МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ

УЧРЕЖДЕНИЕ ОБРАЗОВАНИЯ
«ГОМЕЛЬСКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ»

Кафедра пропедевтики внутренних болезней

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ТЕЗИСЫ ЛЕКЦИЙ ПО ПРОПЕДЕВТИКЕ ВНУТРЕННИХ БОЛЕЗНЕЙ

Учебно-методическое пособие
для студентов 3 курса факультета по подготовке специалистов
для зарубежных стран, обучающихся по специальности «Лечебное дело»

THESES OF LECTURES ON PROPEDEUTICS OF INTERNAL DISEASES

Teaching workbook
for 3rd year students of the
Faculty of General Medicine for Overseas Students
studying in the specialy of «General Medicine»

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Учебно-методическое пособие содержит основные положения лекций по курсу пропедевтики внутренних болезней, изложенные в краткой тезисной форме.

Предназначено для студентов 3 курса факультета по подготовке специалистов для зарубежных стран.

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LECTURE 1
INTRODUCTION IN CLINIC OF INTERNAL DISEASES. INQUIRY AS A METHOD OF CLINICAL RESEARCH INQUIRY OF PATIENTS WITH DISEASES OF THE RESPIRATORY SYSTEM

The propaedeutic of internal diseases is the 1st stage of studying of clinical medicine by students. Clinical disciplines are studied on the basis of clinical medical establishments (clinics).

The clinic (from Greek *«κlinike»* — «doctoring») is a medical establishment or sectioning of medical establishment (for example, a branch of hospital, or a polyclinic) where besides usual medical and preventive work medical preparation is conducted, i. e. theoretical and practical training of students and doctors, and also research work.

Definitions of some concepts which will be used in the lecture:

Health is a condition of complete physical, mental and social well-being, and not just absence of signs of deseases and physical defects.

Disease is impairment of frames and functions of organism because of its insufficient resistibility to constantly changing conditions of external and internal medium.

Etiology (from Greek «*etios*» — «the reason», «*logos*» — «a science») is a science about the reasons and conditions of a course of a disease.

Pathogeny (from Greek «*pathos*» — «suffering», illness; «*genesis*» — «the parentage», «development») is a science about mechanisms of a course of a disease.

Sanogenesis is a doctrine about mechanisms of protection of the organism from action of the damaging agents directed to restoration of function of damaged frames.

Definition of the propedeutics of internal diseases

Propedeutics (from Greek «propaideuo» — «preliminary I train») is introduction in to any science, the preliminary, introduction course regularly stated in the concise and elementary form.

Propedeutics of internal diseases is introduction or introduction course in to internal diseases.

Internal diseases is one of the largest branches of theoretical and applied medicine, the scientific discipline, having the purpose of studying of recognition of diseases of internal organs in their various clinical forms, the reasons of a parentage of these diseases, their prophylaxis and treatment.

Tasks of internal diseases as a scientific discipline

1. Recognition of diseases of internal organs (diagnostics).
2. Studying an etiology and a pathogeny of diseases of internal organs.
3. Development of questions of prophylaxis and treatment of diseases of internal organs.

The primary goals of the chair of propedeutics of internal diseases are training students to:

1. A technique of clinical examination of patients.
2. Symptomatologies of diseases.
3. The bases of laboratory and tool diagnostic researches in diseases of internal organs.
4. The basic nosological units (diseases) and syndromes in terms of training the students to apply the data received on the examination of the patient to diagnostics of specific diseases.

Frame (sections) of the propedeutics of internal diseases

1. Diagnostics.
2. A private pathology.

Diagnostics (from Greek «*diagnosticos*» — «*capable to distinguish*») is a section of medicine, a separate science studying methods of research for recognition of disease and condition of the patient with the purpose of assignment of the necessary treatment and preventive actions.

Frame of diagnostics as a scientific discipline

1. The medical diagnostic technics (actually diagnostics) is the section of diagnostics studying methods of reception of the information on the patient. It includes studying methods of inquiry and objective research of the patient.
2. Semeiology or symptomatology (from Greek «*semeion*» — «an attribute») is a section of diagnostics studying signs, revealed on research of the patient, the doctrine about the diagnostic importance and a parentage of separate signs and syndroms of diseases.
3. The methodology of the diagnosis (medical logic) is a section of diagnostics including studying of features of thinking of the doctor on the establishment of the diagnosis and disease on the basis of the data, received on patient's examination.

Medical diagnostic technics

Methods of research of the patient

Divided into 2 groups

1. Basic.
2. Auxiliary (additional).

The basic methods of the patient's examination

1. Inquiry of the patient (from Latin — «i*nterrogation*») or anamnesis (from Greek «*anamnesis*» — «memoirs»).
2. Survey (from Latin — «examination»).
3. Palpation (from Latin — «palpatio»).

4. Percussion (from Latin — «percussion»).

5. Auscultation (from Latin — «auscultation»).

Auxiliary methods of research:

1. Measurings (body temperatures, body height, weight, a circle of the chest, daily quantity of urine, etc.)

2. Laboratory researches of liquids of organism and its discharges (blood, spinal liquid, gastric juice, bile, urine, exudate in a body cavity, sputum, pus, etc.).

3. Morphological researches of bodies, tissues, liquids of organism and its discharges.

4. Tool researches (electrocardiogram, radiological, researches of function of breath, isotope researches, ultrasonic, endoscopy methods, etc.).

Research methods divide on a way of reception of the information on the patient:

1. Subjective.

2. Objective (physical).

The subjective method of research considers the patient as the subject who recollects and tells about the sensations and events which accompanied it from the onset of the disease. It is the story of the patient about the life and desease, its subjective representations about desease which he suffers.

The doctor obtains subjective data about sick by inquiry method.

Objective (physical) research methods. The doctor examines the patient, guiding by the sense organs and ability to manipulate by a number of practical actions. The patient is object of examination.

Methods of objective research divide on:

1) basic — survey, palpation, percussion and auscultation;

2) auxiliary — various measurements, laboratory, morphological and tool methods.

Semiotics

Symptom (from Greek «*symptomos*» — «the sign», «coincidence») is a sign of any disease, statistically significant deviation of any indicator from norm or occurrence of qualitatively new, not a characteristic phenomenon of a healthy organism.

Types of symptoms on the importance

1. Diagnostic symptoms are peculiar only to one disease.

2. Specific symptoms are characteristic of a group of diseases of one organ or one system.

3. Nonspecific symptoms are characteristic of many diseases of various organs and systems of organs.

4. Symptoms, not characteristic of the given disease are not revealed in the given disease.

Types of symptoms on a method of their revealing

1. Subjective symptoms are symptoms which are revealed on the basis of the description of the patient of the sensations wich arise in desease process. They are revealed by inquiring of patients, i. e. a subjective method of research.

2. Objective symptoms. These are symptoms which are revealed by methods of objective research of the patient: survey, palpation, percussion, auscultation and in the laboratory — tool ways.

Types of symptoms on time of their occurrence

1. Early (initial) — arise in early, initial stages of development of desease.
2. Late — arise in a heat of disease or in recovery.

Types of symptoms on prognostic value

1. Favorable symptoms — indicate in easy or usual current of disease, and also on its permission.

2. Adverse (menacing) — testify the heavy form of desease, about probability of unfavorable outcome.

Syndrome (from Greek «*syn*» — «together», «*dromos*» — «to move», «run» — «to run together») are groups of symptoms which have the general pathogenesis.

A complex of symptoms is a group of symptoms or syndromes which are characteristic of disease, but are not united by the general origin.

Diagnosis methodology

The diagnosis (from Greek «*diagnosis*» — «recognition») is a short medical conclusion about essence of disease and a condition of the patient, expressed in terms of a modern medical science.

Diagnosis structure

1. The basic disease is a disease which has forced the patient to address for medical aid, has served as an occasion for hospitalisation or disease which threatens a life of the patient, is able to lead to death or physical inabilities of the patient independently or through complications.

2. Complications of the basic disease are diseases of other character and aetiology, but connected with it by development mechanisms.

3. Accompanying diseases — are diseases which are found out in the patient simultaneously with the main one, but they are not interconnected with it.

Types of diagnoses

I. On character and the maintenance

1. Anatomic.
2. Pathoanatomical.

3. Pathophysiological (functional).

4. Pathogenetic.

5. Nozological.

6. Etiological.

These types of diagnoses are components which are used at the formulation of the diagnosis of disease.

II. On a way of construction and substantiation.

1. The direct diagnosis (or the diagnosis by analogy — diagnosis morbi. The diagnosis is established by method of comparison of clinic of disease which is available for the patient, with a typical clinical picture which is characteristic of the given disease.

2. The differential diagnosis (diagnosis differentialis). It is the diagnosis by means of comparison of several similar diseases. Here it is necessary to exclude less probable diseases, leaving more probable ones. It is the diagnosis by means of exception (diagnosis per exclusionem).

3. The diagnosis by means of observation (diagnosis per observatione). The diagnosis is established during observation of the patient on occurrence of new symptoms of disease or reception of additional results of research.

4. The diagnosis on medical effect (diagnosis ex juvantibus). The diagnosis is established on the basis of favorable result of treatment with the preparations specifically working at the given disease.

5. The diagnosis by result of harmful action (diagnosis ex nonentibus). This application of medical products or other influences provoking occurrence of symptoms of the disease (allergic tests, physical exertion in stenocardia).

6. The diagnosis on operation (diagnosis sub operatione).

III. By the time of disease revealing.

1. Preclinical diagnosis. It is an attempt to distinguish and determine the condition of the organism, boundary between the norm and pathology.

2. Early diagnosis. Disease is distinguished in its very onset.

3. Late diagnosis. It is established at the height of development of disease or on a section table.

4. Retrospective diagnosis. It is established on the basis of the data which have been revealed earlier, but for some reason have not been taken into account, on the basis of the analysis of the medical documentation, etc.

5. Postmortal diagnosis. It is established after death of the patient and is recorded in postmortal epicrisis.

IV. By the degree of reliability.

1. The rough diagnosis is a working hypothesis arising in the initial stage of examination of the patient by the doctor; it determines the direction of diagnostic search.

2. The preliminary diagnosis is established by results of inquiry and physical examination of the patient. This diagnosis is the basis for administration of prelimi-

nary treatment and determination of the plan of laboratory and tool examination of the patient.

3. The clinical diagnosis is established after laboratory — tool examination of the patient and observation of the patient during the preliminary treatment.

4. The final diagnosis is established by results of observation of the patient during the treatment.

5. The hypothetical diagnosis (in doubt).

6. The incomplete or uncertain diagnosis.

7. The erroneous diagnosis.

Sections of a case history of the patient (in-patient or out-patient)

1. Inquiry of the patient (anamnesis, interrogatio) (passport part, complaints, anamnesis of disease, anamnesis of his life).

2. Objective research (status praesens obectivus).

3. A diary of a case history (cursus morbi).

4. Treatment (therapia).

5. The data of laboratory and tool researches, consultations of experts.

6. Epicrisis.

Value of a case history

1. All the data on the course of disease, risk factors, clinical manifestations of disease, its treatment, current, rehabilitational actions are fixed in the case history.

2. Pathomorphism is studied on the basis of a case changes in clinic and current of the disease during the time.

3. Efficiency of treatment of the patient is estimated on the basis of a case history.

4. A case history is the important legal document. It is used in case of occurrence of disputed situations between the doctor and the patient.

5. A case history ascertains the fact of the presence of the patient in hospital or his references to the doctor.

6. A case history is a financial document (the definition of the correctness of the charge of medicines and other medical agents, validity of distribution of sheets of disability, etc.).

Anamnesis (inquiry) is a set of the data received on medical examination by means of inquiry of the very patient and surveyed and (or) of person knowing him.

The purposes of studying of anamnesis

1. Reception of the information necessary for diagnostics.

2. An estimation of probable gravity of the disease.

3. An establishment of other sources of the information on the patient (the relatives acquaintance, other doctors, etc.).

4. An establishment of confidential relations with the patient.

5. An estimation of the person of the patient and his attitude to his disease.

The circuit of inquiry of the patient

1. The general data on the patient (nameplate data).
2. Complaints of the patient (molestia).
3. A history of the present disease — the anamnesis of disease (anamnesis morbi).
4. A history of life of the patient – the anamnesis of life (anamnesis vitae).

The general data on the patient (nameplate data):

— Surname, name, a patronymic.
— Age.
— Sex.
— Formation.
— Occupation.
— Place of employment.
— Residence.

Complaints of the patient (molestia)

Classification of complaints by their general character

1. Group of the certain, accurate complaints (cough, vomiting, dyspnea, pains, hypostases, rise in temperature) — are revealed at the expressed changes of organs.

2. Group of uncertain, not clear complaints (aches a bit, «I feel heart», «I'm not felling good», «I'm feeling sick», etc.) — are characteristic of insignificant impairments of functions of organs in chronic diseases in remission.

3. Group of numerous and various, extremely detailed and very uncertain complaints are characteristic of the patients, suffering neurosises.

Classification of complaints by the maintenance

1. The complaints characterising changes of size, form, position and appearance of separate parts of the body (hypostases, tumours, deformation of joints and chest etc.). They appear in morphological changes of organs.

2. Complaints of frustration of those or other functions of the organism (vomiting, dyspnea, palpitation, cough, diarrhoeia, etc.). They characterise functional changes.

3. Complaints of abnormal sensations (mental experiences) — pain, weakness, bad state of health, itching, nausea, heartburn, etc.

Classification of complaints by the diagnostic importance

1. The main complaints are those sensations and the phenomena which disturb the patient most of all and are expressed definitely and sharply enough.

2. Additional complaints are all the other complaints.

History of the present disease (the disease anamnesis)

The disease anamnesis is a history of development of disease, feature of its current from the moment of occurrence to the given reference for medical aid.

Value of the anamnesis of disease

1. Revealing of initial manifestation of disease which can differ from those revealed at the moment of examination of the patient.
2. The number of diseases has only for them specific (gout, myocardial infarction, etc.).
3. For many diseases certain sequence of occurrence of symptoms and change of the character of disease processes (rhinitis, conjunctivitis — allergic bronchitis — asthma) are characteristic.
4. The number of diseases has a periodicity of aggravations (pollinosis).
5. The estimation of efficiency of treatment and forecasting of the current of disease.

Sections of the anamnesis of disease

1. Time of occurrence of diseases.
2. A state of health prior to the onset of disease.
3. Prospective causes of disease.
4. 1st signs of disease.
5. Dynamics of occurrence and change of symptoms, occurrences of complications.
6. Frequency and the reasons of aggravations, duration of remissions.
7. The 1st reference for medical aid (when and why).
8. Earlier diagnostic researches and their results.
9. Character and efficiency of earlier treatment.
10. Character of treatment of the patient in home conditions.
11. Dispensary supervision over patients.
12. Terms and the reasons of latest aggravation of disease.
13. Motives of hospitalisation in clinic.
14. The insurance anamnesis: sick-list presence (how long); physical inability group.

History of the patient's life (the life anamnesis)

The life anamnesis is a medical biography which allows to carry out the analysis of physical, mental and social development of the patient in each age period of life, conditions and way of life, for the purpose of revealing of possible risk factors of disease occurrence and progressing.

Sections of the anamnesis of life

1. Short biographic data on children’s and youthful years of life.
2. The labour and household anamnesis.

3. The family and hereditary anamnesis.
4. The saffered diseases.
5. Bad habits.
6. The allergic and medicinal anamnesis.
7. The gynecologic anamnesis.

Complaints of patients with diseases of the respiratory system

Local (specific) complaints

1. Impairments of nasal breath, running nose.
2. Voice changes.
3. Nasal bleedings.
4. Pain and other unpleasant sensations in the throat.
5. Cough.
6. Sputum.
7. Hemoptysis
8. Breathlessness.
9. Dyspnea, asthma.
10. Pains in the thorax.

The general (nonspecific) complaints

1. Fever.
2. Weakness.
3. Drop of working capacity.
4. Sweating.
5. Drop of appetite, etc.

Impairment of respiration through the nose, rhinitis, deterioration of olfaction

They can be caused by local pathological processes:
1. Inflammation (rhinitis, sinusitises).
2. Polyps.
3. Vasculomotor edema of a mucosa.
4. Curvature of the nasal septum, etc.
They can be as manifestations of general disease — allergies.

Sensation of pain, dryness and ticking in the throat, changes of voice

Usually it is attribute of chronic inflammatory processes of the pharynx and the larynx, edema of the larynx, tumours of the larynx, damage of a recurrent nerve, etc.

Cough is a complex reflex act which is caused by presence in airways of foreign bodies, or the bodies got from outside (pieces of food, dust, etc.), or the

bodies formed as a result of inflammatory or other pathological processes (sputum, pus, blood, mucous, etc.).

A reflex arch of reflex cough: from branches of wandering and top laryngeal nerves in mucosas of the larynx, the trachea and the bronchi through the center of cough in the medulla oblongata to motor nerves – lower laryngeal, diaphragmatic and spinal.

Variants of cough on the rhythm

1. Cough as separate jerks (tussiculation). It is observed in laryngitises, tracheobronchitises, in smokers, in the initial stages of pulmonary tuberculosis, sometimes in neurosises, hysteria.

2. Cough as attacks. It is observed in the hit of a foreign body into the airways, whooping-cough, bronchial asthma, pulmonary caverns, augmentation of lymphonoduses of a mediastinum and a prelum of the trachea and large bronchi by them, an endobronchial tumour.

Variants of cough on the timbre

1. Short and cautious cough which is accompanied by a morbid grimace. It is observed in dry pleurites, damages of the pleura of any etiology, fractures of ribs, damage or an inflammation of muscles of the chestl.

2. Barking cough. Appears in swelling of false vocal chords (the voice remains not changed), diseases of the larynx if true vocal chords are not affected, a prelum of the trachea (struma, tumour), hysterias.

3. Hoarse cough. It is attribute of inflammation of true vocal chords. In this case patients' voice is rhonchial.

4. Silent cough. It is characteristic of ulcerations or destructions of true vocal chords (tuberculosis, lues), atonies of vocal chords (the affection of laryngeal and recurrent nerves and muscles of the larynx), the exhausted and sharply weakened patients.

5. Cough with a special indistinct shade can occur in case the patient has a big cavity in the lung or if he coughs with the closed mouth (tuberculosis).

Variants of cough on the character

1. Nonproductive is not accompanied by expectoration. It occers in acute bronchitises (in the initial stage), disease of the larynx, diseases of the pleura, augmentation of lymphonoduses of the mediastinum and a prelum of the trachea and bronchi by them, lung cancer, hysterias.

2. Productive cough is accompanied by discharge of sputum. It is characteristic of bronchites, chronic obstructive pulmonary disease, pneumonias, a bronchoectatic disease, abscess of the lungs, pulmonary tuberculosis with disintegration.

Variants of cough on the time of it's occurrence

1. Morning cough. It is characteristic of chronic inflammatory diseases of the top airways (the nose, the nasopharynx, the fauces, the larynx), smokers and

topers, chronic obstructive pulmonary disease, bronchiectasias, abscesses, caverns of the lungs. It is caused by accumulation of sputum in the airways or cavities within the night.

2. Evening cough — is characteristic of pneumonias and acute bronchitises.

3. Night cough. It is observed in bronchial asthma, augmentation of lymphonoduses of the mediastinum and a prelum of the trachea by them, a pulmonary tuberculosis, etc.

Variants of cough from the point of view of the conditions in which cough occers, or the phenomena by which it is accompanied.

1. The cough arising in connection with the acceptance of a certain position of a body by the patient. It is observed in the presence of cavitary formations in the lungs, communicating with a bronchus.

2. The cough arising in connection with reception of food, especially if the particles of the just accepted food are allocated with sputum. It is characteristic of a fistula between the esophagus and the bronchus or the trachea (a cancer of the esophagus, tuberculosis of lymphonoduses of the mediastinum).

3. The cough which is accompanied by the allocation of a great amount of sputum (a full mouth). It is observed in the emptying of cavities of the lungs (bronchiectasias, an abscess).

4. The cough which is accompanied by vomiting. It is observed in whooping-cough, in some forms of pulmonary tuberculosis and pharyngitises.

5. Cough is a manifestation of tussive syncope (cough-unconscious syndrome) if it is accompanied by loss of consciousness and spasms.

6. Cough is characteristic of whooping cough if it is accompanied by respiratory standstill (reprises).

Cough variants by duration of existence

1. Constant cough. It is characteristic of: chronic obstructive pulmonary disease, chronic diseases of larynx and of pharynx, pulmonary tuberculosis, heart failure.

2. Periodic cough. It is characteristic of: sensitive persons on inhalation of cold air (hyperreactance of bronchial tubes), smokers, chronic diseases of lungs and the bronchial tubes proceeding with alternation of aggravations and remissions, bronchial asthmas, whooping cough.

3. An unitary attack of strong cough. It is characteristic of the presence of foreign bodies in respiratory ways.

Sputum

Sputum is a discharge from the respiratory ways, thrown out on at coughing.

Properties of sputum which are necessary to estimate:

1) quantity (volume);
2) consistence;

3) the character of sputum;
4) the character of discharge of sputum;
5) colour;
6) smell;
7) impurity.

Types of sputum by its character

1. Mucous sputum.
2. Serous sputum.
3. Purulent sputum.
4. Mucopurulent sputum.
5. Serious-mucopurulent sputum.
6. Putrefactive sputum.
7. Bloody sputum.

Hemoptysis (haemoptoe) — discharge of blood with sputum, thus the quantity of blood does not exceed 50 ml a day.

The reasons of hemoptysis:

1) pulmonary tuberculosis;
2) a breaking up tumour of bronchial tubes and lungs;
3) bronchiectasis;
4) hemorrhagic bronchitis;
5) lung abscess;
6) lung infarction;
7) stagnation of blood in pulmonary circulation;
8) croupous pneumonia.

Dyspnea

Breathlessness (dyspnoe) (from Greek «*dys*» — «upset», «bad» and «*pnein*» — «to breathe» — «the complicated», «broken breath») is the patient's sensation of shortage of air, accompanied by change of frequency, depth, a rhythm of breath and duration of phases of inspiration and exhalation.

Types of breathlessness

I. The subjective breathlessness is only shown by sensation of difficulty of breath without objective signs of respiratory dysfunction (neurosises, a hysteria).

II. The objective breathlessness is characterised not only by sensation of shortage of air, but also by objective signs — changes of frequency, rhythm, depth of breath, change of duration of inhalation or exhalation, participation in the act of breath of auxiliary respiratory muscles, cyanosis and other signs.

III. The physiological or working breathlessness occurs in healthy people on considerable physical exertion and disappears in 1–3 minutes of rest.

IV. The pathological breathlessness is caused by presence of any pathological process in the organism.

Pathogenetic variants of a pathological breathlessness

1. The pulmonary breathlessness is connected with impairments of function of the respiratory system.

2. The cardiac dyspnea is caused by blood circulation impairment (especially in pulmonary circulation).

3. Hematogenic breathlessness is connected with changes of a chemical composition of blood (carbonic oxide, methemoglobin former connections, e. g. nitrates), with anaemia.

4. Neurogenic or central breathlessness is caused by functional affection of the central nervous systems (neurosis) or its organic affection.

5. Thoraco-phrenic breathlessness caused by impairment of mobility of the chest and a diaphragm (kyphoscoliosis, Bekhterev's desease, myositis, myopathy).

The reasons of a pulmonary breathlessness

1. The narrowing of top respiratory ways (foreing bodies, a throat spasm, an inflammation, a tumour, pressure from the outside).

2. The narrowing of bronchial tubes (bronchitis, bronchial asthma, foreing bodies, pressure from the outside).

3. The affection of the pulmonary tissue (pneumonia, tuberculosis, emphysema, etc.)

4. Lungs atelectasis (blood, air, etc. in a pleural cavity).

5. Difficulty of falling of lungs (pleural solderings).

6. Difficulty of functioning of lungs (hems in lungs, fibrosis).

Thoraco-phrenic breathlessness occurs

1. In impairment of mobility of the thorax (ossification of ribs, rib fractures, pains in the chest).

2. In impairment of function of the respiratory musculation (botulism, dermatomyositis, myasthenia, etc.)

Inspiratory breathlessness

The inspiratory breathlessness consists of sensation of an obstacle to respiration or difficulties of inspiration.

The reasons of the inspiratory breathlessness

1) Difficulty of entering of air into the larynx, the trachea and large bronchi:

— An edema of the larynx and vocal chords.

— A prelum of the trachea or the larynx by the tumour, a lymphonoduses, or a struma.

— Foreign bodies of the larynx, a trachea and large bronchuses.

2) Impairment of the respiratory function of the chest:

— Dry pleuritis, fractures of ribs (owing to the pain).

— Paralysis of the respiratory musculation.
— A prelum of the lungs by air, liquid or a tumour.
— Subjective sensation of shortage of inspiration — an attribute of neurocirculatory asthenia.

Expiratory breathlessness

The expiratory breathlessness is characterized by the sensation of difficulty of expiration.

The causes of the expiratory breathlessness

— Bronchial asthma.
— Chronic obstructive pulmonary disease.
— Obstructive emphysema of the lungs.
— Expiratory collapse of bronchi and trachea.

The mixed breathlessness

The mixed breathlessness is characterized by difficulty of inspiratory and expiratory phases. It is the most often variant of breathlessness.

Pains in the chest

Pains in the chest can be caused by the following reasons.
1. Pathological process in the chest.
2. Pathological process in the respiratory organs.
3. Pathological process in the heart and the aorta.
4. Irradiation of pains from the spine or the abdominal cavity.

The reasons of the pains caused by a pathology of the chest:

1. A lesion of intercostal nerves (neuralgia). These pains are constant, increase slightly on inspiration, on inclination of the trunk to the sick side. They localize strictly on the intercostal. Thus three especially morbid points are excreted: at the spine; at the level of the axillary fossa; at the breast bone — in places of the exit of branches of a nerve into the skin.

2. A lesion of intercostal muscles (myosites). These pains are whining, increase on inspiration and on inclination of the trunk aside, on palpation of the affected muscles. Pain points are absent.

3. Diseases of ribs and breast bones (periostites, osteomyelites, etc.). This pain localize in bones and does not depend on the act of respiration and movements of the chest. On examination and palpation it is possible to reveal the deformation of ribs and their tenderness in the place of localization of a pathological process.

4. Fractures of ribs. In this case the pain localizes in the area of the damaged rib, it increases on respiration and movements. On palpation it is possible

to reveal a crepitation of fragments of ribs, sharp tenderness, puffiness of surrounding tissues.

The pains caused by a pathology of the pleura:

The reasons: pleurites, metastases of the tumour in the pleura, traumas, pneumothorax.

Localization — in the lower lateral zones of the chest (pain in the side).

They occur or increase at the height of inspiration, on cough, laughter, conversation. Character: sharp, prickly, it is accompanied by nonproductive morbid cough, increases on inclination to the healthy side, weakens on inclination to the sick side and on bracing of a sick half of chest.

The pain arising at the moment of formation of a pheumothorax is sudden, intensive, localizes on the limited area of the chest (corresponds to the place of the rupture of the pleura), accompanied by inspiratory breathlessness, cyanosis, drop of blood pressure. At attack of intensive cough or a physical effort frequently precede this pain appearance.

LECTURE 2
EXAMINATION AND PALPATION AS METHODS OF CLINICAL RESEARCH OF PATIENTS

Examination as a method of clinical research of patients

Examination (inspectio) — the clinical method of research based on reception of the information on the patient by visual perception of the structure and functioning of his organism.

Rules of examination

1. The position of the doctor. The doctor should face the person and be to the right of him. Light should fall from behind the doctor's back.

2. Illumination. Examination is the best way for carrying out at daylight or at white strongly disseminated artificial light which is given with lamps of day time illumination. Illumination should be direct and lateral.

3. Technics of examination. The patient fully of partially undressed (depending on conditions and the attitude of the patient to the examination), should be examined in direct and lateral illumination. It's better to carry on the examination in the patient's standing condition. The examination of the stomach is carried in a standing and lying position.

4. The plan of examination. At the beginning the general examination is made, and then — local examination is made. The general examination is the examination of the patient from head to foot, irrespective of prospective local-

ization of disease process. Local (special, detailed) examination — examination of that area which corresponds to localization of disease process.

The circuit of carrying out of the general examination

On carrying out of the general examination it is necessary to estimate.

1. A general view of the patient.
   1.1. The general condition of the patient.
   1.2. The position of the patient.
   1.3. The consciousness of the patient.
   1.4. The build and constitution.
   1.5. Height.
   1.6. The weight of the body, nutritional state.
   1.7. The bearing.
   1.8. The gait.
   1.9. The examination of the head, the face, the neck.
2. The condition of the skin and its appendages.
3. The condition of a hypodermicall-fatty layer.
4. The condition of lymph nodes.
5. The condition of the muscular system.
6. The condition of the bone system.
7. The condition of joints.

The general condition of the patient

It is integrated indicator characterising a physical and mental condition of the person.

Types of the patient's condition:

1) good;
2) satisfactory;
3) of average gravity;
4) severe;
5) the poorest;
6) agonal.

The severity of the patient's condition is characterised by expressiveness of infringement of functions of the organism, the presence of threat to the patient's life, probability of development dangerous complications in the near future.

Types of the patient's position

Active position is a position in which the patient can change a body pose independently even if movements cause pains and (or) other unpleasant sensations.

Passive position is observed during the unconsciousness of the patient, in extreme weakness of the patient, in extensive damages of the loco-motor system

and the nervous system when the patient is not in condition to change the pose independently.

The compelled position is a position which the patient accepts for simplification of pain or unpleasant sensations.

Consciousness of the patient:

1) clear;
2) dulled;
3) absence of consciousness (coma).

Signs of clear consciousness:

1. Correct orientation in space, in time, in own person.
2. Logic of thinking.
3. Correct and timely answers to questions.

Signs of the dulled consciousness:

1. Detachment of the patient (impossibility of correct perception of the surrounding world).
2. Orientation impairment in space and time.
3. Orientation impairment in own person.
4. Incoherence of thinking.
5. A full or partial amnesia (absence of the memoirs connected with the period of dulled consciousness).

Types of impairments of consciousness

1. Stupor — a condition of obnubilation, from which it is possible to help out of stupor (catalepsy) for short time with a conversation, however the patient orientates badly himself in surrounding conditions, answers questions slowly and with delay.
2. Sopor (hibernation) — the patient is in a dream condition, does not react to the speech turned to him or answers questions tersely («yes», «no»), however reacts to examination, palpation, percussion.
3. Coma is a full loss of consciousness with a sharp decrease or full absence of the basic reflexes.

During the dulled consciousness delirium and hallucinations can be observed.

Delirium — mental impairments shown in false judgements, conclusions.

The hallucination is a deceit of feelings, the false perceptions arising without corresponding irritation.

Build and constitution

The build is a set of morphological features of the organism which has developed on the basis of hereditary and got properties (got properties are often defined by features of environment surrounding the patient).

The constitution (from Latin «*constitution*» — «the device», «addition») is a set of functional and morphological features of the organism which has developed on the basis of hereditary and got properties and defining the organism's reaction to influence of external and internal factors (M. V. Chernorutsky).

Constitutional human body types:

1. Normostenic (average) type.
2. Asthenic type.
3. Hypersthenic type.

Index of Pinhei

It is applied for definition of type of the constitution of the person.

Index of Pinhei is L minus (T plus P), where: L — Height (sm), T — a circle of the chest (sm), P — weight of the body (kg). In the normosthenics the size of index of Pinhei makes up plus 10 — plus 30; in hypersthenics — it is less than plus 10; at asthenics — it is more than plus 30.

Estimation of the condition of the diet (fatness)

The estimation of the condition of the person's diet is made on the basis of comparison of his body height and of the body weight. Thus the following settlement parameters — indexes are used.

Index of Brock

Index of Brock is equal to body height (cm) minus 100 with augmentation for hypersthenics and decrease for asthenics on 10 %.

Ketle index of mass of body (IMB)

Index of mass of body (IMB) is shown in the formyla 1:

$$IMB = \frac{Body\ weight\ (kg)}{Body\ height^2\ (m)} \qquad (1)$$

Size **IMB** in the healthy adult person makes 19–23.

Bearing is a habitual position of the body at standing, walking and sitting.

Pose is an interposition of parts of the body.

Gait is a set of the pose and movements on walking.

The estimation of the condition of the skin and its appendages includes the definition of:

1) Colouring (color) of the skin.

2) Cleanliness of the skin; the clean skin is absence an eruption, scratchings, ecchymoses, cicatrixes, ulcers on the skin.

3) Turgor (elastance) of the skin.

4) Humidities of the skin.

5) Body height and character of the scalp.
6) Condition of nails.

Types of cyanosis

Cyanosis — the cyanotic colouring of the skin caused by increase of concentration of restored hemoglobin is higher than 50 g/l in blood.

1. A peripheric cool cyanosis (Crocq's disease) — a sign of heart failure.
2. Diffuse warm (central) cyanosis — a sign of respiratory failure.
3. Local cyanosis — a sign of local infringement of venous blood-flow.

Types of icteruses

Yellow staining skins (icterus) is a sign of adjournment of pigment of bilirubin in the skin (endogenic, true icterus) or pigments of an exogenous parentage — Carotinum (carrots), citrus, some medicines (exogenous, false icterus).

Turgor (elastance) of the skin

It is examined by capture of the skin with 2 fingers on the back side of the surface of the patient's hand. Normally turgor dermal fold finishes quickly. The turgor of the skin can be: reduced (emaciation, deaquation, advanced age) and increased (edemas, scleroderma).

Condition of the hypodermic fatty layer

We estimate:
— A degree of expressiveness.
— Uniformity of distribution of the hypodermic fatty layer.
— Presence of edemas.
— Presence of lipomas and other tumorous formations.

Edemas

1. Local: discirculatory — are caused by local venous stagnation (clottage of the vein, phlebitis, a prelum of the vein) or lymphostasis (elephantiasis, metastases of cancer); inflammatory — local inflammation of the skin, hypodermic fat (a phlegmon, a furuncle, an abscess); allergic (Quincke's edema).
2. Wide-spread: stagnant (heart failure); renal (in disease of kidneys); dystrophic (protein-free); hepatic (in cirrhosis of the liver); endocrine (hypothyroidism).

Lymph nodes

In norm lymph nodes are not revealed visually and on palpation.
On palpation we estimate:
— the number of enlarged lymphonodes;
— tenderness;
— consistence;
— mobility;
— cohesion among themselves and with environmental tissues.

Palpation as a method of clinical research

Palpation (from Latin «*palpatio*» — «a palpation») — a clinical method of research by means of taction of muscular and spatial (stereometric) feelings with the purpose of studying physical properties of tissues and organs, topographical parities between them, their sensitivities and detection of some functional phenomena in the organism.

