

**MINISTRY OF HEALTH OF UKRAINE**

**ODESA NATIONAL MEDICAL UNIVERSITY**

**Faculty: medical №1**

**Department of propaedeutics of internal diseases and therapy**

**APPROVED BY.**



Deputy-rector for scientific and pedagogical work

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**METHODOLOGICAL DEVELOPMENT  
FOR PRACTICAL CLASSES IN THE DISCIPLINE**

Faculty, course: dentistry , 3

Discipline: Propedeutics of internal medicine

Approved:

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## **Practical lesson No. 1**

**Subject: Admission to the internal medicine clinic. Methods of survey and physical examination of the patient. Additional examination methods. Scheme of medical history.**

**Goal:** To know about the propaedeutics of internal diseases as a science, its purpose and tasks, the role of Ukrainian 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:** propaedeutics, survey, complaints, anamnesis, examination, medical history.

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, spirometry 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.

**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.

**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.

#### 4. Summary:

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#### 5. List of recommended literature (main, additional, electronic online information resources):

##### **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. Methods 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.– 13<sup>th</sup> 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](http://www.ama-assn.org) – American Medical Association /American Medical Association
3. [www.who.int](http://www.who.int) - World Health Organization
4. [www.dec.gov.ua/mtd/home/](http://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](http://www.gmc-uk.org) - General Medical Council (GMC)
7. [www.bundesaerztekammer.de](http://www.bundesaerztekammer.de) – German Medical Association
8. <https://onmedu.edu.ua/>
9. <https://onmedu.edu.ua/kafedra/propedevniki-vnutrishnih-hvorob-ta-terapii/>

## **Practical lesson No. 2**

**Subject: General examination of the patient. Examination of individual parts of the body.**

**Research methods of the respiratory system. Questioning and general examination of a patient with a pathology of the respiratory system.**

**Goal:** 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:** Examination of the patient, palpation, percussion, auscultation, questioning, history, complaints.

**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 Obratsov-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 (inspection)*** 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.

*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** (*soapr*, 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*), *acheiroparesis* (*acheiroparesis 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 hyperkinesia 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 *asgigantism* (*gygantismus*), and may be due to hyperfunction of the anterior lobe of the pituitary gland (secreting somatotrophic hormone) or hypofunction of the gonads (eunuchoidism, hypogonadism). Height below 130 centimeters is considered *asdwarfism* (*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:

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

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 ( $\approx 90^\circ$ ), 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 ( $>90^\circ$ ), 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 ( $<90^\circ$ ), 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 (hypersthensics are characterized by disturbances of metabolism, reactivity, vegetative reactions; for asthenics – insufficient development of connective tissue, disorders of the digestive system).

Clinical significance:

- in hypersthensics - 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 (Bertollet 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 labialis*; 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" (*astravascularia 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 anastomoses 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 *nystagnus* - 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<sub>2</sub>.

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:**

2. **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:**

6. Describe the tasks of the department of propaedeutics of internal diseases.
7. Name the main sections of the ictopia of the disease.
8. What is the significance of general examination in general clinical diagnosis?
9. Plan for general inspection. Conditions and technique of general inspection.
10. The patient's condition and criteria for its assessment.
11. Consciousness, criteria for assessing consciousness.
12. Types of impaired consciousness: blackout, stupor, sopor, coma; their reasons.
13. Name the types of the patient's position.
14. What type of unconsciousness does fainting belong to?
15. For which diseases is facial blushing characteristic: a) unilateral, b) bilateral?
16. What type of cyanosis is observed with mitral stenosis
17. 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.*

- F. Violent attacks
- G. Expiratory shortness of breath
- H. Cough
- I. Chest pain
- J. **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:*

- F. Stupor
- G. **Sopor**
- H. Coma

- I. Delirium
- J. Dusk

*Task 2. What heart disease is characterized by pulsation of the carotid arteries ("carotid dance"):*

- F. Mitral insufficiency
- G. **Aortic insufficiency**
- H. Insufficiency of the tricuspid valve
- I. Aortic stenosis
- J. 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.**

**Practical class no3**

**Subject:** The main syndromes in lung pathology based on the results of the survey and physical examination.

**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.

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.

**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* — 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.

*pains* in the chest in patients with diseases of the respiratory organs often increase during

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.

Examination of the chest is also carried out for the purpose *assessment of its shape, size, symmetry and charactera 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).

*Dynamic examination of the chest* with the use of deep breathing allows you 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.*

*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** (*vocal cord s.peceoral*) – chest tremor caused by the transmission to its surface of low-frequency vibrations of the vocal cords at the moment of pronouncing words containing the letter "r", 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 vocal tremor:*

- 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 tremors (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).

**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 lungs** be 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 plesimeter 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.

**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 readiness acquirers 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 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:** Study of patients with cardiovascular pathology (survey, physical examination, assessment of pulse and blood pressure). Additional research methods (laboratory and instrumental).

