead to volemic and water-electrolyte disorders and to a decrease in muscle mass of patients. At the same time, swelling of the lower extremities may occur. Against the background of progressive hypoproteinemia, patients lose weight and become exhausted. High temperature is replaced by low fever or normalizes, which is a prognostically unfavorable sign, as it indicates a sharp decrease in the body's reactivity. If the abscess becomes chronic, weight loss, anemia, and hypertrophic pulmonary osteoarthropathy may occur. Physical examination of the chest in this chronic phase sometimes does not reveal any deviations from the norm, but in most cases dry and moist rales are heard. With a severe course, when the decay and suppuration of lung tissue continues, against the background of increased intoxication, functional disorders occur on the

part of the cardiovascular system, liver and kidneys. As the disease progresses, organic changes in internal organs can occur, which are characteristic of a septic state.
Data of additional research methods for AL.
1. Hemogram. Changes in the hemogram in the acute period are typical for severe inflammatory processes - leukocytosis with a pronounced shift of the leukocyte formula to the left. In patients with reduced immune reactivity, especially those who have suffered the flu, with the anaerobic nature of the process, leukopenia is often observed, which is an unfavorable prognostic sign. The shift of the leukocyte formula to the left towards myelocytes, promyelocytes exactly corresponds to the severity of the process, is a reliable criterion of the dynamics of the patient's condition. Relative and absolute lymphopenia is characteristic of a severe course of the process. Hypochromic anemia very often accompanies pulmonary destruction, especially of a gangrenous nature.
2. Biochemical research. Violation of protein metabolism is manifested by a decrease in the content of total protein, especially plasma albumins. Hypoproteinemia is caused by protein loss with pus and liver dysfunction. The level of α-globulins (glycoproteins, protein indicators of the "acute phase" of inflammation) and γ-globulins (immunoglobulins and immune complexes) increases. The ratio between the main fractions of blood serum proteins changes, the albumin-globulin ratio decreases. "acute-phase" concentration of glycoproteins, components of the main substance of connective tissue, indicators of "ceruloplasmin, haptoglobin" increases. - sialic acids, C-protein, mucin,
3. Examination of urine. Changes in the urine may appear when "toxic kidney" occurs - moderate proteinuria (selective albuminuria), cylindruria, micropotheinuria. A decrease in the excretory function of the kidneys is manifested by isosthenuria, a decrease in diuresis, and an increase in the serum creatinine level.
4. Examination of sputum. The study of sputum is of great importance for the diagnosis of destructive pulmonitis and control of the dynamics of the process. All sputum is collected every day and its quantity and organoleptic properties are recorded - putrid, purulent, with an admixture of blood. When defending, the sputum of patients with abscesses is divided into 3 layers. The upper layer is foamy - slime with impurities of manure. The middle layer is an opaque viscous liquid, a mixture of saliva and serous liquid. The lower layer is a mixture of crumbly tissue detritus, half-decomposed fragments of lung tissue (Dietrich plugs) and a large amount of manure. The lower layer is heterogeneous, consisting of white-yellow-grayish-brown fragments. Microscopy of sputum sediment reveals leukocytosis, fragments of lung tissue, elastic fibers, crystals of cholesterol, fatty acids, and hematoidin.
5. Bacteriological research. Bacteriological studies are carried out to identify the causative agent of pulmonary suppuration. Bacterioscopy of a sputum smear with preliminary staining by various means allows identification of various microorganisms by their morphological features. The method is accurate, but not sensitive enough. Sowing sputum on various nutrient media for cultivation allows establishing the etiology of suppuration. A quantitative method of sputum research is used to differentiate oral microflora contamination: only microorganisms whose concentration exceeds 10 million microbial bodies in 1 ml of sputum or 10,000 microbial bodies in 1 ml of bronchial tree lavage are considered etiologically significant. The most reliable information about the "microbial landscape" of suppuration is obtained by examining a punctate or aspirate from the area of inflammatory infiltration.

Cultivation of non-spore-forming anaerobic microorganisms has a number of difficulties. The material must be collected under anaerobic conditions (direct puncture of the focus, puncture of the trachea with a special airless syringe), stored "under oil" in a sealed tube for no more than 2 hours. Sowing is carried out on special elective media, cultivation must be carried out in strictly anaerobic conditions. A technically simpler and fairly reliable method of gas-liquid chromatography of manure - the type of pathogen is determined by the composition of anaerobic metabolites (fatty acids).

7. X-ray examination. It is possible to suspect the beginning of an abscess in a patient with severe pneumonia on the basis of changes in the clinical picture and physical data, but the main role in the diagnosis of lung abscesses is played by X-ray examination, which is preferably performed in the vertical position of the patient. In the early period of the disease, a segmental or lobular induration can be seen on chest x-rays, which becomes spherical due to stretching by pus. The appearance of one or more clearings against the background of homogeneous darkening in the lungs indicates the formation of solitary or multiple abscesses. In the future, multiple small cavities can merge into larger ones, in which, after the breakthrough of the abscess into the bronchus and coughing up sputum, fluid levels begin to be determined.

8. Computed tomography of the lungs, computerized abscessography. Additional information about the number and localization of abscesses, as well as the appearance of accompanying pleural effusion, which is poorly visible on radiographs, can be obtained from computer tomography of the lungs. Conventional and computerized abscessography are also used - transthoracic injection of water-soluble radiopaque drugs into the destruction cavity. The location of the purulent focus near the chest wall allows its puncture under ultrasound control.

9. Thoracoscopy. In the presence of empyema of the pleura or pneumothorax, a thoracoscopy is performed, which often allows to detect the presence of bronchopleural fistulas, their location and size, to perform a biopsy of the pleura or lungs to clarify the etiology of the disease. Data of additional research methods at GL. Diagnosis of lung gangrene is based on the features of clinical and radiological manifestations. Lung gangrene on X-rays is characterized by inflammatory infiltration and darkening of the lung tissue, which does not have clear boundaries and covers more than one lobe, often the entire lung. Sometimes, clearings of different calibers are detected at the site of foci of decay, in the largest of them, areas of necrotic tissue can be visualized - pulmonary sequestrations. The rapid addition of X-ray signs of pleural effusion, and then pyopneumothorax, is characteristic.

Complications of lung abscess include pleural empyema, pyopneumothorax, pulmonary hemorrhage, and septic condition. The consequence of an acute lung abscess, in addition to complete recovery with scarring (obliteration) of the cavity, can be the so-called "clinical recovery" with cleaning of the cavity, which is well drained through the bronchus, its stabilization and transformation into an air cyst. With small sizes, such a cyst may be asymptomatic, but under unfavorable circumstances (infection, impaired drainage), fluid may appear in it and recurrence of suppuration may occur. Chronic lung abscess. The main criterion for the chronic course of a lung abscess is the disappearance of acute suppuration while preserving the cavity itself against the background of the development of pneumosclerosis. The main reasons for the transition of an acute abscess into a chronic form are the presence of sequestrations in the cavity and its insufficient drainage through the

bronchi. Perhaps the features of the microflora and the reactivity of the macroorganism are also important. Symptoms depend on the phase of the clinical course of the disease - exacerbation or remission. During remission, the manifestations of the disease are minimal. Exacerbation can be triggered by a viral infection, hypothermia, overwork, stress. In patients, body temperature rises, cough intensifies, shortness of breath, chest pain, and malaise appear. The amount of sputum increases, which acquires an unpleasant smell. It is often accompanied by hemoptysis. With a long course of the disease, patients develop hypoproteinemia, signs of chronic hypoxia and intoxication appear (thickening of the nail phalanges in the form of "drumsticks", nails in the form of watch glasses). X-rays show one or more intrapulmonary cavities with thick walls, uneven contours, empty or with liquid (in case of exacerbation), surrounded by a zone of pneumosclerosis. Bronchi visualized by bronchography or CT in pathologically changed areas of the lungs are deformed, their lumens are unevenly narrowed or widened. As for complications, in addition to local (pulmonary) complications, extrapulmonary ones are also characteristic of chronic abscess: amyloidosis of kidneys and other organs, septicopyemia, subcutaneous and intermuscular emphysema, mediastinal emphysema, pulmonary heart failure. Abscesses in the kidneys, liver, adrenal glands, and brain are possible when pus gets into the vessels of the large blood circulation.

Bronchiectatic disease (BEC) is an acquired (and in some cases congenital) disease characterized by a localized chronic purulent process in irreversibly changed (dilated and deformed) and functionally defective bronchi, mainly in the lower parts of the lungs.
Topicality. The so-called primary bronchiectasis, as a rule, arise as a result of the transfer of acute infectious diseases of the bronchopulmonary system in childhood. Secondary bronchiectasis occur as a complication or manifestation of another disease. According to the distribution of bronchiectasis, they are found in 5% of the sectional material. Among patients with BEH, 60-65% are men.
Etiology and pathogenesis. A decisive role in the occurrence of bronchiectasis is played by the combination of the action of pathogens and genetic deficiency of the bronchial tree. In the pathogenesis of BEH, an important role is played by impaired patency of large (lobar, segmental) bronchi, which lead to the deterioration of their drainage function, retention of secretions, and the formation of obturational atelectasis. Obturation of the bronchus and retention of bronchial secretions lead to the development of a purulent process. Gradually, the wall of the bronchial tubes undergoes a remodeling of the mucous membrane with complete or partial death of the ciliated epithelium, which provides bronchial drainage, degeneration of cartilaginous plates, smooth muscles with their replacement by fibrous tissue. A decrease in the resistance of the bronchial walls to an increase in endobronchial pressure due to coughing, stretching by accumulating secretions, an increase in intrapleural pressure due to a decrease in the volume of an atelectazed lung particle leads to a persistent expansion of the lumen of the bronchi. An important pathogenetic role in the formation of bronchiectasis is played by impaired patency of smaller bronchi located more distally from the expansions that have formed. The degree and nature of obturation of these bronchi can be accompanied by the development of atelectasis, emphysema of the lungs. The links of the pathogenesis of BEH are diseases of the upper respiratory tract (tonsillitis, sinusitis, adenoids), which are a constant source of infection of the respiratory tract, expiratory

stenosis of the bronchi and trachea, and impaired pulmonary circulation.
Cylindrical, bag-shaped, spindle-shaped and mixed bronchiectasis can be distinguished by the form of bronchial expansion, which can be unilateral or bilateral.
Clinical manifestations. Patients with BEH complain of a wet cough with secretion of mucous or mucous-purulent sputum, the amount of which gradually increases to 200-500 ml per day. Sputum is released mainly in the morning, with a "full mouth", has an unpleasant smell, may be accompanied by hemoptysis or pulmonary bleeding. In the case of persistent sputum, it is divided into three layers: the upper one is foamy, the middle one is a mixture of yellow-green pus and brown blood, and the lower one is a crumbly mass, which is a fragment of destroyed lung tissue. In patients, body weight decreases, there is no appetite, body temperature rises, especially with delayed sputum production, sweating, shortness of breath, chest pain may occur. Along with the described typical form, there is a special "dry" form of bronchiectasis, which is characterized by the presence of a single symptom - periodic hemoptysis. Physical examination reveals paleness of the skin and cyanosis, fingers in the form of "drumsticks" and nails in the form of "watch glasses", during percussion over the affected area there is a dulling of sound, during auscultation - constant small and medium-bubble rales over the lower parts of the lungs.
Data of additional research methods.
1. Clinical blood analysis. Leukocytosis with rod-nuclear shift, increased ESR, anemia are observed.
2. Biochemical analysis of blood. The content of sialic acids and seromucoids in the serum increases, SRP appears.
3. Clinical analysis of urine. Urine analysis may be unchanged, but with the development of kidney complications, proteinuria occurs.
4. In the clinical analysis of sputum, erythrocytes, leukocytes, detritus, Dietrich plugs ("casts" of bronchi containing cholesterol, soap, elastic fibers), elastic and collagen fibers are found.
5. Bacterioscopy of sputum reveals a significant number of microbial bodies.
6. When examining the function of external breathing, signs of obstructive-type disorders are revealed, and in the case of a widespread process, a decrease in the LV.
7. On the examination X-ray, focal deformation of the lung pattern, cyst-like clearings are revealed, on the tomograms - areas of carnification, thin-walled cavities, cylindrical expansion of the bronchi. The pathology of the regional bronchi is visualized on bronchograms, and the type of atelectasis is specified.
8. Bronchoscopy is important for assessing the severity of suppuration (endobronchitis), for dynamic monitoring of the course of the process, for endobronchial rehabilitation.
9. Angiopulmonography helps to determine anatomical changes in the vessels of the lungs, to detect hemodynamic disturbances in the small circle of blood circulation.
10. Bronchial arteriography makes it possible to detect blood shunting through bronchial-pulmonary anastomoses.
11. Scanning of the lungs allows detecting a violation of capillary blood flow.

Respiratory failure. Respiratory failure (RD) is a syndrome caused by the inability of the external respiratory system to ensure the normal gas composition of arterial blood. DN also occurs in cases where maintenance of the normal gaseous composition of arterial blood at an

adequate level is achieved due to excessive functional stress of this system. DN can be acute (GDN) and chronic. GDN develops in a short period of time: from several hours to several days.
The main reasons for the development of GDN are sharp pain caused by trauma or surgery; violation of the condition and mobility of the diaphragm; violation of the central mechanisms of breathing regulation in case of injuries and diseases of the brain; violation of tracheobronchial patency, obstruction of the respiratory tract; reduction of the functioning lung surface; circulatory disorders in the small circle - shunting, the development of the so-called shock lung, thromboembolism of the branches of the pulmonary arteries; acute heart failure that leads to pulmonary edema; pulmonary edema with infusion overload, reduction of plasma oncotic pressure, increased permeability of alveolar-capillary membranes.
There are two types of GDN: 1) without retention of carbon dioxide (low PaO2 at low or normal PaCO2). It develops in patients with respiratory distress syndrome in adults, pneumonia of viral or bacterial origin, aspiration pneumonia, fat embolism of branches of the pulmonary artery, pulmonary edema as a result of severe ventilation and perfusion disorders and intrapulmonary shunting; 2) with retention of carbon dioxide (low PaO2 with increased PaCO2). Contains two types of disorders: ventilation-perfusion imbalance and inadequate alveolar ventilation. Patients with type 2 chronic obstructive pulmonary disease, in turn, are divided into two categories: a) with the presence of chronic obstructive pulmonary diseases with the additional influence of infection; b) inadequate lung ventilation is caused by extrapulmonary causes: - violation of breathing control (drug overdose, CNS disease, trauma, stroke); - neuromuscular disorders (polio, myasthenia, Guillain-Barré syndrome); - chest injury.
The assessment of the severity of GDN is mainly based on the indicators of the partial pressure of oxygen and carbon dioxide in the arterial blood
Clinical manifestations.
Signs of GDN: shortness of breath, tachypnea, cyanosis (absent with hemorrhage and anemia), tachycardia, excitement, then dizziness, loss of consciousness, increased moisture of the skin, movements of the wings of the nose, inclusion of auxiliary muscles in breathing. As GDN progresses, hypertension turns into hypotension, bradycardia, arrhythmia often develop, and patients die in the event of cardiovascular failure.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:
1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking workbooks, communicating with a patient with dry and exudative pleurisy, lung abscess and gangrene, bronchiectasis, lung cancer, respiratory failure for the purpose of collecting complaints and anamnesis, general

and local examination, chest palpation, comparative and topographic percussion of the lungs, auscultation of the lungs, assessment of clinical examination data and laboratory-instrumental data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.
2.1. Requirements for the theoretical readiness of applicants to perform practical classes: the applicant must know the etiology of pleurisy, subjective symptoms of dry and exudative pleurisy, objective symptoms of dry and exudative pleurisy

to know the physical, biochemical, morphological characteristics of pleural exudate, the method of collecting instruments for pleural puncture and the technique of pleurocentesis, the main complications of pleurocentesis.

to know the etiology, pathogenesis, clinical manifestations of abscess, lung gangrene, bronchiectasis disease, laboratory, X-ray and instrumental data in this pathology.

List of didactic units:
- to conduct a subjective examination of patients with pleurisy.
- conduct an objective examination of patients with pleurisy.
- to assess X-ray changes in patients with pleurisy.
- to evaluate the laboratory indicators of pleural exudate.
- collect a set of tools and prepare the patient for pleural puncture
- perform a pleural puncture
- master the skills of clinical examination of patients with abscess, lung gangrene, bronchiectasis.
- master the skills and abilities of evaluating clinical data and data of diagnostic studies in these diseases.

2.2. Questions to check basic knowledge on the topic of the lesson:
Questions to test basic knowledge

1. Definition of dry and exudative pleurisy.
2. Classification of dry and exudative pleurisy.
3. Etiology and pathogenesis of abscess of dry and exudative pleurisy.
4. Conducting subjective research:
a. Clarification of complaints.
b. Collection of analysis.
5. The value of methods of objective research of respiratory organs at diagnosis of dry and exudative pleurisy.
a. General overview.
b. Examination of the chest.
V. Palpation of the chest.
Mr. Lung percussion.
d. Auscultation of the lungs.

6. To evaluate the physical and macroscopic properties of pleural exudate.

7. Definition of lung abscess, lung gangrene, bronchiectasis.

8. Classification of lung abscess, lung gangrene, bronchiectasis.

9. Etiology and pathogenesis of lung abscess, lung gangrene, bronchiectasis.

10. Conducting subjective research:

a. Clarification of complaints.

b. Collection of analysis.

11. The value of methods of objective research of respiratory organs at diagnosis of lung abscess, lung gangrene, bronchiectasis.

a. General overview.

12. Examination of the chest.

V. Palpation of the chest.

Mr. Lung percussion.

d. Auscultation of the lungs.

b. The importance of laboratory, instrumental and X-ray methods examination for the diagnosis of purulent lung diseases.

Tests to check basic knowledge

3. Formation of professional skills and abilities:

mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the interview)

formation of the ability to perform a clinical examination of a patient with kidney pathology (the applicant must be able to perform a physical examination of a patient for dry and exudative pleurisy, lung abscess and gangrene, bronchiectasis, lung cancer, respiratory failure),

formation of the ability to evaluate the data obtained during the clinical examination of a patient with dry and exudative pleurisy, lung abscess and gangrene, bronchiectasis, lung cancer, respiratory failure; carry out a clinical interpretation of the main symptoms and syndromes in kidney diseases

formation of the ability to conduct a modern laboratory-instrumental examination of a patient with dry and exudative pleurisy, lung abscess and gangrene, bronchiectasis, lung cancer, respiratory failure (the applicant must be able to prescribe a plan for laboratory-instrumental examination, give an assessment of the examination results)

3.1. Control materials for the final stage of the lesson:

1. Conduct a subjective examination of a patient with dry pleurisy.

2. Conduct an objective examination of a patient with dry pleurisy.

1. Conduct a subjective examination of a patient with exudative pleurisy.

2. Conduct an objective examination of a patient with exudative pleurisy.
3. Define the Damoiseau line.
4. Define the Garland triangle.
5. Define Rauchfuss-Grocco triangle.
6. Determine the indications for pleural puncture.
7. 3 choose instruments for pleural puncture.
10. Prepare the patient for pleural puncture.
11. Assist the doctor during pleural puncture.

Situational tasks:

Tasks of the STEP-2 type

1. After determining the lower border of the right lung, the doctor found that it is 2 cm along all topographic lines. above the norm.
What additional research should be done?
*A - X-ray
In - Bronchophonia
C - Bronchography
D - Biopsy
E - X-ray

2. The patient complains of a painful cough with discharge of up to 600 ml of purulent chocolate-colored sputum with phlegm. He was acutely ill, temperature 390 C, fever of the wrong type. On the X-ray, an area of darkening with a cavity in the center, with irregular contours and a fluid level.
What disease are we talking about?
A. Lung abscess.
B. Tuberculosis.
S. Bronchiectatic disease.
+D. Lung gangrene.
E. Abscessing pneumonia.

3. The disease began suddenly with a fever up to 390C, the wrong type, sharp pains in the chest. The sputum is putrid with an admixture of blood, the presence of a rotten tooth, up to 400 ml per day. When percussing over the affected area, the sound is shortened, voice tremor is increased. Anaerobic streptococcus was isolated in the sputum.
What disease are we talking about?
A. Lung abscess.
+V. Lung gangrene.
S. Tuberculosis of the lungs.
D. Bronchiectatic disease.
E. Abscessing pneumonia.

4. The patient is 47 years old and has been smoking regularly since his youth. Objectively: he

is thin, the chest is cylindrical, the intercostal spaces retract during breathing with a frequency of 28 in 1 minute, when he coughs, he secretes 2 liters of sputum.
What research method is the most expedient to perform to clarify the diagnosis?
A. Collection of anamnesis.
B. Computed tomography.
S. Bronchography.
+D. Roentgenoscopy of the lungs.
E. Percussion.

5. The patient is 47 years old and has been smoking regularly since his youth. Objectively: he is thin, the chest is cylindrical, the intercostal spaces retract during breathing with a frequency of 28 in 1 minute, when he coughs, he secretes 2 liters of sputum.
What is the nature of sputum in this patient?
A. Slime.
V. Gnylnna.
+S. Purulent
D. Muco-purulent.
E. Krovyanista.

6. The patient is 46 years old. He fell ill acutely: chills appeared, cough, first dry, then with mucous-purulent sputum, pain in the right side, temperature 39.8 0 C. On the 12th day of the illness, chills appeared, the temperature rose to 400C, there was about 1.5 liters of sputum. Objectively: the general condition is severe, acrocyanosis. Percussion in the region of the upper lobe of the right lung dullness, in the center of which is tympanitis. Auscultatively in this area, bronchial breath, wet sonorous larynx rales.
Your diagnosis?
A. Acute bronchitis.
+V. Lung abscess.
S. Acute pneumonia.
D. Bronchiectatic disease.
E. Dry pleurisy.

Practical lesson No. 27
Topic: Symptoms and syndromes in aortic heart defects.

Goal: Acquiring knowledge and mastering professional competences during the examination of a patient with aortic heart defects - questioning, physical examination, instrumental research.

Basic concepts: 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.
1. Aortic valve insufficiency syndrome.
1.1. Causes of aortic valve insufficiency.
1.2. Hemodynamic disorders in the presence of aortic valve insufficiency.
1.3. Complaints of patients with aortic valve insufficiency syndrome.
1.4. Data of a physical examination of a patient with aortic 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. 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).

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

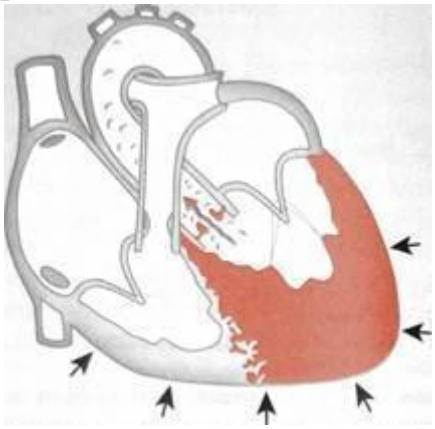


Fig. 3.8.*Aortic stenosis*
insufficiency of the bicuspid valve (moralization of the defect). In the small circle of blood circulation, the filling of venous vessels with blood increases. Next, right ventricular failure develops (stage of AC decompensation).
Classification
Stages of AC: I – complete compensation, 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

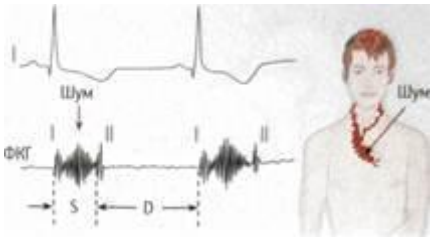


Fig. 3.9.*Systolic murmur in aortic stenosis*

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).

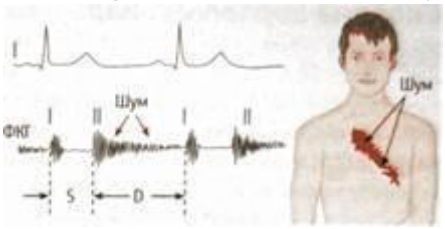


Fig. 3.11.*Diastolic murmur in aortic insufficiency*

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.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:

1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with a patient with aortic heart disease in order to collect complaints and history, general and local examination, palpation of the heart area, percussive determination of the limits of relative and absolute heart dullness, auscultation of the heart and blood vessels, pulse examination and measurement of blood pressure, assessment of clinical examination data and laboratory-instrumental data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for the theoretical readiness of applicants to perform practical classes: the applicant must know the modern definition, etiology, pathogenesis, hemodynamics, classifications of aortic heart defects, subjective and objective data in these diseases to know instrumental (x-ray, ECG, ECHO-CG) data for this pathology.

List of didactic units:

- to conduct a subjective examination of patients with aortic heart defects
- conduct an objective examination of patients with aortic heart defects
- to assess X-ray changes in patients with aortic heart disease
- evaluate ECG, echocardiogram data in patients with aortic heart defects
- master the skills and abilities to assess leading clinical syndromes in patients with aortic heart defects

2.2. Questions to check basic knowledge on the topic of the lesson:

1. Etiology of acquired defects of the aortic valve?
2. What intracardiac hemodynamic disorders do you know in aortic insufficiency?
3. What intracardiac hemodynamic disorders do you know in aortic stenosis?
4. What additional data can be obtained with the help of research tools?

3. Formation of professional skills and abilities:

mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the interview)

formation of the ability to perform a clinical examination of a patient with aortic heart defects (the applicant must be able to perform a physical examination of the patient),

formation of the ability to evaluate the data obtained during the clinical examination of a patient with aortic heart defects; carry out a clinical interpretation of the main symptoms and syndromes in these diseases

formation of the ability to conduct a modern laboratory-instrumental examination of a patient with aortic heart disease (the applicant must be able to prescribe a plan for a laboratory-instrumental examination, give an assessment of the examination results)

3.1. Control materials for the final stage of the lesson:

1. Diagnostic value of ECG, echoscopy, chest X-ray.
2. Etiology of aortic valve damage.
3. Intracardiac hemodynamics in aortic stenosis, aortic valve insufficiency.
4. The main symptoms of the disease.
5. Methods and study scheme of a patient with aortic stenosis, aortic valve insufficiency.
6. General method of questioning patients with aortic valve damage.
7. Diagnostic value of ECG, echoscopy, chest X-ray.
8. Characteristics and mechanisms of the main complaints in patients with aortic valve damage.
9. Course of defects, development of signs of heart failure and possible complications in aortic heart defects.
10. Value of general and local examination of a patient with aortic valve defects.
11. Investigate a patient with an aortic valve lesion.
12. Perform a general and local examination of a patient with aortic valve defects.
13. 3 to take an anamnesis and conduct a subjective examination of the patient.
14. Conduct an objective examination of the patient: examination, palpation, auscultation of the heart, pulse examination and blood pressure measurement.
15. Evaluate changes in the ECG, echocardioscopy, X-ray of the heart.

16. Give a clinical assessment of the results obtained during the examination of a patient with aortic valve defects.

Situational tasks:

Tasks of the STEP-2 type

1. A 50-year-old woman complains of pronounced general weakness, shortness of breath with slight physical exertion, pain in the region of the heart of a constricting nature. He has been sick for 2 years. Objectively: the skin is pale. The apical impulse is palpated in the 6th intercostal space. During auscultation in the region of the tip of the I tone is weakened, the II tone over the aorta is weakened, a coarse systolic noise is heard. Pulse 64 in 1 minute, tardus et parvus. Blood pressure - 110/90 mm Hg. What vice should you think about?
A. Obstruction of the botal duct.
+V. Stenosis of the mouth of the aorta.
C Non-occlusion of the interventricular septum.
D. Insufficiency of the mitral valve.
E. Insufficiency of the tricuspid valve.

2. A 30-year-old patient complains of shortness of breath during physical exertion, palpitations and pain in the region of the heart, occasional loss of consciousness. Objectively: "heart hump" in the area of the left half of the chest. An apical thrust in the 5th intercostal space outwards from the mid-clavicular line. During palpation in the region of the base of the heart "cat's purring". Auscultatory systolic murmur on the aorta. What vice should you think about?
A. Non-occlusion of the interventricular septum.
B. Mitral stenosis.
+S. Stenosis of the mouth of the aorta.
D. Insufficiency of aortic valves.
E. Stenosis of the mouth of the pulmonary artery.

3. An 18-year-old girl complains of an increase in body temperature to 380C over the past 2 months. Recently, shortness of breath during physical exertion, palpitations, pain in the heart area appeared. Objectively: pale skin, pulsation of carotid arteries. The apical impulse is diffuse, localized in the 5th intercostal space. On auscultation, the II tone is weakened in the aorta, in Botkin's vein and in the II intercostal space to the right of the sternum, there is a protodiastolic murmur. What vice should you think about?
A. Insufficiency of the mitral valve.
+V. Insufficiency of aortic valves.
C. Stenosis of the mouth of the aorta.
D. Insufficiency of pulmonary artery valves.
E. Mitral stenosis.

4. A 40-year-old man has been suffering from rheumatism for 15 years. Lately, pronounced shortness of breath and palpitations during physical exertion, pain in the heart region have appeared. Objectively: "dance of the carotid", spilled and apical thrust, which lifts, that is

shifted to the left and below the 6th intercostal space along the mid-clavicular line. A systolic murmur and a soft diastolic murmur are heard in the II intercostal space to the right of the sternum. X-ray aortic configuration of the heart. What vice should you think about?

A. Insufficiency of the mitral valve.
B. Insufficiency of aortic valves.
C. Stenosis of the mouth of the aorta.
+D. Combined aortic malformation.
E. Mitral stenosis.
Question
1. Aortic heart defects - classification, etiology.
2. Insufficiency of the aortic valve - indicate hemodynamic disorders, main symptoms, vascular phenomena.
3. Insufficiency of the aortic valve indicate the data of palpation, percussion and auscultation of the heart.
4. Aortic stenosis (stenosis of the mouth of the aorta) - indicate hemodynamic disorders, main complaints, symptoms.
5. Aortic stenosis - indicate the data of palpation, percussion and auscultation of the heart

3.2. Requirements for work results, including before registration: substantiation of the diagnosis based on complaints, medical history and life data, clinical and laboratory-instrumental examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (*if necessary*).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

List of recommended literature
Basic:

1. Propedeutics of internal medicine: textbook / Yu.I. Decyk, O.H. Yavorskyi, E.M. Neiko and others; under the editorship O.H. Yavorsky – 6th ed., corr. and added - K.: VSV "Medicine", 2020. - 552 p. + 12 p. color incl.

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.

Additional:

1. Methodology of examination of a therapeutic patient: teaching. manual / S.M. Andreychyn, N.A. Bilkevich, T.Yu. Chernets. – Ternopil: TDMU, 2016. – 260 p.

2. 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.

3. Yepishyn A.V. Propaedeutics of internal diseases with care of therapeutic patients / AV. Yepishyn// K. - 2015. 768p.

4. Kovalova OM. Propedeutics of internal medicine/ OM. Kovalova, NA Safargalina-Kornilova// K.: Medicine 2010 - 750p.

5.Macleod's Clinical Examination / Ed. G.Douglas, F.Nicol, C.Robertson.– 13th ed.– Elsevier. 2013. – 471 p.

6.Bates' Guide to Physical Examination and History Taking /Ed. Lynn S. Bickley, Peter G. Szilagyi. – Wolters Kluwer, 2017. – 1066 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/ - Odesa National Medical University
9. https://onmedu.edu.ua/kafedra/propedevtiki-vnutrishnih-hvorob-ta-terapii/ - Department of propaedeutics of internal diseases and therapyOdessa National Medical University

Practical lesson No. 28

Topic: Mitral heart defects. Main symptoms and syndromes based on clinical and instrumental examination methods.

Goal: Acquiring knowledge and mastering professional competences during the examination of a patient with mitral heart defects - questioning, physical examination, instrumental research.

Basic concepts:

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.