By means of a method of a palpation it is possible to determine

1. The character of organ surface.
2. The temperature of the skin of the palpated site of the body.
3. The humidity of the skin or mucosas.
4. The consistence of the organ and tissues covering it.
5. The elastance of the skin, organs and tissues.
6. The form of the organ or pathological formation.
7. The position of organs or pathological formations.
8. The size (sizes) of organs.
9. The mobility of organs and tissues.
10. The mutual relation of organs or structures.
11. The sensitivity and tenderness.
12. The functional phenomena caused by movements of organs and fluctuation of their structures during their work: voice tremor above lungs; apical and cardiac jerks, murmur of heart («the cat's purring»), etc.

Elements of palpation

— Installation of the hand in the initial position.
— A motionless position of the hand.
— Pressure with the tips of one or several fingers.
— Sliding on the skin (stroking) or together with the skin on underlying tissues.
— Gradual, sometimes stage-by-stage immersing of fingers into the stomach on certain depth.
— A push — a fast immersing of fingers on the necessary depth.
— Capture of being palpated tissues or organs between fingers of 1 or 2 hands.
— Displacement of tissues or organs.
— Return of the hand to a starting position.
— Carrying over of the hand to a new position.

Consecutive performance of a set of elements of palpation — a cycle of palpation.

The general rules of palpation

1. It should be warm indoors where the palpation is carried on.
2. Sites of the body which are palpated should be bared.
3. Position of the patient should provide the maximum relaxation of muscles and not cause pain and unpleasant sensations. Position of the patient depends on research objectives.

4. Position of the doctor should be convenient and not cause pressure and exhaustion, should provide freedom of movements.

5. More often the doctor settles down to the right of the patient, facing him.

6. Hands of the doctor should be warm, nails on them — close-cut.

7. Palpating movements should be possible easier and soft, any pressing should be gradual.

8. Palpation should be begun with the healthy part or a body site (if it is known), and then come over to the sick part or body sites.

9. It is always necessary to compare the sick and healthy parts (comparative palpation).

Ways of palpation

— Palpation with a motionless hand.
— Stroking.
— Palpation-pressure.
— Palpation in a fold of tissues.
— Rough palpation of the stomach.
— Superficial sliding palpation of the stomach.
— Deep sliding palpation.
— Conjoined manipulation.
— Jerky palpation.
— Diaphragm-inspiratory palpation.
— Sliding off palpation.
— Palpation with «a double hand».

LECTURE 3
PERCUSSION AND AUSCULTATION AS A RESEARCH METHODS. PERCUSSION AND AUSCULTATION OF LUNGS.

Percussion (from Greek «*percussio*» — «drawing of blows») is a method of objective research by means of percussion on a surface of the patient's body with a simultaneous estimation of sounds arising on this process.

The founder of the method of percussion is Viennese doctor Leopold Auenbrugger. Fundamental propositions about a percussion method are stated in 1761 in the treatise «The New opening allowing to find out the latent chest diseases on the basis of percussion of the chest».

Percussion types

1. Direct percussion consists of percussion with one or several fingers directly on the patient's body.

2. The mediocre (mediated) percussion — drawing of blows is made on plessimeter, enclosed to the patient's body.

Methods of direct percussion

1. The method of L. Auenbrugger.
2. The method of F. G. Yanovsky.
3. The method of V. P. Obraztsov.
4. Stroking (sliding) percussion.

Methods of mediocre percussion

1. With a hammer on plessimeter.
2. With a finger on plessimeter.
3. With a finger on a finger (the method of G. I. Sokolsky).

Percussion types on intensity of percussion blow

1. Deep (strong or loud).
2. Superficial (weak or silent).
3. The quietest (weakest), limiting, threshold percussion — the method of Goldschider.
4. The method of Plesha.

The basic percussion sounds

1. A clear pulmonary sound. It's receive on the percussion of those sites of the chest where the normal pulmonary tissue adjoins directly the chest wall. It's loud, long, low and not tympanic.

2. A dull sound. It occurs over those sites of the chest, where dense parenchymatous organs — the heart, the liver, the spleen adjoin them.

In pathological conditions a dull percussion sound is observed in all cases of considerable reduction airness of lungs, fillings of the pleural cavity with liquid or dense formations. A dull percussion sound is silent, short, high also resembles the sound received on a blow against a tree.

3. A tympanic sound. It's revealed everywhere where the cavities containing air adjoin the chest wall. Tympanic tone possesses a sonorous musical shade and resembles the sound arising on a blow in a drum. In the healthy person it is defined only in one site of the chest — on the left in the lower parts at the front, in the so-called «semilunar space Trawbe».

The mixed percussion sounds

1. The dulled sound (clear pulmonary plus dull).
2. A pulmonary sound with a tympanic shade.
3. Dulled — tympanic.

The general rules of percussion

1. It should be warm and silent in the room where the percussion is carried out.

2. On percussion the position of the patient should be convenient, with the maximal relaxation of muscles.

3. On percussion the position of the doctor should be convenient.

4. It is necessary to percuss a naked body.

5. Hands should be warm.

6. A plessimeter should be a middle or index finger of the left hand.

7. The finger — plessimeter is put to the patients body with the whole palmar surface, but without a strong pressing. Index and fourth fingers are pulled apart.

8. Percussion impacts should be rendered by a pulp of a trailer phalanx of a middle finger of the right hand in the area of the joint between trailer and middle phalanxes of the finger — plessimeter or on its middle phalanx.

9. The finger hammer should be bent so that its trailer phalanx is under the right angle to the basic one. Other fingers do not adjoin it.

10. The nail of a striking finger should be trimmed short.

11. The axis of a trailer phalanx of a striking finger and the direction of percussion impact should be strictly perpendicular to the surface of the finger-plessimeter.

12. Percussion impact should be mild and always of identical force. On percussion the hand should be incurvated only in a radiocarpal joint, and the forearm and the upper arm should be motionless.

13. Percussion impact should be short.

14. Percussion impact should be resilient.

Topographical percussion

The purpose of topographical percussion is to determine the sizes and configuration of various organs (lungs, heart, liver, spleen, etc.) and delimit them from each other.

The general rules of topographical percussion

1. The direction of percussion. Drawing of percussion impacts should be made in the direction from the organ giving a loud percussion sound, to the organ giving a dull sound, i.e. from a clear sound to a dull one.

2. The position of the plessimeter. A finger — plessimeter is located on a being percussed surface in a parallel way to the border of expected dullness.

3. The force of percussion impact. Superficial (weak, silent) percussion is applied to define absolute dullness it. Louder percussion is applied to define relative dullness of the organ.

4. The mark of border of the body is made on the external edge of a finger — plessimeter inverted to the organ, giving a louder sound.

With the help of topographical percussion of lungs we determine:

1. The lower border of lungs.

2. Height of standing of apexes of lungs.

3. Width of fields of Crenig.

4. Excursion of the lower edge of lungs.

The ptosis of the lower border of both lungs is marked:

— In acute or chronic lung expansion (emphysema).
— On the expressed weakening tone of abdomen muscles.
— On a low position of the diaphragm, that more often occurs on a ptosis of the organs of the abdominal cavity (visceroptosis).

The shift of the lower border of lungs upwards from both sides occurs:

— On rising of pressure in the abdominal cavity owing to a clump of liquid (ascites), air (an acute perforation of the ulcer of the stomach and duodenum) in it, owing to meteorism (a clump of gases in the intestines).
— In obesity.
— In bilateral exudative pleuritis.

Shift of the lower lungs border upwards from the one side is observed on:

— Decrease of lung size in pneumosclerosis.
— An atelectasis (fall) of lungs owing to bronchial obstruction.
— Accumulation of liquid in a pleural cavity.
— A substantial growth of the liver sizes (cancer and echinococcosis).
— Augmentation of the spleen.

Pathological deviations of lungs apexes from norm:

1. A lower standing of apexes of lungs and narrowing of fields of Crenig is observed on infiltration of lungs apexes, in presence of pleural adnations in their area, that more often occurs as a consequence of tuberculosis.
2. A higher standing of apexes of lungs and expansion of fields of Crenig is marked in lungs emphysema.

Decrease of mobility of the lower edge of lungs is observed:

— In emphysema.
— On inflammatory infiltration.
— In the presence of plenty of liquid in the pleural cavity.
— On adnation of pleural leaves.

Comparative percussion

The purpose of comparative percussion is to reveal pathological processes in lungs, pleural cavities and the abdominal cavity.

Rules of comparative percussion lungs

1. The percussion should be carried out on strictly symmetric sites of both halves of the chest.
2. If it is possible to suggest beforehand where the pathological process is localised percussion is started with the healthy part.

3. The position of the finger-plessimetr and the force of its pressing to the patient's body should be equal from both parts.

4. Tapping blows should be equal on force from both parts. The force of blow is average. Application of alternately loud and silent percussion in one and the same point is possible.

5. Percussion is carried out on the maximum relaxation of the patient, on smooth quiet breath.

6. Percussion is only carried out on intercostal intervals.

7. In thorax deformation the diagnostic value of comparative percussion is not big.

The reasons of shortening percussion sound

1. Reduction of airness of the pulmonary tissue (pneumosclerosis, tuberculosis of lungs, lobar pneumonia in hepatization stage, the cavity filled with liquid — sputum, pus, echinococcus cyst, tumour, pulmonary infarction, atelectasis).

2. Changes of the pleura complicating of carrying out percussion blow to the pulmonary tissue containing air (pleura thickening — tumours, liquid in the pleural cavity).

Tympanic sound

It is characteristic of: air congestions in the pleural cavity (pneumothorax); emphysemas of lungs; the presence of the containing air cavity in the lungs (an abscess, a cavity) if the cavity is located close to the chest wall and its size is not less than 3–4 cm; high standing of the diaphragm owing to a high arrangement of hollow organs of the abdominal cavity; phrenic hernias — the stomach or gut loops get into the thorax through a hernial aperture; pneumopericardium; hypodermic emphysema.

Variants of tympanic sound

1. Metal percussion tone. To receive of metal tone hammer-plessimeter percussion on the method of Gejbner-Liechtenstern is applied.

2. A box sound.

3. The sound of the burst pot.

4. Dulled tympanic sound.

Dulled tympanic sound

Arises: in the initial stage and the permission stage of lobar pneumonia; in bronchopneumonia when the sites of consolidation of the pulmonary tissue alternate with the sites containing air; in exudate pleurisy, in a lung site over exudate (in the zone of compression atelectasis); in incomplete obturation atelectasis; in a partial prelum of the lung with air, tumour, highly standing diaphragm; in the early stage of hypostasis of lungs.

Auscultation is a method of research consisting of auscultation of the sound phenomena, arising in the organism as a result of fluctuations of its elements, and of judgement about a physical condition of the organs according to a sound.

Auscultation types

1. Direct auscultation — is made by ear applying to the patient's body.

2. The mediocre (mediated) auscultation — is made with the help of a stethoscope (phonendoscope).

General rules of auscultation

1. It should be as quiet, as possible in the room where auscultation is made.
2. It should be warm in the room where auscultation is made.
3. Being listened to parts of the body should be bared.
4. If it is necessary one should shave or moisten with water the being listened to part of the body.
5. The stethoscope bell should be warm.
6. The stethoscope bell should be enclosed to the patient's body with all its edges.
7. Fixing a stethoscope to the patient's body, it is not necessary to touch tubes.
8. The stethoscope cannot be pressed to patient's body too close.
9. Stethoscope chestpiece should closely adjoin the walls of external acoustical meatus, but should not cause pain.
10. It is expedient to use the same stethoscope.
11. The position of the patient and the doctor should be convenient.
12. It is necessary to supervise over the patient his breath, other actions improving the quality of auscultation.
13. Listening to the patient, it is necessary to learn to distract from coming from the outside noise.

Rules of auscultation of lungs

1. Lungs are listened to in the patient's sitting or better standing position.
2. It is necessary to observe the sequence on auscultation (the front surface of the chest, lateral departments, the back surface).
3. It is necessary to use the methods improving the carrying out of the sound:

— on auscultation in armpit region the patient should get his hands behind his head;

— on auscultation on scapular and paravertebral lines the patient should cross his hands on the chest and incline his head slightly forward.

4. The patient should breathe deeply, with regular intervals, slowly, through a half-open mouth.
5. It is necessary to supervise over the patient's respiration.
6. It is originally expedient to carry out comparative auscultation, and then listen to those sites where pathological changes have been noticed in details

The sound phenomena revealed on lungs auscultation

1. Respiratory murmur.
2. Additional acoustic phenomena.
3. Bronchophony.

Classification of respiratory murmurs

1. The basic respiratory murmurs: vesicular respiration; bronchial respiration; mixed (bronchovesicular) respiration.

2. Additional (collateral) respiratory murmurs: rales and rhonchi; crepitation; pleural rub; plueropericarditis murmur.

The basic respiratory murmurs

Vesicular (alveolar) respiration

Vesicular respiration is basic respiratory murmur which results from soothing out of alveoli during an inspiration. Thus air entering the alveoli, causes stretching of their walls. The walls of the alveoli pass from the weakened condition intj intense one that causes the oscillatory movements forming the murmur.

The reasons of weakening of vesicular respiration

Physiological: a thick thoracal wall (obesity, excessive development of musculation); an insignificant layer of pulmonary tissue (the apex, the lower departments of lungs).

Pathological:

1. The syndrome of «obstacle»: pleural exudate, pheumothorax, fibrothorax.
2. Obturation of the trachea or large bronchi, conducting to atelectasis.
3. Drop of an elastance of alveoli (emphysema, interstitial oedema of lungs, etc.).
4. Decrease of respiratory mobility of a chest: pains; a pathology of ribs and a spine; a pathology of a respiratory musculation.

The reasons of intensifying of vesicular respiration

Physiological: deep and often respiration in connection with physical exertion, nervous overexcitement; a thin thoracic wall and a high elastance of lungs — puerile respiration, characteristic of children and teenagers.

Pathological: vicarious or replaceable respiration — is determined above a site of a healthy lung, posed near the pathologically changed site; hyperventilation in hyperthermia, hyperthyroidism; respiration of Kussmaul, characteristic of some types of comas being accompanying with acidosis (uremic coma, diabetic coma, hepatic coma).

Qualitative changes of vesicular respiration

1. Rigid respiration. This intensifying of vesicular respiration which is characterized by a low tone sound distinguished as a rough, sounding, completely occupying inspiratory and exhalation phases. The reason is bronchitis.
2. Rigid respiration with a prolate exhalation — sign of narrowing of lumen of fine bronchi.
3. Saccadic respiration. It is a version of vesicular respiration which is characterized by intermittence of its sounding. It is observed on: non-uniform narrowing of lumen of bronchioles owing to occlusion with mucous, non-uniform reduction of respiratory musculation (myosites, myasthenia, botulism, etc.), nervosisms and nervous overexcitement, frigorism.

Bronchial (laryngotracheal) respiration

Bronchial respiration arises in the larynx on passage of air through a voice rima and is caused by fluctuations of vocal chords and adjoining parts of the larynx.

Zones of auscultation of physiological bronchial respiration

— A front surface of the larynx, in the field of thyroid WHOilage, a bulbar fossa.
— Area of the handle of the breast bone.
— On juxtaspinal lines, at a level from the 7th cervical up to the 2nd – 4th thoracal spine.
— Above apex of the right lung.

Types of pathological bronchial respiration

1. Infiltrative. It occurs in condition of permeability of bronchi to a site of alveoli which are filled with inflammatory exudate, blood or transudate. This is loud, intensively sounding bronchial respiration which is determined in pneumonia, pulmonary tuberculosis, lungs infarct, pulmonary bleedings, lungs edema.
2. Atelectative. It occurs on compression atelectasis of lungs. This bronchial respiration is indistinct, silent, weak.
3. Cavitary. It occurs in presence of air cavitary formations surrounded with a dense elastic wall in the lungs. This is loud, sonorous bronchial respiration. Versions of cavitary bronchial respiration are: 1) amphoric respiration; 2) metal respiration.

Additional respiratory murmurs

Additional (collateral) respiratory murmurs are the murmurs arising in connection with respiratory movements of the chest. They are auscultated though on a background of the basic respiratory murmurs, but separately from them.

Rales and Rhonchi are the additional respiratory murmurs arising in the trachea, bronchi and cavities of lungs.

Rales result from the passage of air through fluid or moisture.

Rhonchi are sounds produced as air passes through narrowed passageways, regardless of the cause, such as exudates, inflammation, spasm, or tumor

Classification of rhonchi

1. Sibilant (high, treble) rhonchi (ronchi sibilantes) — appear in small bronchial tubes at narrowing of their gleam.
2. Sonorous (bass, low) rhonchi (ronchi sonari) — appear in large and average bronchial tubes at a mucous membrane hypostasis, a congestion dense sputum.

Classification of rales

On calibre:

1) fine (small bubbling) rales — occur in small bronchial tubes in the presence of liquid sputum;
2) medium rales — occur in bronchial tubes of average calibre and in small bronchiectasias;

3) coarse (large bubbling) rales — occur in large bronchial tubes, trachea and cavities of lungs.

On sonority:

— sibilant (not sonorous) rales — appear in the bronchial tubes surrounded with a normal pulmonary tissue (bronchitis);

— sonorous rales — appear in the bronchial tubes surrounded with the condensed pulmonary tissue (pneumonia, tuberculosis, pulmonary infarction) or in cavities (bronchiectasias, cavities).

Crepitation

Crepitation arises in the alveoli which walls are moistened with a viscous secretion. On inhaling of a wall of alveoli become unstuck, creating a sound in the form of an easy crash.

The reasons of crepitation occurrence

1. Initial (I) stage of lobar pneumonia — non sonorous initial crepitation (crepitatio indux).
2. Final (III) stage of lobar pneumonia — sonorous final crepitation (crepitatio redux).
3. Focal pneumonia (not frequent phenomenon).
4. Initial stages of tubercular infiltration of lungs.
5. Pulmonary infarction.
6. Initial stages of hypostasis of lungs.
7. Chronic stagnant heart failure.
8. In the lower departments of lungs in the weakened and elderly patients after a long lying.

Noise of friction of pleura

Noise of friction of pleura (affrictus pleurae) is the additional respiratory noise being listened to over the place of inflammation of pleural leaves.

The reasons of pleura friction noise occurrence

1. Dry pleurisy.
2. Exudative pleurisy — after resorption exudate.
3. Pleura tuberculosis.
4. Tumoral defeat.
5. Considerable dehydration and drying of leaves of pleura (cholera, unrestrained vomiting).
6. Uraemia.

Bronchophony

Bronchophony is a carrying out of voice from the throat along the air column of the trachea and bronchial tubes on the chest surface.

Bronchophony occurs on: consolidation of the pulmonary tissue (pneumonia, tuberculosis, etc.); cavitary processes in lungs (an abscess, a cavity).

LECTURE 4
SYMPTOMATOLOGY AND DIAGNOSTICS OF BRONCHIAL ASTHMA, BRONCHITES AND EMPHYSEMAS OF LUNGS

Bronchial asthma is a chronic inflammatory disease of airways with participation of many cells, mediators of allergies and the inflammations, accompanied by hyperreactance and variability, reversible obstruction of bronchial tubes that is shown by attacks of asthma, cough or the complicated breath.

Risk factors of development of bronchial asthma

I. The factors concerning the development of bronchial asthma

1. Predisposing factors.

1.1. Atopy is a tendency to the development of raised quantity of IgE in reply to the contact with environment allergens.

1.2. Heredity — is inherited level of IgE and hyperreactance of bronchi.

2. Causal factors — allergens.

3. The factors promoting occurrence of asthma.

3.1. Smoking (active and passive).

3.2. Virus respiratory infection.

II. The factors causing aggravation asthma (triggers)

1. Allergens.
2. Respiratory infection.
3. Physical activity and hyperventilation.
4. Weather conditions (weather changes, cold air, high humidity, etc.).
5. Emotional loadings.

The mechanism of development of asthmatic fit in bronchial asthma

1. Spastic stricture of a smooth musculation of bronchi.
2. Edema of mucosa of bronchi.
3. Hyperproduction of sputum.

Classification of bronchial asthma

1. Mainly allergic asthma (exogenous).
2. Nonallergic asthma (endogenic).
3. Asthma of a mixed type (a combination of signs of allergic and nonallergic asthma).
4. Not specified asthma.
5. The asthmatic status.

Classification of bronchial asthma on a degree of gravity

1. Mild incidental (intermittent) development of asthma.
2. Mild persistent asthma.

3. Medium persistent asthma.
4. Serious persistent asthma.

Clinic of dyspnea attack

1. The stage of signal symptoms: rhinitis, eye itching and lacrimation, urticaria and skin itching, paroxysmal dry cough.

2. The stage of attack height: progressing expiratory breathlessness, sense of prelum of the chest, dry or unproductive cough, abjointing of viscid sputum.

Examination: diffuse cyanosis, compelled position orthopheoa, puffiness of face, emphysematous chest, infrequent, deep, noisy respiration.

Palpation of the chest: rigidity, dilating of the intercostal spaces, the weakened voice fremitus.

Percussion of the chest: a box sound, dilating of lungs borders, restriction of motility of the inferior edge of lungs.

Auscultation of lungs: rigid respiration, significant elongation of expiratory phase, rhonchi.

3. The stage of the sanction of the attack: appearance of productive cough, abjointing of sputum; decrease and dissappearance of breathlessness.

Laboratory diagnostics

1. The general analysis of blood: leukopenia, eosinophilia, lymphocytosis.

2. The analysis of sputum: sputum is viscid, glassy, eosinophilia, Charcot-Leiden crystal, Curschmann's spirals.

3. Allergological diagnostics: revealing of allergenspecific IgE in blood serum; allergological tests with epicutaneous, intradermal, inhalation introduction of allergens; allergological anamnesis.

The asthmatic status is intensive, long attack of dyspnea, steady against usual methods of the treatment, characterized by nonproductive cough, acute respiratory failure and impairments of gas structure of blood — anoxemia and hypercapnia.

The causes of the asthmatic status

1. Errors in treatment of the patient:
— the termination of corticosteroids intake;
— an overdosage of sympathomimetics;
— sedative preparations intake.
2. Acute respiratory diseases.
3. Psychological stress.
4. Errors in carrying out of deallergization with allergen or making of allergological tests.

Stages of the asthmatic status:

1) relative compensation;
2) a stage of a decompensation or «numb» lungs;
3) a stage of hypoxial hypercapnial comas.

Acute bronchitis is inflammatory process in the trachea, bronchi and (or) bronchioli, described by acute development and diffuse reversible damage mainly of their mucosa.

Etiology of acute bronchitis

1. Contagious factors — viruses of flu, parainfluenza, adenoviruses, mycoplasmas (i. e. originators of acute respiratory diseases).
2. Physical factors — hot air and frigorism, ionizing radiation.
3. Volumetric factors — steams of acids, alkalis, toxicants (sulphur dioxide, oxides of nitrogen).
4. Influence of dust particles.

Contributing factors:

— Smoking.
— Alcoholism.
— Cardiovascular diseases (left ventricle failure).
— Infringements of nasal respiration.
— Locuses of persistent infection in nasopharynx.
— Serious diseases lowering an immune responsiveness of the organism.

Phases of development of acute bronchitis

1. Reactive-hyperemial or nervous-reflex:
— Hyperemia and edema of mucosa.
— Damage of epithelium.
— Oppression of mucocilia clearance.
— Augmentation of mucous production.
2. Contagious phase:
— Bracing of bacterial infection contamination on mucosa.
— Development of purulent inflammation.

Classification of acute bronchitis

I. The etiological factor:
1. Acute contagious bronchitis.
2. Acute noncontagious bronchitis.

II. Character of inflammation:
1. Catarrhal.
2. Purulent.
3. Purulent — necrotic.

III. Localization of lesion:
1. Proximal.
2. Distal.
3. Acute bronchiolitis.

IV. Functional features:

1. Nonobstructive.
2. Obstructive.

V. Development:

1. Acute — till 2 weeks.
2. Lingering — till 4 weeks.
3. Relapsing — arises 3 and more times within a year.

Clinic of acute bronchitis

Complaints:

1. Cough.
2. Abjointing of sputum.
3. Expiratory breathlessness (in the syndrome of bronchial obstruction).
4. Fever.
5. Signs of intoxication.

Examination:

1. Signs of fever: hyperemia of the face, shine of eye, sweating.
2. Diffuse cyanosis (in bronchoobstructive syndrome).
3. The chest is not changed.

Percussion and palpation of the chest:

Pathological changes are not revealed.

Auscultation of lungs:

1. Rigid respiration.
2. Elongation of expiratory phase (in the syndrome of bronchial obstruction).
3. Rhonchi.

Tool methods of diagnostics of acute bronchitis

1. X-ray examination of lungs: intensifying of a pulmonary drawing in radical zones; dilating of roots of lungs.

2. Research of function of external respiration.

For bronchoobstructive syndromethe following signs are characteristic:

— drop of Tiffno index.

— drop of peak expiratory flow.

— moderate drop of a Maximum Voluntary Ventilation (MVV).

Laboratory signs of acute bronchitis

1. The general analysis of blood: neutrofilic leukocytosis with alteration of the nuclear formula of neutrophils to the left; acceleration of ESR.

2. The biochemical analysis of blood: levels of the C-reactive protein, seromucoid, Fibrinogenum, glycoproteins, sialine acids enlarge.

3. Microscopic examination of sputum: plenty of leucocytes with predominance of neutrophils; epithelium of bronchi.

Chronic obstructive pulmonary disease (COPD) is a disease characterized by a chronic diffuse inflammation of bronchi, showing cough with sputum and breathlessness, leading to progressing impairments of a lung ventilation and gas exchange on obstructive type.

Epidemiological definition of chronic obstructive pulmonary disease

Patients with **COPD** are those persons who have cough with a sputum expectoration which lasts not less than 3 months a year within 2 years on end provided that these patients haven't other diseases which can cause the same signs (bronchiectasia, tuberculosis and others).

Aetiology of chronic obstructive pulmonary disease

Risk factors of development of chronic obstructive pulmonary disease are in the table 1.

Table 1 — Risk factors of development of chronic obstructive pulmonary disease

Probability of value	Environmental factors	Internal factors
1. Established	Smoking, increased level of dust and gases in the air connected with professional harmfulness (cadmium, silicon)	Deficiency of alpha-1-antitripsin
2. High probability	Unsuccessful status of environment (SO_2), low social and economic position, alcohol consumption, passive smoking	Family character of disease, low weight at birth
3. Possible probability	Adenoviral an infection, deficiency of vitamin C	Genetic predisposition (1st group of blood, absence of IgA)

Stages of formation of chronic obstructive pulmonary disease

The 1st stage — threats of occurrence of disease.

Presence exogenous and endogenous risk factors: tobacco smoking; long influence of dust and others irritants; frequent acute respiratory viral diseases (more than 3 times a year); infringement of nasal breath; genetic predisposition, etc.

The 2nd stage — predisease.

Changes of mucous membrane of bronchi are characteristic: reorganisation of secretory apparatus; replacement of vibrating epithelium with goblet-like cells; hyperplasia of mucous glands; mucociliary insufficiency.

Clinical displays: cough; long and relapsing current of acute bronchitis.

The 3rd stage — clinically generated chronic obstructive pulmonary disease.

The 4th stage — complications: emphysema of lungs; bronchiectasias; pneumorrhagia; respiratory failure; chronic pulmonary heart.

Components of bronchial obstruction in chronic obstructive pulmonary disease

1. Reversible:

1) Spastic stricture of bronchial muscle.

2) Inflammatory edema, infiltration of mucosa and submucosa of the bronchi.
3) Obturation of respiratory ways by sputum.
2. Irreversible:
1) Fibroplastic changes of bronchial walls.
2) Bronchial stenosis and deformation.
3) Expiratory collapse of fine bronchi.

Classification of chronic obstructive pulmonary disease

Gravity of a disease:
1. Mild — FEV_1 (Forced Expiratory Volume in 1 Second) — 70 % and more.
2. Medium — FEV_1 — 69–50 %.
3. Serious — FEV_1 less than 50 %.

Clinic of chronic obstructive pulmonary disease

Complaints:
1. Cough.
2. Branch of sputum.
3. Expiratory breathlessness.
Examination:
1. A diffuse cyanosis.
2. Emphysematous chest.
3. Participation of auxiliary respiratory musculation in the act of respiration.
Palpation and percussion of the chest:
Pathological changes are not revealed or there are attributes of an emphysema of lungs.
Auscultation of lungs:
1. Rigid respiration.
2. Elongation of expiratory phase.
3. Rhonchi.

Laboratory diagnostics chronic obstructive pulmonary disease

The general analysis of blood (on exacerbation of disease): neutrophfilic leukocytosis with the shift of the nuclear formula to the left; acceleration of ESR.
The biochemical analysis of blood (during an exacerbation of disease): rising of concentration of seromucoid, sialine acids, the C-reactive protein, glycoproteins, Fibrinogenum, globulins fractions.

Tool diagnostics of chronic obstructive pulmonary disease

Function of external respiration: drop of an exhalation capacity; drop of PEF (Peak Expiratory Flow); drop of Tiffno index; drop of MVV; drop of VLC (vital lungs capacity).
X-ray examination of lungs: intensifying and deformation of the pulmonary drawing; expansion and fibrosis of lungs roots; sings of lungs emphysema.

Lungs emphysema

Lungs emphysema (from Greek «*emphysema*» — «the inflation», «filling with air») **is** a lungs condition, characterized by pathological expansion of the air spaces posed more distal of terminal bronchioles.

Forms of lungs emphysema

1. Nonobstructive emphysema — there are no phenomena of obstruction of terminal bronchioles and fine bronchi.

2. Obstructive emphysema — there are impairments of ventilation caused by a collapse (fall) of terminal bronchioles and (or) an obturation of fine bronchi.

Types of nonobstructive lungs emphysemas

1. Compensatory or vicarious lungs emphysema is an inflation of lungs owing to replaceable re-extension of its tissues after a resection of a part of lungs, in atelectases, extensive pneumonias, kyphoscoliosis.

2. Senile (initial, atrophic, senile) lungs emphysema is a consequence of age atrophy of the pulmonary tissue, mainly of resilient fibers.

Types of obstructive lungs emphysemas

1. Functional emphysema — an acute inflation of lungs in a suddenly arisen valval obstruction of bronchi: an aspiration of foreign body, an attack of a bronchial asthma, a bronchiolitis.

2. Chronic substantive lungs emphysema — anatomic damage of lungs, characterized by expansion of air spaces more distal of the terminal bronchioles, accompanying by a destruction of walls of alveoli.

Reasons: 1) COPD; 2) bronchial asthma.

Clinic of lungs emphysema

Complaints:

1. Breathlessness.
2. Undue fatigability.

Examination:

1. Diffuse cyanosis.
2. Emphysematous chest: augmentation of front-back diameter; a horizontal course of ribs; expansion of intercostal spaces; restriction of respiratory excursions.

Palpation of the chest:

1. Rigidity.
2. Weakening of vocal fremitus.

Percussion of the chest:

1. A box sound.
2. High standing of apexes of lungs.
3. Expansion of fields of Crenig.
4. Infraplacement of the lower edge of lungs.
5. Restriction of mobility of the lower edge lungs.

Degrees of gravity of lungs emphysema

1st degree — a feebly marked lungs emphysema.

The lower border of lungs is not changed. Mobility of the lower edge of lungs is diminished up to 4 cm. Absolute dullness of the heart is not determined.

2d degree — lungs emphysema of medium gravity.

The lower lungs border is displaced 1 rib downwards. Mobility of the lower lungs edge diminished up to 2 cm. Insignificant participation of auxiliary respiratory muscles in the act of respiration.

3d degree — sharply expressed lungs emphysema.

The lower lungs border is displaced 2 ribs downwards. There is no mobility of the lower lungs edges. Participation of auxiliary muscles in the act of respiration is sharply expressed. The lower edge of the liver is below the costal arch.

Rentgenological sings of lungs emphysema

1. Barrel or bell-like chest with horizontal back pieces of ribs and expansion of intercostal spaces.
2. Augmentation of the area and increase of a transparence of pulmonary fields.
3. Pulmonary drawing depression and depletion.
4. A low arrangement of a diaphragm, reduction of excursions of her domes.
5. «Small», «drop» heart.

Research of function of external breath at emphysema of lungs:

— Decrease in vital capacity of lungs.
— Decrease in the maximum ventilation of lungs.
— Increase in residual volume of lungs.

LECTURE 5
SYMPTOMATOLOGY AND DIAGNOSTICS OF PNEUMONIAS, ATELECTASES, PLEURITIS AND PNUEMOTHORAXES. SYNDROME OF IMPAIRMENT OF FUNCTION OF EXTERNAL RESPIRATION

Pneumonia is a group various on aetiology, pathogenesis and the morphological characteristic acute focal infectious-inflammatory diseases of lungs with primary involving in pathological process of respiratory departments and obligatory presence intraalveolar inflammatory exudation.

Working grouping of a pneumonia
(The European respiratory society, 1993)

I. Community-acquired (out of hospital) pneumonia (CAP).
II. Hospital-acquired (hospital, nosocomial) pneumonia (HAP).

III. The pneumonia at immunodeficient statuses.
IV. Atypically proceeding pneumonia.

Etiology out of hospital pneumonia:

— *Streptococcus pneumoniae* — 70–90 %
— *Haemophilus influenzae* — 7–16 %.
— *Mycoplasma pneumoniae* — 20–30 % at persons are younger than 35 years, 1–9 % in more senior age groups.
— *Chlamydia pneumoniae* — 10 %.
— *Legionella pneumophila* — 2–10 %.

Etiology of hospital pneumonia:

— *Staphylococcus aureus.*
— *Pseudomonas aeruginosa.*
— *Klebsiella pneumoniae.*
— *Escherichia coli.*
— *Proteus mirabilis.*

Hospital pneumonia develops at the patients who are on treatment in hospital more of 48 hours.

Etiology of pneumonia at immunodeficient statuses:

— *Pneumocystes.*
— *Pathogenic mushrooms.*
— *Cytomegaloviruses.*
— *Staphylococcus aureus.*
— *Escherichia coli.*
— *Pseudomonas aeruginosae.*

Etiology of atypically proceeding pneumonia

— *Mycoplasma pneumoniae.*
— *Chlamydia pneumoniae.*
— *Legionella pneumophila.*

Infection penetration ways

1. Aerogenic and bronchogenic — by respiratory ways with inhaled air.
2. Contact — from the locus of infection in the organs contacting with the lungs (pericardium, pleura, abdominal cavity).
3. Hematogenous — with blood in sepsis.
4. Lymphogenous — by limphatic vessels.

Stages of lobar pneumonia

1st stage — microbic edema or inflammatory inflow.

Amplification of alveolus capillary permeability; serous exudate in the lumen of alveoli with lots of microorganisms.