**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.
- learn additional methods of laboratory and instrumental diagnostics

**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.

**Complaints** with 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, atherosclerosis, 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)

***Swelling*** of 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. 5*

**Subject:**The main syndromes in diseases of the cardiovascular system.

**Goal:**Acquiring knowledge and mastering professional competences during the examination of a patient with diseases of the cardiovascular system - questioning, physical examination, laboratory-instrumental studies.

**Basic concepts: 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 sounds**consists 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).

**Strengthening** of 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.

**Weakening** of 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 cardiomyopathies, 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 aorta** associated with such diseases as primary arterial hypertension, secondary hypertension, atherosclerosis of vessels, ischemic heart disease, syphilitic mesoaortitis.

**Accent II tone over the pulmonary trunk** observed 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 bifurcation** heart 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 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 tone** is 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).

Arterial hypertension

WHO/MTM 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 precordial 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 PECLLO 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 (IV 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 PECLLO 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, isohyponaturia 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 PECCO, 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, renovascular 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 *Istage* characterized only by an increase in HELL (160/95 mm Hg and above), while there are no organic changes in the cardiovascular system.

*GBIn 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 PEKLO 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)

PRESSURE category	Blood pressure	
	systolic	diastole
Optimal	< 120 and < 80	
Normal	< 130 and < 85	
High is normal	130-139 or 85-89	



PRESSURE category	Blood pressure	
	systolic	diastole
Arterial hypertension	140-159 or 90-99  160-179 or 100-109  180 or 110	
I stage		
II stage		
III stage		

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

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

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

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

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

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

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

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.

**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.
7. Pathogenesis of coronary heart disease?
8. Pathogenesis of the development of myocardial infarction?
9. Complaints of patients with different functional classes of angina pectoris?
10. Complaints of patients with anginal form of myocardial infarction?

11. What is the mechanism of diastolic gallop rhythm in myocardial infarction?

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. 6**

**Subject:** Examination of patients with pathology of digestive organs. Survey and general examination of patients with pathology of the digestive system. Additional research methods.

**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 Obratsov-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. 7.**

**Subject:** Basic symptoms and syndromes in gastroenterology based on physical examination data. Changes in the oral cavity in diseases of the gastrointestinal tract (GI).

**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 lamina propria 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. pylori*). 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 <sup>13</sup>C 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 40-50% 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<sup>+</sup> 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 gastrinogenau, 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, 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.

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 fistful 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 atropine or 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, chromogastroscoy 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  $^{14}\text{C}$  or  $^{13}\text{C}$  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. 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-cholinolytic (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  $\mu$ V) and the total power of gastric biopotentials (over 900  $\mu$ V/minute), the gastrogram curves are asymmetric. In case of mediogastric ulcers, on the contrary, hypokinesia and hypodyskinesia 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 trioleate glycerin 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 metoclopramide (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, 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 precordial region, appear 15-30 minutes after eating. Taking antacids helps reduce pain. With ulcers of the cardiac and subcardiac 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 subcardiac 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 subcardiac 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 cyclicality, 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

10. 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

11. 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

12. 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

13. 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

14. 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.

What disease can be assumed in the patient?

- +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.

1.

### **Practical lesson No. 8**

**Subject:** Questioning and examination of a patient with pathology of the liver and biliary tract

**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 Obratzov 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 Obratzov 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 plesimeter 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 ( $\pm 1-2$  cm); – the second straight line (between the 3rd and 4th points) – 8 cm ( $\pm 1-2$  cm); – oblique (between the 3rd and 5th points) – 7 cm ( $\pm 1-2$  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 angiochololiths, 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 Obratsov-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 urokinase-diphosphate-glucosyltransferase (inherited in an autosomal dominant type) or as a result of a defect in the process of binding bilirubin to ligandins (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  $\mu\text{mol/l}$ , rarely up to 140  $\mu\text{mol/l}$ . 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 cytoplasmic 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,  $\beta$ -mercaptopurine, 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  $\mu\text{mol/l}$ ), dysproteinemia, moderate activity of cytolytic enzymes in the blood.

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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  $\mu\text{mol/l}$ , 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  $\mu\text{mol/l}$  and above), hyperglycemia, blood urea rises by 15 to 20 mmol/l and above, creatinine – above 300  $\mu\text{mol/l}$ . 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.

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. 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 plesimeter 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 ( $\pm 1-2$  cm); – the second straight line (between the 3rd and 4th points) – 8 cm ( $\pm 1-2$  cm); – oblique (between the 3rd and 5th points) – 7

cm ( $\pm 1-2$  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), venous blood stasis in the liver (due to circulatory disorders), bile stasis (stone or cancer of the hepatic or common bile duct), hypertrophic liver cirrhosis, and (in the initial stage), liver tumors, its 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 angiochololiths, 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 Obratzsov-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. 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 urokin-diphosphate-glucuronyltransferase (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  $\mu\text{mol/l}$ , rarely up to 140  $\mu\text{mol/l}$ . 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,  $\beta$ -mercaptopurine, 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.

