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**DIAGNOSTICS AND TREATMENT OF INTERNAL  
DISEASES IN THE ELDERLY  
(gastroenterology, pulmonology, hematology)**

Training manual

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and it is recommended for the use in educational process for foreign students.*

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The training manual was prepared in accordance with the requirements of the new program of the discipline "Internal Medicine" for higher medical schools of III-IV levels of accreditation, approved by the Ministry of Health of Ukraine (2016). The manual contains training materials on the topic "Diagnosics and treatment of internal diseases in the elderly." The physiological changes in the aging digestive, respiratory, blood and blood-forming organs, the features of diagnosics and treatment in the elderly, the pharmacokinetics and pharmacodynamics of medications are highlighted. The methodological and educational materials presented in the manual will allow students not only to learn the basic gerontological aspects in the clinic of internal diseases, but also to gain practical skills in the supervision in elderly patients.

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## CONTENT

Introduction.....	4
1. Actuality .....	5
2. Learning objectives .....	6
3. Materials for classroom self-study work .....	8
3.1. List of basic terms and characteristics that student must acquire.....	8
3.2. Content of the topic.....	9
3.3. List of recommended literature .....	40
3.4. Materials for student self-assessment at the classroom stage .....	44
4. Materials for classroom self-study work.....	56
4.1. Practical assignments students carry out on practical training .....	56
4.2. Methodological support for self-study work of students at the stages of practical training.....	58
5. Tasks and materials for extracurricular self-study work .....	58
Annex 1. Abbreviations used in the results of laboratory and instrumental studies.....	59
Annex 2. Main laboratory values and their interpretation.....	62
Annex 3. Schemes of describing the main instrumental studies.....	84
Annex 4. Examples of interpretation of laboratory and instrumental studies .....	91
Annex 5. Examples of situational tasks and rationales for their solving .....	101

## **INTRODUCTION**

The expediency of preparing the training manual is due to the need to make changes to the organization of the educational process in accordance with the requirements of the new curriculum discipline “Internal Medicine”, approved by the Ministry of Health of Ukraine in 2016. The main component of these changes in the program is the addition to the content module-5 the topic of etiology, pathogenesis, and features of the clinical manifestations of the main internal diseases in the elderly, assuming students are to master theoretical knowledge, to acquire skills of supervision, physical examination of elderly patients, interpreting laboratory results and instrumental studies, methods of differential diagnosis and rationale for the clinical diagnosis, prevention and treatment of the elderly patients based on the principles of evidence-based medicine, and to acquire knowledge on issues of medical ethics and deontology.

## 1. Actuality

The planet's population is rapidly aging. According to the WHO, by 2050 the number of elderly people in the world will increase to 38% and times, when such people prevail in the general population, will quickly come. In EU countries, the life expectancy of men is 74 - 77 years, women 80 - 82 years. The level of health and especially morbidity in the elderly are due to an increase in the number of chronic diseases [1-2]. According to the WHO, more than 40% of people aged 50-65 have 4-5 diseases, and 66% of people over 75 have more than 5. The sickness rate in the elderly (60-74 years) is almost 2 times higher, and in senile (75 years and older) - 6 times higher than in young people [3]. Ukraine belongs to the demographically old countries of the world. The proportion of the population, who are 60 years and older, is 20.3%. According to WHO, by the middle of this century, this percentage is expected to increase in Ukraine to 38.1%, and the proportion of people who are 80 years and older will increase by 3.5 times. Aging of the population is closely related to an increase in the prevalence of age-dependent pathology, especially circulatory system diseases, which take first place in prevalence, which determine more than half of all deaths and a third of cases of disability.

The purpose of the publication of this manual is to help students study the aging process, age-related physiological changes in organs and systems, the characteristics of the course of diseases, and acquire skills for diagnostics and treatment in the elderly and senile patients.

## **2. Learning objectives:**

- Students will learn the features of development, course, symptoms, clinic, diagnostics and treatment of diseases in the elderly.
- Students will acquire skills of history taking in the elderly and senile patients.
- To teach students to interpret the results of objective examination, laboratory and instrumental studies in the elderly and senile patients.
- Students will understand features of pharmacodynamics in patients of older age groups.
- Students will study the principles of drug therapy in elderly patients.

### **Student must know:**

- Physiological characteristics of the elderly.
- Features of pathology in the elderly.
- Features of comorbid pathology in the elderly
- Psychological features of the geriatric patient
- Features of the development, course, symptoms, clinic, diagnostics and treatment of diseases in the elderly.
- Features of the collection of anamnesis in elderly patients
- Interpretation of the results of objective examination, laboratory and instrumental studies in the elderly patients.
- Features of pharmacodynamics in patients of older age groups.
- Features of the effect of drugs on the body of an elderly person.
- Principles of drug therapy in the treatment of the elderly.
- Emergency care algorithms for the elderly patients.

### **Student must be able to:**

- Take history, perform physical examination in the elderly patients and diagnose the most common diseases of the digestive, respiratory, blood and blood-forming organs.

- Identify a typical clinical picture of the most common diseases of the internal organs in the elderly.
- Identify types of the course and complications of the most common internal diseases in the elderly.
- Make a preliminary diagnosis for the most common diseases of the internal organs in the elderly.
- Draw up an examination plan, justify the need for use, determine indications and contraindications, possible complications when performing invasive and non-invasive diagnostic methods in elderly patients.
- Interpret laboratory and instrumental studies in elderly patients.
- Make differential diagnosis based on analysis of the results of clinical, laboratory and instrumental examination.
- To reason and formulate a diagnosis for the most common diseases of the internal organs in the elderly.
- Prescribe treatment, carry out primary and secondary prevention of the most common diseases of the internal organs in the elderly.
- Assess the prognosis and performance for the most common diseases in the elderly.
- Diagnose and provide medical care in emergency situations for the most common diseases in the elderly.
- Perform medical manipulations according to the topic of practical training.
- Obtain medical records in accordance with the requirements of the discipline program.
- Demonstrate knowledge of the moral and ethical principles of a medical specialist and the principles of professional subordination.

### **3. Materials for the classroom self-study work.**

#### **3.1. . List of basic terms and characteristics that student must acquire**

Term	Definition
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1. Gerontology	A science that studies the processes of aging in accordance with the biological, physical and spiritual characteristics of people. Their social significance.
2. Geriatrics	a specialty that focuses on health care of elderly people. The main task of geriatrics is to study the features of development, clinic, treatment, prevention of various diseases in elderly and senile patients; elucidation of factors that possibly affect the processes of premature aging.
3. Gerohygiene	A discipline of gerontology that studies the influence of living conditions on human aging processes and develops measures aimed at preventing premature aging, creating conditions that will ensure effective longevity for the population.
4. WHO age classification (2015)	25-44 - young age; 44-60 - average age; 60-75 - old age; 75-90 - senile age; After 90 – long-livers.
5. Chronological age	The period of time (in years, months, days) lived from the day of birth until a certain measured moment.
6. Biological age	The set of anatomical and physiological characteristics of the body that meet the age norms for this population. It depends on the individual growth rate, development and aging of the body. The difference between chronological and biological age at the maturation stage can reach five years, and at the aging stage - up to 20 years. If the biological age is significantly ahead of the chronological, this indicates premature aging.
7. Aging	This is a combination of various mechanisms at the molecular, cellular, organ and systemic levels that lead to involutive changes in organs and tissues with the extinction of body functions.
8. Processes of anti-aging (vitauctus)	Along with the destruction and degradation that are characteristic of the aging process, in the course of evolution processes arose that aimed at the survival and increase of the stability of biological systems. These are the mechanisms of anti-aging, or vitauctus (from: vita - life, auctum - increase). For the first time, an outstanding Ukrainian gerontologist V.V. Frolkis,



	<p>who proposed the term "vitauct." All mechanisms of vitauct can be divided into two groups: genotypic and phenotypic.</p> <p>These are processes that stabilize the life of the body, increase its reliability, aimed at preventing damage to living systems with age and increasing life expectancy.</p>
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### **3.2. Content of the topic.**

In Ukraine, as in many countries of the world, there is a rapid pace of population aging, and the proportion of people of retirement age is increasing [1-3]. This has become a state problem, the solution of which requires an integrated approach. Medicine and education are faced with the task of training specialists who are well versed in the psychology of elderly people, the physiological age-related changes in the aging organism, the course of diseases of various organs and systems in them, emergency care in case of complications, the methods of preventing these diseases, the main principles of pharmacotherapy [5,6].

#### **Structural and functional changes in organs and organ systems in the aging process.**

Structural and functional changes in organs and organ systems during aging are mainly destructive processes and various compensatory-adaptive mechanisms.

**Cardiovascular and circulatory systems.** Blood pressure, mainly systolic, increases with age [5]. The heart rate slows down. Sclerotic processes in the vessels are combined with atherosclerotic changes, which are more pronounced on the lower extremities. The total amount and chemical composition of the blood remains almost unchanged. In the blood coagulation system in the elderly people the dynamic balance of hemostasis decreases with an increased risk of thromboembolic complications. Hypercoagulation is combined with hyperlipidemia. The content of endogenous cholesterol and triglycerides increases. The incidence of myocardial infarction, other forms of coronary heart disease, complications of hypertension, and vascular pathology of the brain is growing. Tolerance to carbohydrates changes, which often coincides with the occurrence of

type II diabetes mellitus. There is a restructuring of neurohumoral regulation, new compensatory-adaptive mechanisms are forming in the circulatory system.

**Respiratory system.** There is a deformation of the chest, atrophy of the intercostal muscles and diaphragm. The lungs are reduced in size, the elasticity of the elastic fibers, the structure of the alveoli changes, accompanied by the disappearance of the interalveolar septa, the expansion of the alveolar ducts. There is a decrease in the gas exchange surface and a decrease in the vital capacity of the lungs, tidal volume, reserve volume of inspiration and expiration, while the frequency of respiration increases. The functionality of the respiratory system decreases with the growth of hypoxia and hypercapnia, especially on physical exertion. Violation of bronchial obstruction, decreased elasticity and the formation of pulmonary fibrosis cause uneven ventilation of the lungs. In old age, a mismatch between ventilation and perfusion may occur, which is accompanied by a decrease in O<sub>2</sub> voltage with an increase in CO<sub>2</sub> in arterial blood.

**Digestive system.** A decrease in the activity of the secretory apparatus of various parts of the gastrointestinal tract, liver and pancreas is observed. Their enzymatic activity decreases. The motor activity of various departments of the gastrointestinal tract decreases, the intensity of the digestion and absorption processes in the intestine decreases.

**Musculoskeletal system.** Dystrophic-destructive changes in bone tissue (osteoporosis, osteochondrosis) develop, skeletal muscle contractility decreases, their atrophy becomes more pronounced, which leads to impaired mobility manifested by slower movements, tremor of the hands, head, difficulty in performing fine movements.

**Nervous system.** Morphological and functional changes in the brain lead to certain changes in mental processes in old age. The process of internal inhibition is weakened, manifested in intemperance, emotional lability, irritability, verbosity.

General features of the course of internal diseases in elderly patients.

Old age, as a general biological process, cannot be equated with a disease.

Asymptomatic age-related changes, malaise, which do not have clinical manifestations of the disease, are often observed in practically healthy elderly people. Diseases in people of old age have a chronic course, frequent complications and concomitant diseases; subjective and objective symptoms are much less pronounced than in people of mature and middle age.

**The list of features of the course of internal diseases in elderly patients (M.D. Strazhesko) [1]:**

1. The symptoms of various diseases (subjective and objective, subjective-objective) in old age are much poorer than in adulthood;
2. All diseases have a latent and prolonged course;
3. During illness, physiological systems capable of 'fighting' a harmful factor are rapidly depleted;
4. During infection, the immune system is not able to provide the rapid development of humoral and tissue immunity and cannot guarantee the course of energy processes in various diseases at the same level as in adulthood.

Chronic diseases predominate in the elderly: atherosclerosis, cardiosclerosis, arterial hypertension, cerebral vascular lesions, mental (hydrocyanic) depression, pulmonary emphysema, chronic atrophic gastritis, cholelithiasis, type 2 diabetes mellitus, eye diseases (cataract, glaucoma), neoplasms and others.

**General pathology features in the elderly. [1, 4]**

- Most patients suffer from chronic diseases (from 4 to 10) - polymorbidity, which complicates their course and conducting rational and safe pharmacotherapy.
- Chronic diseases and their monosymptomatics, latent course dominate.
- Atypicality, areactivity, and latency of the disease course are characteristic.
- The chronicity of acute diseases is characteristic (at first the diseases have a protracted course).
- An organism with immune deficiency demonstrates areactivity.
- In the first place there is a cardiovascular pathology.

- In people of older age groups diagnostics (and so does treatment) requires a different approach than in patients of a young age.

**These features of the pathology in the elderly are often caused by:**

- slower and often masked course of pathological processes of internal diseases (pneumonia, myocardial infarction, diabetes mellitus, etc.);

- other genesis and course of gastric ulcer, which develops on the background of atherosclerosis;

- the influence of clinically pronounced age-related changes in the bones and joints of the spine, causing circulatory disorders in the main vessels and provoking symptoms for the erroneous diagnosis of heart disease;

- the latent course of abdominal catastrophes requiring urgent surgical interventions;

- acute diseases often take a chronic course;

- the severity of damage to the body does not correspond to the slightly expressed manifestations of the disease;

- pharmacokinetics in the elderly is largely due to the development of the anacidic stomach, a violation of the absorption process in the digestive tract, a decrease in the amount of fluid in the body, an increase in the number of fat cells, and a disturbance in the excretion process (the number of functioning nephrons is reduced).

**Features of the clinical course of respiratory diseases in elderly patients.[15]**

**Chronic obstructive pulmonary disease (COPD).** Age-related changes in lung function worsen the course of COPD and, combined with a decrease in cardiac output, and changes in the central nervous system, lead to decreased oxygen saturation of arterial blood, a decreased lung response to hypoxia and hypercapnia, and exercise intolerance. A manifestation of general involutive changes in the lungs is age-related emphysema, in which the main process is a loss of elastic properties of the lung tissue. The increase in air content in the lungs is due to atrophy of the lung tissue (interalveolar septum) and changes in the thoracic

spine. In the history of patients with age-related emphysema, there are no chronic lung diseases and, above all, chronic bronchitis.

Obstructive emphysema (chronic), due to impaired patency of the bronchi with overstretching of the lung tissue, is primary (due to hereditary factors) and secondary - for obstructive pulmonary diseases, often causing chronic pulmonary heart disease.

In the elderly, the severity and value of the main symptoms of COPD is reduced. There is a decrease in the cough reflex. A significant proportion of patients with COPD does not complain of breath shortness at all due to self-restriction of activity, an age-related decrease in the sensitivity of the respiratory center to hypoxia. In older people, coughing can be a manifestation of other diseases (tuberculosis, bronchial asthma, lung cancer), and shortness of breath can be a manifestation of heart failure, anemia, and obesity.

Elderly people with a prolonged course of COPD have cachexia, a decrease in skeletal muscle mass, and osteoporosis. In the progression of osteoporosis in COPD, the leading role is played by the chronic inflammatory process with an increase in the level of pro-inflammatory cytokines and proteolytic enzymes that disrupt bone metabolism, decrease in physical activity, inadequate nutrition, the development of "chronic disease anemia", cytokine-induced anemia. In patients with severe pulmonary insufficiency, compensatory erythrocytosis may develop due to the stimulating effect of hypoxia on the production of erythropoietin. Erythrocytosis causes a violation of the rheological properties of blood, contributes to compensatory hypoxic vasoconstriction with pulmonary hypertension.

Gastroesophageal reflux disease (GERD) can also be a cause of coughing in older people, which requires an upper endoscopy, 24-hour esophageal pH test and a proton pump inhibitor test in parallel with peak flow monitoring of bronchial obstruction. The principles of treatment of COPD in elderly patients are consistent with the new recommendations of GOLD -2017-2018.

**Pneumonia.** The main features of the course of pneumonia:

- absence of hyperthermia, cough;
  - The presence and intensification of breath shortness can be a fairly sensitive symptom of pneumonia.
  - Classic signs - dullness of percussion sound, crepitus - are not always clearly expressed;
  - The prevalence of extrapulmonary symptoms from the central nervous system - apathy, drowsiness, lethargy, loss of appetite, confusion up to the development of a soporous state, and the cardiovascular system - increased shortness of breath, weakness and manifestations of decompensation;
  - No changes in the peripheral blood
  - Lack of typical radiological changes.
  - The treatment of pneumonia in the elderly does not have any significant features.
- Sputum changes in some lung diseases:

Nosological form	Amount of sputum	Sputum characteristic	Macroscopic examination	Microscopic examination
Chronic bronchitis	Different	Mucopurulent, mucopurulent with blood		Leukocytes – large amount; erythrocytes, a lot of bacteria, macrophages
Bronchiectasis	Large, "full mouth"	Mucopurulent, with different layers	Dietrich's plugs	White blood cells - a whole field of view, crystals of fatty acids, crystals of hematoidin, cholesterol; heterogeneous flora, massive
Bronchial asthma	Very small	Mucous	Curschmann's spirals	Columnar epithelium, Charcot-Leyden crystals, eosinophils
Lobar pneumonia	Small in the beginning, massive later	Sticky, rust-colored in the beginning, later mucopurulent	Fibrin clots, altered blood	Macrophages, white blood cells, red blood cells, hematoidin crystals, hemosiderin grains,

				pneumococci
Lung abscess	Massive if abscess ruptures into bronchus	Purulent and fetid	Tissue debris	White blood cells - a whole field of view, crystals of fatty acids, crystals of hematoidin, cholesterol; heterogeneous flora, massive
Lung tuberculosis	Different	Mucopurulent, sometimes with blood	Lentil or rice-like bodies (Koch's lens) in cavernous tuberculosis	Detection of Mycobacterium tuberculosis, elastic fibers and various crystals
Lung cancer	Big amount in tumor rupture	Blood tinged, mucopurulent with blood	Tissue debris	Atypic cells

### **Features of the course of circulatory system diseases in the elderly.**

Age-related changes significantly limit the adaptive capabilities of the physiological systems of the body. The heart rate slows down, on exertion, the necessary acceleration of the rate does not occur, which limits the functional abilities of the heart. With tachycardia, a mismatch quickly arises between the blood supply to the coronary vessels and rapidly enhanced metabolism in the heart, and conditions for ischemia are created.

**Arterial hypertension (AH).** [5, 10-14,16]. In elderly people, isolated systolic hypertension (ISH) is often diagnosed. Of great importance in the formation is hypoxia, atherosclerosis, progressive atherosclerosis. When examining such patients, there is a paucity of symptoms. Patients often complain of weakness, noise in the head and ears, unsteady gait, dizziness, shortness of breath on physical exertion, less often headache. Rarely, but there are hypertensive crises that are dangerous for the possibility of developing a stroke, acute heart failure.

Features of treatment of hypertension:

- Doses of antihypertensive medications should be 2 to 3 times less;
- Blood pressure should be gradually reduced by 30% (reducing blood pressure lower may worsen cerebral and renal failure);
- monitor treatment by direct measurement of blood pressure in the supine and standing position;
- monitor kidney function, electrolyte and carbohydrate metabolism;
- exclude medications that can suddenly lower the pressure and cause orthostatic collapse or carefully monitor patient;
- carefully prescribe beta-blockers, especially in the presence of diabetes mellitus and a tendency to bronchospasm, apply an individual approach taking into account polymorbidity.

**Coronary heart disease (CHD).** In the elderly and senile age, the most common form is stable angina, which can turn into unstable; an atypical course of stable angina pectoris is often found, especially when it occurs on the background of diabetes mellitus or hypothyroidism. In elderly patients, the frequency of painless angina pectoris increases (from 25% at the age of 60 - 69 years, up to 45% at the age of 80 - 89 years). In 40% of patients with a painless form of the disease, cardiac rhythm disturbances are detected.

Features of the course of an attack of angina pectoris: the emotional color of an attack of angina pectoris is dim (a sense of fear of death), often absent; non-intense compressive pain behind the sternum or in the area of the heart, often of an indefinite nature; unusual irradiation of pain (often in the jaw, neck, back of the head), an attack of angina pectoris is often associated with an increase in blood pressure; in some cases, neurological symptoms (cerebrovascular insufficiency) come to the fore; an attack can be triggered by meteorological factors, overeating; IHD (angina pectoris, atherosclerosis) in elderly patients contributes to the development and progression of heart failure and cardiac arrhythmias. To diagnose coronary heart disease, it is necessary to use instrumental examination (ECG at rest



and during physical exertion, echocardiography, coronary angiography).

Treatment is an individual approach according to the MOH protocols. Treatment begins with small doses of drugs, stenting and coronary artery bypass grafting if necessary.

### **Peculiarities of the course of myocardial infarction in elderly patients.**

The frequency of myocardial infarction (MI) increases with aging, repeated heart attacks often occur, and their course becomes more severe.

- The number of painless forms is growing. The pain may not be intense, without typical localization and irradiation. Atypical forms of MI with large necrosis often develop and are combined with impaired cerebral circulation.

- The prognosis of the disease in the elderly patients is significantly worsening.

- Cardiogenic shock can occur in any period of myocardial infarction.

- Deterioration of cerebral circulation leads to ischemic stroke.

- Often there are manifestations of heart failure, which have a severe course, mainly in the left ventricular type.

- In the acute period, rhythm disturbances often develop, which worsen the prognosis.

- Diagnosis of Q-MI with ECG is the same as in patients of a younger age, but in some cases it is not always informative.

- In elderly patients, enzyme activity may be negligible even with a large lesion of muscle tissue.

- The temperature and blood reactions are less pronounced, sometimes completely absent due to a decrease in the reactivity of the body.

The principles of care for patients with myocardial infarction - according to the guidelines of the Ministry of Health of Ukraine for the management of patients with acute coronary syndrome.

In people of older age groups, bed rest adversely affects the function of compensatory mechanisms, contributes to the development of pneumonia,

thrombophlebitis, pulmonary embolism. There is a tendency to develop bedsores, urinary tract infections.

### **Features of the course of digestive diseases in elderly patients [15]**

Atrophy of the face muscles and bones is observed, the bite changes, which makes chewing difficult; teeth are worn down, tooth enamel becomes yellow; tongue papillae are smoothed out; salivary glands atrophy, saliva production decreases, dry mouth, cracked tongue and lips occur.