2nd stage — hepatizations. Filling the lumen of alveoli with exudate, containing a lot of fibrin. Infiltration of the pulmonary tissue.

3rd stage of the outcome. Dilution and excretion of alveolus exudate through bronchi, inflammatory process outcome.

Classification of pneumonias (1995)

I. Variants of pneumonias depending on originating epidemiological conditions:
1. Out of hospital pneumonia.
2. Intrahospital (hospital) pneumonia.
3. The Pneumonia at immunodeficient statuses.
4. Pneumonias in patients with immunodeficient conditions.

II. On localization and extention:
1. Unilateral (it is left, right-hand):
— Total.
— Lobar.
— Segmentary.
— Lobular.
— Central («radical»).
2. Bilateral.

III. On gravity:
1. Serious.
2. Average.
3. Mild or abortive current.

Clinic of lobar pneumonia

I. The onset of the disease is acute, sudden:
— Chill.
— Fever — 39–40 ºC.
— A pleural pain in the chest.
— Nonproductive cough.
— Malaise, drop of working capacity, other signs of intoxication.

II. The height of the disease:
— Fever of constant type with critical drop in 2–7 days.
— Pains in the chest (pleural).
— Cough with branch of sputum.
— Features of sputum: viscid, frequently — hemorrhagic, of brown or red color, volume is no more than 50–100 ml, without smell.
— Breathlessness of the inspiratory or mixed types.
— Signs of intoxication.
— A diffuse cyanosis, hyperemia of the face.
— Herpes labialis.
— Frequent superficial respiration.

— Backlog of a half of the chest on the affected side in the act of respiration.

— Palpation of the chest: pain and intensifying of voice tremor above the center of pneumonia.

— Percussion of lungs: a stage of a microbic edema — a dulled — tympanic sound; a stage of hepatization — the dulled or dull sound; a stage of sanction — a dulled — tympanic sound.

— Auscultation of lungs: a stage of inflow — the weakened vesicular or bronchial-vesicular respiration, sonorous fine vesicular rales, nonsonorous crepitation; a stage of hepatization — bronchial respiration; a stage of sanction — weakened vesicular or bronchial-vesicular respiration, sonorous fine vesicular rales, sonorous crepitation.

Laboratory diagnostics of lobar pneumonia

1. The generalan alysis of blood:

— High neutrophilic leukocytosis.

— The expressed shift of the nuclear formula of neutrophils to the left.

— Aneosinophilia.

— High ESR.

2. The biochemical analysis of blood: augmentation of concentration of fibrinogenum, C-reactive protein, seromucoid, glycoproteins, globulins.

3. The analysis of sputum: viscid, hemorrhagic, contains plenty of neutrophils, erythrocytes, clots of fibrin, originators of the disease.

Bronchial (focal) pneumonia affects the lungs in patches around the tubes (bronchi or bronchioles).

Clinic of focal pneumonia

I. The onset of the disease is gradual, within 3–4 days, frequently clinic of acute bronchitis precedes.

— Fever — 38–39 ^{o}C.

— Nonproductive cough.

— Malaise, drop of working capacity, other signs of intoxication.

II. The height of the disease.

— Fever of the wrong type with lytic drop in 2–7 days.

— Pains in the chest are visceral, dull, whining, nonintensive.

— Cough with branch of mucopurulent sputum, volume is no more than 50–100 ml, without smell.

— Breathlessness of the inspiratory or mixed types - an infrequent sign.

— Signs of intoxication.

— Diffuse cyanosis (seldom), hyperemia of the face.

— Accelerated respiration.

— Backlog of a half of the chest on the affected side in the act of respiration.

— Palpation of the chest: insignificant intensifying of vocal fremitus above the center of pneumonia.

— Percussion of lungs: a dulled — tympanic or dulled sound;

— Auscultation of lungs: the weakened vesicular or bronchial-vesicular respiration, sonorous fine vesicular rales. Crepitation is seldom revealed.

Laboratory diagnostics of focal pneumonia

1. The general analysis of blood: moderate neutrophilic leukocytosis; shift of the nuclear formula of neutrophils to the left; augmentation of ESR.

2. The biochemical analysis of blood: augmentation of concentration of Fibrinogenum, C-reactive protein, seromucoid, glycoproteins, globulins.

3. The analysis of the sputum: mucopurulent, contains plenty of neutrophils.

Syndrom of atelectasis

Atelectasis is a fall of lungs or its parts, observable on the termination of access of air into alveoli.

Types of atelectasis:

— Obturative.
— Compression.
— Distention (functional).
— Mixed.

The reasons of obturative atelectasis:

— Aspiration of the foreign body.
— Occlusion of bronchi with mucus, viscous sputum.
— Bronchi occlusion with endobronchial tumour.
— Prelum of bronchi with a tumour from the outside, lymphonoduses, cicatricial tissue.

The reasons of compressive atelectasis:

— A liquid congestion in the pleural cavity.
— Air congestion in the pleural cavity.
— Tumours of the pleura and mediastinum.
— Aneurysms of large vessels.

Functional atelectasis

It is observed: in the weakened lying patients; on impairments of function of respiratory muscles; on impairment of function of the respiratory centre; on a high standing of the diaphragm with impairment of its mobility (ascites, meteorism, peritonitis, pregnancy).

Pleuritis is infectious or aseptic inflammatory process of various aetiology in pleura, is accompanied by formation of imposings of fibrin on their surface and (or) congestion of exsudate of various character in the pleural cavity.

Etiological classification of pleuritis

1. Infectious pleuritis: bacterial (*Pneumococcus, Streptococcus, Staphilococcus, Haemofillus influenzae, Klebsiella pneumonia, Pseudomonas aeruginosae*, etc.) — parapneumonic and metapneumonic; tubercular; rickettsial; protozoan (amoebas); fungoid; parasitic (echinococcus); virus.

2. Noninfectious (aseptic) pleuritis: tumoral; allergic (in allergoses); in diffusive diseases of connective tissue (acute rheumatic fever, rhematoid arthritis, scleroderma); on thromboembolism of the pulmonary artery and lung infarction (discirculatory); traumatic — a thorax trauma, electroburns, X-ray therapy; in myocardial infarction (Dressler's syndrome); fermentogenic (pancreatic); uraemic; in hemorrhagic diathesis; in familial paroxysmal (familial recurrent) polyserositis.

Penetration ways of the etiological factor

1. Contact — in pneumonia, tuberculosis, abscess of lungs, etc.
2. Hematogenic.
3. Lymphogenous.
4. From the environment — on traumas of the chest, medical manipulations.

Pleural exudate syndrome pathogenesis

Pleural fluid is produced by the parietal layer of pleura and reabsorbed by its visceral layer.

Mechanisms of the pleural exudate formation:

1. Amplification of permeability of pleura vessels (inflammation).
2. Drop of absorptive ability of pleural lists (inflammation).
3. Change of hydrostatic and colloid-osmotic blood pressure in pleural vessels.

Hydrothorax is a pathological syndrome caused by accumulation of non-inflammatory fluid — transudate in the pleural cavity.

Etiology of hydrothorax:

A. Increase of hydrostatic pressure.
— Circulatory insufficiency.
— Tumours of the mediastinum squeezing vena cava and brachiocephalic veins.
B. Drop of oncotic blood pressure owing to hypoproteinemia in:
— Kidneys diseases.
— Liver cirrhosis.
— Nutritional dystrophy.
— Malabsorption syndrome.
— Serious anemia.
C. Miksedema (hypothyroidism).

Types of pleurites

1. Exudative (wet) — is characterized by accumulation of exudate in the pleural cavity.

2. Dry (fibrinous) — there is no exudates in pleural cavity, there are available applying of fibrin on pleura.

Character of exudate in exudative pleuritis

1. Serous.
2. Serofibrinous.
3. Purulent.
4. Putrefactive.
5. Hemorrhagic.
6. Eosinophilic.
7. Cholesteric.
8. Chilic.
9. Mixed.

Clinic of dry pleuritis

I. Complaints:

1. Pain in the chest (pleural):
— Character — acute, stabbing, intensive.
— Location — lower-lateral departments.
— Increases on inspiration, cough, laughter.
— Subsides on expiration, with the patient lying on the sick side.
2. Pleural cough:
— Dry, painful, cautious.
3. Breathlessness of inspiratory type.
4. Fever and other signs of intoxication.

II. Examination:

1. Compelled position:
— Lying on the sick side;
— Sitting — bending to the sick side.
2. Often shallow breathing.
3. Backlog of the half of the chest in the act of respiration on the side of lesion.

III. Palpation of the chest:

1. Tenderness of intercostals.
2. Crepitation — palpatory equivalent of pleural friction rub.
3. Backlog of the half of the chest in the act of respiration on the side of lesion.

IV. Percussion of the lungs:

1. Restriction of excursions of the inferior sick side lung edge.
2. Tenderness.
3. Insignificant dullnesses (spastic stricture of muscles, thickening of pleural lists).

V. Auscultation of the lungs:

1. Weakened vesicular respiration on the sick side.
2. Saccadic respiration.
3. Pleural friction rub.

Roentgenography of the lungs

1. Restriction of motility of the dome of the diaphragm on the sick side.
2. Costal-diaphragmatic sinus is sealed on the sick side.

Clinic of exudative pleuritis

I. Complaints:

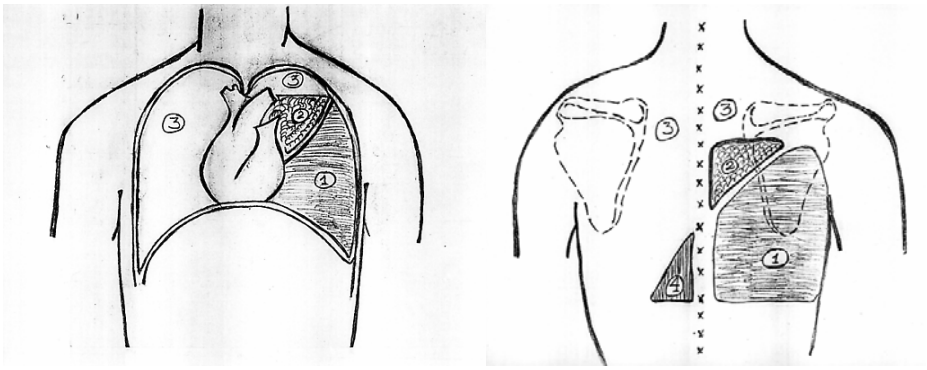
1. Pressure in the chest on the sick side.
2. Nonintensive, dull aches in the chest on the sick side.
3. Inspiratory or mixed breathlessness.

II. Examination:

1. Compelled position: lying on the sick side; sitting - bending to the sick side.
2. Augmentation of the half of the chest on the sick side, dilating and protrusion of intercostal spaces.
3. Accelerated shallow breathing.
4. Backlog of the half of the chest in respiration on the sick side.

Pleural exudate. The data of physical examination

The data of physical examination are shown in the picture 1.



Picture 1 — The data of physical examination
1 — A zone of exsudate; 2 — Harlend's trigone;
3 — A zone of not changed lung; 4 — Roufus-Grokko trigone.

1. A zone of exsudate (1).

Palpation: intercostal intervals rigidity; vocal trembling absence.

Percussion: dull sound.

Auscultation: respiratory noise absence or vesicular respiration is sharply weakened.

2. A zone of compressive atelectasis (Harlend's trigone) (2).

Palpation: vocal trembling is strengthened.

Percussion: dilled-tympanic sound.

Auscultation: bronho-vesicular or bronchial respiration.

3. A zone of not changed lung (3).

Palpation*:* pathological changes are not present.

Percussio clear pulmonary sound (in vicarious emphysema — a pulmonary sound with a tympanic shade).

Auscultation*:* vesicular respiration (in vicarious emphysema — strengthened vicarious respiration).

4. Roufus-Grokko trigone-results from displacement of mediastinum organs (4).

Palpation: weakened vocal trembling.

Percussion: dulled sound.

Auscultation: weakened vesicular respiration.

The differential diagnostic distinctions between pleural exsudate and transudate are shown in the table 2.

Table 2 — Differential diagnostic distinctions between pleural exsudate and transudate

Signs	Exsudate	Transudate
Appearance of liquid	Turbid, frequent hemorrhagic, can be purulent, putrefactive, has a smell	Transparent, slightly yellowish, sometimes colourless, has no smell
Change of appearance of pleural liquid after standing	Grows turbid, flakes of fibrin, pus, can clot	Remains transparent, the deposit is not formed
The protein content	> 30 g/l	< 30 g/l
LDG	> 200 ME/L or > 1,6 g/l	< 200 ME/L or < 1,6 g/l
Density	> 1,018 kg/l	< 1,015 kg/l
Rivalt's, Lucerini tests	Positive	Negative
Amount of leukocytes in pleural liquid	> 1000 in 1 mm^3	< 1000 in 1 mm^3

Syndrome of pneumothorax

Pneumothorax — a pathological status characterised by a congestion of air in the pleural cavity.

Development mechanisms of pneumothorax

1. Rupture of the pulmonary tissue and visceral pleurae.
2. Trauma with damage of parietal or visceral pleurae.

Types of pheumothorax

1. Traumatic.
2. Operational.
3. Spontaneous.
4. Artificial

Spontaneous pneumothorax — air congestion in the pleural cavity, not connected with mechanical damage of the thorax or pulmonary tissue on trauma or medical influences.

Classification of spontaneous pneumothorax

I. By origin:
1. Primary (idiopathic).
2. Symptomatic (secondary).

II. On prevalence:
1. Total.
2. Partial (partial).

III. Depending on presence of complications:
1. Not complicated.
2. Complicated (bleedings, pleurisy, etc.).

Aetiology of primary pneumothorax

1. Limited violent emphysema of lungs.
2. Insufficiency of alpha-1-antitrypsin.
3. Congenital weakness of the pleura.
4. Deep immersing in water, flight on the plane at the big height (sharp pressure differences).

Aetiology of symptomatic pneumothorax

I. 1. Tuberculosis of lungs.
2. Complications of pneumonia: abscess and gangrene of lungs, empyema of pleurae.
3. Chronic obstructive pulmonary diseases.
4. Bronchiectasias.

II. 1. Lungs congenital cysts.
2. Lungs and pleura malignant tumours.
3. Hydatid disease of lung.

Pneumatothorax pathogenetic variants

1. Open.
2. Closed.
3. Valvular (tense).

The syndrome of failure of function of external respiration (FER)

Types of impairments of function of external respiration

1. Ventilating — impairments of ventilation of alveoli owing to impairment of permeability of bronchi or restrictions of respiratory motility of the chest.

2. Parenchymatous — a consequence of lesion of lungs parenchyma: diffusive — impairment of diffusion of gases from alveoli in blood of capillars of lungs; perfused — impairment of pulmonary blood-flow; restrictive — drop of respiratory surface of lungs or restriction of distensibility of lungs.

Respiratory failure (RF) is a pathological condition of the organism in which maintenance of normal gas structure of arterial blood is not provided, or normal gas structure of blood is achieved due to such work of the apparatus of external respiration which results in drop of functionalities of the organism.

Types of ventilating respiratory failure

1. Centrogenous **RF** — is caused by oppression of the respiratory center (pathology of brain, intoxication).
2. Neuromuscular **RF** — is caused by impairment of carrying out or neuromuscular transmission of impulse to respiratory muscles, and also by diseases of muscles (lesion of spinal cord, botulism, myasthenias, myosites).
3. Thoraco-diaphragmatic **RF** — is caused by restriction of motility of the chest or restriction of locomotion of lungs by extrapulmonary causes (kyphoscoliosis, Bekhterev disease, massive pleural adnations, pleural exudate, pheumothorax).
4. Bronchial-pulmonary **RF** — caused by a pathology of bronchi and lungs (this type includes two variants: **obstructive** and **restrictive)**.

Obstructive type of respiratory failure

The occurrence reasons:

1) a spasm of smooth muscles of bronchi;
2) a thickening of the wall of bronchi as a result of hypostasis, cellular infiltration of mucous membrane or fibrosis of bronchial tubes;
3) presence of viscous sputum in bronchi;
4) endobronchial tumour;
5) foreign body in bronchi;
6) prelum of bronchi by a tumour from the outside, by the increased lymph nodes or large vessel aneurysm;
7) expiratory collapse of the trachea and large bronchial tubes.

Changes of external breath function in obstructive type of respiratory failure

1. Decrease in exhalation power.
2. Decrease in exhalation peak speed.
3. Decrease in volume of the Forced Expiratory Volume in 1 Second (FEV_1).
4. Decrease of Tyffno's index (Tyffno's index = FEV /vital capacities of lungs) × 100 %, norm (rate) — 70–80 %).
5. Decrease in the maximum ventilation of lungs (due maximal pulmonary breathing capacity = vital capacities × 35).

Restrictive type of respiratory failure

The occurrence reasons:

1) fibrosis of lungs (pneumoconiosises, scleroderma);
2) emphysema of lungs;

3) pleural adnation;
4) exudative pleurisy, hydrothorax;
5) pneumothorax;
6) alveolitis, pneumonia, tumours of lungs;
7) extraction of a lung site.

Changes of external breath function in restrictive type of respiratory failure

1. Decrease in vital capacity of lungs.
2. Decrease in the maximal pulmonary breathing capacity.

Mixed (obstructively-restrictive) type of respiratory failure

It is characterised by presence of signs of obstructive and restrictive types of respiratory insufficiency in the patient.

Acute respiratory failure

The term of acute RF means:
1) Sudden occurrence of respiratory failure.
2) Gradual development of RF up to the critical status demanding intensive therapy or resuscitation.

Stages of acute respiratory failure

The 1st stage — initial.
It's characterized by:
— The compelled position of the patient — orthopnea.
— Expressed cyanosis of skin and mucous membranes.
— Excitation, trouble, sometimes delirium, hallucinations.
— Accelerated respiration up to per 40 per minute.
— Participation of auxiliary respiratory muscles in the respiratory act.
— Tachycardia up to 120 per minute.
— Moderate arterial hypoxemia (Pa O_2 — 60–70 mm of Hg) and normocapnia (Pa CO_2 — 35–45 mm of Hg).
The 2nd stage — deep hypoxemia.
It's characterized by:
— The poorest condition of patients.
— Superficial respiration, patients convulsively suffice with a mouth air.
— Position — orthopnea.
— Alternation of the periods of excitation with drowsiness periods.
— Frequency of breath exceeds 40 per minute.
— Frequency of cardiac contractions is above 120 per minute.
— Hypoxemia is revealed in blood (P_aO_2 — 50–60 mm of Hg) and hypercapnia (Pa CO_2 — 50–70 mm of Hg).

The 3rd stage — hypercapnic coma.

It's characterized by:

— Loss of consciousness.

— Expressed diffusive cyanosis.

— Cold sticky sweat.

— Pupils are expanded (mydriasis).

— Superficial, rare, often arrhythmic respiration — Chejn-Stoks type.

— Sharp hypoxemia is revealed in blood (P_aO_2 — 40–55mm of Hg) — and expressed hypercapnia ($P_a CO_2$ — 80–90 mm of Hg).

The Stages of chronic respiratory insufficiency are shown in the table 3.

Table 3 — Stages of chronic respiratory insufficiency

Stages	I (compensated)	II (expressed subcompensated)	III (decompensating)
Breathlessness	On the prof. exertion	On daily exertion	At rest
Cyanosis	No	Appears on exertion	Diffusive constant
Participation of auxiliary muscles in the respiratory act	Do not participate	Participation is significant on exertion	Participate at rest
Frequency of respiration (per minute)	May be norm	More than 20 at rest	More than 20 at rest
Ventilating infringements	Decrease in indicators up to 80–50 %	Decrease in indicators up to 50–30 %	Decrease in indicators below 30 %

LECTURE 6
INQUIRY OF PATIENTS WITH CARDIOVASCULAR DISEASES. SURVEY AND PALPATION OF THE HEART AREA AND GREAT VESSELS

The reasons of occurrence of heart and chest pain:

— Ischemic heart disease.

— Myocarditis.

— Pericarditis.

— Cardioneurosis.

— Myocardiodystrophy.

— Aorta pathology (aortitis, dissecting aneurysm of aorta).

Palpitation (heartbeats) is a subjective sensation of heartbeats. Patients complain of severe and loud hearbeats.

Heartbeats and bradycardia

Causes: vagotonia in patients with neurocirculatory asthenia (cardioneurosis); complete atrioventricular heart block and separate variants of atrioventricular heart block of the 2d degree.

Heartbeats in combination with normal rate of the heart rhythm

Causes: cardioneurosis; aortic insufficiency; nonclosure of arterial (Botallo's) duct.

Accelerated heartbeating

Causes:

1) increase of the positive chronotropic effect of the sympathetic nervous system on the heart or decrease of the vagus nerve negative chronotropic effect on the heart (in healthy people may occur normally with exercise, after the emotions, having tea, coffee, alcohol), after smoking or taking activating heart medicines (adrenaline, ephedrine, aminophylline, atropine);

2) heart failure;

3) arterial hypotension;

4) thyrotoxicosis;

5) anemias;

6) fevers;

7) paroxysmal, suddenly arising and ceasing palpitations, of different duration, which testify about paroxysmal tachycardias.

Mechanism of cardiac breathlessness

Decrease of cardiac contractions → slowing down of the blood-flow → hypoxemia, hypercapnia → stimulation of the respiratory center → breathlessness.

Signs of cardiac breathlessness

Character — inspiratory, mixed.

Originating conditions — physical exertion, a prone position.

What gives a relief — physical rest, position orthopoea.

It is accompanied by tachycardia.

Can be paroxysmal (cardiac asthma).

Hemoptysis in patients with cardio-vascular diseases system can be caused by:

1. Penetration of erythrocytes through alveolar-capillary membrane in substantial increase of pressure in veins and capillars of pulmonary circulation.

2. Lung infarction of owing to thromboembolia of the pulmonary artery branches.

3. Rupture of swallen venous vessels in bronchi while coughing.

Mechanism originating cardiac edemas

1. Increase in capillary hydrostatic pressure and slowing down of the blood-flow in them.

2. Impairment of the humoral regulation of the water and salt exchange, hyperaldosteronism, intensifying of natrium reabsorption, retention of fluid, augmentation of blood volume.

3. Decrease of albuminums formation in the liver because of venous stagnation, drop of oncotic blood pressure.

Types of a compelled position of a patient

1. Orthopneoa — in acute left ventricle failure, chronic circulatory unefficiency of II–III stages.
2. Sitting, when bending forward — in pericardites.
3. Standing — during the attack of stenocardia.
4. Compelled body's postural change in myocardial infarction and stratifying aortic aneurysm.

Types of skin coloration

1. Acrosyanosis — cyanosis of finger tips, lips, nose, auricles — in heart failure.
2. Paleness of the skin — in aortal heart diseases (aortal paleness), acute vascular failure (syncope, shock, collapse).
3. Skin hyperemia — in arterial hypertension.
4. Icterus or icteritiousness of sclera — because of impairment of liver function at heart failure.
5. Colour «coffee with milk» — at infectious endocarditis.

Specific kinds of the face:

1) mitral face — the face of patient with mitral stenosis — pallor of the face skin with cyanosis cheeks, lips and youthful appearance;
2) face of Korvizar — the face sick of heart failure — icteric-pale with cyanosis, bloated with dim eyes, a half-open mouth.

At survey of area of heart and large vessels it is possible to reveal

1. Heart hump.
2. Apex beat.
3. Heart beat.
4. Aorta pulsation.
5. Pulmonary artery pulsation.
6. Heart aneurysms pulsation.
7. «Carotid shudder» and other arteries.
8. Venous pulse.

The heart hump (gibbus cardiacus) is protrusion of thorax in the field of the heart, caused by strong increase in its sizes. The occurrence reason - increase in the sizes of heart at children's age.

Apex beat is a pulsation of the limited site of the forward wall of thorax in area of conformity heart apex in the systole beginning, connected with change of the heart form and myocardium density.

The apex beat push is not found out at:

1) thick chest wall;
2) the developed muscles;

3) narrow intercostal intervals;
4) at women with developed mammary glands.

Ways of visual optimisation revealing of apex beat:

1) a trunk inclination forward;
2) a prone position on left to a side;
3) survey after a deep exhalation.

Characteristic properties of the apex beat

1. Width (area): poured, normal, limited.
2. Height: high and low.
3. Force: strengthened, not strengthened, weakened.
4. Resistance (the sensation reflecting density of a myocardium): resistant, not resistant.

Width of apex beat

The width of apex beat is the area of concussion of the thorax made by him.
In norm diameter 1–2 cm.
More than 2 cm — poured.
Less than 1 cm — limited.

A wide apex beat

Results from enlarged heart and, first of all, the left ventricle.

Exocardial causes of increased in the area of the apex beat:

1) a thin thorax;
2) wide intercostal intervals;
3) corrugation of the lower edge of the left lung;
4) heart displacement forward by a tumour of mediastinum;
5) high standing of the diaphragm.

A limited apex beat is observed in:

1) emphysema of the lungs;
2) low standing of the diaphragm;
3) exudative pericarditis.

Symptom of «cat's purring» — is a vibration (or trembling) of the chest wall, resembling the purring of the cat, caused by low-frequency fluctuations of the wall of the heart, caused by a blood stream on a passage through a narrowed (stenosis) valve aperture during the phase of systole or diastole.

Types of «cat's purring»:

1) systolic trembling — coincides with the apex beat and pulse wave on the carotid;

2) diastolic trembling — does not coincide with the apex beat and pulse wave on the carotid;

3) systolic — diastolic trembling of the chest wall.

Diagnostic value of «cat's purring» symptom

— Systolic trembling in the 2nd intercostal space at the breastbone right edge — aortic ostium stenosis.

— Systolic trembling in the 2nd intercostal space at the breastbone left edge — pulmonary artery stenosis.

— Systolic trembling in the 4th intercostal space at the breastbone left edge — a defect symptom of the interventricular septum.

— Diastolic trembling over the heart apex — the symptom of the left atrioventricular canal stenosis (mitral stenosis).

— Diastolic trembling over the basis of the xiphoid process — the symptom of the right atrioventricular canal stenosis (tricuspid stenosis).

— Systolic — diastolic trembling in the 2nd intercostal space at the left edge of the breastbone or jugular fossa — the symptom of an nonclosure arterial (Botallo's) duct.

Venous pulse — periodic swelling of jugular veins and their subsequent fall resulting from the activity of the heart.

Venous pulse is a swelling of veins, but not a pulse wave arising in arteries as arterial pulse wave from the aorta is exhausted in the field of capillaries and does not reach veins.

The origin of venous pulse

Venous pulse results from blood pressure change in the right atrium at heart beat. These fluctuations are not caused by a return current of blood from the auricle into veins during its systole, but only depend on the delay of outflow of blood from veins into the right atrium or its (outflow) of acceleration.

The properties of arterial pulse are shown in the table 4

Table 4 — Properties of arterial pulse

Mains properties	The name of the pulse	The note
I. The rhythm	1. Regular (rhythmical), (pulsus regularis)	In healthy persons
	2. Irregular (arrhythmic), (p. irregularis)	The causes: 1) extrasystole; 2) atrioventricular heart block; 3) fibrillation
II. Pulse rate	3. More than 90 per minute. 4. Less than 60 per minute.	Tachycardia Bradycardia
III. Tension	5. Tense hard pulse (p. durus). 6. Soft pulse (p. mollis)	Explains intra-arterial blood pressure

The continuation of table 4

Mains properties	The name of the pulse	The note
IV. Filling	7. Strong pulse (p. plenus). 8. Pulsus vacuus (p. vacuus)	Explains artery filling; depends on blood volume, vascular permeability, stroke volume, i. e. identical filling both hands (feet) simultaneously — norm, i. e. filling decrease on one artery, e. g. radial artery, occurs in mitral stenosis, thromboembolism, etc.
	9. Identical (p. aequalis). 10. Unequal (p. inaequalis)	This is an identical filling of an artery while examining one artery. It differs from identical pulse when pulse on both radial arteries is simultaneously examined
V. Amount (dilatation degree of artery during passage pulse waves through it)	11. Great (p. magnus) or high (p. altus). 12. Small (p. parvus) or low. 13. Thready pulse (p. filiformus)	Hard and full pulse. In aortic valve insufficiency. Pulse soft and pulsus vacuus or tense and pulsus vacuus. Filling and tension are extremely lowered, in shock, acute heart failure
VI. Form (the velocity of pressure changes in the arterial system)	14. The abrupt pulse (p. celer). or bouncing pulse (p. saliens) 15. Slow (p. tardus).	Fast occurrence and disappearance of pulse waves, is observed in insufficiency of aortic valves. Slow occurrence and disappearance of pulse waves, is observed in aortic ostium stenosis
VII. Other changes	16. Dicrotic or split (p. dicroticus) 17. Paradoxical (p. paradoxus) 18. Alternating (p. alternans) 19. Deficiency of pulse (p. deficiens)	It is perceived as a dual blow due to the presence of additional pulse a push during the decrease of the basic pulse wave. It is observed in the general decrease in the tone of peripheral arteries in patients with serious infectious diseases. Filling of the pulse during a deep breath decreases sharply in exudative and constrictive pericarditis, emphysema of lungs, tumour of mediastinum, pleural effusion in which diastole and its blood filling is limited because of pressure upon the heart. Alternation of pulse waves of a small and normal amplitude; occurs in severe defeats of the myocardium. The frequency of cardiac contractions is more than pulse fluctuations on the peripheral artery that occurs in fibrillation, extrasystole

The characteristic of the normal wall of the artery

1. Soft.
2. Flat.
3. Elastic.
4. Smooth.

The factors influencing the characteristics of the pulse:

1. Sex.
2. Age (newborns — rate — 130–150 per 1 minute, by 20 years of age — norm).
3. Height.
4. Physical exertion (in 1–2 minutes the rate should be normal).
5. Emotions.
6. Body position (transition from horizontal position in a sedentary one — increases on 4–6 beats; transition from a sedentary position into vertical one — increases on 6–8 beats).
7. Digestion.
8. Phases of respiration: inhaling — acceleration; exhalation — delay.

LECTURE 7
ASCULTATION OF THE HEART. HEART TONES IN NORM AND PATHOLOGY. HEART MURMURS

Tones of the heart — short abrupt sounds arising in a healthy heart during its work.
Tone is a sound caused by periodic fluctuations.
Simple (pure) tone is a sound caused by fluctuations of one frequency.
Complex tone — a set of simple tones with the multiple relation of frequencies.

Types of heart tones

1. Constant tones — the 1st and the 2nd tones.
2. Changeable tones — the 3rd, the 4th tones.
3. Additional tones — opening snap of mitrale valve, systolic click, pericardium-tone.

The sequence of ventricle phases cycle of the heart activity

Systole of ventricles (0,33 s.) is shown in the table 5.

Table 5 — Systole of ventricles (0,33 seconds)

Periods	Phases	The note
The pressure period (PP) (0,08 seconds)	Phase of asynchronous contraction (0,05 seconds)	Myocardium fibres are consistently involved in contraction process. The form of ventricles changes, but pressure does not increase. At the end of this phase are AV valves (Q-I tone) close

The continuation of the table 5

Periods	Phases	The note
The pressure period (PP) (0,08 seconds)	Phase of isometric contraction (0,03 seconds)	The contraction period of ventricles on closed valves when pressure in ventricles is less than pressure in the aorta and the pulmonary artery
The exile period (EP) (0,25 seconds)	Phase of fast exile (0,12 seconds)	Begins on pressure in LV = 65–75 mm Hg and in RV = 5-12 mm Hg. It is characterised by emission of 2/3 stroke volume

Diastole of ventricles (0,47 s.) in shown in the table 6.

Table 6 — Diastole of ventricles (0,47 seconds)

Periods	Phases	The note
The relaxation period (0,1–0,13 seconds)	Protodiastolic phase (0,04 seconds)	Time from the beginning of the ventricle relaxation to the aorta and pulmonary artery semilunar valves closing (time spent on closing semilunar valves)
	The phase of isometric relaxation (0,07–0,11 seconds)	Relaxation of ventricles with closed of atrioventricular and of semilunar valves while pressure in ventricles does not decrease lower, than in the atriums by that time filled with blood
The period of ventricle filling (0,25 seconds)	Phase of fast filling (0,09 seconds)	Begins with opening of clack-valves, lasts until the pressure drop termination in atriums. 3nd tone means the end of this phase
	Phase of slow filling (0,016 seconds)	Proceeds till the moment of atrial depolarization
	Phase of atrial systole (0,06–0,09 seconds)	The expulsion of blood from atriums into ventricles during the systole of auricles. Corresponds to 4th tone

The mechanism of 1st tone formation

1st tone consists of 4 components.

The basic is *a valvular component* — formed by fluctuations of cusps atrioventricular valves at accent against them of blood in the phase of the isometric contraction.

The muscular component — is caused by fluctuations of the ventricles myocardium in the phase of isometric contraction.

The vascular component — is connected with fluctuations of initial parts of an aorta and a pulmonary trunk by blood when it is ejected.

The mechanism of 2nd tone formation

2nd tone consists of 2 components.

The basic — *is a valvular component* — formed by beating blood against closed semilunar cusps of the aorta and pulmonary trunk valves in the period of relaxation.

The vascular component — is connected with fluctuations of the initial parts of the aorta and pulmonary trunk by transfer of fluctuations from valves of the aorta and pulmonary trunk.

The mechanism of formation 3rd and 4th tones

3rd tone — protodiastolic tone — is caused by fluctuations of ventricle walls, arising during their fast passive filling with blood in phase of diastole.

4th tone — presystolic — is caused by the fluctuations arising when filling the ventricles with blood during a systole of atriums, it arises at the end of diastole (in the presystole).

Rules of auscultation of the heart

1. It is necessary to listen to the heart in various positions: when the patient is lying, standing, after physical activity.

2. It is better to listen to the heart after a deep breath at a delay of breath and after the subsequent deep exhalation for respiratory noise does not disturb listening.

3. The heart auscultation is necessary to perform in a strict sequence (from 1 to 5 point consistently). Sounding of 2nd tone is necessary to compare with points of auscultation 2 and 3.

4. At revealing any changes in auscultation points all of heart areas are carefully listen to.

5. To improvement auscultation of sound phenomena connected with the mitrale valve pathology, it is necessary to give the patient position on the left side when the apex of the heart approaches the chest wall closer; defeat of the aorta valve is better revealed at auscultation of the patient in a vertical position with the hands crossed and lifted over the head and in a prone position on the right side.

Accessory tones

Opening snap of mitral valve — is listened to in the 4th and 5th intercostal intervals between the left edge of a breastbone and the heart apex, it is connected with an accent of blood and fluctuations of sclerotic cusps of the mitral valve in the initial stage diastole. It is only characteristic of mitral stenosis.

Systolic click — arises at the mitral valve prolapse, it is caused by bending the cusps of the mitral valve into the atrium during the systole of ventricles.