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### Cholangitis

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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 40°C, 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 Obratzov 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. 9**

**Subject: Main symptoms and syndromes in diseases of the liver and biliary tract (portal hypertension syndrome, hepatolienial syndrome, jaundice syndrome)**

**Goal:** Acquiring knowledge and mastering professional competencies during questioning and physical examination of a patient with pathology of the liver and biliary tract (examination, percussive determination of the limits and sizes of the liver according to Obratzov 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:**

**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 Obratzov 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 Obratzov 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.
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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 Obratzov 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

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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?

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B - gastric ulcer

- C - pancreatitis
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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
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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?

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- D - Cholecystitis
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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
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- 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
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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

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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?

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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?

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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

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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. 10*

**Subject: Methods of examination of patients with pathology of urinary organs. Laboratory and instrumental methods of research of the urinary system.**

**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 cavitory 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.

**Atanamnesis 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. *Anuria* - 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 eclampsia - 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 *isuremia (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.

Correct 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, anemic, 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.

Right 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  $\mu\text{mol/l}$ .

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  $\mu\text{mol/l}$ . 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 ( $38^{\circ}\text{C}$ ), 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.

### *Practical lesson No. 11*

**Subject:** Basic syndromes in nephrology. Changes in the oral cavity in kidney diseases.

**Goal:** Acquiring knowledge and mastering professional competences during the examination of the patient for the main syndromes in nephrology.

**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-Kakovskiy, 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;

I 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*** ( $\beta$ -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 ***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 nephrobiopate 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.

**Athypertensive** 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.

**Pyelonephritis** is 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 hectic 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  $30-40 \times 10^9/l$ , 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 -  $>3$ g/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<sup>2</sup> lasting 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<sup>2</sup>);
- CKD II: kidney damage from CKD with a moderate decrease in GFR ( $60-89$  ml/min/1.73

m<sup>2</sup>);

- CKD III: kidney damage from CKD with an average degree of GFR reduction (30-59 ml/min/1.73 m<sup>2</sup>);
- CKD IV: kidney damage from CKD with a significant decrease in GFR (15-29 ml/min/1.73 m<sup>2</sup>);
- CKD V: kidney damage with terminal CKD (< 15 ml/min/1.73 m<sup>2</sup>).

**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. Concept of acute and chronic pyelonephritis.
    3. The main syndromes in pyelonephritis
    4. The main syndromes in glomerulonephritis
    5. Changes in the general analysis of urine and functional tests in patients with glomerulonephritis.

6. Changes in the general analysis of urine and functional tests in patients with pyelonephritis.

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  $\mu\text{mol/l}$ . 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  $\mu$ mol/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  $\mu\text{mol/l}$ , 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 hypoisostenuria. What test detects the concentration function of the kidneys?

A. General analysis of urine.

V. Three-glass test.

+S. Zimnitsky'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 Zimnitskyi, the weight is 1009-1013. Serum creatinine - 420  $\mu\text{mol/l}$ , 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.

11. 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  $\mu\text{mol/l}$ . 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.

12. 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. Pasternatskyi's symptom on the left sharply (+). In the urine - specific weight 1020, protein 0.99 g/l, pyuria (leukocytes 100-120 in p/zr), fresh erythrocytes 20-30 in p/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. 12*

**Topic:** Research methods of patients with pathology of hematopoietic organs. Survey and general examination of patients with pathology of the blood system. Clinical interpretation of general blood analysis. Coagulogram analysis.

**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.

### *Practical lesson No. 13*

**Subject:** Main syndromes in hematology. Manifestations of blood diseases from the mucous membrane of the oral cavity and the dental and jaw system

**Goal:** Acquiring knowledge and mastering professional competences during examination of a patient with anemia, leukemia, hemorrhagic diathesis - questioning, physical examination, laboratory-instrumental studies.

#### **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<sub>12</sub>- 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<sub>12</sub>- folic acid deficiency anemia. The main laboratory signs of V<sub>12</sub>- 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  $4 \times 10^{12}/l$  in men and, respectively, less than 120 g/l and  $3.5 \times 10^{12}/l$  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\* $>100 \mu\text{m}^3$  (fl\*\*), erythrocyte diameter  $> 8 \mu\text{m}$ ).**

This type of anemia includes vitamin B deficiency anemia<sub>12</sub>, folic acid deficiency and paroxysmal nocturnal hemoglobinuria.

**II. Microcytic anemia (MCV $<80 \mu\text{m}^3$  (fl), erythrocyte diameter  $< 6.5 \mu\text{m}$ ).**

This type of anemia includes iron deficiency anemia, thalassemia, Minkovsky-Shafar microspherocytic anemia.