The esophagus is characterized by moderate lengthening due to curvature of the thoracic spine and expansion of the aortic arch; atrophy of the mucous membrane of the esophagus, muscle fibers are replaced by connective tissue, especially in the middle part of the esophagus, which is manifested by some discomfort during eating; due to a decrease in the tone of the esophagus, an increase in the frequency of gastroesophageal reflux is observed.

Age-related changes in the stomach are characterized by a decrease in the thickness of the gastric mucosa, the number of secretory cells, a violation of the blood supply of the wall of the stomach, and atrophy of the muscle layer of the stomach. The secretory and motor functions of the stomach are reduced.

**Gastroesophageal reflux disease (GERD) in elderly patients.** The frequency of GERD and the severity of the course of the disease increases with age. This is due to the greater frequency of risk factors for the development of gastroesophageal reflux: an increase in the patient's body weight, an increase of the pressure in the abdominal cavity, weakening of the esophagus motility and a decrease in the tone of the lower esophageal sphincter, a high frequency of hiatal hernia, weakening of the barrier properties of the esophagus epithelium - the most important factor in protecting the esophagus from aggressive influence of gastric contents, microcirculatory disorders, which lead to a shift in tissue acid-base balance, reducing the amount of saliva and bicarbonate contents in it, taking medication (antihypertensive, nitrates, NSAIDs, acetylsalicylic acid). Erosive

esophagitis with atypical symptoms (anorexia, dysphagia, vomiting, progressive decrease in body weight) is more common, and the clinical manifestations of GERD in the elderly can be exclusively extra-esophageal: cardiac and pulmonary.

GERD can be a trigger for a cascade of pathological processes that initiate destabilization of coronary blood flow, myocardial ischemia due to viscerocardial reflex, which becomes the reason for the differential diagnosis of chest pains. Gastroesophageal reflux in patients with combined pathology often provokes potentially dangerous changes: ST segment depression and ventricular extrasystole. In patients with extraesophageal manifestations of GERD, a PPI test is of great diagnostic value.

Treatment is carried out according to standards, but the likelihood of drug interactions (clopidogrel and PPI, which are metabolized by cytochrome P450) increases.

**Chronic gastritis (CG).** In elderly patients, forms of gastritis with the presence of secretory insufficiency of the stomach predominate, atrophic gastritis (atrophic or atrophic with intestinal metaplasia) is more common, special forms are rigid antral gastritis, polypous and cystic gastritis, which do not have specific clinical symptoms and often occur latently or with the predominance of dyspeptic syndrome.

**Diagnostics.** First of all, to exclude gastric cancer, upper endoscopy with histomorphological confirmation of changes in the gastric mucosa, gastric pH test, X-ray examination.

CG treatment is carried out according to the standards, but depends on the state of the secretory function of the stomach - replacement therapy in the absence of hydrochloric acid and pepsin in the stomach (natural gastric juice 1 tablespoon 3-5 times a day in a glass of water with meals) or - diluted hydrochloric acid with pepsin, acidin-pepsin (during or after meals), 1 tab. 3-4 times a day, dissolving the tablet in 1/3 cup of water.

**Peptic ulcer.** Peptic ulcers in the elderly occur 3 times more often than in

the duodenum.

The main age-related factors of the development of peptic ulcer include: an impairment of the neurohumoral regulation of the stomach and duodenum; decrease in blood supply of the gastric mucosa; violation of trophism and salivation, reduced regenerative capabilities of epithelial cells of the mucous membrane.

The following ulcerative lesions are distinguished: "chronic" ulcer that has arisen at a young and middle age and exists for a long time; "Late" peptic ulcer, which developed in old or senile age; "Senile" peptic ulcers, which are symptomatic ulcers in chronic diseases of the circulatory system, respiratory system, and prolonged use of medications.

Localization of ulcers in the stomach is frequent. For a "long-standing" ulcer seasonal exacerbations are rarely detected and a constantly progressive type of the course of the disease with long periods of exacerbations is more common. Often, exacerbation is complicated by massive bleeding or perforation. Scarring of an ulcerative defect in elderly takes more time, and the frequency of exacerbations decreases.

"Late" ulcer has the following features:

- Some patients complain mainly of constant moderate pain, that does not depend on food intake;
- In some cases there is no pain, but dyspeptic syndrome predominates;
- Seasonality of relapses is absent;
- Ulcers are not deep, localized mainly in the upper parts of the stomach;
- Often for a long time they are not scarred and complicated by malignancy, perforation or bleeding;
- In almost 50% of elderly patients, peptic ulcer is accompanied by hidden bleeding, perforation;
- History and objective examination are uninformative;
- For diagnostics, it is advisable to carry out x-ray and endoscopic

examination, which complement each other.

Before performing upper endoscopy, it is necessary to record an ECG in order to avoid complications in the presence of contraindications.

“Senile” ulcers (the result of vascular lesions by atherosclerosis, vascular walls become rigid, do not contract, refraction is difficult – bleeding) large, but not deep, clear contours, thin superficial necrotic layer in the bottom, moderate inflammatory cell infiltration. Treatment of peptic ulcer is carried out according to standards taking into account age-related features of the course and comorbidity.

### **Intestinal diseases in elderly patients.[1-3, 6]**

Age-related changes in the intestines: an increase of the total length, especially of the large intestine; mucosal atrophy, impairing the digestion of food, the absorption of carbohydrates, proteins, fats; intestinal microbiota changes: the number of lactic acid bacteria decreases, and the number of putrefying bacteria increases significantly. This contributes to the growth of endotoxin production and pathological processes in the intestine.

**Abdominal ischemia syndrome** is caused by severe atherosclerosis of the abdominal aorta and its branches. Blood supply is disturbed in the left parts of the large intestine - ischemic colitis. Dyspeptic syndrome, periumbilical pain, swelling, constipation, weight loss dominate. Clinical manifestations of maldigestion syndrome and malabsorption are present. The appetite is not altered, the "small portions" syndrome is observed (in order to avoid pain). After exercise, eating, abdominal pain intensifies. Ulcers form in the mucosa, with a mild course, rapid epithelization occurs, with a severe course, the ulcer deepens, involves deep layers of the intestine. After scarring, narrowing of the lumen of the intestine is possible. Treatment requires the prescribing anti-ischemic drugs (calcium antagonists, nitrates), antiplatelet agents, enzyme medications, antispasmodics, statins. Eating in small portions is recommended. One of the features of pathology in humans is the rather frequent involving of both small and large intestines, then it is possible to develop their infarction and gangrene with widespread necrosis on all layers of

the intestine. With transient disorders (vanishing colitis syndrome), morphological changes are seen only in the superficial layers of the intestinal mucosa (ulcers, foci of atrophy and destruction). For the chronic variant of the abdominal ischemic syndrome, such changes in the mucous membrane of the small intestine are observed: atrophy of villi and crypts; decrease in the number of crypts; reduction in the number of goblet cells; presence of an irregular shaped low villi, secretions of the mucous membrane with the absence of villous and atrophic crypts, with partial localization in the splenic bend; superficial necrosis of the mucous membrane with remaining of the crypt epithelium over a large extent; presence of blood clots or fibrin clots in the small arteries; hemosiderin-laden macrophages; replacement of the muscularis mucosa with connective tissue. In 15.0% of cases, malabsorption develops with diarrhea, steatorrhea and with a decrease in body weight before cachexia.

**Chronic colitis (HC)** is accompanied by impaired intestinal motor function, colon atony and intestinal anomalies. Prolonged constipation over time leads to the inflammatory process of the colon mucosa, mainly sigmoiditis, proctitis, which are manifested by constipation with pain in the left half of the abdomen, bloating and the large bowel diverticulitis.

**Diverticulitis** is confirmed by X-ray examination, sigmoidoscopy. Manifestations of acute diverticulitis: pain in the left iliac region; fever, peritoneal irritation symptoms; rectal bleeding; constipation and diarrhea. Complications: perforation of the intestinal wall with peritonitis; massive bleeding; intestinal obstruction (swelling of the wall, stenosis), fistulas. Treatment of HC and diverticulitis: a diet with a restriction of carbohydrates and fats; antispasmodics (papaverine, no-shpa, trimebutin, mebeverin). In the inflammatory process - rifaximin, phthalazole, norfloxacin in short courses of 7-10 days, probiotics. Complications require immediate surgery.

**Nonspecific ulcerative colitis (UC)** in 10-11% of patients begins after 50 years and has the following features: acute form is rare; a chronic course with

exacerbations is typical, the duration of which is much longer than in young people; the onset is uncharacteristic for UC: short-term diarrhea, moderate abdominal pain, blood in the formed feces, constipation. Common complications: perforation of the intestinal wall with abscess or peritonitis, intestinal bleeding, bowel stenosis. Treatment: mesalazine, rifaximin, corticosteroids.

**Constipation and the principles of their treatment.** In the elderly, stool retention (senile constipation) is promoted by low physical activity, the progression of existing diseases typical of the elderly (atherosclerosis, abdominal ischemia, parkinsonism, depression), age-related features (hypoxia, decreased reparative processes, degeneration of the ganglia responsible for gastrointestinal motility, decreased synthesis of regulatory gastrointestinal peptides, dysbiosis, frequent use of medications that contribute constipation. Constipation can be independent (primary) and symptomatic (secondary). Primary constipation is mainly caused by hormone imbalance, changes in the sensitivity of the receptor apparatus, impaired autonomic intestinal innervation and psychogenic effects. Symptomatic constipation is a sign of organic diseases, developmental abnormalities not only of the intestines, but also of other organs or systems.

If constipation occurs, a diagnostic research must be performed to exclude cancer: a) interpreting patient's history and results of physical examination; b) an X-ray examination of the intestine (allows you to assess the anatomical state of the colon: inflammation or its normal structure with functional disorders, tumors, abnormalities or megacolons characteristic for obstruction, hypogangliosis, idiopathic expansion) c) colonoscopy, histological and histochemical studies of biopsy samples of the intestinal mucosa. The treatment for constipation depends on its type and the factors that cause it. It should be remembered that in the elderly, the incidence of diseases of the intestines and stomach is increasing. Gastric cancer appears mainly at the age of 40-70 years.

**Diseases of the gallbladder and liver in elderly patients.** Human aging is accompanied by a decrease in liver volume, blood flow, a decrease in the

metabolism of drugs and regenerative ability and changes in immune functions, a decrease in the function of hepatocytes involved in the metabolism of proteins, fats, hydrocarbons, pigments and that perform an antitoxic function.

The gallbladder in the elderly increases in size, the tone of its muscles and motor function decrease; there is bile stasis and a lot of cholesterol is secreted. All this contributes to the development of cholelithiasis (gallstone disease).

**Chronic hepatitis (CG) [8,9]** in people of older age groups is usually a continuation of the chronic viral process that arose at a young age. Frequent variants of the course of chronic hepatitis C: asymptomatic, low-symptomatic (persistent), active. Complications: cirrhosis and hepatocellular carcinoma. Treatment of chronic viral hepatitis is carried out according to generally accepted standards.

**Autoimmune hepatitis (AIH)** develops in almost 20% of patients older than age 60, the disease is progressive and appears unexpectedly, since ascites and cirrhosis are typical manifestations of this disease at the time of diagnosis. There are no differences in clinical presentation and treatment between young and elderly patients with AIH.

**Non-alcoholic fatty liver disease (NAFLD).** The disease mainly manifests itself in middle or old age. A significant part of the cases of cryptogenic cirrhosis may be associated with the terminal stage of NAFLD, and, possibly, age is a risk factor for liver fibrosis and a high mortality rate among patients with NAFLD. Elderly patients have much more risk factors for developing NAFLD, in particular: hypertension, obesity, diabetes and hyperlipidemia. Old age is a risk factor for drug damage to the liver, as older people are more prone to adverse reactions to drugs.

**Chronic cholecystitis [7]** in the elderly is usually the continuation of a long-term process that began at a young age. Noncalculous chronic cholecystitis in geriatric age is rare. Clinically characterized by moderate intensity of pain attacks. With a latent course - dyspeptic syndrome. After diet violation - biliary colic.

**Chronic calculous cholecystitis** often has an atypical course: dyspeptic



syndrome with complaints of lack of appetite, bitterness in the mouth, nausea, vomiting, discomfort in the upper abdomen. Typical biliary colic attacks are rare. Patients report only mild pain or a feeling of pressure in the right hypochondrium, more often after a diet violation.

Atypical course is due to:

- decreased muscle tone of the bladder and sphincters; age-related atrophy;
- a combination of chronic cholecystitis with a chronic inflammatory process in the stomach, duodenum, pancreas or intestines.
- gall bladder reflexes can trigger angina attacks.

Complications of gallstone disease: obstruction of the bile duct and hydrops of the gallbladder; empyema; obstructive jaundice with the development of secondary biliary cirrhosis; gallbladder gangrene with peritonitis and perforation of the gallbladder.

Surgical treatment is indicated in empyema and hydrops of the bladder, obstructive jaundice, gangrene and peritonitis, with frequent episodes of biliary colic. In other cases, conservative treatment: antibiotics, antispasmodics, anti-inflammatory, painkillers.

**Age-related changes in the pancreas.** Atrophy begins at the age of 40: the number of cells secreting hormones decreases, and the concentration of insulin, on the contrary, increases. At the same time, blood glucose levels also increase; pancreatic secretion production is reduced.

Clinical manifestations of pancreatic dysfunction are signs of maldigestia and malabsorption syndromes, which require constant use of enzyme replacement therapy.

The nutrition of the elderly should be not only complete, but also balanced. Due to the nutritional characteristics of older people, the ratio of proteins, fats and carbohydrates in it is 1: 0.8: 3, in contrast to the usual ratio of 1: 1: 4 for people of younger age groups. With age, the daily need for vitamins increases. It is necessary to limit the intake of salt, and the one that is added to the dish is limited to 3-4 g

per day.

### **Features of the course, diagnostics and treatment of diseases of blood and blood-forming organs in the elderly**

**Age-related changes in the hematopoietic system.** [1,2] In the bone marrow, spleen, lymph nodes, tonsils after 65-70 years, the mass of all components of the blood system is significantly reduced. In the peripheral blood, the count of T- and B-lymphocytes is low, their functional activity is decreased. In the bone marrow, the stroma grows, the number of reticular and collagen fibers increases, vessels obliterate, the hematopoietic tissue partially replaces with fat and the number of blasts decreases. In peripheral blood, the number of leukocytes is within normal limits, the number of eosinophils and stab neutrophils is reduced. Changes in thrombocytopoiesis: the number of megakaryocytes decreases, the number of "old" and degeneratively altered cells increases. But remaining young megakaryocytes provide the necessary number of blood platelets and, therefore, no thrombocytopenia is observed in elderly. Age-related changes are accompanied with a decrease in the functional activity of the bone marrow, especially the erythropoietic activity, which contributes to the development of anemia.

**Anemia in the elderly.** An increase in the frequency of anemia in the elderly is associated with an increase in the number of chronic diseases that contribute to its development (kidney and oncological diseases, lesions of the stomach and duodenum). The development of anemia in the elderly is accompanied by a significant deterioration in the quality of life, aggravates the course of the existing pathology and poses a threat of premature death. In elderly people, iron deficiency anemia (IDA) and anemia with a complex pathogenetic mechanism (anemia of a chronic patient or anemia of a chronic disease (ACD)) are the most common. Anemia of a chronic patient occurs during processes accompanied by inflammation or toxic effects on the body (chronic infectious and inflammatory diseases, oncological process, chronic kidney disease, chronic heart failure, cirrhosis of the liver, chronic hepatitis, systemic diseases, endocrine

pathology - hypothyroidism and others). They are caused by iron deficiency due to chronic bleeding from erosions and gastrointestinal ulcers during long-term therapy with NSAIDs, as well as due to disregulated hemostasis caused by NSAIDs. Elderly patients may have deficiency of vitamin B 12 due to a decrease in the formation of an intrinsic factor in the stomach fundus, including NSAID-associated gastropathy, an increase in the level of inflammatory cytokines - interleukins-1 and 6, TNF-alpha (there is a correlation between the level of cytokines and the content of serum iron). In anemia of a chronic disease, the total iron-binding capacity (TIBC) can be reduced (due to suppression of transferrin synthesis in the chronic inflammatory process) or be within normal limits. With IDA, TIBC is increased, but the proportion of saturated transferrin is much lower than normal. A very important sign of ACD is the lack of an effect of iron supplement therapy. Treatment is carried out by correcting the state of the underlying disease, a balanced diet, iron supplements, with chronic kidney disease - erythropoietin, with a deficiency of vitamin B12 - parenteral administration.

**Iron deficiency anemia:** muscle weakness, trophic disorders: dry skin, angular cheilitis, thin and brittle nails, koilonychia; perversion of taste and smell, shortness of breath, tachycardia, swelling in the legs, anemic systolic murmur at the apex of the heart, arrhythmia (extrasystole, block), the voltage of the ECG waves drops, sweating, tinnitus (ringing in the ears) appears.

Blood tests: decreased hemoglobin, color index 0.8-0.7, microcytosis, neutropenia, thrombocytopenia, moderate reticulosis, elevated ESR, decreased concentration of serum iron.

To find out the causes of anemia, it is necessary to perform a series of examinations: X-ray of the gastrointestinal tract, upper endoscopy, rectal examination, sigmoidoscopy, colonoscopy, feces occult blood test, gynecological examination for women, urological examination for men, the chest X-ray, bronchoscopy, abdominal and pelvic ultrasonography.

Treatment: diet therapy - meat, fish, cheese, fruit, vegetables; - iron

supplements (ferrocovenum, ferbitolum, ferrum lek) for 2-3 months under the control of blood iron.

**B-12 deficiency anemia, folic acid deficiency anemia.** B-12-DA is more common than FDA, sometimes there may be a combination of them. Etiology: insufficient content of B-12 and folic acid in food; imbalanced diet (low in protein); impaired absorption in the intestine, decreased secretion of an intrinsic factor in the stomach in atrophic gastritis in old age; gastric resection - after 5-8 years or more; liver diseases (liver is a storage place for B12), helminthic invasion (tapeworm), heredity, chronic enteritis, intestinal diverticulosis, and malignant tumors of the stomach.

Clinical characteristics: anemic syndrome (weakness, fatigability, palpitations during physical exertion, shortness of breath), damage to the nervous system - funicular myelosis - paresthesia, decreased vibration sensitivity, abnormal gait; gastrointestinal manifestations: glossitis – atrophic glossitis, burning tip of the tongue, decreased stomach secretion, liver and spleen enlargement with an elevated blood bilirubin concentrations and mild yellowness of the skin; heart failure (dyspnea, tachycardia, edema), changes in peripheral blood (anisocytosis, schizocytosis (fragments of red blood cells), megakaryocytes, poikilocytosis, hyperchromia, color index up to 1.2-1.5, Howell-Jolly bodies, Cabot rings, thrombocytopenia, leukopenia. Bone marrow biopsy shows erythroid hyperplasia; erythroid precursors show megaloblastic features. In order to determine the causes of anemia, such examinations are carried out: gastrointestinal fluoroscopy, sigmoidoscopy, gastroscopy, gastrointestinal ultrasound, liver function tests (bilirubin, transaminases, blood protein), feces occult blood test, and the feces analysis for helminth eggs.

Treatment: Vitamin B-12 at 200-500 mcg once a day for 6 weeks, then 1 time / week for 2-3 months, 2 times / month for 6 months; maintenance therapy - 1-2 times / year with short courses of 5-6 injections; with a deficiency of folic acid: 5-15 mg / day.

**Aplastic anemia.** Etiology and risk factors: a natural decrease in the functional activity of the bone marrow, decreased compensatory abilities in old age, the effects of toxic substances, drugs, ionizing radiation, infectious diseases, viruses that easily cause hematopoiesis disorders. Clinical characteristics: anemic, hemorrhagic syndrome (thrombocytopenia), frequent infectious or inflammatory processes (due to leukopenia, neutropenia), a more severe course than in young people. Blood tests: anemia, leukopenia, thrombocytopenia, bone marrow – low count of all poietic cells.

Treatment: transfusion of red blood cells, when hemorrhagic syndrome is present - transfusion of platelet concentrate; in inflammatory processes - antibiotics, splenectomy. If the conservative therapy is ineffective, bone marrow transplantation is indicated.

**Leukemia.** Among all blood diseases in older people, leukemia accounts for up to 55%. Often there are chronic lymphocytic leukemia, chronic monocytic leukemia, chronic myeloid leukemia and acute leukemia.

**Acute Leukemia** Clinical features: complaints of shortness of breath, palpitations, general weakness, bleeding; hemorrhagic syndrome manifestations are nose and gum bleeding, and bruises. There also may be hemorrhages in the internal organs. Blood tests: severe anemia, thrombocytopenia, leukopenia. Lymph nodes are swollen and spleen is enlarged in 50% of patients. Often complications: pneumonia, sepsis, necrotizing ulcerative tonsillitis.

The treatment is similar to the treatment of the young, but the doses of cytotoxic drugs are 1/2 - 1/3 of the dose for the young. At the beginning of treatment, 1-2 cytostatics are used, with a rapidly progressing course - a combination of 4-5 drugs (VAMP, CAMP regimens).

**Chronic lymphocytic leukemia** is a benign tumor of lymphoid tissue. Elderly men are mostly affected. The course is benign, in a smaller number of cases - malignant.

Clinical characteristics: swollen lymph nodes, enlargement of spleen, liver,

often lymphoid skin infiltration; blood test: leukocytosis with lymphocytosis, Gumprecht cells(shadows), anemia, thrombocytopenia; infectious complications due to decreased immunity - defective lymphocytes. There are 3 stages: initial, manifesting, terminal.

Treatment; in the initial stage special treatment is not carried out. If the condition worsens - primary stimulating therapy: 1 cytostatic - chlorbutin or cyclophosphamide. In the manifesting stage: a combination of cytostatics - VAMP, CAMP, COP schemes. With large sizes of lymph nodes or spleen - radiation therapy. In the inter-course period - maintenance therapy with one of the cytostatics. With infectious complications - antibiotic therapy.