Pericardtone — arises at adhesive pericarditis, it is connected with fast and sharp stretching of ventricles by adhesions at the beginning of the diastole.

«Quail rhythm» is a triple rhythm, characteristic only of a mitral stenosis:

— loud 1st sound on the apex beat;

— the 2nd sound is not changed or there is an accent of the 2nd sound over the pulmonary artery;

— opening snap of mitral valve.

«The gallop rhythm» is a triple rhythm listened to against tachycardia and reminds gallop of the running horse on its sounds, testifies a considerable decrease myocardium tone.

We distinguish:

— protodiastolic gallop (1st, 2rd and 3rd sounds);

— middiastolic gallop (1st, 2rd, 3rd and 4th sounds);

— presystolic gallop (1st, 2rd and 4th sounds).

Exocardial reasons of strengthening of both tones

— Exhaustion, a thin chest wall.

— Corrugation of anterior parts of the left lung.

— A tumour of back mediastinum (removes the heart anteriad).

— Infiltration of adjacent edges of lungs to the heart.

— Big air cavities in lungs.

Exocardial reasons of easing of both tones

— Adiposity.

— Excessive development of muscles.

— A hypodermic emphysema.

— Left-side hydrothorax.

— An emphysema of lungs.

— Exudative pericarditis.

Cardiac reasons of strengthening of both tones:

— Increase of influence of the sympathetic nervous system on the heart (tachycardia caused by thyrotoxicosis, anemia).

The cardial reasons of easing of both tones:

— myocarditises;

— myocardium dystrophy;

— cardiosclerosis;

— vascular collapse, arterial hypotension;

— myocardial infarction;

— decrease of contractive activity of the myocardium.

Pathological conditions it which easing of the 1st tone over the apex of the heart is observed

— Mitrale valve insufficiency.

— Aorta valves insufficiency.

— Aortal stenosis.
— Hypertrophy of the left ventricle.
— Myocardium pathology (myocarditises, dystrophies, cardiosclerosis).

Pathological conditions in which easing of the 1st tone over the basis of xiphoid process is observed:

— insufficiency of the tricuspid valve;
— insufficiency of pulmonary artery valves;
— stenosis of pulmonary artery;
— hypertrophy of right ventricle.

Pathological conditions in which strengthening of the 1st tone over the apex of the heart is observed:

— mitrale stenosis;
— extrasystole;
— full atrioventricular block;
— fibrillation.

Pathological conditions in which strengthening of the 1st tone at the basis of xiphoid process is observed:

— stenosis of right atrioventricular aperture (tricuspid stenosis).

Pathological conditions at which easing of the 2nd tone over the aorta is observed:

— insufficiency of aorta valves;
— low arterial pressure;
— aortal stenosis.

Pathological conditions in which easing of the 2nd tone over the pulmonary artery is observed:

— insufficiency of pulmonary artery valves;
— low pressure in the pulmonary circulation;
— stenosis of pulmonary artery.

Pathological conditions in which strengthening (accent) of the 2nd tone over aorta is observed:

— arterial hypertensia;
— aorta atherosclerosis;
— syphilitic aortitis.

Pathological conditions in which strengthening (accent) of the 2nd tone over the pulmonary artery is observed:

— the diseases accompanied by hypertensia in pulmonary blood circulation (mitrale heart diseases, pulmonary heart, primary pulmonary hypertensia).

Cardiac murmurs are periodic, prolonged sounds, listened to in the heart area, synchronously connected with its activity, not having the accurate beginning and the end, revealed within interval between tones, and not only on the site of valves projection, but also on the big space of the cardial and exocardial areas.

Rules of auscultation of the heart on revealing of murmurs

1. In the presence of heart murmur, auscultation is carried on not only in the points of auscultation of valves, but also over the whole surface of the heart.
2. The heart auscultation should be carried on in patient's position standing and lying.
3. For improvement of auscultation of the heart murmurs connected with the mitral valve pathology, it is necessary to lay to the patient on the left side when the apex of the heart approaches the chest wall closer.
4. For improvement of auscultation of the heart murmur connected with the aorta valve defeat, it is necessary to give the patient a vertical position with the hands crossed and lifted over the head, a prone position on the right side.
5. For improvement of auscultation of the heart murmur connected with the right atrioventricular valve defeat, the patient should be given a position on the right side or on a back with the legs lifted up.
6. It is better to listen to cardiac murmurs at a breath delay after a usual (superficial) breath so that respiratory murmur does not disturb listening.
7. Special tests: Muller's test, Valsalva's test, test with a dosed out physical activity, test with medicinal preparation (for example nitroglycerine) are carried on to change intracardiac haemodynamics conditions.

Classification of cardiac murmurs by occurrence place

1. Intraheart (intracardiac) murmurs — are formed in the heart.
2. Exocardial (extracardiac) murmurs — are formed outside the heart.

Variants of intracardiac murmurs

— Organic.
— Inorganic (functional).

The reasons of organic intracardiac murmurs

— Anatomic changes in the valves (shutters, chords).
— Nonclosure of embryonic apertures — oval window, Botallo's duct, foramen interventriculare.

The causes of functional intracardiac murmurs

— Anemias — with low number of formed elements of blood and acceleration of intracardiac bloodstream (anemic murmurs).
— Changes of intracardiac haemodynamics with acceleration of the bloodstream (haemodynamic murmurs) in case of fevers, nervous stress, thyrotoxicosis.
— Functional changes of the valvular apparatus without its morphological changes — inflammatory, dystrophic changes or heart muscle necrosis, a valvular ring stretching, papillary muscle dysfunction (muscular murmurs).

The reasons of exocardial murmurs

— Visceral and parietal pericardium leave friction in inflammation (pericardial friction murmur).

— The friction of the leaves of the inflammed pleura, adjacent to the pericardium (pleuropericardial murmurs).

— The stretch of the pulmonary tissue pressed by the heart during heart systole (cardiopulmonary murmur or the so-called systolic breath).

Classification of cardiac murmurs in relation to phases of the cardiac cycle:

1) systolic murmurs — heard between 1st and 2nd heart tones during a systolic pause, coincide with a apex beat and pulse wave on a carotid;

2) diastolic murmurs — heard between 2nd tone and 1st tone of the following warm cycle during diastolic pauses, do not coincide with the apex beat and the pulse wave on the carotid;

3) systolic-diastolic murmurs are heard during systolic and diastolic pauses.

The causes of systolic murmurs

— Mitrale valve insufficiency.
— Tricuspid regurgitation, tricuspid insufficiency.
— Aortic ostium stenosis.
— Pulmonary artery ostium stenosis.

The causes of diastolic murmurs

— Left atrioventricular ostium stenosis.
— Right atrioventricular ostium stenosis.
— Aorta valves insufficiency.
— Pulmonary artery valves insufficiency.

The Classification of murmurs by force and loudness in shown in the table 7.

Table 7 — Classification of murmurs by force and loudness (by Friman-Lievayn in modification by Tsukerman)

Loudness degree on a scale	Murmurs is listened
I	Only in the epicentre after some adaptation
II	In the epicentre right away without adaptation
III	Through a back surface of the palm applied to the murmur epicentre
IV	On the wrist if the palm is placed on the murmur epicentre
V	On a forearm if the palm is placed on the murmur epicentre
VI	Through an air layer between the thorax and the phonendoscope

Classification of murmurs according to loudness change from the moment of occurrence till termination:

1) decreasing murmurs — their loudness gradually decreases (decreases the majority of cardiac murmurs);

2) increasing murmurs — their loudness gradually amplifies (presystolic murmurs in case of mitrale stenosis);

3) decrescent-accruing (middiastolic and presystolic murmurs in mitral stenosis);

4) accruing-decreasing (diamond-shaped and fusiform) — exile murmurs in stenosis of aorta and the pulmonary artery;

5) monotonous murmurs — their loudness is the same on all extent (systolic murmur in mitral insufficiency).

Classification of murmurs by duration:

1) short — proceed less than half of cardiac cycle phase;

2) long — last more than half of cardiac cycle phase;

3) pansystolic (pandiastolic) — occupy all cardiac cycle phase (a systole or diastole).

The rules defining a direction of carrying out of murmur

1. Murmur arises not only in the area of narrowing, but also on both sides of it.

2. Murmur is better carried on in the direction of blood current.

3. If on both sides of the place of narrowing the gleams of cavities are different then a louder murmur is listened to over the wider cavity, than over the narrower one.

Differences of functional murmurs from the organic

1. Functional murmurs are more often systolic (exceptions — Flint's murmur, Graham Steell's murmur, Coombs' murmur which are diastolic).

2. Functional murmurs are listened to over the pulmonary artery more often, especially at children's and teenage age; and also over the apex of the heart.

3. Functional murmurs are labile, their properties depend on the position of the body, physical activity, nervous overstrain, inhalation and exhalation phases.

4. Functional murmurs are not carried on in the direction typical for organic murmurs.

5. Functional murmurs are blowing, soft, not musical.

6. Functional murmurs are not accompanied by «cat's purring».

7. Functional murmurs do not occupy the whole systole or diastole.

8. On functional murmurs there are not other signs of valvular heart diseases (change of tones, additional tones, etc.).

The reasons for murmur of friction of the pericardium:

— Inflammation of the pericardium (pericarditis).

— Tumoral defeat of leaves of the pericardium.

— Dehydration of the organism and drying of leaves of the pericardium (vomiting, diarrheas, etc.).

— Small hemorrhages in pericardium (leucoses, hemorrhagic diathesis, vasculitises).

Difference of murmur of friction of the pericardium from intracardial murmurs

1. Pericardium friction murmur not always accurately coincides with a phase of a cardiac cycle — systole or diastole.
2. Pericardium friction murmur is listened to in the zone of absolute dullness of the heart.
3. Pericardium friction murmur is not carried on («dies where it is born»).
4. Pericardium friction murmur amplifies on an inclination of the trunk of the patient forward and on pressing on the chest with a stethoscope.
5. Pericardium friction murmur amplifies on a breath in on throwing back of the head.
6. Pericardium friction murmur is felt more close to the ear, than valve murmurs.

Pleuropericardial friction murmur

Arises in inflammation of mediastinal pleurae, but it is synchronous to heart activity.

Differences of pleuropericardial friction murmur from pericardium friction murmur

1. Pleuropericardial murmur is listened to in the area of the left edge of relative dullness of the heart.
2. Together with pleuropericardial murmur it is possible to listen to a typical friction murmur of the pleura simultaneously.
3. Exhalation pleuropericardial murmur weakens or disappears sharply, on inhalation — it amplifies.

Characteristics of cardiopulmonary murmur:

— It is a short systolic murmur.
— The murmur is gentle, like breath in on a vesicular respiration.
— It is listened to on the edge of relative dullness of the heart.
— It is better listened to in a breath in phase, on a breath delay it disappears or decreases sharply.

LECTURE 8
SYMPTOMATOLOGY AND DIAGNOSTICS OF ACUTE RHEUMATIC FEVER

Acute rheumatic fever (ARF) — a postinfectious complication of A-streptococcal tonsillitis (quinsy) in the predisposed persons, caused by the development of autoimmune answer to antigenes of streptococcus and cross reactance with similar antigenes of the person tissue (in the skin, joints, heart, brain).

Aetiology

The activator of acute rheumatic fever is β-hemolytic streptococcus of group A.

Risk factors of the development of acute rheumatic fever

— Acute and chronic focal streptococcal infection.
— Chronic tonsillitis in relatives.
— Household factors — bad living conditions and bad working conditions.
— Seasonal change of weather.
— Age-sexual factors (children of 7–15 years of age, and persons of a young age, women).
— Genetical predisposition.
— A carriage of antigens HLA — A 2, B 7, B 35.

Pathogeny of acute rheumatic fever

The modern theory of pathogenesis of ARF is toxic-immunopathologic.

Fundamental propositions

1. Ancestral predisposition — dysfunction of the immune system, failure of T-suppressor activity.
2. Formation of plenty of antibodies to antigens of streptococcus (etiotypes of M-protein).
3. Structural resemblance of antigens of streptococcus and antigens of heart, brain and synovial membranes of the person (molecular mimicry).
4. Interaction of antibodies to streptococcus with antigens of tissues of the person, development of immune inflammation in joints (arthritis), myocardium, endocardium (carditis), pericardium, pleura (serositis), brain (chorea).
5. Damage of endocardium of valves of the heart with the formation of heart diseases.

Phases of disorganization of the connecting tissue in acute rheumatic fever

— Mucoid edema — a lesion of the basic material of connecting tissue, accumulation of mucopolysaccharides.
— Fibrinoid necrosis — disorganization of elastic and collagenic fibers, necrosises, deposit of fibrinoid.

— Granulomatous phase — formation of granulomas of Ashoff consisting of hystiocytes, lymphocytes, plasmocytes and fibroblasts.

— Sclerosis — development and inspissation of connecting tissue, deformation and shortering of cusps of heart valves.

The Classification of acute rheumatic fever in shown in the table 8/

Table 8 — Classification of acute rheumatic fever

Clinical Variants	Clinical manifestations		A degree of activity	An outcome	FC of CHF on NYHA
	Basic	Additional			
Acute Rheumatic Fever Repeated Acute Rheumatic Fever	Carditis. Arthritis. Chorea. Annular erythema. Rheumatic nodules	Fever. Arthral-gias. Serosites. Abdominal syn drome	1 — minimal; 2 — moderate; 3 — high	Convalescence Chronic rheumatic disease of heart: — Without a heart disease. — Heart disease	0 1 2 3 4

Examples of formulation of the clinical diagnosis

1. Acute rheumatic fever: a carditis, a polyarthritis, the 3rd degree of activity, CHF FC 1.

2. Acute rheumatic fever: a chorea, the 1st degree of activity.

3. Repeated acute rheumatic fever: a carditis, the 2nd degree of activity. Combined mitral heart disease with predominance of a stenosis. CHF FC 2.

4. Chronic rheumatic disease of heart: postinflammatory regional fibrosis of cusps of the mitral valve. CHF FC 0.

5. Chronic rheumatic disease of heart: combined mitral-aortal heart disease (combined mitral fault with predominance of failure, aortal failure of the 1st degree). CHF FC 2.

Clinic of acute rheumatic fever

1. The onset of the disease — in 2–4 weeks after tonsillitis or pharyngitis.

2. The 1st sign of the disease — fever 38–39 ºC, accompanied with sweating, malaisey.

Clinic of arthritis

1. Joint pain, intensifying on movements.

2. Edema, hyperemia and rise in temperature of the skin above joints.

3. Symmetric lesion of joints.

4. Talocrural, patellar, ulnar, humeral and radiocarpal joints are more often affected.

5. Duration of arthritis is 5–10 days.

6. After the sanction of arthritis joints remain not changed.

Clinic of carditis

Variants of damage of heart:

1) Myocarditis.
2) Myocarditis + endocarditis.
3) Myocarditis + endocarditis + pericarditis (pancarditis).

Clinic of myocarditis

I. Complaints: pains in the heart area; palpitation, faults in the cardiac work; breathlessness of mixed character; orthopneoa; edemas on the legs.

II. Examination: position orthopneoa; paleness of skin, cyanosis, edemas of the legs.

III. Palpation: accelerated pulse of a weak filling; the apex beat is displaced to the left, wide, weakened.

IV. Percussion of the heart: borders of relative dullness of the heart are displaced to the left.

V. Auscultation of the heart: weakening of the I tone on the apex of the heart; the «gallop» rhythm; systolic apex murmur.

Electrocardiographi — signs of myocarditis

1. Decrease of voltage of wave T or inversion of wave T.
2. Depression of segment ST.
3. Arrhythmias: atrioventricular blocks, extrasystole, atrial fibrillation.

Clinical signs of endocarditis

1. Intensifying and elongation of systolic apex murmur during the 4–5 weeks of the disease.
2. Further weakening of the 1st tone on the apex of the heart.
3. Appearance of a transient diastolic apex murmur (edema of cusps of the mitral valve).
4. Appearance of diastolic murmur on the aorta.
5. Echocardiography: thickening, roughness of cusps of valves.

Clinic of dry (fibrinous) pericarditis

I. Complaints.

1. Acute, pricking pains in the heart.
2. Reflex distresses: faults in the cardiac work, palpitation, breathlessness, drop of the blood pressure (BP).

II. Examination. The compelled position of the body — sitting, having bent forward.

III. Palpation of the heart area.

1. Pain in the heart area.
2. Crepitation — sensation of friction of the leaves of the pericardium.

IV. Percussion of the heart. Changes are absent.

V. Auscultation of the heart.

1. Pericardial murmur.

2. Sign of Poten — intensifying of pericardial murmur on inspiration.

3. Sign of Gerce — intensifying of pericardial murmur on throwing back of the head.

Clinic of exudative pericarditis

I. Complaints.

1. Sense of gravity, pressure or dull ache in the heart area.

2. Breathlessness.

II. Examination.

1. Smoothness or a protrusion of intercostal spaces in the heart area.

2. Collar of Stocks — swelling of cervical veins, puffiness and cyanosis of the neck and the face.

3. Sign of Winter — the abdomen does not participate in the act of respiration.

III. Palpation of heart.

Jardin's symptom — apex beat is abatement, his position to the right of the left edge of relative dullness of the heart.

IV. Percussion of heart: enlargement of heart borders to the left and to the right; increase in the sizes of absolute heart dullness; configuration of heart-trapezoid; Ebstein's sign — obtuse angle between right contour of the heart and of the liver.

V. Heart auscultation.

1. Considerable easing of heart tones.

2. Murmur of pericardium friction.

Echocardiography

Echocardiogram of the space around heart — parietal pericardium leaf is removed from the heart with exudate.

Skin defeat in acute rheumatic fever

1. Erythema annulare — light pink stains of annular forms on the skin of the trunk and hips.

2. Rheumatic nodules — dense, painless nodes in diameter of 2–10 mm in subcutaneous fat, in fasciae, aponeurosis (extensor surfaces of elbow joints, knee joints, in the field of anklebones, nape).

Lesions of the nervous system in acute rheumatic fever (chorea minor)

It is caused by defeat of nuc. striatum, nuc. caudatum, nuc. lenticularis.

Signs:

— Hyperkinesis — involuntary movements.

— Statics and coordination impairments.
— Mental instability.
— Muscular weakness.

Defeat of other organs in acute rheumatic fever

Respiratory organs: pleuritis, pneumonites.
Kidneys: glomerulonephrites.
Abdominal organs: peritonitis.

Laboratory diagnostics of acute rheumatic fever

I. Inflammation signs.

1. General analysis of blood: neutrophilic leukocytosis; shift of the nuclear formula of neutrophils to the left; acceleration of erythrocyte sedimentation rate.

2. Biochemical analysis of blood: increase in concentration of C-reactive protein, sialic acids, glycoproteids; dysproteinemia — increase in concentration of alpha-2 and gamma-globulins

II. Signs of carditis: activity increase of cardiospecific enzymes - kinase of phosphocreatine, lactate dehydrogenase ($LDG_{1,2}$); concentration increase of myoglobin.

III. Signs of streptococcal infection: increase of caption of antistreptococcal antibodies in blood — antistreptolysine — 0 (ASL), antistreptokinase (ASK), antistreptohyaluronidase (ASH).

The criteria of diagnostics of acute rheumatic fever in shown in the table 9.

Table 9 — Criteria of diagnostics of acute rheumatic fever

Big criteria	Small criteria
Carditis. Arthritis. Chorea. Erythema annulare. Hypodermic rheumatic nodules	*Clinic:* Artralgia. Fever. *The laboratory:* Increase of ESR. Increase of level of C-reactive protein. *Tool:* Lengthening of PR interval on the electrocardiogram. Signs of mitrale or aortal regurgitation on Echo-CG
The data confirming the preceding A-streptococcal infection. Raised or raising caption of antistreptococcus antibodies. Positive A-streptococcal culture, allocated from pharynx, or positive test of fast definition of the A-streptococcal antigene	

Diagnostic rule

Presence of 2 big criteria or 1 big and 2 small criteria in the combination to the data confirming the previous infection with beta-hemolityc streptococcus of group A, testifies to high probability of acute rheumatic fever.

LECTURE 9
SYMPTOMATOLOGY AND DIAGNOSTICS OF MITRAL AND AORTAL VALVULAR DISEASES

Heart disease — a proof pathological change of the heart structure or the main vessels, breaking its function.

Classification of heart diseases

I. On occurrence terms:
   1. Congenital defects.
   2. The acquired defects.

II. On character of defeat of heart valves:
   1. Stenoses of apertures.
   2. Insufficiency of valves.
   3. Combined defect (stenosis and insufficiency).

III. On quantity of affected valves:
   1. Single-valvular (simple, combined).
   2. Multi-valvular (combined).

Clinical syndromes in heart diseases

1. Syndrome of valvular defeats.
2. Syndrome of pathological process which caused the development of heart disease.
3. Syndrome of impairments of the systemic blood circulation.

Syndrome of defeat of the valve (group of symptoms)

1. Valvular (direct) symptoms: change of heart tones; additional tones; murmurs; «the cat's purring».
2. Indirect symptoms: compensatory hypertrophy and dilatation of heart departments; blood-flow impairment in various vascular areas.

Terminology of heart diseases

1. Insufficiency of the valve.
2. Aperture stenosis.
3. Defect with prevalence of insufficiency.
4. Defect with prevalence of stenosis.
5. Defect without accurate prevalence of stenosis or insufficiency.

The nomenclature of defects of the mitrale valve

— Insufficiency of the mitrale valve.
— Mitrale stenosis.
— Mitrale defect with prevalence of insufficiency of the mitrale valve.

— Mitrale defect with prevalence of stenosis of the left atrioventricular aperture.

— Mitrale defect without accurate prevalence of mitrale insufficiency or mitrale stenosis.

Stenosis of the left atrioventricular aperture

Stenosis of the left atrioventricular aperture (mitrale stenosis) is the heart disease caused by a pathology of the mitrale valve, characterised by narrowing of the left atrioventricular aperture, that creates an obstacle to a current of blood from the left atrium into the left ventricle.

Aetiology of mitrale stenosis

1. Acute rheumatic fever.
2. Congenital mitrale stenosis (isolated or Lutembacher's syndrome).
3. Infectious endocarditis.
4. Atherosclerosis of the mitrale valve.
5. Malignant carcinoid.
6. Systemic lupus erythematosus.
7. Heart amyloidosis.
8. Narrowing of mitral orifice can be caused by: thrombus of the left atrium, myxoma of the left atrium.

Hemodynamics in mitral stenosis

1. The narrowed mitral orifice serves as an obstacle for expulsion of blood from the left atrium and conducts to its superfluous blood filling, dilatations of sinus of the left atrium and its hypertrophy.
2. The augmentation of pressure in the left atrium leads to retrograde rising of pressure in pulmonary veins and capillaries that invokes spastic stricture of arterioles in lungs — Kitaev's reflex.
3. The spastic stricture of branches of the pulmonary artery produces the development of pulmonary hypertension, overload of right ventricle by pressure, hypertrophy of its walls, and in the subsequent — dilatation.
4. Decompensation on the systemic circulation.

Clinic of mitral stenosis

I. Complaints.

1. Breathlessness on physical exertion.
2. Cough, abjection of small amount of brown or hemorrhagic sputum (pneumorrhagia).
3. Malaise, undue fatiguability.
4. Palpitation on physical exertion, fault in the heart in patients with atrial fibrillation.
5. Pains in heart area.

II. Examination.

1. The mitral face (facies mitralis) on the background of acyanotic colouring of the face skin, cyanochroic colour of cheeks.

2. Heart hump.

3. Pulsing in heart area on the left of the breast bone in II–III intercostals, epigastric pulsing.

III. Palpation.

1. Absence or weakening of apex beat.

2. Diastolic «cat's purring».

3. Heart beat, pulsing in the epigastric area.

4. The pulse in sharply expressed mitral stenosis is small (pulsus parvus), is Popov's sign is revealed decrease of the pulse on the left radial artery (pulsus differens).

IV. Percussion. Borders of relative dullness of the heart are displaced upwards and to the right.

V. Auscultation.

1. The 1st tone on the apex of the heart is strengthened, clapping.

2. The 2nd tone on the pulmonary artery is strengthened, stressed, bifurcated.

3. The opening snap of the mitral valve on the apex.

4. Quail rhythm.

5. There is diastolic murmur in the apex area.

6. In sharply expressed pulmonary hypertension there is diastolic pulmonic murmur (Graham-Steell murmur).

VI. Rentgenological examination.

1. Augmentation of the left atrium, the right ventricle.

2. Dilating of the pulmonary artery.

3. Flattening of the heart owing to protrusion of the 3rd arch on the left.

VII. Electrocardiogram. Signs of hypertrophy of the left atrium and hypertrophy of the right ventricle.

VIII. Echographic signs of mitral stenosis:

— The unidirectional advance of anterior and posterior cusps of the mitral valve (in norm the posterior cusp of the valve in diastole is displaced backward.

— The sinus of the left ventricle is not enlarged.

— Dilating of sinus of the left atrium and the right ventricle.

— Cusps of the mitral valve are dwarfed.

— Cone-like form of the mitral valve.

— Decrease of the area of the mitral orifice.

Diagnostic criteria of mitral stenosis
Direct (valval) signs:

— Clapping 1st tone.

— Opening snap of mitral valve.

— Diastolic murmur.

— Diastolic tremor.

— Echocardiographic signs.

Indirect signs:

1. Left atrium:

— Radiographic and echoscopic signs of hypertrophy and dilatation of the left atrium.

— ECG signs of hypertrophy and dilatation of the left atrium.

2. Pulmonary:

— Breathlessness on physical exertion.

— Attacks of cardiac asthma.

— Protrusion of fulcrum of the pulmonary artery.

— Dilating of branches of the pulmonary artery.

3. Rightventricular:

— Pulsing in epigastrium.

— Rentgeno, echo, ECG signs of hypertrophy and dilatation of the right ventricle.

— Right heart failure.

Mitral insufficiency

It is a heart disease characterized by incomplete closing of cusps of the mitral valve and therefore entering of part of blood from the left ventricle into the left atrium during a systole.

Etiology of relative mitral insufficiency

1. Dilating of the sinus of the left ventricle and its fibrous ring of any genesis (arterial hypertension, faults of the aortal valve, coarctation of aorta, myocardites, dilatational cardiomyopathies, aneurysms of the left ventricle, etc.).

2. Prolapse of the mitral valve — superfluous locomotion of mitral cusps into the sinus of the left atrium during a ventricular systole.

3. Dysfunction of papillary muscles (ischemia, cardiosclerosis, myocardial infarction).

4. Breakage of valval chordas.

5. Calcification of the valval ring (in elderly people), breaking its narrowing during a ventricular systole.

Etiology of organic mitral insufficiency

1. Rheumatic endocarditis — (75 % of cases).

2. Myxomatous transformation of cusps of the valve.

3. Contagious endocarditis.

4. Diffuse diseases of connecting tissue — systemic lupus erythematosus, pseudorheumatism.

5. Atherosclerosis.

Hemodynamics in mitral insufficiency

1. Incomplete closing of cusps of the mitral valve causes anatropic blood flow from the left ventricle into the left atrium during a ventricular systole.

2. The left atrium tests an overload in volume. The walls of it are hypertrophied, the sinus dilatates.

3. The enlarged inflow of blood into the left ventricle produces its dilatation and hypertrophy.

4. The pulmonary hypertension, hyperfunction and hypertrophy of the right ventricle in weakening of the left atrium the pressure in its sinus raises and is retrogradely transmissed to pulmonary veins.

At weakening the left atrium pressure in its sinus raises and retrograde is transferred to pulmonary veins.

Clinic of mitral insufficiency

I. Complaints.

1. Breathlessness on physical exertion**.**

2. Palpitation.

3. Cough is dry or with sputum, frequently with an impurity of blood (hemoptysis).

II. Examination.

1. Cyanosis of lips.

2. Heart beat.

3. The apex beat in the 5th intercostal is outward of the left midclavicular line.

III. Palpation.

1. The apex beat is strengthened, of a raising character, wide, displaced to the left.

2. Heart beat.

IV. Percussion.

The shift of borders of relative dullness of the heart to the left and upwards (dilatation and hypertrophy of the left ventricle and the left atrium).

V. Auscultation.

1. The 1st tone on the apex is weakened.

2. Accent of 2nd tone on the pulmonary artery.

3. Systolic murmur on the heart apex, radiates to the left axillary area or along the breast left edge to the heart basis.

4. Pulse and arterial pressure are not changed.

VI. Radiological examination.

1. The left atrium increase.

2. The left ventricle increase.

3. General sizes of heart are increased.

VII. Electrocardiogram. The left atrium and the left ventricle hypertrophy signs.

Echocardiogram signs of mitrale insufficiency

— Discordance of movements of anterior and posterior cusp's of the mitrale valve.

— Fibrosis of the anterior cusp.

— Increase in the sizes of the left atrium and the left ventricle, thickening of the wall of the left ventricle.

— Increase of diastolic divergences of cusps of the mitrale valve.

Diagnostic criteria of mitrale insufficiency

Direct (valvular) signs: systolic murmur on the apex; easing of 1st tone.

Indirect (haemodynamic) signs: increase in the left ventricle; increase in the left atrium.

The nomenclature of defects of the aortal valve

— Insufficiency of the aortal valve.

— Stenosis of the aorta mouth.

— Aortal defect with prevalence of stenosis of the aorta mouth.

— Aortal defect with prevalence of insufficiency of the aorta valve.

— Aortal defect without accurate prevalence of insufficiency of the aorta valve or stenosis of the aorta mouth.

Insufficiency of the aorta valve

Insufficiency of the aorta valve is a pathological condition in which cusps of the semilunar valve do not close aortal aperture completely and during the diastole there is a return current of blood from the aorta into the left ventricle.

Aetiology of aortal insufficiency

Organic aortal insufficiency:

— rheumatic endocarditis;

— infectious endocarditis;

— calcification of the valve (degenerate process in elderly people);

— trauma of the heart area;

— congenital anomaly;

— aorta atherosclerosis (disputably);

— diffuse diseases of the connective tissue;

— nonspecific aorto-arteritis (Takayasu's disease);

— ankylosing spondylitis (Bekhterev'sillness);

— antiphospholipid syndrome.

Relative aortal insufficiency:

— Degenerate dilatation of the aorta (in elderly people).

— Aneurysm of the aorta of any genesis.

— Marfan's syndrome, Ehlers-Danlos' syndrome.

— Imperfect osteogenesis (Lobshtein's disease).
— Syphilitic mesaortitis.
— Arterial hypertensia.
— Uraemia.

Haemodynamics impairment in aortal insufficiency

1. Regurgitation of blood from the aorta creates a turbulent movement of blood in the zone of valvular defect, protodiastolic murmur arises. Owing to regurgitation in the aorta pressure falls sharply. Similarly pressure in large arteries will vary. High systolic and low diastolic blood pressure are fixed, high pulsation of arteries is expressed.

2. Regurgitation of blood in the left ventricle causes an increase diastolic pressure and its stretching with the subsequent increase in the force of left ventricle contraction; dilatation and hypertrophy of the left ventricle arise.

3. Further on relative insufficiency of the mitrale valve is formed, pressure in the left auricle, then and in the pulmonary circulation raises.

Clinic of aortal insufficiency

I. Complaints.
1. Strong pushes of the heart.
2. Tachycardia.
3. Pulsation of neck vessels and other peripheral arteries.
4. Dizzinesses, episodes of weakness and faints.
5. Pains in heart of angina pectoris type.
6. Cardiac dyspnea.

II. Examination:
1. Paleness of skin.
2. The strengthened pulsation arteries.
3. Musset's symptom.
4. Pulsation of uvula and tonsils (Muller's symptom).
5. Systolic narrowing and diastolic expansion of pupils — Landolfi's sign.
6. Pseudo-capillary Quincke's pulse.
7. A chest vigorous impact in the zone of heart relative dullness.
8. The apex beat is strengthened, displaced to the left and downwards in the 6–7th intercostal space.

III. Palpation:
1. The resistant dome-shaped apex beat in the 6 or 7th intercostal spaces, is displaced to the left.
2. Pulsation of the aorta arch.

IV. Percussion:
1. Heart dullness expansion to the left and downwards.
2. Aortal heart configuration.

V. Auscultation:

1. The 1st tone on the apex is a little weakened.
2. The 2nd tone on the aorta is weakened or absent.
3. The 3rd tone is heard on the apex.
4. Diastolic murmur in Erba-Botkin's point and in the projection place of aortal valve.
5. There is diastolic Austin Flint murmur on the heart apex.

VI. Pulse.

Rapid, fast (pulsus celer), jumping up (pulsus saliens), short, high (pulsus altus), big (pulsus magnus) and tense (pulsus durus).

VII. Arterial pressure:

1. Systolic pressure is raised (up to 160–180 mm Hg).
2. Diastolic pressure is lower 50 mm Hg.
3. Pulse pressure makes 80–100 mm Hg (30–50 mm Hg in norm).

VIII. X-ray examination:

1. The left ventricle sharp increase.
2. The aorta shade is diffusely expanded.

IX. Electrocardiogram. Signs of hypertrophy of the left ventricle.

Echocardiogram signs of aortal insufficiency

— Vibration of the front cusp of the mitrale valve during the diastole period.
— Aortal valve change.
— The left ventricle dilatation and hyperkinesis of its walls.

Diagnostic criteria aortal insufficiency

1. Direct (valvular) signs:

— Diastolic murmur.
— Easing or disappearance of the 2rd tone.

2. Indirect (left ventricular) signs:

— The apex beat is strengthened diffuse, displaced downwards and to the left.
— Relative dullness of heart expansion to the left; the left ventricle increase revealed by radiological examination and echocardiogram.
— Signs of hypertrophy of the left ventricle on ECG.

3. Indirect signs caused by drop of diastolic pressure in the aorta:

— Low diastolic pressure.
— Pains the heart area.
— High rapid pulse.
— Double sound of Traube and double Duroziez's murmur on peripheric arteries.
— Capillary sphygmus.

Stenosis of the aorta ostium

The stenosis of the aorta ostium is subaortic stenosis which creates interrupting to blood flow from the left ventricle in to the aorta.

Etiology of stenosis of the aorta ostium

I. Congenital valval aortal stenosis.
II. Acquired stenosis of the aorta ostium.
— Acute rheumatic fever.
— An age idiopathic degenerative calcification of the aortal valve.
— Atherosclerosis of the valve of the aorta.
— Contagious endocarditis.
— Pseudorheumatism (an infrequent cause).

Impairments of hemodynamics in aortal stenosis

1. In stenosis of the aorta ostium there is interrupting in the path of the blood flow from the left ventricle in to the aorta that leads to rising of systolic pressure in the ventricle.

2. The systole of the left ventricle is extended, the rate of blood-flow through a stenosed aorta ostium is enlarged, systolic murmur is formed.