**III. Normocytic anemia (MCV  $81-99 \mu\text{m}^3$  (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 ( $1 \text{ fl} = 10^{-15}\text{l} = 1 \mu\text{m}^3$ )

MCV (mean corpuscular volume) is the average volume of an erythrocyte, calculated according to the formula:

Calculation example: hematocrit 0.3 (30%), erythrocytes  $3.5 \times 10^{12}/\text{l}$

**Variants of morphological changes of erythrocytes:**

- o Anisocytosis - a change in the size of erythrocytes.
- o Poikilocytosis – a change in the shape of erythrocytes.
- o Microcytosis is a condition in which microcytes predominate in the peripheral blood (erythrocytes  $< 6.5 \mu\text{m}$  in diameter).
- o Microspherocytosis - the presence of round microcytes.
- o Schizocytosis – the presence of very small particles of erythrocytes ( $2-3 \mu\text{m}$ ).
- o Planocytes – erythrocytes with reduced thickness.
- o Annulocytes are erythrocytes that look like a ring (with significant lightening in the center).
- o Ovalocytes – erythrocytes, oval in shape, without light in the center.
- o Stomatocytes – erythrocytes with a linear lumen in the center in the form of a mouth.
- o Xerocytes – erythrocytes with reduced cell volume.
- o 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<sub>12</sub>- deficiency and foliodeficiency anemia.

#### **Note:**

Color indicator (CP) is a value reflecting the hemoglobin content in an erythrocyte, calculated according to the formula:

$$CP = \frac{\text{Hemoglobin (g/l)}}{\text{Erythrocytes (10}^{12}/\text{l)}} \times 10$$

Calculation example: hemoglobin 90 g/l, erythrocytes  $3.5 \times 10^{12}/\text{l}$

### ***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 B<sub>12</sub> 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<sub>12</sub>

### **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 hypotransferrinemia

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 (< $4 \times 10^{12}/l$  in men, < $3.5 \times 10^{12}/l$  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  $\mu\text{mol}/L$  in men, <11.5  $\mu\text{mol}/L$  in women)

2. Decreased serum ferritin concentration (<12  $\mu\text{g}/l$ )

3. The total iron-binding capacity of blood serum is increased (>70  $\mu\text{mol}/l$ )

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<sup>51</sup>Cr erythrocytes.

### **IN<sub>12</sub>-DEFICIENCY ANEMIA.**

**IN<sub>12</sub>- deficiency anemia** is an anemia caused by a violation of DNA synthesis in erythrokaryocytes due to vitamin B deficiency<sub>12</sub> and is characterized by the megaloblastic type of hematopoiesis.

Addison-Birmer anemia (pernicious anemia) is considered a classic variant of B<sub>12</sub>-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<sub>12</sub>- 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<sub>12</sub> 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<sub>12</sub>. (invasion by a helminth - tapeworm is wide, bacterial infection with the "blind loop" syndrome, etc.)

IV. Increased consumption of vitamin B<sub>12</sub> (multiple pregnancy, hemolytic anemia, myeloma disease, etc.)

V. Violation of vitamin B intake<sub>12</sub> with food (strict vegetarianism).

VI. Depletion of vitamin B reserves<sub>12</sub> (cirrhosis).

VII. Violation of transport of vitamin B<sub>12</sub> (absence of transcobalamin II or the appearance of antibodies to it).

### **Pathogenesis B<sub>12</sub>- deficiency anemia.**

Vitamin B<sub>12</sub> 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<sub>12</sub>- 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<sub>12</sub>- 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<sub>12</sub>+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<sub>12</sub>, which is labeled<sup>60</sup>Co. After an hour, vitamin B is administered<sub>12</sub> 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<sub>12</sub> in the intestine

**Instrumental research with V<sub>12</sub>- 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

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 hepatosplenomegaly 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 VIII 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, sarcomatous 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 myeloma should alert the doctor. Suspicion of CML is confirmed by eosinophilia, basophilia, and hyperthrombocytopenia.

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  $50 \times 10^9/l$ . 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  $500 \times 10^9/l$  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 myelemeia, 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 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 anemia, leukemia, hemorrhagic diatheses
- conduct an objective examination of patients with anemia, leukemia, hemorrhagic diatheses
- 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.
8. Classification of leukemias.
9. Classification of coagulation disorders.
10. Define hemophilia.

11. Name the main etiological causes of leukemia.
12. Name the main etiological causes of coagulation disorders.

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 30-year-old patient is in the ward for Werlhoff's disease. Objectively: pale, has petechial hemorrhages on the extensor surfaces of the forearms. Ps – 92/min, BP – 100/60 mm Hg. Art. The lower edge of the spleen is at the level of the navel. In the blood: er. – 2.81012/l, Hb – 90 g/l, Ht – 0.38, blood clot. – 30109 /l. The patient is preparing for a splenectomy. What transfusion medium should be chosen first of all for preoperative preparation?