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

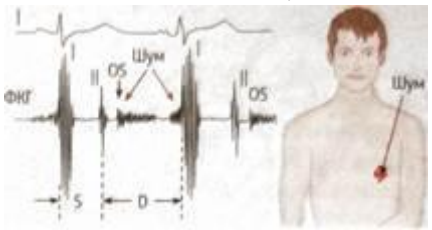


Fig. 3.5. *"Quail" rhythm and diastolic murmur in mitral stenosis*
night (EVS deviation to the right, increase in R waves in V1-V2 and S waves in V5-V6). On**FCG**diastolic noise, increased I tone, Q interval – I tone continues for more than 0.07 s (which indicates delayed closing of the mitral valve 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

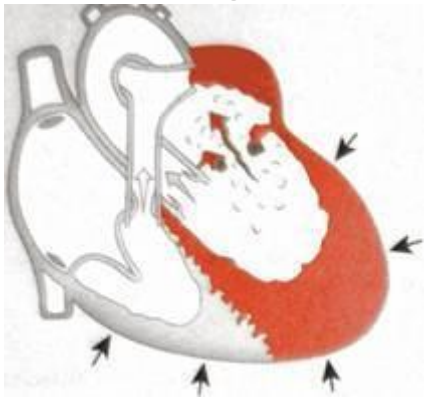


Fig. 3.6.*Mitral insufficiency*

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.
Plan:
1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with a patient with mitral valve disease in order to collect complaints and history, general and local examination, palpation of the heart area, percussive determination of the limits of relative and absolute cardiac dullness, auscultation of the heart and vessels, examination of the pulse and measurement of blood pressure, evaluation of clinical examination data and laboratory-instrumental data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.
2.1. Requirements for the theoretical readiness of applicants to perform practical classes: the applicant must know the modern definition, etiology, pathogenesis, hemodynamics, classifications of mitral heart defects, subjective and objective data in these diseases to know instrumental (x-ray, ECG, ECHO-CG) data for this pathology.

List of didactic units:
- to conduct a subjective examination of patients with mitral heart defects
- conduct an objective examination of patients with mitral heart defects
- to evaluate radiological changes in patients with mitral heart defects
- evaluate ECG, echocardiogram data in patients with mitral heart defects
- master the skills and abilities to assess leading clinical syndromes in patients with mitral heart defects

2.2. Questions to check basic knowledge on the topic of the lesson:

1. Etiology of acquired defects of the mitral valve?
2. What intracardiac hemodynamic disorders do you know with mitral insufficiency?
3. What intracardiac hemodynamic disorders do you know with mitral stenosis?
4. Complaints of patients with mitral valve insufficiency?
5. Complaints of patients with mitral stenosis?
6. What are the mechanisms of shortness of breath in mitral defects?
7. What borders of the heart are enlarged with mitral stenosis?
8. What borders of the heart are increased in mitral insufficiency?
9. Describe the apical palpitation in mitral stenosis?
10. Describe the apical palpitation in mitral insufficiency?
11. Give an auscultatory characteristic of mitral stenosis?
12. Give an auscultatory characteristic of mitral insufficiency? 13. Mechanism of occurrence of "cat's purring" in mitral stenosis?
14. What is the mechanism of heart failure in mitral heart defects?
15. Explain changes in pulse and blood pressure in mitral heart defects?
16. What additional data can be obtained with the help of research tools?

Tests to check basic knowledge

1. During palpation, the apical impulse is normal, a "cat's purr" is detected, the upper and right limits of relative cardiac dullness are increased, and a diastolic murmur is heard. What kind of heart defect can you think of? Mitral stenosis.
2. During palpation, the apical impulse is strengthened, increases in the left, upper and right limits of relative cardiac dullness are determined, and a systolic murmur is heard. What kind of heart defect can you think of? Insufficiency of the mitral valve.

3. Formation of professional skills and abilities:

mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the interview)

formation of the ability to perform a clinical examination of a patient with mitral heart defects (the applicant must be able to perform a physical examination of the patient),

formation of the ability to evaluate the data obtained during the clinical examination of a patient with mitral heart defects; carry out a clinical interpretation of the main symptoms and syndromes in these diseases

formation of the ability to conduct a modern laboratory-instrumental examination of a patient with mitral heart defects (the applicant must be able to prescribe a plan for a laboratory-instrumental examination, give an assessment of the examination results)

3.1. Control materials for the final stage of the lesson:

1. Etiology of mitral valve damage.

2. Intracardiac hemodynamics in mitral stenosis, mitral valve insufficiency.
3. The main symptoms of the disease.
4. Methods and study scheme of a patient with mitral stenosis, mitral valve insufficiency.
5. General method of questioning patients with mitral valve damage.
6. Diagnostic value of ECG, echoscopy, chest X-ray.
7. Characteristics and mechanisms of the main complaints in patients with mitral valve damage (dyspnea, palpitations, orthopnea).
8. Course of defects, development of signs of heart failure and possible complications in mitral heart defects.
9. Value of general and local examination of a patient with mitral valve defects.
10. The main symptoms of the disease.
11. Diagnostic value of ECG, echoscopy, chest X-ray.
12. 3 to take an anamnesis and conduct a subjective examination of the patient.
13. Conduct an objective examination of the patient: examination, palpation, auscultation of the heart, pulse examination and blood pressure measurement.
14. Evaluate changes in the ECG, echocardioscopy, X-ray of the heart.

Situational tasks:

Tasks of the STEP-2 type

1. During auscultation of the apex of the heart of a 30-year-old patient, who is on the dispensary record in the polyclinic for rheumatism, the doctor heard a weakened first sound. What disease should you think about?
A. Stenosis of the mitral orifice.
+B. Insufficiency of the mitral valve.
C. Thyrotoxicosis.
D. Anemia.
E. Neuro-circulatory dystonia.

2. A 20-year-old patient has been suffering from rheumatism since he was 10 years old. He complains of palpitations, shortness of breath during exertion. When examined - "Facies mitralis". During auscultation at the apex of the heart, there is a loud "caressing" tone. What disease should we think about?
A. Insufficiency of the aortic valve.
B. Myocarditis.
+C. Stenosis of the mitral orifice.
D. Hypertensive disease.
E. Myocardial infarction.

3. During auscultation of the apex of the heart of a patient suffering from rheumatism, the doctor identified a "clapping" first sound. additional Ш tone /"opening pin" of the mitral valve/. What disease is characterized by a similar "over-sung" rhythm?
A. Hypertensive disease.
B. Myocarditis.
C. Exudative pericarditis.

+D. Stenosis of the mitral orifice,
E. Acute bronchitis.

4. In what disease can a presystolic gallop rhythm occur?
A. Dry pleurisy.
B. Dry pericarditis.
+C. Stenosis of the mitral orifice.
D. Chronic bronchitis.
E. Chronic glomerulonephritis.

5. A 26-year-old patient is bothered by pain in the right hypochondrium. During the examination of the patient, the presence of acrocyanosis, "heart hump", was revealed, and during palpation of the apex of the heart, a diastolic "cat's purr" was determined. Which of the following diagnoses is the most reliable?
A) Chronic hepatitis.
C) Acute cholecystitis.
C) Insufficiency of aortic valves.
+D) Mitral stenosis.
E) Myocarditis.

6. In a sick 15-year-old girl, an upward and rightward shift of the percussive borders of relative and absolute dullness of the heart was established during the examination. What previous diagnosis is most appropriate?
A) Myocardial infarction.
B) Myocarditis.
+C) Mitral stenosis.
D) Lung emphysema.
F) Anemia.

7. A 32-year-old female patient complains of pain in the heart area and a feeling of "interruptions". During the examination, the presence of atrial fibrillation with a pulse deficit of 15 beats per minute was established. It was found out that in childhood she often suffered from sore throats. What is the most reliable diagnosis?
+A) Mitral stenosis.
C) Aortic stenosis.
C) Myocarditis.
D) Vegeto-vascular dystonia.
E) Cardiosclerosis.

8. In a 27-year-old patient with mitral stenosis, a presystolic murmur was heard at the apex of the heart for five years. During the next examination, it was established that this noise had disappeared. What is the most likely cause of this condition?
A) Left atrial hypertrophy.
C) Complete atrio-ventricular blockade.
+C) Atrial fibrillation.

D) An increase in pressure in the small circle of blood circulation.
F) Insufficiency of the mitral valve.

9. Patient T., 25 years old, was brought to the reception department of the hospital with complaints of pain in the extrathoracic region, palpitations, shortness of breath. The examination revealed the presence of Popov's symptom. What is the cause of this symptom?
A) Dilation of the right pupil
C) Dilation of the left pupil.
+C) Less filling of the pulse on the left radial artery than on the right.
D) The appearance of pulsation of the jugular veins when pressing on the liver area.
E) Presence of Vynogradov-Durozier noise.

10. In a patient with an established diagnosis of mitral stenosis, an instrumental examination revealed a violation of cardiac hemodynamics, which indicates the appearance of a functional heart defect. What is the most likely complication for this patient?

A) Relative insufficiency of the bistable valve.
+B) Relative insufficiency of the tricuspid valve.
C) Relative stenosis of the mouth of the aorta.
D). Relative stenosis of the tricuspid opening.
E) Relative insufficiency of aortic valves.

3.2. Requirements for work results, including before registration: substantiation of the diagnosis based on complaints, medical history and life data, clinical and laboratory-instrumental examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (*if necessary*).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 29
Topic: Symptoms and syndromes in hypertension and secondary arterial hypertension. Hypertensive crises.

Goal: Acquiring knowledge and mastering professional competences during the examination of a patient with arterial hypertension and ischemic heart disease - questioning, physical examination, laboratory and instrumental studies.

Basic concepts: WHO/MTH definitions for arterial hypertension, essential hypertension (hypertensive disease) and symptomatic hypertension. The main risk factors of hypertension and mechanisms of its development. Classification of hypertensive disease according to blood pressure level and target organ damage. The main complaints of a hypertensive patient, examination data, palpation of the precardial region, percussion of the borders of cardiac dullness and auscultation of the heart. ECG signs of myocardial changes in hypertension. Symptomatic hypertension. Complicated and uncomplicated hypertensive crises.
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)

RE category	ssure	
	l < 80	
	l < 85	
ɔrmal	139 or 85-89	
ypertension		
	ɔr 90-99	
	ɔr 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.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:

1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with a patient with arterial hypertension and ischemic heart disease in order to collect complaints and history, general and local examination, palpation of the heart area, percussive determination of the limits of relative and absolute cardiac dullness, auscultation of the heart and blood vessels , pulse research and blood pressure measurement, evaluation of clinical examination data and laboratory-instrumental data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for the theoretical readiness of applicants to perform practical classes:

the applicant must know the modern definition, etiology, pathogenesis, classification of hypertension and symptomatic arterial hypertension, CHD (angina and myocardial infarction), subjective and objective data in these diseases
to know laboratory and instrumental data for this pathology.

List of didactic units:
- to conduct a subjective examination of patients with arterial hypertension and coronary artery disease
- conduct an objective examination of patients with arterial hypertension and coronary artery disease
- appoint a plan for additional laboratory and instrumental examinations of patients with arterial hypertension and coronary artery disease
- evaluate the results of laboratory tests in patients with arterial hypertension and coronary artery disease
- evaluate ECG, echocardiogram data in patients with arterial hypertension and coronary artery disease
- master the skills and abilities to assess leading clinical syndromes in patients with arterial hypertension and coronary artery disease

2.2. Questions to check basic knowledge on the topic of the lesson:
1.

3. Formation of professional skills and abilities:

mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the interview)

formation of the ability of clinical examination of a patient with arterial hypertension and coronary artery disease
(the applicant must be able to perform a physical examination of the patient),

formation of the ability to evaluate the data obtained during the clinical examination of a patient with arterial hypertension and coronary artery disease; carry out a clinical interpretation of the main symptoms and syndromes in these diseases

formation of the ability to conduct a modern laboratory-instrumental examination of a patient with arterial hypertension and coronary artery disease (the applicant must be able to prescribe a plan of laboratory-instrumental examination, give an assessment of the examination results)

3.1. Control materials for the final stage of the lesson:
1. Etiology and mechanism of development of arterial hypertension.
2. Risk factors of arterial hypertension.
3. Classifications of hypertension and symptomatic arterial hypertension.
4. Clinical picture of hypertension.
5. Complication of hypertensive disease.

6. Basic laboratory and instrumental methods of diagnosis of hypertension.

Situational tasks:

Tasks of the STEP-2 type

1. Patient M., 52 years old, complains of headache, palpitations. High BP for about 5 years. She suffered a large myocardial infarction a year ago, 6 months ago - a cerebral stroke. Objectively: the state of average weight. The left border of the heart is displaced outward by 2.5 cm. Accent of the II tone on the aorta, systolic murmur at the apex. The pulse is 96 per minute, non-rhythmic. Blood pressure - 190/105 mm Hg. Your diagnosis? 1. Stenosis of the mouth of the aorta;
2. Insufficiency of the aortic valve;
3. Coarctation of the aorta;
*4. Hypertensive disease of the III century;
5. Atherosclerosis of the aorta.

2. The patient is 70 years old and has been suffering from hypertension for more than 20 years. Woke up at night from a sudden attack of shortness of breath with difficulty breathing. Objectively: orthopnea. Pulse 108 bpm, rhythmic. Moist rales in the lower parts of the lungs. What complication of hypertension did the patient have?
1. Myocardial infarction;
2. Acute violation of cerebral circulation;
*3. Cardiac asthma;
4. Fainting;
5. Lung infarction.

3. A 70-year-old patient complains of shortness of breath, swollen legs, and has been suffering from hypertension for 10 years. Two years ago - myocardial infarction. Objectively: pulse 110 bpm, arrhythmic, deficit 20. Percussion: enlargement of the left ventricle of the heart. Blood pressure - 190/100 mmHg. The first tone is weakened, the accent of the second tone is over the aorta. In the lower parts of the lungs - small and medium alveolar moist rales. What brought the patient to the doctor?
1. Myocardial infarction;
2. Hypertensive crisis;
3. Lung edema;
4. Pneumonia;
*5. Heart failure.

3.2. Requirements for work results, including before registration: substantiation of the diagnosis based on complaints, medical history and life data, clinical and laboratory-instrumental examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (*if necessary*).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 30
Subject: Ischemic heart disease: symptoms and syndromes in angina pectoris and myocardial infarction.

Goal: Acquiring knowledge and mastering professional competences during the examination of a patient with coronary heart disease - questioning, physical examination, laboratory-instrumental studies.

Basic concepts:
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.

Stable angina pectoris

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.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:
1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with a patient with arterial hypertension and ischemic heart disease in order to collect complaints and history, general and local examination, palpation of the heart area, percussive determination of the limits of relative and absolute cardiac dullness, auscultation of the heart and blood vessels , pulse research and blood pressure measurement, evaluation of clinical examination data and laboratory-instrumental data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.
2.1. Requirements for the theoretical readiness of applicants to perform practical classes: the applicant must know the modern definition, etiology, pathogenesis, classification of hypertension and symptomatic arterial hypertension, CHD (angina and myocardial infarction), subjective and objective data in these diseases

to know laboratory and instrumental data for this pathology.

List of didactic units:
- to conduct a subjective examination of patients with arterial hypertension and coronary artery disease
- conduct an objective examination of patients with arterial hypertension and coronary artery disease
- appoint a plan for additional laboratory and instrumental examinations of patients with arterial hypertension and coronary artery disease
- evaluate the results of laboratory tests in patients with arterial hypertension and coronary artery disease
- evaluate ECG, echocardiogram data in patients with arterial hypertension and coronary artery disease
- master the skills and abilities to assess leading clinical syndromes in patients with arterial hypertension and coronary artery disease

2.2. Questions to check basic knowledge on the topic of the lesson:

1. Pathogenesis of coronary heart disease?
2. Pathogenesis of the development of myocardial infarction?
3. Complaints of patients with different functional classes of angina pectoris?
4. Complaints of patients with anginal form of myocardial infarction?
5. What is the mechanism of diastolic gallop rhythm in myocardial infarction?
6. What electrocardiographic signs do you know during an attack of angina pectoris?
7. What electrocardiogram will be outside an attack of angina pectoris?
10. What electrocardiographic signs of myocardial infarction do you know?
11. In which leads will there be changes on the electrocardiogram in case of posterior myocardial infarction?
12. In which leads will there be changes on the electrocardiogram with anterior lateral localization of myocardial infarction?
13. What laboratory data confirm the presence of an acute myocardial infarction?
14. What additional data can be obtained with the help of research tools?
15. What effect does alcohol abuse, tobacco smoking have on the development and course of hypertensive disease?

Tests to check basic knowledge

1. During palpation, the apical impulse is normal, a "cat's purr" is detected, the upper and right limits of relative cardiac dullness are increased, and a diastolic murmur is heard. What kind of heart defect can you think of? Mitral stenosis.
2. During palpation, the apical impulse is strengthened, increases in the left, upper and right limits of relative cardiac dullness are determined, and a systolic murmur is heard. What kind of heart defect can you think of? Insufficiency of the mitral valve.

3. Formation of professional skills and abilities:

mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the interview)
formation of the ability of clinical examination of a patient with arterial hypertension and coronary artery disease
(the applicant must be able to perform a physical examination of the patient),
formation of the ability to evaluate the data obtained during the clinical examination of a patient with arterial hypertension and coronary artery disease; carry out a clinical interpretation of the main symptoms and syndromes in these diseases

formation of the ability to conduct a modern laboratory-instrumental examination of a patient with arterial hypertension and coronary artery disease (the applicant must be able to prescribe a plan of laboratory-instrumental examination, give an assessment of the examination results)

3.1. Control materials for the final stage of the lesson:
1. Etiology and mechanism of development of arterial hypertension.
2. Risk factors of arterial hypertension.
3. Classifications of hypertension and symptomatic arterial hypertension.
4. Clinical picture of hypertension.
5. Complication of hypertensive disease.
6. Basic laboratory and instrumental methods of diagnosis of hypertension.
7. Etiology and mechanism of development of ischemic heart disease.
8. Classification of coronary heart disease, functional classes of angina pectoris.
9. Clinical picture of angina pectoris.
10. Basic laboratory and instrumental methods of diagnosing angina pectoris.
11. The influence of tobacco smoking and alcoholism on the course of coronary heart disease.
12. Etiology and mechanism of development of myocardial infarction.
13. Clinical picture of various variants of the course of myocardial infarction.
14. Laboratory diagnosis of myocardial infarction.
15. Main electrocardiographic changes in myocardial infarction.
10. Main complications of myocardial infarction, course of the disease.

Situational tasks:
Tasks of the STEP-2 type
1. The patient, 55 years old, complains of squeezing pains behind the sternum, which appear during physical exertion of low intensity (walking at a slow pace around the ward). The pain lasts for about 3-4 minutes and goes away on its own when physical activity is stopped (rest), or you are forced to take nitroglycerin, a tablet of which stops the pain in 2-3 minutes. He notes similar pains for 2 days. He is being treated in the infarct department for a Q-myocardial infarction that occurred 10 days ago. During the first 7 days of treatment, when the pain after the heart attack stopped, he did not notice such pains. On the ECG - signs of the acute stage of Q-myocardial infarction. Your previous diagnosis?
A) Repeated myocardial infarction
C) Stable angina pectoris of IV functional class
C) The first developed angina

*D) Postinfarction angina
E) Stable angina of functional class II

2. The patient, 55 years old, complains of a burning pain that first appeared behind the sternum after physical exertion (he lifted a 50 kg oxygen cylinder at work). The pain lasted about 15 minutes and went away on its own after stopping physical activity. The pain returned with less intensity when walking and is present at this time. The patient's ECG was recorded in the company's medical center. On the ECG, in leads J, II, aVL, V4-V6, there is an elevation of the S-T segment, which almost merges with the T wave, forming a "monophasic curve". Your diagnosis?
*A) Myocardial infarction
C) Stable angina pectoris of IV functional class
C) Angina occurred for the first time
D) Post-infarction angina
E) Stable angina P functional class

3. The patient, 68 years old, turned to the district doctor in the morning with complaints of shortness of breath that appeared during previously accessible physical exertion of low intensity (walking around the room), which she previously noted only when climbing stairs or uphill. The night before, she had an attack of inspiratory dyspnea, which stopped with orthopnea and taking 2 nitroglycerin tablets in 40-60 minutes. The patient has a history of type II diabetes for 10 years. On the ECG in leads V1-V4 there is a Q wave, 0.04 seconds wide, elevation of the S-T segment, which almost merges with the T wave; in leads III, aVF - reciprocal changes. Your diagnosis?
*A) Myocardial infarction
C) Stable angina pectoris of IV functional class
C) Angina occurred for the first time
B) Postinfarction angina
E) Stable angina of functional class II
7. The patient, who has been in the cardiology department for the 17th day due to a non-Q-myocardial infarction, began to complain of renewed attacks of angina pectoris. What research is more appropriate to do?
*A) Coronarography
B) Velergometry
C) Step test
D) EKG study at rest
E) Echocardioscopy

8. A bicycle ergometry was performed on a patient complaining of extrathoracic pain radiating to the left arm during high-intensity exercise. Upon reaching the submaximal load, the patient developed pain behind the sternum, during this period no changes on the ECG indicating myocardial ischemia were registered. What do the test results indicate?
A) Angina is confirmed
C) Exclude angina pectoris
*C) Do not exclude angina pectoris

D) Variant angina is confirmed
E) Variant angina is excluded

9. In a 54-year-old patient suffering from stable angina P of the functional class, anginal attacks intensified, lengthened and became more frequent, they began to appear in response to less physical activity. Your diagnosis?
A) Myocardial infarction
C) Stable angina pectoris of IV functional class
C) Stable angina pectoris of functional class
D) Stable angina P functional class
*E) Progressive angina pectoris

10. The patient, 54 years old, complains of squeezing pains behind the sternum, which appear during physical exertion of medium intensity, which was previously available (walking at an average pace of 600-700 meters, climbing stairs above the 4th floor). The pain lasts for about 4-5 minutes and goes away on its own when physical activity is stopped (rest). The pain appeared for the first time about 5-6 days ago, the patient had not noted such pain before. No pathological changes were detected on the ECG registered at rest.
Your previous diagnosis?
A) Myocarditis
B) Myocardial infarction
*C) Angina that occurred for the first time
D) Variant angina pectoris
E) Stable angina of functional class II

3.2. Requirements for work results, including before registration: substantiation of the diagnosis based on complaints, medical history and life data, clinical and laboratory-instrumental examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (*if necessary*).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 31.

Subject: Clinical-instrumental and laboratory research of patients with chronic gastritis, peptic ulcer disease of the stomach and duodenum. Main symptoms and syndromes. Tumors of the stomach.

Goal: Acquiring knowledge and mastering professional competences during the examination of a patient for chronic gastritis, peptic ulcer disease of the stomach and duodenum, chronic hepatitis and cirrhosis of the liver - questioning, physical examination, laboratory-instrumental studies.

Basic concepts: Definition and modern classification of gastritis and peptic ulcer of the stomach and duodenum. The main etiological factors of these diseases. Prevalence of helicobacteriosis, conditions of damage to the mucous membrane of the stomach and duodenum. The main complaints of patients with chronic gastritis and peptic ulcer. Peculiarities of the pain syndrome depending on the localization of the pathological focus and the state of the acid-producing function of the stomach. Manifestations of dyspeptic syndrome in chronic gastritis and peptic ulcer of the stomach and duodenum. Possibilities of instrumental and laboratory examination of patients. The main complications of peptic ulcer of the stomach and duodenum. Gastric bleeding syndrome.
The main complaints of patients with cholecystitis and cholangitis. Data of physical examination of patients with chronic cholecystitis and cholangitis. Definition and principles of modern classification of chronic hepatitis and liver cirrhosis. The main etiological factors of the development of hepatitis and liver cirrhosis. The mechanism of liver damage in hepatitis of viral etiology. The main complaints of patients with hepatitis and liver cirrhosis, features of examination results and physical examination data. Morphological and biochemical signs of liver damage. The main complications of liver cirrhosis.

Chronic gastritis (CHG) CHG is a disease with a chronic relapsing course, the basis of which are inflammatory and dystrophic, dysregenerative lesions of the mucous membrane of the stomach, which are accompanied by a violation of its secretory, motor-evacuatory and incretory function.
ChG is a clinical and morphological concept, but the final diagnosis becomes valid only after histological confirmation Prevalence: among all diseases of the gastrointestinal tract - 35% of cases are chG; among diseases of the stomach - 85%. ChG affects 40-50% of adults worldwide.
The prevalence of HG depends on the place and living conditions of people, which is clearly related to infection with H. pylori.
Etiopathogenesis and pathomorphology In clinical practice, three types of CG are most common:
1. Superficial with a predominant lesion of the antral part of the stomach, most often associated with H. pylori (type B gastritis), in which normal or even increased secretion of hydrochloric acid is disturbed for a long time.
2. Autoimmune fundus gastritis (type A gastritis), in the formation of which autoimmune mechanisms take part. It is characterized by the detection of antibodies to parietal cells and intrinsic factor, as well as a high level of gastrin in blood serum with a tendency to achlorhydria of gastric juice.
3. Chemical, reflux gastritis (gastritis type C), which is characterized by a focal lesion of the fundal part of the stomach as a result of the cytotoxic effect on the mucous membrane (CO) of the contents of the duodenum in duodenogastric reflux. It often develops in the 19th stump of an operated stomach with small intestinal reflux. Close to this type of gastritis is gastritis caused by medication damage to the gastric mucosa.
Morphological changes in CG: These include inflammation, atrophy, impaired cellular renewal, including metaplasia and dysplasia. Chronic inflammation is indicated by the infiltration of the personal plate and epithelium with mononuclear elements. CO atrophy is

characterized by a decrease in the number of normal glands. The biological basis of atrophic gastritis is a violation of proliferation and apoptosis due to various pathogenic factors (including H. Rylori). In case of atrophy, together with the irreversible loss of the gastric glands, their replacement by metaplastic epithelium or fibrous tissue occurs. Intestinal metaplasia (replacement of gastric epithelium with intestinal epithelium) is quite common, and in elderly people it is found even in practically healthy people. With atrophic gastritis, it is almost 100% cured. Dysplasia. It should be considered as a precancerous condition. It distinguishes two degrees: low and high.
Classification. It was adopted in 1990 at the IX International Congress of Gastroenterologists (Australia), which was called the "Sydney system". In 1998, modified L.I. Aruin et al.
CG type B.
Clinic: manifested by symptoms characteristic of peptic ulcer disease, hunger and night pains in the epigastrium, nausea, vomiting, acid belching, heartburn. Characteristic tendency to constipation.
All symptoms are caused by an increase in acid-forming function in response to damage to the antral part of the stomach. The disease can have an asymptomatic course.
The diagnosis is established on the basis of a characteristic clinical picture; endoscopic examination, which makes it possible to clarify the localization, character of changes in the CO of the stomach. The absolute diagnostic criterion for the disease is the detection of H. pylori itself and the products of its vital activity, as well as morphological signs of chronic gastritis in biopsies.
CG type A. Clinic: Most often, the course was asymptomatic until B12-deficient megaloblastic anemia developed. Sometimes the clinic is characterized by symptoms of gastric dyspepsia (dull pain and heaviness in the epigastrium after eating, belching, nausea, unpleasant taste in the mouth); signs of intestinal dyspepsia (flatulence, diarrhea). Fatigue, drowsiness, burning of the tongue, paresthesias of the limbs appear during the development of pernicious anemia.
Objectively: lacquered tongue, pale skin, possible subicteric sclera, as well as loss of vibrational sensitivity, impaired gait, etc. Type A gastritis is often combined with other autoimmune diseases - Hashimoto's thyroiditis, Addison's disease, hypoparathyroidism, etc.
The diagnosis is established on the basis of the clinical signs listed above and the characteristic endoscopic picture (pale CO of the bottom and body of the stomach, through which the vascular pattern is clearly visible). The most characteristic feature is the presence of antibodies against parietal cells and intrinsic factor.
ChH type C Clinic. Pain and a feeling of heaviness in the epigastrium during sleep or immediately after eating, nausea, vomiting, heartburn. It often has an asymptomatic course.
The diagnosis can be verified by the morphological study of the gastric biopsy specimen.
Radiation CG – the severity of stomach damage depends on the dose of radioactive radiation. Moderate or severe coagulation necroses of CO of the stomach with secondary inflammatory infiltration are revealed. Moderate changes are usually reversible and regress as CO is updated to the norm over a period of 4 months. Ulcers, fibrosis and obiter hyalinosis of vessels are formed in case of severe damage.
Lymphocytic gastritis - has a clear histological characteristic: mainly infiltration by interepithelial lymphocytes (normally 3-5 per 100 epithelial cells, with lymphocytic gastritis - increases to 30-50)

Granulematous gastritis - occurs as a manifestation of Crohn's disease, sarcoidosis, parasitic invasions, foreign body ingestion. The morphological substrate is epithelioid cell granulomas, sometimes with giant multinucleated cells.
Eosinophilic (allergic) gastritis is characterized by pronounced infiltration of CO and other layers of the stomach wall by eosinophils. The antral region is mainly affected.
Other infectious gastritis (not Helicobacter pylori-associated). They are caused by viruses, microbes, fungi, parasites. They are rare.
Menetrier's disease is a hypertrophic gastropathy. The main morphological feature of the disease is giant folds mainly in the area of the bottom and body of the stomach. It is clinically manifested by anorexia, nausea, vomiting, gastrointestinal bleeding, diarrhea, weight loss up to 25 kg, pain in the epigastrium, hypochlorhydria, hypoalbuminemia (before the development of non-protein edema in 20-100% of patients).
Diagnostics
Methods of instrumental diagnosis of HG:
- functional diagnostics: determination of gastric secretion by the method of fractional sounding or by the method of intragastric pH-metry; - endoscopic examination;
- morphological study; x-ray examination of organs of the gastrointestinal tract;
- diagnosis of H. pylori infection (bacteriological examination - culture of a CO biopsy on a differential diagnostic medium; morphological: histological - staining of bacteria in a histological preparation of CO according to Giemse, Wartyna-Starry, Ghent, toluidine blue; cytological - staining of bacteria in smears-imprints of CO stomach according to Giemse, Gram;
determination of the products of H. Pylori's vital activity: urease - determination of urease activity in a CO biopsy of the stomach in a liquid or gel-like environment that contains a substrate, a buffer and an indicator; respiratory - determination of C or 13C isotopes in exhaled air, which are released as a result of the splitting of labeled urea in the patient's stomach under the action of the urease of the H. pylori bacterium; immunoenzymatic - determination of antibodies to H. pylori; PCR - determination of H. pylori using the polymerase chain reaction in feces.