3. The augmentation of the work of the left ventricle invokes its concentric hypertrophy.

4. In stenosis the pressure in the aorta is reduced.

Clinic of stenosis of the aorta ostium

I. Complaints.

1. Giddinesses, syncopes, dimness in eyes on physical exertion.
2. Palpitation.
3. Pains in the heart area are of anginal character.
4. Breathlessness, attacks of cardiac asthma.

II. Examination.

1. Paleness of skin.
2. Pulsing in the left precardiac area.
3. Shift of apex beat to the left in to the 5th and the 6th intercostal.

III. Palpation.

1. The apex beat is slow, rising, high, resistant, displaced to the left in to the 5th, less often into the 6th intercostal.
2. In 2 intercostal at the left at edge of breast bone - a systolic tremor.

IV. Percussion.

1. The left border of the heart is displaced to the left.
2. Aortal configuration of the heart.

V. Ascultation.

1. The 1st tone on the heart apex is weakened.
2. The 2nd tone on the aorta is weakened or absent.
3. In the 2nd intercostal on the right at the edge of the breast bone there is a rasping, intensive systolic murmur.

VI. Pulse. Slow (pulsus tardus), mild, of a small filling (pulsus mollis), small or low (pulsus parvus seu humilis).

VII. Arterial pressure (AP). Systolic AP is reduced.
VIII. Rentgenological examination.
1. Dilating of the left ventricle.
2. Dilating of the ascending part of the aorta**.**
IX. Electrocardiogram. Signs of hypertrophy of the left ventricle.

Echocardiografic signs of stenosis of the aorta ostium

— Thickening of cusps of the aortal valve.
— Decrease of degree of disclosing of the aortal valve.
— Thickening of the left ventricle wall.
— Augmentation of the left ventricle sinus.

Diagnostic criteria of stenosis of the aorta ostium

1. Valval (direct) signs:
— Systolic murmur on the aorta.
— Systolic tremor.
— Weakening of the 2nd tone on the aorta.
— Change of valves on echocardiogram.
2. Leftventrical signs:
— Strengthened apex beat.
— Dilating of relative dullness of the heart to the left.
— Augmentation of the left ventricle according to roentgenoscopy, echo-cardiographies.
— Syndrome of hypertrophy of the left ventricle on ECG.
3. Signs dependent on drop of cardiac outlier
— Undue fatiguability.
— Headaches, giddinesses.
— Sense of faintness.
— A low systolic pressure.
— Small, slow pulse.

LECTURE 10
SYMPTOMATOLOGY AND DIAGNOSTICS OF ARTERIAL HYPERTENSIONS

Arterial hypertension (AN) is a stable rising of arterial pressure (AP) — systolic from above or equal to 140 mm Hg and-or diastolic up to the level from above or equal to 90 mm Hg on the data not less than double measuring on Korotkov's method on two or more consecutive visits of the patient with interval not less than 1 week.

Types of arterial hypertensions

1. Essential, initial.
2. Symptomatic, secondary.

Essential (initial) AN — chronically proceeding disease of unknown etiology with the ancestral predisposition, arising owing to interaction of genetical factors and environmental factors, characterized by stable rising of AP on the absence of organic lesion of the organs and systems regulating it.

Symptomatic (secondary) AN is rising of arterial pressure, etiologically connected with the definite, as a rule, clinically well determined diseases of the organs and systems participating in the regulation of arterial pressure.

The classification of AP levels (mm Hg) in shown in the table 10.

Table 10 — Classification of arterial pressure levels (mm Hg)

Categories of the AP	Systolic AP	Diastolic AP
Optimum AP	< 120	< 80
Normal AP	120–129	80–84
Categories of the AP	Systolic AP	Diastolic AP
High normal AP	130–139	85–89
Hypertension of the 1st degree of gravity	140–159	90–99
Hypertension of the 2nd degree of gravity	160–179	100–109
Hypertension of the 3rd degree of gravity	> 180	> 110
Isolated systolic hypertension	> 140	< 90

Note.

1. If the levels systolic and diastolic AP correspond to different categories, on the level of AP the given person is referred to a higher category.

2. In isolated systolic hypertension it is also possible to distingwish 3 degrees of gravity due to on the level of systolic AP.

3. The given classification of levels of AP is applicable only to persons who do not receive antihypertensive preparations.

Factors determining the forecast at arterial hypertension

A. Risk factors of cardiovascular diseases which should be considered at assessment of risk.

— Levels of the systolic and diastolic BP (AH of 1st, 2nd and 3nd degrees of gravity).

— Men older than 55 years old.

— Women older than 65 years old.

— Smoking.

— Dyslipidemia (general Cholesterin more than 6,5 mmol/l).

— Indicatings on the precocity of cardiovascular disease in the family hystory (at the age of younger than 55 years old for men or younger than 65 years old for women).

— Transabdominal obesity (waist less than 102 cm for men or less than 88 cm for women).

— C-reactive protein (high level).

B. Lesion of ogans-targets.

— Hypertrophy of the left ventricle (electrocardiography, echocardiography).
— Ultrasonic signs of thickening of the artery wall or the atherosclerotic plaque.
— A slight rise of serumal creatinine (115–133 mcmol/l).
— Microalbuminuria (30–300 mg/d).
— Plasma levels of glucose on empty stomach more than 7,0 mmol/l.
— Plasma levels of glucose after meal more than 11,0 mmol/l.
C. Clinical conditions associated with arterial hypertension.
— Vascular diseases of the brain: ischemic insult, hemorrhagic insult, transient infringement of the cerebral circulation.
— Heart diseases: myocardial infarction, stenocardia, revascularization of coronary arteries, stagnant heart failure.
— Kidney diseases: diabetic nephropathy, renal failure.
— Diseases of the peripheral arteries — obliterating atherosclerosis of vessels of the inferior extremities.
— Severe retinopathy: hemorrhages or exudate of retina, papilledema.
— Diabetes mellitus.
The table risk of complication development in shown in the table 11.

Table — Risk of complication development

Risk factors or diseases	AH of 1st degree	AH of 2nd degree	AH of 3rd degree
There are no risk factors	1st — low	2nd — moderate	3rd — high
1–2 factors	2nd — moderate	2nd — moderate	4th — very high
3 and more risk factors, a lesion of «organs – targets»	3rd — high	3rd — high	4th — very high
Concomitant diseases	4th — very high	4th — very high	4th — very high

Causes of arterial hypertension

1. Family predisposition.
2. Risk factors:
— Superfluous consumption of salt (adequate quantity is 3,5g).
— Insufficient consumption of calcium and magnesium with meal and water.
— Smoking.
— Use of alcohol.
— Superfluous body mass.
— Hypodinamia.
— Low social and economic condition.
— Psyhoemotional stressful situations in combination with character.

Types of symptomatic arterial hypertension

1. Renal:
— Renalparenchymatous develops in glomerulonephritis, pyelonephritis, congenital anomalies and other kidney diseases.
— Renovascular — develops in fibromuscular dysplasia of renal arterias, nonspecific aorto-arteritis, atherosclerosis.
— Renoprival — renectomy.

2. Endocrine — at pheochromocytoma, Cushing's disease/syndrome, initial hyperaldosteronism (Conn's syndrome), hyperthyroidism, acromegalia.

3. Hemodinamic — at the coarctation of aorta, atherosclerosis of aorta, failure of the aortal valve, complete AV-blockage.

4. Neurogenic — at organic lesion of the brain — tumours, bruises, traumas.

5. Exogenous — taking medicinal preparations, products with tryptophan.

Lesion of organ-targets at arterial hypertension

Heart lesion (hypertonic heart).

Hypertonic heart is a complex of the anatomical, biochemical and physiological changes forming in the myocardium in AH development from the beginning of the disease when these changes are not evident, up to the final stage resulting heart failure.

It is characterized by the left ventricle myocardium hypertrophy with the subsequent development of heart failure, cardiac rhythm impairments, atherosclerotic lesion of coronary arteries.

Affected kidneys — «hypertonic nephropathy» («primarily contracted kidney»).

Affected retinal vessels: spastic stricture of arterioles and arteries; retinal apoplexies; exudate in the retina.

Impairments of the brain: acute (ischemic and hemorrhagic insult, transitional ischemic attack); chronic (hypertonic discirculatory encephalopathy).

Clinic of arterial hypertension

Complaints:

— Onset of the disease — general malaise, sleeplessness, pains, heavy headache, difficulty in concentration at work, pain in the heart area, sensation of pulsation in the head, sweating, tremor, buzz in the ears, head noises, flashing of «front sights» before eyes.

— Later — breathlessness on physical exertion, swollen legs.

General examination: hyperemia of face skin, visible pulsation of the head and neck vessels.

Apex beat: positive, wide, displaced to the left, strengthened, resistant, quite often domed.

Borders of absolute and relative heart dullness are displaced to the left, dimension of heart diameter is enlarged, dimension of diameter of vascular bundle is enlarged, enlarged aortal configuration.

Auscultation of the heart: weakening of 1st tone on apex, aortic accent of the 2nd tone, systolic murmur on heart apex.

Changes of eyeground: central retinal artery occlusion, dilated veins of retina; large and fine hemorrhages, exudates; edema of papillas of optic nerves, edema of retina, subitaneous loss of vision.

ECG: signs of hypertrophy of the left ventricle and the left atrium.

Roentgenogram of the chest: elongation and protrusion of the 4th arche of the left contour of the heart (hypertrophy of the left ventricle); aortal configuration; elongation and protrusion of the 1st arch of the left contour (dilatation of aorta).

LECTURE 11
SYMPTOMATOLOGY AND DIAGNOSTICS OF ATHEROSCLEROSIS AND ISCHEMIC HEART DISEASE

Atherosclerosis — the pathological process characterized by deposit of lipids of blood in walls of arteries that is accompanied by formation of the fibrous (atherosclerotic) plaque narrowing the lumen of the vessel.

Risk factors of development of atherosclerosis

A. Modified (can be eliminated).

1. Dislipidemia (hypercholesterinemia, atherogenous hyperlipoprotein emia).
2. Smoking.
3. Arterial hypertension.
4. Obesity.
5. Hypodynamia.
6. Diabetes.
7. Frequent psychoemotonal stresses.
8. Hyperhomocycteinemia.

B. Nonmodified (ineradicable).

1. The burdened heredity.
2. The male, age is older than 60 years.

The classification of Stages of atherosclerotic lesions is shown is the table 12.

Table 12 — Classification of Stages of atherosclerotic lesions (Stary, 1995)

Stage	The characteristic
Stage I — initial lesions	Changes of endothelium, presence of separate foamy cells
Stage II — lipide strips	Clumps of the foamy cells overloaded with lipids, forming stains and strips
Stage III — transitive lesions	Is similar to Stage II, but there are extracellular lipide deposits
Stage IV — atheroma	The big clumps of lipids with formation of a lipide nucleus
Stage V — fibroatheroma	Has a lipide nucleus and a cover
Stage VI — complicated	Breakage (anguish) of a plaque, hemorrhage in plaque, intramural clottages

Stages of atherosclerosis (A. L. Myasnicov, 1965)

1. Initial (preclinical) period — formation of atherosclerotic plaques is observed, lipidemia takes place, but there are no clinical manifestations.

2. The period of clinical manifestations:

I — ischemic stage.

II — thrombonecrotic stage.

III — fibrous stage.

Clinic of atherosclerosis

General manifestations

1. Signs of the expressed senilism.
2. Early canities of hair on the head and thorax (in men).

3. Plural xanthomas.

4. Sign of Franc — erect or diagonal fold on ear.

5. Sign of Gabrioly — a heavy growth of hair on auricles.

6. A senile arch of the eye iris.

Laboratory diagnostics of atherosclerosis

1. General analysis of blood — without changes.

2. Biochemical analysis of blood:

— Augmentation of the level of general cholesterin, triglycerides; cholesterin of low-density lipoprotein (LDL).

— Drop of cholesterin of high-density lipoprotein (HDL).

— Augmentation of atherogenic quotient (AQ): AQ = the general cholesterin – cholesterin of HDL / cholesterin of LDL. Norm 3–3,5.

Tool diagnostics of atherosclerosis

Impairments of blood-flow in arteries, thickening and roughness of walls, decrease of size of the lumen of arteries are revealed with the following methods.

1. Radiopaque angiography.

2. Echoangiography — ultrasonic scanning is applied to examine somnolent and spinal arteries.

3. Magneto-reverberatory angiography.

Ischemic heart disease

Ischemic heart disease (IHD) — an acute or chronic lesion of heart caused by decrease or the arrest of delivery of blood to myocardium in connection with an atherosclerotic lesion of coronary arteries that breaks equilibrium between coronary blood-flow and needs of myocardium for Oxygen.

The syndrome of coronary failure is disharmony of coronary blood-flow to needs of myocardium in Oxygen, leading to the ischemia or necrosis of myocardium and development of cardiosclerosis.

Nonatherosclerotic causes of coronary failure

1. Arteritis (nodous periarteritis, Takaiasu disease, systemic lupus erythematosus, lues, etc.).

2. Traumas of coronary arteries.

3. Spastic stricture of coronary arteries.

4. Embolism of coronary arteries (contagious endocarditis, intracardiac thrombuses implanted valves, etc.).

5. Congenital anomalies of coronary arteries.

6. Other causes — aortal heart diseases, thyrotoxicosis, hypercoagulation, complications of catheterization of heart.

Pathogeny of ischemic heart disease

The need of myocardium for Oxygen depends of:

— Frequencies of cardiac reductions.
— Myocardial contraction.
— Strains of the left ventricle during systole.

Supply of the myocardium with Oxygen depends on the size of coronary blood-flow which is defined by:

— The size of resistance of coronary arteries (diameter, elastance).
— The size of perfused pressure in the phase of diastole (the difference between diastolic pressure in the aorta and diastolic pressure in the left ventricle).

The pathogenetic factors promoting to the development of ischemia of the myocardium

1. Organic narrowing of the lumen of the coronary artery by atherosclerotic process (plaques, units of thrombocytes, thrombuses).
2. The spastic stricture of coronary arteries on the background of atherosclerosis which alters the reactivity of arteries and makes their hypersensitive to the influence of neurogenic stimulants and environmental factors.
3. Drop of ability of coronary arteries to extend adequately under the influence of the metabolites arising in conditions of rising of need of myocardium in Oxygen (adenosine, lactic acid, Inosinum, hypoxanthine).
4. The dysfunction of endothelium caused by atherosclerosis, characterized by predominance of coagulator and vasoconstrictive factors.

Classification of ischemic heart disease

1. Subitaneous coronary death (an initial cardiac arrest).
2. Stenocardia.
2.1. Stenocardia of effort.
2.1.1. For the 1st time arisen stenocardia of effort.
2.1.2. Stable stenocardia of effort (with the indicating of functional class I, II, III, IV).
2.1.3. Progressing stenocardia of effort.
2.2. Spontaneous stenocardia.
3. Myocardial infarction.
3.1. Macrofocal (transmural).
3.2. Microfocal.
4. Postmyocardial infarction cardiosclerosis.
5. Impairments of cardiac rhythm.
6. Heart failure.

Subitaneous coronary death as a form ischemic heart disease

The subitaneous coronary death as a form IHD is death at the presence of the witnesses, which occurred instantly or within 1–6 hours, caused most frequently by fibrillation of ventricles and not connected with the presence of the signs, allowing to establish any another diagnosis, except IHD.

Clinical signs of subitaneous coronary death:

— Loss of consciousness.
— Apnoea.
— Absence of pulsus on carotid arteries.
— Absence of cardiac tones.
— Dilated pupils.
— Acyanotic-grey shade of the skin.

Stenocardia

Stenocardia is the form of IHD characterized with paroxysmal pains in the heart area, caused by ischemia of the myocardium (without development of necrosis).

Signs of stenocardia

1. Character of pains — tightening, berning, pressing.
2. Localization — substernal area or atrial zone to the left of the breast bone.
3. Causes — physical exertion or emotional strain.
4. Duration of pain sensations — 2–3 m (not less than 1 minutes and not more than 20 minutes).
5. Fast and complete stoping effect of Nitroglycerine — in 3-5 m, but not more than 10 minutes.

Stenocardia of effort is the clinical form of stenocardia characterized by transient attacks of pain in the heart area which are causes by physical exertion, stress or other factors raising the need of the myocardium in oxygen (increased BP, tachycardia).

Stable stenocardia of effort (SSE)

SSE is stenocardia existing more than 1 month and described by stereotype pains in the heart area resulting from the same load.

Functional classes (FC) of stable stenocardia:

I class — the patient tolerates well usual physical exertion, attacks of stenocardia arise only at the excessive loads, carried out in a long-term and fast rate. Vigor of the mastered load (W) not less than 750 kgm/min (125 Wt), double product (DP) — not less than 278.

II class — small restrictions of usual physical activity. Attacks of stenocardia arise at walking on a flat surface covering a distance more than 500 m, at rising more than on one floor. W = 450–749 kgm/min (75–100 Wt), DP = 210–277.

III class — the expressed restriction of usual physical activity. Attacks arise at walking in normal rate on a flat surface on a distance less than 100–500 m, at rise on one floor. W = 300–449 kgm/min (50 Wt), DP = 151–209.

IV class — a stenocardia arises at small physical exertion, walking on a flat surface on distance less than 100 m. Originating attacks at rest, in one's sleep typical. W = 150 kgm/min (25 Wt), DP ≤ 150.

Progressing stenocardia

Progressing stenocardia of effort is subitaneous augmentation of frequency, gravity and duration of attacks of stenocardia in reply to a usual load for a patient.

Signs of progressing stenocardia

— Augmentation of intensity and duration of pains.
— Dilating zones of pain irradiation.
— Decrease of exertion causing pain.
— Drop of the effect of Nitroglycerine.
— Appearance of new signs (breathlessness, arrhythmia, weakness, sweating, etc.).
— Stenocardia of effort is accompanied stenocardia on rest.

Spontaneous stenocardia

Spontaneous stenocardia (alternative, vasospastic, Prinzmetal's) is a form of IHD, characterized by stenocardia causes by the spastic stricture of coronary arteries.

Clinical manifestation of spontaneous stenocardia

— Pains are not connected with physical exertion.
— Night and morning attacks of pains.
— Series of 2–5 attacks with intervals of 5–60 minutes.
— Provoking influence of aboriginal cooling and hyperventilation.
— Passing lightly rise of ST on ECG.
— Good acceptability of exercise in the absence of attack.

Loading electrocardiography-Tests

1. Tests provoking ischemia of myocardium by rising oxygen consumption:
— veloergometria;
— tredmil-test;
— transesophageal atrial pacing;
— dobutamin test.
2. Tests provoking ischemia of myocardium by drop of Oxygen delivery:
— test with dipiridamolum;
— test with adenosine.

Other tool methods of ischemic heart disease diagnostics

1. Holter's monitoring — prolonged registration of electrocardiography (ECG) in conditions of free (habitual) for a tested activity with the subsequent analysis of the received findings on the decoder.

2. Scintigraphy of myocardium with 201 Tl. Radioactive thallium is absorbed by the normal myocardium proportionally to the degrees of its blood supply. In constrictive atherosclerosis on the scintigram there are focal defects of accumulation of isotope.

3. Coronary angiography — «the gold standard» of coronary artery lesion and atherosclerosis diagnostics. It is carried out with the help of the angiograph by introduction into of coronary artery ostium of a contrast agent through a catheter with the subsequent roentgenography.

Myocardial infarction

Myocardial infarction (MI) — a form of IHD is characterized by the development of a local myocardium necrosis caused by an acute disharmony of coronary blood-groove to the needs of myocardium.

Etiology of the myocardial infarction

1. Atherosclerosis of coronary arteries and development of clottage in them. This variant of MI is an independent nosological unit, a form of IHD.

2. Nonatherosclerotic lesion of coronary arteries. This variant of MI is not an independent nosological unit, and it is considered a syndrome of other diseases.

Pathogeny of the myocardial infarction

Pathophysiological triad of MI:

— Breakage of atherosclerotic plaque.
— Clottage of a coronary artery.
— Coronary spasm.

Classification of myocardial infarction

1. Depth and extensiveness of necrosis:

1.1. Macrofocal or Q-infarct:
— Transmural (QS).
— Nontransmural (with pathological wave Q).
1.2. Microfocal or non-Q-infarction.

2. Localization:

— MI of the left ventricle.
— MI of the right ventricle.
— MI of the atriums.

3. Periods of MI:

— Prodromal.
— Peracute.
— Acute.
— Subacute.
— Postmyocardial infarction cardiosclerosis.

4. Clinical course features:

4.1. Character of it's development flow.

— Continued MI — recurrence of pain syndrom, ST rise within the 1st 2–3 days of the disease.

— Relapsing MI — originating of new locuses of necrosis within 3–28 days.
— Recurrence MI — originating of new locuses of necrosis after 28 days.
4.2. Typical and atypical forms of MI.
4.3. Complicated or uncomplicated MI.

Periods of myocardial infarction

1. Premyocardial (prodromal) period — can be absent or last from several hours up to about a month. Proceeds as one of the variants of astable stenocardia.

2. The peracute period is the period continuing from the moment of occurrence of coronary occlusion and sharp ischemia caused by it, prior to the beginning of formation of necrosis of the myocardium. Duration from 30 minutes till 2 hours.

Provoking factors of MI development:
— Intensive physical exertion.
— Psychoemotional stress.
— Traumas and surgical interventions.
— The expressed frigorism and overheating.
— Hypoglycemia in diabetes.
— The sexual intercouse.

Atypical forms of MI:
1. Painful atypical forms of MI:
— Transabdominal.
— Peripheric.
2. Painless atypical forms of MI:
— Asthmatic.
— Cerebral.
— Arithmic.
— Hydropic.
— Asymptomatic (erased).

3. The acute period — typically final formation and restriction of necrosis, myomalacia, gradual resorption of necrotic masses and their replacement with quaggy granulomatous tissue. Duration varies — from 2 day up to 14 days.

Signs of resorptional-necrotic syndrome:
1. Fever.
2. Laboratory signs: leukocytosis, augmentation of ESR, biochemical markers of inflammation:
— Rising level of C-RP.
— Rising of seromucoids level.
— Rising of level of Fibrinogenum.
— Rising of level of a2-, γ globulins.
3. Signs of destruction of cardiomyocytes:
— MB-KFK.
— Myoglobin.

— Troponin I.
— AST, ALT.
— LDG1.

4. The subacute period — is characterized by a complete replacement of necrotic masses with granulomatous tissue and formation of cicatrix at the place of necrosis. Duration of the period — 6–8 weeks.

5. The postmyocardial infarction cardiosclerosis period — is characterized by complete strengthening of cicatrix and acclimatization of cardiovascular system to new operating condition.

Duration of the period — 2–6 months.

The topical diagnostics of MI in the left ventricle in shown in the table 13.

Table 13 — Topical diagnostics of myocardial infarction in the left ventricle

Localization of myocardial infarction	Catching abductions
I. MI of anterior wall, including anterior-lateral areas:	
— anterior-septal;	V1, V2 (V3);
— anterior-apical;	V3, V4;
— anterior-lateral;	I, aVL, V5, V6;
— top-lateral;	aVL;
— wide-spread (extensive) anterior;	V1–V6;
— wide-spread (extensive) anterior-lateral	I, aVL, V1–V6
II. MI of posterior wall, including posterior-lateral areas:	
— posterior-septal	II, III, aVF, V1–2;
— posterior-diaphragmal (lower)	II, III, aVF;
— wide-spread (extensive) posterior-lateral	II, III, aVF, V4–V6
III. Circular infarct of the apex	I, II, III, aVL, aVF, V3–V6

Other tool methods of myocardial infarction diagnostics

1. Echocardiography. Akinesia, hypokinesia, dyskinesia of the heart ventricle.

2. Radioisotope scintigraphy of myocardium with 99Tc-pyrophosphate. Radioactive Tc collects only in the zone of necrosis («a hot stain»).

3. MRI, CT. Allow to reveal ischemic zones of myocardium, the sizes of cavities of heart, endocardiac thrombuses, aneurysm of heart.

Acute coronary syndrome

Acute coronary syndrome (ACS) — any combination of clinical signs, forcing to suspect MI or astable stenocardia.

This syndrome includes:
— Myocardial infarction with the rise of segment ST.
— Myocardial infarction without the rise of segment ST.
— Astable stenocardia.

The general anatomic substratum **in basis of ACS** — clottage of various degree of expressiveness of coronary arterias above the anguish of atherosclerotic plaque or anabrosis of endothelium with the subsequent clottage, vasoconstriction and microembolism.

LECTURE 12
THE CIRCULATORY INSUFFICIENCY

The syndrome of circulatory insufficiency is a group various on the mechanism of development of pathological conditions in which the cardiovascular system during short time or gradually slowly loses the ability to provide an adequate blood supply of organs and tissues.

Variants of circulatory insufficiency

1. Heart failure.
2. Vascular failure.
3. Cardiovascular failure.

Heart failure (HF) — is a complex multisystem syndrome in which initial impairment of function of the heart causes a lot of hemodynamic, nervous and humoral reactions directed to the maintenance of the circulation according to needs of the organism which originally have an adaptive, and then — pathological value.

Etiology of heart failure

I. Cardiovascular reasons:

1. IHD.
2. AH.
3. Heart diseases.
4. Cardiomyopathies and myocardites.
5. Arrhythmias.
6. Chronic drunkenness.
7. Constrictive and exudate pericardites.
8. Infectious endocarditis.

II. Exocardial reasons:

1. Diseases of organs of respiration with the pulmonary hypertension.
2. Diseases of thyroid gland.
3. Diffuse diseases of connecting tissue.
4. Infiltrative defeats of myocardium in sarcoidosis, lardaceous, hemochromatosis.
5. Anemic syndromes.
6. Radial therapy on the area of mediastinum.
7. Deficiency of selenium and avitaminosises.
8. Cardiotoxic action of medicinal preparations.

Pathogeny of heart failure

The basic mechanisms:

1. Primary impairment of the myocardium (primary myocardial insufficiency): ischemic heart disease, myocarditis, dilated cardiomyopathy, myocardium dystrophy.

2. Resistance with overload (postloading increase) — a systolic overload of heart comparments: arterial hypertention, aortic ostium stenosis, pulmonary artery stenosis, aorta coarctation, mitral stenosis.

3. An overload with volume (preloading increase) — diastolic overload of the heart comparments as a result of over flow of superfluous quantity of blood to them: insufficiency of heart valves, defects of the heart septum, open arterial (Botallo's) duct, increase in the volume of circulating blood (glomerulonephritis, renal insufficiency, introduction of great volumes of liquid).

4. Decrease in filling the ventricles with blood (diastolic insufficiency): myocardium hypertrophy (hypertrophic cardiomyopathy), cardiosclerosis, amyloidosis, sarcoidosis, hemochromatosis, constrictive and exudative pericarditises, isolated mitral stenosis, tachyarrythmia.

Classification of heart failure

According to the period of development:

— Acute heart failure.

— Chronic heart failure.

According to localisation of a pathological process:

— Left-side (left ventricular, left atrial).

— Right- side (right ventricular, right atrial).

— Total (is left-right-sided, congestive, biventricular).

Acute heart failure

Acute heart failure — rather fast, quite often sudden development of heart failure which reaches such a degree of manifesation that leads to a number of organs failure and represents a direct threat to the patient's life.

Variants:

— Acute left ventricular (left atrial) insufficiency.

— Acute right ventricular (right atrial) insufficiency.

— Total insufficiency.

Acute left ventricular insufficiency

Acute left ventricular insufficiency — is quickly, at times suddenly developed, sharply manifested disfunction of the left ventricle of the heart, causing blood congestion in the pulmonary circulation, cardiac asthma and an alveolar hypostasis of the lungs.

Left ventricular insufficiency aetiology

1. Myocardial infarction, stenocardia.
2. Arterial hypertension.
3. Diffuse myocarditis.
4. Aortic heart disease.
5. Mitral stenosis (left atrial insufficiency).
6. Myocardium dystrophies (alcohol, thyrotoxic and others).
7. Cardiosclerosis (atherosclerosis, postmyocardal and others).

8. Excessively physical activities.
9. Transfusion of great volumes of liquid.
10. Acute decrease of urine output.

Acute right ventricular insufficiency

Acute right ventricular insufficiency — is sharply occurred manifested dysfunction of the right ventricle (atrium) of the heart, causing congestion of blood in the venous part of systemic circulation.

Acute right ventricular insufficiency aetiology

I. Acute pulmonary hypertension (acute pulmonary heart):
— Severe thromboembolia of the pulmonary artery branches.
— Persistent attack of bronchial asthma or an asthmatic condition.
— Pneumothorax, especially valvular.
— An extensive pneumonia.
II. The acute overload of the right ventricle in volume:
— Myocardial infarction with the rupture of the ventricular septum and sharply occurred blood shunting the from left to the right.
— Infectious endocarditis of the tricuspid valve with acute tricuspid insufficiency development.

Classification of chronic heart failure (N. D. Strazhesko and V. H. Vasilenko, 1935)

1st stage of **chronic heart failure (CHF)**. The initial, latent blood circulation insufficiency, with symptoms appearing only on physical activity.
2nd stage of **CHF**. A manifested long term blood circulation insufficiency, haemodynamics impairment (congestion in pulmonary and systemic circulation).
The period A. Haemodynamics impairments only in one pulmonary or systemic circulation.
The period B. Vivid haemodynamics impairments involving the whole of the cardiovascular system.
3rd stage **CHF**. Final, dystrophic with severe impairments of haemodynamics, proof impairments of metabolism and irreversible changes in structure of organs and tissues.

Classification of chronic heart failure (NYHA, 1964)

1. FC — asymptomatic dysfunction of the left ventricle of the heart, without restriction of physical activity.
2. FC — mild chronic heart failure. Patients with heart diseases causing a slight restriction of physical activity. In everyday life there is palpitation, weakness, dyspnea.
3. FC — average degree of chronic heart failure. Patients with an expressed restriction of physical activity. Palpitation, weakness, dyspnea are caused by small physical exertion.
4. FC — severe CHF. Patients has complaints at rest.

The 6-minutes test of walking

Within 6 minutes a patient can pass in a convenient rate the following distances:
— 0 FC — 551 m and more.
— 1 FC — 426–550 m.
— 2 FC — 300–425 m.
— 3 FC — 150–300 m.
— 4 FC — less than 150 m.

Pulmonary hypertension

Pulmonary hypertension is a pathological syndrome characterized by BP rise in the pulmonary arteries.

Forms of the pulmonary hypertension according to localization of disturbance blood-flow in the pulmonary circulation:

1. Artery-arteriol form (precapillary).
2. Capillary form.
3. Venule-venous (postcapillary) form.
4. Arteriovenous form.
5. Obscure forms.

Pulmonary heart

Pulmonary heart — is hypertrophy and/or dilatation of the right ventricle of the heart, owing to hypertension in the pulmonary circulation, caused by lungs diseases, deformation and impairments of chest motility or lesion of pulmonary vessels.

Etiology of the pulmonary heart

1. Diseases primarily affecting airways and lung parenchyma.
2. Diseases primarily affecting the chest and causing its deformation and motility restriction.
3. Diseases primarily affecting pulmonary vessels.

Types of the pulmonary heart

Accordine to the rates of development:

— Acute — arises within several minutes to several o'clock (tromboembolia of the pulmonary artery branches, valval pheumothorax, asthmatic condition, etc.).

— Subacute — arises within several weeks to several months (recurrent embolia of fine pulmonary artery branches, alveolites, etc.).

— Chronic — it is formed within years, decades (COPD, bronchial asthma, kyphoscoliosis, Bekhterev's disease, etc.).

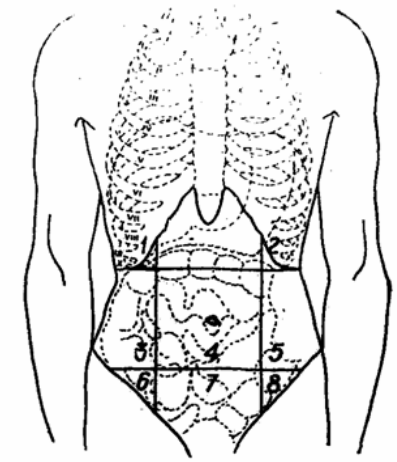
Under indemnification:

— Compensated — there are no signs of heart failure.

— Decompensated — there are signs of heart failure.

LECTURE 13
INQUIRY AND EXAMINATION OF PATIENTS WITH DISEASES OF GASTROINTESTINAL TRACT. SUPERFICIAL AND DEEP PALPATION OF THE ABDOMEN

The topographical areas of gaste are shown in the picture 2.



Picture 2 — Topographical areas of gaste

Epigastric area: 1 — Subcostal areas; 2 — Epigastric area; *Mesogastric area:* 3 — Iliac areas (flanks); 4 — Umbilical region; Hypogastric area: 5 — Inguinal areas; 6 — Suprapubic area.

Classification of patients complaints with diseases of gastrointestinal tract (GIT)

I. Local complaints:

1. «Esophageal» complaints: dysphagia, esophageal pain, esophageal bleeding, esophageal vomiting.

2. «Gastric» complaints: stomach pain, stomach discomfort, stomach dyspepsia, stomach bleeding.

3. «Intestinal» complaints: intestinal pains, intestinal dyspepsia, impairments of defecation, intestinal bleeding, intestinal vomiting.

II. General complaints:

1. Appetite impairments.
2. Changes of taste.
3. Growing thin.

III. Additional general pathological complaints:

1. Undue fatiguability.
2. Drop of work capacity.
3. Muscular delicacy.
4. Neurotic distresses.
5. Signs of intoxication, etc.

Dysphagia (from Greek *dys* — «impairment», «phagein» — «to swallow», «to eat») is impairment of the act of swallowing and transition of food along the esophagus owing to narrowing of lumen of esophagus or its peristalsis impairment.

The causes of organic narrowing (stenosis) of the esophagus

— Foreign body in the esophagus.
— Tumours of the esophagus.
— Cicatrix of the esophagus.
— Pressure upon the wall of esophagus of food which has got stuck in diverticulum.
— Prelum of esophagus with the tumour of mediastinum, aortic aneurysm, abnormal vessels, pericardiac exudate, the enlarged thyroid gland.
— Achalasia of cardial department of the esophagus.

The causes of functional narrowing (spastic stricture) of the esophagus

1. Spastic stricture of the esophagus musculation: the reflex irritations, starting with other organs (mediastinum, gall bladder, intestines and stomach); impairments of vegetative innervation of the esophagus muscular lauer; neurose; tetanus.
2. Impairments of the esophagus peristalsis: myasthenias; botulism; dermatomyositis; scleroderma.

Signs of organic dysphagia:

— dysphagia is constant, it gradually and continuously strengthens;
— the swallowing of hard food primarily impairs, on progressing of stenosis there are difficulties in reception of cereal-like food, and further on in fluid reception.