+A. Platelet mass.

D. Erythrocyte suspension.

B. Canned blood.

E. Washed erythrocytes.

C. Native erythrocyte mass.

2. A 27-year-old patient suffers from hemophilia. Hospitalized with scaly, pale skin. Objectively: Ps - 110/min, BP - 100/60 mm Hg. Art. In the blood: Hb – 80 g/l; Er. – 2.81012/l. The appointment of which of the following therapeutic agents is the first priority in this case?

+A. Cryoprecipitate.

D. Dicynone.

B. Canned blood.

E. Epsilon-aminocaproic acid.

C. Erythrocyte mass.

3. A 42-year-old woman complains of bruises on her legs and prolonged menstruation, general weakness, and noise in her head. Objectively: a large number of spotty hemorrhages on the legs and trunk. Tachypnea, tachycardia, systolic murmur at all points. Blood pressure - 75/50 mm Hg. Art. In the blood: er. – 1.91012/l, Hb – 60 g/l, CP – 0.9, leuk. – 6.5109 /l, tr. – 20109/l, ESR – 12 mm/h. Duration of bleeding according to Duque – 12 minutes. In the bone marrow, there is a large number of young, immature forms of megakaryoblasts without signs of platelet detachment. What is the most likely diagnosis?  
+A. True thrombocytopenic purpura. D. Acute B. Hemophilia A. megakaryoblastic leukemia. C. Willebrandt's disease. E. Hemophilia B

4. An 18-year-old patient developed pain and swelling in the ankle-foot joints and diffuse pain in the abdomen, which worsens after eating, periodic liquid hemorrhagic diarrhea, and a papulo-hemorrhagic rash on the lower extremities, 2 weeks after acute coronary syndrome. In the blood: er. – 3.01012/l, Hb – 95 g/l, ESR – 40 mm/h, platelets are normal. What is the most likely diagnosis?

+A. Hemorrhagic vasculitis.

D. Infectious endocarditis.

B. Werlhof's disease.

E. Rheumatoid arthritis.

C. Systemic lupus erythematosus.

5. A 27-year-old patient complains of nosebleeds, multiple bruises on the skin of the front surface of the trunk and extremities, severe general weakness. In the blood: Hb - 74 g/l, reticulocytes - 16%, erythrocytes - 2.51012/l, platelets - 30109 /l, ESR - 25 mm/h. What is most effective in the treatment of thrombocytopenia?

+A. Splenectomy.

D. Cytostatics.

B. Iron preparations.

E. Vitamins of group B.

C. Blood transfusion

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 16-year-old patient has been sick since the age of 5, when for the first time after an appendectomy massive and prolonged bleeding from a postoperative wound was observed. 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

11. 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. 14**

**Topic: Research methods and semiotics of allergy manifestations. Auxiliary research methods in allergology. The main syndromes in allergology.**

**Goal:** Mastering modern methods of examination of patients with allergic diseases. Know the general principles and methods of examination of patients with allergic diseases. Know the main clinical symptoms of allergic diseases. To be able to conduct a subjective examination of patients with allergic diseases, to highlight the features of complaints and anamnesis of these patients. Be able to make a plan for additional examination of these patients. Formation of deontological skills when communicating with patients with allergic diseases.

### **Basic concepts:**

Types of allergic reactions.

Etiology, pathogenesis of allergic diseases.

Main complaints, anamnesis, objective manifestations of allergic diseases.

Angioneurotic edema (Angioedema), definition, manifestations, features of localization and course.

Urticaria, definition, manifestations, features of localization and course.

Anaphylactic shock, definition, precursors, manifestations, features of the course.

Hay fever, definition, harbingers, manifestations, features of the course.

Care of patients with allergic diseases.

Clinical syndromes in allergology.

Laboratory and instrumental research methods in allergology.

### **.1. Requirements for theoretical readiness of applicants and performance of practical classes:**

- the acquirer must know the mechanisms of formation of various types of allergic reactions
- the applicant must know the method of examining a patient with an allergic reaction,
- the applicant must know the algorithm for collecting complaints and the history of a patient with an allergic reaction,
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### **2.2. Questions to check basic knowledge on the topic of the lesson:**

Patient A., 46 years old, turned to the doctor with complaints that every year at the end of April - May he has burning eyes, runny nose, headache, and fever. The disease worsens when leaving the city. He cannot work at this time. He was treated for acute catarrh of the upper respiratory tract, but without success.

1. What is the disease associated with? Justify your assumptions.
2. Explain the mechanism of these disorders.
3. Your recommendations to the patient.

The patient developed acute glomerulonephritis 2 weeks after purulent angina.