Definition of HC - a chronic disease of the stomach or duodenum with a recurrent course, prone to progression, which is based on the formation of an ulcerative defect in the mucous membrane of the stomach or duodenum in the period of exacerbation with subsequent scarring.
ETIOLOGY VK is a multifactorial disease with a polygenic type of heredity. In the presence of a "critical" number of genetically determined signs, a predisposition to VH is formed, which can be realized when the body is affected by a complex of adverse environmental factors. Hereditary tendency to peptic ulcer is found in 4050% of patients. The risk of developing HF in blood relatives of patients is 3-4 times higher than in the general population. "Family ulcer syndrome" - ulcers of the same localization, more often duodenal, are found in parents and their children, all of them have 0 (I) blood group.
CAUSING FACTORS OF THE EXTERNAL ENVIRONMENT
1. Psychoemotional stress, chronic overstrain of the nervous system, in which a focus of "stagnant excitement" is formed in the subcortical structures, cortical-subcortical

relationships are disturbed with the development of dystonia of the autonomic nervous system, hormonal imbalance.
2. Harmful habits - smoking and alcoholism, 95% of peptic ulcer patients smoke. Nicotine causes hyperplasia of lining cells in the mucous membrane of the stomach, suppresses the formation of bicarbonate by the pancreas, increases the level of pepsinogen-1 in the blood, suppresses mucus formation in the stomach and duodenum, strengthens the motility of the stomach, causes spasms, duodeno-gastric reflux. Alcohol damages the mucous barrier of the stomach, enhances the back diffusion of H+ ions through the mucous membrane. Low concentrations of ethanol stimulate gastric secretion, high concentrations suppress it, but cause erosion of the mucous membrane. Systematic alcohol consumption is accompanied by dystrophy and atrophy of the gastric mucosa with the appearance of areas of intestinal metaplasia.
3. Alimentary factors - systematic violation of the stereotype of nutrition - liquid and irregular food intake, dry, hasty food, unbalanced diet with a deficiency of proteins and vitamins, abuse of rough, spicy foods, strong coffee.
4. The infectious factor (Helicobacter pylori - HP) plays an auxiliary role in the development of HC. Helicobacter-dependent form is duodenal ulcers associated with chronic type B gastritis.
5. Unfavorable meteorological factors - sharp fluctuations in meteorological conditions, characteristic of autumn and spring, cause dysfunction of the neurohypophysis-pituitary-adrenal cortex system, during this period the acid-forming function of the stomach increases.
PATHOGENESIS There are significant genetic, clinical, functional, and pathogenetic differences between gastric ulcer (mediogastric ulcer) and duodenal ulcer (pyloroduodenal ulcer). However, in both cases, the decisive moment is the violation of the balance between the factors of "protection" of the mucous membrane and the factors of "aggression", which cause self-digestion of the mucous membrane with the formation of an ulcer defect.
"AGGRESSION" FACTORS
1. Hypertonus of the vagus nerve. An increase in the tone of the parasympathetic nervous system is accompanied by an excess release of the neurotransmitter acetylcholine, which directly stimulates the parietal and main lining cells of the gastric glands, and also increases the release of gastrin from G-cells of the stomach and histamine from mast cells. The vagus nerve stimulates (albeit to varying degrees) all three phases of gastric secretion, especially the brain phase. Hypervagotonia is observed in 2/3 of patients with duodenal ulcers.
2. Sufficient production of gastrin. Gastrin is a gastrointestinal hormone that is synthesized by G-cells of the pyloroantral part of the stomach. The secretion of gastrin is stimulated by distension of the stomach with food and the effect of products of partial hydrolysis of food proteins. Gastrin is the main mediator that ensures the gastric phase of hydrochloric acid secretion. In addition, the hormone has a trophic effect on the mucous membrane of the stomach - it causes hyperplasia of the fundal glands.
3. Hyperhistamineemia. Histamine is the final mediator that mediates the effect of gastrin on the gastric glands, one of the strongest stimulators of gastric secretion. When using blockers of H-2 histamine receptors, gastric secretion, which was stimulated by both histamine and pentagastrin, is inhibited. Histamine is produced in the mast cells of the mucous membrane

of the stomach and affects the histamine H-2 receptors of the lining cells of the gastric glands.
4. Increase in the mass of lining cells of the stomach. Reconstruction of the mucous membrane of the fundal part of the stomach with sharp hyperplasia and an increase in the mass of lining cells can be congenital or acquired.
5. Acidopeptic factor. Acid-peptic aggression consists in long-term hyperchlorhydria and increased content of enzymes in gastric juice. 7 types of proteolytic enzymes of gastric juice were identified. Of these, 5 fractions are combined into the group of pepsinogen-1 (or pepsinogen A), which exhibits maximum activity in an acidic environment at pH 1.5-2.0. 2 fractions of enzymes form a group of pepsinogen-2 (pepsinogen C, or gastrixinogenau, their maximum activity is observed at pH 3.2-5.0. Proteolytic enzymes cathepsin D and C are active at pH 2.0-3.5. Stimulators of pepsinogen secretion are acetylcholine and, to a lesser extent, gastrin and histamine, inhibitors - somatostatin, prostaglandin E-2, anticholinergic substances. In duodenal ulcer, in 60% of cases, the level of pepsinogen-1 in gastric juice, blood, and urine is elevated, with gastric ulcer, as a rule, normal The ulcerogenic action of pepsin-1 is realized through damage to the protective mucus.
6. Gastroduodenal dyskinesia. Violation of the motor and evacuation function of the gastroduodenal zone increases the duration of contact of "aggressive" gastric juice with the mucous membrane. 7. Violation of the mucous barrier by Helicobacter pylori. The mucous barrier is a system of protective factors of the gastroduodenal zone: mucus, high regenerative capacity of the surface epithelium, adequate regional blood circulation. The mechanism of action of the mucous barrier is to prevent the back diffusion of H+ ions through the mucous membrane. When the mucosal barrier is damaged 5, the retrodiffusion of H+ increases dramatically, which is one of the key factors in ulcer formation. Mucus is formed by additional cervical cells of the fundal glands of the stomach and all cells of the covering epithelium, in the duodenum - by goblet cells and Brunner's glands. Mucoal-bicarbonate barrier is the first line of defense of the mucous membrane of the stomach. The second line of defense is the surface highly prismatic epithelium of the stomach, which is characterized by high regenerative activity, non-stop restoration of cell membranes. With HC, the rate of proliferation of epitheliocytes is sharply increased, and they are unable to perform their protective functions. Adequate regional blood circulation is necessary for the last energy supply, the supply of plastic substances. Microthrombosis, microcirculation disorders, ischemia zones contribute to ulcer formation.
8. Neurotrophic disorders. Violation of trophic processes in the mucous membrane of the gastroduodenal zone, caused by a change in the tone and reactivity of the sympathetic-adrenal system, is characteristic of VK. The sympathetic division of the autonomic nervous system has an ergotropic effect, improves regional blood circulation and tissue trophicity, increases the formation of protective mucus, the level of cAMP, prostaglandins. The pathogenic effect is manifested both by the excessive accumulation of catecholamines in the mucous membrane of the gastroduodenal zone, and by the depletion of catecholamine reserves.
PROTECTIVE FACTORS
1. Mucous-bicarbonate barrier of the stomach and duodenum.
2. Active physiological regeneration of the surface epithelium - complete renewal of the cells of the covering and glandular epithelium takes place within 1-5 days, up to 500,000 cells are

formed every minute from the pit epithelium and cells of the cervical part of the gastric glands. The cells of the surface epithelium are closely adjacent to each other, their apical membrane contains lipoproteins, which prevent the penetration of ions and water-soluble substances into the cells. On the surface of the mucous membrane of the stomach, the pH is 2.2, and on the surface of the covering epithelium covered with a mucous-bicarbonate layer, it is 7.6.
3. Duodenal inhibitory mechanism - inhibition of gastric secretion when chyme enters the intestinal mucosa due to the release of gastrointestinal hormones - secretin, somatostatin, GIP, VIP, cholecystokinin-pancreozymin. These hormones block the secretion of gastrin. Atrophic duodenitis, which is accompanied by a decrease in the production of gastrointestinal hormones by the duodenum, leads to hyperacidity and the development of duodenal ulcers.
CLINICAL MANIFESTATIONS
PAIN SYNDROME The leading clinical manifestation of HF is pain syndrome. Pain sensations in HC are caused by an increase in intragastric and intraduodenal pressure with irritation of pain baroreceptors, spastic contraction of the cardiac and pyloric sphincters with ischemia of their walls and intense shooting pains, reactive periulcerous inflammation of the mucous membrane, irritation of the ulcer with active gastric juice, inflammation of the serous membranes. Pain syndrome in peptic ulcer disease has a number of pathognomonic features.
1. Pains have a steady daily rhythm caused by the diet. The interval between eating and the episode of pain caused by it depends on the location of the ulcer. The "higher" the ulcer is located, the faster pain occurs after eating.
- "EARLY PAINS" appear 15-30 minutes after eating, develop with mediogastric ulcers.
- "LATE PAINS" occur 45 minutes - 2 hours after eating, characteristic of pyloroduodenal ulcers.
- "HUNGER PAINS" appear on an empty stomach, 3-4 hours after the last 6 meals, and disappear after eating any food, even after the patient drinks a glass of water, typical for duodenal ulcers.
- "NIGHT PAIN" is a variant of "hunger pain", relieved by eating. Patients with night pains always have something from food on the bedside table - a glass of milk or white bread.
-Depending on the localization of the ulcer, the nature of the relationship between pain and food in patients is different. - With mediogastric ulcers, there is a characteristic stereotype: "food - pain, hunger - relief." With duodenal symptoms, there is a stereotype: "hunger - pain, food - relief." 2. Pains are localized in a small, limited area, the patient can accurately indicate their location.
The pain zone is limited to 1-2 cm. The most typical pain zones:
1) epigastric region, below the xiphoid process along the midline of the abdomen or slightly to the right (duodenal ulcers);
2) epigastric region, below the xiphoid process, to the left of the midline of the abdomen (mediogastric ulcer). At the height of a pronounced pain attack, the pain zone may slightly increase.
3. Cyclical nature of each pain attack - gradual increase in pain, reaching maximum severity, slow decrease in pain intensity until its complete elimination. The duration of one pain cycle can reach 2-3 hours.

4. The expression and character of the pain are very variable. Pains of minimal severity can be perceived as a painful, aching feeling of hunger. A feeling of discomfort in the epigastrium, a feeling of fullness, distension is possible. The intensity of sensations can increase, creating a feeling of squeezing pain, burning, pinching, drilling, reaching the intensity of cutting, tearing pain. The intensity of pain depends on the activity of the ulcer process, the involvement of serous membranes, the severity of the perifocal inflammatory process, the pain threshold and the reactivity of the patient.

5. Persistence, stereotypy of pain sensations, their daily rhythm are preserved with each subsequent relapse.

6. Irradiation of pain appears only when complications arise. Penetration (germination) of an ulcer into neighboring organs, the development of an adhesion process (perigastritis, periduodenitis), the occurrence of concomitant diseases (cholecystitis, pancreatitis) are accompanied by a violation of the usual rhythm, nature and localization of pain. Penetration of a duodenal ulcer into the head of the pancreas or a deep ulcer of the back wall of the stomach reaching the serous membrane is accompanied by pain that radiates to the back at the level of the 10th thoracic - 1st lumbar vertebra. Penetration of the ulcer into the lesser omentum is clinically manifested by radiating pain in the right (rarely in the left) hypochondrium. Penetration of the ulcer into the hepatoduodenal ligament is characterized by pain radiating into the right half of the chest. Penetration of the ulcer into the gastrosplenic ligament is accompanied by radiation of pain in the left half of the chest. Irritation n. phrenicus subcardial ulcer is manifested by the spread of pain beyond the sternum, to the precardiac, supraclavicular region on the left.

7. Factors that eliminate a pain attack: 1) taking soda, alkalis, alkaline mixtures (open the pylorus, stimulate evacuation, cause relieving air belching and reduce intragastric pressure); 2) appointment of cholinolytics or myotropic antispasmodics; 3) local heat (eliminates spasm of sphincters, reduces smooth muscle tone, improves microcirculation, lowers intra-organ pressure); 4) spontaneous or induced vomiting, probing the stomach. 7

8. Seasonality of exacerbations and pain - autumn and spring. Exacerbation lasts 3-5 weeks, accompanied by a characteristic pain syndrome. After that, remission develops, sometimes even spontaneously, without adequate therapy. Circadian rhythms of the neurohumoral regulation systems, seasonal changes in the nature of nutrition are the basis of seasonality of HC.

DYSPEPTIC SYNDROME Dyspeptic syndrome may not be present at all in uncomplicated HF.

BURNING is pronounced, unbearable, can cause a feeling of burning pain. Constant heartburn, especially its intensification when lying on the back, is a sign of insufficiency of the cardioesophageal sphincter or hernia of the esophageal opening of the diaphragm with gastroesophageal reflux of acidic gastric contents. With duodenal ulcers, heartburn occurs in 80% of cases, with mediogastric ulcers - in 30-40%.

ERUPTION (50-60% of patients) on the internal organs. Air belching can be caused by aerophagia - swallowing when breathing a significant amount of air. The second condition for the development of the symptom is a decrease in the tone of the cardiac sphincter of the esophagus. Sour belching and heartburn are characteristic of duodenal ulcers ("acidosis syndrome"). Belching bitter is a sign of duodeno-gastric bile reflux. Belching of recently eaten food or with the "smell of rotten eggs" is observed with long-term retention of food in

the stomach: organic pyloroduodenal stenosis, inflammatory edema, and long-term spasm of the pylorus. Gastrostasis is accompanied by colonization of the stomach by microorganisms, microbial breakdown of food protein with the formation of hydrogen sulfide.
NAUSEA precedes vomiting.
VOMITING is a classic symptom of VT (40-60% of cases), occurs spontaneously at the height of a pain attack and relieves or completely eliminates pain. In the absence of spontaneous vomiting, patients often induce it artificially by pressing on the root of the tongue.
APPETITE in patients with HC is preserved or increased. Anorexia develops with pyloroduodenal stenosis or VL with reduced acidity. Sitophobia (fear of eating) is characteristic of a severe exacerbation.
SPASTIC CONSTIPATION (in 50% of patients with VH) - delay of defecation for 2-3 days, complicated act of defecation, release of hard feces in the form of dense small balls ("sheep feces"). Disturbance of intestinal function is caused by vagotonia, increased segmenting peristalsis, spasms of the large intestine, as well as a slag-free diet and hypodynamia of patients.
METEORISM (50% of patients), caused by secondary dysbacteriosis with the appearance of hemolyzing strains, fungi, staphylococci, sharp inhibition of bifidobacteria and lactobacilli. Secondary colitis often develops, mostly proctosigmoiditis.
ASTENO-NEUROTIC SYNDROME In VH, characteristic features of the psycho-emotional status: increased anxiety, egocentrism, demonstrativeness, high level of requests. Psychopathological syndromes are often found - anxiety-depressive, anxiety-phobic, hypochondriac, asthenic with hysterical reactions, often there are neurotic-neurosis-like states. Increased sensitivity to changes in meteorological factors - meteotropism.
PHYSICAL EXAMINATION During an attack of pain, patients often adopt a characteristic forced position - lying on their side (or back) with their knees drawn to their stomach or squatting, thus exerting pressure on the fossa under the breast with a fisted hand. In the non-acute period, the general condition of the patient is satisfactory, the condition is active. An examination of the abdominal wall may reveal pigmentation of the epigastrium as a result of the systematic use of local heat (heating pads) to eliminate spastic pain. The tongue with mediogastric ulcers, which are most often accompanied by gastritis, has a grayish-yellow plaque. The tongue with duodenal ulcers is clean, moist, with 8 well-defined papillae. A dry, covered with abundant brown coating of the tongue is observed in the case of complications of HCV penetration into neighboring organs or perforation.
PALPATORY in the epi- and mesogastria, increased sensitivity of the skin (hyperesthesia-hyperalgesia) is revealed. In case of exacerbation of the disease, the appearance of protective muscle tension, increased resistance of the abdominal wall, caused by the reaction of the parietal sheet of the peritoneum to the active ulcer process, is possible. With deep palpation in the area where the ulcer defect is located (especially with palpation of the "niche" area behind the X-ray screen, during X-ray examination), local tenderness is determined. Pyloroduodenal stenosis, which complicates the course of VDC, is manifested by visible peristaltic waves of the stomach, going from left to right, after a shock-like palpation in the epigastrium. Push-like palpation of the epigastrium leads to the appearance of a "slapping noise" in patients with pyloroduodenal stenoses and severe gastroptosis. Sometimes, deep palpation reveals a spasmodic, dense, painful sigmoid colon.

MENDEL'S SYMPTOM - local pain upon percussion in the area of the ulcer in the epigastrium. During inhalation, short jerky blows are applied with two bent hands (index and middle) of the right hand in symmetrical areas of the abdominal wall in the epigastrium - under the xiphoid process, to the right and to the left of it. With a positive symptom, the patient feels pain in a limited area. The expression of the symptom (+, ++, +++) corresponds to the activity of the process. Mendel's positive symptom is associated with irritation of the zone of the parietal leaf of the peritoneum adjacent to the ulcer.
SYMPTOM OF OBRAZTSOVA-STRAZHESKO - persistent tympanitis when percussing the medial part of the right costal arch, appears with adhesions between the stomach, duodenum and liver (perigastritis, periduodenitis).
OPENHOVSKY'S SYMPTOM - pain when pressing on the area of spinous processes of 8-9 thoracic vertebrae.
BOAS SYMPTOM - pain when pressing on both sides of the spine at the level of 10 thoracic - 1 lumbar vertebrae, observed with penetration of the ulcer.
LAENEK'S SYMPTOM - pain during palpation in the epigastric area with a retracted abdomen.
BERGMAN'S SYMPTOM - disappearance of abdominal pain following the onset of gastrointestinal bleeding.
BRUNNER'S SYMPTOM - friction noise under the costal arch during ulcer perforation.
HYUNTZBURG'S SYMPTOM - localized rumbling between the gallbladder and pylorus, a possible symptom of duodenal ulcer.
REICHMAN'S SYMPTOM - excessive secretion of gastric juice, sour belching, unbearable heartburn, vomiting on an empty stomach with gastric juice (often at night), breath due to reflex spasm of the vocal cords. It is observed with duodenal ulcers or stenosis of the pylorus.
BENEDICT'S SYMPTOM - the patient is given a saturated sodium bicarbonate solution to drink and auscultation of the stomach is performed: pronounced crepitation is heard with hyperacidity, moderate crepitation with normoacidity, and no crepitation with anacidity.
UDEN'S SYNDROME - reflex disorders of the heart: a feeling of pressure in the area of the heart with radiation to the left shoulder, angina pectoris, hypotension, shortness of breath, aerophagia, flatulence.
SCHLISENGER'S SYMPTOM - variable displacement of the navel to the side of the lesion when the patient exerts himself, observed in prepyloric ulcers.
EFLEIN'S SYMPTOM - contraction of the back muscles at the level of 7-10 thoracic vertebrae upon percussion of the patient in the supine position.
TRINITY SYMPTOM - triple cyclicity of ulcer pain: change of pain during the day depending on food intake, change of pain during the year depending on the season, alternating periods of exacerbation and remission.
ADDITIONAL EXAMINATION METHODS
X-RAY EXAMINATION X-ray examination is the most common method of diagnosing ulcer disease, as it has practically no contraindications and is widely available. However, the sensitivity of x-ray examination for ulcers is 75-85%. X-ray of the stomach and colon with contrast with barium suspension is most often used. Special X-ray examination methods have a higher diagnostic sensitivity: 1) double contrast with additional air insufflation into the stomach; 2) parietography with additional gas injection into the stomach cavity and abdominal cavity; 3) relaxation duodenography under artificial hypotonia of the duodenum

with additional administration of aeron or atropine, metacin to the patient - at the same time, functional spasms are eliminated, contrast transit is slowed down. X-ray examination should be polypositional, in vertical and horizontal positions of the patient to achieve "tight" filling of all parts of the stomach and duodenum with X-ray contrast. SYMPTOM "NICHES" is considered a direct X-ray sign of an ulcer. A contour "niche" is distinguished - a depot of barium mass with clear contours, conical, elongated or trapezoidal shape, which is determined by the contour of the gastric shadow. The "relief-niche" is located on the front or back wall of the stomach or duodenum and is a stable depot of barium among the folds of the mucous membrane - a "spot" on the relief. The "niche" may not be determined during tamponade of the ulcer crater with fibrin or blood clots. Other X-ray symptoms are indirect, among them a number of functional symptoms are distinguished. FASTING HYPERSECRETION - incomplete filling of the stomach with contrast due to the presence of an endogenous secretion in the fasting stomach. DISORDERS OF MOTICS AND TONE of the stomach and the stomach - characteristic hypermotility and hypertonus of the stomach, acceleration of contrast evacuation from the stomach and its rapid passage through the stomach in combination with short-term pylorospasms and gastroduodenal or gastroesophageal reflux. Hypotonus and hypomotility are more often observed in peptic ulcer disease, aperistaltic zones can be determined in the area of deep ulcers. LOCAL PAIN in the "niche" area is observed during deep palpation of the patient behind the x-ray screen. LIMITATION OF MOBILITY of the DPC bulb due to periduodenitis and the formation of adhesions. The presence of indirect signs of HC is also taken into account. "FILLING DEFECT in the area of the ulcer, which is formed when there is a significant "inflammatory wall" around the ulcer and the closing of the swollen folds of the mucous membrane above the ulcer crater. CONVERGENCE OF THE FOLDS OF THE MUCOUS MUSCLE as a result of periulcerous muscle spasm and cicatricial pulling of the mucosa towards the ulcer defect. SYMPTOM " "INDEX FINGER" or contralateral de Quervain's spasm - a filling defect on the opposite side of the ulcer on the side of the stomach or duodenum due to spasm or scarring of the circular or oblique muscles. The symptom of finger-like retraction can be functional or permanent, determined by the great curvature of the stomach, in the outlet of the stomach or in the bulb of the DPC. SCART-ULCER DEFORMATION of the stomach and DPC is caused by cicatricial tightening of muscle fibers. The most common form of deformation of the bulb of the DPC is fine jaggedness of the wall contours, uneven narrowing of the lumen of the duodenum, the formation of some diverticula-like pockets in the shape of a shamrock, a butterfly, a hammer. Relaxation duodenography is performed to differentiate morphological changes from functional ones. X-ray diagnosis of complications of IV is possible. The proof of the penetration of the stomach ulcer into the lesser omentum is the irregular shape of the "niche", its large dimensions, the straightening of the contour of the minor curvature and its fixation on a large area. The diagnosis of penetration of the DPK ulcer is complicated, the most frequent signs are large, irregularly shaped, with indistinct contours of the niche, which do not change their shape after the administration of antispasmodics. The occurrence of ulcer perforation is accompanied by the accumulation of free gas in the most painful part of the abdominal cavity under the diaphragm in a standing position, i.e. punctured pneumoperitoneum. When the position of the patient changes, the gas moves. A sign of organic cicatricial stenosis of the pylorus is the presence of contrast in the stomach after 24 hours. after research. Deformations are often observed in ulcers of the

pyloric canal - angular or knee-like curvature of the canal, spasm or its persistent expansion, "flaring" of the pylorus.
ENDOSCOPIC EXAMINATION Fibrogastroduodenoscopy (FGDS) is the most sensitive and informative method of detecting ulcers, which allows you to reliably determine the localization and size of ulcers, determine the stage of the ulcer process, and carry out dynamic control over the scarring process. Ulcer formation takes 4-6 days and is conditionally divided into several stages: - the red spot stage (1-a) is characterized by the formation of a limited area of pronounced hyperemia on the mucous membrane. - 2nd stage of erosion: in the zone of hyperemia, numerous point hemorrhages appear, merging with each other and erosion. - 3rd stage - an ulcer with flat edges, has an irregular shape and extends only to the depth of the mucous membrane. - 4th stage, a complete ulcer deepens to the muscular and serous membrane, the "inflammatory shaft" forms a deep crater around the perimeter.
The ACUTE PHASE of a chronic ulcer is characterized by a round or oval shape of the ulcer defect. If the submucosa is fibrosed due to scarring of previous ulcers, the shape of an acute ulcer may be polygonal or slit-like. The edges of a fresh ulcer are clear, smooth, juicy, and bleed easily when in contact with a fibroscope. The bottom of the ulcer is covered with yellowish-gray fibrin overlays. The mucous membrane around the ulcer is sharply swollen, hyperedematous, an "inflammatory ridge" is formed around the ulcer. The folds of the mucous membrane are thickened, they do not straighten completely during insufflation of air, often perifocally, numerous fine-point erosions are determined.
The subacute phase of a chronic ulcer is characterized by a decrease in the severity of periulcerous edema and the depth of the "inflammatory shaft", the bottom is filled with granulation tissue, the ulcer becomes flattened, its diameter decreases, and the folds of the surrounding mucous membrane converge to the edges of the ulcer. Such ulcers are often not detected radiologically.
THE CRACKING PHASE is manifested by the flat topography of the ulcer, the matching of its edges with the formation of a slit-like defect or several defects.
THE RED SCAR PHASE is accompanied by the formation of a bright red linear or star-shaped scar at the site of the ulcer, surrounded by a hyperemic mucous membrane with pronounced converging folds.
THE WHITE SCAR PHASE is characterized by a permanent white scar of a linear shape, elongated, often surrounded by converging white-gray strands. Perifocal hyperemia disappears. Duodenal ulcers differ from gastric ulcers in their shallower depth and more complex shape - polygonal, star-shaped. An ulcer is formed within 4-6 11 days, and the process of self-limitation and scarring takes 4-6 weeks for a duodenal ulcer, 6-8 weeks for a mediogastric ulcer. In 1/3 of patients with duodenal ulcer, cicatricial ulcer deformation of the duodenal bulb is formed, which is manifested by pronounced thickening and deformation of the folds of the mucous membrane, uneven narrowing of the lumen of the duodenum. In the course of endoscopic examination, chromogastroscopy with additional injection of dyes into the stomach is often performed in patients with gastric cancer. Methylene blue (15-20 ml of a 0.5% solution) stains the zones of intestinal metaplasia and the tumor guest blue-blue. Congo red (30-40 ml of 0.3% solution) colors areas of active acid formation black, acid-forming areas bright red. It is mandatory to examine the biopsy material for Helicobacter infection,

for this, samples of the mucous membrane of the pyloric department and zones of gastric metaplasia in the duodenum are taken and special staining is performed.
MORPHOLOGICAL EXAMINATION Morphological examination complements the endoscopic examination: through a fibrogastroduodenoscope, under visual control, a targeted biopsy is performed from the bottom and edges of the ulcer, from the surrounding areas of the mucous membrane. During the morphological examination of the punctate from the edges and bottom of the ulcer, detritus is revealed - an accumulation of mucus, exfoliated epithelium and necrotized cells, under which there are necrotized collagen fibers. Signs of an acute inflammatory process are observed in the peri-ulcer zone - swelling, blood vessels, leukocyte infiltration, proliferation of fibroblasts, swelling and necrosis of vessel walls, dystrophy and necrosis of nerve elements. In the healing phase, the ulcer defect is filled with granulation tissue, inflammatory infiltration decreases and the ulcer epithelizes - a layer of single-layer epithelium from the edges of the ulcer "crawls" onto the ulcer defect.
DIAGNOSTICS OF H. RYLORI INFECTION Bacteriological examination - culture of a biopsy sample on a differential diagnostic medium; Morphological: histological – staining of the bacterium in the histological preparation CO according to Giemse, Vartyna-Starry, Gente, toluidine blue; cytological - staining of bacteria in smears-imprints of CO of the stomach according to Giemse, Gram; Determination of the products of H. Pylori's vital activity: urease - determination of urease activity in a CO biopsy of the stomach in a liquid or gel-like medium containing a substrate, a buffer and an indicator; respiratory - determination of 14C or 13C isotopes in the exhaled air, which are released as a result of the splitting of labeled urea in the patient's stomach under the action of the urease of the bacterium H. Rylori; Immunoenzymatic - determination of antibodies to H. Pylori; PCR - determination of H. Pylori using the polymerase chain reaction in feces
STUDY OF THE SECRETORY FUNCTION OF THE STOMACH 1. ASPIRATION-PROBE METHOD The study of gastric secretion is carried out according to the standard method, with the study of 3 phases of the secretory cycle: fasting, basal and stimulated by standard stimuli. The nature of acid formation disorders is significantly different in ulcers of different localization. The following pattern is observed with HC: the "higher", the more proximal the ulcer is in the gastroduodenal zone, the lower the indicators of acid formation and pepsin secretion. Duodenal ulcers are characterized by a significant increase in acid production in all phases of gastric secretion. The panhyperchlorhydric type of gastric secretion is most often detected, with an increase in both basal acid production (BAPC) and maximal stimulated acid production (MAPC). A more significant 12 increase in BKP is characteristic, which leads to a change in the ratio of BKP: MKP - it is 1: 4 - 1: 3 (1: 6 in healthy people). The output of hydrochloric acid in the basal phase of secretion (BKP) in patients with duodenal ulcers exceeds 5-7 mmol/h, sometimes reaching 12-15 mmol/h, which does not occur in other stomach diseases (with the exception of Zollinger-Ellison syndrome). The increase in acid production is combined with a 3-4 times increase in pepsin output, up to 100-200 mg/h due to an increase in the number and functional activity of the main (pepsin-producing) cells of the gastric mucosa. . Ulcers of the pyloroantral region are characterized by sufficiently high indicators of acid formation and secretory function of the stomach. Ulcers of the pyloric canal are close to duodenal ulcers in terms of the intensity of acid formation. Indicators of the acid-forming function of the stomach in patients with antral ulcers are usually lower than in patients with duodenal ulcers, but higher than in patients with

ulcers of the body of the stomach and cardia. Mediogastric ulcers are accompanied by characteristics or changes of the hyporeactive type. normal functional Ulcers of the subcardial and cardiac departments are distinguished by reduced indicators of secretion of hydrochloric acid and pepsin. However, pronounced achlorhydria, the absence of a secretory response to maximum stimulation are not characteristic, requiring the exclusion of the primary ulcerative form of gastric cancer.

2. INTRAGASTRONIC PH-METRY The study allows separate determination of pH in different departments of the gastroduodenal zone and long-term monitoring of indicators. Pyloroduodenal ulcers are characterized by a syndrome of continuous acid formation in the stomach, which continues even at night in the absence of food stimulation. If in healthy people under basal conditions in the morning the reaction in the body of the stomach is weakly acidic, pH 3.0-6.9, on average 4.5 units, then in patients with duodenal ulcer the basal acidity is sharply increased in the morning, no more than 2.0, reaching 0 .9-1.0 units. The second functional feature of these ulcers is the "acidic decompensated stomach" syndrome - there is no normal acidity gradient between the pyloroantral and fundal parts of the stomach. In healthy people, the pH difference between these departments is 4.0 units or more, with pyloroduodenal ulcers it is absent at all ("acidic decompensated stomach") or is 1-1.5 units ("acidic subcompensated stomach"). With a pyloric-fundal pH difference of 1.5-2.0 units, an "acidic compensated stomach" is diagnosed. The loss of the acid-neutralizing function of the pyloric glands in combination with the increased activity of the acid-forming fundal glands of the stomach leads to "acidification" of the duodenum. In healthy people, a slightly alkaline environment (pH 7.2-8.0) with periodic "peaks of acidification" (1 in 20 seconds) is found in the initial part of the duodenum during the evacuation of gastric contents. In patients with duodenal ulcers, persistent acidification of the duodenal medium to pH 3.4-2.8 is observed. The insufficiency of the alkalinizing function of the gastroduodenal zone is also confirmed by a significant reduction in the alkaline time (AL). Normally, under basal conditions of secretion, the alkaline nature of the stomach environment after taking 1 g of soda in 30 ml of water persists for 20-25 minutes, under conditions of pentagastrin stimulation - 8-10 minutes. In patients with duodenal ulcers, the alkaline time in the basal period is reduced to 7-10 minutes. Pharmaco-secretory tests performed during pH monitoring make it possible to find out the specifics of the mechanism of hypersecretion in a given patient. With reflex vagus-dependent hypersecretion, a positive chlorosyl (atropine) test is observed - after the subcutaneous injection of a standard dose of M-cholinelytic (1 ml), acid formation is partially blocked, intragastric pH values increase by 1.5-2.5 units. In the treatment of these patients, it is advisable to use M-cholinolytics. In patients with pyloroduodenal ulcers, a more informative study of basal secretion is the pH values, the value of the alkaline time when stimulating tests (histamine, pentagastrin) change slightly, because the secretory apparatus is constantly functioning at the limit of its capabilities, a significant increase in acid formation is no longer possible. With mediogastric and cardiac ulcers, on the contrary, the state of secretory processes is more accurately reflected by research under histamine or pentagastrin stimulation. Basal secretion in these patients may be normal or slightly increased (pH 2.0-4.0), or moderately reduced to pH 4.5-6.0. For ulcers of any localization, true anacidity with a pH of more than 6.0 is not characteristic. The results of intragastric pH-metry are reflected in the detailed clinical diagnosis. Criteria for the main variants of the state of gastric secretion after standard stimulation: pH 0.9-1.2 - pronounced hyperacidity; pH 1.3-1.5 -

hyperacidity; pH 1.6-2.2 - normal acidity; pH 2.5-3.5 - moderate hypoacidity; pH 3.6-6.0 - pronounced hypoacidity; pH greater than 6.0 - anacidity.
ELECTROGASTROGRAPHY Duodenal ulcers are characterized by a hyperdyskinetic state of the gastroduodenal zone. During electrogastrography, an increase in the frequency of gastric contraction waves (over 3 pulses/minute), an increase in the average amplitude of peristaltic waves (over 300 μV) and the total power of gastric biopotentials (over 900 μV/minute), the gastrogram curves are asymmetric. In case of mediogastric ulcers, on the contrary, hypokinesis and hypodyskinesis of the stomach are observed.
RADIOISOTOPIC SCANNING OF THE STOMACH The most informative and accessible method of assessing the evacuatory function of the stomach is a radionuclide study - scanning of the stomach after giving a "trial breakfast" of porridge containing trioleateglycerin labeled with I-131. In patients with duodenal ulcers, evacuation from the stomach is uneven - accelerated during the first hour, after 75 minutes it slows down, possibly due to reactive pylorospasm. With mediogastric ulcers, there is a tendency to slow down the evacuation function of the stomach. The research is complemented by pharmacological tests, which make it possible to identify the leading mechanism of motor-evacuation disorders and to predict the effectiveness of therapy for these disorders. A positive chlorosyl (atropine) test - slowing of evacuation with normalization of the rhythm - is typical for patients with duodenal ulcers with a leading vagal mechanism of pathogenesis. In these cases, M-cholinolytics show a stable therapeutic effect. A negative test indicates resistance to treatment with these drugs. With mediogastric ulcers, as a rule, positive tests with metoclopamid (Cerucal), a blocker of dopamine receptors. The drug stimulates and normalizes the motility of the gastroduodenal zone.
CLINICAL VARIANTS OF VH Gastric and duodenal ulcers of various localization have significant differences in clinical symptoms, different detectability by basic diagnostic methods, and are characterized by various secretory and motor abnormalities. Taking into account these features is mandatory for the correct diagnosis and treatment of VH.
ULCERS OF THE CARDIAL AND SUBCARDIAL SECTION OF THE STOMACH Ulcers of the cardiac section of the stomach are located 2-3 cm below the border between the epithelium of the esophagus and the stomach, ulcers of the subcardiac section - 0.5-5 cm below. Ulcers of this localization are combined into a single group, their frequency is 3-5% among all forms of HC. This area of the stomach has a powerful muscle layer, when it spasms, intense pain appears, which has a squeezing, pressing, burning, distending character, 14 often mimics angina pectoris. The horizontal position of the patient can contribute to the appearance of pain - at the same time, the contact time of the food chyme with the ulcerative defect increases. The pains are localized at the level of the xiphoid process, behind the sternum or slightly to the left, often refer to the precardial region, appear 15-30 minutes after eating. Taking antacids helps reduce pain. With ulcers of the cardiac and subcardial part of the stomach, the blocking function of the cardiac mass ("insufficiency of the cardia") is disturbed, which determines the nature of dyspeptic phenomena - typical are signs of gastroduodenal reflux: heartburn, belching, nausea. A decrease in the secretory function of the stomach is characteristic of ulcers of this location. X-ray and endoscopic diagnosis of cardiac and subcardial stomach ulcers is complicated. Shielding by the costal arch and the xiphoid process, the rapid flow of barium suspension causes a significant number of false-negative results during X-ray of the stomach. It is necessary to study in the vertical and

horizontal positions of the patient, in oblique and lateral projections, with the provision of additional portions of contrast and the execution of a series of pictures for the next study. Ulcers of the subcardial department are characterized by rapid scarring and long periods of remission, but there is a high risk of malignancy (8% of cases) and gastrointestinal bleeding (18%), which are of significant intensity.