**Chronic myeloid leukemia** is more common in middle and old age. Pathogenesis: a tumor from myeloid cells that do not lose their ability to differentiate to mature cells. After 60 years, the course is benign. In the initial stage, there are no clinical symptoms, only initial changes in the peripheral blood; in the expanded stage: enlarged spleen, liver, leukocytosis in the blood, WBC left shift, presence of metamyelocytes, myelocytes, promyelocytes, myeloblasts, eosinophilic-basophilic association; first erythrocytosis and thrombocytosis, then anemia and thrombocytopenia. In the terminal stage: a blast crisis with a malignant course resembling acute leukemia.

Treatment: the tactic is similar to chronic lymphocytic leukemia. From the group of cytostatics - myelosan, myelobromol, dopan.

**Chronic monocytic leukemia** occurs mainly in old age. It is characterized by bone marrow hyperplasia with proliferation of monocytes. Clinical features: benign course, enlarged spleen, hemorrhagic syndrome; blood test - monocytes up to 20-40%, bone marrow: polymorphic cell hyperplasia with an increase in the number of cells of the monocytic series; later anemia, thrombocytopenia develops.

Treatment: symptomatic in the initial and advanced stages (blood transfusion, glucocorticoids in hemorrhagic syndrome), cytostatics (as in acute leukemia) in the terminal stage.

**Lymphogranulomatosis:** a malignant granuloma that occurs in the lymph nodes and in the internal organs. Most patients are often between the ages of 16-30 and after the age of 50. Clinical features: swollen lymph nodes, lesions of the internal organs (symptoms of damage to the stomach, lungs, bones), general intoxication: fever, chills, itchy skin, sweating, exhaustion; neutrophilic leukocytosis in the blood, high ESR, eosinophilia, monocytosis, in the terminal stage - a decrease in lymphocyte, erythrocyte and leukocyte count. Puncture biopsy: lymph nodes contain Berezovsky-Sternberg cells or their predecessors - Hodgkin cells.

Treatment: chemotherapy, radiation therapy, combination therapy (chemo- and radiation), surgical treatment.

**Hemorrhagic diathesis (HD)** is a group of diseases that are different in etiology, diagnostic methods and treatment. Their common feature is a tendency to increased bleeding and bleeding, which occurs spontaneously or due to minor injuries (that cannot cause bleeding in a healthy person). According to the mechanisms of development, HD are divided into 3 groups: the first - HD associated with changes in platelets, the second - HD are caused by blood coagulation system disorder, the third - HD, resulting from damage to the vascular wall.

**Thrombocytopenic purpura (TP) (essential thrombocytopenia, Verlhof's disease)** is a common disease among all HD. Women are affected 3 times more often than men in adolescence or in premenopausal age. An increased platelet destruction is a main feature. Normally, the platelet life is 6-8 days, and with thrombocytopenia - from several hours to a day. Etiological factors: heredity, viral diseases, bacterial infections, intoxication, hypersensitivity to certain foods or medicines. The acquired form of the disease is almost 10 times more common than hereditary.

Clinical characteristics. Complaints of multiple hemorrhages in the skin and subcutaneous tissue (mainly on the arms and legs, and on the trunk, too), that

appear without cause or with the slightest injury. Hemorrhages are of different sizes, often large. They do not occur simultaneously, so they have a different color - purple, cherry blue, brown, yellow. After a few days, the elements of the rashes disappear, and new ones appear instead. The patient's skin becomes spotty - resembles the skin of a leopard. Uterine bleeding is a common complaint; the disease can begin from this manifestation, and sometimes this manifestation is the only one for many years. Often there are nosebleeds, sometimes pulmonary, gastrointestinal, and renal. Blood loss, especially uterine and nasal, can be significant and lead to the development of hypochromic anemia. Hemorrhages in the brain or in the retina are very dangerous. They occur in acute forms of the disease. Prolonged bleedings are observed arising from tooth extraction and other surgery; they are very difficult to stop. On physical examination, in addition to changes in the skin, in 1/3 of the cases, an enlarged spleen is found. With an exacerbation of the disease, an increased body temperature can be observed.

Laboratory examination: peripheral blood - a decreased platelet count of  $50 \times 10^9$  in 1 liter, but during remission, the platelet count may become normal, low RBCs and hemoglobin count, bleeding time increases to 15-20 minutes. In the chronic course, low platelet counts are constantly observed and new hemorrhages constantly occur. The latent form of the disease is characterized by periodic single bruises on the skin, prolonged menstruation, sometimes there are nosebleeds. The platelet count in such cases is slightly decreased. Cases of recovery, prolonged remissions are possible. Death may occur due to massive bleeding and hemorrhage in vital organs.

Treatment: bed rest, high-calorie diet, medication therapy (glucocorticoids - 30-40 mg / day); immunosuppressive drugs if the previous treatment is ineffective; splenectomy, if there is no result with the drug therapy; local treatment - tamponade with epsilon-aminocaproic acid, hemostatic sponge, fresh plasma, thrombin solution, calcium chloride; blood or platelet mass transfusion if indicated.

**Hemophilia** is a hemorrhagic diathesis (HD), which develops due to



congenital insufficiency of blood coagulation factors in the blood. The hemophilia gene is localized on the X chromosome. Most patients are male. The disease is transmitted to the grandchildren through clinically healthy daughters in whom (carrier) half of the sons are born with hemophilia and half of the daughters can become carriers of the disease.

Clinical features. The course of the disease is chronic. The most difficult periods are childhood and adolescence. In people older than 30-50 years, bleeding occurs less frequently. A tendency to bleeding is observed from the first hours after the birth of a child: hemorrhage from the umbilical cord, skin hemorrhage, cephalohematomas, prolonged bleeding during teething and loss of milk teeth. Patients often have massive hemarthrosis of big joints; contractures, ankylosis, and atrophy of nearby muscles are formed. Often there are nose and gum bleeding, sometimes gastrointestinal, renal (hematuria), hemorrhages in the brain, into the pleural cavity (hemothorax), into the abdominal cavity (symptoms of acute abdomen appear). Hemorrhages in the tongue root or larynx can lead to asphyxiation and death of the patient. Due to constant blood loss, patients develop symptoms of chronic posthemorrhagic anemia: pallor of the skin, dizziness, fatigue.

Laboratory examination: coagulogram - prolongation of blood coagulation time; peripheral blood - a decrease in the number of RBCs, a decrease in hemoglobin, a decrease in the color index.

Treatment: fresh-frozen plasma transfusion. The antihemophilic factor is unstable, therefore, only fresh-frozen plasma must be transfused with a shelf life of no more than 24 hours. If blood loss is significant, up to 2 liters of plasma could be transfused, and with less serious bleeding - 250-500 ml. Antihemophilic globulin is 20 times more active than plasma; Cryoprotein is an even more active drug, it is obtained from frozen plasma. With fresh hemarthrosis, blood from the joint cavity is aspirated, a pressure bandage is applied and the joint is immobilized. Glucocorticoids (25-30 mg) are prescribed to relieve swelling around the joint. For

bleeding, a hemostatic sponge is used topically. Intramuscular injections for hemophilia patients are contraindicated.

**Hemorrhagic vasculitis (Henoch–Schönlein purpura (HSP))** develops due to increased permeability of the vascular wall due to the immune inflammatory process with the destruction of microvessels and thrombosis; hemorrhagic syndrome is developed. Etiological factors: viruses, bacteria, vaccination, food allergies, medications, trauma, cooling, parasitic infections.

Clinical features: the skin form of the disease has an acute onset, less often the course is protracted or recurrent. Complaints: an increase in temperature to 39-40 degrees, the appearance of rashes on the skin and mucous membranes of a hemorrhagic-papular nature (a vesicle filled with bloody fluid, sometimes with necrosis in the center), symmetrically on the right and left sides of the limbs and trunk. When pressed with a finger, the rash does not disappear. Sometimes there are itchy skin and swelling. Skin pigmentation at the site of the rash is not changed.

Joint syndrome is manifested by pain in large joints, periarticular tissues swelling, movement is limited. The pain goes away after a few days, sometimes recurs.

Hemorrhagic rashes on the gastric mucosa and peritoneum lead to the development of the abdominal syndrome. The patient's complaints of severe constant or periodic abdominal pain lasting 1-3 hours, nausea, vomiting of blood, diarrhea (may be mixed with blood). The abdomen is tense (acute abdomen syndrome). After 1-4 weeks from the onset of the disease, renal syndrome can develop: increased blood pressure, swelling, in the urine - protein up to 30%, cylinders, red blood cells, kidney failure risk.

Cerebral form: thrombovasculitis of the brain, meninges with meningeal syndrome, epileptic seizures.

Pulmonary form with pulmonary hemorrhage. These forms are severe and often lead to death.

Laboratory examination: leukocytosis, high ESR, positive tests for the

activity of the inflammatory process, increased levels of circulating immune complexes in the blood plasma; urine analysis - protein, red blood cells, cylinders.

Treatment: bed rest for 3-4 weeks, glucocorticoids (if the process is intense and especially if there are lesions of the gastrointestinal tract) - 30-40 mg per day; heparin therapy with individual selection of doses of the drug. Heparin is administered in equal doses throughout the day under mandatory laboratory control of the blood coagulation system. In the presence of painful joint syndrome, NSAIDs are prescribed, 50% dimexide with heparin is used in places of massive rashes.

### **Pharmacokinetics of drugs in elderly and senile patients**

The process of passage of drugs in the body (pharmacokinetics) has the following steps: introduction (intake) of the drug into the body, penetration (absorption) through biological membranes into the vascular bed, then to the tissues and cell receptors, distribution in biological fluids, organs and tissues, biotransformation (metabolism of drugs with a change in their pharmacokinetic properties and the formation of metabolites), excretion (elimination) from the body. The absorption of drugs decreases with age, their excretion from the body changes. The intensity of metabolic processes is weakened, the excretory function of many organs is reduced, pharmacological substances are slowly excreted from the body, and circulate longer. A decrease in the detoxifying function of the liver contributes to the accumulation in the body of the drugs, their metabolites, and contributes to the drug intoxication. This makes it necessary to halve the initial dose of most drugs in elderly patients than in young people.

### **Features of pharmacodynamics in the elderly**

The pharmacological effects of drugs and their mechanisms of action (pharmacodynamics) during aging change due to age-related changes in receptors, their sensitivity to drugs, the content of metabolites, enzyme activity, and the reactivity of the internal environment of the body. Their effect on specific

receptors, membranes, and cell enzymes changes the functioning of body systems and cells.

Under the influence of **beta - adrenoblockers** in elderly patients, an increase or weakening of pharmacological effects, an increase in the central nervous system side effects and disturbances in peripheral arterial circulation can be observed.

**Sedatives and hypnotics**, especially barbiturates, often contribute to the development of anxiety, irritability and insomnia; prolonged administration to persons with latent renal failure leads to accumulation and intoxication, inhibition of the excitability of the respiratory center. Long-term administration of chlorpromazine may be accompanied by the development of jaundice, impaired digestive function. In the elderly, morphine causes depression of the respiratory center. If necessary (e.g. myocardial infarction) intravenous administration is possible in significantly lower doses (4–6 mg), rather than 10–15 mg as usual. Morphine should be used very carefully in patients with asthma, chronic obstructive pulmonary disease, significant chest deformity and kyphoscoliosis. Morphine can also cause urinary retention.

**Nonsteroidal anti-inflammatory drugs** enhance side effects (gastrotoxicity, nephrotoxicity, hepatotoxicity). Elderly and senile people may experience abdominal pain, nausea, vomiting, tinnitus, exacerbation of peptic ulcer, sodium retention, edema; the risk of bleeding increases. Long-term use of salicylates causes hearing loss, confusion

**Antibiotics and sulfa drugs**: delayed excretion and increased concentration in the blood is observed. Ototoxic and nephrotoxic effects are inherent in streptomycin and aminoglycosides. Sulfanilamides are contraindicated in elderly patients with urolithiasis, parenchymal kidney disease, and chronic renal failure. When elderly patients use broad-spectrum antibiotics, vitamins, antifungal agents, and probiotics are prescribed due to the increased risk of candidiasis and dysbiosis.

**Corticosteroids** in elderly people more often cause an imbalance of the

electrolytes, sodium and water retention, hyperglycemia, an increased tendency to impaired immune response, the formation of peptic ulcers and bleeding. Mostly they are used in acute conditions.

**Cholinolytics, antispasmodics** - atropine, belladonna preparations, scopolamine, metacin are contraindicated in glaucoma. Anticholinergics often interfere with urination and contribute to urinary retention in the elderly.

**Adrenaline and sympathomimetic drugs**, when exceeding individual dosing, cause spasm of the vessels of the brain, kidneys, peripheral parts of the cardiovascular system, therefore, it is especially necessary to prescribe them carefully to people with brain disorders, hypertension, atherosclerosis, and severe organic heart diseases. In such cases, their use is possible only in life-threatening situations.

**ACE inhibitors** - reduced sensitivity to hypotensive effect due to a decrease in RAAS activity in elderly patients, and in **loop diuretics** - electrolyte imbalance.

#### **Features of geriatric pharmacotherapy**

- Consider polymorbidity, chronic degenerative incurable diseases.
- Pharmacotherapy of the leading syndrome.
- Slowing down the absorption, metabolism and elimination of drugs from the body.
- Individual dosing and drug selection with the transition to a maintenance dose.
- Patients are prone to self-medication and polypharmacy.
- The therapeutic dose is not higher than 50% of the dose of patients of young and middle age.
- High risk of cumulation of drugs and their metabolites with side effects.
- In complex therapy include geroprotectors, adaptogens and anabolic hormones.
- Simple medication intake with a written explanation of the timing and

dosing.

- Take into account the mental and social situation of the patient: loneliness, depression, dementia, impoverishment, encephalopathy, etc.

### **Principles of treatment for the elderly**

- Continually maintain an elderly patient's sense of self-worth.
- Encourage the patient and those close to him to communicate closely with the medical staff regarding treatment. It is advisable that the patient periodically brings his medications for examination.
- Inform the patient and his relatives about medications, side effects when choosing / replacing drugs. Clearly write the schedule and doses of drugs, explain the purpose of their prescribing.
- It is advisable for the patient to keep a diary of his condition, the presence and frequency of pathological manifestations (pain, swelling, blood pressure, heart rate, etc.), as well as the list and amount of drugs used.
- Avoid polypharmacy. The most important rule of geriatric pharmacology is the individualization of doses.
- Take into account the existing risk factors for complications: liver, kidney, heart failure, anemia, weight loss and possible negative effects of medications.
- It is advisable to start treatment with small doses of drugs, selecting the minimum sufficient to obtain the effect.
- To increase the patient's adherence to treatment, use the minimum number of drugs and the frequency of administration in a form convenient for the patient.
- It is necessary to take into account nutrition, water and salt diet, the amount of urine excreted per day, adherence to prescribed medication regimen regarding food.

### **The main risks of polypharmacy:**

- drug interactions;
- use of potentially unsuitable drugs;

- an independent risk factor (death, hip fracture, and also the risk of falls - especially when using drugs that affect the central nervous system)
- risk of a “prescription cascade”: an adverse reaction is perceived as a new disease;
- taking many drugs reduces patients' adherence to therapy.

### **The principles of non-drug therapy**

The main principle of non-drug therapy for elderly patients is the preventive and therapeutic focus: diet, physical and mental activity (L. Ena, 2016). It is necessary to do everything possible to activate patients, stimulate them to useful actions (dressing, toilet, eating), prescribe physiotherapy exercises, etc. However, it is necessary to avoid prolonged hospitalization and strict bed rest in every possible way: excessively strict and prolonged bed rest is categorically undesirable, as it leads to a high risk:

hypostatic pneumonia,

thromboembolic complications;

disorders of the urinary system: decreased detrusor tone, vesicoureteral reflux, hydronephrosis, accelerated calculus formation, exacerbation of pyelonephritis - a “care disease” (maladie de sorti);

defecation disorder: hypomotor intestinal dyskinesia, coprostasis, fecal stone formation, diverticula, complicated by hemorrhoids;

violation of bile secretion: hypomotor dyskinesia of the gallbladder and sphincter of Oddi, accelerated stone formation, cholecystitis;

decreased appetite and catabolism: progressive alimentary dystrophy with inevitable consequences;

increased joint stiffness: accelerated cartilage degeneration, secondary synovitis, purulent arthritis, progressive osteoporosis, aseptic bone necrosis;

disturbance of sleep and psyche: changing biorhythms, mixing up day and night, confusion).

### **Features of the Long-Livers' Nutrition**

(Grigorov Yu.G., Kuznetsova S.M., 2003)

- Low-calorie diet (1500-2000 kcal).
- Dairy and vegetable direction (intestinal microflora of long-livers and healthy children are almost identical).
- Fiber-rich foods.
- Adequate amino acids (methionine, cysteine, glutamic acid).
- High content of polyunsaturated fatty acids (linoleic), vitamins, natural antioxidants and capsaicin (thermoregulation).
- Active use of tea containing catechins (decrease in the level of  $\beta$ -amyloid).
- Moderate alcohol consumption.

### **3.3. RECOMMENDED LITERATURE**

#### **Basic:**

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### **Electronic resources:**

www \ moz.gov.ua

www \ testcentr.org.ua

www \ zsmu.zp.ua

<http://ukrmed.org.ua/>

<http://ukrcardio.org/>

<http://www.consilium-medicum.com/>

<http://www.medscape.com/>

Up To Date <http://www.uptodate.com>

BMJ Clinical Evidence <http://clinicalevidence.bmj.com>

Medscape from WebMD <http://www.medscape.com>

National Guideline Clearinghouse <https://www.guideline.gov/>

Centers for Disease Control and Prevention (CDC) <https://www.cdc.gov/>

The Cochrane Collaboration The Cochrane Library <http://www.cochrane.org/>

Clinical Knowledge Summaries (CKS) <http://prodigy.clarity.co.uk/>

The Finnish Medical Society Duodecim <https://www.duodecim.fi/>

The Association of the Scientific Medical Societies in Germany

<http://www.awmf.org>

The French National Authority for Health <http://www.has-sante.fr/>

National Institute for Health and Clinical Excellence (NICE)

<https://www.nice.org.uk/>

Canadian Medical Association InfoBase (CMA InfoBase: Clinical Practice

Guidelines (CPGs) <http://www.cma.ca/>

The National Health and Medical Research Council (NHMRC)

<https://www.nhmrc.gov.au>

Royal College of Physicians <https://www.rcplondon.ac.uk/>

AMA (American Medical Association) <https://www.ama-assn.org/>

American Academy of Family Physicians <http://www.aafp.org/home.html>

American Academy of Pediatrics Policy (AAP Policy) Clinical Practice

Guidelines <https://www.aap.org>

European Pediatric Association, the Union of National European Pediatric

Societies and Associations (EPA/UNEPSA) <http://www.epa-unepsa.org/>

American College of Cardiology <http://www.acc.org/>

American Heart Association <http://news.heart.org/>

European Society of Cardiology <http://www.escardio.org/>

### **3.4. Materials for student self-assesment for classroom stage.**

### **Theoretical questions for self-assessment:**

1. Age-related physiological changes in the respiratory system.
2. Features of the course of respiratory diseases in elderly patients (bronchial asthma, chronic non-obstructive bronchitis, COPD).
3. Age-related physiological changes in the circulatory system.
4. Features of the course of diseases of the circulatory system in the elderly and senile age (arterial hypertension, coronary heart disease, myocardial infarction)
5. Age-related physiological changes in the digestive system.
6. Features of the course of diseases of the digestive system in elderly patients.
7. Age-related physiological changes in blood and blood-forming organs.
8. Features of the course of diseases of the blood and blood-forming organs in the elderly.
9. Features of pharmacodynamics in patients of older age groups.
10. Features of the use of drugs in elderly and senile patients.