1. What type of allergic reaction is the basis of this disease?
2. Explain the mechanism of development of this disease.

The patient was hospitalized with exacerbation of chronic bronchitis. Treatment with antibiotics is prescribed. On the 4th day, the general condition worsened: the fever persisted, shortness of breath and cough intensified, itchy rashes appeared on the skin, and auscultation showed scattered dry rales. In the blood - eosinophilia (10%). The

deterioration of the patient's condition is due to the development of:

- A) Allergoid reaction
- B) Bronchopneumonia
- C) Bronchial asthma
- D) Asthmatic bronchitis
- E) Drug allergic reaction

A young man, 20 years old, has an injured right testicle. What danger does this pose to the left (healthy) testicle?

- A) Antigen unmasking and antibody damage
- B) Development of the infectious process
- C) Development of atrophy
- D) Development of hypertrophy
- E) Does not threaten anything

The patient, 27 years old, instilled drops containing penicillin into his eyes. After a few minutes, itching of the body, swelling of the lips, eyelids, cough with whistling appeared, blood pressure decreased. What class of immunoglobulins are involved in the development of this allergic reaction?

- A) Iq M and Iq G .
- B) Iq E.
- C) IqA.
- D) IqM.
- E) IqG.

A woman, 43 years old, suffers from pneumonia. After 10 min. after an injection of ampicillin, the patient complained of sharp weakness, burning in the face and hands, cough, shortness of breath, and chest pain. Objectively: cyanosis, swelling of the eyelids, face with red rashes, heart rate - 120/min., blood pressure - 90 mmHg. What is the most likely reason for the sudden deterioration of the patient's condition?

- A) Anaphylactic shock.
- B) Urticaria.
- C) Quincke's edema.
- D) Asthma attack.
- E) Thromboembolism of the pulmonary artery.

The guinea pig was injected with 0.1 ml of horse serum for the purpose of sensitization. What are the external signs of sensitization?

- A) Rash on the skin.
- B) Joint swelling.

- C) There are no external manifestations
- D) Increase in body temperature.
- E) Pain.

The patient turned to the doctor with complaints that every spring, during the flowering period of plants, he has a headache, runny nose, weakness, and an increase in temperature. What type of allergic reaction according to Yell and Coombs is observed in the patient?

- A) Cytotoxic complement-dependent type.
- B) Antibody-dependent cellular cytotoxicity.
- C) Immunocomplex type.
- D) Cell-mediated type.
- E) Anaphylactic type

Patient D., 15 years old, was admitted to the allergy department with a diagnosis of "bronchial asthma". The formation of antibodies of which class determines the development of the main clinical symptoms:

- A) IgA
- B) IgG
- C) IgM
- D) IgD
- E) IgE

During the examination of the patient, who previously suffered from angina in a severe form, edema, increased blood pressure, and decreased diuresis were found. These symptoms are characteristic of acute glomerulonephritis, which is based on damage to the basal membrane of the glomeruli, often by the mechanism:

- A) Allergies of the cytotoxic type
- B) Allergies of the anaphylactic type
- C) Delayed-type hypersensitivity
- D) Immunocomplex allergic reaction
- E) Allergic reaction of the stimulating type

A few minutes after the local anesthesia of the tooth with novocaine by the dentist, the patient suddenly developed weakness, itching of the skin, and sharp spasm-like pains in the abdomen. Objectively: hyperemia of the skin, urticaria, tachycardia, blood pressure drop to 70/40 mmHg. What type of allergic reactions does the described pathology belong to?

- A) Anaphylactic type
- B) Cytotoxic type
- C) Stimulating type

- D) Cell-mediated type
- E) Immunocomplex type

The nurse of the manipulation room with 20 years of experience developed contact dermatitis of the upper extremities. What type of allergic reactions according to Gell et Coombs does this disorder belong to?

- A) Anaphylactic type
- B) Cell-mediated type
- C) Cytotoxic type
- D) Stimulating type
- E) Immunocomplex type

A 45-year-old man complains of burns that appeared on exposed parts of the body after a short stay in the sun (no more than 10-20 minutes), severe headache, nausea, dizziness. Explain the cause of this pathology.

- A) Sunstroke
- B) Heat stroke
- C) Photochemical burn
- D) Overheating
- E) Photoallergy

The girl, 15 years old, suffers from bronchial asthma. In the spring, during the flowering period of the grasses, she developed a severe attack of expiratory shortness of breath. Which of the biologically active substances reliably caused a spasm of the bronchial muscle tissue in this case?

- A) Thromboxane A<sub>2</sub>.
- B) Prostacyclin.
- C) A mixture of leukotrienes C<sub>4</sub>D<sub>4</sub>Well<sub>4</sub>
- D) Bradykinin.
- E) Serotonin.

After 2 hours after a transfusion of allogeneic plasma, a patient with burns in the stage of toxemia developed pain in the joints, lower back, hemorrhagic rash on the skin, and the temperature rose. Which of the allergic reactions occurs in this case?