ULCERS OF THE MINOR CURVATURE OF THE STOMACH (MEDIOGASTRIC) The most common gastric ulcers - 40-68% of cases, are localized mostly in the middle third or in the corner of the stomach. Mediogastric ulcers are characterized by a vague relationship with eating - episodes of both early and late, hungry and night pain are possible, although the dominant form of the pain syndrome is pain 1-1.5 hours after eating. The pain has an aching character, moderate intensity, is localized in the epigastric region to the left of the midline, often radiates to the left half of the chest, right and left hypochondrium, lumbar region (in 1/3 of patients). Exacerbation of VH and, therefore, ulcer pains have a pronounced seasonal dependence. When complications of peptic ulcer disease occur, the nature of the pain changes, the usual relationship with food rhythms disappears. Dyspepsia phenomena of reflux genesis are often observed - heartburn, belching, less often vomiting, nausea. Acid formation is normal or reduced, so a decrease in appetite and body weight is possible. A combination with atrophic antral gastritis is typical. The most common complication is bleeding (14%), penetration of the ulcer into the omentum is often observed.

CHARACTERISTICS OF THE COURSE OF VC In most cases, the course of VC is characterized by pronounced cyclicity, which allows distinguishing active and inactive stages of the disease. Phasing is more clearly expressed in duodenal ulcers. The active stage is conventionally divided into 3 phases: the acute and subacute phases of relapse and the phase of incomplete remission.

The ACUTE PHASE of the active stage is characterized by pronounced clinical symptoms of exacerbation, local pain upon palpation and percussion, resistance of the abdominal wall. During endoscopy, there is an ulcer defect surrounded by an inflammatory mass, accompanying gastritis, duodenitis. The average duration of the phase is 10-14 days.

The SUB-ACUTE PHASE of peptic ulcer relapse is characterized by a reduction in the manifestations of pain and dyspeptic syndromes, and the absence of changes in the functional examination. Endoscopically - decrease in the size and depth of the ulcer, disappearance of the inflammatory shaft, growth of granulation tissue. The duration of the phase is also on average 10-14 days.

THE PHASE OF INCOMPLETE REMISION is characterized by the complete absence of clinical symptoms of HC in the presence of endoscopic changes corresponding to the red scar phase.

INACTIVE STAGE is accompanied by complete clinical and anatomical remission.

DEGREE of severity of the disease - mild, moderate and severe.

MILD COURSE is characterized by rare exacerbations (once every 2-3 years) with mild clinical symptoms, a small and shallow ulcer (up to 0.5-1.5 cm in diameter), which scars within 4-6 weeks. In the stage of remission, working capacity is not impaired.

MEDIUM COURSE is characterized by more frequent relapses (every year, 1-2 times a year), which occur with pronounced clinical symptoms, the diameter of the ulcer is more than 1.5 cm. Scarring of the ulcer occurs after 6-8 weeks of inpatient treatment, a long phase of incomplete remission with limitation of work capacity .

A SEVERE COURSE is accompanied by frequent relapses with practically no permanent complete remissions. Clinical manifestations and functional disorders are pronounced and persistent. Characteristic are resistance to conservative treatment and the presence of complications, a significant decrease in working capacity.
COMPLICATIONS 1. Bleeding 2. Perforation 3. Penetration 4. Stenosis 5. Malignancy 6. Perigastritis and periduodenitis

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:
1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with a patient with chronic gastritis, peptic ulcer disease of the stomach and duodenum, chronic hepatitis and liver cirrhosis for the purpose of collecting complaints and anamnesis, physical examination of the patient; evaluation of clinical examination data and laboratory instrumental data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.
2.1. Requirements for the theoretical readiness of applicants to perform practical classes:
the applicant must know the modern definition, etiology, pathogenesis, classification of chronic gastritis, peptic ulcer disease of the stomach and duodenum, chronic hepatitis and liver cirrhosis, subjective and objective data in these diseases
to know laboratory and instrumental data for this pathology.

List of didactic units:
- to conduct a subjective examination of patients with chronic gastritis, peptic ulcer disease of the stomach and duodenum, chronic hepatitis and liver cirrhosis
- conduct an objective examination of patients with chronic gastritis, peptic ulcer disease of the stomach and duodenum, chronic hepatitis and liver cirrhosis
- appoint a plan for additional laboratory and instrumental examinations of patients with chronic gastritis, peptic ulcer disease of the stomach and duodenum, chronic hepatitis and liver cirrhosis
- evaluate the results of laboratory tests in patients with chronic gastritis, peptic ulcer disease of the stomach and duodenum, chronic hepatitis and liver cirrhosis
- master the skills and abilities to assess the leading clinical syndromes in patients with chronic gastritis, peptic ulcer disease of the stomach and duodenum, chronic hepatitis and liver cirrhosis

2.2. Questions to check basic knowledge on the topic of the lesson:
1. Definition of gastritis, peptic ulcer disease of the stomach and duodenum.
2. Classification of gastritis, peptic ulcer disease of the stomach and duodenum.
3. Etiology and pathogenesis of gastritis, peptic ulcer disease of the stomach and duodenum.
4. Definition of chronic hepatitis and liver cirrhosis.

5. Classification of chronic hepatitis and liver cirrhosis.
6. Etiology and pathogenesis of chronic hepatitis and liver cirrhosis.
7. Conducting subjective research: clarification of complaints; history taking.
8. The importance of methods of objective examination of the digestive organs in the diagnosis of chronic hepatitis and liver cirrhosis:
a) general overview;
b) examination of the oral cavity (mucous membrane, gums, teeth, tongue, tonsils, pharynx);
c) examination of the abdomen (size, configuration, external tumors, hernias, subcutaneous veins, navel, visible peristalsis, participation in breathing);
d) superficial palpation (detection of soreness, resistance, muscle tension, peritoneal symptoms, discrepancy of rectus abdominis muscles, hernias, tumors);
e) deep palpation of the abdomen;
f) determining the size of the liver and spleen according to the Kurlov method;
g) determination of free fluid in the abdominal cavity.
9. Characteristics of these laboratory-instrumental methods of research in chronic hepatitis and liver cirrhosis.

3. Formation of professional skills and abilities:
mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the interview)
formation of the ability to perform a clinical examination of a patient for chronic gastritis, peptic ulcer disease of the stomach and duodenum, chronic hepatitis and cirrhosis of the liver (the applicant must be able to perform a physical examination of the patient),

formation of the ability to give an assessment obtained during a clinical examination of a patient with chronic gastritis, peptic ulcer disease of the stomach and duodenum, chronic hepatitis and liver cirrhosis
given; carry out a clinical interpretation of the main symptoms and syndromes in these diseases

formation of the ability to conduct a modern laboratory-instrumental examination of a patient with chronic gastritis, peptic ulcer disease of the stomach and duodenum, chronic hepatitis and cirrhosis of the liver (the applicant must be able to prescribe a plan of laboratory-instrumental examination, give an assessment of the examination results)

3.1. Control materials for the final stage of the lesson:
Situational tasks:
Tasks of the STEP-2 type
1. During the examination, an increase in the abdomen was detected, more in the lower part. The navel is bulging. On the skin of the abdomen, tortuous strands of dilated veins are visible, which run radially from the navel. Percussion - dulling of sound in the lower abdomen. What reason can you think of for the increase in the belly?
A. Flatulence
B. Obesity
+S. Ascites

D. Peritonitis
E. Swelling of the abdominal wall

2. Patient K., 35 years old, complains of poor appetite, nausea, stool disorder, weakness, aching pain in the right hypochondrium, low-grade fever, yellowness of the skin and sclera. Urine is the color of beer, and the stool is discolored. About: an increase in the liver by 4 cm. It is moderately dense, painful. In the urine, bilirubin +++, urobilin +++. In the blood, direct bilirubin is 60 mmol/l, indirect bilirubin is 42 mmol/l.
What disease can be assumed in the patient?
+A - hepatitis
B - gastric ulcer
C - pancreatitis
D - Cholecystitis
E – Gastritis

3. Patient K., 35 years old, complains of poor appetite, nausea, stool disorders, weakness, aching pain in the right hypochondrium, low-grade fever, yellowness of the skin and sclera. Urine is the color of beer, and the stool is discolored. About: an increase in the liver by 4 cm. It is moderately dense, painful. In the urine, bilirubin +++, urobilin +++. In the blood, direct bilirubin is 60 mmol/l, indirect bilirubin is 42 mmol/l.
What type of jaundice does the patient have?
+A - parenchymatous
B - mechanical
C - hemolytic
D is false

4. Patient P., 56 years old, complains of weakness, low-grade fever, nausea, lack of appetite, abdominal distension, constant pain in the right hypochondrium. 12 years ago, he suffered from Botkin's disease, after which dull pains remained in the right hypochondrium. After 7 years, an increase in the liver was detected. About: jaundice, the liver protrudes 5 cm. from under the costal edge, dense, painful. On the skin, individual vascular stars, single small hemorrhages.
What disease could the doctor think of?
+A - cirrhosis of the liver
B - gastric ulcer
C - pancreatitis
D - Cholecystitis
E – Gastritis
6. Patient S., 50 years old, came with complaints of a feeling of weight, sometimes pain in the right hypochondrium, bitterness in the mouth, unstable stool, weakness, quick fatigue. In the course of 10 years, he ate irregularly and drank alcohol. Ob-but: "hepatic palms", the abdomen is enlarged in volume, on the skin the expansion of veins is visible. The liver protrudes 2 cm from under the costal arch, is soft, with a smooth surface, and is sensitive to palpation. The size of the spleen according to Kurlov - 12x18 cm.
What disease can be assumed in the patient?

+A- cirrhosis of the liver
B - gastric ulcer
C - pancreatitis
D - Cholecystitis
E – Gastritis

7. Patient S., 50 years old, came in with complaints of a feeling of heaviness, sometimes pain in the right hypochondrium, bitterness in the mouth, unstable stool, weakness, quick fatigue. In the course of 10 years, he ate irregularly and drank alcohol. Ob-but: "hepatic palms", the abdomen is enlarged in volume, on the skin the expansion of veins is visible. The liver protrudes 2 cm from under the costal arch, is soft, with a smooth surface, and is sensitive to palpation. The size of the spleen according to Kurlov - 12x18 cm.
For which clinical syndrome are these changes characteristic?
A - cholestasis syndrome
+B - portal hypertension syndrome
C - cytolysis syndrome
D - jaundice
E - hepatorenal syndrome

8. The patient was delivered in serious condition. Consciousness is confused. Skin and mucous membranes are jaundiced. Kussmaul's breath. "Hepatic" smell from the mouth. The liver is not enlarged. What syndrome is there?
A. cholestasis syndrome
B - portal hypertension syndrome
C - cytolysis syndrome
+D - hepatocellular failure syndrome
E - hepatorenal syndrome

9. The patient was delivered in serious condition. Consciousness is confused. Skin and mucous membranes are jaundiced. Exhausted. Kussmaul's breath. "Hepatic" smell from the mouth. The abdomen is enlarged, dilated veins are visible on the skin. The liver is not enlarged. Your diagnosis?
+A- cirrhosis of the liver
B - gastric ulcer
C - pancreatitis
D - Cholecystitis
E – Gastritis

Orientation map.

	ts	ns	
	gastritis - etiology, tion, main s.		
	gastritis - data of		

	aboratory and ıtal studies.		
	lcer disease and :er disease - main symptoms, istics of the painful :.		
	lcer disease and :er - clinical, y and instrumental		
	ımors - tion, atology, diagnosis.		

3.2. Requirements for work results, including before registration: substantiation of the diagnosis based on complaints, medical history and life data, clinical and laboratory-instrumental examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (*if necessary*).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

List of recommended literature

Basic:

1. Propedeutics of internal medicine: textbook / Yu.I. Decyk, O.H. Yavorskyi, E.M. Neiko and others; under the editorship O.H. Yavorsky – 6th ed., corr. and added - K.: VSV "Medicine", 2020. - 552 p. + 12 p. color incl.
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.

Additional:

1. Methodology of examination of a therapeutic patient: teaching. manual / S.M. Andreychyn, N.A. Bilkevich, T.Yu. Chernets. – Ternopil: TDMU, 2016. – 260 p.
2. 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.
3. Yepishyn A.V. Propaedeutics of internal diseases with care of therapeutic patients / AV. Yepishyn// K. - 2015. 768p.
4. Kovalova OM. Propedeutics of internal medicine/ OM. Kovalova, NA Safargalina-Kornilova// K.: Medicine 2010 - 750p.
5.Macleod's Clinical Examination / Ed. G.Douglas, F.Nicol, C.Robertson.– 13th ed.– Elsevier. 2013. – 471 p.
6.Bates' Guide to Physical Examination and History Taking /Ed. Lynn S. Bickley, Peter G. Szilagyi. – Wolters Kluwer, 2017. – 1066 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/ - Odesa National Medical University
9. https://onmedu.edu.ua/kafedra/propedevtiki-vnutrishnih-hvorob-ta-terapii/ - Department of propaedeutics of internal diseases and therapyOdessa National Medical University.

Practical lesson No. 32

Topic: Symptoms and syndromes in chronic hepatitis and liver cirrhosis.

Goal: Acquiring knowledge and mastering professional competences during the examination of a patient with chronic hepatitis and cirrhosis of the liver - questioning, physical examination, laboratory-instrumental studies.

Basic concepts: Definition and principles of modern classification of chronic hepatitis and liver cirrhosis. The main etiological factors of the development of hepatitis and liver cirrhosis. The mechanism of liver damage in hepatitis of viral etiology. The main complaints of patients with hepatitis and liver cirrhosis, features of examination results and physical examination data. Morphological and biochemical signs of liver damage. The main complications of liver cirrhosis.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:

1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with the patient with chronic hepatitis and liver cirrhosis for the purpose of collecting complaints and history, physical examination of the patient; evaluation of clinical examination data and laboratory and instrumental data*),*conducting a test control, solving a clinical problem, written solution of Step-2 type problems, face-to-face survey, discussion, role play on the subject of the lesson.

2.1. Requirements for the theoretical readiness of applicants to perform practical classes: the applicant must know the modern definition, etiology, pathogenesis, classifications of chronic hepatitis and liver cirrhosis, subjective and objective data in these diseases to know laboratory and instrumental data for this pathology.

List of didactic units:

- to conduct a subjective examination of patients with chronic hepatitis and liver cirrhosis
- conduct an objective examination of patients with chronic hepatitis and liver cirrhosis
- appoint a plan for additional laboratory and instrumental examinations of patients with chronic hepatitis and cirrhosis of the liver
- evaluate the results of laboratory tests in patients with chronic hepatitis and liver cirrhosis
- master the skills and abilities to assess leading clinical syndromes in patients with chronic hepatitis and liver cirrhosis

2.2. Questions to check basic knowledge on the topic of the lesson:

1. Definition of chronic hepatitis and liver cirrhosis.

2. Classification of chronic hepatitis and liver cirrhosis.

3. Etiology and pathogenesis of chronic hepatitis and liver cirrhosis.

4. Conducting subjective research: clarification of complaints; history taking.

5. The value of methods of objective examination of the digestive organs in the diagnosis of chronic hepatitis and liver cirrhosis:

a) general overview;

b) examination of the oral cavity (mucous membrane, gums, teeth, tongue, tonsils, pharynx);

c) examination of the abdomen (size, configuration, external tumors, hernias, subcutaneous veins, navel, visible peristalsis, participation in breathing);

d) superficial palpation (detection of soreness, resistance, muscle tension, peritoneal symptoms, discrepancy of rectus abdominis muscles, hernias, tumors);

e) deep palpation of the abdomen;

f) determining the size of the liver and spleen according to the Kurlov method;

g) determination of free fluid in the abdominal cavity.

6. Characteristics of these laboratory-instrumental methods of research in chronic hepatitis and liver cirrhosis.

3. Formation of professional skills and abilities:

3.1. Control materials for the final stage of the lesson:
Situational tasks:

Tasks of the STEP-2 type

1. During the examination, an increase in the abdomen was detected, more in the lower part. The navel is bulging. On the skin of the abdomen, tortuous strands of dilated veins are visible, which run radially from the navel. Percussion - dulling of sound in the lower abdomen. What reason can you think of for the increase in the belly?
A. Flatulence
B. Obesity
+S. Ascites
D. Peritonitis
E. Swelling of the abdominal wall

2. Patient K., 35 years old, complains of poor appetite, nausea, stool disorder, weakness, aching pain in the right hypochondrium, low-grade fever, yellowness of the skin and sclera. Urine is the color of beer, and the stool is discolored. About: an increase in the liver by 4 cm. It is moderately dense, painful. In the urine, bilirubin +++, urobilin +++. In the blood, direct bilirubin is 60 mmol/l, indirect bilirubin is 42 mmol/l.
What disease can be assumed in the patient?

+A - hepatitis
B - gastric ulcer
C - pancreatitis
D - Cholecystitis
E – Gastritis

3. Patient K., 35 years old, complains of poor appetite, nausea, stool disorders, weakness, aching pain in the right hypochondrium, low-grade fever, yellowness of the skin and sclera. Urine is the color of beer, and the stool is discolored. About: an increase in the liver by 4 cm. It is moderately dense, painful. In the urine, bilirubin +++, urobilin +++. In the blood, direct bilirubin is 60 mmol/l, indirect bilirubin is 42 mmol/l.
What type of jaundice does the patient have?
+A - parenchymatous
B - mechanical
C - hemolytic
D is false

4. Patient P., 56 years old, complains of weakness, low-grade fever, nausea, lack of appetite, abdominal distension, constant pain in the right hypochondrium. 12 years ago, he suffered from Botkin's disease, after which dull pains remained in the right hypochondrium. After 7 years, an increase in the liver was detected. About: jaundice, the liver protrudes 5 cm. from under the costal edge, dense, painful. On the skin, individual vascular stars, single small hemorrhages.
What disease could the doctor think of?
+A - cirrhosis of the liver
B - gastric ulcer
C - pancreatitis
D - Cholecystitis
E – Gastritis
6. Patient S., 50 years old, came with complaints of a feeling of weight, sometimes pain in the right hypochondrium, bitterness in the mouth, unstable stool, weakness, quick fatigue. In the course of 10 years, he ate irregularly and drank alcohol. Ob-but: "hepatic palms", the abdomen is enlarged in volume, on the skin the expansion of veins is visible. The liver protrudes 2 cm from under the costal arch, is soft, with a smooth surface, and is sensitive to palpation. The size of the spleen according to Kurlov - 12x18 cm.
What disease can be assumed in the patient?
+A- cirrhosis of the liver
B - gastric ulcer
C - pancreatitis
D - Cholecystitis
E – Gastritis

7. Patient S., 50 years old, came in with complaints of a feeling of heaviness, sometimes pain in the right hypochondrium, bitterness in the mouth, unstable stool, weakness, quick fatigue. In the course of 10 years, he ate irregularly and drank alcohol. Ob-but: "hepatic palms", the

abdomen is enlarged in volume, on the skin the expansion of veins is visible. The liver protrudes 2 cm from under the costal arch, is soft, with a smooth surface, and is sensitive to palpation. The size of the spleen according to Kurlov - 12x18 cm.
For which clinical syndrome are these changes characteristic?
A - cholestasis syndrome
+B - portal hypertension syndrome
C - cytolysis syndrome
D - jaundice
E - hepatorenal syndrome

8. The patient was delivered in serious condition. Consciousness is confused. Skin and mucous membranes are jaundiced. Kussmaul's breath. "Hepatic" smell from the mouth. The liver is not enlarged. What syndrome is there?
A. cholestasis syndrome
B - portal hypertension syndrome
C - cytolysis syndrome
+D - hepatocellular failure syndrome
E - hepatorenal syndrome

9. The patient was delivered in serious condition. Consciousness is confused. Skin and mucous membranes are jaundiced. Exhausted. Kussmaul's breath. "Hepatic" smell from the mouth. The abdomen is enlarged, dilated veins are visible on the skin. The liver is not enlarged. Your diagnosis?
+A- cirrhosis of the liver
B - gastric ulcer
C - pancreatitis
D - Cholecystitis
E – Gastritis

3.2. Requirements for work results, including before registration: substantiation of the diagnosis based on complaints, medical history and life data, clinical and laboratory-instrumental examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (*if necessary*).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 33

Topic: Main symptoms and syndromes in kidney diseases - acute and chronic glomerulonephritis.

Goal: Acquiring knowledge and mastering professional competences during the examination of a patient with acute and chronic glomerulonephritis - questioning, physical examination, laboratory-instrumental studies.

Basic concepts: Definition and modern classification of glomerulonephritis. The main mechanisms of development of glomerulonephritis. Complaints of patients with kidney damage and results of physical examination of patients with glomerulonephritis. Edema syndrome and arterial hypertension syndrome in kidney diseases. Possibilities of instrumental diagnosis of kidney pathology. Laboratory examination of urine, analysis and interpretation of the results of general clinical analysis of urine, examination of urine according to Nechiporenko, Ambyurzhe, Adis-Kakovskyi, Zimnytskyi. Urinary, nephrotic syndromes in kidney diseases. The results of a biochemical study of blood in kidney pathology. Renal failure and renal colic syndromes. Definition and classification of chronic kidney disease.

Glomerulonephritis

Bilateral kidney disease of an immune-inflammatory nature with predominant damage to the glomeruli and involvement of the renal tubules, interstitium, and renal vessels in the pathological process.

According to modern ideas, GN is a heterogeneous group of diseases that includes many different pathological conditions in terms of etiology, pathogenesis, morphology, and prognosis. In most cases, this term refers to an independent kidney disease, however, GN is quite often a consequence of kidney damage in other diseases (collagenosis). Cases of HH are observed less often than HC.

In Ukraine, the classification of GN is accepted (L.A. Pyrig et al., 1995), according to which clinical forms of glomerulonephritis are distinguished:

acute (with a protracted course when the disease lasts more than 4 months),

subacute (rapidly progressing with pre-azotemic and azotemic stages),

chronic GN,

Stages of chronic GN:

prehypertensive (antihypertensive at the suggestion of T.D. Nikula (1981),

hypertensive

the stage of renal failure (its four stages are distinguished);

Course options:

Urinary syndrome;

l Nephrotic syndrome;

Phases of chronic glomerulonephritis:

Aggravation;

Remissions.

Acute glomerulonephritis is the most common clinical form of diffuse immune-inflammatory kidney damage, which is characterized by a first-onset acute nephritic syndrome that developed after a streptococcal or other infection.

GG occurs more often in children aged 3 to 7 and in adults aged 20-40, more often in men, mainly in the cold season.

Its reason is, as a rule,

streptococcal infection (β-hemolytic streptococcus group A, nephritogenic strains of streptococcus 1, 4, 8, 12, 49)

staphylococcal infection,

less often***viruses***(hepatitis B, C, chicken pox, measles, Coxsackie, rubella, HIV, influenza. Therefore, acute glomerulonephritis begins mainly after angina, pharyngitis, endocarditis, scarlet fever, pneumonia, influenza, acute respiratory syndrome.

Acute hypothermia of the body contributes to the occurrence of the disease, especially in conditions of high air humidity.

Vaccination is also one of the factors in the occurrence of GG, and in 2/3 of cases the lesion occurs after 2 or even 3 injections of the vaccine.

The disease develops under the influence of infection against the background of a particularly changed sensitivity of the body. The basis of the disease is the process of deposition of immune complexes and complement components in the kidney glomeruli, which is accompanied by a violation of microcirculation in the kidneys, the occurrence of immune inflammation, destruction of the walls of glomerular capillaries, infiltration of kidney glomeruli by neutrophils and monocytes. In the emergence of GG, the infection plays the role of only a "starting mechanism". The disease develops as a kind of chain reaction, which the infection can only support, although not always.

The occurrence and progression of glomerulonephritis can lead to the presence of**foci of chronic infection in the body**: carious teeth, inflammation of the tonsils (tonsillitis), additional sinuses (sinusitis, frontitis), as well as cooling, susceptibility to allergic reactions, overtiredness.

Acute glomerulonephritis usually develops 8-14 days after an infectious disease, less often "for no reason".

So: HH can be suspected if erythrocytes and protein are detected in the urine in combination with hypertension and edema 1-4 weeks after a streptococcal or other infection.

Often develops**acute and chronic forms of glomerulonephritis**, less often - **rapidly progressive and subacute** (malignant).

In approximately half of the cases, the disease is characterized***a decrease in the amount of urine, a change in its color ("meat slop"), swelling of the body that occurs quickly, sometimes over several hours and is localized on the face, trunk, limbs***. They not only appear quickly, but also disappear within 10-14 days. Swelling can be from minor on the face to widespread throughout the body, edema of the chest, abdomen. Sometimes ascites, hydrothorax, hydropericardium develops. It is also observed***increased blood pressure (hypertension).*** With a favorable course, the pressure normalizes in 3-4 weeks. Long-term and persistent hypertension is a poor prognostic sign. Heart rate slowed down for 2-3 weeks. This is an important diagnostic feature. Tachycardia is noted in most diseases that are accompanied by edema. During the examination, pallor of the skin, puffiness of the face, cyanosis of the lips, and acrocyanosis are determined. Often the patient does not notice the onset of the disease. General malaise, weakness, fatigue, undefined pain in the lower back can be considered as residual effects of a cold. In such cases, the diagnosis of acute glomerulonephritis can be made only on the basis of the result of a urine analysis. This should be remembered, especially since such patients sometimes feel practically healthy, often ignore the doctor's instructions and advice, do not follow the appropriate regime, diet, are not treated, and refuse control examinations.

So: characteristic**diagnostic criteria** acute glomerulonephritis - hypertension, edema, bradycardia.

Laboratory studies

- **In the clinical analysis of urine** reveal a combination*proteinuria* (from 1 to 3 g/day for 2-8 weeks) and*hematuria* (in the field of vision 5-50 erythrocytes, urine takes on the appearance of "meat slops"), expressed at the beginning of the disease, which gradually decreases after a few days.

IN**general blood analysis** they reveal mild anemia (decrease in the number of erythrocytes and hemoglobin), moderate leukocytosis, eosinophilia, lymphopenia, an increase in ESR up to 20-50 mm/h.

In biochemical analysis reveal hypoproteinemia (60 g/l and below), dysproteinemia, the appearance of C-reactive protein, accumulation of sialic acids, an increase in the titer of antistreptolysin antibodies, accumulation of creatinine, urea, residual nitrogen, hypercholesterolemia, hyperlipidemia.

Special studies

- A biopsy followed by a pathomorphological examination of the nephrobioptate allows a nosological diagnosis to be made.

- Ultrasound of the kidneys: contours are smooth, dimensions are unchanged and increased (with ARF), echogenicity is reduced.

- ECG in hypertension reveals overload of the left ventricle and possible rhythm disturbances.

Forecast. Recovery in 85-90% of cases.

Complications of GG:

1.-eclampsia (angiospastic encephalopathy);.

2.- acute heart failure;

3.-acute renal failure

Subacute and rapidly progressing glomerulonephritis are severe forms of the disease and are relatively rare. Often, their occurrence is due to untimely treatment, non-compliance with its methodology, violation of the diet, regime in more benign forms of glomerulonephritis. In 20-80% of cases, GG becomes chronic.

Chronic glomerulonephritis

HG is a group concept that includes diseases of the glomeruli with a common, most often immune mechanism of damage with a gradual decrease in kidney function and the development of kidney failure.

A distinction is made between primary-chronic and secondary-chronic glomerulonephritis (with nodular arteritis, systemic lupus erythematosus).

Chronic glomerulonephritis it is diagnosed in the event that 2-3 months after GG there is a mildly expressed urinary syndrome or swelling, an increase in blood pressure.

Primary chronic glomerulonephritis they are usually detected under random circumstances (urine examination or pressure measurement when issuing a sanatorium-resort card, military commission, etc.). Often, this type G is established already in the stage of chronic obstructive pulmonary disease.

Signs of CG are diverse and depend on the clinical form, stage of the disease, and the state of renal function.

There are nephrotic, hypertensive, mixed, latent, hematuric forms of the disease.

Latent hematuric forms are characterized by a benign gradual course. CKD does not develop with these forms for many years - 10-20 years or more. The only manifestation may be changes detected in the an. urine (slight proteinuria, erythrocyturia, cylindruria), the patient has no unpleasant sensations, there is no edema. Only slight puffiness under the eyes can be determined. Blood pressure is normal. If appropriate treatment and prevention recommendations are followed, this condition can last for years and decades. The occurrence of hypertension with HCG is accompanied by headache, dizziness, shortness of breath. The development of hypertension with HCG indicates the transition from the antihypertensive stage of the disease to the hypertensive stage, in which the patient must adhere more strictly to the regime and diet, constantly take medication for hypertension. It should be noted that with kidney diseases, hypertension is much easier to tolerate than with hypertension. Therefore, hypertension of renal origin is often detected during random blood pressure measurement.

mixed the form is characterized by the most severe course and rapid progression. Symptoms of chronic renal failure appear 5-7 years after the onset of the disease. It is characterized by a combination of nephrotic and hypertensive syndromes (massive proteinuria, hypo- and dysproteinemia, increased blood pressure, fundus changes, left ventricular hypertrophy).

ChG can manifest itself in a variant with urinary or nephrotic syndrome.

For**nephrotic**the syndrome is characterized by swelling of the body. increased excretion of protein in the urine (more than 3.5-4 g/day), changes in some blood parameters. The nephrotic variant of HCG is more dangerous, it progresses faster, and is accompanied by a significant decrease in the body's resistance to infections due to a decrease in the level of gamma globulins. Intercurrent diseases (furunculosis, pneumonia, dysentery) are poorly treated, they contribute to the progression of the disease and often cause the death of patients. Therefore, such patients require close supervision and treatment. Infectious diseases, hypothermia, surgical interventions can be the reasons for the transition of urinary syndrome to nephrotic syndrome.

At**hypertensive** the form of progression of the disease is determined by the severity of hypertension and the presence of cerebral and cardiac complications - angina pectoris, arrhythmia, HF, eye disorders (retinopathy), encephalopathy.

There is almost no complete recovery from HCG. The main task of treatment is to slow down and stabilize the course of the disease, to preserve the patient's ability to work and live comfortably.

Mortality from this disease depends on the rate of development of kidney failure. Death is possible from uremia, as well as from complications: hypovolemic shock, nephrotic crisis, thrombosis, severe infections.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:
1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with a patient with acute and chronic glomerulonephritis, for the purpose of collecting complaints and anamnesis, physical examination of the patient; evaluation of clinical examination data and laboratory and instrumental data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.
2.1. Requirements for the theoretical readiness of applicants to perform practical classes: the applicant must know the modern definition, etiology, pathogenesis, classifications of acute and chronic glomerulonephritis, subjective and objective data in these diseases, know laboratory and instrumental data in this pathology.