### **A. Test exercises for self-check of theoretical knowledge.**

1. Constipation in people of old age is not caused by:
  - A. low fluid intake
  - B. physical inactivity
  - C. Weakening of intestinal motility
  - D. a change in the intestinal microflora
  - E. Decrease in the number of beta cells of the islet pancreatic apparatus
2. The main features of the course of diseases in elderly patients are:
  - A. decrease the number of diseases
  - B. polymorbidity, chronic and atypical course
  - C. prevalence of acute forms of diseases
  - D. the predominant effect of external etiological factors
  - E. the prevalence of infectious diseases

3. A peptic ulcer in the elderly is often localized in:
- A. stomach
  - B. duodenum
  - C. Cecum
  - D. colon
  - E. rectum
4. Characteristic disturbances of the function of the gallbladder and biliary tract in patients of the elderly are:
- A. Increased tone of the sphincter of Oddi in the presence of a juxtapapillary diverticulum
  - B. Increased the contractile capacity of the gall bladder
  - C. Increase in the volume of the gallbladder in the study on an empty stomach
  - D. reduction of the contractile capacity of the gallbladder
  - E. Increased tone of the sphincter of Oddi: expansion of the common bile duct
5. What foods contain more fiber needed by elderly patients?
- A. bran
  - B. dried fruits
  - C. vegetables
  - D. white bread
  - E. meat
6. Old age:
- A. 67-79
  - B. 75-89
  - C. 60-80
  - D. 45-59
  - E. up to 45
7. Atrophy of mucous membranes with age leads to:

- A. Reduction of secretion
  - B. increase of protective properties
  - C. Epithelial growth
  - D. Increased secretion
  - E. The barrier function does not change
8. Geriatrics is:
- A. The science of aging the body
  - B. science that studies the features of combined pathology in the sloping and aging age
  - C. science studying the state of the elderly
  - D. science that studies the peculiarities of the course of diseases in the sloping and aging age
  - E. science studying the aging age
9. Gastroesophageal reflux disease in the elderly is primarily due to:
- A. with the action of aggressive factors of reflux (HCl, pepsin, bile acids)
  - B. increased intraabdominal pressure
- 13
- C. Increased clearance of the esophagus
  - D. decrease of antireflux barrier function
  - E. violation of stomach emptying
10. Specify the principles of treatment for constipation in patients of the elderly:
- A. giving laxatives
  - B. Providing Prokinetics
  - C. provision of serotonin-related receptor-related drugs
  - D. a proper diet that includes dietary fiber and a complete water balance
  - E. dosed gymnastics, abdominal massage, physiotherapy
11. Indicators that progressively decrease with age:
- A. Oncotic pressure

- B. Function of digestive glands
- C. sensitivity of organs to hormones
- D. intracerebral pressure
- E. everything is right

12. In elderly patients, polymorbidity is most often manifested by the presence of:

- A. 1 disease
- B. 2 diseases
- C. 2-3 diseases
- D. 4-6 diseases
- E. no dependence

13. Symptomatic ulcers in the elderly are ulcers:

- A. Due to the violation of blood circulation as a result of diseases of the cardiovascular, respiratory and other systems
- B. due to infection with *Helicobacter pylori*
- C. long-term existing ulcers that have arisen in young and middle age
- D. all listed
- E. none of the above

14. In the occurrence of peptic ulcers in the elderly, the greatest importance is:

- A. genetic predisposition
- B. violation of microcirculation in the wall of the stomach
- C. increase in the acid-forming function of the stomach
- D. psycho-emotional overload
- E. physical overload

15. The course of illness in elderly patients is not inherent:

- A. Autism of the course of diseases
- B. irradiance
- C. expressiveness of clinical manifestations

- D. Reduced expressiveness of clinical manifestations
  - E. multiplicity of pathological processes
16. Aging of the digestive system is manifested:
- A. increased appetite
  - B. appearance of hepatic colic
  - C. increased taste sensations
  - D. reduction of taste sensations
  - E. increase of enzymatic processes
17. Constipation in elderly patients is often conditioned:
- A. using a large amount of liquid
  - B. increase in physical activity
  - C. increased intestinal motility
  - D. weakening of the intestinal motility
  - E. sphincter spasm
18. Recommendations for nutrition for elderly patients include:
- A. the consumption of high-calorie food
  - B. the use of animal fats
  - C. Vegetarian nutrition
  - D. the exclusion of vitamins from the diet
  - E. eating small portions 4-5 times a day
19. In the elderly, the total daily caloric content of food:
- A. equals the need for patients with mature age
  - B. decreases
  - C. rises
  - D. does not matter
  - E. equals the need for childhood
20. At the keel of the esophageal aperture of the diaphragm in the elderly, the leading symptom is:
- A. decrease in appetite



- B. heartburn
  - C. air burst
  - D. pain behind the sternum in a horizontal position after eating
  - E. vomiting
21. The rules of pharmacotherapy for the elderly:
- A. A reduced dose of medication is prescribed
  - B. an increased dose is prescribed
  - C. medicines are only taken parenterally
  - D. All medicines are taken at once
  - E. only oral application
22. Before age-related changes of the liver and its functions include:
- A. weight reduction of the liver
  - B. Increased cholesterol synthesis in the liver
  - C. decrease in the synthesis of bile acids
  - D. increase in liver size
  - E. reduction of activity of monooxygenase system
23. The main causes of cholestasis in the elderly can be:
- A. liver disease (hepatitis, cirrhosis, cholangiocarcinoma), tumors of the pancreatoduodenal zone
  - B. thalassemia
  - C. Obturation of hepatic and total bile ducts by stone, postoperative strictures of the biliary tract
  - D. hemolytic anemia
  - E. Congenital arterio-edema dysplasia
24. The elderly with cholelithiasis may be shown the following treatment method:
- A. Extracorporeal shock-wave lithotripsy
  - B. endoscopic cholecystectomy
  - C. Oral litholytic therapy or contact litholysis

D. prophylactic cholecystectomy

E. cholecystectomy with hepatoneurovascular anastomosis

**Answers:**

A. Tests for self-assessment of the level of theoretical knowledge

1	2	3	4	5	6	7	8	9	10	11	12
E	B	A	D	A	B	A	D	D	D	B	D
13	14	15	16	17	18	19	20	21	22	23	24
A	B	C	D	D	E	B	D	A	A	C	B

**B. Tests for the final control**

1. Patient K., 72 years old, complains of intense pain in the right hypochondrium, nausea, vomiting, bitterness in the mouth. Palpation of the abdomen shows pain in the projection of the gall bladder, positive symptoms of Murphy, Ortner. Which disease is most likely?

A. Acute pancreatitis

B. stomach ulcer

C. Gastro-esophageal reflux

D. dyskinesia of the biliary tract

E. gallstone disease

2. The patient is 75 years old. During the last 3 months there were complaints of discomfort in the epigastric area, sometimes on the sternum, nausea, vomiting. In the last 2 years, the patient received treatment with nonsteroidal anti-inflammatory drugs for rheumatoid arthritis. Skin covers pale pink, hemodynamics are stable, with palpation of the abdomen - local pain in the epigastric region. What is the disease?

A. peptic ulcer of the stomach

B. gall-stone disease

C. Pulmonary disease of the 12th gullet

D. angina pectoris

E. non-specific ulcerative colitis

3. The patient is 72 years old. Within 1 month, there is a pain in the epigastric region, sometimes for the sternum, nausea. In the last year the patient received ibuprofen 400 mg for osteoarthritis. Objectively: AT 130/80 mm Hg, pulse 75 per minute, with palpation of the abdomen - local morbidity under the urinary tract. Possible cause of this disease?

A. Receiving NSAIDs

B. Disorders of the diet

C. exacerbation of osteoarthritis

D. age

E. pancreatitis

4. The patient has 64 years of complaints of heartburn on the sternum with the body tilt, as well as a sneezing at night. Objectively: the pallor of the skin, the tongue is densely covered with a white bloom; Moderate pain in palpation in the epigastrium. EPGDS-mucous membrane of the esophagus is swollen, hyperemic, cardia is not blocked, in the lower third of the esophagus - a lot of small erosions. Which of the following diagnoses is most probable?

A. Cholelithiasis

B. chronic gastritis

C. Gastroesophageal Reflux Disease

D. chronic cholecystitis

E. chronic pancreatitis

5. A 66-year-old patient with chronic heart failure receives digoxin (0.25 mg / day), furosemide (20-40 mg / day), and veroshpyrone (250 mg / day) for 2 months. In 2 days before the hospitalization, abdominal pain, muscle weakness, paresthesia in arms and legs appeared. An increase in the amplitude of the T wave, expansion of the QRS complex is noted on the ECG. Cause of deterioration:

A. Hypokalemia

- B. Hypercalemia
- C. Hyponatremia
- D. Intoxication with cardiac glycosides
- E. Hypomagnemia

6. A patient K., 67 years old, was hospitalized in the surgical department on the third day of the disease with complaints of fever to 39° C, pain in the right hypochondrium, jaundice. In the ultrasound study, the concrements in the gallbladder are absent, the diameter of the choledoch is 4 mm. Your previous diagnosis?

- A. Acute cholangitis
- B. Acute cholecystitis
- C. Acute pancreatitis
- D. Carole syndrome
- E. Acute viral hepatitis

7. A 62 year old patient, for 12 years, marks a periodic pain in the left hypochondrium with irradiation in the back, in connection with which he is compelled to adhere to a diet with restriction of greasy, roasted, sharp, smoked dishes. During the last 1.5 years the attachment of bloating is noted; a chair up to 2-3 times a day, abundant, smelly, with a shiny surface, with the remains of undigested food. Changing symptoms in a patient due to joining:

16

- A. Exocrine pancreatic insufficiency.
- B. Endocrine pancreatic insufficiency.
- C. Cholestatic syndrome.
- D. Irritable bowel syndrome.
- E. Insufficiency of gastric secretion.

8. A 65 year old patient complains of pain in the epigastrium with irradiation in the back, vomiting without relief, diarrhea. Objectively: pallor, dry skin, tachycardia, abdominal pain above the navel of 3 cm. Blood analysis: leuk.  $10 \times 10^9 / l$ , ESR

18mm / h, urine diastase 128 units. Ultrasound - the pancreas is hyperherogenic, the head is 24 mm, the tail is 37 mm. The most probable diagnosis?

- A. Chronic pancreatitis
- B. Stomach ulcer
- C. Erotic Duodenitis
- D. Tuberculous mesodenitis
- E. Enterocolitis

9. The patient, 70 years old, arrived at the department with complaints about permanent disrupting and, often, spastic abdominal pain, severe bloating, delayed emptying for 4 days, vomiting 4 times in the last day, general weakness (4 months). For 5 years - constipation, the last month in feces marked the appearance of traces of blood that binds to hemorrhoids. Objectively: reduced eating, skin and mucous membranes with an iridescent tint. AT 160/90 mm Hg. Art. Pulse 88 beats per minute. The abdomen is tense, pronounced flatulence, in the right flank the peristalsis of the colon loops is visually determined. In the blood test: erythrocytes  $3,0 \times 10^9 / l$ , ESR 45 mm / h. On the review X-ray of the abdominal cavity against the background of bloated loops of the intestine, numerous horizontal levels of fluid are determined. Your previous diagnosis?

- A. Nonspecific ulcerative colitis.
- B. Atherosclerosis of mesenteric vessels
- C. Cancer of the descending colon.
- D. Cancer of the ascending department of the colon.
- E. Diverticulum of the small intestine.

10. Patient D., 71 years old, complains about the alternation of diarrhea and constipation, a feeling of incomplete emptying of the intestine after the act of defecation, emptying of the mucus, periodic pain in the abdomen, increased exhaust gases. It is observed at the therapist for 3 months with regard to intestinal dysbiosis of 3 degrees. Abdomen is mild, pain in palpation along the large intestine. Irygосcopy: colitis. What drugs should be prescribed?

A. Probiotics + Spasmolytics

B. Prokinetics.

C. Enzyme preparations.

D. True A, B.

E. True A, C.

B. Tests for the final control

1	2	3	4	5	6	7	8	9	10
E	A	A	C	B	A	A	A	C	E

### C. Tasks for self-assessment

Task #1. A man, 53 y/o, suffers from peptic ulcer of the stomach. Recently, frequent profuse vomiting of stomach contents with a fetid smell, tonic convulsions with loss of consciousness appeared. Emergency care was provided to the patient, followed by preparation for surgery on the stomach.

1. What complication of chronic gastric ulcer caused the development of a serious condition of the patient?
2. What is the mechanism of tonic seizures development?
3. What was the emergency care provided to the patient?
4. What surgery on the stomach provided effective care for the patient?

Task #2. An elderly woman was treated in the cardiology department for acute myocardial infarction. Suddenly there was a profuse vomiting of "coffee grounds", loss of consciousness and death. At autopsy of the deceased, in addition to transmural myocardial infarction of the apex of the heart, a round defect of the wall of the lesser curvature of the stomach, filled with brown masses, was found.

1. What did the pathologist find in the stomach?
2. What pathological process arose in a patient in the stomach?
3. Indicate the immediate cause of death of the patient.

Task #3. A 64-year-old man suffered from chronic atrophic gastritis. Gastroscopy was performed with a biopsy of the sections of the gastric mucosa. Histological studies of biopsy specimens revealed cellular atypia with pronounced polymorphism of cells forming trabecular structures.

1. What was revealed during a histological examination of a biopsy specimen of the gastric mucosa?
2. What processes in the mucous membrane of the stomach became the basis for the development of a new pathology?
3. Further actions of the doctor.

**C. Answers to the self-assessment tasks:**

Task number 1.

1. Gastric tetany, as a manifestation of complete cicatricial stenosis of the pylorus.
2. The development of hypochloremic uremia, which occurred during blood thickening due to loss of fluid and electrolytes with vomit.
3. Parenteral administration of isotonic sodium chloride solution.
4. Restoring patency of the stomach through resection of its part.

Task number 2.

1. Acute stomach ulcer.
2. Acute gastric ulcer is a complication of acute myocardial infarction.
3. The cause of death is acute posthemorrhagic anemia.

Task number 3.

1. Cancer of the stomach.
2. Dysplasia and metaplasia of the epithelium of the gastric glands.
3. Refer the patient to the oncology clinic.

**4. Materials for classroom self-study work**

#### **4.1. Practical assignments students carry out on practical training**

1. Master the skills of collecting an anamnesis, identifying all risk factors that preceded the disease.
2. Master the methodology of an objective examination of elderly patients with respiratory, cardiovascular, gastrointestinal and blood diseases.
3. Supervise patient with respiratory, cardiovascular, gastrointestinal and blood diseases.
4. Assess the general condition of an elderly patient, taking into account the history, physical examination, laboratory and instrumental methods of examination.
5. Formulate a preliminary diagnosis.
6. Justify the need for laboratory and instrumental studies to establish a clinical (final) diagnosis.
7. Make a differential diagnosis of diseases of the corresponding organ system in an elderly patient.
8. Formulate the main clinical diagnosis and its complications, determine the concomitant disease on the basis of a comprehensive assessment of physical examination, data from clinical, laboratory and instrumental methods of examination.
9. Determine the need for consultation of other specialists.
10. Make a treatment plan for the patient.
11. Write prescriptions for medications.
12. Determine the prognosis of the disease
13. Make a plan for prophylaxis measures and prevention of disease relapse.
14. Write a protocol for patient supervision according to the next scheme:

Patient's full name

Age \_\_\_\_\_ Sex \_\_\_\_\_ Occupation \_\_\_\_\_

Presenting complaint:

\_\_\_\_\_

\_\_\_\_\_



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Anamnesis morbi:

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Anamnesis vitae:

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Physical examination results:

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Preliminary diagnosis:

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Examination plan:

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Lab and instrumental examination results:

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Clinical diagnosis rationale:

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Clinical diagnosis:

Main disease

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Concomitant diseases

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Complications

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Treatment:

1. Regimen

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2. Diet

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3.

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Student's signature

**4.2. Methodological support for self-study work of students at the stages of practical training:**

- Department's training manuals (ZSMU repository)
- Method manuals for practical trainings (doc.zsmu.edu.ua)
- Lectures presentations (Department's e-resources doc.zsmu.edu.ua)
- Normative documents: national, international recommendations, standards and guidelines (E-resources, The Internet)
- Equipment: electrocardiographs, tonometers, equipment for diagnostic rooms according to the profile of the departments.
- Visual materials: patient history, situational tasks, laboratory tests, X-rays, spiograms, ECGs, etc.

**5. Tasks and materials for extracurricular self-study work:**

- Write an library-research paper on the topic: functional dyspepsia, COPD, as independent nosological units, anemia in chronic diseases.

- Make an algorithm for diagnosing GERD, gastric ulcer, chronic pancreatitis, irritable bowel syndrome, asthma and COPD.
- Fill in the comparative table for chronic gastritis, peptic ulcer of the stomach and duodenum, anemias.

## **Annex 1**

### **Abbreviations used in the results of laboratory and instrumental studies**

#### **General Blood Test:**

WBC - White Blood Cells

GRA - Granulocytes

LYM - Lymphocytes

MON - Monocytes

RBC - Red Blood Cells

HGB - Hemoglobin

HCT - Hematocrit

MCV - Mean Cell Volume

MCH - Mean Corpuscular Hemoglobin - mean hemoglobin content in a red blood cell;

MCHC - Mean Corpuscular Hemoglobin Concentration

RDW – Red blood cell Distribution Width - a measurement of the range in the volume and size of red blood cells

PLT - Platelets

MPV - Mean Platelets Volume

PCT - Plateletcrit

PDW - Platelet Distribution Width

#### **Biochemical parameters:**

TBIL - Total bilirubin

DBIL - Direct bilirubin

TP - Total protein

ALB - Albumin

URE - Urea  
CRE - Creatinine  
ALT - Alanine transaminase  
AST - Aspartate transaminase  
GLU - Glucose  
CHOL - Cholesterol  
TG - Triglycerides  
HDL - High-density lipoproteins  
LDL - Low-density lipoproteins  
LDH - Lactate dehydrogenase  
URIC - Uric acid  
LIPA - Lipase  
AMIL - Amylase  
ALP - Alkaline phosphatase  
GGT - Gamma-Glutamyl Transpeptidase;  
CK (MB) - Creatine kinase  
FE - Iron  
Ferritin  
Folate  
TRF - transferrin  
TIBS - Total iron binding capacity  
CRP - C-reactive protein  
RF - Rheumatoid factor  
ASO - Anti-streptolysin O  
HBA1c - Glycated hemoglobin  
MAU - Microalbuminuria  
BNP - Brain natriuretic peptide  
AMM - Ammonium  
PSA - Prostate specific antigen

AFP - Alpha-fetoprotein

CEA - Carcinoembryonic antigen

Ostase - Bone alkaline phosphatase

INR - International normalized ratio

APTT - Activated partial thromboplastin time

TSH - Thyroid-stimulating Hormone

T4 - total thyroxine

T3 - total Triiodothyronine

FrT4 – free thyroxine

TgAB - Thyroglobulin antibodies

**Acid-base status.**

pH- potential of Hydrogen

pCO<sub>2</sub> - partial pressure of carbon dioxide

pO<sub>2</sub> - partial pressure of oxygen

SO<sub>2</sub> - arterial oxygen saturation

Lac - lactate

BUN - blood urea nitrogen

Calculated parameters of acid-base homeostasis:

BE-ECF - base excess extra cellular fluid

BE-B - base excess blood

SBC - standard bicarbonate

HCO<sub>3</sub> - bicarbonate ion concentration

TCO<sub>2</sub> - carbon dioxide content

O<sub>2</sub>Ct - oxygen content

AaDO<sub>2</sub> - alveolar-arterial oxygen gradient

**Spirometry.**

VC - vital capacity

ERV - expiratory reserve volume

IRV - inspiratory recerve volume

TV- tidal volume

FVC - forced vital capacity

FEV1 - Forced expiratory volume at the end of the first second of forced expiration

FEV1/VC - Tiffeneau index

FEV1/FVC - Gensler index

PEF – peak expiratory flow

**Annex 2.**

## **Main laboratory values and their interpretation**

### **Age-related changes in blood**

Changes in the blood system in elderly and senile people occur in the bone marrow, spleen, lymph nodes and tonsils. After 65-70 years, the mass of all components of the blood system is significantly reduced.

In the peripheral blood, the white blood cell count remains normal. There is a decrease in the number of eosinophils and stab neutrophils, lymphocytes of T and B populations, their functional activity also decreases.

In the bone marrow, hematopoietic tissue and adipose tissue are replaced, stroma grows, the number of reticular and collagen fibers increases, vessels supplying blood to the bone marrow are obliterated. The number of blasts in the bone marrow decreases, but they mature in a timely manner. Changes in thrombocytopoiesis occur: the number of megakaryocytes decreases, the number of "old" and degeneratively altered cells increases. At the same time, a small number of young megakaryocytes adequately provides the necessary number of blood platelets, and therefore, thrombocytopenia is not observed in old age.

In general, the functional activity of the bone marrow decreases, especially erythropoiesis, which contributes to the development of anemia.

### **Complete blood count**

- White blood cell count(WBC).
- Red blood cell count(RBC).

- Hemoglobin concentration (HGB).
- Hematocrit (HCT).
- RBC indices: mean corpuscular volume (MCV), mean cell hemoglobin (MCH), mean cell hemoglobin concentration (MCHC), RBC distribution width-coefficient of variation(RDW-CV).
- Platelet count (PLT).
- Thrombocrit (PCT).
- Thrombocyte indices: mean platelet volume (MPV), platelet distribution width (PDW).
- Leukocyte formula.

**White blood cells** form in the bone marrow and lymph nodes. These are nuclear cells that provide immunity. In adults, the blood contains  $4-9 \times 10^9 / l$  (4000 - 9000 in 1  $\mu$ l) white blood cells. Their numbers in the blood of a healthy person change depending on the time of day and the functional state of the body. 2 groups of leukocytes are distinguished: granulocytes (granular), these include neutrophils, eosinophils and basophils; agranulocytes (non-granular) - lymphocytes and monocytes. Clinically, the number of leukocytes and changes in the ratios between individual groups and types of leukocytes are of the diagnostic value. The percentage of leukocyte types is called the **leukocyte formula**. When evaluating the results of a leukogram, one should always consider not only the content of certain types of white blood cells, but also their absolute values. This is due to the fact that an increase in the blood absolute content of any one type of white blood cell leads to a decrease in the percentage of other forms of white blood cells.

### **Types of white blood cells**

**Neutrophils** make up 50-75% of all WBCs. The main function of neutrophils is to protect the body from microbial infections and toxins of microorganisms. Neutrophils carry out phagocytosis and other antimicrobial reactions, production of interferon in case of viral infection.

**Eosinophils** (1-5% of all WBCs). Their main function is to neutralize toxins

of protein origin, foreign proteins, antigen-antibody complexes. Eosinophils also phagocytize granules of basophils and mast cells that contain a lot of histamine.

**Basophils** (0-1% of all WBCs), like mast cells of connective tissue, produce histamine and heparin. The number of basophils increases during the regenerative (final) phase of acute inflammation. Heparin from basophils prevents blood coagulation (anticoagulant) in the focus of inflammation, and histamine expands the capillaries (vasodilator), which contributes to resorption and healing.

**Monocytes** (2-10% of all WBCs) - capable of amoeboid movement, exhibit pronounced phagocytic and bactericidal activity. After migration to tissues, monocytes turn into macrophages, which are involved in phagocytosis and the formation of specific immunity.

**Lymphocytes** (20-40% of all WBCs). Lymphocytes are able not only to penetrate into tissues, but also to return back to the blood, they are responsible for the formation of specific immunity, carry out the function of immune surveillance in the body, and maintain the genetic constancy of the internal environment. Lymphocytes are divided into T - lymphocytes (thymus-dependent) and B - lymphocytes (Bursa-dependent).

**T - lymphocytes** play an important role in immune surveillance. With the weakening of their functions, the risk of developing tumors, autoimmune diseases increases, the tendency to various infections increases.

**B - lymphocytes** are formed in the bone marrow, but in mammals they undergo differentiation in the lymphoid tissue of the intestine, appendix, palatine and pharyngeal tonsils. The main function of B-lymphocytes is to create humoral immunity by producing antibodies. After meeting with antigen, B-lymphocytes migrate to the bone marrow, spleen and lymph nodes, where they turn into cells that are antibodies producers - immune  $\gamma$  - globulins (immunoglobulins).

**Leukocytosis** is a condition in which the number of leukocytes exceeds 10,000 in 1  $\mu$ l of blood. There are physiological and reactive leukocytosis:

a) physiological leukocytosis is due to the redistribution of leukocytes



between the vessels of various organs and tissues. It is characterized by a slight increase in the number of leukocytes, by the absence of changes in the leukocyte formula and by short duration.

b) reactive leukocytosis develops in inflammatory and infectious diseases.