Signs of functional dysphagia:

— dysphagia is paroxysmal, the periods of dysphagia can be alternated with the periods of normal swallowing;
— it is provoked by smoking, hot and cold food, alcohol, spicy food, stress;
— the hard food is swallowed easier, than fluid (paradoxical dysphagia);
— after the introduction of spasmolytic and cholinolytic agents dysphagia decreases.

Eructation is a sudden, sometimes sonorous consensual abjection into the oral cavity of the stomach contents.

Types of eructation:

— air eructation or empty eructation (eructatio);
— food eructation (regurgitatio) — small vomiting.

Heartburn (pyrosis) is a sensation of fever or a burning sensation on the course of the esophagus, in the substernal or epigastric areas caused by the hit of stomach contents into the esophagus.

Signs of esophageal vomiting

1. It occurs without a preliminary nausea, it is preceded with the sensation of food delay in the esophagus.
2. It arises right after meal.
3. Vomitive masses are not volumetric, consist of undigested food, have no acidic taste and odour.
4. In big diverticula of the esophagus vomitive masses can consist of the food taken long time ago and has a putrefactive odour.

The causes of esophageal bleedings

— Breakage of varicose dilated veins of the esophagus.
— Mellori-Veis syndrome (breakage of mucosa of the cardial department of the esophagus on vomiting).
— Breaking up tumour of the esophagus.
— Ulcers of the esophagus.
— Traumas of the esophagus.
The basic sign of esophageal bleedings is vomiting with not changed blood.

Variants of «stomach pains»

1. Spastic pains (spastic stricture of smooth musculation of the stomach).
2. Distensive pains (distention of the stomach wall with its contents).
3. Irritation of intramural ganglia of the stomach.
4. Ventroptosis (gastroptosis).
5. Perigastritis — adhesive process around the stomach.

Signs of «stomach pains»

1. Localization — epigastric area.
2. There is a distinct connection with the reception of food.
3. Seasonal prevalence of pains is characteristic.

Dyspepsia (from Greek *dis* — «impairment», *pepsis* — «food») is a groop of signs (syndromes) arising owing to impairment of processes of digestion.

Types of dyspepsia:
— Gastric.
— Intestinal.
— Hepatic.
— Pancreatic.
— Of a mixed character.

Gastric dyspepsia is a pathological syndrome caused by insufficient or supersecretion of hydrochloric acid and pepsin, excessively fast or sharply time-lapse evacuation of food from the stomach.

Signs of stomach dyspepsia

1. Gastric discomfort is unpleasant sensation in the epigastrium which is not regarded as pain by the patient, it has a set of shades:

— The feeling of overfilled stomach after meal.
— The feeling of gravity in the epigastrium.
— Early saturation.
— Inflation in the upper half of the abdomen.
— Nausea.
— Rumble, sensation of splash, etc.

2. Nausea is a burdensome pressure in anticardium and simultaneously an unpleasant sensation in oral cavity, accompanied with paleness of the skin, giddiness, hypersalivation, cold snap of extremities, drop of arterial pressure and, sometimes, semisyncopal state.

3. Vomiting is the complex reflex act caused by exaltation of the vomitive center during a stomachal contents ejects consensually through a mouth (less often — and through nasal courses).

Pathogenetic variants of vomiting

1. Central (nervous, cerebral) vomiting.
2. Hematogenous toxic vomiting.
3. Peripheric (visceral) vomiting.

Signs of central vomiting

— Appears suddenly without previous nausea.
— It has no connection with taking meals.
— Vomitive masses are scanty, without any odour.
— Does not give the patient a relief.
— Does not stop after empting the stomach.
— There are no other signs of the GIT diseases.
— There are signs of CNS impairments — headaches, giddiness, consciousness disturbances.

Causes of stomach vomiting

— Inflammation of the stomach mucosa.
— Affecting the stomach with stimulating chemical matters, drugs, toxins, rotten products.
— Spastic stricture of the gatekeeper (pylorospasm).
— Organic stricture of the gatekeeper (pylorostenosis).

Causes of stomach bleeding

— Peptic ulcer and acute stomach ulcers.
— Erosive lesions of the stomach mucosa.
— Carcinoma of the stomach with disintegration of the tumour.

— Mellory-Veis's syndrome.
— Traumas and burns of the stomach.
— Hemorrhagic diathesis.

Basic manifestations of the stomachal bleeding: vomiting, brown emesis (colour of «coffee grounds»), black feces (melena).

Variants of intestinal pains

1. Spastic pains.
2. Distensional pains.
3. Mesenteric pains caused by the intestine mesentery lymph nodes inflammation.
4. Irritation of intramural nerve endings by inflammation, cicatrix, tumour.
5. Ischemic pains caused by impairments of circulation in the intestine (atherosclerosis, clottage, embolisms).
6. Adhesive pains caused by adhesive desease of the intestine.

Differences between intestinal and stomach pains

1. Localization is more often in the inferior and lateral areas of the abdomen, paraumbilical area.
2. They do not depend on taking food.
3. There is no dependence on defecation (can arise before, during and, less often, after defecation).
4. Decrease of pains after defecation or passage of flatus.

Features of distension pains

1. Periodicity is not characteristic.
2. Accompanied by abdominal distention (meteorism).
3. Long-term, gradually become dull during a long continuous abdominal distention.
4. Whining, monotonous.
5. Localized.

Features of spastic pains (colics)

1. Paroxysmal pains, as repeated attacks — scrambles.
2. Begin and finish suddenly.
3. Intensive, stinging.
4. Quickly change localization.
5. More often pains localize in the paraumbilical area.
6. Decrease after heating, taking spasmolytics.

Intestinal dyspepsia is impairment of the digestion in the intestine as a result of enzimal failure in intestine, pancreas, liver or accelerated passage of meals in the intestine, and also owing to dysbacteriosis.

Signs of intestinal dyspepsia

1. Meteorism (from Greek *meteorismos* — «raising upwards») is the abdominal distention arising owing to superfluous formation of gases in the digestive tube (flatulencia), impairment of adsorption and passing.

Causes of meteorism:

1) impairment of digestion owing to enzimal failure of the stomach, intestine, pancreas;

2) dysbacteriosis;

3) aerophagy;

4) inflammation and atrophy of the intestinal mucosa with impairment of adsorption of gases.

2. Rumble in the abdomen (borborygmi) are hums and splash, arising from interference of gases and fluid at their simultaneous passing through narrowing in the intestine.

3. Disorders of defecation:

1) Diarrhoea (from Greek *diarrhea* — «efflux») is augmentation of volume of the feces masses more than 250 g daily, as a rule, with frequency more than 3 times a day, discharging in nonplastic, fluid or semifluid form with changed physico-chemical properties.

Causes of diarrhoea

Diseases of:

— intestine (intestinal);

— stomach (gastrogenic);

— pancreas (pancreatogenic);

— liver (hepatogenic);

— endocrine glands (diabetes, Addisonis disease, thyrotoxicosis) — endocrine;

— metabolic disorders (uremia, lardaceous) — metabolic;

— neurosises (neurogenic);

— taking drugs (medicamental).

Mechanism originating of diarrhoea:

— Accelerated peristalsis in the intestine (with fast transit of its contents).

— Decrease of adsorption of water and electrolit in the intestine.

— Increased transsudation in lumen of the intestine.

— Intensified myxopoiesis.

— Impairment of scission of the food components up to products which can be reabsorbed in the intestine.

Signs of enteral diarrhoea:

— Rather rare (4–6 times a day) evacuation of the intestine.

— Painless evacuation of the intestine.

— Polyexcrements owing to the insufficient intestinal adsorption (malabsorbtion) or intestinal digestion impairment (maldigestion).

Signs of colitic diarrhoea:

— Frequent (10 and more times a day) evacuation of the intestine.

— Small volume of excrements.

— Mucous and blood in feces.

— False feeling defecation.

— Tenesmus.

2) Constipation (obstipation) is evacuation of intestine of 3 and less times a week, the act of defecation being accompanied by additional efforts, secretion of nonplastic condensed feces which does not give satisfaction.

Causes of constipations:

1. Dietary habits (low-fiber diet, restriction of fluid) — alimentary constipations.
2. Intestinal dyskinesia — atonic or spastic constipations.
3. Hypodynamia.
4. Inflammatory processes in the intestine — inflammatory constipations.
5. Mechanical obstacles — tumour, cicatrical strictures, dolichosigma, megacolon — mechanical constipations.
6. Intoxications (lead, nicotine) — toxic constipations.
7. Endocrine diseases (hypothyroidism, acromegalia) — endocrine constipations.
8. Imbalance of water and salt exchange (dehydration).

Appetite (from an armour *appetites* — «desire») is the sensation connected to food requirement.

Variants of impairments of appetite:

1. Complete loss of appetite (anorexia).
2. Drop of appetite.
3. Kept appetite.
4. Rising of appetite (polyphagia).
5. Distortion of appetite.

Taste is the sensation arising on the influence of various solvable matters on gustatory receptors, mainly posed in the tongue.

Changes of taste

— Unpleasant taste in the mouth.

— Obtusion of gustatory sensations.

— Absence of gustatory sensations.

General principles of objective examination of abdomen

1. The general physical examination of abdominal cavity organs begins with examination of the abdomen, consistently passing to palpation, percussions and in rare cases to auscultation.

2. Physical examination is carried out in two positions of the patient - standing and lying.

3. The examination of the abdomen should be carried out on good illumination at daylight or artificial white light.

4. The light source should be behind of the doctor's back and illuminate the completely naked abdomen in regular intervals.

5. On examination of the patient in a lying position the doctor should sit in front of the patient.

6. On examination of the abdominal cavity in a lying position the patient should lie on a semihard couch.

Examination of oral cavity

Examination of oral cavity allows to estimate: state of teeth, state of mucosas, state of the tongue, the odour from the mouth.

Diagnostic value of changes of oral cavities and pharynx mucosa

1. The chronic inflammation of palatine tonsils and mucosa of pharynx is the locus of infectious contamination in the organism.

2. Decondensation of gums and bleeding from them is a sign of hypovitaminosis C.

3. Inflammatory and dystrophic changes of the tongue are shown as features of its surface and the fur on it:

— Wet and pure tongue is a sign of uncomplicated duodenum ulcer, pyloric part of the stomach.

— Tongue coated with a gray-white fur is a sign of acute gastritis.

— Dry tongue with a dirty — grey fur is a sign of peritonitis, acute pancreatitis, cholecystitis, deaquation of the organism.

— Tongue with flattened papillas (varnished) is a sign of autoimmune gastritis, vitamin — B12-, folic-, iron deficient anemias.

— «Geographical», with the inflamed fields tongue (Hunters glossitis) is a sign of vitamin — B12- deficient anemia and parasitizing of tenial helminth — wide fishworm.

Changes of the form of the abdomen are

—Retraction (pulling into).

— Diverticulum (projection).

The causes of symmetric diverticulum of the abdomen are

1. Obesity.
2. Meteorism.
3. Ascites.
4. Pregnancy.
5. Loose-hanging abdomen is uniform diverticulum of the abdomen in the inferior departments owing to delicacy of muscles of prelum abdominale (women who gave birth many times).
6. Overstretch of the urinary bladder on its overflow with urine — diverticulum in the inferior departments of the abdomen.

Distinguishing signs of ascites, meteorism and obesity

Ascites:

1. The skin of the abdomen is thin, nitidous.
2. The umbilicus projects, there can be umbilical hernia.
3. Diastases of direct muscles of the abdomen can be observed.
4. Dermal folds in the inferior departments of the abdomen («apron») are absent.
5. Augmentation of the abdomen is uniform (in initial stages the inferior part projects).
6. In a prone position the abdomen is «froggy», in a standing position — loose-hanging.
7. On percussion of the abdomen — in the center — tympanic note, in sloping places — blunt.

Obesity:

1. Skin of usual characteristics, strips of distention in lateral and inferior departments can be revealed.
2. The umbilicus is pulled inward.
3. In the inferior departments of the abdomen in a standing position — there are skins folds («apron»).
4. The dull thympanitis is percussionly revealed above all the areas of the abdomen.

Meteorism:

1. Skin of usual characteristics.
2. The umbilicus is pulled inward.
3. Dermal folds in the inferior departments of the abdomen are absent.
4. In a standing position the abdomen is not loose-hanging.
5. There can be a non-uniform protrusion of the abdomen.
6. The tympanic note is percussionly revealed above all the areas of the abdomen.

Dissymmetric protrusions of the abdomen:

There are consequences of the following pathological conditions:

1. Augmentations of abdominal cavity organs (liver, spleen, kidneys, pancreas).
2. Tumours of abdominal cavity organs.
3. Cysts of abdominal cavity organs (ovaries, pancreas, liver).
4. Distention of the stomach (pylorostenosis) or loops of intestine (an intestinal obstruction).

Symmetric retraction of the abdomen:

1. Sharp general exhaustion of patients (cancer cachexia, pituitary cachexia).
2. Exhausting diarrheas.
3. Pernicious vomiting.
4. Stenosis of cardial department of the esophagus and the pylorus.
5. Tubercular meningitis, lead colica (scaphoid abdomen).
6. Sometimes — in wide peritonitis.

Signs of pathological peristalsis

— It is visible above the place of obstruction.
— Local.
— It is frequently accompanied by a loud rumble.
— It is visible through a normal abdominal wall.
— It is caused by a mild beating on anterior abdominal wall.
— Waves of antiperistalsis (an intestinal obstruction) can take place.

Other signs determined on examination

1. Hernial diverticulum.
2. Phlebectasia of the anterior abdominal wall — «a head of the Jellyfish» — a sign of a portal hypertension, the syndrome of inferior vena cava.
3. Lipomas, other tumours and metastases of tumours.
4. Edemas (diseases of kidneys, heart failure).
5. Postoperative cicatrixes.

Palpation (from an armour *palpatio* — «palpation») — is a clinical method of examination by means of taction, muscular and regional (stereometric) senses with the purpose of studying of physical properties of tissues and organs, topographical interrelations among them, their sensitivities and detection of some functional phenomena in the organism.

General rules of palpation of the abdominal cavity

1. The investigating person should sit on the right of the patient, facing him.
2. The seat of chair the examining person sits on should be settled down at the level of patient's bed.
3. The examining person should take the most convenient for carrying out of examination position, allowing to provide the least strain of muscles, the maximal volume of motions of hands.
4. The hand of the examining person should be warm, nails should be trimmed short.
5. The patient should lay flat on his back on a rigid bed with weakened musculation.
6. The head of the being examined person should rest on a low pillow, and, if it is possible, it is better without it.
7. The legs and head of the being examined person should not rest against the backrest of the bed, and overhang from the bed, legs should lie straight or slightly bent in knee joints.
8. The being examined person should breathe smoothly and quietly, it is better through his mouth with his abdomen, that reduces strain of the abdominal wall. If the patient is not able to breathe with his abdomen, he should be taught it.
9. The abdomen of the being examined person should be naked from xiphoid process up to pubis.

10. Palpation should be begun with the painless not affected with pathological process area.

11. In case of hypersensibility of being examined person to palpation, his attention should be distracted with conversation.

12. Palpating motions should be as mild as possible. Any effort of motion should be gradual in order to prevent reflex spastic stricture of musculation. If possible the palpation should be painless.

The superficial palpation of the abdomen allows

1. To determine the degree of strain of muscles of the abdominal wall.

2. To determine the presence of tenderness of the whole abdominal wall or its separate fields.

3. To distinguish the puffiness of abdominal wall from accumulation of adeps or from its strain in it in ascites or meteorism (in puffiness — there are dents of dactyls).

4. To reveal apostatis (diastases) of direct muscles of the abdomen.

5. To reveal inspissation, units, metastases of tumours, hernial diverticula in the abdominal wall.

6. To distinguish tumours of the abdominal wall from tumours inside the abdominal cavity (if to ask the patient to strain his abdomen, intra-abdominal tumours cease to be palpated).

Differences of resistance of the abdominal wall from its muscular strain (defans, muscular protection)

1. In muscular defans the strain of the abdominal wall reaches the great degree, at perforation of the ulcer of stomach it reaches almost stone density (abdomen is like a board).

2. In muscular strain even the superficial palpation is accompanied by sharp tenderness that is not present in resistance.

3. In resistance of the abdominal wall it is frequently possible to achieve disappearing of resistance by derivation of the patient by conversation or at long mild stroking of abdominal wall. It does not happen in muscular strain.

The deep palpation allows

1. To make topographical differentiation of organs of the abdominal cavity from each other.

2. To determine: size, form, position, character of surface, consistence, tenderness (sensitivity), motility of organs of the abdominal cavity, property of the wall and the character of contents (for hollow organs).

Ways of deep palpation of the abdomen

1. Hands with slightly bent fingers are placed on the area of being palpated organs perpendicularly its longitudinal axis (installation of the hand).

2. Formation of a dermal fold.

3. Dipping of the hand during several expirations up to the back wall of the abdominal cavity.

4. Sliding along the organ in the direction of transversal axis of the organ.

The order of palpation of organs of the abdominal cavity (N. D. Strazhesko)

1) sigmoid intestine;
2) caecum;
3) appendix;
4) final piece of the ileal intestine;
5) ascending department of the colon;
6) descending department of the colon;
7) transversal colon;
8) hepatic and splenic flexures of the colon;
9) big curvature of the stomach;
10) pylorus;
11) pancreas;
12) the edge of the liver;
13) spleen;
14) kidney.

LECTURE 14
SYMPTOMATOLOGY AND DIAGNOSTICS OF CHRONIC GASTRITIS AND PEPTIC ULCER OF THE STOMACH AND DUODENUM. SYMPTOMATOLOGY AND DIAGNOSTICS OF INTESTINE DISEASES

Chronic gastritis (CG) is a collective concept, uniting various on etiology and pathogeny inflammatory and degenerative lesions of mucosa of the stomach, characterized by its cellular infiltration, impairments of physiological regeneration of epithelium and there of by an atrophy of glandular epithelium, its metaplasia, disorders of secretory, motor and incretory functions of the stomach (P. J. Grigorjev, A. V. Vdovenko, 1998).

Etiology of chronic gastritis

1. Infection *Helycobacter pyloris* (H. pyloris).
2. Autoimmune factor.
3. Duodenum-gastric reflux.
4. Treatment with gastrotropic preparations (Salicylases, other nonsteroid anti-inflammatory preparations — Indomethacinum, diclohpenac; potassium Sodium chloridum, Reserpinum, antituberculous remedies).

5. Other factors (provoking exacerbation):

— Nutritional errors.

— Abuse with alcohol.

— Smoking.

— Professional harmfulnesses.

— Internal causes — persistent infections, hypercorticoidism, gout, deficiency of iron and vitamin B12, hypoxia, renal failure, etc.

Classification of chronic gastritis («the Sydney system», 1990)

I. On etiology.

1. Gastritis associated with *H. pyloris*.

2. Autoimmune gastritis.

3. Reactive (chemical) gastritis caused by duodenum-gastric reflux and reception of medicinal preparations.

4. Special forms of gastritis:

— granulematous (in Crohn's disease, sarcoidosis, tuberculosis and foreign bodie of the stomach);

— eosinophilic, as a possible manifestation of allergy;

— lymphocytic — the nature of it is not clear (probably, *H. pyloris* is guilty, intolerance of gluten — Gee's disease).

II. In dependence on topography of inflammatory process in the stomach:

— gastritis of antrum;

— gastritis of the stomach body;

— pangastritis.

III. Morphological types:

— Nonatrophic.

— Atrophic.

— Special forms.

Clinic of chronic gastritis, associated with Helycobacter pyloris

1. Pains in the epigastrium, more often late, night.

2. Gastric dyspepsia (acidic eructation, heartburn; nausea, vomiting (seldom)).

3. Palpatory tenderness in the epigastriums.

Methods of diagnostics of chronic gastritis, associated with Helycobacter pyloris

1. FGDS (signs of inflammation of mucosa and anabrosis in the antral department) with biopsy of mucosa.

2. Research of the gastric secretion (rising of secretory function of the stomach).

3. Diagnostics of infection contamination *H. pyloris* (urease test, microscoping of mucosa, Unguenta — impresses, C-urease test).

Features of autoimmune gastritis

1. Family character of disease.
2. Pain syndrome is rare, the stomach discomfort is more often marked.
3. Achylic diarrheas and sprue.
4. Vitamin — B12-deficient, iron-deficient anemias.
5. FGDS — atrophy of mucosa of stomach body.
6. Drop of secretory function of the stomach.
7. Hypergastrinemia.

Peptic ulcer is chronic relapsing disease in which as a result of impairment of regulating nervous and hormonal mechanisms and disorders of stomach digestion the ulcer in the stomach or duodenum is formed.

Etiology of peptic ulcer

— Stresses.
— Heredity.
— Abuse with alcohol.
— Smoking.
— Infection contamination *H. pyloris.*
— Reception of medicinal preparations (Aspirinum, corticosteroids).

Pathogeny

The predominance of factors of aggression over factors of protection is shown in the table 14.

Table 14 — Predominance of factors of aggression over factors of protection

Factors of aggression	Factors of protection
— Hyperproduction of HCL — Autoimmune aggression — *H. pyloris* — Proulcerogenic nutritional factors — Duodenum-gastric reflux	— Optimum circulation — Antiulcerogenic nutritional factors — Synthesis of protective prostaglandina, endorphins — Mucosal-bicorbonate barrier

Classification of peptic ulcer

1. On localization: stomach ulcer, duodenum ulcer, combined ulcer.
2. On the dimensions (small, average, big, huge).
3. On stage (an exacerbation, a remission).
4. Complications.

Clinic of peptic ulcer

— Pain;
— Heartburn;
— Eructation;
— Vomiting and nausea.

Diagnostics of peptic ulcer

1. FGDS.
2. X-ray examination.

Complications of peptic ulcer

1. Bleeding.
2. Perforation.
3. Penetration.
4. Stenosis.
5. Malignancy.

Perforation of ulcer

1. Period of the pain shock (a «knife-like» abdominal pain, drop of BP, strain of muscles of abdominal wall).
2. Period of imaginary well-being.
3. Peritonitis.

Bleeding

1. Hematemesis.
2. Melena.
3. Hemorrhagic shock.

Functional diseases of GIT are various combinations of chronic or relapsing signs which are not explained by structural or biochemical changes.

The 3rd Roman classification of functional disorders of gastrointestinal tract

A. Esophagus disorders.
B. Gastroduodenal disorders.
C. Intestinal disorders.
D. Functional abdominal pains.
E. Biliary disorders.
F. Anorectal disorders.
G. Children's functional disorders.

Classification of functional disorders of the intestine

C1. Irritable bowel syndrome
C2. Functional abdominal distention.
C3. Functional constipation.
C4. Functional diarrhea.
C5. Nonspecific functional disorders of the intestine.

Irritable bowel syndrome (IBS) is a functional GI disorder characterized by abdominal pain and altered bowel habits in the absence of specific and unique organic pathology.

The **Rome III criteria** (2006) for the diagnosis of IBS require that patients must have recurrent abdominal pain or discomfort at least 3 days per month during the previous 3 months that is associated with 2 or more of the following:

— Relieved by defecation.
— Onset associated with a change in stool frequency.
— Onset associated with a change in stool form or appearance.

Supporting symptoms include the following:

— Altered stool frequency.
— Altered stool form.
— Altered stool passage (straining and/or urgency).
— Mucorrhea.
— Abdominal bloating or subjective distention.

Inconsistent symptoms are alert to the possibility of organic pathology. Symptoms not consistent with irritable bowel syndrome include the following:

— Onset in middle age or older.
— Acute symptoms: IBS is defined by chronicity.
— Progressive symptoms.
— Nocturnal symptoms.
— Anorexia or weight loss.
— Fever.
— Rectal bleeding.
— Painless diarrhea.
— Steatorrhea.
— Lactose and/or fructose intolerance.
— Gluten intolerance.

Functional constipation is a group of functional disorders which are manifested as persistent labored, infrequent or apparent incomplete defecation.

Functional diarrhea, as a rule, is often, more than 2–3 times a day emptying of intestines with abjection of fluid or cereal-like excrements.

Inflammatory diseases of intestines

Noncontagious enterites and colites are a group of various on etiology and pathogeny of inflammatory — degenerative diseases, more often of mucosa of small intestine and (or) colon with impairment of all functions of intestines.

Classification of inflammatory diseases of intestines

The international classification of diseases

— K50 Crohn's disease.
— K51 Nonspecific ulcerative colitis.
— K52 Other noncontagious gastroenterites and colites.
— Radiative colitis and gastroenteritis.
— Toxic colitis.
— Allergic gastroenteritis and colitis.

Types of intestinal pains in coloenterites

— Spastic pains.
— Distension pains.
— Mesenterial pains.
— Pains owing to ganglionitis.

Types of intestinal dyspepsia

— Fermentative dyspepsia (impairment of digestion of carbohydrates).
— Putrefactive dyspepsia (impairment of proteopepsis).
— Fatty dyspepsia (impairment of digestion of fats).

Palpatory pain points in coloenterites

— Point of Porges — a little bit more to the left and above umbilicus at the level of XII thoracal and I lumbar spondyles.

— Point of Shternberg: 1 — ileocecal area; 2 — is higher than umbilicus on the right at the level of II lumbar spondyle.

The syndrome of maldigestion (from French *mal* — «disease», *digestion* — «digestion») is a morbid condition caused by impairment of digestion of alimentary matters owing to deficiency of alimentiry enzymes on intestinal membranes and in the lumen of small intestine, deficiency of bilious acids, insufficient blending of food with bile and juice of pancreas.

Malabsorbtion (from French *mal* — «disease», *absorbtion* — «adsorption») is a syndrome which is a manifestation of impairment of adsorption of alimentary matters from small bowel. It leads to expressed disorders of the patient's diet.

Aboriginal signs of malabsorbtion:

— Intestinal pains.
— Intestinal dyspepsia (meteorism, rumble, diarrhea).

Signs of enteric coprologic syndrome:

— Polyexcrements — augmentation of feces more than 300 g amounts.
— Lientery — undigested particles of food in feces.
— Steatorrhea — neutral fats in feces.
— Creatorrhea — typical fibers in feces.
— Amylorrhea — starch and fat in feces.

General signs of malabsorbtion:

— Drop of body mass.
— Deaquation.
— Polyhypovitaminosis.
— Impairments of proteins, lipid, electrolitic exchanges.

— Anemia.
— Hypoproteinemia.
— Hypolipidemia.

Nonspecific ulcerative colitis (NUC) — a disease of unknown etiology characterized by development of necrotizing inflammation of colon mucosa with formation of ulcers, hemorrhage and pus.

The etiology is unknown.

Pathomorfologic – the hall colon, but most frequently — straight and distal departments of the colon may be affected. The inflammation of mucosa, formation of anabroses, ulcers and pseudopolypuses, contact bleeding are characteristic.

Clinic of nonspecific ulcerative colitis

I. Complaints.

1. Diarrhea: often up to 20 times a day with an admixing of blood, mucous, pus.
2. Abdominal pains in the area of projections of the colon.
3. Intoxication: weakness, depression, fever, drop of appetite.

II. Examination.

1. Weighf loss.
2. Paleness of skin and mucosas.
3. Abdominal distention.

III. Palpation of the abdomen. Tenderness on the colon course, resistance of abdominal wall, spastic or atonic dialated departments of colon, tender.

IV. Systemic exhibitings of NUC:

— Arthritis of talocrural and knee joints.
— Nodulose erythema.
— Dermatitis.
— Iritis, iridocyclitis, episcleritis, keratitis.
— Lesion of liver: fatty dystrophia, hepatitis, cirrhosis.
— Lardaceous of kidneys.
— Autoimmune thyroiditis.

Laboratory and tool diagnostics

1. General analysis of blood: hypochromic anemia; neutrofilic leukocytosis with shift of the nuclear formula to the left; acceleration of ESR.

2. Biochemical analysis of blood: drop of protein level; augmentation of α2- and γ-globulins; drop of iron level.

3. Analysis of feces. Plenty of leucocytes, erythrocytes, cells of intestinal epithelium.

4. Endoscopic examination: diffuse hyperemia of colon mucosa; anabroses of mucosa; superficial ulcers covered with pus; bleeding especial after the contact with endoscope; pseudopolypuses.

LECTURE 15
SYMPTOMATOLOGY AND DIAGNOSTICS OF CHRONIC PANCREATITIS

Chronic pancreatites (CP) are chronic inflammatory — dystrophic diseases of pancreas causing impairment of permeability of its ducts, sclerosis of parenchyma and appreciable impairments of exocrine and endocrine functions in progressing of a pathological process.

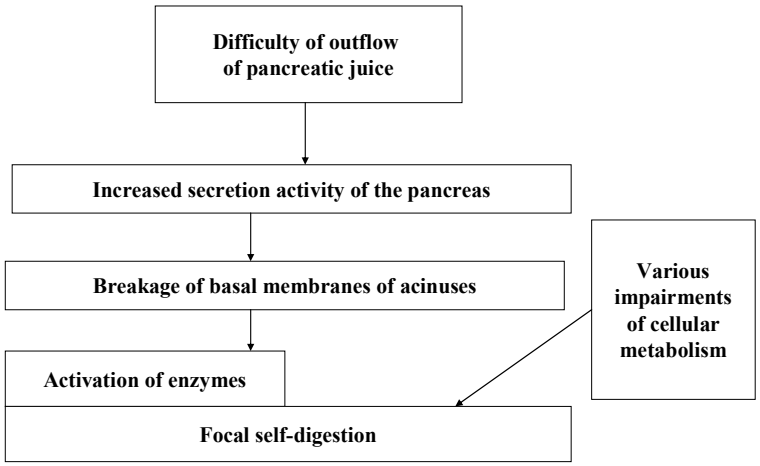
Etiology of chronic pancreatitis

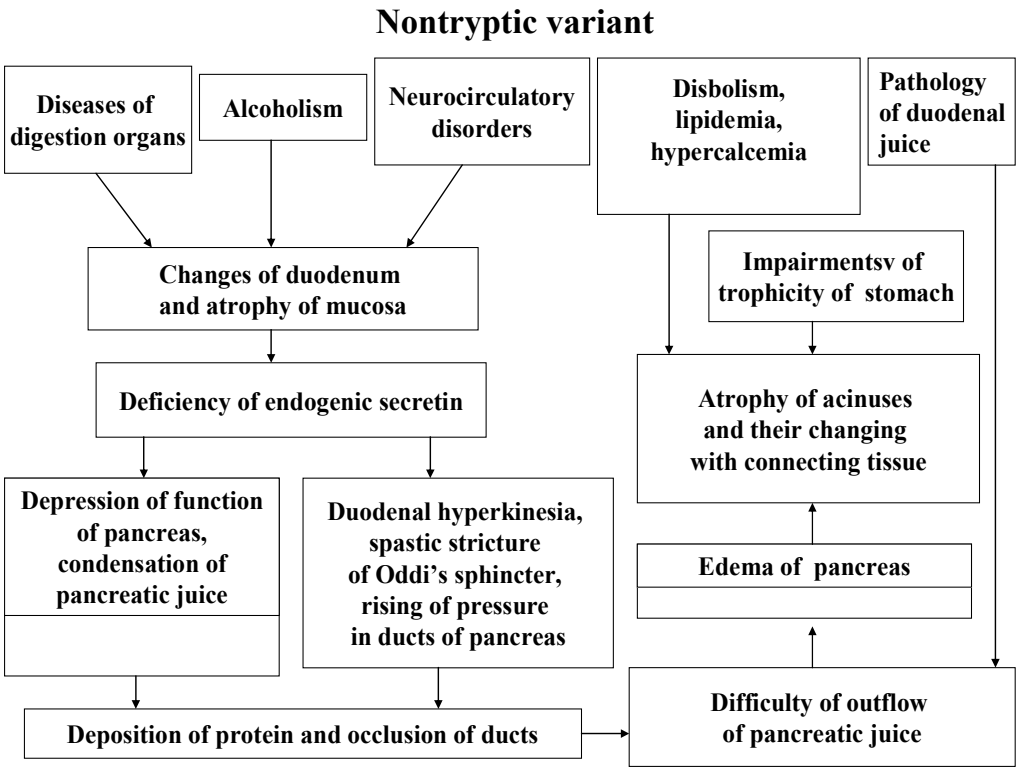
1. Abuse with alcohol.
2. Diseases of cholic pathes.
3. Diseases of duodenum and papilla of Vateri (duodenutis, hypotonia of sphincter of Oddi).
4. Nutritional factor:
1) Superfluous use of adipose nutrition.
2) Insufficient entering of protein with food.
3) Polyhypovitaminosis.
5. Infrequent causes:
1) Virus infection contamination (epidemic parotitis, hepatitis B, virus of Cocsaci).
2) Change of pancreatic vessels (atherosclerosis).
3) Hyperlipoproteinemia.
4) Hyperparathyroidism.
5) Acute pancreatitis.
6) Genetical predisposition, inheritable diseases (mucoviscidosis, hemochromatosis).
7) Medicamental influences (cytostatics, tetracyclinums, sulfanilamidums).

Pathogeny of chronic pancreatitis

Pathogenetic variants:
1. Nontryptic.
2. Tryptic.
Pathogenetic variants of chronic pancreatitis are shown on the picture 3.

Tryptic variant





Picture 3 — Pathogenetic variants of chronic pancreatitis

Classification of chronic pancreatitis (A. L. Grebenev, 1982)

I. On etiological sign:
— initial chronic pancreatitis;
— secondary chronic pancreatitis.
II. On features of clinic:
— polysymptomatic form;
— pain form;
— pseudo-tumoral form;
— dyspeptic form;
— latent (asymptomatic) form.
III. On flow of the disease:
— mild degree (1st stage, initial);
— average degree (2nd stage);
— severe degree (3rd stage, terminal, cachectic).

Clinic of chronic pancreatitis

1. Pains:
— Localization — in the epigastric range, surrounding.
— Conditions of originating — reception of meals, especially adipose, alcohol.
— Day-night rhythm — is more expressed in the 2nd half of a day.
— Character — intensive, acute, stinging.
2. Pancreatic dyspepsia:

— increased salivation;

— eructation with air or eaten meals;

— persistent nausea;

— vomiting, at times repeated, not invoking simplification of state, and at times — intensifying pains;

— loss of appetite;

— disgust for adipose meals.

3. Intoxication and general signs: moderate, subfebric fever (in the expressed exacerbation of the disease), weakness, sweating, drop of work capacity, depression.

Examination

Position of the patient: lying on the abdomen; sitting, having bent forward; genucubital.

«Color» dermal symptoms of exacerbation of chronic pancreatitis:

— The syptom of the Grotto — atrophy of hypodermic fatty tissue, brown or cyanotic shade of the skin in the area of projection of pancreas.