List of didactic units:
- to conduct a subjective examination of patients with acute and chronic glomerulonephritis
- carry out an objective examination of patients with acute and chronic glomerulonephritis appoint a plan of additional laboratory and instrumental examinations of patients with acute and chronic glomerulonephritis evaluate the results of laboratory tests in patients with acute and chronic glomerulonephritis possess the skills and abilities to assess leading clinical syndromes in patients with acute and chronic glomerulonephritis

2.2. Questions to check basic knowledge on the topic of the lesson:
1. Concept of acute and chronic glomerulonephritis.

2. Give brief information about the etiology, pathogenesis, clinical picture and course of HGN.

3. Characteristics of the main symptoms and syndromes of HGN.

4. Changes in the general blood analysis and biochemical blood test results of patients with HGN and HGN.

5. Changes in the general analysis of urine and functional tests in patients with glomerulonephritis.

6. Instrumental research methods used in patients with glomerulonephritis.

7. Give a clinical assessment of the results of blood, urine, instrumental methods of research in patients with kidney pathology.

3. Formation of professional skills and abilities:

mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the interview)

formation of the ability to perform a clinical examination of a patient with acute and chronic glomerulonephritis (the applicant must be able to perform a physical examination of the patient),

formation of the ability to evaluate the data obtained during the clinical examination of a patient with acute and chronic glomerulonephritis;
carry out a clinical interpretation of the main symptoms and syndromes in these diseases

formation of the ability to conduct a modern laboratory-instrumental examination of a patient with acute and chronic glomerulonephritis (the applicant must be able to prescribe a plan of laboratory-instrumental examination, give an assessment of the examination results)

3.1. Control materials for the final stage of the lesson:
Situational tasks:

Tasks of the STEP-2 type

1. An 18-year-old patient developed swelling, headaches, and vomiting 3 times during the day, 2 weeks after angina. About: swelling of the face, eyelids, blood pressure increase, urine the color of "meat slop".

What is the most likely diagnosis?

A. Urethritis.

+V. Glomerulonephritis.

S. Cystitis.

D. Pyelonephritis.

E. Urinary stone disease.

Task 2. A 60-year-old patient was prescribed gentamicin (80 mg every 6 hours) due to fever after cholecystectomy. After 10 days, the patient's creatinine increased to 310 μmol/l. AT-130/80 mm Hg. Daily diuresis - 1.2 l. Urinalysis without pathology. Ultrasound of the buds: the size of the buds is normal. What is the most likely cause of kidney failure?

A. Acute glomerulonephritis.

B. Hepatorenal syndrome.

S. Inadequate fluid infusion.

D. Cortical necrosis of the kidneys.

+E. Nephrotoxicity of gentamicin.

Task 3. A 40-year-old patient complains of pronounced swelling of the face, trunk, limbs, and general weakness. Suffers from chronic osteomyelitis of the left lower leg. Ob-no - paleness of the skin, swelling of the whole body. Swellings are soft, shift when changing body position. Blood pressure -105/70 mm Hg. In urine - body weight 1030, proteinuria 9.9 g/l, cylinders - epithelial, granular, waxy 6-8 in p/zr. Hypoproteinemia in the blood (total protein 42 g/l). What syndrome should you think about?

A. Hypertensive.

V. Hematuric.

+S. Nephrotic.

D. Anemic.

E. Azotemic.

Task 4. The patient is worried about weakness, shortness of breath, thirst. History of chronic tonsillitis. About - the skin is pale, dry. Blood pressure 180/110 mm Hg. HR-96 in 1 min. The heart is enlarged to the left, accent A 2. In the general blood analysis: ER-2.4 T/l, Hv-80 g/l, ESR-50 mm/h. In the analysis of urine: urine weight-1006, protein-0.99 g/l, leukocytes-2-3 in p/zr., er.-15-20 in p/zr, leached. Serum creatinine is 752 μmol/l. Which diagnosis is more likely?

+A. Chronic glomerulonephritis, CKD III stage.

B. Acute glomerulonephritis, nephrotic syndrome.

S. Hypertensive disease.

D. Chronic glomerulonephritis.

E. Hemorrhagic vasculitis, renal form.

Task 5. A 24-year-old patient complains of swelling of the face and eyelids. In the anamnesis, a sore throat was transferred 2 weeks ago. General analysis of urine - specific gravity 1018, protein 4.2 g/l, er. Leached 20-30 in p/zr, hyaline, waxy, epithelial cylinders 8-10 in p/zr. In blood serum, cholesterol is 7.2 mmol/l, total protein is 54 g/l, albumin is 32.1 g/l. Your diagnosis?

+A. Acute glomerulonephritis, nephrotic syndrome.

B. Acute pyelonephritis.

S. Acute glomerulonephritis, hematuric syndrome.

D. Chronic pyelonephritis.

E. Glomerulosclerosis.

Task 6. An 18-year-old patient complains of an increase in blood pressure to 200/120 mm Hg. discharge of large amounts of urine at night. There is a history of frequent sore throats. General analysis of urine - ud. weight 1008, protein-0.99 g/l, leached erythrocytes - 8-14 in p/zr, hyaline, waxy cylinders - 5-7 in p/zr. Changes in urine were detected for the first time 10 years ago. Your diagnosis?

A. Hypertensive disease

B. Chronic pyelonephritis, arterial hypertension.

+S. Chronic glomerulonephritis, secondary arterial hypertension.

D. Renal amyloidosis.

E. Metabolic nephropathy.

Task 7. A 17-year-old patient complains of dull pain in the lumbar region. It is known from the anamnesis that she suffered from angina 10 years ago. General analysis of urine: body weight 1017, protein 1.65 g/l, erythrocytes leached 10-15 in p/zr, hyaline cylinders 8-10 in p/zr. Serum creatinine-90 μmol/l, urea-5.3 mmol/l. What is the most likely diagnosis?

+A. Acute glomerulonephritis, urinary syndrome.

B. Acute glomerulonephritis, nephrotic syndrome.

S. Acute pyelonephritis.

D. Chronic pyelonephritis.

E. Kidney amyloidosis.

Task 8. A 24-year-old patient has been suffering from systemic lupus erythematosus for 10 years. General analysis of urine - weight 1012, white - 6.6 g/l, er. leached 10-14 in p/zr, hyaline cylinders 6-10 in p/zr, waxy, epithelial - 2-4 in p/zr. According to which of the specified options, kidney disease developed?

A. Chronic glomerulonephritis.

+V. Lupus-nephritis, nephrotic syndrome.

S. Lupus amyloidosis.

D. Pyelonephritis.

E. Metabolic nephropathy.

9. A 26-year-old patient complains of pain in the lumbar region. For 2 years, urine tests reveal hypoisosthenuria. What test detects the concentration function of the kidneys?

A. General analysis of urine.

V. Three-glass test.

+S. Zimnytsky's trial.

D. Nechiporenko's test.

E. Addis-Kakovsky sample.

Task 10. A 28-year-old patient will be examined in connection with urinary syndrome: ud. weight 1010, protein-0.66 g/l, leukocytes 4-6 in p/zr, erythrocytes leached 20-30 in p/zr, hyaline cylinders 2-4 in p/zr. In the test according to Zimnytskyi, the weight is 1009-1013. Serum creatinine - 420 μmol/l, creatinine clearance (glomerular filtration) - 40 mol/min. Your diagnosis?

A. Acute glomerulonephritis.

B. Acute pyelonephritis.

C. Chronic glomerulonephritis without chronic renal failure.

D. Chronic glomerulonephritis, chronic obstructive pulmonary disease 1 st.

+E. Chronic glomerulonephritis, CKD III stage.

3.2. Requirements for work results, including before registration: substantiation of the diagnosis based on complaints, medical history and life data, clinical and laboratory-instrumental examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (*if necessary*).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 34
Topic: Main symptoms and syndromes in kidney diseases - acute and chronic pyelonephritis.

Goal: Acquiring knowledge and mastering professional competences during the examination of a patient with acute and chronic pyelonephritis - questioning, physical examination, laboratory-instrumental studies.

Basic concepts: Definition and modern classification of pyelonephritis. The main mechanisms of development of pyelonephritis. Complaints of patients with kidney damage and results of physical examination of patients with pyelonephritis. Edema syndrome and arterial hypertension syndrome in kidney diseases. Possibilities of instrumental diagnosis of kidney pathology. Laboratory examination of urine, analysis and interpretation of the results of general clinical analysis of urine, examination of urine according to Nechiporenko, Ambyurzhe, Adis-Kakovskyi, Zimnytskyi. Urinary, nephrotic syndromes in kidney diseases. The results of a biochemical study of blood in kidney pathology. Renal failure and renal colic syndromes. Definition and classification of chronic kidney disease.

Pyelonephritisis a non-specific infectious and inflammatory disease of the kidneys with damage to the calyces and bowls, tubules, connective tissue base of the kidneys with subsequent damage to blood vessels and glomeruli.

Primary pyelonephritis develops against the background of unchanged urine outflow, accounting for 20% of all cases.**Secondary** pyelonephritis caused by urinary stasis is observed in 80% of patients.

The second most common kidney disease. Pyelonephritis is the most common disease of the kidneys and urinary tract, second in prevalence after upper respiratory tract infection. Pyelonephritis is much more common than glomerulonephritis. In women, this disease is observed 3-4 times more often than in men.

Pyelonephritis is caused by various microorganisms, in particular Escherichia coli. Bacteria enter the kidney via lymphatic vessels. From the urinary tract, as well as from nearby organs (the appendix, intestines, genitals). Microorganisms can enter the kidneys from distant foci of infection (tonsils, additional sinuses, carious teeth, bile ducts).

The possibility of development of pyelonephritis is increased by the presence of obstacles to the outflow of urine - stones, scars, bends of the ureters, prolapse of the kidney, congenital anomalies, enlargement of the prostate gland. The possibility of pyelonephritis is increased by diseases of the circulatory system, diabetes, various debilitating diseases, hypothermia, constipation, abuse of some drugs (diuretics, phenacetin, acetylsalicylic acid, analgin, amidopyrine), as well as pregnancy, which causes a violation of urine outflow. . Pyelonephritis can be acute or chronic, unilateral or bilateral.

Acute pyelonephritis is an acute inflammatory process in the renal parenchyma and glomerular system.

Clinical manifestations:

Intoxication syndrome. The onset of pyelonephritis resembles an acute infectious disease: the temperature suddenly rises, chills appear, general weakness, muscle pain, joint pain, headache, often accompanied by nausea, vomiting, clear pulse and breathing, thirst. Diarrhea often reaches 39-40-41°C, constant or hecteric type. Profuse sweats appear 1-2 hours after the onset of prolapse. After sweating, the temperature drops slightly, sudden weakness, adynamia, and hypotension may develop.

Local inflammation syndrome. Local signs of the inflammatory process appear - pains in the lower back of varying intensity, in the area of the kidneys, which spread downwards to the inguinal and suprapubic areas. Irradiation to the upper abdomen, left hypochondrium, and back is rarely observed. The pains worsen at night, when coughing, when moving the corresponding leg.

Dysuric syndrome. Pain is accompanied by dysuric phenomena - heartburn, cutting, frequent urges to urinate.

When examining patients with acute pyelonephritis, they are found**signs of general intoxication syndrome** - facial hyperemia, sweating, muscle soreness, as well as hysterical sclera.

During bimanual palpation, tenderness is determined in the hypochondrium and lumbar region. Muscle tension, increased muscle tone in the lumbar region and hypochondrium on the affected side may be detected. Pasternacki's symptom is positive or sharply positive in most cases, but it can also be negative. In the first days of the disease, an enlarged, painful kidney is palpated.

Data of additional examination methods.

In the general blood test pronounced leukocytosis up to 30-40x10·9/l, a neutrophilic shift of the leukocyte formula to the left with an increase in the number of rod-nucleated leukocytes, the appearance of young forms is observed. A characteristic increase in ESR up to 40-80 mm/h, a possible decrease in hemoglobin.

In the general analysis of urine - proteinuria and leukocyturia (the most characteristic laboratory and signs of acute glomerulonephritis)

Proteinuria in most cases does not exceed 1.0 g/l. Sometimes it reaches 2-3 g/l or is completely absent.

Leukocytes (pyuria) are the most characteristic feature of pyelonephritis. Leukocytosis can be determined by separate clusters when examined under a microscope or cover the entire field of view. Leukocyturia may be transient. A normal content of leukocytes in the urine is possible in the case of obstruction of the outflow tract from the affected kidney - blockage of the ureter by a stone or tumor.

Erythrocyturia is not always observed. The cause of erythrocyturia is stone trauma or necrosis of the renal papillae.

Cylindruria is found in the severe course of the disease. Granular or waxy cylinders are characteristic.

Microbiological examination of urine. Bacteriuria is the characteristic and earliest laboratory sign of acute pyelonephritis, and is found in most cases. The diagnosis of GP is confirmed only by true bacteriuria - at least 50-100 thousand microbial bodies in 1 ml of urine.

Violation of the nitrogen-excreting function of the kidneys.

Immunological disorders

Survey urography - reveals the presence of radiopaque stones and their localization.

Excretory urography makes it possible to detect concretions and signs that are not detected during the examination, dyskinesia of the urinary tract (pelvis and ureters), their expansion and narrowing.

Ascending pyelography is used very rarely, as it can worsen the course of the disease.

Radioisotope rheography =- allows you to assess the functional state of both kidneys.

Radioisotope scanning.

Ultrasound of the kidneys.

Thermography

Endoscopic examination (cystoscopy, chromocystoscopy).

Clinical forms of acute pyelonephritis

- Apostematous nephritis
- Kidney abscess
- Kidney carbuncle.
- Necrosis of renal papillae

CHRONIC PYELONEPHRITIS

Most often, it is a consequence and continuation of acute.

Clinical manifestations of CP can be quite diverse. They depend on the spread of the process (unilateral or bilateral), the presence or absence of urodynamic disorders, the virulence of the pathogen, and the state of the body's immunological reactivity.

Clinical and laboratory signs of chronic pyelonephritis are most pronounced in the period of exacerbation of the disease and insignificant in the period of remission. In some cases, CP, like glomerulonephritis, can develop from the very beginning as a primary-chronic process, it is detected during a random urinalysis or blood pressure measurement. Not experiencing any disturbances, patients do not consult a doctor for a long time.

Suspect HP it is possible on the basis of periodic, seemingly "causeless", sudden increase in body temperature, which sometimes lasts even half a day. After that, patients do not always find it necessary to seek medical help. It also happens that an increase in body temperature up to 37.2-37.50C is observed for weeks and months. In such cases, pain in the lower back area is interpreted as a manifestation of sciatica, and decreased work capacity, appetite, weakness, and rapid fatigue are signs of overfatigue. In the case of pain in the lower back, especially one-sided, cutting and pain during urination, frequent urges to it, the patient should be examined in the laboratory. Unpleasant sensations during the act of urination are associated with the fact that pyelonephritis often leads to inflammatory processes in other parts of the urinary system. in the later stages of CP, patients develop hypertension, which is accompanied by a headache, unpleasant sensations in the area of the heart, and shortness of breath. At the same time, anemia, pallor of the skin, shortness of breath may develop. in patients with primary chronic pyelonephritis, anemia and hypertension are the first signs of the disease that force the patient to consult a doctor.

Clinical and laboratory manifestations of CHD resemble acute pyelonephritis. An increase in body temperature appears, often up to 38-39 0C, the general condition worsens, there are pains in the lumbar region, dysuric phenomena, anorexia, nausea, vomiting, abdominal pain, headaches.

SYNDROMES

General intoxication (sometimes the only manifestations of the disease) - fatigue, sweating, headaches, lack of appetite, weight loss, monotonous subfibrillation. With a long course of CP, signs of intoxication acquire a persistent character - constantly increased fatigue, reduced work capacity, lethargy, drowsiness, periodic headaches, decreased appetite, weight loss. Constant dyspeptic phenomena appear with the addition of CNN. Body temperature is normal, sometimes in the evening it rises to subfebrile numbers.

Local pain syndrome. (characteristic localization and irradiation).

Dysuric phenomena - x-on polyuria with the release of 2-3 liters of urine per day, nocturia, caused by a violation of the concentration ability of the kidneys.

Arterial hypertension in 10% of patients with CP, there is an increase in blood pressure, changes in the vessels of the fundus, hypertrophy of the left ventricle, hypertension is more

often transient, and in CKD it becomes persistent. Adding hypertension disrupts the course of the disease. LV hypertrophy, LV overload, ischemia, angina attacks develop.

Physical examination: puffiness of the face, pastiness or puffiness of the eyelids, especially under the eyes. Sometimes swelling on the neck. Typical morning swelling maximum. Pale skin. When CNN joins - dry skin, grayish-yellow, earthy color. Swelling is growing. The tongue is dry, with a dirty brown coating, the mucous membrane of the lips is dry and rough. Mr. Pasternacki is positive, but not always.

Additional examination methods.

Clinical blood analysis: leukocytosis, accelerated ESR. The severity of the changes depends on the manifestations of the inflammatory process. Normochromic anemia, increased number of reticulocytes, anemia resistant to treatment.

Clinical analysis of urine: proteinuria (>1 g/l), leukocyturia, active leukocytes, hyaline and granular cylinders, hematuria are detected.

Examination of daily urine for protein - in typical cases does not exceed 1g/l, with nephrotic syndrome - >3g/l.

Samples of Nechiporenko, Kakovsky-Addis -

Who's winter rehearsal?

Bacteriological examination of urine

Immunological studies.

Biochemical studies:

Chronic kidney disease is a disease characterized by long-term (at least 3 months) structural and/or functional renal changes according to clinical, laboratory, instrumental, and morphological studies, which at the same time provide grounds for excluding the acute nature of the pathological process in the kidneys.
So, the criteria for defining CKD are:
1) kidney damage lasting more than 3 months, the manifestations of which are structural or functional disorders of the organ with the presence/absence of a decrease in the glomerular filtration rate (GFR). The lesion is manifested by pathomorphological changes in kidney tissue or changes in blood or urine;
2) GFR < 60 ml/min/1.73 m^2lasting 3 months or more in the absence of other signs of kidney damage.

Five stages are distinguished in the course of CKD:
• CKD I: kidney damage with normal or increased GFR (90 ml/min/1.73 m^2);
• CKD II: kidney damage from CKD with a moderate decrease in GFR (60-89 ml/min/1.73

m^2);
• CKD III: kidney damage from CKD with an average degree of GFR reduction (30-59 ml/min/1.73 m^2);
• CKD IV: kidney damage from CKD with a significant decrease in GFR (15-29 ml/min/1.73 m^2);
• CKD V: kidney damage with terminal CKD (< 15 ml/min/1.73 m^2).

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:
1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with a patient with acute and chronic pyelonephritis in order to collect complaints and anamnesis, physical examination of the patient; evaluation of clinical examination data and laboratory and instrumental data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.
2.1. Requirements for the theoretical readiness of applicants to perform practical classes: the applicant must know the modern definition, etiology, pathogenesis, classifications of acute and chronic pyelonephritis, subjective and objective data in these diseases, know laboratory and instrumental data in this pathology.

List of didactic units:
- to conduct a subjective examination of patients with acute and chronic pyelonephritis
- conduct an objective examination of patients with acute and chronic pyelonephritis
- appoint a plan for additional laboratory and instrumental examinations of patients with acute and chronic pyelonephritis
- evaluate the results of laboratory tests in patients with acute and chronic pyelonephritis
- master the skills and abilities to assess leading clinical syndromes in patients with acute and chronic pyelonephritis

2.2. Questions to check basic knowledge on the topic of the lesson:
1. Concept of acute and chronic pyelonephritis.

2. Give brief information about the etiology, pathogenesis, clinical picture and course of acute pyelonephritis.

3. Characteristics of the main symptoms and syndromes of HGN.

4. Changes in the general blood analysis and the results of the biochemical blood test of patients with acute and chronic pyelonephritis.

5. Changes in the general analysis of urine and functional tests in patients with glomerulonephritis.

6. Instrumental research methods used in patients with glomerulonephritis.

7. Concept of acute and chronic pyelonephritis.

8. To describe subjective and objective methods of examination of acute and chronic pyelonephritis.

10. Give a clinical assessment of the results of blood, urine, instrumental methods of research of patients with kidney pathology.

3. Formation of professional skills and abilities:

mental examination, give an assessment of examination results)

3.1. Control materials for the final stage of the lesson:
Situational tasks:

Task 1. Gentamicin (80 mg every 6 hours) was prescribed to a 60-year-old patient after cholecystectomy due to fever. After 10 days, the patient's creatinine increased to 310 μmol/l. AT-130/80 mm Hg. Daily diuresis - 1.2 l. Urinalysis without pathology. Ultrasound of the buds: the size of the buds is normal. What is the most likely cause of kidney failure?

A. Acute glomerulonephritis.

B. Hepatorenal syndrome.

S. Inadequate fluid infusion.

D. Cortical necrosis of the kidneys.

+E. Nephrotoxicity of gentamicin.

Task 2. A 40-year-old patient complains of severe pain in the lumbar region on the left, radiating to the left inguinal region, an increase in t (38, cloudy urine. During the examination, pallor of the skin, swelling of the eyelids were revealed. Pasternacki's symptom on the left sharply (+). In the urine - specific gravity 1020, protein 0.99 g/l, pyuria (leukocytes 100-120 in n/zr), fresh erythrocytes 20-30 in n/zr. What is your diagnosis?
Correct answer Urinary stone disease, secondary pyelonephritis. IN Chronic glomerulonephritis.

S Chronic pyelonephritis.

D Congestive kidney.

AND Urinary stone disease.

Task 3 During the X-ray examination, patient K., 58 years old, showed a decrease in the size of one of the kidneys. This can be beneficial:

Right answer chronic pyelonephritis

IN acute pyelonephritis

S the presence of cysts in the kidney tissue

D amyloidosis of the kidneys

AND acute glomerulonephritis

3.2. Requirements for work results, including before registration: substantiation of the diagnosis based on complaints, medical history and life data, clinical and laboratory-instrumental examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (*if necessary*).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 35
Subject: Symptoms and syndromes in anemia.
Goal: Acquiring knowledge and mastering professional competences during examination of a patient with anemia - questioning, physical examination, laboratory-instrumental research.

Basic concepts:
Definition and modern classification of anemias. Basic laboratory criteria of anemia. The mechanism of iron deficiency in the body and the occurrence of iron deficiency anemia. Main clinical manifestations of sideropenic and general hypoxic syndromes in iron deficiency anemia. Laboratory criteria of iron deficiency anemia. Causes and pathogenesis of V_{12}- folic acid deficiency anemia. Manifestations of the general anemic syndrome, syndromes of damage to the digestive organs, funicular myelosis and peripheral blood damage in B_{12}- folic acid deficiency anemia. The main laboratory signs of V_{12}- folic acid deficiency anemia. Congenital and acquired hemolytic anemias: manifestations of general anemia, jaundice syndromes, splenomegaly and hemosiderosis of internal organs. Basic laboratory criteria of hemolytic anemias and features of bilirubin metabolism disorders. Analysis and interpretation of a general clinical blood test.
The main components of the blood coagulation system. Development factors of bleeding and causes of hemorrhagic syndromes - thrombocytopenia, coagulopathy, hemorrhagic vasculitis. Characteristics of the hemorrhagic syndrome in hemophilia, thrombocytopenic

purpura and Schönlein-Henoch disease. Manifestations of articular, abdominal, renal and anemic syndromes in these diseases. Basic methods of laboratory diagnosis of hemorrhagic syndromes.

Anemia is a clinical and hematological syndrome characterized by a decrease in the content of hemoglobin and erythrocytes per unit volume of blood, which leads to the development of tissue oxygen starvation.

Anemia is diagnosed when the hemoglobin level in the blood is less than 130 g/l and the number of erythrocytes is less than 4×10^{12}/l in men and, respectively, less than 120 g/l and 3.5×10^{12}/l in women. Having common features, anemias at the same time represent a heterogeneous group of diseases, which are characterized by their own etiology, pathogenesis, features of the clinical picture, diagnostic criteria and methods of treatment. Within the scope of this topic, the main clinical forms of anemia are considered: iron deficiency, V_{12}- deficient, foliodeficient, hemolytic, hypoplastic, posthemorrhagic.

It is important for the formation of clinical thinking***pathogenetic classification of anemias***:

I. Anemia due to blood loss (posthemorrhagic).

1. Acute posthemorrhagic anemia.
2. Chronic post-hemorrhagic anemia.

II. Anemia due to impaired formation of erythrocytes and hemoglobin.

3. Iron deficiency anemia.
4. Iron redistribution anemia.
5. Iron-rich anemia.
6. Megaloblastic anemias associated with impaired DNA synthesis.

6.1. IN_{12}- and foliodeficiency anemias.

6.2. Megaloblastic anemia caused by a hereditary deficiency of enzymes involved in DNA synthesis.

6.3. IN_{12}- achrestic anemia

7. Hypoproliferative anemias.
8. Anemias associated with bone marrow failure.

8.1. Hypoplastic (aplastic) anemia.

8.2. Refractory anemia in myelodysplastic syndrome.

9. Metaplastic anemias.

9.1. Anemia with hemoblastosis.

9.2. Anemia with cancer metastases in the bone marrow.

10. Dyserythropoietic anemia.

III. Anemia due to increased blood loss.

11. Hereditary anemias.

11.1. Associated with a violation of the structure of the erythrocyte membrane (microspherocytic anemia of Minkovsky-Shaffar, ovalocytosis, acanthocytosis).

11.2. Associated with deficiency of enzymes in erythrocytes.

11.3. Associated with a violation of hemoglobin synthesis (sickle cell anemia, hemoglobinosis, thalassemia).

12. Acquired anemia.

12.1. Autoimmune anemia.

12.2. Paroxysmal nocturnal hemoglobinuria (Markiafava-Micheli disease).

12.3. Medical anemia.
12.4. Traumatic and microangiopathic anemia.
12.5. Anemia due to poisoning with hemolytic poison and bacterial toxins.

V. Anemias are mixed.

During the diagnostic search, it is advisable to determine the nature of anemia according to the morphology of erythrocytes, color index, as well as the ability of the bone marrow to compensate for anemia.

Morphological classification of anemias:

I. Macrocytic anemia (MCV*>100 μm^3 (fl), erythrocyte diameter > 8 μm).**

This type of anemia includes vitamin B deficiency anemia$_{12}$, folic acid deficiency and paroxysmal nocturnal hemoglobinuria.

II. Microcytic anemia (MCV<80 μm^3 (fl), erythrocyte diameter < 6.5 μm).

This type of anemia includes iron deficiency anemia, thalassemia, Minkovsky-Shafar microspherocytic anemia.

III. Normocytic anemia (MCV 81-99 μm^3 (fl), the diameter of erythrocytes is 7.2-7.5 microns).

This type of anemia includes hypoplastic anemia, most hemolytic anemias, and acute posthemorrhagic anemia.

Note:

fl – femtoliter (1 fl = 10^{-15}l = 1 μm^3)

MCV (mean corpuscular volume) is the average volume of an erythrocyte, calculated according to the formula:

Calculation example: hematocrit 0.3 (30%), erythrocytes 3.5×10^{12}/l

Variants of morphological changes of erythrocytes:

- Anisocytosis - a change in the size of erythrocytes.
- Poikilocytosis – a change in the shape of erythrocytes.
- Microcytosis is a condition in which microcytes predominate in the peripheral blood (erythrocytes < 6.5 μm in diameter).
- Microspherocytosis - the presence of round microcytes.
- Schizocytosis – the presence of very small particles of erythrocytes (2-3 μm).
- Planocytes – erythrocytes with reduced thickness.
- Annulocytes are erythrocytes that look like a ring (with significant lightening in the center).
- Ovalocytes – erythrocytes, oval in shape, without light in the center.
- Stomatocytes – erythrocytes with a linear lumen in the center in the form of a mouth.
- Xerocytes – erythrocytes with reduced cell volume.
- Acanthocytes are erythrocytes with spikes on the surface.

Classification of anemias by color index

I. Hypochromic anemia (color index <0.8)

This type of anemia includes iron-deficiency anemia, iron redistribution anemia, iron-saturated anemia, and thalassemia.

II. Normochromic anemia (color index 0.85-1.05).

This type of anemia includes hypoplastic anemia, most hemolytic anemias, acute posthemorrhagic anemia, and metaplastic anemias.

III. Hyperchromic anemia (color index >1.05)

This type of anemia includes B_{12}- deficiency and foliodeficiency anemia.

Note:

Color indicator (CP) is a value reflecting the hemoglobin content in an erythrocyte, calculated according to the formula:

, where 1 g%=10 g/l

Calculation example: hemoglobin 90 g/l, erythrocytes 3.5×10^{12}/l

Classification of anemias according to the bone marrow's ability to compensate.

I. Regenerative anemia (number of reticulocytes 0.5-5%)

This type of anemia includes iron deficiency anemia.

II. Hyperregenerative anemia (number of reticulocytes >5%)

This type of anemia includes acute posthemorrhagic anemia, hemolytic anemia, and iron-saturated anemia.

III. Hyporegenerative anemia (reticulocyte count <0.5%)

This type of anemia includes B12 deficiency anemia, foliodeficiency anemia, hypoplastic anemia

IRON DEFICIENCY ANEMIA

Iron deficiency anemia is an anemia characterized by a deficiency of iron in blood serum, bone marrow and depots (spleen, liver).

Latent iron deficiency is a condition characterized by a decrease in the amount of iron in the depot and a decrease in transport iron (ferritin) with still normal indicators of hemoglobin and erythrocytes.

20% of the world's population suffers from iron deficiency anemia and latent iron deficiency.

Etiology of iron deficiency anemia.

I. Chronic blood loss

uterine blood loss (dysfunctional uterine bleeding, uterine myoma, endometriosis, malignant uterine tumors, presence of intrauterine contraceptives, etc.)

blood loss from the digestive tract (stomach and duodenal ulcers, stomach cancer, cancer of the small or large intestine, diverticula, polyps, varicose veins of the esophagus, hemorrhoids, etc.)

blood loss in closed cavities (endometriosis, Goodpascher's syndrome)

hemoptysis (tuberculosis, lung cancer)

Hematuria (glomerulonephritis, urolithiasis, tuberculosis, bladder cancer, etc.)

nosebleeds (hypertension)

hemorrhagic diathesis (blood loss can be from any source)

hookworm infestation

II. Increased need for iron

· pregnancy, childbirth and lactation

· the period of puberty and growth

· intense sports activities

· on the background of vitamin B treatment$_{12}$

III. Insufficient initial level of iron

in newborns and young children whose mothers had anemia

IV. Insufficient supply of iron with food

· strict vegetarianism

· low standard of living

Anorexia

V. Impaired iron absorption

Chronic enteritis with malabsorption syndrome

Resection of the small intestine

Gastric resection according to the Billroth II method

VI. Violation of iron transport

Hereditary hypotransferinemia

Hypoproteinemia regardless of genesis (nephrotic syndrome, malabsorption syndrome, liver cirrhosis, chronic hepatitis, etc.)

Appearance of antibodies to transferrin and its receptors

Pathogenesis of iron deficiency anemia.

Under the influence of etiological factors, iron deficiency develops, which manifests itself in a decrease in iron reserves in the red bone marrow, spleen and liver. Over time, iron concentration in blood serum and hemoglobin synthesis decrease, trophic disorders of epithelial tissues occur. The consequence of these changes is the appearance of clinical manifestations of iron deficiency anemia.

Clinical picture of iron deficiency anemia.

The clinical picture consists of anemic (signs of hypoxia) and sideropenic (signs of epithelial tissue dystrophy) syndromes.

Anemic syndrome includes cardiovascular syndrome, astheno-vegetative syndrome, syndrome of damage to the gastrointestinal tract.

· Cardiovascular syndrome: complaints - palpitations, pain in the area of the heart, shortness of breath during physical exertion, objectively - pallor of the skin and visible mucous membranes, tachycardia, hypotension, muffled heart sounds, soft systolic murmur at all auscultatory points, possible expansion of the limits relative cardiac dullness to the left and nonspecific ECG changes. Sometimes there are pastosities of the lower legs, feet, and face.

Astheno-vegetative syndrome: complaints - general weakness, fatigue, reduced work capacity, drowsiness, dizziness.

Gastrointestinal tract damage: complaints - decreased appetite, belching, constipation or the presence of mushy stools, objectively - signs of atrophic gastritis with achlorhydria.

Sideropenic syndrome includes: trophic changes in the skin and its derivatives (skin - pale and dry, nails - brittle, sometimes with a spoon-shaped depression, hair - dry, without shine); muscle weakness; progressive caries; hoarseness of voice; rhinitis; glossitis - a

bright crimson tongue with atrophy of the papillae, painful; angular stomatitis ("crush"); spoiled taste (pica chlorotica) - desire to eat earth, clay, chalk, ice (pagophagia); deterioration of the sense of smell - addiction to gasoline, exhaust gases; the symptom of "blue sclera" - a blue tint of the eyes due to thinning of the sclera and translucency of the choroid; imperative urges to urinate; sideropenic dysphagia, sideropenic subfebrile; susceptibility to infectious and inflammatory processes.