A white blood cell count of less than 4000 in 1  $\mu\text{l}$  of blood is called leukopenia.

**Agranulocytosis** - the content of neutrophils below  $0.75 \times 10^9 / \text{L}$  with a decrease in the total number of leukocytes below  $1 \times 10^9 / \text{L}$ .

**Red blood cells** are produced in the body of an adult in the red bone marrow, deprived of a nucleus, have the form of a biconcave disc with peripheral thickening and retraction in the center. The diameter of red blood cells is 7.2 - 7.5 microns, the average thickness ranges from 2.1 to 2.4 microns, the volume is 86 - 90  $\text{mm}^3$ , the total surface is 140 - 145 microns. About 10% of red blood cells circulating in the blood are destroyed daily. This occurs mainly in the cells of the phagocytic mononuclear system in the spleen, liver, bone marrow

Red blood cells with a diameter of 7.2 - 7.5 microns are called **normocytes**. In healthy people, there are about 70% of normocytes in the peripheral blood.

**Anisocytosis** is the presence of red blood cells, which vary in size, with the predominance of small diameter red blood cells (**microanisocytosis**) or large diameter (**macroanisocytosis**). Normally, anisocytosis is 11.5-14.5% of total RBCs. There are degrees of anisocytosis severity. Mild anisocytosis (+): approximately 25% of red blood cells differ from normal red blood cells; Moderate anisocytosis (++) : about 50% of red blood cells differ in size from normocytes. Severe anisocytosis (+++), in which 70 - 75% (or more) differs from normocytes. With extreme anisocytosis (++++), almost all red blood cells differ in size from normal red blood cells. It is important to note the size red blood cells: microcytes, macrocytes, or mixed. The predominance of microcytes in the peripheral blood (red blood cells with a diameter of 6.5-7.0 microns) often occurs due to a lack of iron in the body. Microcytes appear in iron deficiency, sideroblastic anemia, with

thalassemia, tumors. Macrocytosis (a large content of red blood cells with a diameter of 8 - 9 microns) is observed after blood loss, increased decay of red blood cells, in cancer, stomach polyps, leukemia, and pregnancy anemia. Macrocytes are detected in blood regeneration, deficiency of B12 and folic acid, liver diseases (especially cirrhosis), hypothyroidism, leukemia, cytostatic and immunosuppressive therapy and alcoholism. Megalocytosis (red blood cells with a diameter of more than 9 microns) occurs due to a deficiency of vitamin B12, folic acid. It can be in pregnancy anemia and in other cases of macrocytosis.

Erythrocytes by the **color index**: normochromic (CI = 0.9 - 1.1), hypochromic (CI less than 0.85) and hyperchromic (CI more than 1.15). **Hypochromia** indicates a decrease in the hemoglobin content in red blood cells, typical of iron deficiency, sideroblastic anemia, thalassemia. Red blood cell **hyperchromia** manifests itself with a deficiency of vitamin B12, folic acid. Erythrocyte **normochromia** occurs in acute posthemorrhagic anemia (on the first day after blood loss), hypo- and aplastic, non-spherocytic hemolytic anemia and anemia in case of kidney diseases, chronic infections.

**Anisochromia** - the different color intensity of red blood cells in a peripheral blood preparation, it is a sign of a violation of hemoglobin synthesis.

**Poikilocytosis** is variation in cell shape of erythrocytes. Spherocytes (spherical red blood cells) occur with hereditary microspherocytosis, hemolytic anemia caused by blood incompatibility, DIC, septicemia, after surgical interventions on the heart (installing artificial heart valves).

**Ovalocytes** (oval red blood cells) are found in hereditary ovalocytosis, hemolytic anemia, severe iron deficiency, megaloblastic anemia, leukemia.

**Leptocytes** (target erythrocytes) are detected in thalassemia, iron deficiency, sickle cell anemia, liver disease with jaundice, obstructive jaundice, alcoholism, after splenectomy.

**Stomatocytes** (erythrocytes with central enlightenment in the form of a narrow linear strip) are found with hereditary stomatocytosis (a form of hemolytic

anemia), immune forms of hemolytic anemia, after blood transfusions, in cirrhosis and liver tumors, obstructive jaundice, acute alcohol poisoning.

**Acanthocytes** are found in hereditary acanthocytosis (a form of hemolytic anemia), severe liver diseases, after splenectomy, during heparin therapy and in alcoholism.

**Echnocytes** (erythrocytes with multiple outgrowths (as if covered with thorns) are detected in acute bleeding, uremia, thrombocytopenic purpura, stomach cancer, acute bleeding.

**Teardrop-shaped** red blood cells form in toxic hepatitis, myelofibrosis.

**Sickle** erythrocytes - characteristic of sickle cell anemia.

**Schistocytes** - fragments of destroyed red blood cells are found in severe anemia, DIC, vasculitis, glomerulonephritis, uremia, after endoprosthetics.

**Inclusion bodies of red blood cells.** Normal red blood cells do not contain inclusions. In pathologic conditions, there can be basophilic granularity. Normally, their number is from 0 to 3 - 4 per 10,000 red blood cells. Red blood cells with basophilic granularity are found in large numbers in toxic damage to the bone marrow (lead, zinc, bismuth, mercury poisoning), megaloblastic anemia, thalassemia, poisoning with hemolytic poisons, after splenectomy.

**Cabot rings** are the remnants of the nuclear envelope in the form of a circle, eight-shaped, bagel, of red and purple color. May occur in severe forms of anemia (megaloblastic, metaplastic), leukemia, polycythemia, heavy metal poisoning.

**Siderocytes:** red blood cells with the inclusion of hemosiderin, ferritin (look like small blue granules). An increased number of siderocytes (more than 1%) is determined in sideroblastic anemia, enhanced hemolysis of red blood cells, after splenectomy.

**Heinz bodies (Heinz-Erlich):** 1-3 inclusions of a round shape in the periphery of red blood cells, 1 - 2 microns in size. They appear during the oxidation of hemoglobin by toxic substances (anidine, nitrobenzene, phenylhydrazine, potassium chlorate, nitroglycerin, sulfonamides) and

methemoglobinemia in patients with hemolytic anemia, radiation sickness.

**Schüffner's dots** are small dark pink or red inclusions in the red blood cells of patients with three-day malaria.

**Maurer's clefts:** granularity of various sizes (10 - 15 inclusions) in red blood cells of patients with tropical malaria.

**Reticulocytes** belong to polychromatophilic cells. Normally, the number of reticulocytes is 0.5 - 1% of the total content of red blood cells. This is a population of newly formed red blood cells lacking a nucleus. Reticulocyte after 2 days in the bloodstream becomes a mature red blood cell. The number of reticulocytes in the blood reflects the erythropoietic activity of the bone marrow. In women, the content of reticulocytes is slightly higher than in men. An increase in their amount in the blood indicates the activation of hematopoiesis in the bone marrow and is observed in blood loss (a reticular crisis occurs on the 3rd - 5th day after acute blood loss); hypoxia, treatment with iron supplements, vitamin B12 and folic acid.

A **decrease** in the number of reticulocytes in the blood reflects a decrease of hematopoietic activity, which is typical for aplastic and hypoplastic anemias; anemia that develops due to iron, vitamin B12 or folic acid deficiency, in thalassemia, sideroblastic anemia, bone metastasis, radiation sickness, radiation and cytostatic therapy.

**The erythrocyte sedimentation rate (ESR)** is normally 2 - 15 mm/h in women and 1 - 10 mm/hour in men. This is a non-specific indicator of the pathological condition of the body. Accelerating ESR factors: a decrease in the number of red blood cells, a decrease in plasma viscosity, an increase in the level of fibrinogen, gamma and beta-globulins, C-reactive protein, paraproteinemia, hypercholesterolemia. An increase in ESR can be observed in physiological conditions, for example, after eating (up to 25 mm / h), pregnancy (up to 45 mm / h), in the postpartum period. An increase in ESR is observed in inflammatory processes in the body and in conditions accompanied by severe intoxication. This occurs in infectious and inflammatory diseases, myocardial infarction, rheumatism,

rheumatoid arthritis, liver damage, kidney diseases (nephrotic syndrome), endocrine disorders, blood system diseases (anemia, lymphogranulomatosis, myeloma disease), anemia, malignant granulomas and monoclonal gamma myelomas (myeloma) , Waldenstrom macroglobulinemia, immunoproliferative disorders, hyperfibrinogenemia, hypercholesterolemia, exposure to certain drugs - morphine, dextrin, methyldopa, viamine A, poisoning with chemical substances (lead, arsenic), and others. Decreased ESR observed in erythrocytosis. Factors that slow down ESR include hyperbilirubinemia, hypofibrinogenemia, and an increase in bile acid levels.

The **color index** (CI) shows the relative hemoglobin content in the red blood cell. Normally, its value is 0.85-1.05. CI is calculated by the formula:  $CI = 3 \times Hb \text{ g\%} / RBC.$ , Where: Hb - the amount of hemoglobin in g/l, RBC - the number of red blood cells in 1 l. CI is clinically similar to the average erythrocyte hemoglobin content (MCH). An increase in CI is hyperchromia of red blood cells, a decrease in CI is hypochromia of red blood cells.

#### General Blood Test

Parameter	Normal values	
	male	female
<b>RBC x 10<sup>12</sup> / l</b>	4,0-5,0	3,7-4,7
Hemoglobin(HGB), g/l	130-160	120-140
Hematocrit (HCT),%	40-48	36-42
Mean cell volume (MCV), fl	<b>75-96</b>	
Mean cell hemoglobin (MCH), pg	27-31	
Mean cell hemoglobin concentration (MCHC),%	33-37	
Reticulocytes ,%	0,2-1,2	
Leukocytes (WBC), x10 <sup>9</sup> / л	4-9	
Platelets (PLT), x10 <sup>9</sup> / л	180-320	
<b>ESR, mm / h</b>	1-10	2-15

#### Leukocyte formula

Parameter	Normal values
-----------	---------------

	%	$\times 10^9 / l$
<u>Neutrophils</u> :		
Stab neutrophils	1-6	0,04-0,3
Segmented neutrophils	45-72	2,0-5,5
Eosinophils	0,5-5	0,02-0,3
Basophils	0-1	0-0,065
Monocytes (MON)	3-9	0,09-0,6
Lymphocytes( LYM, LY%)	18-40	1,2-3,0

### **The main types of changes in leukocyte formula**

- Neutrophilic-eosinopenic; characterized by an increased content of leukocytes, neutrophils, a decrease in eosinophils, lymphocytes, monocytes; observed in cancer, pneumonia, peritonitis, septic infections;
- Neutrophilic-eosinophilic; leukocytosis, a neutrophilic shift to the left, a decrease content of lymphocytes (lymphopenia), monocytes (monocytopenia), an increased content of eosinophils are detected; occurs in pulmonary tuberculosis, lymphogranulomatosis, scarlet fever.
- neutropenic; characterized by a decreased content of leukocytes and neutrophils, a "degenerative" shift to the left, relative lymphocytosis; seen in infectious diseases (typhoid fever, measles, brucellosis, flu, etc.).
- Lymphatic and monocytic reactions; shifts corresponding to this type are characterized by leukocytosis, lymphocytosis, monocytosis; found in infectious diseases.
- Protozoal: accompanied by leukopenia, neutropenia (with a neutrophilic shift to the left), lymphopenia; observed with malaria, leishmaniasis.

### **Biochemical blood test**

#### **Carbohydrate metabolism:**

glucose:

blood 3,33-5,55 mmol / l

plasma 4,22-6,11 mmol / l

Glycated hemoglobin 4,5-6,1%

## Glycemia test and interpretation

Result	Oral Glucose Tolerance Test (OGTT)
Normal	less than 140 mg/dl
Prediabetes	140 mg/dl to 199 mg/dl
Diabetes	200 mg/dl or higher

### *1999 WHO Diabetes criteria – Interpretation of Oral Glucose Tolerance Test*

Glucose levels	NORMAL		Impaired Fasting Glycaemia		Impaired Glucose Tolerance		Diabetes Mellitus	
			(I.F.G.)		(I.G.T.)		(D.M.)	
Venous Plasma	Fasting	2 hrs	Fasting	2 hrs	Fasting	2 hrs	Fasting	2 hrs
(mmol/l)	< 6.1	< 7.8	≥6.1 & <7.0	<7.8	< 7.0	≥ 7.8	≥ 7.0	≥11.1
(mg/dl)	< 110	< 140	≥110 & <126	<140	< 126	≥ 140	≥ 126	≥ 200

Type of Diabetes	Normal glucose tolerance	Hyperglycemia	
		Pre-diabetes*	Diabetes Mellitus
		Impaired fasting glucose or impaired glucose tolerance	Not required for insulin control
Type 1			
Type 2			
Other specific types			
Gestational Diabetes			
Time (years)			
FPG	<5.6 mmol/L (100 mg/dL)	5.6–6.9 mmol/L (100–125 mg/dL)	≥7.0 mmol/L (126 mg/dL)
2-h PG	<7.8 mmol/L (140 mg/dL)	7.8–11.0 mmol/L (140–199 mg/dL)	≥11.1 mmol/L (200 mg/dL)
HbA1C	<5.6%	5.7–6.4%	≥6.5%

Source: D. L. Kasper, A. S. Fauci, S. L. Hauser, D. L. Longo, J. L. Jameson, J. Loscalzo: Harrison's Principles of Internal Medicine, 19th Edition. www.accessmedicine.com  
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**Protein metabolism:**

Total protein 65-85 g/l  
Albumin: 30-50 g/l  
Globulin: 20-30 g/l  
 $\alpha_1$  3-6%  
 $\alpha_2$  5-8%  
 $\beta$  9-13%  
 $\gamma$  15-22%  
Seromucoid 0,13-0,2 un.  
Creatinine 50-115  $\mu\text{mol} / \text{l}$   
Urea 4,2-8,3  $\mu\text{mol} / \text{l}$   
Uric acid  
    male 214-458  $\mu\text{mol} / \text{l}$   
    female 149-404  $\mu\text{mol} / \text{l}$   
Thymol turbidity test 0-5 un.

**Lipid metabolism:**

Total cholesterol 3,9-5,2 mmol / l  
HDL 0,9-1,9 mmol / l  
LDL under 2,2 mmol / l  
 $\beta$ -lipoproteins 35-55 un.  
Triglycerides 0,45-1,7 mmol / l

**Dyslipidemia is determined according to lipid profile.**

- Low-density lipoprotein cholesterol (LDL) contains up to 2/3 of plasma cholesterol and transports it to tissues. LDL is calculated using the Friedwald formula: in mmol / L -  $\text{LDL} = \text{total cholesterol} - \text{HDL} - \text{C} (0.45\text{TG})$ , and in mg / dl -  $\text{LDL} = \text{total cholesterol} - \text{HDL} (0.2\text{TG})$ , while it is necessary that the TG are less 4.5 mmol / L.
- High Density Lipoprotein Cholesterol (HDL) captures cholesterol from the tissues and transports it to the liver where bile acids are formed.
- Klimov's atherogenicity index is:  $(\text{Total Cholesterol} - \text{HDL}) / \text{HDL} = 3.0-3.5$
- Non-HDL cholesterol is calculated by simple subtraction of HDL from total cholesterol and, unlike LDL, does not require TG to be less than 5 mmol / L.



**Table: Fredrickson Classifications of Dyslipidemia**

Diagnosis	Fredrickson classification	Lipids	Lipoproteins	Genetics
Familial Hyperchylomicronemia	Type 1	↑TG	↑Chylomicrons	Autosomal recessive due to 2 mutant alleles of <i>LPL</i> , <i>ApoC2</i> , <i>ApoA-V</i> , <i>LMF-1</i> , <i>GPIHBP1</i>
Familial Hypercholesterolemia	Type 2A	↑TC	↑LDL	Autosomal codominant; heterozygous form results from 1 mutant allele of LDL receptor, ApoB, or PCSK9; homozygous form results from 2 mutant alleles of these genes.
Familial Combined Hyperlipoproteinemia	Type 2B	↑TC, ↑TG	↑VLDL, ↑LDL	Polygenic
Dysbetalipoproteinemia	Type 3	↑TC, ↑TG	↑IDL	Apo E2/E2 homozygosity; or heterozygous rare mutations in ApoE.
Primary Hypertriglyceridemia	Type 4	↑TG	↑VLDL	Unknown
Mixed Hypertriglyceridemia	Type 5	↑TC, ↑TG	↑VLDL, ↑Chylomicrons	Polygenic

- Triglycerides lead to an increase in VLDL, have a toxic effect on the endothelium, and reduce the level of HDL. Possible causes of hypertriglyceridemia: genetic predisposition, obesity, type 2 diabetes, alcohol abuse, a diet high in simple carbohydrates, kidney disease, hypothyroidism, pregnancy, autoimmune disorders, medications (corticosteroids, estrogens, thiazides, beta-blockers, antiretroviral, psychotropic drugs).
- DLP I - hyperchylomicronemia. It usually develops in childhood, has a family character (inherited deficiency of the LPL enzyme). The development of atherosclerosis is not characteristic of this type of DLP, the clinical signs are hepatosplenomegaly, xanthomatosis, abdominal colic, acute pancreatitis.
- DLP II - divided into two subtypes. Type IIa DLP is hyperbetalipoproteinemia characterized by a high LDL content. Type IIb DLP – hyperbeta- and hyperprebetalipoproteinemia, which is characterized by a high content of both LDL and VLDL (a hereditary disease due to the lack of LDL

receptors). Type II DLP is often manifested in CHD in the event of sudden death in childhood and adolescence due to myocardial infarction. Usually, such a fatal outcome occurs in individuals with homozygous heredity. In individuals with heterozygous heredity, coronary heart disease develops later and is not so acute.

- Type III DLP - dysbetalipoproteinemia, or “floating” DLP. Lipoproteins with extremely high cholesterol and high electrophoretic mobility (“floating” lipoproteins) appear in blood serum. They accumulate in the blood due to impaired conversion of VLDL to LDL. Patients with type III DLP have a pathological tolerance to carbohydrates, that is, a carbohydrate diet leads to a persistent increase in the level of triglycerides in the blood. This type of DLP is often combined with manifestations of atherosclerosis, including coronary heart disease and damage to the vessels of the lower extremities, mainly in adults.
- Type IV DLP - hyperprebetalipoproteinemia. Its sign is an increased level of VLDL. It is determined in elderly people with atherosclerosis of coronary arteries, obesity, diabetes mellitus, etc. Some patients with DLP of this type have a decrease in carbohydrate tolerance.
- Type V DLP - hyperprebetalipoproteinemia and hyperchilomicronemia. Clinically, this type has the same symptoms as type I DLP, sometimes combined with latent or mild diabetes. In contrast to type I DLP, with type V DLP, the activity of the LPL enzyme is only moderately reduced. IHD with this type of DLP, as a rule, is not detected.

**Atherogenic hyperlipidemia:**

- increased total cholesterol;
- increased LDL and VLDL;
- increased triglycerides;
- decreased HDL cholesterol.
- By the types of hyperlipidemia, this is type II A, II B, type IV.

### **Clinical classification of dyslipidemia (CNT, 2007)**

- Hypercholesterolemia (corresponds to Type IIa by D. Fredrickson).
- Combined dyslipidemia (corresponds to Type IIb, III according to D. Fredrickson).
- Hypertriglyceridemia (corresponds to Type IV by D. Fredrickson).

### **Bile pigment metabolism:**

Total bilirubin 8,5-20,5 mmol / l  
Direct bilirubin 0, -5,1 mmol / l  
Indirect bilirubin до 16,5 mmol / l

### **Mineral metabolism:**

Sodium 135-152 mmol / l  
Potassium 3,6-5,2 mmol / l  
Total Calcium 2,2-2,75 mmol / l  
Ionized Calcium 1,0-1,15 mmol / l  
Magnesium 0,7-1,2 mmol / l  
Inorganic Phosphate 0,81-1,55 mmol / l  
Serum Iron 12,5-30,4  $\mu\text{mol}$  / l  
TIBC 50-84  $\mu\text{mol}$  / l  
Ferritin:  
    male 15-200  $\mu\text{g}$  / l  
    female 12-150  $\mu\text{g}$  / l

### **Enzymes:**

AST 0,1-0,45  $\mu\text{mol}$  / (h x ml)  
ALT 0,1-0,68  $\mu\text{mol}$  / (h x ml)  
Diastase 12-32  $\mu\text{g}$  / (h x ml)  
GGT:  
    male to 800 nmol / (s x l)  
    female to 580 nmol / (s x l)  
Alkaline phosphatase to 120 IU / l  
Acidic phosphatase to 167 nmol / (s x l)  
LDH 0,8-4,0  $\mu\text{mol}$  / (h x ml)  
CPK 24-170 U / l  
MB-CPK 0-24 U / l

### **Coagulogram:**

Prothrombin time (PTT) 12-20 s  
Prothrombin index (PTI) 90-105%  
Total Fibrinogen 2-4 g / l  
Fibrinogen B - none

APTT 35-50 s  
Blood clotting time 5-10 min.  
Plasma recalcification time 60-120 s  
Citrated plasma heparin tolerance 10-16 min.

### **Gastric secretion test**

Gastric juice:

Volume per day - 2-3 L  
Specific gravity - 1005  
Reaction, pH - 1,6-1,8  
Fasting gastric contents:  
Volume - 5-40 ml  
Total acidity - no more than 20-30 mmol / l  
Free hydrochloric acid - to 15 mmol / l  
Pepsin - 0-21 mg%

Basal secretion of gastric juice:

Total content volume, collected in 4 portions during 60 min after aspiration of fasting portion - 50-100 ml  
Total acidity - 40-60 mmol / l  
Free HCl - 20-40 mmol / l  
Bound HCl - 10-15 mmol / l  
Total HCl debit - 1,5-5,5 mmol / h.  
Free HCl debit - 1,0-4,0 mmol / h.  
Pepsin debit - 4-40 mg/h.

Stimulated by histamine (submaximal):

60 min portion volume - 100-150 ml  
Total acidity - 80-100 mmol / l  
Free HCl - 65-85 mmol / l  
Bound HCl - 10-15 mmol / l  
Total HCl debit - 8-14 mmol / h.  
Free HCl debit - 6,5-12 mmol / h.  
Pepsin debit - 50-90 mg/h.