- A) Urticaria
- B) Quincke's edema
- C) Anaphylaxis
- D) Serum sickness
- E) Autoimmune vasculitis

4. Summary:

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## **Practical lesson No. 15**

**Subject:** Principles of patient care in the internal medicine clinic and providing first medical and pre-medical aid

**Goal:** Get acquainted with the essence of the concept of "patient care". Acquire theoretical knowledge about the importance of patient care of a therapeutic profile, scope and features of care methods in the therapeutic department; responsibilities of middle and junior medical personnel in carrying out care measures. To master the skills of performing the simplest care measures: hygiene of the ward, beds, change of underwear and bed linen, personal hygiene. To master the simplest skills of compliance with hospital, medical-security and sanitary-hygienic regimes. Master the technique and technique of performing the simplest resuscitation measures - mouth-to-mouth and mouth-to-nose artificial respiration, external (indirect) heart massage. Learn the concepts of clinical and biological death. To learn basic concepts of medical ethics, deontology and aesthetics, basic deontological principles of patient care in reception and therapeutic departments.

### **Basic concepts:**

An idea of the rights and responsibilities of junior medical staff of reception and therapeutic departments. Rules of ethics and deontology, medical and protective regime in the work of junior medical staff. Techniques for preparing disinfectant solutions, means of sanitizing patients, premises, furniture and medical accessories. Techniques of artificial respiration "mouth to mouth", "mouth to nose", indirect heart massage, signs effectiveness of resuscitation.

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

1. Get acquainted with the essence of the concept of "patient care".
2. To acquire theoretical knowledge about the importance of caring for patients of a therapeutic profile, the scope and features of care methods in the therapeutic department; responsibilities of middle and junior medical personnel in carrying out care measures.
3. Master the skills of performing the simplest care measures: hygiene of the ward, beds, changing underwear and bed linen, personal hygiene.
4. To master the simplest skills of compliance with hospital, medical and protective and sanitary and hygienic regimes.
5. To master the methodology and technique of execution the simplest resuscitation measures - mouth-to-mouth and mouth-to-nose artificial respiration, external (indirect) heart massage.
6. Learn the concepts of clinical and biological death.
7. To learn basic concepts of medical ethics, deontology and aesthetics, basic deontological principles of patient care in reception and therapeutic departments.

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

### *Question.*

1. What should be understood by the terms "hospital", "clinic"? What is their difference?
2. What should be understood by the medical and protective regime and what measures are taken to comply with it?
3. What structural units does the therapeutic department consist of?
4. What is the department's sanitary and hygienic regime and what are the requirements for its observance?
5. What is the nursing position of the department and what are its functions?
6. What are medical ethics and deontology?
7. What elements are included in measures to observe the patient's personal hygiene?
8. What is the purpose of the reception department?
9. What is the sequence in the work of the reception department?
10. What documentation does the nurse of the reception department fill out?
11. How should the hair be treated when pediculosis is detected?
12. Describe the medical documentation: title page of medical history, patient registration logs.
13. Anthropometric measurements.
14. Transportation, adequate to the patient's state of health.
15. Structure and function of a therapeutic hospital.
16. Preparation and use of disinfectant solutions
17. Features of cleaning the premises of the therapeutic department.
18. Care of hair, skin, nails, eyes, mouth, ears, nose, bed preparation, change of linen, prevention of bedsores.
19. The role and responsibilities of junior medical personnel in treatment and prevention activities.
20. What is the concept of medical ethics and morals of the work of a therapeutic hospital?
21. What are the signs of clinical and biological death?
22. Concept of resuscitation.



23. Methodology and technique of artificial respiration using the mouth-to-mouth and mouth-to-nose methods
24. Methodology and technique of indirect heart massage.
25. Signs of the effectiveness of resuscitation measures.
26. Complications during resuscitation.

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

#### *Test and tasks.*

- What moral and ethical qualities characterize a medical worker?

- A. a sense of duty and vocation to one's profession
- B. high morality
- S. benevolence and compassion
- D. self-demanding and the ability to keep medical secrets

That is, All are listed.

- What problems of the patient can be classified as physiological?

- A. sleep disturbance
- B. breathing disorder
- C. lack of attachments, friends
- D. lack of movement

- You need to prepare a disinfectant solution. Chloramine belongs to which group of chemicals?

- A. Group of oxidizers
- V. Haloids
- S. Lugi
- D. Acids
- E. Spirits

- In which treatment and prevention institution, not only treatment, but also teaching and research work is carried out?

1. In the clinic
2. B dispensary
3. In the clinic
4. In the hospital
5. In the sanatorium

- When distributing medicines, one of the bottles was found to have no pharmacy label; how should a nurse behave in such a situation?