Laboratory data in iron deficiency anemia.

Clinical blood analysis:

1. Decreased hemoglobin (<130 g/l in men, <120 g/l in women)

The severity of anemia is determined mainly by the level of hemoglobin:

Lung - hemoglobin content 90-120 g/l;

Medium - hemoglobin content 70-90 g/l;

Severe - hemoglobin content below 70 g/l.

2. Decrease in the number of erythrocytes (<4×10^{12}/l in men, <3.5×10^{12}/l in women)
3. Decrease in color index (<0.8)
4. Morphological changes of erythrocytes (predominance of microcytes, anisocytosis, poikilocytosis)
5. Normal content of reticulocytes, with significant bleeding can be increased
6. Tendency to leukopenia, with significant blood loss, thrombocytosis is possible
7. With significant anemia, a moderate increase in ESR is possible (up to 25 mm/h)

Biochemical analysis of blood:

1. Decreased serum iron concentration (<13 μmol/L in men, <11.5 μmol/L in women)
2. Decreased serum ferritin concentration (<12 μg/l)
3. The total iron-binding capacity of blood serum is increased (>70 μmol/l)
4. Ferritin iron saturation is reduced (<25%)

Instrumental diagnostic methods in iron-deficiency anemia, they are used mainly to identify the source of chronic blood loss (FGDS, colonoscopy, rectoromanoscopy, X-ray examination, ultrasound of the liver, spleen, pelvic organs, etc.).

In order to detect the presence of bleeding, a gynecological examination, urine examination, stool examination for occult blood (Gregersen's reaction), determination of occult blood loss with the help of labeled ^{51}Cr erythrocytes.

IN_{12}-DEFICIENCY ANEMIA.

IN_{12}- deficiency anemia is an anemia caused by a violation of DNA synthesis in erythrokaryocytes due to vitamin B deficiency$_{12}$ and is characterized by the megaloblastic type of hematopoiesis.

Addison-Birmer anemia (pernicious anemia) is considered a classic variant of B_{12}-deficiency anemia and associated with the production of antibodies to the parietal cells of the stomach.

The development of this disease is characteristic mainly for people aged 60-70 years. Prevalence among the population as a whole - 0.1%.

Etiology V_{12}- deficiency anemia.

I. Violation of secretion by the stomach of the "internal factor" - gastromucoprotein (antibodies to parietal cells and gastromucoprotein, gastrectomy, stomach cancer, etc.)

II. Violation of absorption of vitamin B_{12} in the intestine (malabsorption syndrome, intestinal resection, tumors and granulomatous diseases of the intestine, selective malabsorption of cobalamin - Imerslund's syndrome, etc.).
III. Competitive costs of vitamin B_{12}.(invasion by a helminth - tapeworm is wide, bacterial infection with the "blind loop" syndrome, etc.)
IV. Increased consumption of vitamin B_{12}(multiple pregnancy, hemolytic anemia, myeloma disease, etc.)
V. Violation of vitamin B intake$_{12}$ with food (strict vegetarianism).
VI. Depletion of vitamin B reserves$_{12}$ (cirrhosis).
VII. Violation of transport of vitamin B_{12} (absence of transcobalamin II or the appearance of antibodies to it).

Pathogenesis B_{12}- deficiency anemia.

Vitamin B_{12} fulfills its biological role in the form of two coenzymes - methylcobalamin and deoxyadenosylcobalamin.
Deficiency of methylcobalamin leads to disruption of DNA synthesis and maturation of rapidly growing cells: bone marrow cells, gastrointestinal tract epithelium. These changes are most pronounced on the part of the red hematopoietic germ. The number of megaloblasts increases in the bone marrow. Megaloblastic erythropoiesis is characterized by delayed maturation of erythrocyte nuclei compared to cytoplasmic hemoglobinization, increased decay of megaloblasts in the bone marrow, and reduced erythrocyte lifespan.
A deficiency of deoxyadenosylcobalamin leads to a violation of the metabolism of fatty acids and the accumulation of methylmalonic and propionic acids, which are toxic to the nervous system, which leads to damage to the posterior and lateral trunks of the spinal cord and to a decrease in the synthesis of myelin.

Clinical picture of V_{12}- deficiency anemia.

Damage to the hematopoietic system: complaints of general weakness, dizziness, ringing in the ears, darkening of the eyes, flickering of butterflies before the eyes, palpitations and shortness of breath, objectively - the skin is pale, often with a lemon-yellow tint, sometimes an increase in body temperature is possible, tachycardia, extrasystole, muffled tones heart, systolic murmur on the turntable, non-specific ECG changes.
Damage to the digestive system: complaints of loss of appetite, a feeling of heaviness in the epigastrium after eating, belching food and air, nausea, pain and burning in the tongue, objectively - the tongue is smooth "lacquered", red in color (Ganter's glossitis), possible signs of aphthous stomatitis, atrophic gastritis, atrophic changes in the intestinal mucosa with malabsorption phenomena, enlargement of the liver and spleen.
Damage to the nervous system (funicular myelosis): complaints of weakness in the legs, a feeling of "tingling" and numbness of the legs, objectively - impaired sensitivity, decreased tendon reflexes, atrophy of the muscles of the lower extremities, dysfunction of the pelvic organs (incontinence of urine and feces).

Laboratory data at V_{12}- deficiency anemia.

Clinical blood analysis:

1. Color index >1.1.
2. An increase in the diameter of erythrocytes (macrocytosis).
3. Anisocytosis.
4. Preservation of the remnants of the nucleus of erythrocytes (Jolly bodies, Cabot rings).

5. Reticulocytopenia.
6. Leukopenia, hypersegmentation of neutrophils.
7. Thrombocytopenia.
Myelogram (key examination for diagnosis)
1. Hyperplasia of the red hematopoietic germ.
2. The appearance of megaloblasts in the bone marrow.
3. Hypersegmentation of neutrophils.
Biochemical analysis of blood:
1. Unconjugated hyperbilirubinemia is possible.
2. It is possible to increase the enzymes LDH1 and LDH2 (lactate dehydrogenase) in the blood.
Immunological analysis of blood: detection of antibodies to parietal cells of the stomach, to gastromucoprotein or complex "vitamin B_{12}+gastromucoprotein".
Analysis of urine and feces: with the development of hemolysis - urobilin appears in the urine, stercobilin increases in the feces.
Schilling's test: positive.
During the test, the patient takes vitamin B inside$_{12}$, which is labeled^{60}Co. After an hour, vitamin B is administered$_{12}$ intravenously to saturate the liver depot. A positive test - a decrease in the excretion of radioactive vitamin with urine - indicates a violation of the absorption of vitamin B_{12} in the intestine
Instrumental research with V_{12}- deficiency anemia.
Esophagogastroduodenoscopy: diffuse atrophic gastritis, duodenitis, less often - atrophic esophagitis.
Study of gastric secretion: a sharp decrease in the amount of gastric juice, a decrease in hydrochloric acid and pepsin.
Ultrasound of the liver and spleen: a slight increase in the size of the spleen, sometimes the liver.
Roentgenoscopy of the stomach: violation of the evacuation function, flattening and smoothing of the folds of the mucous membrane
Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:
1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with a patient with anemia in order to collect complaints and history, physical examination of the patient; evaluation of clinical examination data and laboratory and instrumental data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.
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- to conduct a subjective examination of patients with anemia
- conduct an objective examination of patients with anemia
- appoint a plan for additional laboratory and instrumental examinations of patients with anemia, leukemia, hemorrhagic diatheses

2.2. Questions to check basic knowledge on the topic of the lesson:
1. Classification of anemias.
2. Etiology of iron deficiency anemia.
3. Clinical signs of sideropenic syndrome.
4. Blood parameters in iron deficiency anemia.
5. Etiology and pathogenesis of V_{12}- deficiency anemia.
6. Clinical manifestations of V_{12}- deficiency anemia.
7. Blood parameters at V_{12}- deficiency anemia.

3. Formation of professional skills and abilities:
mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the interview)
formation of the ability to perform a clinical examination of a patient with anemia, leukemia, hemorrhagic diathesis (the applicant must be able to perform a physical examination of the patient),

formation of the ability to evaluate the data obtained during the clinical examination of a patient with anemia, leukemia, hemorrhagic diathesis;
carry out a clinical interpretation of the main symptoms and syndromes in these diseases

formation of the ability to conduct a modern laboratory-instrumental examination of a patient with anemia, leukemia, hemorrhagic diathesis (the applicant must be able to prescribe a plan of laboratory-instrumental examination, give an assessment of the examination results)

3.1. Control materials for the final stage of the lesson:

1. A 21-year-old patient came to the clinic because of painful and prolonged menstruation, which takes the form of bleeding. General weakness, dizziness, tinnitus, shortness of breath, hair loss and brittle nails are a concern. Blood analysis: HB - 56 g/l; er-2.8 t/l; KP- 0.6; leuk.-2.5 g/l; ESR - 14 mm/hour, platelets 139 g/l; reticulocytes - 4.5%; anisocytosis with a tendency to microcytosis, hypochromia. Which of the diagnoses is probable?

+A iron deficiency anemia;

B12 deficiency anemia;

Autoimmune anemia;

D aplastic anemia;

E Werlhof's disease.

2. A 69-year-old patient complains of weakness, dizziness, burning of the tongue, decreased appetite. 5 years ago - gastric resection. Objectively: the skin is pale, the language is crimson, smooth; systolic murmur at the apex of the heart. In the blood analysis: er. - 2.2 t/l; HB - 76 g/l; KP-1,1; leukopenia; ESR 30 mm/hour. Jolly's bulls, Cabot's rings. Your diagnosis?

And stomach cancer

In cirrhosis of the liver

With hemolytic anemia

+D B12 deficiency anemia

E iron deficiency anemia.

Practical lesson No. 36
Topic: Symptoms and syndromes in leukemias and hemoblastoses.

Goal: Acquiring knowledge and mastering professional competences during the examination of a patient with leukemia - questioning, physical examination, laboratory and instrumental research.

Basic concepts: Diagnostic criteria of leukemia

Diagnostic criteria of acute leukemia (HL).

The clinical picture of GL can be different, which depends on the suppression of normal hematopoiesis.

In the course of the disease, three stages of the course are distinguished:

1) Initial - can be diagnosed retrospectively.

2) The stage of the developed clinical picture.

3) Terminal stage.

There are several variants of the initial stage:

The acute onset of the disease is observed in half of the patients and is characterized by high t of the body (sometimes with symptoms), intoxication, inflammatory weakness, joint pain, abdominal pain, pain when swallowing. The onset of the disease can be interpreted as flu, sore throat, rheumatism, acute appendicitis, acute appendicitis. Sometimes patients are mistakenly admitted to the infectious disease department.

The onset of the disease with pronounced clinical manifestations is observed in 10% and is characterized by profuse bleeding (nasal bleeding), etc.

- Slow onset - manifested by general weakness, fatigue, malaise, minor hemorrhages in the form of bruises and slight enlargement of L/nodes.

- Latent period of flow – observed in 5% of patients, the disease is discovered by chance (when examining peripheral blood during preventive examinations).

Phases of the course of the disease are characteristic of HL

1. Aleukemic (without the release of blast cells into the blood).

2. Leukemic (with the release of blast cells into the blood)

The stage of the developed clinical picture includes the main syndromes.

1) Hyperplastic.
2) Hemorrhagic.
3) Tumor intoxication syndrome.
4) Anemic syndrome.
5) Immunodeficient.
6) Neuroleukemia
7) Ulcerative-necrotic changes

- hyperplastic syndrome: characterized by an increase in lymph nodes, pain in the bones, heaviness and pain in the left and right hypochondrium, hepatosplenomegaly, development of ulcer-necrotic syndrome. Other symptoms may appear; such as headache, shortness of breath, cough, sciatica, etc

- anemic syndrome: characterized by dizziness, flickering of flies before the eyes, shortness of breath during physical exertion, palpitations, noise in the head and ears - symptoms that are characteristic of anemia)

- hemorrhagic syndrome:

Characteristic: skin hemorrhages, bleeding gums, nosebleeds (for example, such symptoms as in thrombocytopenia, hemorrhagic vasculitis). In the anamnesis, there may be infections - flu, contact with chemical preparations, poisonous chemicals.

It is characterized by an increase in body temperature, weakness, changeability, lack of appetite, ossalgia. Such symptoms make it possible to suspect infectious diseases (tuberculosis, sepsis, etc.).

-Immunodeficiency syndrome is characterized by the addition of severe pneumonia, which very often leads to the death of patients. That is, there is a violation of cellular and humoral immunity.

Acute leukemia is characterized by the development of manifestations of other organs and systems.

1) Neuroleukemia: characterized by the development of leukemic infiltration in the membranes and substance of the brain and spinal cord. There are several forms of neuroleukemia:

In addition, leukemic infiltration of the brain, prostate, bladder, lesions of the gastrointestinal tract, esophagus, stomach, liver, kidneys, and heart may occur.

The diagnostic criteria for HL are the study of peripheral blood and bone marrow punctate.

The main criterion for acute leukemia is the presence of more than 30% blast cells in the bone marrow (blastemia).

Their number can be up to 80-90%). In the aleukemic phase, GL blasts may be absent in the blood. In such cases, the diagnosis is made based on the results of the bone marrow examination.

Cytochemical diagnosis of acute leukemias

The form of acute leukemia is determined using a cytochemical study.

Among the instrumental methods of research, the following are used: tomography or X-ray of the chest, ECG, ultrasound, FGDS, in which changes characteristic of leukemic infiltration of the relevant organs and tissues are found.

Diagnostic criteria for chronic lymphocytic leukemia (CLL)

The diagnosis is usually made on the basis of the results of a general clinical examination of the patient and morphological analysis of peripheral blood smears. In the course of CLL, initial, advanced and terminal stages of the disease are distinguished (according to the old classification).

The initial stage of CLL is moderate lymphadenopathy, the clinic develops slowly and imperceptibly. Complaints are mainly asthenic in nature.

The disease at this stage is detected by chance during a routine blood test, when an increase in the relative (40-50%) and absolute number of lymphocytes is detected with a slight leukocytosis.

It should be emphasized that there are no "causeless" lymphocytes, and leukemic reactions of the lymphoid type (infectious mildly symptomatic lymphocytosis, lymphocytosis with whooping cough, rubella) are found only in children, infectious mononucleosis - a disease also mainly of young age - is diagnosed by the blood count. In which lymphocytes are found, which differ from the usual large size, a wide rim of cytoplasm around the nucleus and its brighter color.

An increase in the absolute and relative number of lymphocytes should always alert the doctor.

The majority of peripheral blood cells are represented by mature lymphocytes. Individual Botkin-Gumprecht shadows (semi-destroyed nucleus of lymphocytes with remnants of nucleoli) can also be detected. They form when a smear of blood is applied to a glass slide, when easily injured tumor lymphocytes are crushed, losing cytoplasm.

Expanded (pronounced clinical and hematological manifestations).

In this stage, asthenic complaints intensify. Characteristic lymphoproliferative syndrome: there is a generalized lesion of lymph nodes in the following sequence: first cervical, then axillary, then conglomerates of nodes appear in the mediastinum, in the abdominal cavity and inguinal areas. Lymph nodes have a pasty-elastic consistency. More often, they are painless, not welded together and with the skin, without ulcers and do not suppurate.

Characteristic hepatosplenomegalic syndrome. Changes in the lungs are manifested by frequent banal pneumonias and specific leukemic infiltration. The cardiovascular system, gastrointestinal tract, and genitourinary system are affected.

In CLL, infiltration of the UIII pair of cranial nerves is often observed, with hearing loss, congestion, and tinnitus.

The terminal stage is characterized by exhaustion, a significant deterioration of the general condition, the development of complications, an increase in anemia (not only due to the suppression of erythropoiesis, but also due to the occurrence of cases of autoimmune hemolytic anemia), the appearance of hemorrhagic syndrome, a significant increase in lymph nodes and spleen, refractoriness to the therapy. The transition of CLL to the terminal stage is often accompanied by sarcomatous growth in the lymph node.

Such lymph nodes begin to grow rapidly, acquire a stony density, infiltrate and squeeze neighboring tissues, causing swelling and pain syndrome. Often, sarcoid growth in the

lymph nodes is accompanied by an increase in temperature. Sometimes such nodes are located in the subcutaneous tissue of the face, trunk, limbs, under the mucous membrane in the oral cavity, nose, and the vessels that develop in them give them the appearance of hemorrhages. One of the manifestations of the terminal stage of the disease can be severe renal failure as a result of infiltration of the parenchyma of the organ by tumor cells. Herpetic infection is a serious, often fatal complication.

In the clinical picture, 2 syndromes are distinguished:

1. lymphoproliferative, caused by lymphadenopathy, splenomegaly and lymphoid proliferation of the bone marrow:

a) general symptoms caused by intoxication, the growth of leukemic cells in the bone marrow, spleen (itching, fever, sweating, pain in the bones, spleen and liver);

b) hepato- and splenomegaly;

c) leukemic infiltrates in the skin (leukemias);

d) symptoms are associated with an increase in regional lymph nodes (mediastinal, mesenteric);

e) characteristic changes in bone marrow and peripheral blood.

2. Syndrome of complications:

a) purulent - inflammatory;

b) autoimmune (autoimmune hemolytic anemia, autoimmune thrombocytopenia).

direct causes of death of patients with CLL are most often intercurrent infection, severe anemia, hemorrhages in vital organs and intoxication.

Diagnosis of CLL:

Leukocytosis with absolute lymphocytosis in the blood.

More than 30% of lymphocytes in the bone marrow punctate with diffuse lymphoid hyperplasia in the bone marrow trepanation.

Enlargement of the lymph nodes and spleen is an optional symptom of CLL, but when these organs are involved in the process, a diffuse proliferation of lymphocytes is observed.

An additional diagnostic feature is Huprecht's shadow in a blood smear.

The benign form of CLL causes a very slow, noticeable only over years, but not months, increase of lymphocytes in the blood in parallel with the growth of leukocytes.

A very slow increase in lymphocytosis to a noticeable increase in lymph nodes can last for years and decades. All this time, the patients are on the "D" record, and may need cytostatic therapy.

Progressive (classical) form.

CLL starts as benign, but the number of white blood cells increases from month to month along with the size of the lymph nodes.

Tumor form of CLL.

A feature of this form is a significant increase and dense consistency of lymph nodes with low leukocytosis.

The splenomegalic form of CLL is characterized by predominant enlargement of the spleen with widespread enlargement of lymph nodes and varying levels of leukocytosis. This form is prognostically more favorable.

Bone marrow form of CLL. rapidly progressive pancytopenia, total or partial replacement of the bone marrow by mature lymphocytes. The lymph nodes are not enlarged, the spleen, with rare exceptions, is also not enlarged, the liver is also of normal size. Prognostically favorable form.

Diagnostic criteria for chronic myelogenous leukemia (CML)

Chronic myeloid leukemia is a classic hematological disease that goes through three stages of development: the chronic stage, the acceleration stage, and the stage of transformation into acute leukemia.

Diagnosis of the chronic phase

CML develops mostly latently. The clinical picture is manifested by various symptoms. Typical complications occasionally unmask CML.

1. Circumstances of discovery

- In most cases, the disease is detected by chance or thanks to a systematic general blood test.

- General symptoms: asthenia; emaciation; a feeling of discomfort in the left hypochondrium; deterioration of the general condition of varying degrees of severity, but sufficient for the appointment of a general blood test.

- Clinical examination. Palpable splenomegaly is the main classic symptom. Now this symptom occurs less and less due to early diagnosis of the disease.

- Complications: priapism, gout attack, splenic infarction, hemorrhagic symptoms.

Granulocytic Neutrophilic Hyperleukocytosis with myelemia should alert the doctor. Suspicion of CML is confirmed by eosinophilia, basophilia, and hyperthrombocythemia.

The final diagnosis is established after the discovery of the Philadelphia chromosome or its molecular equivalent - a gene.

2. Additional examination

- An ordinary general blood test is often enough to establish a diagnosis. He reveals significant hyperleukocytosis, often more than 50 x 109/l. Hyperleukocytosis is associated with an increase in the absolute number of neutrophilic granulocytes. The increase in the absolute number of basophils and especially eosinophils is less proportional. Myeloma consists mainly of mature precursor cells (myelocytes and metamyelocytes). Young cells (pro-myelocytes and myeloblasts) occasionally occur. The absolute number of other blood leukocytes remains unchanged, although in percentage terms their number decreases significantly. Other abnormalities of the general blood test include slight normocytic anemia. The increase in the number of platelets over 500 x 109/l is much more pronounced. Thrombocytopenia is rare at diagnosis.

- Biochemical blood analysis reveals hyperuricemia proportional to hyperleukocytosis and a high level of lactate dehydrogenase. In granulocytes with CML, the level of neutrophil alkaline phosphatase is significantly reduced, the enzymatic activity of which is determined on blood smears based on 100 neutrophils. Detection of a reduced level of alkaline leukocyte phosphatases is not sufficient for a definitive diagnosis of CML. The method has many false negative errors and is not specific. In classic cases of CML, elevated levels of vitamin B12 and histamine are found.

- Coagulation and homeostasis. Prolongation of bleeding time reflects acquired thrombocytopathy, which also occurs in cases of other myeloproliferative syndromes. The prolongation of Kwik's time can be associated with a deficiency of the U-th factor.

- A myelogram must be done, although it helps little in confirming the diagnosis. The myelogram reveals only an increase in the cellularity of the bone marrow with a pronounced predominance of elements of the neutrophilic series. It helps during differential diagnosis and is especially important for studying the karyotype.

- Bone marrow biopsy confirms the diagnosis of myeloproliferative syndrome with narrowing of fat depots. At the time of diagnosis, she does not show significant fibrosis (no signs of collagen fibrosis, but there is reticular fibrosis).

The presence of myeloid cells in the peripheral blood. Cells of the first four rows of the modern scheme of hematopoiesis. A collection of eosinophils, basophils and neutrophils.

Study of factors of the external pathway of blood coagulation in the presence of excess tissue thromboplastin. Formula of cellular elements of bone marrow.

• Chronic myelomonocytic leukemia. Differential diagnosis is sometimes extremely difficult. Only a karyotype or a molecular biological study can establish a true diagnosis. In clinical practice, it is necessary to distinguish chronic myelomonocytic syndrome, which belongs to the group of myelodysplasias, and chronic myelomonocytic leukemia, which belongs to myeloproliferative syndromes. Clinical symptoms of leukemic progression are clearly expressed (exudative serositis, specific skin lesions}, in addition, bone marrow failure (anemia, thrombocytopenia) is very often diagnosed. A general blood test reveals anemia, monocytosis, mild myelemia, and thrombocytopenia. Myelogram and bone marrow biopsy confirm diagnosis Blood and urine lysozyme is significantly elevated.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:

1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with a leukemia patient in order to collect complaints and history, physical examination of the patient; evaluation of clinical examination data and laboratory and instrumental data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for the theoretical readiness of applicants to perform practical classes: the applicant must know the modern definition, etiology, pathogenesis, classifications of anemias, leukemias, hemorrhagic diatheses, subjective and objective data in these diseases, know laboratory and instrumental data in this pathology.

List of didactic units:

- to conduct a subjective examination of leukemia patients
- conduct an objective examination of leukemia patients,
- appoint a plan for additional laboratory and instrumental examinations of patients with hemorrhagic diatheses
- evaluate the results of laboratory tests in patients with
- leukemia
- master the skills and abilities to assess the leading clinical syndromes in patients with leukemia

2.2. Questions to check basic knowledge on the topic of the lesson:

1. Classification of leukemias.

2. Etiology and pathogenesis of leukemias.

3. Main clinical syndromes in leukemia.

4. Blood parameters in acute and chronic leukemias.

3. Formation of professional skills and abilities:

mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the interview)

formation of the ability to perform a clinical examination of a patient with anemia, leukemia, hemorrhagic diathesis (the applicant must be able to perform a physical examination of the patient),

formation of the ability to evaluate the data obtained during the clinical examination of a patient with anemia, leukemia, hemorrhagic diathesis;
carry out a clinical interpretation of the main symptoms and syndromes in these diseases

formation of the ability to conduct a modern laboratory-instrumental examination of a patient with anemia, leukemia, hemorrhagic diathesis (the applicant must be able to prescribe a plan of laboratory-instrumental examination, give an assessment of the examination results)

3.1. Control materials for the final stage of the lesson:
Situational tasks:

Tasks of the STEP-2 type

3. The patient turned to the doctor with complaints of general weakness, pain in the tubular bones, high temperature. The examination revealed a systemic increase in lymph nodes, hepato-lienal syndrome. In the blood test: er.-2.6 t/l; HB-67 g/l; platelets - 45 g/l; leukocytes 56 g/l, blasts 87%; p/y-1%; s/y-7%; lymphocytes-5%; ESR 55 mm/hour; Your diagnosis?

+ A Acute leukemia

In Chronic myeloid leukemia

From Eritrea

D Myeloma disease

4. In a 60-year-old patient, during a preventive examination, it was found in the blood test that the number of leukocytes was 60 g/l, and in the formula, the number of lymphocytes was 89%. Enlargement of cervical and inguinal lymph nodes. Slight weakness. Your diagnosis?

And tuberculosis of the lymph nodes;

+ In chronic lymphocytic leukemia;

With infectious mononucleosis;

D infectious lymphocytosis;

Is lymphogranulomatosis.

5. A 68-year-old man complains of fatigue, sweating, and enlarged cervical, submaxillary, and axillary lymph nodes. In the blood: leukocytes - 35 g/l, of which 60% are lymphocytes; Botkin-Gumprecht corpuscles; the level of hemoglobin and the number of platelets are normal. In the myelogram, lymphocytes are -40%. What is the most likely diagnosis?

And acute leukemia;

In lymphogranulomatosis;

+ With chronic lymphocytic leukemia;

D tuberculous lymphadenitis;

It is chronic myelogenous leukemia.

6. A 48-year-old man complains of weakness, sweating, and heaviness in the upper abdomen. During the examination, hepatosplenomegaly was found in him. In the blood analysis: neutrophilic leukocytosis, eosinophil-basophil association, single blast cells. Liver biopsy showed myeloid infiltration. Your diagnosis?

+A chronic myelogenous leukemia

In chronic lymphocytic leukemia;

With acute leukemia;

D liver cirrhosis;

E leukemoid reaction.

7. A 65-year-old man has been ill for several years, notes an increase in cervical and axillary lymph nodes, sweating, weakness. The condition worsened 2 weeks ago: the liver, spleen, and lymph nodes increased. In the blood test: er.-2.8 t/l; HB - 92 g/l; L-68 g/l; lymph - 86%; ESR - 48 mm/hour. Botkin-Gumprecht cells in peripheral blood smear. Which of the diagnoses is correct?

And chronic myeloid leukemia

In subleukemic myelosis

With lymphogranulomatosis

D tuberculosis

+E chronic lymphocytic leukemia

8. The patient has a systemic increase in lymph nodes, hepato-lienal syndrome, pronounced jaundice. In the blood test: r.-2.4 t/l; HB - 58 g/l; reticulocytes - 10%; platelets-145g/l; leukocytes - 56 g/l; lymphocytes-87%; p/y-1%; s/y-7%; m-5%; ESR-55mm/hour. What is your underlying medical diagnosis? What complication of the main disease can be suspected based on clinical and laboratory indicators?

And aplastic anemia

+ In chronic lymphocytic leukemia. Symptomatic hemolysis

With toxic neutropenia

D agranulocytosis

E B12 deficiency anemia.

9. A 23-year-old man became acutely ill. During the week, the temperature is 39-40 C, weakness, sweating, dizziness, nose and gum bleeding, hemorrhage on the skin of the body. In the blood test: HB-72 g/l; r.-2.3 t/l; leukocytes 7.6 g/l; with 5%; l-9%; blast cells 86%; ESR - 23mm/hour. Which of the following is the most reliable?

+ And acute leukemia

I have sepsis

From tuberculosis

D hemorrhagic vasculitis

E systemic lupus erythematosus

10 A 27-year-old patient has been complaining of fatigue, sweating, and heaviness in the left hypochondrium for about a year, especially after eating. Objectively: enlargement of the spleen, liver. In the blood: er. – 3.21012/l, Hb – 100 g/l, CP – 0.87, leuk. – 100109 /l, b. - 7%, e. - 5%, m. - 15%, y. - 16%, p. - 10%, p. – 45%, lymph. - 2%, mon. - 0%, reticle. - 0.3%, thrombus. – 400109/l, ESR – 25 mm/h. What is the most likely diagnosis?

A.+ Chronic myelogenous leukemia.

D. Erythremia.

B. Chronic lymphocytic leukemia.

E. Liver cirrhosis.

C. Acute leukemia.

11. A 46-year-old patient complains of itching of the skin, sweating, especially at night, temperature rise to 38.6°C. Objectively: on the skin of the chest there are traces of scratching, supraclavicular lymph nodes the size of a pigeon's egg, not fused to the skin. Which research is the most appropriate?

+A. Puncture of an enlarged lymph node.

B. General blood analysis.

C. Overview X-ray of the chest.

D. Imunogram.

E. Total protein and protein fractions

11. A 63-year-old patient complained of lower back pain. A course of physiotherapeutic treatment was carried out for sciatica. However, the patient's condition did not improve. The patient underwent X-ray of the bones of the spine and pelvis, which revealed osteoporosis and significant bone defects. Moderate normochromic anemia in the blood, proteinuria in the urine. Total blood protein – 10.7 g/l. What disease should be thought of first of all?

+A. Myeloma disease.

D. Bone metastases.

B. Urinary stone disease.

E. Systemic osteoporosis.

C. Acute sciatica.

3.2. Requirements for work results, including before registration: substantiation of the diagnosis based on complaints, medical history and life data, clinical and laboratory-instrumental examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (*if necessary*).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 37
Topic: Symptoms and syndromes in hemorrhagic diatheses.

Goal: Acquiring knowledge and mastering professional competences during examination of a patient with hemorrhagic diathesis - questioning, physical examination, laboratory and instrumental studies.

Basic concepts:

The main components of the blood coagulation system. Development factors of bleeding and causes of hemorrhagic syndromes - thrombocytopenia, coagulopathy, hemorrhagic vasculitis. Characteristics of the hemorrhagic syndrome in hemophilia, thrombocytopenic purpura and Schönlein-Henoch disease. Manifestations of articular, abdominal, renal and anemic syndromes in these diseases. Basic methods of laboratory diagnosis of hemorrhagic syndromes.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:

1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with a patient with hemorrhagic diatheses in order to collect complaints and history, physical examination of the patient; evaluation of clinical examination data and laboratory and instrumental data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.

2.1. Requirements for the theoretical readiness of applicants to perform practical classes: the applicant must know the modern definition, etiology, pathogenesis, classifications of hemorrhagic diatheses, subjective and objective data in these diseases, know laboratory and instrumental data in this pathology.

List of didactic units:

- to conduct a subjective examination of patients with hemorrhagic diatheses
- conduct an objective examination of patients with hemorrhagic diatheses
- appoint a plan for additional laboratory and instrumental examinations of patients with hemorrhagic diathesis
- evaluate the results of laboratory tests in patients with anemia, leukemia, hemorrhagic diathesis
- master the skills and abilities of assessment of leading clinical syndromes in patients with hemorrhagic diathesis

2.2. Questions to check basic knowledge on the topic of the lesson:

1. Classification of hemorrhagic diatheses.
2. Etiology and pathogenesis of hemorrhagic diatheses.
3. Clinical signs of hemorrhagic diatheses.

4. Blood parameters in hemorrhagic diatheses.

3. Formation of professional skills and abilities:

mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the interview)

formation of the ability of clinical examination of patients with hemorrhagic diathesis (the applicant must be able to perform a physical examination of the patient),

formation of the ability to evaluate the data obtained during the clinical examination of a patient with hemorrhagic diathesis;
carry out a clinical interpretation of the main symptoms and syndromes in these diseases

formation of the ability to conduct a modern laboratory-instrumental examination of a patient with anemia, leukemia, hemorrhagic diathesis (the applicant must be able to prescribe a plan of laboratory-instrumental examination, give an assessment of the examination results)

3.1. Control materials for the final stage of the lesson:
Situational tasks:

Tasks of the STEP-2 type

6. A 16-year-old boy was admitted to the hematology department with complaints of pain in the right shoulder joint, which occurred after the joint was bruised. It is known from the anamnesis that such a clinical picture has been observed repeatedly since early childhood. Objectively: the joint is enlarged, sharply painful on palpation. In the blood: er. – 3.71012/l, Hb – 110 g/l, blood clot. – 115109 /l, lake. – 6.9109/l, ESR – 25 mm/h. Prothrombin index – 90%, recalcification time – 280 min, blood coagulation time – 38 min, fibrinogen – 3.5 g/l. What is the most likely diagnosis?