Fasting gastric content microscopy:

Starch grains - turn out to be single  
Muscle fibers - none  
Fat - none  
Plant cells - none  
Squamous epithelium - small amount

Red blood cells - none  
White blood cells - small amount, changed  
Yeast - solitary  
Sarcina - none  
Lactic acid rods – none

### **Duodenal aspiration**

Portion «A» (duodenal content):  
Volume - 20-35 ml (1 ml / 1 min.)  
Colour – golden yellow  
Transparency - transparent  
Specific gravity - 1007-1015  
Reaction - alkalescent  
Portion «B» (gallbladder bile):  
Volume - 30-60 ml  
Colour – dark brown (olive)  
Transparency - transparent  
Specific gravity - 1016-1032  
Reaction - alkaline  
Portion «C» (hepatic ducts bile):  
Volume - 30 ml  
Colour – golden brown  
Transparency - transparent  
Specific gravity - 1007-1010  
Reaction - alkaline

### **Bile portions microscopic examination**

Portion «A»:  
Epithelium – small amount  
WBCs - 1-2 in FoV  
Mucus – small amount  
Crystals of Cholesterol and Calcium Bilirubinate - none  
Culture - negative  
Portion «B»:  
Epithelium – small amount  
WBCs - 2-3 in FoV  
Mucus – small amount  
Crystals of Cholesterol and Calcium Bilirubinate - solitary  
Culture - negative  
Portion «C»:  
Epithelium – small amount  
WBCs - 2-3 in FoV  
Mucus – small amount

Crystals of Cholesterol and Calcium Bilirubinate - none  
Culture - negative

### **Fractional duodenal aspiration**

Phase I – Common bile duct: bile of portion «A». Excretion time 10-20 min. Volume 20 ml.

Phase II – closed sphincter of Oddi: 2-6 min., no bile.

Phase III – bile of portion «A» from distal part of common bile duct: excretion 3-5 min., volume 3-5 ml.

Phase IV - portion «B»: excretion 20-30 min., volume 30-50 ml.

Phase V - portion «C»: excretion 20-30 min., volume 10-30 ml.

### **Sputum examination**

#### **Macroscopic examination**

The **amount** of sputum depends on the kind of the pathological process in the bronchi and lungs. Sputum amount can vary from 2-5 ml in one cough, or daily up to half a cup. It is caused by an increased secretion of the mucous membrane due to bronchitis, focal pneumonia. A coughing up to 200 ml, or daily up to 1.5 l, is observed in abscess, bronchiectasis, tuberculosis and lung cancer.

**Character:** mucous, mucopurulent, mucopurulent-bloody, serous, serous-purulent, bloody-mucous, etc.

Dividing into layers is observed when large cavities in the lung are emptying: the lower layer (dense) of pus, detritus, the upper one is liquid, the third layer is often foam.

**Color:** grayish, yellow or green (presence of pus), rusty, red, brown (impurities of blood and its decay products), gray and black (coal and dust), white (flour dust), etc.

**Sputum consistency:** viscous (presence of mucus), liquid - serous fluid, sticky (large amount of fibrin).

There is usually no smell. A fetid or putrid odor appears with an abscess, gangrene, bronchiectasis, and the decay of malignant tumors.

#### **Microscopic examination**

**White blood cells** are always present in sputum. Their number is greatest if the sputum is purulent.

**Eosinophils** are found in large clusters in separate areas in bronchial asthma, in the presence of helminths, echinococci or neoplasms.

There could be solitary **red blood cells** in any sputum. A large number of them is found in hemoptysis.

**Squamous epithelial** cells are from the oral cavity, nasopharynx. They have no diagnostic value.

Cells of **cylindrical ciliated epithelium** are found in large numbers in inflammatory processes of the respiratory tract.

**Alveolar macrophages** are often detected in sputum mucosa with a low content of pus (pneumonia, bronchitis, tuberculosis). Siderophages - alveolar macrophages containing hemosiderin (a breakdown product of erythrocyte hemoglobin) are found in congestive lung conditions and pulmonary infarction.

The presence of **elastic fibers** indicates the destruction of lung tissue (abscess, tuberculosis, gangrene, tumor).

**Curschmann's spirals and Charcot-Leiden** crystals are formed in the presence of bronchial asthma.

**Cholesterol crystals** form during the breakdown of sputum fat in cavities as a result of congestion.

**Dietrich plugs** (small grains with an unpleasant odor) are contained in purulent sputum in the cavities (lung abscess, bronchiectasis).

**Actinomyces druses** - plexuses of the thin mycelium with a bulbous extension on the ends.

**Atypical cells** are a sign of malignant tumors.

**Bacterioscopic examination:** a preparation is made from purulent particles of sputum and stained according to Ziehl–Neelsen (to detect mycobacterium tuberculosis) and Gram (to detect coccal flora). A more reliable method for the recognition of microflora in sputum is the bacteriological method, which allows to identify the causative agent of the disease.

## **Pleural fluid examination**

**Character.** Pleural fluid can be serous, serous-purulent, purulent, putrefactive, serous-fibrinous, hemorrhagic, chylous, pseudochylosis, cholesterol.

Transudate is observed in chronic heart failure in the stage of decompensation or liver cirrhosis. It is a transparent serous, almost colorless or yellowish liquid.

Serous exudate differs little from transudate, but when standing, a fibrin clot (serous-fibrinous) forms; often seen in tuberculosis.

Serous-purulent exudate (pleural empyema): a turbid, yellow liquid with a loose sediment of gray color.

Purulent exudate (pleural empyema): cloudy, yellow-green, thick consistency.

Putrid exudate occurs in lung gangrene with a breakthrough into the pleural cavity: cloudy, gray-green with a putrefactive odor, contains a lot of detritus, bacteria.

Hemorrhagic exudate is formed in malignant neoplasms, hemorrhagic diathesis, and traumatic injuries. It is a cloudy, red, or brownish liquid. To detect pus, Petrov test is carried out: distilled water is added to the studied exudate, which causes hemolysis of RBCs. If the exudate is purely hemorrhagic, then it becomes transparent, and if there is an admixture of pus, the liquid remains cloudy.

Chylous exudate is observed if there is a rupture of large lymphatic vessels. A milky, turbid liquid contains a large amount of fat. When ether is added, it becomes enlightened.

The **color** of the pleural fluid can also be different. Transudates and serous exudates are light yellow in color, purulent - yellow-green in color. A large admixture of blood gives the liquid a reddish-brown tint. Milky white color is characteristic of chylous and pseudochylous exudates.

Transudates and serous exudates are transparent, others are cloudy.

**Specific gravity** of pleural fluid is determined using a urometer, it ranges from



1.002 to 1.025. The lower specific gravity is observed in transudates and does not exceed 1.015. Determination of chemical properties. The protein content in transudate is 5 - 2.5 g / l, exudates - 30-50 g / l. Albumins predominate in transudates, lipoproteins predominate in exudates. The Rivalta test is used to determine transudate and exudate: in a cylinder with a capacity of 100 ml with distilled water, add 2-3 drops of concentrated acetic acid and 1 - 2 drops of the studied pleural fluid. If a white cloud forms upon adding liquid, dropping to the bottom of the cylinder, the sample is positive. The exudate contains serosomucin, which gives a positive test.

Microscopic examination: neutrophilic leukocytes predominate in purulent exudate, lymphocytes predominate in serous exudate, the presence of eosinophils in serous exudate is considered as a manifestation of an allergic process and is observed with rheumatic effusions, tuberculosis, and parasitic diseases. Plasma cells - can also be in serous and purulent exudate with prolonged inflammatory processes. Red blood cells - are present in any effusions in a small amount and are associated with a traumatic admixture of blood at the time of puncture. Hemorrhagic exudates contain a large number of red blood cells. Tumor cells (atypical) are always in the form of conglomerates, typical of the diagnosis of malignant neoplasms.

#### **General urine test**

Color	Straw yellow
Transparency	transparent
Reaction	acescent/neutral
Specific gravity	1,008- 1,026
Protein	0,033 g / l
Glucose	none
Acetone, ketone bodies, bile pigments	none
<b>Urine sediment microscopy</b>	
Squamous epithelium	small amount
Renal epithelium	non
WBC	0-5 in the FoV for females 0-3 in the FoV for males
RBC	0-1 in the FoV

Hyaline casts	1-2 in the FoV
Mucus	small amount
Bacteria	none
Salts	small amount

### **Fecal examination**

Amount per day - 100-250 g

Consistency – smooth and soft, may have cracks on its surface

Shape is cylindrical (sausage-shaped)

Brown colour

Reaction - neutral or alkalescent

Mucus, blood - none

Fecal Microscopy:

Muscle fibers - none or solitary (digested, without transverse striation)

Connective tissue - none

Neutral Fat - None

Fatty acids - none

Soaps - none

vegetable fiber:

digested - single cells or cell groups

undigested - contained in different quantities

Starch - none

Iodophilic flora - none

Mucus, epithelium - none

White blood cells - none

### **Annex 3**

### **Schemes of describing the main instrumental studies**

#### **Scheme for the description and analysis of electrocardiograms (ECG)**

Waves and intervals.

Normally:

P wave is < 0.11 sec., PQ interval is 0.12 - 0.20 sec.,

Q wave is < 0.04 sec, amplitude of Q wave is not > ¼ amplitude of R wave,

QRS complex is no longer than 0.10 sec.,

QT interval is 0.35 - 0.45 sec.

### Plan of ECG description:

1. Rhythm (rhythm source) \_\_\_\_\_,  
(regularity)\_\_\_\_\_.

2. Heart rate\* \_\_\_\_\_ / min.

\* Formula for heart rate calculation:

Heart rate =  $60 : (RR \text{ in mm} \times 0,02 \text{ sec})$  if the speed is 50 mm / sec

Heart rate =  $60 : (RR \text{ in mm} \times 0,04 \text{ sec})$  if the speed is 25 mm / sec

3. Position of the electric axis of the heart \_\_\_\_\_.

4. Intervals duration: PQ = \_\_\_\_ sec, QRS = \_\_\_\_ sec, QT = \_\_\_\_ sec .

5. Criteria of the heart chambers hypertrophy:

Left atrium hypertrophy \_\_\_\_\_ (criteria in this ECG)

Left ventricle hypertrophy \_\_\_\_\_ (criteria in this ECG)

Right atrium hypertrophy \_\_\_\_\_ (criteria in this ECG)

Right ventricle hypertrophy \_\_\_\_\_ (criteria in this ECG)

Criteria for myocardial ischemia, acute MI, scar \*\*:

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\*\* in the presence of these changes, you need to describe them, specify the localization. If it is an acute MI, the localization and stage of the infarction should be indicated.

6. Heart rhythm disorders (describe) \_\_\_\_\_

7. Conduction disturbances (describe) \_\_\_\_\_

### Method for ECG analysis.

Check the correctness of the ECG registration technique, the amplitude of the control millivolt (norm 10 mm).

Assess the following ECG characteristics.

**Heart rhythm regularity.**

Measure the duration of several adjacent R - R intervals and compare them with each other. If the duration of the R - R intervals differ from each other no more than 0.10 - 0.15 s, then the heart rhythm is correct, regular.

**Determine heart rate.**

If the rhythm is regular: 60 divided by the duration of the R - R interval, expressed in seconds (heart rate =  $60 : R-R$ ). The normal heart rate (HR) in healthy individuals at rest is from 60 to 90 per minute. If the rhythm irregular (R - R intervals differ by more than 0.15 s), use the average duration of several R - R intervals, or determine the arithmetic average of the maximum and minimum heart rates.

**The determination of the source of excitation** is carried out according to the positions of the P waves in relation to the QRS complexes.

Criteria for sinus rhythm:

- P wave is positive in the II standard lead and in leads I, aVF, V2-V6. P wave is negative in aVR lead.
- The P wave is located in front of each ventricular complex and is recorded at the same distance to the QRS in each cardiac cycle.
- P wave is permanent, of the same shape in the same lead.

Conductivity Function Assessment:

- P wave duration (normal up to 0.10 s).
- the duration of the P-Q interval (normal from 0.12 s to 0.20 s, depending on the heart rate).
- the duration of the QRS complex (norm from 0.06 s to 0.10 s).
- the duration of the interval of internal deviation in the chest leads is in V 1 (normal to 0.03 s) and in V 6 (normal to 0.05 s).

**Determination of the electric axis of the heart** (position in the frontal plane):

Visually determine the electric heart here by the characteristics of the R and S

waves in standard leads. Variants of the electric axis position: normal (angle  $\alpha$  from  $+30^\circ$  to  $+69^\circ$ ):  $R_{II} > R_I \geq R_{III}$ , if the S wave in I and III leads is less than the R wave in the same leads; horizontal (angle  $\alpha$  from  $0$  to  $+29^\circ$ ):  $R_I \geq R_{II} > R_{III}$ , in the III lead the S wave is registered less in amplitude of the R wave in this lead; vertical (angle  $\alpha$  from  $+70^\circ$  to  $+99^\circ$ ):  $R_{III} \geq R_{II} > R_I$ , in the I lead the S wave is less in amplitude than the R wave in this lead.

#### **Atrial P wave analysis:**

- P wave duration (normal up to 0.10 s).
- P wave amplitude (norm up to 2.5 mm).
- determine the polarity of the P wave (normal is always positive in leads I, II, aVF, V 2-6 and always negative in the aVR lead).
- evaluate the shape of the P wave and note possible deformations (all positive P waves have the shape of a semi-shaft with smooth contours).

#### **QRS complex analysis.**

Q wave - normally does not exceed  $\frac{1}{4}$  of the R wave height in the same lead, its duration is normal to 0.03 s.

R wave - the amplitude is normal in the limb leads to 20 mm, in the chest leads - up to 25 mm. Compare with the amplitudes Q and S in the same lead, measure the interval of internal deviation in the leads V 1 (normal up to 0.03 s) and V 6 (normal up to 0.05 s), evaluate the shape of the wave.

S wave: amplitude up to 20 mm. Compare with the R wave height R in the same lead, evaluate the shape and duration (normal to 0.04 s).

#### **S-T segment analysis.**

Normally, the S - T segment is isoelectric, the deviation from the isoelectric line can be  $\pm 0.5-1$  mm.

In leads V1-2, normally can be a rise in the ST segment by 1-2 mm.

In leads V5-6, normally shift of the ST segment is possible above and below the isoelectric line up to 0.5 mm. It is necessary to determine and describe the shape of the possible displacement of the S - T segment.

### **T wave analysis:**

- T-wave polarity (normally always positive in leads I, II, aVF, V 2-6 and always negative in aVR lead).
- the shape of the T wave (normally the wave is asymmetrical, has a sloping ascending part and a slightly steeper descending part).
- the amplitude of the T wave (normally, in the limb leads, the amplitude of the wave is up to 6 mm, in the precordial leads - up to 17 mm).

### **Q - T interval analysis:**

Q - T interval (electrical systole of the heart) is measured from the beginning of the QRS complex to the end of the T wave. It is necessary to compare the duration of the obtained Q - T interval with the data of the table, nomogram, or calculate according to the Bazetta formula:  $Q - T = K \times \sqrt{R - R}$ , where  $K = 0.37$  for men and  $0.40$  for women (normally the duration of the Q - T interval is on average from  $0.35$  to  $0.44$  s and depends, primarily, on the heart rate - decreases with increasing heart rate).

### **Echocardiogramm description (EchoCG)**

Patient's name \_\_\_\_\_

Clinical data: \_\_\_\_\_

#### **Parameters (M-mode, 2D EchoCG)**

		Normal values
Right ventricle		0,9-2,6
Interventricular septum		0,6-1,1 cm
Left ventricle (diastole)		3,5-5,7 cm
Left ventricular wall (diastole)		0,6-1,1 cm
Left atrium		1,9-4,0 cm
Ascending aorta		2,0-3,7 cm

diameter		
Left ventricle injection fraction		> 55%

### Heart valves

	mitral	aortic	tricuspid	pulmonary
insufficiency, degree 1 + -4 +				
stenosis, degree 1 + -4 +				
calcinosis				
additional data				

Pulmonary

hypertension \_\_\_\_\_

Pericardial effusion \_\_\_\_\_

Segmental left ventricular contractility (normo-, hypo-, dyskinesia)

Segments	Anterior septal	Anterior	Lateral	Posterior	Inferior	Septal
Basal						
Mid						
Apical	X			X		

Disorders of diastolic function \_\_\_\_\_

Other Features \_\_\_\_\_

Conclusion: \_\_\_\_\_

\_\_\_\_\_

—

## **Spirogram analysis**

### **Main parameters of external respiration**

#### **A. Static parameters of the external respiration**

1. TV – tidal volume - 0,25-0,5 l.
2. IRV – inspiratory reserve volume - 1,5-2,0 l.
3. ERV – expiratory reserve volume - 1,5-2,0 l.
4. RV – residual volume - 100 ml-1, 5 l.
5. FRC – functional residual capacity (characterizes the degree of lung tissue elasticity) - 2,5-3,0 l.
6. VC (vital capacity): male - 3,5-4,5 l; female - 2,5-4,0 l.
7. TLC – total lung capacity - 4,5-6,5 l.

TLC = VC + RV - the volume of gas in the lungs at maximum inspiration.

#### **B. Dynamic parameters of the external respiration.**

1. Respiratory rate - 12-18 respiratory movements in 1 min.
2. Respiratory minute volume (RMV) - the volume of air entering the lungs: at rest - 6-8 l / min, on maximum physical exertion - 50-60 l / min, for athletes - 120-180 l / min;  $RMV = TV \times \text{Respiratory rate}$ .
3. MVV (Maximal voluntary ventilation): - volume of air entering the lungs in 1 min. in forced breathing; normally equal to 80-200 l / min; sharply decreases with diseases of the respiratory system and the cardiovascular system; athletes - 120-200 l / min.
4. FEV1(Forced expiratory volume) – bronchial patency parameter; it is the volume of air that can forcibly be blown out in first 1 second, after full inspiration; for 20-60 y/o males - 70-85%.
5. PIFR – peak inspiratory flow rate; PEFR – peak expiratory flow rate;  $PEFR = 4-8 \text{ l / s}$ .
6. DS – dead space;  $DS = ADS + PDS$ , где ADS (anatomic dead space) - 0,12-0,18 l - part of the space of the airways (oral cavity, pharynx, trachea, bronchi), that is not involved in gas exchange. PDS (physiologic dead space) air of



the alveoli, which receive an insufficient amount of blood and do not participate in gas exchange.

7. AV – alveolar ventilation;  $AV = (TV - DS) \times RR$  it is about 70-80% of total lung ventilation.

8. Effective ventilation - an integral parameter characterizing the ratio of the volume of air involved in gas exchange to the volume of air, ventilating the lungs due to muscular effort.  $EV = AV / RMV \times 100$ .

9. Total oxygen uptake 0,2 l / min.

10. VO<sub>2</sub> max – maximal oxygen uptake - the amount of oxygen consumed by the body in 1 min. in maximal breathing; VO<sub>2</sub> max = 3-5 l / min или 50-60 ml / min / 1 kg of body mass.

11. RQ- respiratory quotient - calculated from the ratio of carbon dioxide produced by the body to oxygen consumed by the body.

RQ with the oxidation of carbohydrates - 1.0; proteins - 0.8; fats - 0.7; mixed food - 0.82-0.87.

### **The main spirogram parameters in healthy individuals**

Tidal volume (TV)- 0,3-0,9 L

Inspiratory reserve volume (IRV) - 1-1,5 L

Expiratory reserve volume (ERV) - 0,8-1,5 L

Vital capacity (VC) - 3,5-5 L

Forced vital capacity (FVC) - vital capacity from a maximally forced expiratory effort

Forced expiratory volume (FEV<sub>1</sub>) is a volume that has been exhaled at the end of the first second of forced expiration - >85% of the required value.

Tiffeneau index (FEV<sub>1</sub> / FVC,%) - >70% of the required value

### **Criteria for various types of ventilation failure**

#### Obstructive Type:

Decreased forced expiratory volume in 1 second, i.e., FEV<sub>1</sub>

Tiffno index decrease (FEV1 / VC ratio)

Total lung capacity (OEL) is normal or increased

Restrictive Type:

Criterion - decrease in total lung capacity

### **Scheme for describing lung X-Rays**

**Projection** of the image (PA, lateral, AP, decubitis, supine, oblique).

**Special conditions** of radiography (position of patients: sitting or lying down due to the severity of their condition, with dynamic respiratory blur of the image if patient is unconscious, etc.).

**Evaluating** image quality (physical and technical characteristics: optical density, contrast, image sharpness, lack of artifacts).

The **condition of the chest soft tissues** (volume, structure, the presence of foreign bodies or air after injuries, etc.).

The condition of the chest and shoulder girdle **bones** (position, shape, size and structure of the bones: ribs, sternum, visible cervical and thoracic vertebrae, clavicles, shoulder blades, heads of the humerus).

**Comparative evaluation** of pulmonary fields (area, shape, transparency). If symptoms of pathology are detected (extensive or limited dimming or clarification, foci, round or annular shadow), describe in detail their positions, shapes, sizes, shadow density, structure, and contours.

The state of the **lung pattern** (distribution of elements, architectonics, caliber, nature of the contours).

The state **lung roots** (position, shape, size, structure, contours of the elements, the presence of additional formations).

The state of the **mediastinum** (position, shape and width of it as a whole and the characteristic of individual organs).

X-ray (clinical and radiological) conclusion.

### **Calculation of body mass index**

Body mass index (BMI) was developed by A. Quetelet in 1869. The body mass

index is calculated by the formula: divide your weight in kilograms by the square of height in meters:  $BMI = \text{weight (kg)} : [\text{height (m)}]^2$  and measured in  $\text{kg} / \text{m}^2$ .

According to the WHO, body mass index is the most effective way to measure overweight and obesity at the population level, as it can be applied to all adults regardless of gender and age. The indicator may vary for a particular ethnic group. Normal BMI values for the population of developed countries correspond to the interval of 20-25  $\text{kg} / \text{m}^2$ , and for the population of developing countries - 18.5-25  $\text{kg} / \text{m}^2$ .