— Mandor's syptom — violet stain on the face.

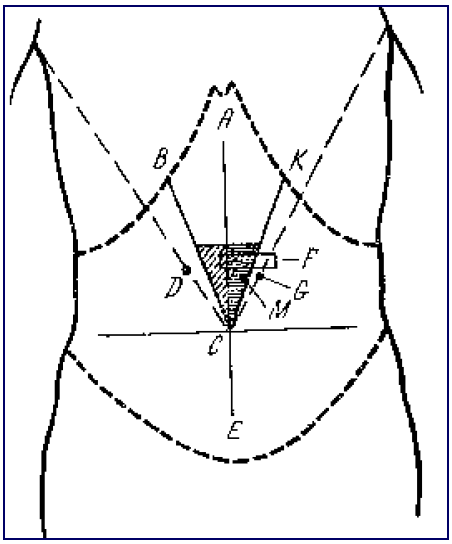
— Holsted's syptom — cyanosis of the anterior abdominal wall.

— The syptom of Heating-Turner — cyanosis of lateral walls of abdomen, especially the left one.

— The syptom of Culen-Johnson — yellow-cyanochroic shade of the skin in area of umbilicus.

Palpatory phenomena and pain points in pancreas diseases

Palpatory phenomena and pain points in pancreas diseases in shown in the picture 4.



Picture 4 — Palpatory phenomena and pain points in pancreas diseases:
ABC — zone of Shoffar — pain in lesion of the head of the pancreas;
АСК — zone of Gubergrits-Sculsky — pain in lesion of the body of the pancreas;
G — Gubergrits's point — pain in lesion of the tail of the pancreas; F — palpated pancreas

Syndrome of extrasecretory failure of pancreas

1. Pancreatogenous diarrheas, polyexcrements, steatorrhea.
2. Signs of intestinal dyspepsia.
3. Sprues and maldigestion, thinness, polyhypovitaminoses, deaquation, electrolytic impairments, anemias.

Syndrome of intrasecretory (incretory) failure of pancreas — diabetes.

Laboratory and tool diagnostics

General analysis of blood: in exacerbation — neutrophilic leukocytosis with deviation to the left, augmentation of ESR, anemia.

The general analysis of urine: in exacerbation there is rising of level of alpha amylase (diastase).

Biochemical analysis of blood.

1. Diagnostics of activity of inflammatory process in the pancreas:

a) amylase test — activity of amylase raises during the first hours of exacerbation of CP.

b) Lipase test — activity of lipase raises from the end of the 4th day.

c) Augmentation of «proteins of acute phase of inflammation» — gamma-globulins, sialine acids, seromucoid.

2. Diagnostics of syndrome of intrasecretory failure of the pancreas — augmentation of glucose level.

Research of extrasecretory functions of pancreas:

a) Definition of lipase, alpha amylase, Trypsinum, bicarbonate causeticities in duodenal contents.

b) Secretin — pancreozimin test: definition of lipase, alpha amylase, Trypsinum, bicarbonate causeticities in duodenal contents on empty stomach (basal secretion) and after intravenous introduction of secretin and pancreozimin.

c) Definition of elastase in feces.

Tool diagnostics

1. Ultrasound research of pancreas:

Signs of chronic pancreatitis:

— Illegibility of pancreas frame with hyperechogenic areas.
— Calcification of the pancreas and stones of its ducts.
— Non-uniform dilating of ducts of the pancreas.
— Rough contour of the pancreas.
— Diffuse rising of echogenic of the pancreas.

2. Rentgenologic examination of the pancreas:

— Calcinate of pancreas.
— The developed arch of the duodenum.
— Augmentation of retrogastric space.

LECTURE 16
SYMPTOMATOLOGY AND DIAGNOSTICS OF GALL BLADDER AND CHOLIC DUCTS DISEASES. SYMPTOMATOLOGY AND DIAGNOSTICS OF CHRONIC HEPATITIS AND LIVER CIRRHOSIS

Chronic noncalculous cholecystitis is chronic polyetiologic inflammatory disease of gall bladder combined with motor dysfunctions of bile ducts, impairment of outflow of bile, changes of its physical and chemical properties and biochemical structure.

Aetiology of chronic cholecystites

I. Bacterial infection.

Sources of infection:

— Diseases of nasopharynx (tonsillitises, sinusitises).
— Diseases of oral cavity (stomatites, ulites, parodontosis).
— Diseases of the urin system (urethrites, cystites, pyelonephrites).
— Diseases of the reprodactive system (prostates, adnexites, endometrites).
— Infectious disease of intestines.
— Virus lesions of the liver.
— Chronic pancreatitis.

Ways of penetration of infection into the gall bladder:

1. Hematogenous.
2. Ascending.
3. Lymphogenous.

II. Parasitogenic invasions: opisthorchiasis; lambliasis.

III. Duodenum-cholic reflux: failure of sphincter Oddi; duodenal stasis with rising pressure in the duodenum; chronic pancreatitis.

IV. Allergy.

V. Chronic diseases of digestion organs: hepatitis, cirrhosis of liver, disease of intestines and pancreas.

VI. Acute cholecystites.

VII. Dysfunctional disoders of biliary tract (biliary dysfunctions), DDBT.

DDBT is the unmatched, delayed, insufficient or superfluous motor and tonic function of the gall bladder and sphincter of Oddi.

Classification of dysfunctional disorders of biliary tracts (Rome, 1999)

1. On localization.

A. Dysfunction of the gall bladder.
B. Dysfunction of sphincter Oddi.

2. On etiology.

A. Initial.
B. Secondary.
3. On functional state.
A. Hyperfunction.
B. Hypofunction.

Etiology of dysfunctional disorders of biliary tracts

— Cholecystectomia.
— Psychoemotional stresses.
— Obesity.
— Inactive mode of life.
— Impairments of diet.
— Pregnancy and disovary disorders.
— Reflex influences of abdomen organs.
— Congenital anomalies of development of the gall bladder and bile ducts.

The basic stages of pathogeny of chronic ducts diseases

DDBT → impairments of outflow of bile from gall bladder → augmentation of concentration of bile → aseptic «chemical» cholecystitis → infectioning of the gall bladder, «contagious» cholecystitis → changes of physical and chemical properties of bile → cholelitiasis.

Classification of chronic cholecystites (Cimmerman J. S., 1992)

1. On etiology and pathogeny:
1.1. Bacterial.
1.2. Virus.
1.3. Parasitogenic.
1.4. Nonmicrobic (aseptic).
1.5. Allergic.
1.6. «Fermental».
1.7. Of obscure etiology.
2. Clinic-morphological variants:
2.1. Chronic noncalculous cholecystitis.
2.2. Chronic calculous cholecystitis.
3. Phases of disease:
3.1. Exacerbation (decompensation).
3.2. Fading exacerbation (subindemnification).
3.3. Remission (indemnification).

Clinical syndromes of chronic cholecystitis

1. Syndrome of dysfunction of the gall bladder and sphincter of Oddi (dyskinesias of bile ducts) — it is clinically manifested by cholic pain.

2. Syndrome of hepatic dyspepsia — nausea, vomiting, bitter eructation, drop of appetite.

3. Syndrome of cholestasia — in hyperfunction of sphincter of Oddi (it is rare).

4. Inflammatorily — intoxication syndrome.

Palpatory and percussion symptoms in diseases of chronic pancreatitis (J. S. Cimmerman)

I group. Segmentary reflex symptoms.

1. Visceral — cutaneus pain points and zones:

— Point of Maccensy — crossing of outside edge of the direct muscle of abdomen with dextral costal arch.

— Point of Boas — is localized on juxtaspinal line on the right at the level of X–XI of thoracal spondyles.

— Zones of Zahariyn-Ged — zones of hypersensitivity and tenderness around of points of Boas and Maccensy.

2. Skin (cutaneus) — visceral symptoms.

— The symptom of Aliev — pain on pressing on points of Boas and Maccensy.

— The 1st symptom of Aizenberg — a short impact with the edge of the palm to lower part of the angle of dextral scapula invokes pain in the right hypochondrium.

II group. Reflex pain zones and the points located in the dextral half of the body outside the segments of innervation of the biliary system:

— Point of Bergman (orbital point) — at upperinner edges of the orbit.

— Point of Ionash (occipital point) — in the place of affixion of trapezoid muscle to the back of the head.

— Point of Mussi-Georgievsky (frenicus-symptom) — between legs of musculus sternocleinomastoideus.

— Point of Haritonov (interscapular point) — in the middle of the horizontal line drawn through the middle of intrinsic edge of dextral scapula.

— Point of Lapinsky (femoral point) — in the middle of intrinsic edge of dextral hip.

— Point of dextral popliteal space.

— Plantar point — on the back of autopodium.

III group. The symptoms connected with the irritation of the gall bladder (irritative symptoms):

— The symptom of Ker — pain in the right hypochondrium in the point Maccensy on deep palpation;

— The symptom of Murphy — discontinuing of inspiration during deep palpation of the gall bladder;

— The symptom ofLepene-Vasilenco — pain on delivering of abrupt blows with the tips of fingers below dextral costal arch during an inspiration.

— The symptom of Ortner-Grecov — pain on drawing short impacts with the edge of the palm on dextral costal arch.

— The 2nd symptom of Aizenberg — in a standing position the patient raises tiptoes, and then quickly stands on his heels; thus there is pain in the right hypochondrium.

Signs of dysfunctions of bile ducts, revealed by duodenal intubation

1. Hyperfunction of bile ducts:
— More than 6 minutes elongation of 2nd stage (of time of sphincter of Oddi).
— Elongation of 3rd stage — a stage of sphincter of Lutkens.
— Time of 4th stage (stage of gall bladder) is 20 minutes shorter.
— Decrease of volume of the portion «B» is less than 30 ml.
2. Hypoactivity of bile ducts:
— Time of 2nd stage of bile secretion is 2 minutes and less.
— Time of 4th stage is more than 40 minutes.
— Volume of the portion «B» is more than 80 ml.
— Volume of residual bile is more than 20 ml.

Signs of chronic cholecystitis accoding to results of laboratory investigation of bile of the portion «B»

— Opacification, flakes of mucous in bile.
— Plenty of neutrofilic leucocytes.
— Clumps of leucocytes on flakes of mucous.
— Layers and clumps on mucous of cells of cylindrical or prismatic epithelium of the gall bladder.
— Drop of bile pH.
— Drop of relative density of bile owing to its delution with inflammatory exudate (in norm 0,016–0,035 kg/S).
— Drop of concentration of cholic acids.
— Drop of cholato-cholesteric coeffitient is lower than 10.

Ultrasonic of the gall bladder

Signs of chronic cholecystitis:
— Thickening of walls of the gall bladder is more than 2 mm.
— Inspissation of walls of the gall bladder.
— Non-uniformity and deformation of the contour of the gall bladder.
— Restriction of motility of the gall bladder on respiration and its displacement decrease (symptoms of pericholecystitis).
— Augmentation or decrease of volume of the gall bladder and its emptying (signs of DDBT).
— Dilating of general bile duct is more than 12 mm.

Gallbladder disease (cholelithiasis) is metabolic disease of the hepato-bile system, characterized by formation of stones in the gall bladder or bile ducts.

Etiological factors of cholelithiasis

1. Cholecystitis, cholangitis.
2. Stagnation of bile.
3. Impairments of bilirubin metabolism (hemocatheretic anemias) and cholesterin (obesity, diabetes, lipidemias, gout, etc.).

Clinical forms of cholelithiasis

1. Dyspeptic form (clinic of hepatic dyspepsia).
2. Pain torpid form (clinic of chronic noncalculous cholecystitis).
3. Pain paroxysmal form (attacks of hepatic colic).

Complications of cholelithiasis

1. Mechanical icterus.
2. Acute cholecystitis.
3. Empyema of the gall bladder.
4. The switched — off gall bladder.
5. Edema of the gall bladder.
6. Acute and chronic pancreatitis.

Chronic hepatitis is a diffuse polyetiological inflammatory process in the liver, caused by an initial lesion of liver cells, not resolved during 6 months and evolving or not evolving in liver cirrhosis.

Etiological classification of chronic hepatitis (Los Angeles, 1994)

1. Chronic hepatitis of virus etiology (chronic virus hepatitis C, B, D, G, caused by unknown virus).
2. Chronic alcoholic hepatitis (manifestation of alcoholic disease of the liver).
3. Chronic drug-induced hepatitis.
4. Chronic autoimmune hepatitis.
5. Chronic cholestatic hepatitis.
6. Chronic hepatitis in Wilson's disease.
7. Chronic hepatitis in α_1 — antitrypsin deficiency.
8. Initial sclerosing cholangitis.
9. Cryptogenic hepatitis.

Clinic-morphological variants of chronic hepatitis

1st degree. Chronic hepatitis of minimal activity.

2nd degree. Slightly manifested chronic hepatitis (activity of serumal ALT is enlarged up to 3 norms).

3rd degree. Moderate chronic hepatetis (activity of serumal ALT makes 3–10 norms).

4th degree. Serious chronic hepateitis (activity of serumal ALT exceeds 10 norms).

Clinical syndromes in chronic hepatitis

— Astenic-vegetative syndrome.

— Dyspeptic or transabdominal (pains in the right hypochondrium, hepatic dyspepsia).

— Syndrome of hepatic icterus (rubinicterus, brown urine, clarification of feces, hyperbilirubinemia due to conjugated and nonconjugated fractions, bilirubinuria).

— Syndrome of cholestasia (dermal itch, melasicterus, xanthomatosis, bleeding, osteoporosis, conjugated hyperbilirubinemia, bilirubinuria).

— Syndrome of hepatomegalia (less often — splenomegaly).

Features of clinic of autoimmune chronic hepatitis

Typical features:

— Girls and young women (10–20 years) are ill more often.

— High activity of inflammatory process which does not react to therapy traditional for hepatitis.

— Pains in large joints of the lower and upper extremities.

— Lesions of skin in the form of relapsing purpura, less often — «lupoid butterfly», nodulose erythema, focal scleroderma.

— Chronic glomerulonephrites, serosites (pleuritis, pericarditis), myocardites, thyroidites.

— Generalized lymphoadenopathy and splenomegaly.

Clinic-laboratory syndrome of chronic hepatitis

Syndrome of cytolysis

Augmentation of activity of display enzymes in blood serum:

— ALT.

— AST.

— GGT.

— GDG.

— LDG 4, 5.

Augmentation of concentration in blood serum of:

— Ferrum.

— Vitamin B12.

— Conjugated bilirubin.

Mesenchymal — inflammatory syndrome

(syndrome of immune inflammation)

We reveal in blood:

— Augmentation of concentration of crude protein.

— Augmentation of levels of α2-, β-, γ fractions of globulins.

— Augmentation of levels of IgA, IgM, IgG.

— Positive sedimentary assays — thymolic, sublimate.
— Positive C-reactive protein.
— Antibodies to the tissue of the liver.
— LE-cells.
— Leukocytosis, acceleration of ESR.

Syndrome of cholestasia

Augmentation of activity of excretory enzymes in blood serum:
— Alkaline phosphatase.
— GGT.
Augmentation of serumal concentration:
— Cholic acids.
— Cholesterin.
— β-lipoproteins.
— Conjugated bilirubin.

Syndrome of hepatic-cellular failure

(failures of synthetic function of the liver)

Activity of the following secretory enzymes is reduced in blood serum:
— Cholinesterases.
— Pseudocholinesterases.
Concentrations of the following elements are reduced in blood serum:
— Albuminums.
— Cholesterin.
— Ureas.
— Thrombinogen;
— Fibrinogenum.
— V, VII, IX, X blood-coagulation factors.
— Hepatocuprein.

Laboratory signs of autoimmune hepatitis

Revealing of the following elements in blood:
— Antimitochondion antibodies.
— Antinuclear antibodies.
— Antibodies to smooth musculation.
— LE-cells.
— The rhematoid factor.

Laboratory signs of virus hepatitis

Revealing of:
— In hepatitis B — HBsAg, HBcAg, HBeAg and antibodies to them.
— In hepatitis C — antiHCV.

Liver cirrhosis is a diffuse process characterized by fibrosis and rearrangement of normal architectonics of the liver leading to formation of structural — abnormal noduses and formation of portal hypertension.

Aetiology of liver cirrhosis

— Virus hepatitises C, B, D, G.
— Alcoholism.
— Autoimmune hepatitis.
— Ancestral diseases: hemochromatosis, Wilson's disease, a_1 — antitrypsin deficiency, glycogenoses.
— Diseases inside — and of extrahepatic bile ducts — initial and secondary bile cirrhosis.
— Obstruction of venous outflow from the liver in the syndrome of Badd-Kiary and right heart failure.
— Action of toxins and medicines: four-chloride Carboneum, Chloroformium; benzene, nitrocompounds, intoxication with Hydrargyrum or mushroom poison.
— Rendu-Osler-Weber disease.

Morphological variants of liver cirrhosis

— Micronodal cirrhoses with the diameter of abnormal nodes 1–3 mm.
— Macronodal cirrhoses with the diameter of abnormal nodes more than 3 mm.
— Mixed macro-micronodal cirrhoses.
— Incomplete septal cirrhosis.

Stages of liver cirrhosis

— Stage of indemnification.
— Stage of decompensation.
Types of decompensation:
— hepatic-cellular;
— porto-vascular.

Clinical syndromes of liver cirrhosis

— Astenic-vegetative syndrome.
— Dyspeptic or transabdominal.
— Syndrome of hepatic icterus.
— Syndrome of cholestasia.
— Syndrome of hepatosplenomegalia.
— Syndrome of portal hypertension (ascites, «the head of Jellyfish», varicose veins of esophagus, rectum, splenomegaly, hypersplenism).
— Syndrome of hepatic failure (angiomatosis, pulm erythema, gynecomastia, hemorrhagic syndrome, thinness, encephalopathy).

Hepatic (hepatogenic) encephalopathy is a potentially reversible impairment of function of the brain, arising due to acute hepatic failure, chronic diseases of the liver and (or) portosystemic shuntings.

Factors of hepatic encephalopathy pathogeny

— *The factor of severe parenchymatous failure* — sharp, below critical, drop of mass of functioning hepatocytes with the impairment of their decontaminating function (true hepatic encephalopathy).

— *The factor of portosystemic shuntings* (encephalopathy of hepatic shunting or hepatic portal encephalopathy).

Stages of hepatic encephalopathy

— stage I (precoma I);
— stage II (precoma II, stupor);
— stage III (coma I, sopor);
— stage IV (coma).

LECTURE 17
INQUIRY AND EXAMINATION OF PATIENTS WITH DISEASES OF KIDNEYS AND URINARY TRACTS. PALPATION AND PERCUSSION OF KIDNEYS AND THE URINARY BLADDER

Classification of complaints of patients with diseases of urinary organs

A. Renal complaints.

I. Complaints of changes of organic properties of urine.
1. Changes of intensity of urine colouring.
2. Discolorations of urine.
3. Changes of transparence of urine.

II. Complaints of dysuric disorders.
1. Changes of volume of abjointed urine.
2. Changes of frequency of emictions.
3. Changes of rhythm of emictions.
4. Changes of quality of emictions.

III. Complaints of pains in the lumbar area, in the abdomen, along ureters and above the pubis.

IV. The complaints caused by renal failure (uremia).

B. Extrarenal complaints.

I. Basic complaints: edemas; manifestations of syndrome of arterial hypertension.

II. General complaints.

Changes of intensity of urine colouring

1. Drop of intensity of urine colouring.
1.1. Renal causes.
1.1.1. With augmentation of volume of urine (polyuria):
— Chronic pyelonephritis.
— Chronic renal failure of II–III stages.
— Polyuric stage of acute renal failure.
1.1.2. With drop of urine volume (oliguria):
— Chronic renal failure of IV (terminal) stage.
1.2. The extrarenal causes:
— The use of superfluous amount of fluid.
— Sugar and non-sugar diabetes.
— Usage of diuretics.
— Convergence of edemas and resorption of exudate from sinuses.
— Sympathicotonia (stresses, attacks of pheochromocytoma, paroxysmal supraventriculus arrhythmias).
2. Augmentation of intensity of urine colouring.
2.1. The renal causes:
— Acute glomerulonephritis oligoanuric stage.
— Acute renal failure oligoanuric stage.
2.2. Extrarenal causes:
— Formation of edemas in heart failure.
— Acute infectious deseases with fever.
— Diseases accompanied with diarrhea and vomiting.
— Other types of deaquation (increased sweating, restrictions of fluid consumption).

Variants of discoloration of urine

1. Red colour — gross hematuria, the use of beet, some medicines (Rifampicinum, Antipyrinum).
2. Brown colour — icterus.
3. Black colour — hemocatheretic anemias.

Causes of gross hematuria

Renal:
1. Gross hematuria after attack of renal colic: urolithiasis, infarction of kidney.
2. Nonpain, abundant, persistent, long gross hematuria: tumours of kidneys and the urinary bladder.
3. Nonpain, unstable, transient gross hematuria: tuberculosis of kidneys.
4. Acute and chronic glomerulonephrites.
5. Acute pyelonephritis.
6. Hemorrhagic vasculitis.
7. Trauma of kidney.

Extrarenal:

1. Overdosage of anticoagulants.
2. Hemorrhagic diathesises.
3. Cystites.
4. Traumas of ureters, urinary bladder.

Polyuria — increased (more than 2 000 ml) abjection of urine for a day.

Variants of polyuria:

— Physiological polyuria.
— Pathological polyuria.

Variants of pathological polyuria

Extrarenal polyuria:

— In sugar and non-sugar diabetes.
— In hyperaldosteronism.
— In sympathicotonia.
— In the period of convergence of edemas.
— In resorption of exudates and transudates from perigastria.
— On usage application of diuretics.

Renal polyuria:

— In chronic renal failure of II–III stages.
— In polyuric period of acute renal failure.
— In stage of the sanction of acute glomerulonephritis.

Oliguria — drop (less than 500 ml) of abjection of urine a day.

Variants of oliguria:

— Physiological oliguria.
— Pathological oliguria.

Causes of physiological oliguria:

— Restriction of consumption of fluids.
— Intensive sweating.

Types of pathological oliguria:

— Prerenal.
— Renal.
— Postrenal.

Causes of prerenal oliguria:

1. Decrease of volume of circulating blood owing to:

— Delays of fluid in tissues and perigastria.

— Loss of plenty of fluid in extrarenal way: abundant vomiting and diarrheas, extensive burns and frostbites, great blood loss.

— Diseases leading to restriction of reception of fluids (burns and wounds of the oral cavity, the pharynx, the esophagus, stenoses of the esophagus).

2. Drop of level of arterial pressure in shock, collapse, heart failure.

Causes of renal oliguria:

— Acute glomerulonephrites (oligoanuric period).

— Diseases of kidneys proceeding with a hydropic — nephrotic syndrome.
— Poisoning with nephrotoxic poisons (etilenglycol, mushroom poisons, etc.).
— Traumas and infarction of kidneys.
— Chronic renal failure in end-stage.

Causes of postrenal (subrenal) oliguria:

Particulate obstruction of ureters with mucous or pus, stone, clot, owing to excess in nephroptosis, prelum with tumour, hematoma, cicatrical stenosis.

Anuria is complete arrest of abjection of urine by kidneys or abjection of urine which is less than 100 ml a day. There is no physiological anuria. Types of pathological anuria and causes are the same, as in oliguria.

Pollakiuria — often emiction (over 6 times a day) in which often desires occur not only in the daytime, but also at night.

Causes: cystites, prostatites and adenoma of prostate, stones of the urinary bladder, neoplasm of the urinary bladder, prelum of the urinary bladder with an enlarged uterus (pregnancy, fibromyoma), chronic pyelonephrites, polyuria.

Strangury — emiction in small portions (drops) owing to its sharp difficulty.

Causes: inflammation of the urethra and the cervix of the urinary bladder, tumour of the urinary bladder, adenoma and cancer of the prostate, stones of the urinary bladder.

Ischuria — morbid condition in which the patient is not able to empty the urinary bladder.

Causes: tumours and inflammatory processes of small pelvis; tumours, traumas, infarction of the brain and spinal cord; unconsciousness.

Types of the compelled position of the body in kidneys and urinary tracts diseases

— In paranephritis — lying on the sick side with bent in knee and coxofemoral joints and a leant to the abdomen leg.
— The compelled change of the body — in renal colic.
— Cramps — in uremic coma, eclampsia.
— Orthopnoe — in heart failure and expressed hydropic syndrome.
— Position typical of dry pleuritis — lying on the sick side, sitting having bent in to the «sick» side.
— Typical of dry and exudate pleuritis position — having bent forward, sitting.

Pain points revealed at pathology of urinary tracts

Back points:

1. Costovertebral — in the angle, formed by XII rib and the spinal column.
2. Costal-lumbar — in the place of crossing of XII rib with lumbar muscle.

Anterior points:

1. Subcostal — at 1st edge of X rib.

2. Top ureteric — at eternal edge of direct muscle of the abdomen at the level of the umbilicus.

3. Average ureteric — in the place of crossing linea biiliaca with the vertical line drawn through sp. ossis pubis.

Degrees of nephroptosis

1st degree — the inferior pole of the kidney is palpated.

2nd degree — kidney is entirely palpated.

3rd degree — kidney is completely palpated and displaces into the other half of the abdomenae cavity (regarding the spinal column).

LECTURE 18
SYMPTOMATOLOGY AND DIAGNOSTICS OF ACUTE AND CHRONIC GLOMERULONEPHRITES, RENAL FAILURE

Glomerulonephritis is genetically caused immune inflammation of kidneys with a primary initial lesion of glomuli and involvement in the pathological process of all renal structures, and clinically manifesting itself with renal and extrarenal signs.

Etiology of acute glomerulonephrites

I. Contagious factors.

1. Poststreptococcal glomerulonephritis.

2. Nonpoststreptococcal glomerulonephritis:

— Caused by staphylococci, pneumococci, enterococcuses, bacillus of Friedlander, brucella, typroid bacillus, meningococci.

— Caused by viruses of hepatitis B, contagious mononucleosis, epidemic parotitis, chicken pox, viruses of Coxaci, ECHO.

— Caused by the elementary (toxoplasma, malarial plasmodium) and treponema (acyanotic treponema, leptospira).

II. Noncontagious factors.

1. General diseases: systemic lupus erythematosus, vasculites — Henoch-Schoenlein's disease, Guodpastureis pulmonary — renal syndrome.

2. Usage of vaccines, sera.

3. Influence of alcohol.

4. Cooling.

Pathogenetic variants of acute glomerulonephrites

1. Immunocomplex.

2. «Microimmune», pauci-immune.

3. Caused by antibodies to basal membrane of glomuli.

4. Caused by antigenic mimicry.

Basic syndromes of glomerulonephrites

Extrarenal:

— Hydropic syndrome (typical renal edemas).

— Cardiovascular syndrome (AH and its complications).

— Cerebral syndrome (eclampsia, brain edema).

Renal:

— Syndrome of acute inflammation of renal glomuli (basic manifestations — pain in lumbar region of loin, urinary syndrome — hematuria, proteinuria, cylindruria).

Clinical forms of acute glomerulonephritis

1. The classical triad developed form (hydropic, cardiovascular and urinary syndromes).

2. The bisyndromeic form (urinary and hydropic syndromes or urinary and cardiovascular syndromes).

3. The monosyndromeic form (an isolated urinary syndrome).

4. The nephrotic form (a nephrotic syndrome).

Causes of development of hydropic syndrome — Hyperaldosteronism developing owing to ischemia of kidneys and activization of the renin — angiotensin-aldosteron system.

— Rising of permeability of vessels owing to high activity of Hyaluronidasum.

— Loss of protein with urine, development of hypoalbuminemia and drop of oncotic blood pressure.

Causes of development of arterial hypertension syndrome

1. Activation of juxtaglomerular apparatus owing to renal ischemia with the subsequent rising of synthesis of renin and activation of the renin - andgiotensn mechanism.

2. Hyperaldosteronism with delay of sodium and water in organism and augmentation of circulating blood volume.

Clinical forms of chronic glomerulonephrites

— The latent form (isolated urinary syndrome).

— The nephrotic form (nephrotic syndrome).

— The hypertonic form (the syndrome of AH and moderate urinary syndrome).

— The mixed form (combination of nephrotic and hypertenthion syndrome).

— The hematuric form (a urinary syndrome with minimal proteinuria and expressed hematuria).

A nephrotic syndrome is a clinic-laboratory symptomocomplex caused by structural damage of basal membranes of glomulus capillaries with sharp rising of their permeability and clinically manifesting itself with the combination of massive proteinuria and hypoproteinemia with wide-spread edemas, lipidemia and drop of resistance to infection.

Chronic renal failure (CRF) is a pathological symptomocomplex caused by sharp decrease of the number and function of nephrons that results in impairment of excretory and incretory functions of kidneys, disorder of all types of metabolism, activity of organs and systems, acid-alkaline and electrolitic equilibrium.

Etiology of CRF:

1. Chronic glomerulonephrites.
2. Chronic pyelonephrites.
3. Polycystosis of kidneys.
4. Diabetic nephropathy.
5. Lardaceous of kidneys.
6. Tuberculosis of kidneys.
7. Lesion of kidneys in DDCT, vasculites.
8. Arterial hypertension (primarily contracted kidney and stenosis of renal arteries).
9. Obstructive diseases of urinary tracts.

The stages of chronic renal failure are shown in the table 15.

Table 15 — Stages of chronic renal failure (N. A. Lopatkin, I. N. Kuchinskij)

Clinic-laboratory signs	Latent	Compensated	Intermitent	Terminal
Diuresis	Norm	Mild polyuria	Expressed polyuria	Oligoanuria
Haemoglobin, g/l	More than 100	100-80	80–65	Less than 60
Zimnitsky test	Norm	Fluctuations of density is less than 8	Hypoisosthenuria	
Urea of blood, mmol/l	Up to 8,8	8,8–10	10–20	More than 20
Creatinine of blood, mmol/l	Up to 0,18	0,2–0,3	0,3–0,6	More than 0,6
Glomerular filtration, ml/minutes	60–45	40–30	30–20	20
Electrolits of a blood	Norm	Seldom a hyponatremia	Hyponatremia, hypokalemia, hypocalcemia	Hyperkalemia
Metabolic acidosis	Is absent	Is absent	Moderate	Expressed

LECTURE 19
SYMPTOMATOLOGY AND DIAGNOSTICS OF DIABETES, HYPERTHYROIDISM AND HYPOTHYROIDISM

Diabetes is a state of chronic hyperglycemia which is caused by absolute or relative deficiency of insulin in the organism and leads to pathological changes in various organs and tissues of the organism and to impairment of exchange of proteins, fats and carbohydrates.

The name of this disease arose from Latin words *diabetes* — «course», *mellitus* — «sweet», «honey».

Etiological classification of impairments of glycemia (World Health Organization, 1999)

1. Diabetes of the 1st type: a) autoimmune; b) idiopathic.
2. Diabetes of the 2nd type (impairment of sensitivity of insulin receptors, synthesis of abnormal insulin).
3. Other specific types of diabetes (secondary diabetes) — the most often variants are: a) diseases of the exocrine pancreas (pancreatitis, tumours, hemochromatosis, traumas, etc.); b) endocrynopathy (pheochromocytoma, hypercorticoidism, thyrotoxicosis, acromegalia); c) diabetes induced by medicines or chemicals (adrenoblockers, glucocorticoids); d) infections (rubella, parotitis).
4. Gestational diabetes (diabetes in the pregnant).

Major factors of risk of diabetes

— Superfluous mass of the body.
— Irrational feeding (use of easy assimilable carbohydrates, fat food).
— Inactive mode of life.
— Presence of arterial hypertension.
— Ancestral anamnesis of diabetes.
— Pregnancy pathology: hydramnion, multiple pregnancy during pregnancy.
— Child's weight at birth is more than 4 kg.
— Smoking.
— Abuse with alcohol.
— Regular reception of clophelinum and others central cympatholytics, peroral contraceptives.
— Often stressful situations.
— Autoimmune diseases.

«Big symptoms of diabetes»

— General and muscular weakness.
— Thirst (polydipsia).
— Dryness in mouth.

— Often and abundant emiction.
— Thinness (it is typical of diabetes of the 1st type).
— Rising of appetite (polyphagia).

«Small symptoms of diabetes»

— Causeless dedentition, parodontosis, alveolar pyorrhea — pyoinflammatory lesion of small cavities of teeth.
— Generalized dermal itching with primary localization in the area of perineum.
— Parkinsonism.
— Causeless trophic disorders, furunculosis.
— Polyhypovitaminosis, etc.

Laboratory diagnostics of diabetes

In biochemical analysis of blood the determination of glucose is carried on with the following standard methods:
— glucosooxidase — normal contents of glucose in capillary blood — 3,5–6,1 mmol/l;
— orthotoluidin — 3,3–5,5 mmol/l (it is used more often);
— iit's 0,1 mmol/l lower in venous blood.
The laboratory criteria of diagnostics of impairments of carbohydrate metabolism in shown in the table 16.

Table 16 — Laboratory criteria of diagnostics of impairments of carbohydrate metabolism

Diagnosis	Glycemia in capillary blood, determined with orthotoluidin method, mmol/l	
	On empty stomach	In 2 hours after carrying out of load with glucose
Impairment of glycemia on empty stomach	More or equal to 5,6	Less than 7,8
Diabetes	More or equal to 6,1	More or equal to11,1
Impairment of tolerance to glucose	Less than 6,1	More or equally 7,8 and less than 11,1

Complications of diabetes

Late complications:
— microangiopathy(retinopathy, nephropathy);
— macroangiopathy(atherosclerosis of arteries);
— neuropathy;
— universal angiopathy — combination microangiopathy and macroangiopathy.
Acute complications — diabetic comas, a hypoglycemic coma.

Diabetic nephropathy

Clinical manifestations: microalbuminuria, proteinuria, nephrotic syndrome, chronic renal failure.

Clinical rule

If in unitary analysis of urine (in one portion) we repeatedly reveal more than 20 mg/l of albuminuria, and in the urine collected with a day the contents of Albuminum is more than 30 mg, and it is proved in the analyses of urine repeated in 6 and 12 weeks, it is necessary to expose the diagnosis of beginning diabetic nephropathy.

Types of diabetic (hyperglycemic) comas

1. Ketoacidotic.
2. Hyperosmolar.
3. Lactacidemic.

Causes of hyperglycemic comas

1. Revealed for the 1st time of diabetes which was not diagnosed and treated in time.
2. Delayed introduction of insulin because of error of the patient or abandoning of introduction of insulin.
3. Introduction of insufficient dose of insulin as in the subsequent morbid conditions the need of it is enlarged.

— Intercurrent (concomitant) diseases — infectious diseases, exacerbations of chronic processes of the liver, kidneys, the cardiovascular system, and etc.