+A. Hemophilia.

D. Immune thrombocytopenia.

B. Immune coagulopathy.

E. Hemorrhagic vasculitis.

C. Thrombocytopathy.

7. A 25-year-old patient is seen by a hematologist for hemophilia A. Acute hemarthrosis of the knee joint occurred after falling from a horizontal bar. Objectively: the right knee joint is sharply enlarged, the skin over it is hyperemic. Bleeding time according to Duke – 3 minutes, blood clotting time according to Lee-White – 20 minutes. Which drug is the most effective for the treatment of this patient?

+A. Recombinant factor VIII.

B. Recombinant X factor.

C. Thromboconcentrate.

D. Ambien.

E. Aminocaproic acid.

8. The patient received radiation exposure. He complains of weakness, frequent nosebleeds, "bruises" on the body, palpitations, shortness of breath. He often suffers from respiratory diseases. Blood analysis: er. – 1.21012/l, Hb – 54 g/l, leuk. - 1.7109 /l, e. - 0%, p. - 0%, p. - 32%, l. - 62%, m. - 6%; ESR – 52 mm/h; clot. – 30109 /l. What treatment is most appropriate in this case?

+A. Bone marrow transplantation.

D. Transfusion of whole blood.

B. Platelet mass transfusion.

E. Antilymphocytic

C. Transfusion of erythrocyte mass. immunoglobulin.

9. The 16-year-old patient has been sick since the age of 5, when massive and prolonged bleeding from the postoperative wound was observed for the first time after appendectomy. Later, bleeding after minor injuries was noted. 4 years ago - hemarthrosis of the knee joint. 3 years ago, he was treated for an extra-abdominal hematoma. Deficiency of blood coagulation factor III was detected. The most reliable diagnosis?

+A hemophilia A

In hemophilia B

With thrombocytopenic purpura

D hemorrhagic vasculitis

E congenital thrombocytopenia

10. A 32-year-old woman complains of unexplained bruising, weakness, bleeding gums, and menorrhagia. Objectively: mucous membranes and skin are pale with hemorrhages of various ages. Lymph nodes are not enlarged. Pulse-100 beats. in one minute; BP – 110/70 mm. mercury Art. There are no changes from the internal organs. Blood analysis: er. - 3.0 t/l; HB - 92 g/l; KP- 0.9; anisocytosis, poikilocytosis; leuk. - 10 g/l; e.-2%; p. - 12%; p. - 68%; l.-10%; m.-7%; ESR - 12 mm/hour.

Additional determination of which indicator is appropriate for establishing a diagnosis?

And the blood clotting time

In fibrinogen

From reticulocytes

D osmotic resistance of erythrocytes

+E platelets

3.2. Requirements for work results, including before registration: substantiation of the diagnosis based on complaints, medical history and life data, clinical and laboratory-instrumental examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (*if necessary*).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 37
Topic: Symptoms and syndromes in hemorrhagic diatheses.
Goal: Acquiring knowledge and mastering professional competences during examination of a patient with hemorrhagic diathesis - questioning, physical examination, laboratory-instrumental studies.

Basic concepts:

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:
1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking of workbooks, communication with a patient with anemia, leukemia, hemorrhagic diatheses for the purpose of collecting complaints and anamnesis, physical examination of the patient; evaluation of clinical examination data and laboratory and instrumental data*),*conducting a test control, solving a clinical problem, written solution of problems of the Step-2 type (10 problems), frontal survey, discussion, role-playing on the subject of the lesson.
2.1. Requirements for the theoretical readiness of applicants to perform practical classes: the applicant must know the modern definition, etiology, pathogenesis, classifications of anemias, leukemias, hemorrhagic diatheses, subjective and objective data in these diseases, know laboratory and instrumental data in this pathology.

List of didactic units:
- to conduct a subjective examination of patients with hemorrhagic diatheses
- conduct an objective examination of patients with hemorrhagic diatheses
- appoint a plan for additional laboratory and instrumental examinations of patients with anemia, leukemia, hemorrhagic diatheses

- evaluate the results of laboratory tests in patients with anemia, leukemia, hemorrhagic diathesis
- master the skills and abilities to assess leading clinical syndromes in patients with anemia, leukemia, hemorrhagic diatheses

2.2. Questions to check basic knowledge on the topic of the lesson:
1. Classification of anemias.
2. Etiology of iron deficiency anemia.
3. Clinical signs of sideropenic syndrome.
4. Blood parameters in iron deficiency anemia.
5. Etiology and pathogenesis of V_{12}- deficiency anemia.
6. Clinical manifestations of V_{12}- deficiency anemia.
7. Blood parameters at V_{12}- deficiency anemia.

8. Classification of leukemias.
9. Etiology and pathogenesis of leukemias.
10. Main clinical syndromes in leukemia.
11. Blood indicators in acute and chronic leukemias.
12. Classification of hemorrhagic diatheses.
13. Etiology and pathogenesis of hemorrhagic diatheses.
14. Clinical signs of hemorrhagic diatheses.
15. Blood indicators in hemorrhagic diatheses.

3. Formation of professional skills and abilities:
mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the interview)
formation of the ability to perform a clinical examination of a patient with anemia, leukemia, hemorrhagic diathesis (the applicant must be able to perform a physical examination of the patient),

formation of the ability to evaluate the data obtained during the clinical examination of a patient with anemia, leukemia, hemorrhagic diathesis;
carry out a clinical interpretation of the main symptoms and syndromes in these diseases

formation of the ability to conduct a modern laboratory-instrumental examination of a patient with anemia, leukemia, hemorrhagic diathesis (the applicant must be able to prescribe a plan of laboratory-instrumental examination, give an assessment of the examination results)

3.1. Control materials for the final stage of the lesson:
Situational tasks:
Tasks of the STEP-2 type
1. A 37-year-old patient has had frequent nosebleeds, pronounced metrorrhagia, and periodic formation of bruises on the skin for the past 6 years. 10 days ago, after a significant nosebleed, weakness increased, dizziness and palpitations appeared. Objectively: the skin is pale, there are widespread petechial hemorrhages and single ecchymoses on the front surface

of the trunk, legs, and arms. In the blood: Hb - 80 g/l, er. – 4.01012/l, CP – 0.7; leyk – 5.3109/l; p. - 2%, p. - 65%, e. - 2%, l. - 24%, m. - 5%, thrombus. – 10109/l, ESR – 15 mm/h. What is the most likely diagnosis?
+A. Werlhof's disease.
D. Iron deficiency anemia.
B. Hemophilia.
E. Aplastic anemia.
C. Hemorrhagic vasculitis

2The 16-year-old patient has been sick since the age of 5, when massive and prolonged bleeding from the postoperative wound was observed for the first time after appendectomy. Later, bleeding after minor injuries was noted. 4 years ago - hemarthrosis of the knee joint. 3 years ago, he was treated for an extra-abdominal hematoma. Deficiency of blood coagulation factor III was detected. The most reliable diagnosis?
+A hemophilia A
In hemophilia B
With thrombocytopenic purpura
D hemorrhagic vasculitis
E congenital thrombocytopenia

3. A 32-year-old woman complains of unexplained bruising, weakness, bleeding gums, and menorrhagia. Objectively: mucous membranes and skin are pale with hemorrhages of various ages. Lymph nodes are not enlarged. Pulse-100 beats. in one minute; BP – 110/70 mm. mercury Art. There are no changes from the internal organs. Blood analysis: er. - 3.0 t/l; HB - 92 g/l; KP- 0.9; anisocytosis, poikilocytosis; leuk. - 10 g/l; e.-2%; p. - 12%; p. - 68%; l.-10%; m.-7%; ESR - 12 mm/hour.
Additional determination of which indicator is appropriate for establishing a diagnosis?
And the blood clotting time
In fibrinogen
From reticulocytes
D osmotic resistance of erythrocytes
+E platelets

3.2. Requirements for work results, including before registration: substantiation of the diagnosis based on complaints, medical history and life data, clinical and laboratory-instrumental examination.

3.3. Control materials for the final stage of the lesson: solving two clinical problems on the topic of the lesson, answering 10 tests (*if necessary*).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 38
Topic: Symptomatology of hypothyroidism, thyrotoxicosis. Syndromes. Data analysis of instrumental research methods.

Goal: Acquiring knowledge and mastering professional competences during the examination of a patient with hypothyroidism, thyrotoxicosis, diabetes mellitus - questioning, physical examination, laboratory-instrumental research.

Basic concepts:

Hypothyroidism – the syndrome, the development of which is caused by thyroid hypofunction, develops as a result of a decrease in the amount of functional thyroid tissue and is characterized by a reduced content of thyroid hormones and an increase in the level of thyrotropin in blood serum. In 95% of patients hypothyroidism is primary.

Clinical epidemiology. According to M.P.J. Vanderpump et al. (1995), the frequency of spontaneous hypothyroidism during a 20-year follow-up was 3.5 per 1000 female and 0.8 per 1000 male population. The average age of diagnosis is 60 years. It is worth noting that the frequency of hypothyroidism increases significantly in goiter endemic areas.

Etiology. By etiology, hypothyroidism can be acquired or congenital. Congenital hypothyroidism develops due to aplasia or hypoplasia of the thyroid gland. The most common cause of hypothyroidism is iodine deficiency. Thyroiditis, thyroidectomy, treatment with radioiodine, thyroid irradiation, thyrostatic therapy, long-term treatment with excess iodine, violation of the biosynthesis of thyroid hormones are among the reasons for the development of the syndrome. It is also accepted to distinguish secondary and tertiary (hypothalamic) hypothyroidism.

Pathogenesis. The basis of the pathogenesis of all forms of hypothyroidism is a decrease in the level of thyroid hormones in the blood. The development of secondary hypothyroidism is caused by insufficient production of thyrotropin in the pituitary gland, and tertiary hypothyroidism is caused by a lack of thyroliberin. Hypothyroidism can also develop due to the resistance of peripheral tissues to the action of thyroid hormones. Deficiency of thyroid hormones, which is observed in all forms of hypothyroidism, leads to metabolic disorders and changes in the activity of all systems and organs.

Clinical picture. The main clinical symptoms of hypothyroidism are general weakness, fatigue, drowsiness; poor tolerance to cold, lowering of body temperature; decrease in memory and interest in others; dry skin, hair; weight gain with reduced appetite; low hoarse voice; constipation; disorders of the menstrual cycle; paresthesias, arthralgias, etc.

Classification and examples of formulation of the diagnosis:

Etiological classification of hypothyroidism (A. M. Okorokov, 2000)

I. Primary hypothyroidism (associated with damage to the thyroid itself)

1. Congenital:

• thyroid hypoplasia or aplasia;

• hereditary defects of biosynthesis of thyroid hormones (congenital defects of enzyme systems, defects of thyroglobulin biosynthesis). 2. Acquired:

• postoperative;

•post-radioiodine or as a result of damage by ionizing radiation;

• due to insufficient supply of iodine to the body;

• as a result of the action of drugs (thyrostatic drugs, cordarone);

• as a result of the transferred inflammatory process of the thyroid (autoimmune, viral thyroiditis);

• neoplastic processes of the thyroid gland.

II. Secondary (with damage to the pituitary gland and decreased secretion of thyrotropin):
• adenohypophysis ischemia after bleeding (trauma, childbirth);
• inflammatory processes of the pituitary gland;
• tumors of the brain, pituitary gland;
• autoimmune hypophysitis;
• medicinal effects (treatment with large doses of reserpine, bromocriptine, levodopa).
III. Tertiary hypothyroidism (with damage to the hypothalamus and decreased secretion of thyroliberin): • inflammatory processes of the hypothalamic zone;
• craniocerebral injuries;
• brain tumors;
• long-term treatment with serotonin drugs.
IV. Peripheral hypothyroidism (in case of inactivation of thyroid hormones in the circulation or insensitivity of peripheral tissues to them:
• familial form of decreased sensitivity of receptors of peripheral target tissues to thyroid hormones;
• inactivation of thyroid hormones by antibodies in the circulation process;
• violation of the conversion of thyroxine into triiodothyronine in the liver and kidneys;
• selective resistance to thyroxine (defect of transport of thyroxine through the plasma membrane to the cytosol of the cell).

Clinic. There are subclinical and clinically obvious forms of the disease. Subclinical hypothyroidism is established in the absence of clinical symptoms, an increase in the level of blood thyrotropin and normal levels of thyroxine and triiodothyronine. Clinically obvious (manifest) hypothyroidism is diagnosed in the presence of typical clinical symptoms, an increase in the level of thyrotropin, a decrease in the content of thyroxine and triiodothyronine in the blood.

The clinical diagnosis indicates hypothyroidism, its etiological form, and degree of severity. The mild form is characterized by the presence of drowsiness during the day, edema on the face, hypodynamia, weight gain, decreased memory and attention, a feeling of mild muscle weakness, bradycardia (< 60 bpm) and the presence of thickening of the mitral valve chords on ultrasound of the heart, as well as extrasystoles (up to 15 per hour), psychasthenia.

Moderate hypothyroidism - complaints and clinical signs of the disease indicate manifest hypothyroidism. Characteristic bradycardia (pulse - less than 50 bpm), arterial hypotension, on the ECG - a decrease in the amplitude of the T wave and depression of the ST segment, extrasystole of moderate intensity (15-20 per hour); an ultrasound of the heart reveals asymmetric septal hypertrophy, systolic and diastolic dysfunction of the heart, and a decrease in the ejection fraction. The presence of myopathies, slowing of reflexes, neuropathy is possible. Patients note a decrease in memory and intelligence.

A severe form of hypothyroidism - complaints and clinical manifestations of the disease are clearly defined. Characteristic are bradycardia (up to 40 heart contractions per minute) and arterial hypotension, extrasystole (over 30 per hour), a decrease in the voltage of the waves on the ECG, the presence of asymmetric septal hypertrophy, systolic and diastolic dysfunction, a decrease in the ejection fraction, and an increase in peripheral vascular resistance. Sharply reduced muscle tone and strength. Apathy, hypochondria, melancholy, depressive states, decreased memory and intelligence are characteristic.

Diagnosis of the disease is based on the registration of typical complaints of the patient. Physical examination reveals bradycardia, decreased pulse rate, changes in the cardiovascular system (enlargement of heart borders, hypotension or a slight increase in blood pressure due to activation of the renin-angiotensin-aldosterone system), paleness and dryness of the skin, brittle hair and nails; tendon reflexes are reduced, extremities are cold, swelling is found. One of the early symptoms is an increase in the tongue, a decrease in the tone of the voice. Quite early, patients note a decrease in physical activity, drowsiness. Constipation often occurs, appetite decreases, and body weight increases. During the objective examination of patients, bradycardia and changes in blood pressure are determined.

Laboratory and instrumental studies

On the ECG - bradycardia, low amplitude of R, P waves and the entire QRS complex. When studying biochemical indicators, hypercholesterolemia and hyperlipidemia are determined; increased blood concentration of creatine phosphokinase, lactate dehydrogenase. Decreased glomerular filtration rate of kidneys (in some cases up to 75% of normal). In the urine - moderate proteinuria. A general blood test can detect anemia due to a decrease in the blood content of vitamin B12, folic acid, and iron. In some cases, there is a relative lymphocytosis, an increase in ESR. During the hormonal examination of patients, the level of thyrotropin is increased (typical for primary hypothyroidism), and thyroxine and triiodothyronine are decreased. In secondary and tertiary hypothyroidism, the levels of thyrotropin, thyroxine and triiodothyronine are reduced. In women, there may be disturbances of the menstrual cycle due to changes in the secretion of follitropin, lutropin and an increase in the secretion of prolactin (this may be the reason for the detection of a specific syndrome of lactorrhea-amenorrhea - the Van-Wyck-Ross-Hennes syndrome, in children and adolescents - the Van-Wyck-Grombach syndrome). The time of the Achilles reflex decreases. The size of the thyroid gland on ultrasound can be normal, reduced (aplasia or hypoplasia) or increased in the hypertrophic form of autoimmune thyroiditis. For the diagnosis of the latter, it is important to determine the content of antibodies to thyroperoxidase, thyroglobulin in blood serum (there is an increase in these indicators). Hypothyroidism is characterized by a decrease in the level of ionized and total calcium in the blood, a violation of mineralization of the skeleton.

Diffuse toxic goiter

Definition. Diffuse toxic goiter (Bazedov's disease) is a disease based on hyperfunction of the thyroid gland, its hyperplasia and hypertrophy, and is primarily characterized by changes in the cardiovascular and nervous systems.

The disease occurs more often among women aged 20-50. The ratio among sick men and women is 1:51:7.

Etiology: mental trauma (80%); neurocirculatory dystonia; infection (flu, sore throat, chronic tonsillitis, measles, whooping cough, rheumatism, SLE); hereditary factor (genetic); influence of physical and hormonal factors – overheating in the sun, pregnancy, menopause, hypothalamus pathology. But in general, the disease is considered as an autoimmune process with delayed type hypersensitivity. This is evidenced by the following facts: the presence of protein thyrostimulator in the blood of patients; increase in the titer of antibodies to thyroglobulin, microsomal fraction; violation of cellular immunity; infiltration of lymphocytes and plasma cells of the thyroid gland and retrobulbar tissue; hyperplasia of the

thymus gland; a decrease in the absolute and relative number of T-suppressors and an increase in B-lymphocytes. Leukocyte antigens HLA-B8 are detected much more often.
A thyrostimulating agent is an immunoglobulin (antibody) of class G (IgG), which is formed in B-lymphocytes under the stimulating effect of T-lymphocytes. It was called a long-acting thyroid stimulator (LTTS or LATS). Recently, a TDTS-protector specific only for humans has been found.
Under the influence of TDTS, a factor that acts through TSH receptors of the thyroid gland, hyperproduction of thyroid hormones occurs - the main link in**pathogenesis** diffuse toxic goiter.
Pathogenesis clinical symptoms are due to the effect of thyroid hormones on the nervous, cardiovascular systems, digestive organs, and various types of metabolism.
Exchange violation:
- carbohydrates is manifested by an increase in the absorption of glucose in the intestines, inhibition of the transition of carbohydrates into fats; - proteins: increasing protein breakdown; the occurrence of a negative nitrogen balance.
- fats: strengthening the mobilization of fat from the depot and weight loss of patients.
In addition, there is a violation of vitamin metabolism and water-salt metabolism, including micro- and macroelements.
Pathomorphology. The altered thyroid gland is made up of hyperplastic follicles, the epithelium of which grows from single-layered to multilayered cylindrical. The connective tissue stroma of the gland is excessively vascularized, infiltrated by lymphoid cells. The electrogram shows significant changes in epithelial cells: an increase in the number of colloid droplets, the size of mitochondria and the Golgi complex, and the spread of tubules of the endoplasmic reticulum.
Parenchymatous organs undergo dystrophic changes, areas of necrosis and sclerosis are found in them. This applies to the heart muscle (myocardiodystrophy, myocardiosclerosis), skeletal muscles and liver (fatty degeneration with toxic hepatitis). The central nervous system, gonads, adrenal cortex also undergo changes.

Classification(WHO, 2001)

- 0 - no goiter
- I - the goiter is palpated, but not visualized.
- II - the goiter is palpable and accessible for examination.

According to the degree of severity, toxic goiter is divided into light, medium and severe forms.
Light form is characterized by neurological symptoms, moderate tachycardia (HR about 100 per 1 min.), loss of body weight up to 10%, increase in basic metabolism (up to +30%), total thyroxine up to 190-200 nmol/l and absorption of triiodothyronine (PET3) by erythrocytes on 10-12%.
Form of medium difficulty characterized by significant emotional and vegetative disorders, tachycardia up to 120 per 1 min., heart failure of the I-IIA stage, loss of body weight up to

20%, increase in basic metabolism up to 40%, total thyroxine up to 245-258 nmol/l, PET3 by 12 -13%.

Heavy form – HR more than 120 per 1 min., arrhythmia, heart failure II-III stage, loss of body weight by 30%, increase of thyroxine to 258-270.9 nmol/l, PET3 more than 14%.

Examples of formulation of the diagnosis.

1. Diffuse toxic goiter of the III degree, medium severity, state of decompensation (compensation). Thyrotoxic heart, NC I-IIA st. 2. Diffuse toxic goiter of the II degree, severe form, state of decompensation (compensation). Thyrotoxic heart, atrial fibrillation, tachysystolic form of NK IIA-IIV century. Anovulatory cycles.

Clinical picture. Already during a conversation with the patient, attention is drawn to fussiness, body tremors, verbosity, tearfulness, weight deficit, and in severe cases, muscle volume decreases.

The skin is warm, moist, pigmented. Body temperature is elevated. Hair is brittle. The thyroid gland is enlarged to degrees I-III. Eye symptoms occur in 70% of patients. Eye slits are wide open, a characteristic look reminiscent of anger, surprise (s-mDelrymple), exophthalmos up to 25-26 mm (normally 12-14 mm), eye shine, symptoms of Graefe, Kocher, Mobius, Elinek. Eye symptoms appear as a result of a violation of the tone of the eye muscles due to increased activity of the sympathetic-adrenal system, accumulation of glycosamines in the retrobulbar tissue with its subsequent swelling/

Cardiovascular system. The main symptoms in the clinic of diffuse toxic goiter are changes in the cardiovascular system. "One should never forget that a patient with thyrotoxicosis is, first of all, a patient with heart damage, and taking care of his heart is the main task" (N.A. Shereshevsky). Subjective and objective signs of cardiac disorders, which accompany diffuse toxic goiter, are united by the general term "thyrotoxic heart" (dystrophy, hypertrophy, cardiosclerosis, HNK).

Patients are disturbed by the feeling of frequent heartbeat, pain, suffocation. Tachycardia is the most persistent symptom. Pulse — more than 90 in 1 minute. High pulse pressure is characteristic. Tones are loud, strengthening of the II tone over the pulmonary artery, functional systolic murmur at the top of the heart. Atrial fibrillation is often detected - an indication for urgent radical measures. It occurs as a result of increased excitability of the atrial muscle. In the elderly, thyrotoxicosis can be manifested exclusively by attacks of atrial fibrillation, and between them the pulse rate can be normal, which creates difficulties for diagnosis. Sometimes there are angina attacks as a result of the increased demand of the myocardium for oxygen. Ultimately, a "thyrotoxic heart" occurs, which leads to heart failure.

Digestive organs. The frequency of defecation is noted. Sometimes a spasm of the pylorus is observed - an imitation of an ulcer, pancreatitis. Liver function is often impaired. As a result of thyrotoxicosis, there is an increased permeability of hepatocyte membranes with the development of serous hepatitis and liver cirrhosis.

Nervous System. One of the important manifestations of diffuse toxic goiter is a disturbance of the nervous system. Even Graves (1835) was one of the first to describe this disease under the name "hysteria". Patients are capricious, conflicted, do not get along with others. Their mood often changes, tears easily appear. Marie's positive symptom (hand tremors), as well as the whole body, is a "telegraph pole" symptom. The tremor is constant, it interferes with

work, writing, drawing. On the part of the central nervous system — thyrotoxic encephalopathy: headache, photophobia, double vision, sleep disturbances.
Muscular and endocrine systems. In severe forms, thyrotoxic myopathy develops — weakness of the proximal muscles of the limbs ("cell" symptom).
Disorders of the endocrine sphere are also observed - amenorrhea, hypocorticism, decreased potency and libido.
Definition of hormones. The level of thyroid hormones in the blood is increased, and the level of 0.2 mod/l, the level of T3≤thyroid-stimulating hormone of the pituitary gland is reduced (the level of TSH is increased more than 3 nmol/l, T4 - more than 150 nmol/l).
Thyrotoxic crisis — a life-threatening worsening of the condition of a patient with an accident, which develops mainly in persons with a severe form of the disease. Crises develop in the summer. Reasons - thyroidectomy, use of J131 for therapeutic purposes, intensive thyroid palpation, psychotrauma. It develops when the euthyroid state is not reached. With undiagnosed toxic goiter, in the absence of its treatment, thyrotoxic crisis can be provoked by infections, intoxications, surgical interventions, reactions to various medications.
The crisis develops quickly (hours), less often - gradually (days) due to a sharp increase in the tone of the sympatho-adrenal system, the release of a significant amount of T3 and T4 into the blood, and a decrease in the function of the adrenal cortex. Main symptoms: arrhythmia, temperature up to 40°C, sharp tachycardia (heart rate 200 1 or hypotension, increasing cardiovascular insufficiency. Gastrointestinal disorders rapidly increase (nausea, continuous vomiting, profuse diarrhea, abdominal pain), diffuse sweating, which leads to dehydration of the body. The patient's condition is aggravated by mental agitation, hallucinations, impaired consciousness. In some cases, jaundice develops. Blood analysis reveals leukocytosis, increased ESR, pathological liver tests (cytolytic and cholestatic syndromes). Ultrasound reveals hepatosis, hepatitis.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:
1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking workbooks, communication with a patient with hypothyroidism, thyrotoxicosis, diabetes for the purpose of collecting complaints and anamnesis, physical examination of the patient; evaluation of clinical examination data and laboratory and instrumental data), conducting test control, solving a clinical problem, written solving tasks of the Step-2 type (10 tasks), face-to-face survey, discussion, role-playing on the topic of the lesson.
2.1. Requirements for the theoretical readiness of applicants to perform practical classes: the applicant must know the modern definition, etiology, pathogenesis, classifications of hypothyroidism, thyrotoxicosis, diabetes, subjective and objective data in these diseases, know laboratory and instrumental data in this pathology.

List of didactic units:
- to conduct a subjective examination of patients with hypothyroidism, thyrotoxicosis, and diabetes
- conduct an objective examination of patients with hypothyroidism, thyrotoxicosis, and diabetes
- appoint a plan for additional laboratory and instrumental examinations of patients with hypothyroidism, thyrotoxicosis, diabetes
- evaluate the results of laboratory tests in patients with hypothyroidism, thyrotoxicosis, and diabetes
- master the skills and abilities to assess leading clinical syndromes in patients with hypothyroidism, thyrotoxicosis, diabetes

2.2. Questions to check basic knowledge on the topic of the lesson:
1. Classification of hypothyroidism.
2. Etiology and pathogenesis of hypothyroidism.
3. Clinical signs of hypothyroidism.
4. Methods of laboratory-instrumental diagnosis of hypothyroidism.
5. Classification of thyrotoxicosis.
5. Etiology and pathogenesis of thyrotoxicosis.
6. Clinical manifestations of thyrotoxicosis.
7. Methods of laboratory-instrumental diagnosis of hypothyroidism.

8. Classification of diabetes.
9. Etiology and pathogenesis of diabetes.
10. Main clinical syndromes in diabetes
11. Comatose states in diabetes.
12. Chronic complications of diabetes.

3. Formation of professional skills and abilities:
mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the interview)
formation of the ability to perform a clinical examination of a patient for hypothyroidism, thyrotoxicosis, diabetes (the applicant must be able to perform a physical examination of the patient),

formation of the ability to evaluate the data obtained during the clinical examination of a patient with hypothyroidism, thyrotoxicosis, diabetes; carry out a clinical interpretation of the main symptoms and syndromes in these diseases

formation of the ability to conduct a modern laboratory-instrumental examination of a patient with hypothyroidism, thyrotoxicosis, diabetes (the applicant must be able to prescribe a plan of laboratory-instrumental examination, give an assessment of the examination results)

3.1. Control materials for the final stage of the lesson:
Situational tasks:

Tasks of the STEP-2 type

1. Patient L., 28 years old, complains of sudden general weakness, tearfulness, irritability, palpitations, insomnia, stabbing pains in the heart area, weight loss.
Objectively: height 168 cm, weight 45 kg. The skin is wet. Pulse 128 in 1 min. Blood pressure -150/60 mm Hg. The limits of cardiac dullness have not changed. Heart sounds are increased, systolic murmur at the apex. The thyroid gland is visible when swallowing, when palpating both parts of it are soft and elastic. Pronounced tremor of the fingers. Basic Exchange +40%. On the ECG - sinus tachycardia.
Your previous diagnosis?
A. Myocarditis.
+V. Thyrotoxicosis.
C. Hypothyroidism.
D. Diabetes.
E. Nodular goiter.

2. Patient P., 40 years old, came with complaints of low-grade fever, pronounced general weakness, tremors of the whole body, increased appetite, frequent loose stools. Objectively: malnourished, fussy. The language is hasty, fast. Exophthalmos. The thyroid gland is enlarged. Cardiac activity is arrhythmic, tones are increased.
What disease can be assumed in the patient?
A. Hostria gastroenteritis.
V. Neurosis.
+S. Thyrotoxicosis.
D. Myocarditis.
E. Hypothyroidism.
Q. A 48-year-old patient complains of general weakness, sluggishness, memory impairment, hair loss, dry skin, and long-term constipation. He considers himself a patient for 5 years. The examination revealed swelling of the face, limbs, and trunk. Swelling is dense. The skin is dry, rough, thickened, cold to the touch. There is hair loss in the outer parts of the eyebrows. The voice is low. Pulse 45 in 1 min. Blood pressure 90/50 mm Hg.
What disease should be assumed?
A. Chronic nephritis.
B. Myocarditis.
C. Chronic colitis.
+D. Hypothyroidism.
E. Thyrotoxicosis.

4. A 40-year-old patient under the supervision of an endocrinologist at a regional polyclinic for diffuse toxic goiter underwent tooth extraction. After the operation, the patient's condition suddenly worsened: there was a sharp psychomotor disturbance, hallucinations, hyperemia of the face, an increase in body temperature up to 40°C, pronounced exophthalmos, profuse sweating, uncontrollable vomiting, diarrhea. Pulse 140 in 1 min., arrhythmic. Blood pressure 170/50 mm Hg.
Your diagnosis?
A. Hypertensive crisis.

+V. Thyrotoxic crisis.
C. Acute gastroenteritis.
D. Intoxication delirium.
E. Anaphylactic shock.

5. Patient M., 40 years old, complains of thirst (drinks up to 4 liters of water per day), dry mouth, weakness, itching of the skin. Objectively: height-170 cm, weight-58 kg. Skin and mucous membranes are dry. Tones of the heart are sonorous, clear. Pulse-90 in 1 min. rhythmic, blood pressure -120/80 mm Hg. In the lungs - without features. The language is dry. Abdomen is soft, painless.
What disease should the doctor think about?
A. Chronic nephritis.
B. Myocarditis.
+S. Diabetes.
D. Hypothyroidism.
E. Thyrotoxicosis.

6. Patient M., 40 years old, complains of thirst (drinks up to 4 liters of water per day), dry mouth, weakness, itching of the skin. Objectively: height-170 cm, weight-58 kg. Skin and mucous membranes are dry. Tones of the heart are sonorous, clear. Pulse-90 in 1 min. rhythmic, blood pressure -120/80 mm Hg. In the lungs - without features. The language is dry. Abdomen is soft, painless.
What laboratory parameters should be investigated?
A. Glucose content in urine.
B. The content of glucose in the blood, acetone in the urine.
S. The content of glucose in the blood.
D. Acetone content in urine.
+E. The content of glucose in the blood, glucose and acetone in the urine.

3.2. Requirements for work results, including before registration: substantiation of the diagnosis based on complaints, medical history and life data, clinical and laboratory-instrumental examination.

3.3. Control materials for the final stage of the class: solving two clinical problems on the subject of the class, answering 10 tests (if necessary).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.

Practical lesson No. 39
Topic: Symptoms and syndromes in diabetes.
Goal: Acquiring knowledge and mastering professional competences during the examination of a patient with hypothyroidism, thyrotoxicosis, diabetes mellitus - questioning, physical examination, laboratory-instrumental research.