The body mass index allows you to assess the degree of correspondence of a person's mass and his height and determine whether the mass is insufficient, normal, excess (obesity). BMI is the simplest, most informative and most popular indicator for assessing the level of overweight and the degree of obesity.

Interpretation of BMI:

- 20-25 - normal body weight, there is no health risk;
- 25-30 - overweight, increased health risk;
- 30-35 - obesity - high health risk;
- 35 and more - pronounced obesity, the risk to health is very high

#### **BMI Obesity Classification (WHO, 1997)**

<b>Classification</b>	<b>BMI, <math>\text{kg} / \text{m}^2</math></b>	<b>Health risk</b>
Body weight deficiency	<18,5	Low (high risk for other diseases)
Normal body weight	18,5-24,9	Average
Overweight	25,0-29,9	Mildly Increased
Obesity Class I	30,0-34,9	Moderate
Obesity Class II	35,0-39,9	Severe
Obesity Class III	40,0 and >	Very Severe

#### **Annex 4**

#### **Examples of interpretation of laboratory and instrumental studies**

##### **General Blood Test**

**Date** \_\_\_\_\_

**Patient's name:**

Parameters	Result	Reference values
WBCs	5.6	4.0-9.0 x 10 <sup>9</sup> / l
Hemoglobin	87	M: 130-160 g / l Ж: 120-140 g / l
RBCs	2.1	M 4,0 - 5,0 x10 <sup>12</sup> / l F: 3.7- 4.7 x10 <sup>12</sup> / l
Color index	1.15	0.85-1.05
Reticulocytes	0.5	0.5-1.0%
Mean cell volume (MCV)	113	81 - 99 μm <sup>3</sup>
Mean cell hemoglobin (MCH)	57	27.0-36.0 pg
Mean cell hemoglobin concentration (MCHC)	43	32.0-36.0 g / dl
Hematocrit	30	M.43-54% F.36-47%
Platelets	167	150-390 x10 <sup>9</sup> / l
ESR	32	M: <10 F <15 mm / h

### White blood cell count

Parameters	Result %	Reference value %	Absolute count	Reference value
Eosinophils	1	0,5-5,0	0,06	0-0,5 x 10 <sup>9</sup> /l
Basophils	1	0-1	0,06	0-0,2 x 10 <sup>9</sup> /l
Myelocytes	0	0	0	0
Metamyelocytes	0	0	0	0
Band neutrophils	4	1-6	0,23	0,1-0,6 x 10 <sup>9</sup> /l
Segmented neutrophils	67	50-70	3,94	2,0-7,2 x 10 <sup>9</sup> /l
Lymphocytes	86	19-40	0,98	1,2-3,2 x 10 <sup>9</sup> /l
Monocytes	8	3-10	0,52	0,3-0,8 x 10 <sup>9</sup> /l

**Answer.** There is a decrease in hemoglobin, red blood cells, an increase in

color index, red blood cell indices (MCV, MCH, MCHC) and a normal level of reticulocytes are determined. The revealed changes indicate that the patient: hyperchromic, normoregenerative B12 - folic acid deficiency anemia. It can be a consequence of a disease of the gastrointestinal tract (peptic ulcer, helminthiasis, cancer).

### **Interpretation of myelogram**

#### **An increased count of WBCs is observed in the next conditions:**

- An increased number of megakaryocytes in the bone marrow puncture sample - myeloproliferative processes, metastases of malignant neoplasms in the bone marrow.
- Increased white blood cell / red blood cell ratio: chronic myelogenous leukemia, subleukemic myelosis, leukemoid reactions.
- Increased neutrophil maturation index: blast crisis, chronic myelogenous leukemia.
- Increased blasts more than 20%: acute leukemia.
- Blasts are increased up to 20%: acute leukemia, myeloid forms of chronic leukemia, myelodysplastic syndrome.
- Increased myeloblasts by more than 20%: blast crisis, chronic myelogenous leukemia.
- Myeloblasts are increased to 20%: blast crisis, chronic myelogenous leukemia, myelodysplastic syndrome.
- Increased promyelocytes: leukemoid reactions, chronic myelogenous leukemia, promyelocytic leukemia.
- Increased neutrophilic myelocytes: leukemoid reactions, chronic myelogenous leukemia, subleukemic myelosis.
- Increased neutrophilic metamyelocytes: leukemoid reactions, chronic myelogenous leukemia, subleukemic myelosis.
- Increased stab neutrophils: leukemoid reactions, chronic myelogenous

leukemia, subleukemic myelosis syndrome of "lazy" white blood cells.

- Elevated segmented neutrophils: leukemoid reactions, chronic myelogenous leukemia, subleukemic myelosis, lazy white blood cell syndrome.
- Eosinophils are elevated: allergic reactions, helminthiasis, malignant tumors, acute leukemia, chronic myelogenous leukemia, lymphofanulomatosis.
- Elevated basophils: basophilic leukemia, chronic myelogenous leukemia, erythremia.
- Elevated lymphocytes: chronic lymphocytic leukemia, aplastic anemia.
- Elevated monocytes: chronic myelogenous leukemia, leukemia, monocytic leukemia, tuberculosis, sepsis.
- Increased plasma cells by more than 20%: myeloma.
- Increased plasma cells to 20%: myeloma, aplastic anemia, infections, immune agranulocytosis.
- Increased erythroblasts: hemolytic, posthemorrhagic, folic acid deficiency and B12-deficient anemia (lack of folic acid and vitamin B12), acute erythromyelosis.

**An decreased count of WBCs is observed in the next conditions:**

- Decreased number of megakaryocytes in a bone marrow puncture sample: hypoplastic and aplastic immune and autoimmune processes, radiation and cytostatic cytopenia.
- Decreased white blood cell / red blood cell ratio: hemolysis, blood loss, erythremia, acute erythromyelosis.
- Decreased erythroblast maturation index: B12-deficient anemia, "ineffective" erythropoiesis in hemolysis, and blood loss.
- Decreased promyelocytes: aplastic anemia, as a result of the action of cytostatics, ionizing radiation, immune agranulocytosis.
- Decreased neutrophilic myelocytes: aplastic anemia, as a result of the action of cytostatics, ionizing radiation, immune agranulocytosis.

- Decreased neutrophilic metamyelocytes: aplastic anemia, as a result of the action of cytostatics, ionizing radiation, immune agranulocytosis.
- Decreased stab neutrophils: aplastic anemia, as a result of the action of cytostatics, ionizing radiation, immune agranulocytosis.
- Decreased segmented neutrophils: aplastic anemia, as a result of the action of cytostatics, ionizing radiation, immune agranulocytosis.
- Decreased erythroblasts: aplastic anemia, as a result of the action of cytostatics, ionizing radiation, partial red blood cell aplasia.

### Myocardial necrosis markers test (troponin I)

Test date \_\_\_\_\_

Patient's name \_\_\_\_\_

Parameter	Result	Reference values
Troponin I	19,0	до 0,5 нг / мл

**Answer.** An increase in plasma levels of troponin I indicates the presence of myocardial necrosis, regardless of the mechanism and cause of its occurrence. The degree of its increase is high. In the presence of coronary changes on the ECG, clinical manifestations of coronary heart disease, this is a sign of the development of acute myocardial infarction (normalization of the level of troponin I is observed after 10-12 days).

### Biochemical blood test

Test date \_\_\_\_\_

Patient's name \_\_\_\_\_

Parameter	Result	Reference values
Total protein	48 g / l	66-87 g / l
Albumin	32 g / l	36-50 g / l
Creatinine	200 $\mu$ mol / l	F: 44-80 $\mu$ mol / l; M: 62-106 $\mu$ / l
Cholesterol	7,8 mmol / l	< 5,2 mmol / l
Potassium	4,9 mmol / l	3,5-5,3 mmol / l
Sodium	135 mmol / l	135-148 mmol / l
Calcium	2,2 mmol / l	2,2-2,75 mmol / l

Phosphate	1,23 mmol / l	0,81-1,55 mmol / l
Glomerular filtration rate (GFR)	55 ml / min / 1,73 m <sup>2</sup>	≥ 90 ml / min / 1,73 m <sup>2</sup>

**Answer.** The given changes in biochemical parameters are characteristic for chronic kidney disease: increased creatinine to 200 μmol / L and decreased in glomerular filtration rate (55 ml / min / 1.73 m<sup>2</sup>). A decrease in the level of total protein and albumin with an increase in cholesterol is a sign of the presence of nephrotic syndrome characteristic of chronic glomerulonephritis, possibly amyloidosis, Kimmelstil-Wilson syndrome.

### Biochemical blood test (lipid profile)

Test date \_\_\_\_\_  
 Patient's name \_\_\_\_\_, male.

	Biochemical parameters	Results
1.	Total cholesterol	5,3 mmol / l
2.	Triglycerides	1,09 mmol / l
3.	HDL Cholesterol	1,32 mmol / l
4.	LDL Cholesterol	3,48 mmol / l

**Answer.** The level of total cholesterol exceeds the target levels, both for the general population (<5.0 mmol / L) and for patients with high cardiovascular risk (<4.5 mmol / L) and very high cardiovascular risk (<4.0 mmol / l).

Increased LDL cholesterol for patients with moderate cardiovascular risk (<3 mmol / L) and for patients with high cardiovascular risk (<2.5 mmol / L) and very high cardiovascular risk (<1.8 mmol / l). Determining HDL cholesterol and triglycerides is important in determining cardiovascular risk.

Urine analysis № _____
" _____ " _____ 200 _____ г.
Name . _____ department _____
Volume _____ 120 ml_Color red . _____ Reaction_alkaline
Tranparency_cloudy_Specific gravity _____ 1020 _____
Protein _____ 2,4 g / l _____ Glucose _____ negative _____
Acetone _____ negaive _____ Bile pigments _____ negative _____
Admixtures: _____ negative _____



**Sediment microscopy**

Epithelium: squamous \_\_\_\_\_ negative \_\_\_\_\_  
 transitional \_\_\_\_\_ negative \_\_\_\_\_  
 renal \_\_\_\_\_ negative \_\_\_\_\_  
 WBCs \_\_\_\_ 2-3 in the FoV \_\_\_\_\_  
 RBCs: not changed \_\_\_\_ negative \_\_\_\_\_  
 changed \_\_\_\_\_ 20-30 in the FoV \_\_\_\_\_  
 Casts: hyaline \_\_\_\_ 2-4 in the FoV \_\_\_\_\_  
 granular \_\_\_\_\_ negative \_\_\_\_\_  
 waxy \_\_\_\_\_ 1-2 in the FoV \_\_\_\_\_  
 leukocyte \_\_\_\_ negative \_\_\_\_\_  
 Mucus \_\_\_\_\_ negative \_\_\_\_\_  
 Salts \_\_\_\_\_ negative \_\_\_\_\_  
 Bacteria \_\_\_\_\_ negative \_\_\_\_\_

**Answer.** Proteinuria, erythrocyturia, cylindruria (in the absence of leukocyturia) are determined. Kidney damage is indicated by the presence of cylindruria in combination with significant proteinuria, which is a glomerular one. Significant proteinuria can occur in acute and chronic glomerulonephritis, renal amyloidosis. Changes in the presented urinalysis are more characteristic for glomerulonephritis, since erythrocyturia is detected.

**ECHOCARDIOGRAPHIC EXAMINATION**

Name \_\_\_\_\_ male \_\_\_\_\_ Age \_\_\_\_\_ 40 y/o

	gradient pressure	area	regurgitation	other
Mitral valve			II degree	No special
Aortic valve	2,4 mm Hg		absent	No special
Tricuspid valve			I degree	No special
Pulmonary valve			absent	No special

Aortic root (N <3.5 cm) \_\_\_\_ 3.5 cm \_\_\_\_ Left atrium (N <4 cm) \_\_\_\_ 4.8 cm \_\_\_\_

Left ventricle: EDV (N f: 56-104 ml, m: 67-155 ml) \_\_\_\_\_ 215 ml \_\_\_\_\_

ESV (N f: 19-49 ml, m: 22-58 ml) \_\_\_\_\_ 147 ml \_\_\_\_\_

EF \_\_\_\_ 31.6% \_\_\_\_

Right atrium (N <2.5 cm) \_\_ 3.5 cm \_\_ Right ventricle (N <2.5 cm) \_\_ 3.1 cm

Systolic pressure in the pulmonary artery \_\_\_\_ 35 \_\_\_\_ mmHg; \_\_\_\_\_

degree

The thickness of the walls and the region's contractile capacity

IBP \_\_\_\_0.8\_\_\_\_ cm \_\_\_\_\_

Posterior \_\_\_\_0.8\_\_\_\_ cm \_\_\_\_\_

Lateral \_\_\_\_\_ cm \_\_\_\_\_ diffuse hypokinesis

Front \_\_\_\_\_ cm \_\_\_\_\_

Top \_\_\_\_\_ cm \_\_\_\_\_

Aneurysm \_\_\_\_\_

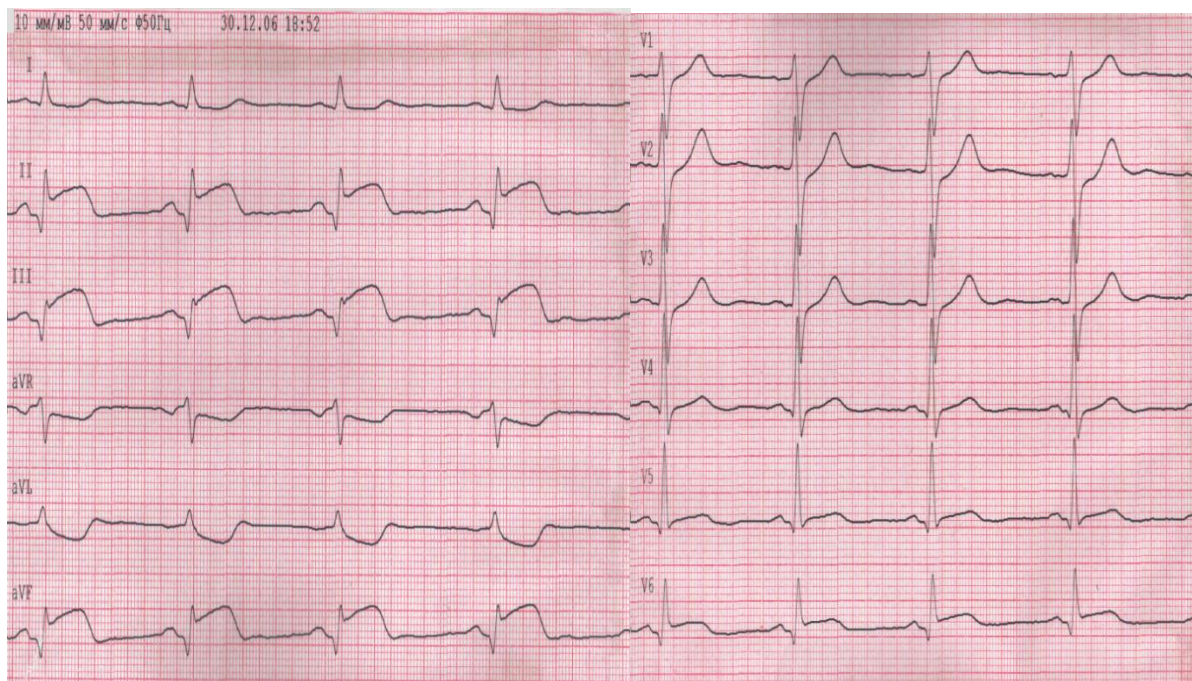
Other: separation of the pericardium leaves to 0.4 cm in diastole.

**Answer.** Significant dilatation of the cavity of the left atrium, left ventricle, right ventricle. Significant diffuse decrease in the contractile capacity of the left ventricle. Relative failure of the mitral valve of the II degree. Pulmonary hypertension of the 1st degree. These changes are possible in dilated cardiomyopathy, acute diffuse myocarditis.

## Electrocardiography

Date \_\_\_\_\_

Patient's data: male, 70 y/o. Blood pressure 110/ 60 mm Hg



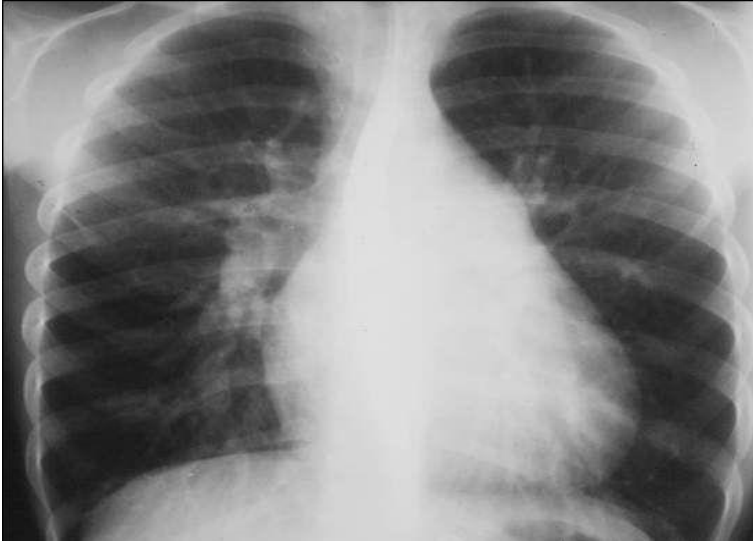
### Example of answer

PQ	0,12
QRS	0,11
QT	0,37

RR	0,90
HR	66

Sinus rhythm. A pathological Q wave in II, III, avF leads, and elevation of the ST II, III, avF segment are registered, which is a sign of Q-myocardial infarction of the inferior wall of the left ventricle.

## Chest X-ray



**Answer.** Pulmonary fields are of normal airiness. Pulmonary vascular pattern is increased. The sinuses are free. The shadow of the heart is located normally. Signs of an enlarged left atrium and left ventricle. Swelling the pulmonary artery trunk, dilation of the right heart.

**Examples of situational tasks and rationales for their solving**  
**Task 1.**

Patient K., 63 years old, complains of pain in the epigastric region, which occurs 1-1.5 hours after eating, on an empty stomach at night or in the morning, heartburn, sour belching, constipation. Considers himself ill for two years, exacerbations - every spring and autumn. The nature of the work is associated with frequent business trips and violations of diet. Smokes, previously not sick. Father suffers from peptic ulcer.

Objective examination. Asthenic type physique, normal body weight. The tongue is coated with white plaque. Hemodynamic parameters are normal. Heart and lungs are without pathological abnormalities. The abdomen is soft, with epigastral pain during superficial palpation. Deep palpation reveals pain in the pyloroduodenal zone. Positive symptoms of Mendel, Openhovsky. The lower edge of the liver is at the level of the costal arch. Symptoms of Kehr, Ortner and phrenicus-symptom are negative.

Data from additional studies. General blood test: RBCs -  $3.3 \cdot 10^{12} / l$ ; hemoglobin - 92 g / l; WBCs -  $4.7 \cdot 10^9 / l$ ; eosinophils - 1%, stab neutrophils - 2%, segmented neutrophils - 68%, lymphocytes - 28%, monocytes - 1%; ESR - 3 mm / hour. Urinalysis - unchanged. Stool examination: helminth eggs - negative, occult blood - negative.

Biochemical blood analysis: glucose - 4.3 mmol / l, bilirubin - 15.69  $\mu\text{mol} / l$ , total protein - 77 g / l, amylase - 25 g / (l · h), creatinine - 67  $\mu\text{mol} / l$ .

Endoscopy: the esophagus is normal. In the stomach there is a significant amount of fluid, the gastric mucosa is hyperemic, the folds are hypertrophied, the duodenal bulb is deformed, on the front wall there is a mucosal defect with a diameter of 0.5 cm. The pH of the gastric content is 1.3.

**Questions:**

1. Determine the patient's aggression factors of the duodenal mucous

membrane:

- A. Smoking, hypersecretion, heredity, malnutrition.
- B. Hypersecretion.
- C. Heredity.
- D. malnutrition.
- E. Stressful situations.

2. Determine the severity of the disease and the nature of the ulcer:

- A. Mild course, acute ulcer.
- B. Moderate course, chronic ulcer.
- C. Severe course, chronic ulcer.
- D. Mild course, chronic ulcer.
- E. Moderate course, acute ulcer.

3. Which drug is a proton pump inhibitor?

- A. Osid.
- B. Quamatel.
- C. Ranisan.
- D. Gastrozepin.
- E. Solcoseryl.

4. When, how and what mineral water do you recommend this patient to

take:

- A. In the phase of remission, water of the Mirgorodskaya type, heat, before meals.
- B. In the remission phase, the water is like Polyana Kvasova, cold, after eating.
- C. In the phase of remission, water of the Polyana Kvasova type, heat, without gas, 1.5 hours before meals.
- D. In the acute phase, water of the Mirgorodskaya type, without gas, 15 minutes before meals.
- E. In the remission phase, the water is like Polyana Kvasova, cold, without

gas, 15 minutes before meals.

5. Which of the following drugs is contraindicated in peptic ulcer disease?

A. Omez.

B. Misoprostol.

C. Eglonil.

D. Naproxen.

E. Peritol.

**Answer for Task 1.**

**Correct answers: 1B, 2B, 3A, 4C, 5D.**

**Rationale for the answers:**

1. Heredity and concomitant pathology are not the factors of aggression; they can determine a predisposition to this pathology. Hypersecretion of hydrochloric acid is one of the important factors of aggression. Nicotine is a strong stimulant of secretion.

2. The chronic course of an ulcer is indicated by the recurrent course of the disease and the presence of deformation of the duodenal bulb. The moderate course is determined by the frequency of relapses (twice a year).

3. Oside - the brand name of the drug omeprazole, which is an inhibitor of the proton pump.

4. Mineral water in case of peptic ulcer with hypersecretion is prescribed warm, without gas 1.5 hours before meals, low mineralization, bicarbonate. The components of water during meals are in the intestines and exhibit an inhibitory effect on gastric secretion.

5. Naproxen is a drug from the group of non-steroidal anti-inflammatory drugs, which, according to their side-effect mechanism, are ulcerogenic.

Clinical diagnosis: peptic ulcer, exacerbation phase, moderate severity, active duodenal ulcer, bulb deformity, chronic gastritis with increased secretory and acid-forming function without impaired gastrointestinal motor function.