— Stress, traumas, operations, burns and scalds.

— Pregnancy.

— Impairment in diet (abuse with easy assimilable carbohydrates).

— Reception of diuretics.

— Diarrhea.

Hypoglycemia is a state which causes are drop of glucose level in blood below 2,8 mmol/l or sharp difference of glycemia level more than 10 mmol/l.

Causes of hypoglycemia

— Superfluous dose of insulin.

— The patient has introduced insulin and has not had meals.

— Physical exertion which reduces needs of the organism of insulin.

— Stressful situations.

— Development of hepatic or renal failure results in deficiency of insulinase and long circulation of insulin in blood.

— Reception of alcohol.

The relative characteristic of comas in diabetes in shown in the table 17.

Table 17 — The relative characteristic of comas in diabetes

Signs	Diabetic coma (ketoacidotic)	Hypoglycemic coma
The onset	Slow	Subitaneous
Consciousness	It is lost gradually, mental depression can precede it	It is lost quickly, exaltation, delirium can precede it
Tone of muscles, reflexes	Hypomyotonia, flaccidity of tendon reflexes, sometimes areflexia	Muscular hypertonia, muscle tension, pathological reflexes
Cramps	Absent	Characteristic
Tone of eyeglobes	Lowered	Normal or increased
Pupils	Narrowed	Wide
Skin	Dry, acyanotic	Wet
Arterial pressure	Lowered	Normal or increased
Breathing	Kussmaul, odour of acetone	Superficial
Cardiovascular system	Heart sounds are dull, tachycardia, weak pulse	Sounds are clear, bradycardia
Digestive system	Tongue is dry, nausea, vomiting	Tongue is wet, there is no vomiting
Urine	Glucosuria, ketonuria	There is no glucosuria and ketonuria
Biochemical analysis of blood	Hyperglycemia is up to 30 mmol/l	Hypoglycemia is less than 3 mmol/l

Terminology

1. Struma is augmentation of dimensions of the thyroid gland:
— Diffuse — uniform augmentation of all departments of the thyroid gland.
— Nodal — presence of nodal formations in the thyroid gland.
2. Euthyroidism — production of normal quantity of thyroid hormones.
3. Hypothyroidism — drop of production of thyroid hormones.
4. Hyperthyroidism — augmentation of production of thyroid hormones.
5. Thyrotoxicosis — clinical manifestation of hyperthyroidism.

Diffuse toxic struma (DTS) is autoimmune disease of the thyroid gland developing in genetical predisposed to it persons, characterized by diffuse augmentation and hyperfunction of the thyroid gland, and also by toxic changes of organs and systems owing to hyperproduction of thyroid hormones.

Etiology of diffuse toxic struma

1. Ancestral predisposition (the basic etiological factor). A mode of inheritance — multifactorial (polygenic). Inheritance is connected with a carriage of antigens HLA-B8, DR3, DW3.

2. The factors provoking development of DTS: mental traumas, stressful situations; diseases of nasopharynx; infectious — inflammatory diseases of other localizations; craniocerebral traumas.

Basic pathogenetic factors of diffuse toxic struma

1. Ancestral deficiency of T-supressing function of lymphocytes.

2. Expression of thyreocites HLA-DR-antigens on the surface.

3. Appearance of forbidden clones of T-lymphocytes with helping activity that promotes formation of the following antibodies to receptors of thyritropic hormone:

— LATS factor (it is a long reacting thyreostimulator) — IgG, stimulating the function of the thyroid gland with substantial growth of production of T3 and T4;

— GSI (growth stimulating immunoglobulins), promoting diffuse growth of thyroid gland.

Pathogeny of basic symptoms of diffuse toxic struma

Excess of thyroid hormones causes:

— Rising sensitivity of the cardiovascular system to catecholamins (tachycardia, impairments of heart rhythm, dystrophia of the myocardium, rising of the level the of BP).

— Augmentation of production of heat (sense of fever, sweating, subfebrile fever).

— Rising of standard metabolism.

— Augmentation of glycemia (intensifying of adsorption of glucose in intestines, stimulation of glyconeogenesis and glycogenolysis).

— Intensifying of catabolism of fats and protein (loss of body weight, atrophy of muscles, thinning of skin).

— Intensifying of motility of the stomach and an intestines (abdominal pain, diarrhea, nausea, vomiting).

— Rising excitability of the nervous system (irritability, tearfulness, changeability of mood, impairment of dream).

— Depression of function of adrenal cortex.

Ophthalmopathy, exophthalmia are consequences of autoimmune lesion oculomotor muscles, superfluous synthesis of glucoseaminoglycanes and others connective tissue components in retrobulbar fat.

Ophthalmic symptoms of thyrotoxicosis

1. The symptom of Crause — increased shine of eyes.

2. The symptom of Dalrimpl — wide disclosing of palpebral fissure (the surprised look).

3. The symptom of Grefe — on vision bracing of the subject alighting downwards slowly the field of sclera between upper eyelid and edge of cornea is exposed.

4. The symptom of Coher — the same (the symptom of Grefe), but on moving of a subject from below upwards.

5. The symptom of Moebius — impairment of convergence of eyeglobes.

6. The symptom of Shtelvag — infrequent nictitation.

7. The symptom of Jofrua — absence of wrinking of the forehead on the sight upwards.

8. The symptom of Rosenbah — tremor of eyelids on closed eyes.

9. The symptom of Brown — absence of narrowing of palpebral fissure on laughter.

10. The symptom of Elinec — pigmentation of eyelids.

11. The symptom of Stasincky or «a red cross» — injection of vessels of scleras, injected vessels depart upwards, downwards, to the left and to the right from of cornea that gives an impression of cross.

Signs of infiltrative ophthalmopathy:

— Exophthalmia — true shift of eyeglobe forward.

— Tumescence of eyelids, their infiltration.

— Conjunctivitis — swelling and reddening of conjunctiva, sense of colic, «sand» in eyes, lachrymation, photophobia.

— Impairment of movements of eyeglobes to the sides.

— Impairment of closing of eyelids that results in dryness of corneas, keratitis, helcomas.

Classification of degrees of struma (Word Health Organization, 1994)

0 degree — struma is not present.

1st degree — struma is palpated, but not seen.

2nd degree — struma is palpated and determined visually.

Laboratory diagnostics of DTS: the level of thyroxin (T4), triiodthyronine (T3) is increased, level TTH is reduced in blood.

Hypothyroidism — heterogenous syndrome characterized by drop or complete abaissement of function of the thyroid gland and changes of functions of various members and systems, caused by the insufficient maintenance of thyroid hormones in the organism.

Etiological classification of hypothyroidisms

I. Initial hypothyroidism — is caused by a pathology of the thyroid gland.

1. Ancestral: hypoplasia or aplasia of TG; ancestral defects of biosynthesis of thyroid hormones or thyreoglobulin.

2. Acquired: postoperative (strumectomy); postradiative (treatment with radioiodine, irradiating of TG); inflammatory diseases of TG (thyroidites); insufficient entering of Iodum into the organism (endemial struma, cretinism); influence of medicines (thyreostatics, cordaronum); tumours of TG.

II. Secondary hypothyroidism — is caused by lesion of the pituitary and failure of TTH: ischemia of the pituitary owing to abundant hemorrhage; inflammatory processes in the pituitary; tumour from thyreotropine producing cells of the pituitary; medicinal influences (Reserpinum, Levodopum, Parlodelum); autoimmune lesion of the pituitary.

III. Tertiary hypothyroidism — is caused by lesion of the hypothalamus and thyroliberinum failure: inflammatory processes in the area of the hypo-

thalamus; craniocerebral traumas; tumours of the brain; usage of preparations of serotonin.

IV. Peripheric hypothyroidism — is caused by inactivation of thyroid hormones during circulation and drop of sensitivity of peripheric tissues to them: inactivation of hormones by antibodies; ancestral drop of sensitivity of receptors to thyroid hormones; impairment of conversion of T3 in T4 in the liver and kidneys; selective resistance to T4 (defect of transport through the membrane).

Pathogeny of hypothyroidism

1. Impairment of metabolism, retardation of synthesis and disintegration of proteins.

2. Mucin, hyaluronic and chondroitin sulfuric acids collect in tissues. They are capable to detain water and invoke myxedema of tissues and organs (myxedema), invoke development of hydrothorax and hydrocardia.

3. Impairment of disintegration of lipids and development of lipidemia, atherosclerosis.

4. Impairments of metabolism of carbohydrates shows hypoglycemia owing to decrease of glucose absorption in intestines and retardations of its recycling by cells.

5. Impairment of energy formation, expressed dystrophic changes in all organs and tissues, impairment of the functional state of the nervous system.

6. Impairment of the function of intestines (constipations, impairment of vitamin B12 ant iron adsorption).

Laboratory diagnostics of hypothyroidisms: blood concentrations of T3 and T4 are reduced, the level of TTH is increased in initial and peripheric ones, reduced in secondary and tertiary hypothyroidisms.

Thyrocardiac crisis — serious, menacing to life of the patient complication of the toxic struma, showing sharp exacerbation of symptoms of thyrotoxicosis.

Causes of development of thyrocardiac crisis:

— Long absence of treatment of thyrotoxicosis.
— Treatment of toxic struma with radioiodine and surgical treatment.
— Infectious — inflammatory diseases.
— Serious mental trauma.
— Serious exercise stress.
— Surgical interventions.

LECTURE 20
SYMTOMATOLOGY AND DIAGNOSTICS OF ACUTE ALLERGOSIS

Allergy is immune reaction of the organism accompanied by damage of its own tissues.

Aetiology of allergosis

I. Primary etiological factor is allergen.

Classification of allergens:

1. Noninfectious:

— Household — house dust, pillow feather, library dust, etc.

— Animal — epidermal and food.

— Insective — poisons, a body of insects.

— Vegetative — pollen, food, etc.

— Industrial — chemical substances, industrial dust.

— Medicinal preparation.

2. Infectious:

— Fungoid — mycelium and spores.

— Bacterial.

— Protozoa.

— Helminthiasis.

— Viruses.

II. Factors promoting development of allergosis.

1. Hereditary predisposition. Allergic diseases more often develop in persons with the generated allergic phenotype (IgE concentration is increased, the density and affinity of receptors to IgE on cells — targets are increased) is more often.

2. External factors: increase in quantity of allergens (products of manufacture of chemical industry); pollution of the environment; increase in the number of medicines; total vaccination of the population; adverse social factors (stress, hypodynamia).

Pathogenesis of allergosis

Ways of allergens receipt into the organism: inhalation, injection, enteral, contact.

The mechanism of allergy development: corresponds to mechanisms of the basic types of allergic reactions (Dzhell and Coombs):

— 1st type — anaphylactic (reagin, mediated IgE).

— 2nd type — cytotoxic.

— 3rd type — mediated by immune complexes.

— 4th type — cellular (of adelayed type, mediated by T-lymphocytes).

— 5th type — antireceptor.

Nettle-rash (urticaria — from Latin *urtica* — «nettle») is a disease characterised by more or less widespread skin eruption of itching blisters, representing hypostasis of the limited area, mainly, papillary stratum of derma (skin).

Aetiology of nettle-rash

1. Allergic nettle-rash.

It develops owing to allergy to exogenous and endogenous allergens. Practically all types of allergens can cause nettle-rash.

2. Pseudo-allergic nettle-rash.

It arise under the influence of exogenous and endogenous factors causing formation and allocation of allergy mediators by nonimmune (nonspecific) mechanism.

3. Mixed nettle-rash.

Allergic and pseudo-allergic mechanisms participate in the development of these nettle-rashes.

Classification of nettle-rash

On clinical course:

1) acute — duration is till 3–4 days;
2) subacute — duration is till 6 weeks;
3) chronic recurrent — duration is over 6 weeks.

On prevalence:

1) focal;
2) generalized.

Clinic of nettle-rash

1. Urticaria (blisters).
2. Itching.
3. Blood eosinophil.

Quincke's edema (angioneurotic edema, giant urticaria) is accurately localised hypostasis of a part of the skin and subcutaneous fat.

Pathogenesis: as in nettle-rash.

Localisation: eyelids, lips, gullet, throat, scrotum. Differences from nettle-rash: there is no itching.

Pollinosis is a chronic allergic disease, caused by pollen of the plants which most typical manifestations are acute rhinitis, conjunctivitis, bronchitis, bronchial asthma, rarer — allergic reactions of other organs and tissue, characterized by seasonal periods of aggravations dependent on presence of pollen of plants in the air.

Anaphylactic shock is acute immediate allergic reaction, arising in reply to repeated introduction of the allergen into organism as a result mediators, causing impairments of functions of vital organs and systems (cardiovascular, respiratory, central nervous, etc.) are allocated.

Anaphylactoid shock is acute generalized nonspecific on external influences, inducing formation and allocation of mediators of immediate hypersensitivity, causing semiology similar to that in anaphylactic shock reaction.

Classification of anaphylactic and anaphylactoid shocks

Clinical variants:

1. Classical (arterial hypotension — collapse, impairment of consciousness up to coma, bronchospasm, involuntary dejection and urination).

2. Haemodynamic (pain in the heart as in stenocardia, arrhythmia, collapse).

3. Asphyctic (an attack of bronchial asthma or asthmatic status).

4. Abdominal (clinic of «acute stomach», peritonitis, pancreatitis, cholecystitis, etc., arising suddenly).

5. Cerebral (convulsive syndrome, vision impairments, short-term loss of consciousness, etc.).

LECTURE 21
SYMTOMATOLOGY AND DIAGNOSTICS OF ANEMIAS

Anaemia (from Greek *an* — «negation», *haema* — «blood») is a pathological condition characterised by reduction of maintenance of haemoglobin in blood volume unit, more often in simultaneous reduction of red blood cells (RBC) quantities that leads to development of oxygen starvation of tissues.

Classification of anemias

On a pathogenetic sign (on the occurrence mechanism)

1. Anemias owing to hemorrhage — posthemorrhagic anemias:
1) acute;
2) chronic.
2. Anemias owing to deranged formation of red blood cells and haemoglobin:
1) asiderotic anemia;
2) nutritional anemia due to vitamine B12 deficiency;
3) folate-achrestic anemia;
4) hypo — and aplastic (medullary insufficiency);
5) metaplastic (hemoblastosis, cancer metastasises in a bone marrow);
6) dyserythropoietic – nephrogenic.
3. Anemias owing to increased hemolysis-hemolytic:
1) hereditary, acquired;
2) acute, chronic.
4. Anemias of mixed genesis.

On a severity level:

1) slight — haemoglobin 110–90 g/l;
2) average — haemoglobin 90–70 g/l;
3) severe — haemoglobin less than 70 g/l.

On haemoglobin maintenance in red blood cells:

1. Normochromal — color index of blood (CI) — 0,85–1,05; average maintenance of Hb in RBC (MCH) — 26–34 (pg), average concentration of Hb in one red blood cell (MCHC) — 31–37 %.

2. Hypochromic — color index of blood (CI) — 0,8 and lower low; MCH — less than 26 pg; MCHC less than 31 %.

3. Hyperchromic anemia — color index of blood — more than 1,1; MCH — more than 34 pg; MCHC — more than 37 %.

On the size of RBC:

1. Normocytic anemia — diameter — 7,2–7,5 microns; average volume of RBC (MCV) — 81 99 mcm^3.

2. Microcytic anemia — diameter — is less than 6,5 microns; MCV — less than 80 mcm^3.

3. Macrocytic anemia — diameter — more than 8 microns; MCV — more than 100 mcm^3.

Laboratory signs of anemic syndrome

— Decrease in level of haemoglobin and quantity of RBC.
— Change of color index of blood.
— Changes of the sizes and the form of RBC.
— ESR acceleration.

Acute posthemorrhagic anaemia

It is anaemia, which develops as a result of massive unitary or repeated (during a short term) of hemorrhage (not less than 300–400 ml of blood).

Reasons of acute posthemorrhagic anemia:

1) traumas, operations;
2) gastrointestinal bleedings;
3) pulmonary bleedings;
4) metrorrhagia;
5) nephritic bleedings;
6) hemostasipathy.

Clinical syndromes of acute posthemorrhagic anemias

1. Bleeding syndrome — external or internal.

2. Syndrome of acute cardiovascular collapse (syncope, collapse, posthemorrhagic shock).

3. Aanemic syndrome.

Haemogram in acute posthemorrhagic anemia

1st phase — phase of reflex, vascular compensatory — the 1st 1–2 days after hemorrhage. Changes are absent or insignificant: moderate decrease in concentration of haemoglobin and the number of RBC; color index of blood and hematocrit are normal (as before the hemorrhage).

2nd phase — phase of hydremic compensation (lasts for about 4–5 days): haemoglobin, RBC and hematocrit levels are considerably lowered; color index of blood is normal (normochromal anaemia).

3rd phase — a phase of marrowy compensation (last for 5–7 days): haemoglobin, RBC and hematocrit level sare lowered; reticulocytosis, normoblastosis; anisocytosis and poikilocytosis of RBC; hypochromia of RBC (after massive hemorrhage); neutrophilic leukocytosis with a shift of the nuclear formula to the left; thrombocytosis (is possible).

Chronic posthemorrhagic (asiderotic) anaemia

It is anaemia owing to obvious or imperceptible (concealed) chronic hemorrhage which finally leads to considerable loss of iron and development of asiderotic conditions.

Aetiology of asiderotic anemias

I. Hemorrhage — primary etiological factor.

Reasons of chronic hemorrhage:

1. Voluminous menstruation bleedings.
2. Delivery.
3. Concealed bleedings from the gastrointestinal tract (ulceration, erosion, tumours).
4. Bleedings in hemostasipathy.

II. Other reasons of occurrence of asiderotic conditions.

1. Hereditary deficiency of iron — low level of iron in the organism of mother during pregnancy.
2. Disturbance of absorption of iron in stomach pathology (achlorhydric condition) and intestines (enterites, diarrhoeia).
3. Pregnancy, lactation.
4. Chronic infectious-inflammatory and autoimmune diseases.
5. Tumours of any localization.
6. Alimentary insufficiency — lack of meat food, an unbalanced food.
7. Excessive physical activities.
8. Impairments of transport of iron (decrease in concentration and functions of transferrin).

Clinic of asiderotic anemias

It includes 2 syndromes — anemic and sideropenic.

Features of anemic syndrome:

— original colour of the skin — pale with a flavovirent shade (chlorosis);

— flavovirent colouring of the skin round the mouth or in the area of nasolabial triangle («yellow moustaches of the chlorotic» — symptom of Heano de Mussi).

Sideropenic syndrome — syndrome caused by reduction of iron concentration in the organism.

Normal maintenance of iron in blood serum: men — 8,8–28,6 mmol/l; women — 7,2–26,8 mmol/l.

Signs of syndrome sideropenic:

1. Change of taste (pica chlorotica).
2. Change of olfaction.
3. Xerosis and desquamation of extremities skin, angular stomatitis.
4. Changes of nails — koilonychia (spoon nail).
5. Changes of hair.
6. Decrease in gastric secretion.
7. Muscular weakness.
8. Weakness of smooth muscles (urine incontience, imperative desires for urination, dysphagy).
9. Pain and burning in the tongue, its reddening, smoothness of papillae (the «varnished» tongue).
10. Symptom of «dark blue scleras» of Osler.
11. Mild pyrexia.
12. Susceptibility to infection (manifestations of immunologic deficiency).

Laboratory diagnostics of asiderotic anemias

Haemogram:
— Decrease of haemoglobin content and quantity of RBC.
— Color index of blood is less than 0,8 (hypochromic anaemia).
— Anisocytosis and poikilocytosis of RBC, tendency to microcytosis.
— Insignificant leukocytopenia.
— Quantity of platelets is normal.
Myelogram. Characteristic changes are not present.
Biochemical analysis of blood:
— Decrease of iron serum concentration.
— Decrease of ferritin serum concentration.
— Increase of general iron binding capacity of blood serum.

Aetiology of nutritional anemia due to vitamine B12 deficiency

I. Disturbance of vitamin B_{12} intestinal absorption.

Reasons: atrophy of stomach mucous membrane — autoimmune gastritis, diffuse Hp-associated gastritis, regular reception of concentrated spirit ethyl; a resection of not less than ¾ of the stomach or gastrectomy; severe enteritis or other diseases of intestines which are manifested by diarrhoeia, malabsorption syndrome; resection of ileum guts.

— Syndrome of Imerslund-Gresbeka — hereditary disturbance of vitamin B_{12} absorption, combined with proteinuria.

II. Competitive expense of vitamin B_{12}.

Reasons: invasion of broad tapeworm (Dyphylobotrius latus); diverticulosis of intestines or syndrome of «blind loop»; repeated pregnancy.

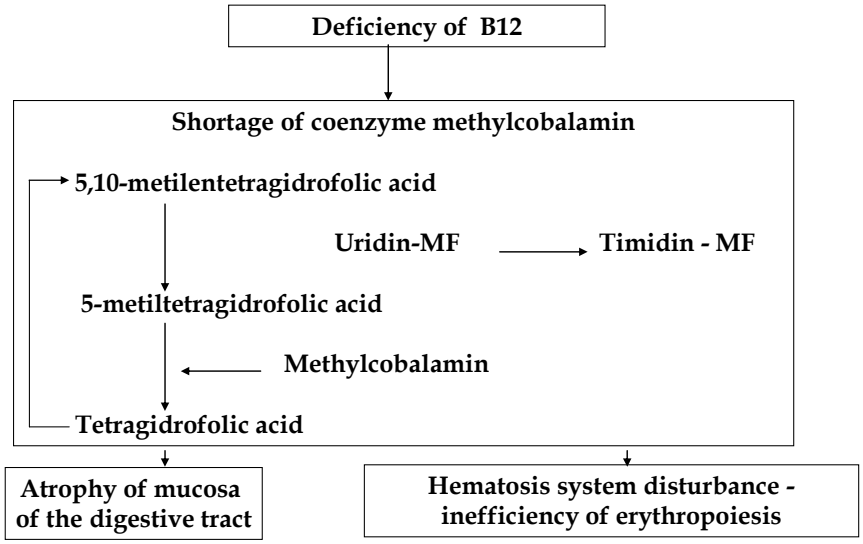
III. Impairment of vitamin B_{12} transport by blood.

Reasons: hereditary insufficiency of transcobalamins; deranged biosynthesis of transcobalamins in the liver (hepatites and cirrhoses).

IV. Impairment of vitamin B_{12} deposition.

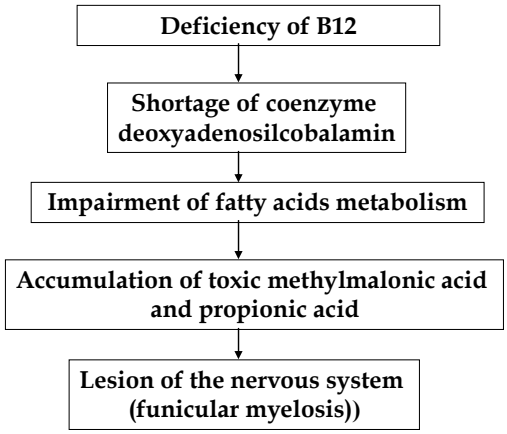
Reasons: severe chronic diseases of the liver (hepatites and cirrhoses).

The scheme of vitamin B_{12} deficiency pathogenesis is shown in the picture 5.



Picture 5 — The scheme of vitamin B_{12} deficiency pathogenesis

The pathogenesis scheme of funicular myelosis is shown in the picture 6.



Picture 6 — Pathogenesis scheme of funicular myelosis

Clinical syndromes of nutritional anemia due to vitamine B_{12} deficiency

1. Anemic.
2. Gastroenterologic (glossitis, atrophic gastritis and enterocolitis).
3. Neurological (funicular myelosis).

Haemogram in nutritional anemia due to vitamine B_{12} deficiency

— Sharp decrease of RBC and haemoglobin.

— Color index of blood always exceeds 1,1 (hyperchromic anemia).

— Macrocytosis or megalocytosis of RBC, displacement of Price-Jonsa curve to the right.

— There are Kebot's rings, Jolly's corpuscles, basophilic stippling in RBC.

— Anisocytosis and poikilocytosis of RBC.

— Decrease of reticulocytes quantity up to their full absence.

— Leukocytopenia, granulocytopenia, aneosinophilia, monocytopenia, giant polysegmental neutrophils.

— Relative lymphocytosis.

— Thrombocytopenia.

Features of the biochemical analysis of blood:

— Increase of unconjugated bilirubin level.

— Iron level is normal or raised.

Myelogram in nutritional anemia due to vitamine B_{12} deficiency

— Quantity of myelokaryocytes is increased or normal ($40–240 \times 10^9$/l).

— Red shoot of bone marrow is extended: ratio leyko/eritro — 1:1 – 0,5:1 (norm — 4:1 – 3:1).

— Type of hematosis is megaloblastic.

Aetiology of folate-achrestic anemias

1. Disturbance of folic acid absorption.

Reasons: resection of big part of the small intestine, especially jejunum;

— chronic diarrhoeia; celiac disease; a long period of time use of anticonvulsants (difenine, phenobarbital), of biseptol, of metotrexate, of cycloserine; alcoholism.

2. Insufficient receipt of folic acid with foodstuff.

Reasons: insufficient use of meat and vegetables; inn newborns at prematurity, impairment of intestinal absorption, rearing with goat's milk.

3. Raised expense of acid folic.

Reasons: chronic inflammatory diseases; hemolytic anemias; exfoliative dermatitis; pregnancy; diverticulosis of intestines, syndrome of «blind loop»; intestines tumours.

Features of clinic of folate-achrestic anemias

1. There is no neurologic syndrome.

2. The gastroenterological syndrome is less expressed.

3. Features of myelogram: megaloblasts are not painted with red alizarin.

LECTURE 22
SYMTOMATOLOGY AND DIAGNOSTICS OF HEMORRHAGIC DIATHESIS

Hemorrhagic syndrome is a pathological complex of symptoms, characterised by liable to bleeding and recurrent bleedings arising both spontaneously, and under the influence of insignificant traumas, not capable to cause bleeding in a healthy person.

Hemorrhagic hemostasiopathy are impairments of hemostasis system, occuring spontaneously or after traumas with bleedings owing to hereditary or acquired defect of coagulation, thrombocyte or vascular components of this system.

Hemostasis system is the system of the organism with functional feature: on the one hand, of prevention and stop of bleedings by maintening structural integrity of vessel walls and fast local thrombus formation in vascular injuries, and on the other hand, insuring liquid condition of blood.

The basic functions of hemostasis system:

— Regulation of resistance and permeability of a vascular wall.

— Maintenance of blood in a liquid condition.

— Arrest of spontaneous and posttraumatic bleedings and hemorrhages in the tissue.

Functional-structural components of the hemostasis system are:

— A vascular wall — the borders of the system.

— Thrombocytes and other blood cells; structural forms.

— Plasma and tissue factors — blood coagulation system and anticoagulation blood system.

— Fibrinolytic system.

Classification of hemorrhagic hemostasiopathy

1. Pathology of thrombocyte hemostasis.

1.1. Thrombocytopenia — decrease of thrombocytes quantity below 150×10^9/l: hereditary, acquired.

1.2. Thrombocytopathy — structural and functional impairments of the thrombocytes: hereditary, acquired.

2. Pathology of blood coagulation system or coagulation hemostasis (coagulopathy): hereditary, acquired.

3. Vascular wall pathology (angiopathy): hereditary, acquired.

4. Mixed (polycomponents) hemostasiopathy: hereditary, acquired.

Thrombocyte vascular hemostasis

The indicators characterising tromboocyte vascular hemostasis:

1. Quantity of thrombocytes (norm 180-320 $\times 10^9$/l): hypocoagulation — less than 140×10^9/l; hypercoagulation - more than 450×10^9/l.

2. Duration of a bleeding by Duke (norm — 2–3 minutes): hypercoagulation — is not present; hypocoagulation — more than 4 minutes.

3. Tests to define of thrombocyte aggregative activity.

4. Tests on resistance (fragility) of capillaries — excisional test, tourniquet test, cup test, Nesterov's, Konchalovsky's.

5. Retraction a blood clot (norm — 48–60 %): hypercoagulation — less than 48 %; hypocoagulation — more than 60 %.

Thrombocytopenia — decrease of thrombocyte quantity in peripheral blood lower 150×10^9/l.

Types of thrombocytopenia:

— Immune thrombocytopenia.

— Thrombocytopenia in marrowy hematosis oppression (marrow insufficiency) — aplastic anemias, influence of chemical substances and ionising radiation, vitamin B_{12} and folic acid deficiency.

— Metaplastic thrombocytopenia, caused by replacement of bone marrow by tumour cells in hemoblastosis and growth of the tumours in the bone marrow.

Clinic of thrombocytopenia

1. Petehial-spotty (microcirculatory) type of hemorrhagic diathesis — petehial rash and cyanotic skin and mucous membranes, long and plentiful nasal, stomatorrhagia, metrostaxis, nephritic, intestinal bleedings.

2. General analysis of blood: thrombocytopenia, posthemorrhagic anemia.

3. Melogram: the increased quantity of megalokaryocyte, infringement of thrombocyte separation.

Coagulation hemostasia

Haemocoagulation phases

1st phase — formation of prothrombinase.

2nd phase — formation of thrombin.

3rd phase — formation of fibrin.

4th phase — postcoagulation.

Coagulogram volume

1st phase of blood coagulation.

1. Blood coagulation time (norm — 6–12 minutes): hypercoagulation — less than 3 minutes; hypocoagulation — more than 12 minutes.

2. The autocoagulation test at minute 8 or 10 minute (norm — 7–11 seconds): hypercoagulation — less than 5 seconds; hypocoagulation — more than 13 seconds.

3. Activated partial time of thromboplastin (norm — 35–42 seconds): hypercoagulation — less than 30 seconds; hypocoagulation — more than 42 seconds.

4. Time of plasmas recalcification (norm — 80–140 seconds): hypercoagulation — less than 60 seconds; hypocoagulation — more than 140 seconds.

2nd phase of blood coagulation.

1. Prothrombin index (norm 0,85–1,05): hypercoagulation — more than 1,1; hypocoagulation — less than 0,7.

3rd phase of blood coagulation.

1. Concentration of fibrinogen A in plasma (norm — 2–4 g/l): hypercoagulation — more than 4 g/l; hypocoagulation — less than 1,5 g/l.

2. Fibrinase activity of, XIII factor (norm — 60–80 seconds): hypercoagulation — more than 100 seconds; hypocoagulation — less than 60 seconds.

3. Thrombin time (norm — 12–16 seconds): hypocoagulation — more than 16 seconds; hypercoagulation — less than 12 seconds.

4th phase of blood coagulation (postcoagulation).

1. Blood clot retraction (norm — 48–60 %): hypercoagulation — less than 48 %; hypocoagulation — more than 60 %.

2. Spontaneous fibrinolysis — fibrinolytic activity (norm — 10–20 %): hypercoagulation — less than 10 %; hypocoagulation — more than 20 %.

Clinic of coagulopathy (hemophilias A, B, C)

Macrocirculatory (hematoma) type of hemorrhagic diathesis: hematomas and hemarthroses spontaneous or after traumas; bleedings from mucous membranes (mouth, pharynx, stomach, intestines) spontaneous or in an inflammation; late bleedings.

Clinic of hereditary angiopathy (hemorrhagica telangiectasia - Rondju-Veber-Osler's disease)

Angiomatous type of hemorrhagic diathesis: burdened hereditary anamnesis, nasal bleedings, long, plentiful, frequent, leading to posthemorrhagic anemias and iron deficiency, cirrhosis.

Clinical picture of acquired angiopathy (hemorrhagic vasculitis — Schönlein-Henoch disease)

Vaskulitis-purpuric type of hemorrhagic diathesis: 1st — the purple, then painted in red-brown colour papule, is more rare — with necrosis, then — flowering purples. Localisation: hips, shins, but other sites of the body are possible.

LECTURE 23
SYMTOMATOLOGY AND DIAGNOSTICS OF HIV-INFECTION

Human immunodeficiency virus (HIV)-infection — infectious process in the human organism, caused by a HIV, characterised by a slow current, lesion of the immune and nervous systems, subsequent development on this background of opportunist infections, neoplasms, leading the infected with HIV to a lethal outcome.

AIDS — the terminal phase of HIV-infection coming in most cases after a rather long period from the moment of catching the virus.

Features of Human immunodeficiency virus

— A spherical form with mace-shaped receptors (superficial receptors), possessing complementation to the cells of the owner, having on their surface fiber-receptor CD-4 (helper T-lymphocyte, monocyte — macrophages in the skin, CNS, intestines, vessels).

— Has enzyme **reverse transcriptase** which «transforms» virus RNA into DNA.

— Enzyme **integraase** «builds in» virus DNA into the owner's cell DNA (a fatal moment for the sufferer). The received defective DNA becomes a part of a hereditary substance of the owner!

Clinical classification of the Human immunodeficiency virus-infection

I. Acute infection.

Appears mostly within 6–12 weeks after the patient becomes infected, but can appear in a week or 8–12 months, and more. A clinical picture is mostly manifested by mononucleosis-like syndrome with or without aseptic meningitis, or this stage proceeds in a subclinical form.

II. Asymptomatic infection (virus infection carrier state).

It is characterized by the absence of any clinical manifestations and HIV-infection symptoms.

Persons are referred to this group on the basis of epidemiological history and laboratory findings data. The latter include search for antibodies to HIV in serum, the analysis of formed elements of blood with the purpose of revealing lymphopenia and thrombocytopenia, immunological methods, defining quantity and correlation of helper T-lymphocyte and T-suppressor, the analysis of antibody fractions.

III. Persistent generalized lymphadenopathy.

It is characterized by expressed lymphadenopathy within 3 and more months in persons with epidemiological data indication possibility of HIV infection in the absence of any other infections and visible reasons.

IV. AIDS — associated symptom complex (pre-AIDS).

In this stage of the disease signs of opportunistic infections or tumoral malignancy characteristic of developed AIDS presentation, are not still observed.

It is characterized by:

1) «constitutive» conditions:

— Weight loss 10 % and more.

— Inexplicable sub-or febrile fever within 3 months and more.

— Unmotivated diarrhoea lasting more than 1 month.

— Syndrome of chronic weariness.

2) secondary diseases:

— Fungoid, virus, bacterial defeats of the skin and mucous.

— Repeated or disseminated zoster, localised Kaposi's sarcoma.

— Pilary leukoplakia.

— Recurrent pharyngitises and a sinusitis.

— Lung tuberculosis.

— Recurrent or persistent viral, bacterial, fungous, protozoic diseases of internal organs.

V. AIDS.

It is characterised by the development of opportunistic infections and tumours, resulting from deep cellular immunodeficiency. All these stages can appear inconsistently and their presence is not absolutely necessary in all infected.

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БОЛЕЗНЕЙ
(на английском языке)

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