Basic concepts:
Definition and modern classification of diabetes. The main risk factors and mechanisms of the development of type 1 and type 2 diabetes. Complaints, features of examination of patients and data of physical examination in type 1 and type 2 diabetes. Modern laboratory diagnosis of diabetes mellitus, diagnostic value of the content of glycosylated hemoglobin and immunoreactive insulin. The most frequent comatose states in diabetes (hyperketonemic, hypoglycemic coma), mechanism of development, clinical manifestations, first aid.
The main etiological factors of thyroid gland damage. Hyperthyroid and hypothyroid syndromes in diseases of the thyroid gland. Diffuse toxic goiter. The main complaints of the patient, the results of physical, instrumental and laboratory examination. Concept of thyrotoxic crisis. The main clinical manifestations of myxedema, the concept of hypothyroid coma.
Diabetes mellitus (DM) is defined by WHO experts as a state of chronic hyperglycemia caused by impaired insulin production or action. This is a chronic endocrine-metabolic disease caused by an absolute (impairment of formation) or relative (impairment of action) insulin deficiency as a result of the action of various endogenous (genetic) and exogenous factors, which is accompanied by a violation of all types of metabolism, primarily carbohydrate with its most characteristic manifestation - hyperglycemia, damage to various organs and tissues, primarily blood vessels and nerves.
EPIDEMIOLOGY OF DIABETES.
The prevalence of CD in individual countries varies: in Japan, China - 0.7 - 0.9%, in Ukraine of Western Europe 3-8%, among some American Indian tribes 15-20%. Today, there are more than 170 million people with diabetes in the world.
At the end of 2006, there were about 1 million people with diabetes in Ukraine.
As of January 1, 2000, 902,700 patients with diabetes mellitus were registered in Ukraine, of which 118,100 patients suffered from type I diabetes mellitus, and 784,600 patients suffered from type II diabetes mellitus.
The costs of treating patients are significant. Thus, in the USA (1992), the losses from diabetes amounted to 102 billion dollars (direct costs for treatment - 45 billion dollars, and indirect costs, as a result of disability, etc. - 47 billion dollars). DM is the leading cause of blindness, 50% of all amputations (without trauma). Patients with diabetes are 2-3 times more likely to be diagnosed with CHD, and mortality from CHD is 2-5 times higher, compared to the general population. There is a constant increase in the number of patients with diabetes in the world, which increases by 1.5-2 times in 10 years.
Compared with other diseases, DM is characterized by the earliest disability. Thus, 60-85% of associations of the blind are formed by patients with diabetes mellitus. Approximately 50% of patients with type 1 diabetes die from CKD. Among such patients, myocardial infarction is diagnosed 3-5 times more often than in the population of the same age, and stroke is 2-3 times more often. Gangrene of the lower extremities occurs 200 times more often, 50-70% of amputations of the lower extremities not related to trauma occur in
The reasons causing the increase in the incidence of diabetes are as follows:
an increase in the population structure of persons with a hereditary predisposition to diabetes;

· an increase in the average life expectancy of people with an increase in the percentage of elderly people who are more likely to suffer from diabetes;
· intensification of life;
deterioration of the environmental and social situation, especially in developing countries;
treatment that ensures life extension of diabetic patients;
the nature of the population's diet, which, in combination with hypodynamia, leads to an increase in the number of obese people;
increase in the frequency of chronic cardiovascular diseases (hypertensive disease, atherosclerosis), which are also risk factors;
improving disease detection.

Risk factors for diabetes

Persons with risk factors for the development of diabetes include:

1. – elderly and senile persons;
2. – identical twins, one of whom has diabetes;
3. - persons in whom both or one of the parents suffers from diabetes and there are patients with diabetes in the pedigree of the other parent;
4. - women who gave birth to a live child weighing 4.5 kg or more;
5. – mothers of children with developmental disabilities;
6. women with glucosuria during pregnancy, as well as after miscarriage or stillbirth;
7. - persons suffering from obesity, atherosclerosis, hypertension, hyperuricemia, gout;
8. – patients with manifestations of the metabolic syndrome (insulin resistance, hyperinsulinemia, dyslipidemia, arterial hypertension, hyperuricemia, microalbuminuria, increased aggregation ability of platelets, central or android obesity);
9. patients with diseases of the liver and biliary tract, pancreas, chronic infections of the urinary tract, chronic lesions of the kidneys, respiratory organs;
10. persons with renal and alimentary glycosuria, episodic glycosuria and hyperglycemia, manifested in stressful situations;
11. patients with persistent periodontal disease and furunculosis;
12. patients with neuropathies of unclear etiology;
13. patients with spontaneous hypoglycemia.

In its development, diabetes usually goes through 3 stages, which are more clearly distinguished in patients with type 2 diabetes.

1. Prediabetes – the state (period of the patient's life) that precedes the disease. Laboratory indicators do not go beyond the norm. Risk factors for diabetes mellitus are often observed in patients:

the presence of diabetes in relatives (if both relatives have diabetes, the risk of developing it in children increases to 50%)
obesity (risk increases with increasing degree of obesity)
hyper- and dyslipoproteinemia
birth of a child with a large (over 4 kg) body weight - both for the child and for the mother (the risk increases with increasing weight)
in women, glucosuria during pregnancy; miscarriages and stillbirths
viral diseases
acute and chronic stress
atherosclerosis, hypertension

acute and chronic intoxications
acute and chronic diseases of the liver and pancreas
iatrogenic factors (thiazide diuretics, glucocorticoids, etc.)

2. Violation of glucose tolerance (latent diabetes) is a condition in which there is no clinical picture of diabetes, fasting normoglycemia, and aglucosuria. Violations are detected during STTG. During this period, patients may show the so-called "small symptoms" of diabetes:
chronic gingivitis, stomatitis, periodontitis, tooth loss
chronic pustular skin diseases, furunculosis
itching of the skin, genitals
skin trophic disorders, skin wounds that do not heal for a long time
unmotivated sexual weakness, violation of the menstrual cycle
polyneuritis
cataract

3. Explicit (manifest) diabetes mellitus is a condition when clinical symptoms become manifest, changes in laboratory indicators, primarily hyperglycemia and glycosuria. Clinical symptoms of diabetes, primarily complaints of patients, are caused by the disease itself, the degree of its compensation and complications.

Patients are observed:
polydipsia, thirst, dry mouth;
polyuria, frequent urination, leading to dehydration:
muscle weakness, increased fatigue;
weight loss (type 1 diabetes), weight gain (type 2 diabetes);
loss of appetite (polyphagia is possible at first);
pain in the legs, paresthesias, loss of sensitivity;
itching (local - perineum, scalp or generalized);
impaired vision, impaired sexual function;
chronic pustular or fungal skin lesions

Depending on the clinic and laboratory indicators, there are 3 degrees of severity of diabetes:**light, medium and heavy**

Clinical manifestations of diabetes

• Hyperglycemia is a cardinal manifestation of the disease. Diabetes is chronic hyperglycemia.
• Glucosuria -
• Polyuria –
• Polydipsia-
• Loss of body weight-
• Polyphagia
• Hyperketonemia
• Ketonuria

Type 1 diabetes (insulin-dependent) is a polyetiological disease. The factors that cause its development most often act in a complex way: environmental factors (viruses, toxins, stress) act on a genetically predisposed organism. Several genes are involved in the implementation of the action, and only a certain combination of them leads to a predisposition to diabetes. A proven link between the development of diabetes and HLA - DR3 antigens; DR4; Dw3; Dw4; B8; B18, Bw15; DQB10302; DQA10301, in the presence of which the risk of

developing diabetes increases 7-14 times (the greater the number of antigens, the higher the risk of developing diabetes).
According to modern concepts, IZD is considered an autoimmune disease. Against the background of genetic predisposition, various diabetogenic factors of the external environment act on b-cells - viruses (Coxsackie, retroviruses, mumps, hepatitis A), toxins, food factors (excessive amount of nitrates and nitrites in food, etc.), drugs (cimetidine, glucocorticoids, indomethacin etc.), which lead to the destruction of b-cells. The formed antigens act on the multinuclear phagocytic system, which leads to their inclusion in immune and inflammatory processes and modulation of the immune response through T- and B-lymphocytes (B-lymphocytes produce antibodies, and T-lymphocytes produce growth factors - interleukin IL, g-interferon , tumor necrosis factor). As a result, the production of autoantibodies to b-cells in macrophages increases, which leads to their destruction and continuation of the autoimmune reaction. When type 1 diabetes lasts up to 5–6 years, b-cells in the islets of Langerhans disappear almost completely. The clinic of diabetes is manifested when more than 2/3 of b-cells are destroyed.
Classification of type I diabetes
There are two main subtypes of type I diabetes - autoimmune and idiopathic. With autoimmune diabetes, specific autoantibodies to insulin-producing cells are detected in the blood of patients
Type I diabetes is diagnosed only with typical clinical symptoms (**progressive decrease in body weight, development of ketoacidosis, progressive physical weakness**), **detection of glycemia**.
A diagnosis of diabetes is made with fasting blood glucose >6.1 mmol/L or with glycemia >11 mmol/L in capillary blood at any time of the day. When performing TSH, the diagnosis of diabetes mellitus is made when the blood glucose level is >11.1 or more mmol/l (after 2 hours from the start of the test
Criteria for compensation of carbohydrate metabolism in type I diabetes
• Compensation: Glycated hemoglobin 5-7%, fasting glycemia 5-6 mmol/l, postprandial glycemia 7.5-8 mmol/l, bedtime glycemia 6-7 mmol/l.
• Subcompensation: Glycated hemoglobin 7.1-7.5%, fasting glycemia 6.1-6.5 mmol/l, postprandial glycemia 8.1-9 mmol/l, bedtime glycemia 7.1-7.5 mmol/l.
• Decompensation:**Glycated hemoglobin >7.5%, fasting glycemia >6.5 mmol/l, postprandial glycemia >9 mmol/l, bedtime glycemia >7.5 mmol/l.**
Type 2 diabetes is also a polyetiological disease, but its causes and pathogenesis differ from type 1 diabetes
. is a heterogeneous disease, the basis of which is insulin resistance and insufficient function of ß cells. This type of disease is based on genetic predisposition, and the main provoking factor is obesity. Risk factors are burdened heredity, obesity, hypertension, gestational diabetes, increased triglycerides, decreased HDL cholesterol.
Persons with risk factors for the development of diabetes include:
elderly and senile persons;
– identical twins, one of whom has diabetes;
- persons in whom both or one of the parents suffers from diabetes and there are patients with diabetes in the genealogy of the other parent;
- women who gave birth to a live child weighing 4.5 kg or more;

- mothers of children with developmental disabilities;
- women with glycosuria during pregnancy, as well as after miscarriage or stillbirth; - persons suffering from obesity, atherosclerosis,
hypertension, hyperuricemia, gout;
- patients with manifestations of metabolic syndrome (insulin resistance, hyperinsulinemia, dyslipidemia, arterial hypertension, hyperuricemia, microalbuminuria, increased aggregation ability of platelets, central or android obesity);
-patients with diseases of the liver and biliary tract, pancreas;-persons with renal and alimentary glycosuria, episodic glycosuria and hyperglycemia, which manifests itself in stressful situations;- patients with persistent periodontal disease and furunculosis;– patients with neuropathies of unclear etiology;– patients with spontaneous hypoglycemia.

DIABETES CLINIC

Type 2 diabetes is characterized by a slow development of the disease, especially in the elderly, when the clinical picture is erased, diabetes is not noticeable for years and appears accidentally, against the background of already existing diabetic vascular or nerve damage. Complaints caused by diabetes decompensation are not so demonstrative, they can be episodic. Thirst, polyuria intensifies in the evening, after a meal and becomes pronounced only against the background of pronounced decompensation. However, type 2 diabetes can manifest itself quite acutely, especially when it manifests against the background of infection, intoxication, trauma, etc. It is not uncommon for the development of obvious stages of diabetes, especially type 2, to be preceded by a long period of hidden hypoglycemic states of varying severity caused by hypersecretion of insulin. Clinically, they are manifested by a feeling of hunger, weakness, sweating, tremors, headache, occur after long breaks in food or on the background of physical exertion, are leveled by taking food, especially with carbohydrate content. Clinical manifestations of type 2 diabetes are often characterized by polymorphic symptoms from the first years, reflecting the presence of angioneuropathy of varying degrees of severity, progression, and localization. Decompensation of diabetes mellitus, especially pronounced, is characterized by a clinic common to both types of diabetes mellitus, however, in severe type 1 diabetes, it develops faster, has a more severe course, and is more difficult to treat.

An objective, stable indicator of the state of compensation for diabetes is glycosylated (glycated) hemoglobin (or glycohemoglobin, or HbA1c - a test where Hb is hemoglobin, A1c is attached glucose). Hemoglobin and other proteins combine with glucose in a slow, non-enzymatic reaction that depends on the concentration of glucose. The more glucose is in the blood, the more glycosylated hemoglobin accumulates in erythrocytes. The test for the determination of glycosylated hemoglobin reflects the average level of glucose in the blood during the life of erythrocytes for the last 2-3 months, during which the interaction of hemoglobin and glucose takes place. Normally, the content of HbA1c in the blood is 5-7% of the total level of hemoglobin.

So, hyperglycemia is a cardinal symptom of type 2 diabetes, develops and progresses due to three main mechanisms:
decrease in insulin secretion due to functional insufficiency of the insular apparatus; - tissue resistance to insulin and insufficient utilization of glucose;
- compensatory increase in glucose production by the liver.

The European group on insulin-dependent diabetes in 1993 proposed the following biochemical parameters of control (compensation of diabetes).

Lung (I degree) the form of the disease is characterized by low levels of glycemia, which do not exceed 8 mmol/l on an empty stomach, when there are no large fluctuations in the blood sugar content during the day, insignificant daily glucosuria (from traces to 20 g/l). The state of compensation is supported by dietary therapy. With a mild form of diabetes, angioneuropathy of the preclinical and functional stage can be diagnosed.

At secondary level (II degree) with the severity of diabetes, fasting blood glucose rises, as a rule, to 14 mmol/l, there are fluctuations in blood glucose during the day, daily glucosuria usually does not exceed 40 g/l, ketosis or ketoacidosis develops episodically. Compensation of diabetes is achieved by following a diet and taking oral hypoglycemic agents or by administering insulin (in the case of secondary sulfamide resistance) in a dose not exceeding 40 units per day. Diabetic angioneuropathy of various localization and functional stage may be detected in these patients.

Severe (III degree) the form of diabetes is characterized by high levels of glycemia (fasting more than 14 mmol/l), significant fluctuations in blood sugar during the day, high level of glucosuria (over 40-50 g/l). Patients require constant insulin therapy at a dose of 60 IU or more, they have various diabetic angioneuropathy. With type 2 diabetes, the development of severe organic lesions of blood vessels with impaired vision, kidney, heart, brain, and lower limbs function makes it possible to consider the disease as severe, regardless of glycemia and glycosuria indicators, the dose and nature of hypoglycemic therapy.

When diabetes is detected for the first time, a conclusion about the severity of the disease can be made only on the basis of dynamic observations against the background of adequate therapy. With a long-term course of type 2 diabetes, especially against the background of the development of severe vascular lesions, the clinical picture is practically no different from the symptoms of type 1 diabetes, patients are forced to be treated with insulin for a number of reasons, despite the original, non-insulin-dependent nature of diabetes. In such patients, in the diagnosis, the original terminology for determining the type of diabetes should be left, that is, diabetes, as before, should be classified as type 2 non-insulin-dependent diabetes with secondary sulfamide resistance.

Indications for a glucose tolerance test

• Temporary disorders of carbohydrate metabolism:
• - glucosuria of pregnant women
• - hyperglycemia after a meal up to 9.9 mmol/l
• - reactive hypoglycemia
• 2. Presence of diabetes risk factors:
• - hereditary predisposition
• - overweight
• - pathological pregnancy and childbirth (miscarriage,
• polyhydramnios, stillbirth, toxicosis of pregnant women,
• weight of newborns >4.1 kg)
• - damage to peripheral vessels, arterial hypertension
• - chronic infections
• - dermatopathies
• hyperlipidemia, hyperuricemia

• retinopathy and neuropathy of unknown cause

Diagnostics

For the diagnosis of diabetes, assessment of the severity and state of compensation of the disease, it is important to determine the level of sugar in the blood, its repeated determinations throughout the day, the study of daily and fractional glucosuria in separate portions, the determination of the content of ketone bodies in urine and blood, the study of the dynamics of the level of glycemia in various forms glucose tolerance test.

Overt diabetes mellitus is diagnosed based on the detection of an increase in blood sugar and the appearance of sugar in the urine. Blood is examined on an empty stomach. Glucosuria is determined in daily urine or daytime, or in a portion of urine collected 2 hours after a meal. Examining only morning urine is not indicative, since glucosuria is usually not detected in urine collected on an empty stomach in mild forms of diabetes. In the case of a slight increase in the level of fasting blood sugar, the diagnosis can only be established if unambiguous results are obtained again, supported by the detection of glucosuria in the daily urine or in its separate portions. To clarify the diagnosis in such cases, determination of glycemia during the day against the background of the patient's food consumption helps. With untreated overt diabetes, the blood sugar level during the day exceeds 10 mmol/l (180 mg%), which is the basis for the appearance of glucosuria, since the renal threshold for glucose permeability is on average 9.5 mmol/l (170-180 mg%) .

If there is a slight increase or normal values of blood sugar in combination with episodic glucosuria during repeated examinations, or there are any other doubts about the diagnosis, a glucose tolerance test (TSG) is used. Even an incidental, one-time finding of hyperglycemia or glycosuria requires careful investigation to rule out or confirm diabetes.

If the subject has an infectious disease or fever, the test cannot be performed. TSH indicators are affected by the following factors: prolonged bed rest, diseases of the digestive tract with impaired glucose absorption, malignant diseases, taking certain drugs (adrenaline, glucocorticoids, caffeine, diuretin, morphine, thiazide diuretics, diphenine, psychotropic drugs and antidepressants). They should be taken into account during the diagnosis.

According to the report of the WHO Expert Committee "Diabetes" (1999), the criteria for diagnosing diabetes are: fasting capillary blood glucose > 6.1 mmol/l (in blood plasma > 7.0 mmol/l); 2 hours after glucose load in capillary blood > 11.1 mmol/l (in blood plasma > 11.1 mmol/l); glucose concentration in a randomly taken blood sample > 11.1 mmol/l in combination with characteristic clinical symptoms (polydipsia, polyuria, weight loss).

Criteria for diabetes mellitus and impaired glucose tolerance (WHO Expert Committee on Diabetes Mellitus)

An auxiliary diagnostic criterion for verifying the diagnosis of diabetes is the determination of glucose in urine collected after a glucose load. People with suspected DM should not have TSH. In such patients, it is advisable to examine blood glucose levels after a meal, only with their normal fluctuations (7.7-8.0 mmol/l) to conduct the test. If one of the indicators of the 2-hour simplified TSH is elevated, it is recommended to conduct a classic TSH after 1 month under the condition of normal nutrition. At least 3 days before the test, the subject should receive 150-200 g of carbohydrates per day, including easily digestible ones.

SKIN, MUSCLE AND BONE DAMAGE IN PATIENTS WITH DIABETES.

Skin lesions in patients with diabetes mellitus are diverse and are united under the single term "diabetic dermopathy": dry skin, rubeosis on the face, yellowness of the palms and soles

(due to impaired carotene metabolism), hyperkeratosis (primarily on the feet), thickening of the nails. Often pustular diseases that are poorly treated. Xanthomatosis - yellowish fat nodules on the palms and feet, the back of the elbow joints.
Lipoid necrobiosis is characteristic - painful red-purple nodes, smooth to the touch with a waxy sheen and clear edges, more often on the lower legs. Merging, they can form rings with a diameter of up to 10 cm. An ulcer can form in the center of the focus. It is believed that this is a kind of metabolic disorder in the skin with deposition of glycogen and lipids against the background of microangiopathy of the skin.
Damage to the bone system is a consequence of metabolic disorders and is observed in the majority of patients with a long course. Systemic osteoporosis, osteoarthritis, osteoarthropathies are characteristic. There is a gradual destruction of the bones. Pathological dislocations and deformations are possible, mainly in the feet. Patients complain of pain in the lower back, in the bones, often pain when percussing the bones. Radiologically, osteoporosis, osteosclerosis, gradual destruction of the bone structure and their reconstruction are observed. Schmorghl's hernia and Dupuytren's contracture occur 2-3 times more often in patients with diabetes.

INFRINGEMENT OF INTERNAL ORGANS (GASTROINTESTINAL TRACT, CARDIOVASCULAR SYSTEM, LUNGS, URINARY SYSTEM) AND FEATURES OF THEIR COURSE IN PATIENTS WITH DIABETES

1. Damage to the digestive organs in diabetes is often observed. Patients have tooth loss, gingivitis, stomatitis. The acid-forming and secretory functions of the stomach are reduced (as a result of hyperglycemia, lack of insulin, microangiopathy of the stomach, etc.). Chronic gastritis, gastroduodenitis, and possible gastroparesis with impaired evacuation are observed in 60% of patients with a disease duration of more than 4 years. Chronic diseases (ulcer disease, cholecystitis, appendicitis, etc.) in patients with diabetes mellitus often have a mild course of symptoms.
Damage to the cardiovascular system is the most frequent in patients with diabetes and in the long course reaches 100%. Atherosclerosis in patients with diabetes mellitus is observed much more often than among the general population and has its own characteristics:
generalized character;
early onset (at a young age in patients with type 1 diabetes);
heavy run;
equally often in patients of both sexes.
Atherosclerotic lesions of coronary vessels, brain vessels and lower extremities are most often observed. Atherosclerosis is the main reason for the development of myocardial infarction, cerebral strokes and gangrene of the lower extremities in patients with diabetes. These lesions are the most common cause of death.
In patients with diabetes mellitus, acute myocardial infarction (MI) develops 3–5 times more often and its course, compared to persons without diabetes mellitus, has its own characteristics:
MI clinic is asymptomatic, often painless forms or with atypical pain syndrome;
often atypical variants (abdominal, asthmatic, arrhythmic, colaptoid, etc.);
frequent MI is transmural, large-focal, recurrent (recurrent MI is 1.5–2 times more likely);
the posterior wall of the myocardium is more often affected;

the course of MI is more severe and the prognosis is worse (mortality is twice as high);
inconsistency between clinic and ECG: ECG – dynamics are slowed down, negative "T" is formed late;
complications often occur: shock, aneurysm, arrhythmias, pulmonary edema, etc.

Features of the course of coronary heart disease (angina) in patients with diabetes mellitus:

• CHD occurs equally often in men and women
• attacks of little intensity, atypical (its manifestation may be weakness, sweating, palpitations, shortness of breath, which are relieved by nitroglycerin);
• irregular, atypical irradiation;
• attacks often occur with hypoglycemia, at rest.

CHD in patients with diabetes should be differentiated from diabetic cardiomyopathy, which is characterized by:

• occurs more often at a young age;
• mainly in patients with type 1 diabetes with a duration of more than 5 years
• often in persons without increased blood pressure and obesity
• pain in the heart according to the type of cardialgia, not angina, often without pain;
• pronounced microangiopathies;
• more often with a decompensated, labile course;
• CNS is more often of the right ventricular type;
• existing diabetic autonomic neuropathy;
• ECG: the voltage is reduced, T±, a test with dosed exercise without characteristic changes (in case of coronary heart disease, a decrease in the S–T segment, inversion and discordance of T), a decrease in coronary reserve, a positive test with K, a negative test with obzidan)

Features of the course of bronchopulmonary pathology in patients with diabetes:

1. Chronic processes often occur, pneumonias have a propensity for a protracted course, the development of abscesses and gangrene. The course is often atypical, with few symptoms.
2. Tuberculosis of the lungs:

a) more often in young people;
b) localization of the process is atypical - in the center or at the gates of the lungs, often bilateral damage;
c) the course is mildly symptomatic with the formation of "silent" caverns;
d) the process progresses rapidly, often the development of exudative forms (up to 50%).

Damage to the urinary and reproductive systems.

`1. Infection of the urinary tract - acute and chronic pyelonephritis, suppuration of the kidneys (carbuncle, abscess, apostematosis), which can lead to bacteremic shock. A feature of their course is mild symptoms, often latent forms, without a temperature reaction with severe intoxication. The course of diabetes mellitus becomes more severe, the infection contributes to decompensation of diabetes mellitus, ketoacidosis.
2. Diabetic nephropathy.
3. Diabetic cystopathy - the basis is visceral neuropathy, which can lead to bladder paresis. The urge to urinate is rare, the bladder is full (up to 500-1000 ml).
4. The function of the genital organs is disturbed. Men often have a decrease in libido, potency; in women - infertility, miscarriages, stillbirths, amenorrhea, vulvitis, vaginitis.

Diabetic angiopathy is a generalized lesion of blood vessels in patients with diabetes mellitus. Damage to small vessels (arterioles, capillaries, venules) is called microangiopathy

and has a specific character, damage to large vessels is called macroangiopathy and is considered early and widespread atherosclerosis.
1. Diabetic nephropathy
Diabetic nephropathy is one of the main causes of disability and mortality in patients with diabetes mellitus: up to 50% of patients with diabetes mellitus type 1 die before the age of 50 due to nephropathy. Nephropathy occurs more often in patients with type 1 diabetes (40-50% versus 20-30% in patients with type 2 diabetes). Histologically, nodular (intercapillary) glomerulosclerosis is pathognomonic for nephropathy.
Proteinuria is an early clinical sign. which indicates deep damage to the kidneys, which is irreversible. The main preclinical criterion of nephropathy is microalbuminuria (30-300 mg/day), which is determined by the radioimmunoassay method or with the help of special strips.
Diabetic retinopathy (DR) is the main cause of blindness, which occurs 25 times more often in patients with diabetes compared to the general population, and visual disability occurs in more than 10% of patients. DR develops in patients with both types of diabetes.
Diabetic foot is one of the manifestations of damage to the lower extremities in patients with diabetes. Diabetic foot is a complex complex of anatomical and functional changes caused by micro- and macroangiopathies and neuropathy in the long course of diabetes. It is based on neuropathy, ischemia and infection. It occurs in 10-20% of patients. There are 3 clinical forms of diabetic foot syndrome: neuropathic, ischemic and mixed.
To**gestational diabetes** include only diabetes detected during pregnancy (about 2% of pregnant women). In general, the manifestations of gestational diabetes disappear after childbirth and carbohydrate tolerance normalizes. However, after 5-15 years, 20-40% of women with gestational diabetes develop clinical diabetes. Pregnancy with gestational diabetes has an increased risk of perinatal mortality and fetopathy.

Equipment:study room, multimedia presentation on the topic of classes, laptop, multimedia projector.

Plan:
1. Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).
2. Control of the reference level of knowledge (checking workbooks, communication with a patient with hypothyroidism, thyrotoxicosis, diabetes for the purpose of collecting complaints and anamnesis, physical examination of the patient; evaluation of clinical examination data and laboratory and instrumental data), conducting test control, solving a clinical problem, written solving tasks of the Step-2 type (10 tasks), face-to-face survey, discussion, role-playing on the topic of the lesson.
2.1. Requirements for the theoretical readiness of applicants to perform practical classes: the applicant must know the modern definition, etiology, pathogenesis, classifications of hypothyroidism, thyrotoxicosis, diabetes, subjective and objective data in these diseases, know laboratory and instrumental data in this pathology.

List of didactic units:

- to conduct a subjective examination of patients with hypothyroidism, thyrotoxicosis, and diabetes
- conduct an objective examination of patients with hypothyroidism, thyrotoxicosis, and diabetes
- appoint a plan for additional laboratory and instrumental examinations of patients with hypothyroidism, thyrotoxicosis, diabetes
- evaluate the results of laboratory tests in patients with hypothyroidism, thyrotoxicosis, and diabetes
- master the skills and abilities to assess leading clinical syndromes in patients with hypothyroidism, thyrotoxicosis, diabetes

2.2. Questions to check basic knowledge on the topic of the lesson:
1. Classification of hypothyroidism.
2. Etiology and pathogenesis of hypothyroidism.
3. Clinical signs of hypothyroidism.
4. Methods of laboratory-instrumental diagnosis of hypothyroidism.
5. Classification of thyrotoxicosis.
5. Etiology and pathogenesis of thyrotoxicosis.
6. Clinical manifestations of thyrotoxicosis.
7. Methods of laboratory-instrumental diagnosis of hypothyroidism.

8. Classification of diabetes.
9. Etiology and pathogenesis of diabetes.
10. Main clinical syndromes in diabetes
11. Comatose states in diabetes.
12. Chronic complications of diabetes.

3. Formation of professional skills and abilities:
mastering communication skills (collecting complaints, detailing complaints, collecting anamnesis, evaluating the results of the interview)
formation of the ability to perform a clinical examination of a patient for hypothyroidism, thyrotoxicosis, diabetes (the applicant must be able to perform a physical examination of the patient),

formation of the ability to evaluate the data obtained during the clinical examination of a patient with hypothyroidism, thyrotoxicosis, diabetes; carry out a clinical interpretation of the main symptoms and syndromes in these diseases

formation of the ability to conduct a modern laboratory-instrumental examination of a patient with hypothyroidism, thyrotoxicosis, diabetes (the applicant must be able to prescribe a plan of laboratory-instrumental examination, give an assessment of the examination results)

3.1. Control materials for the final stage of the lesson:
Situational tasks:
Tasks of the STEP-2 type

1. Patient L., 28 years old, complains of sudden general weakness, tearfulness, irritability, palpitations, insomnia, stabbing pains in the heart area, weight loss.
Objectively: height 168 cm, weight 45 kg. The skin is wet. Pulse 128 in 1 min. Blood pressure -150/60 mm Hg. The limits of cardiac dullness have not changed. Heart sounds are increased, systolic murmur at the apex. The thyroid gland is visible when swallowing, when palpating both parts of it are soft and elastic. Pronounced tremor of the fingers. Basic Exchange +40%. On the ECG - sinus tachycardia.
Your previous diagnosis?
A. Myocarditis.
+V. Thyrotoxicosis.
C. Hypothyroidism.
D. Diabetes.
E. Nodular goiter.

2. Patient P., 40 years old, came with complaints of low-grade fever, pronounced general weakness, tremors of the whole body, increased appetite, frequent loose stools. Objectively: malnourished, fussy. The language is hasty, fast. Exophthalmos. The thyroid gland is enlarged. Cardiac activity is arrhythmic, tones are increased.
What disease can be assumed in the patient?
A. Hostria gastroenteritis.
V. Neurosis.
+S. Thyrotoxicosis.
D. Myocarditis.
E. Hypothyroidism.
Q. A 48-year-old patient complains of general weakness, sluggishness, memory impairment, hair loss, dry skin, and long-term constipation. He considers himself a patient for 5 years. The examination revealed swelling of the face, limbs, and trunk. Swelling is dense. The skin is dry, rough, thickened, cold to the touch. There is hair loss in the outer parts of the eyebrows. The voice is low. Pulse 45 in 1 min. Blood pressure 90/50 mm Hg.
What disease should be assumed?
A. Chronic nephritis.
B. Myocarditis.
C. Chronic colitis.
+D. Hypothyroidism.
E. Thyrotoxicosis.

4. A 40-year-old patient under the supervision of an endocrinologist at a regional polyclinic for diffuse toxic goiter underwent tooth extraction. After the operation, the patient's condition suddenly worsened: there was a sharp psychomotor disturbance, hallucinations, hyperemia of the face, an increase in body temperature up to 40°C, pronounced exophthalmos, profuse sweating, uncontrollable vomiting, diarrhea. Pulse 140 in 1 min., arrhythmic. Blood pressure 170/50 mm Hg.
Your diagnosis?
A. Hypertensive crisis.
+V. Thyrotoxic crisis.

C. Acute gastroenteritis.
D. Intoxication delirium.
E. Anaphylactic shock.

5. Patient M., 40 years old, complains of thirst (drinks up to 4 liters of water per day), dry mouth, weakness, itching of the skin. Objectively: height-170 cm, weight-58 kg. Skin and mucous membranes are dry. Tones of the heart are sonorous, clear. Pulse-90 in 1 min. rhythmic, blood pressure -120/80 mm Hg. In the lungs - without features. The language is dry. Abdomen is soft, painless.
What disease should the doctor think about?
A. Chronic nephritis.
B. Myocarditis.
+S. Diabetes.
D. Hypothyroidism.
E. Thyrotoxicosis.

6. Patient M., 40 years old, complains of thirst (drinks up to 4 liters of water per day), dry mouth, weakness, itching of the skin. Objectively: height-170 cm, weight-58 kg. Skin and mucous membranes are dry. Tones of the heart are sonorous, clear. Pulse-90 in 1 min. rhythmic, blood pressure -120/80 mm Hg. In the lungs - without features. The language is dry. Abdomen is soft, painless.
What laboratory parameters should be investigated?
A. Glucose content in urine.
B. The content of glucose in the blood, acetone in the urine.
S. The content of glucose in the blood.
D. Acetone content in urine.
+E. The content of glucose in the blood, glucose and acetone in the urine.

3.2. Requirements for work results, including before registration: substantiation of the diagnosis based on complaints, medical history and life data, clinical and laboratory-instrumental examination.

3.3. Control materials for the final stage of the class: solving two clinical problems on the subject of the class, answering 10 tests (if necessary).
4. Summary, announcement of assessment results, announcement of the topic of the next lesson.