The diagnosis of peptic ulcer was established on the basis of the patient's

complaints of hunger and night pain, the presence of dyspeptic syndrome, the symptoms of which indicate hypersecretion, seasonal exacerbations and the nature of endoscopic changes (the presence of an active ulcer in the duodenal bulb). The course of moderate severity is justified by the frequency of relapses twice a year.

Hypertrophy and hyperemia of the folds of the stomach during endoscopy confirm the diagnosis of gastritis type B. However, for a final confirmation of the gastritis diagnosis, a gastric mucosa biopsy is necessary.

The pH-test of the stomach (1.3) indicates an increased secretory and acid-producing function of the stomach. The absence of duodenogastric or gastroesophageal reflux indicates normal gastric motility.

Peptic ulcer - a chronic disease of the stomach or duodenum, characterized by the occurrence of a peptic ulcer in the mucous membrane, proceeds cyclically and is prone to progression.

### **Task 2.**

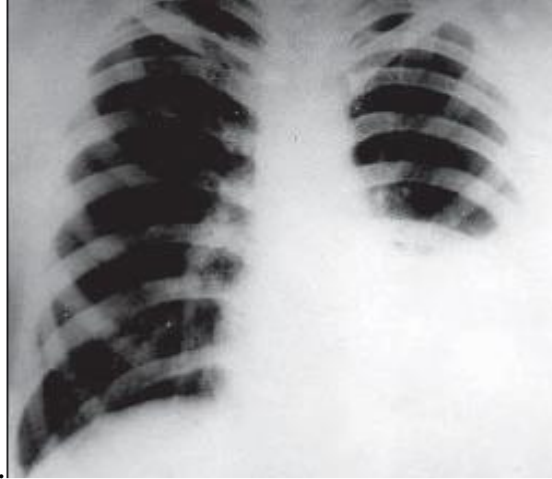
Patient P., 64 years old, complaints of fever up to 40 ° C, dry cough, stitching pain in the left half of the chest, which intensifies with breathing and coughing, general weakness. Sick for 3 days.

Objective data. The state of moderate severity. The position in bed is forced, on the left side. BH - 24 per minute, there is a lag in the left half of the chest during breathing. The skin is cyanotic. On palpation - weakening of voice trembling on the left, with percussion - there is also a shortening of percussion sound from the middle of the scapula down. Auscultatory - there is no breathing on the left in the lower parts, over other parts of the lungs - vesicular. In the study of the cardiovascular system and digestive organs, pathology was not detected.

Data from additional studies. Complete blood count: erythrocytes -  $3.9 \cdot 10^{12}$  / L, hemoglobin - 128 g / L, CI - 0.9, white blood cells -  $11.4 \cdot 10^9$  / L, eosinophils - 4%, stab neutrophils - 9%, segmented neutrophils - 41%, lymphocytes - 37%, monocytes - 8%, ESR - 33 mm / hour. Urinalysis: traces of protein, white blood cells - 4-7 in the field of view. A chest x-ray is attached.



Analysis of pleural fluid: protein 8.4 g / l; white blood cells 25-32 in the field of



view, specific gravity 1,022.

Patient's X-ray

**Questions:**

1. Interpret radiological changes:
  - A. Infiltration of lung tissue.
  - B. The presence of fluid in the left pleural cavity.
  - C. Neoplasm on the left in the lower sections.
  - D. Pleural stratifications.
  - E. Focal pneumosclerosis.
2. What is the most likely diagnosis in a patient:
  - A. Exudative pleurisy.
  - B. Left-sided lower lobe pneumonia complicated by pleurisy.
  - C. Peripheral cancer of the left lung. Carcinomatous pleurisy.
  - D. Exudative pleurisy of tuberculosis origin.
  - E. Heart attack pneumonia.
3. What examinations are needed to establish a definitive diagnosis?
  - A. Sputum culture.
  - B. Tomography.
  - C. Pleural puncture followed by exudate examination.
  - D. Bronchography.

- E. Computed tomography.
4. Describe the result of the pleural fluid examination.
    - A. Stagnant effusion.
    - B. Tuberculous effusion.
    - C. Exudative effusion.
    - D. Carcinomatous effusion.
    - E. Post-traumatic effusion.
  5. Determine the rational combination of drugs to treat the patient:
    - A. Antibacterial therapy, physiotherapy.
    - B. Pleural puncture, antibiotic therapy.
    - C. Antibacterial and glucocorticosteroid therapy.
    - D. Anti-inflammatory and vitamin therapy.
    - E. Pleural puncture and physiotherapy.

**Answers for task 2.**

**Correct answers: 1B, 2A, 3C, 4C, 5B.**

**Rationale for the answers:**

1. An intense homogeneous dense shadow with an oblique line indicates the presence of fluid in the pleural cavity.

2. Considering intoxication-inflammatory syndrome (fever up to 40 ° C, leukocytosis -  $11.4 \cdot 10^9 / l$ , stab neutrophils - 9%, ESR - 33 mm / h), lung tissue syndrome (dry cough, stitching pain in the left half of the chest, which increases with respiratory movements, shortness of breath, cyanosis, lag of the left half of the chest during breathing, weakened voice trembling, shortening of percussion sound on the left, lack of breathing in the same place, the presence of fluid in the pleural cavity radiologically), in the patient - levostoro Nij pleural effusion.

3. Given the presence of fluid in the pleural cavity, intoxication-inflammatory syndrome, it is necessary to carry out pleural puncture for therapeutic and diagnostic purposes and examine the resulting fluid.

4. Exudate is characterized by a large amount of protein (more than 3 g / l),

inflammatory changes are evidenced by leukocytosis (25-30 in the field of view) and specific gravity (1,022).

5. In the presence of a large amount of fluid (up to 3-4 ribs), one must carry out pleural puncture, prescribe antibiotics of a wide spectrum of action. In addition, it will be rational to prescribe a patient desensitizing agents, vitamin therapy and physiotherapeutic treatment (only in the absence of fluid in the pleural cavity) to prevent the formation of a mooring line.

Clinical diagnosis: left-sided exudative pleurisy, RF I.

Exudative pleurisy is an acute inflammatory bacterial lesion of the pleural sheets with exudation into the pleural cavity, characterized by intoxication-inflammatory and pain syndromes, lung lesion syndrome (shortening of the pulmonary sound, lack of breathing, the presence of intense dimming with a clear upper level during X-ray examination) and restrictive respiratory failure.

### **Task 3.**

Patient N, 70 years old. Complains of general weakness, fatigue, pain in the tongue, epigastrium, right hypochondrium, nausea, diarrhea, which alternate with constipation, a feeling of numbness of the lower extremities.

Objective data. The skin and visible mucous membranes are clean, lemon yellow in color. Atrophy of the papillae is observed in the tongue. Temperature - 37.2 ° C. Pulse - 90 in 1 min, rhythmic, of satisfactory filling and tension. Blood pressure - 110 and 70 mm Hg. Cardiac activity is rhythmic. Ist heart tone is weakened, above the apex there is a systolic murmur. Lungs - vesicular breathing. The abdomen is soft, with palpation slightly painful at the point of Kehr's. The lower edge of the liver is palpated 1.5 cm below the edge of the right costal arch. At the edge of the left costal arch, the lower pole of the spleen is palpated. No swelling.

Data from additional studies. Complete blood count: erythrocytes - 1.22 · 10<sup>12</sup> / L, basophilic puncture phenomena, Cabot and Joli bodies, macrocytosis (Fig. 1), anisocytosis were revealed; hemoglobin - 52 g / l, CI - 1.3, white blood

cells -  $4.0 \cdot 10^9 / l$ , stab neutrophils - 7%, segmented neutrophils - 63%, lymphocytes - 24%, eosinophils - 2%, basophils - 2%, monocytes - 2%, polysegmentation of neutrophils is noted (Fig. 2), platelets -  $60 \cdot 10^9 / l$ , ESR - 9 mm / hour. In the myelogram, the cells shown in Fig. 3.

Fecal analysis: diphylobitriosis diagnosed.

Biochemical blood test: total bilirubin -  $40 \mu\text{mol} / l$ ; direct -  $13 \mu\text{mol} / l$ ; indirect -  $27 \mu\text{mol} / l$ .

Upper endoscopy - atrophy of the gastric mucosa, microscopic examination of the mucous membrane (express biopsy) revealed lymphocytic infiltration of the mucous membrane.

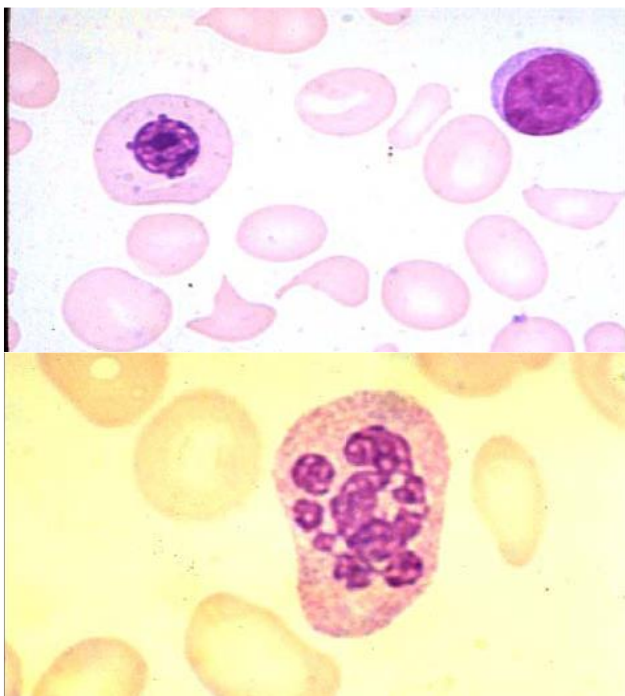


Fig. 1. Patient B's blood. Fig. 2. Patient B's blood.

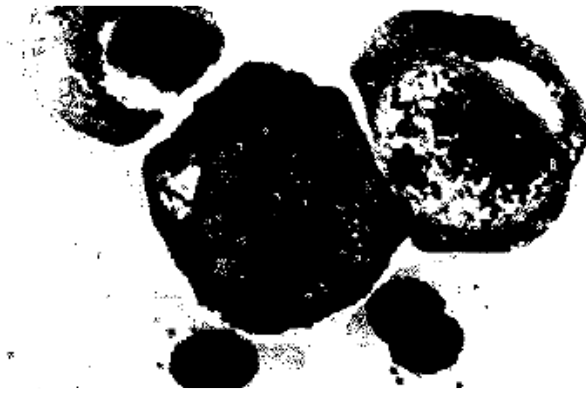


Fig. 3. Patient B's myelogram

Questions:

1. What are the names of large cells with blue cytoplasm found in the myelogram (Fig. 3)?

- A. Blasts.
- B. Megakaryocytes.
- C. Megaloblasts.
- D. Mononuclear cells.
- E. Prolymphocytes.

2. What type of hematopoiesis is observed in the patient?

- A. Megaloblastic.
- B. Normoblastic.
- C. Erythroblast.
- D. Megacaryoblastic.
- E. Erythromyeloblastic.

3. What syndrome treatment should be started first?

- A. Thrombocytopenic.
- B. Anemic.
- C. Diphylobotrious.
- D. Hepatolienal.
- E. Syndrome of the defeat of the cardiovascular system.

4. Consultations of which specialists are necessary for additional

examination of the patient:

- A. Neurologist.
- B. Infectionist.
- C. Cardiologist.
- D. Neurologist, infectious disease specialist, cardiologist.
- E. Patient does not require consultation.

5. What is the patient's diagnosis?

- A. At 12, deficiency anemia.
- B. Iron deficiency anemia.
- C. Folic acid deficiency anemia.
- D. Congenital hemolytic anemia.
- E. Acquired hemolytic anemia.

**Task 3 answers.**

**Correct answers: 1D, 2C, 3B, 4C, 5A.**

**Rationale for the answers:**

1. Large cells with blue cytoplasm, found in sternal punctate (Fig. 3) - megaloblasts.

2. The revealed megaloblasts in the myelogram indicate the presence in the patient of a megaloblastic type of hematopoiesis, which is actually a morphological substrate of B12 deficient anemia.

3. The patient must be prescribed treatment for anemic syndrome.

4. In connection with the presence of symptoms of diphyllbothriasis, signs of damage to the cardiovascular system and possible damage to the nervous system, consult a neurologist, helminthologist, cardiologist.

5. The diagnosis of B12-deficient anemia was established on the basis of the detection of megaloblastic type of hematopoiesis in the analysis of bone marrow aspiration biopsy.

**Clinical diagnosis:** Diphyllbotriosis. At 12, severe deficiency anemia. Anemia caused by a deficiency of vitamin B12, regardless of the causes of this

deficiency, is characterized by the appearance of megaloblasts in the bone marrow, intracerebral destruction of erythrocytes, hyperchromic macrocytic anemia, thrombocytopenia and neutropenia, atrophic changes in the mucous membrane of the gastrointestinal tract and funicular myelosis of the nervous system.

**Task 4.**

Patient M., 60 years old. Complains of general weakness, headache, dizziness, decreased visual acuity, lower back pain.

Objective data. The condition is satisfactory. The skin is pale. The thyroid gland is not enlarged. Pulse - 88 in 1 min, rhythmic. Blood pressure- 130 / 85 mm Hg. The left border of the heart is expanded by 2 cm, the activity of the heart is rhythmic, Ist tone is weakened. In the lungs - vesicular breathing. The abdomen is soft, painless. Palpation of the abdomen revealed no pathological changes.

Data from additional studies. Complete blood count: erythrocytes -  $2.4 \cdot 10^{12} / l$ , hemoglobin - 80 g / l, CI - 1.0, leukocytes -  $6.2 \cdot 10^9 / l$ , stab neutrophils - 2%, segmented neutrophils - 60%, eosinophils - 2%, basophils - 1%, lymphocytes - 28%, monocytes - 7%, platelets -  $80.0 \cdot 10^9 / l$ , ESR - 72 mm / hour.

Urinalysis: clear, the reaction is slightly acidic, relative density 1.019, protein 1.2 g / l, red blood cells 3-4, white blood cells 0-5 in the field of view.

Biochemical analysis of blood: urea - 6.4 mmol / l, total protein - 106 g / l, calcium - 4.2 mmol / l, sodium - 134 mmol / l. During protein electrophoresis, an M-paraprotein gradient was detected.

X-ray of the bones of the skull is



given.

Questions:

1. What changes were detected during x-ray of the skull?
  - A. Pathological changes were not detected.
  - B. Enhanced vascular pattern.
  - C. Osteodestructive changes.
  - D. Osteolytic changes.
  - E. Deformation of the Turkish saddle.
2. What is the most likely diagnosis in a patient?
  - A. Chronic lymphocytic leukemia.
  - B. Chronic myeloid leukemia.
  - C. Plasmacytoma.
  - D. Lymphogranulomatosis.
  - E. Acute leukemia.
3. Indicate which of the indicated signs allow you to verify your preliminary diagnosis?
  - A. Morphological confirmation of the tumor process and biochemical identification of the product of the synthesis of tumor cells.
  - B. Antibody deficiency syndrome, hemorrhagic diathesis, high viscosity syndrome, peripheral sensory neuropathy.
  - C. Hypercalcemia, antibody deficiency syndrome, hemorrhagic diathesis, high viscosity syndrome.
  - D. Hypercalcemia, antibody deficiency syndrome, hemorrhagic diathesis, increased viscosity syndrome, peripheral sensory neuropathy.
  - E. Visceral lesions, hypercalcemia, antibody deficiency syndrome, hemorrhagic diathesis, high viscosity syndrome, peripheral sensory neuropathy.
4. What stage of the disease is defined in this case?
  - A. Initial.
  - B. Deployed.
  - C. Stage I.
  - D. Stage II.



E. Stage III.

5. Which of the following treatment protocols should be chosen.

A. Sarcolysin - 10 mg per day (250-350 mg per course), prednisone - 10-15 mg per day, retabolil - 50 mg intramuscularly 1 time per week.

B. Vincristine - 1 mg / m<sup>2</sup> once every 2 weeks, Sarkolizin - 10 mg per day (250-350 mg per course), prednisone - 10-15 mg per day, retabolil - 50 mg intramuscularly 1 time per week.

C. Cyclophosphamide - 400 mg per day (course dose - 8-10 g), prednisone - 10-15 mg per day, retabolil - 50 mg intramuscularly 1 time per week.

D. Vincristine 1 mg / m<sup>2</sup> once every 2 weeks, cyclophosphamide - 400 mg per day (course dose - 8-10 g), prednisone - 10-15 mg per day, retabolil - 50 mg intramuscularly 1 time per week.

E. Melphalan - 10 mg per day (250-350 mg per course), prednisone - 10-15 mg per day, retabolil - 50 mg intramuscularly 1 time per week.

#### **Task 4 answers**

**Correct answers: 1D, 2C, 3A, 4E, 5C.**

#### **Rationale for the answers:**

1. On the x-ray osteolytic changes are detected.

2. Based on the detection of changes in flat bones (osteolysis), hyperproteinemia - 106 g / l (normal - 65-85 g / l), changes in the electrolyte balance: calcium - 4.2 mmol / l, as well as the presence of paraproteins during electrophoresis proteins (M-paraprotein gradient), proteinuria (protein - 1.2 g / l), one might think that the patient has a plasmacytoma.

3. The criteria for the diagnosis of myeloma is morphological confirmation of the tumor process (in the myelogram - an increase in the number of plasma cells) and biochemical identification of the product of the synthesis of tumor cells (M-gradient).

4. The stage of the disease is established by the classification of Salmon-Duri. The presence of hemoglobin in the patient below 85 g / l, hypercalcemia (4.2

mmol / l), and bone osteodestruction suggest that the patient has stage III disease.

5. The treatment protocol (cyclophosphamide - 400 mg per day (course dose - 8-10 g), prednisone - 10-15 mg per day, retabolil - 50 mg intramuscularly 1 time per week) can be selected based on the fact that according to the classification Salmon-Durie diagnosed stage III myeloma.

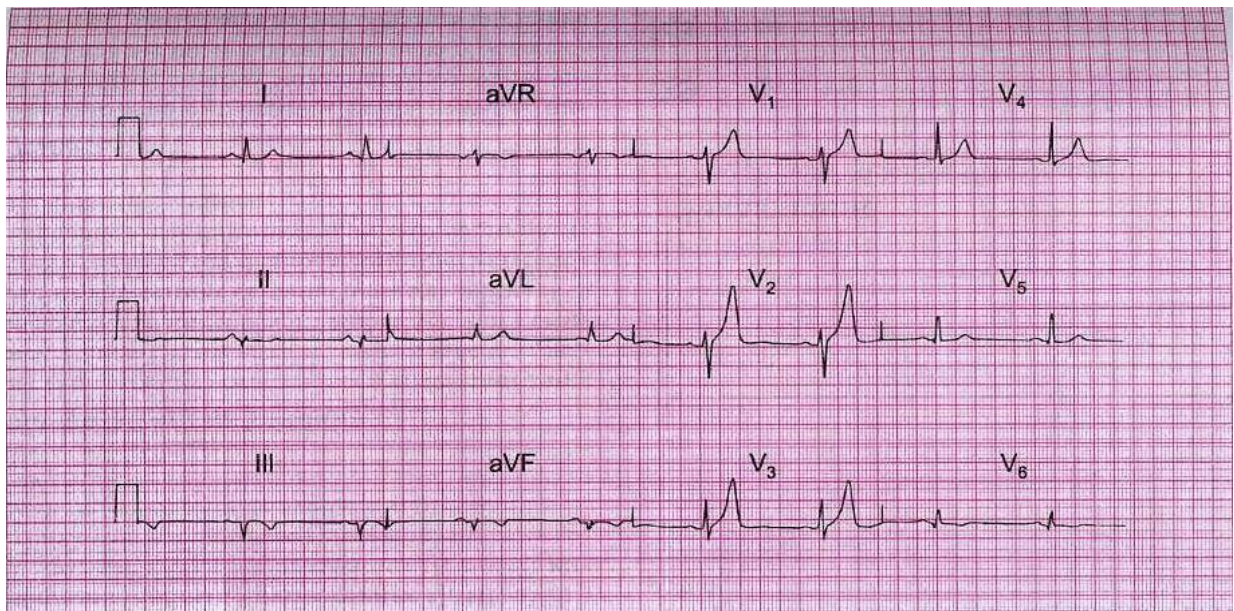
**Clinical diagnosis:** myeloma, diffuse-focal form, stage III.

Myeloma (plasmacytoma) belongs to the group of B-lymphoproliferative diseases - tumor processes in the system of plasma cells.

Solitary (focal) plasmacytomas (bone and extraosseous), generalized plasmacytomas, multiple tumor forms, diffuse-nodular forms, diffuse forms are distinguished. A mandatory sign of a plasma cell tumor is the identification of its synthesis product - PIg - in the blood serum or in the urine of patients, with the so-called non-secretive myelomas, PIg is determined in the cells.

#### **Task 5.**

A 60-year-old man went to the doctor with complaints of severe pain behind the sternum during exercise, which lasts 1-2 minutes. At rest, there was no pain behind the sternum.



Questions:

1. Perform an ECG analysis.

2. Identify pathological changes on the ECG.
3. Determine the preliminary diagnosis.
4. What studies need to be done to confirm the clinical diagnosis.
5. Define treatment tactics.

**Task 5 answers:**

1. ECG - sinus rhythm, heart rate - 100 per min., The electrical axis of the heart is normal, minor Q waves in leads II, III, aVF, T wave inversion in leads III and aVF. High pointed T waves in the lead V 1 - V 3.

2. Clinical rationale for ECG changes: Q waves in the “lower” leads in combination with T wave inversion indicate transferred lower (posterior diaphragmatic) myocardial infarction. It is likely that the patient suffered a previous myocardial infarction and the pain behind the sternum may be due to myocardial ischemia. It is necessary to pay attention to the presence of risk factors (smoking, increased blood pressure, hypercholesterolemia).

3. Coronary heart disease. Postinfarction cardiosclerosis (transferred posterior diaphragmatic myocardial infarction). Angina pectoris. CH 0.

4. A cardiac stress test is indicated, depending on its results, decide on the performance of coronary angiography.

5. It is advisable to recommend to the patient prolonged use of aspirin and statins.