- A. Compare with similar ones and continue to give to patients

- B. Remove from application
- C. Consult a doctor
- D. Consult with the nurse
- E. Consult with the patient

- The patient was admitted to the reception department in a state of severe cardiovascular insufficiency. What amount of sanitary treatment is shown to the patient?

- A. Taking a bath
- B. Partial sanitation
- C. Wiping the face and exposed parts of the body with a towel moistened with an aqueous solution of table vinegar
- D. Send to the intensive care unit urgently without sanitation
- E. Do not send to the intensive care unit without sanitation.

- The patient is brought to the ward on a stretcher in such a way that the head end of the stretcher is attached to the foot end of the bed. How to properly place the stretchers?

- A. That their head end was attached to the foot end of the bed
- B. That their foot end is placed against the head end of the bed
- C. That their foot end is placed against the foot end of the bed
- D. That their foot end is placed in the middle of the bed
- E. That their head end should be placed in the middle of the bed

- Diseases caused by negative relationships between medical staff and patients are called

- A. social
- B. iatrogenic
- C. somatogenic
- D. professional
- E. psychogenic

- Resuscitation measures are an integral part of medical care. Who must be able to conduct it?

- A. Only doctors and nurses of intensive care units.
- V. All specialists with medical education.
- C. The entire adult population.
- D. acquirers of medical facilities.
- E. Ambulance team.

- You count heart rate and pulse per minute. What should they be normally?

- A. 40-60
- V. 50-80
- P. 60-90
- D. 50-120
- E. 70-100

- In front of your eyes, a person fell to the floor, there is no pulse and breathing. What is the maximum duration of clinical death?

- A. 10-15 minutes.
- B. 5-7 minutes.
- S. 2-3 minutes.
- D. 1-2 minutes.
- E. 1 year.

- A 37-year-old man was dragged to the seashore: the skin is extremely pale, there are no movements, no breathing, there is no pulsation in the vessels of the neck. Specify the rhythm in which artificial respiration is performed without the use of devices:

- A. 3 lung inflations per minute.
- B. 6 lung inflations per minute.
- C. 12 lung inflations per minute.
- D. 24 lung inflations per minute.
- E. 48 lung inflations per minute.

- The man was admitted to the intensive care unit in serious condition, unconscious after electrocution. What are the main signs of cardiac arrest?

- A. Wide pupils, no pulse on the carotid artery, no independent breathing.
- B. Wide pupils, lack of independent breathing.
- C. Absence of a pulse on the carotid artery, narrow pupils.
- D. Narrow pupils.
- E. Wide pupils, no pulse on the carotid artery, no independent breathing, narrow pupils, convulsions.

- A 40-year-old woman was taken to the intensive care unit in an unconscious state after electrocution. Indicate the location of the resuscitator's folded hands during indirect heart massage:

- A. The left half of the chest.
- B. Handle of sternum.
- C. The lower third of the sternum.
- D. The xiphoid process.
- E. The middle part of the sternum.

- A 35-year-old woman was found unconscious on the beach. The urgent phase of cardiopulmonary resuscitation consists of: 1. ensuring patency of the respiratory tract; 2. conducting artificial lung ventilation, 3. restoring blood circulation; 4. gastric lavage.

- A. Ensuring the patency of the respiratory tract, carrying out artificial ventilation of the lungs, restoring blood circulation.
- B. Ensuring patency of the respiratory tract, restoration of blood circulation.
- C. Performing artificial lung ventilation, gastric lavage.
- D. Gastric lavage.
- E. Ensuring the patency of the respiratory tract, carrying out artificial ventilation of the lungs, restoring blood circulation, washing the stomach.

- A 45-year-old patient was brought to the reception department in an unconscious state after electrocution. Indicate the rhythm in which indirect heart massage is performed in adults:

- A. 10-20 clicks per minute.
- B. 30-50 beats per minute.
- C. 60-80 beats per minute.
- D. 90-120 beats per minute.
- E. More than 120 beats per minute.

- In front of the door of the clinic, you saw a person on the asphalt. Select the manifestations of clinical death:

- A. Loss of consciousness.
- B. Absence of pulse.
- C. Shortness of breath or shortness of breath.
- D. Absence of blood pressure.
- E. Any of the above symptoms.

- A 16-year-old boy was dragged to the sea shore without signs of life: the skin is extremely pale, there are no movements, there is no breathing, no pulsation of the vessels of the neck is observed. Friends without special medical training provide assistance: one rubs the skin, another presses the fist of his right hand in the middle of the sternum, the third intensively performs "mouth to mouth" breathing. At the same time, with each breath, the belly increases in size, and the chest does not rise. What is the reason for this situation?

- A. Obstruction of the upper respiratory tract.
- B. Ascites.
- C. Weakness of the cardiac sphincter of the stomach.
- D. Carrying out mechanical ventilation with positive inspiratory pressure.
- E. Weakness of the muscles of the anterior abdominal wall.

4. Summary:

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