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Кафедра поликлинической терапии и общей врачебной практики
с курсом дерматовенерологии

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ПОЛИКЛИНИЧЕСКАЯ ТЕРАПИЯ

Учебно-методическое пособие
для студентов 5 курса факультета по подготовке специалистов
для зарубежных стран медицинских вузов

POLYCLINIC THERAPY

Teaching workbook
for the 5th year students of the Faculty on preparation of experts
for foreign countries of medical higher educational institutions

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В учебно-методическом пособии представлены современные аспекты этиологии, патогенеза, классификаций, клинических проявлений, диагностики и лечения заболеваний внутренних органов в практической деятельности участкового врача-терапевта. Предназначено для студентов 5 курса факультета по подготовке специалистов для зарубежных стран медицинских вузов.

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CONTENT

TOPIC 1. Bronchial obstruction: differential diagnosis. Diagnosis and treatment of COPD and asthma in the outpatient setting, medical tactics, medico-social examination, clinical examination, primary prevention. Emergency care in asthma attack and the developing asthmatic status GP-therapist.

The concept of the bronchial obstruction syndrome, major diseases, accompanied by the syndrome .Diagnostic algorithm search with Bronchial syndrome.

Bronchial syndrome (BOS) — a clinical syndrome caused by impaired patency of air bronchi due to narrowing or occlusion of the airway with a consequent increase in airway resistance to inhaled air flow. Biofeedback is one of pathophysiological disorders that can affect the outcomes and progress for many acute and chronic bronchopulmonary diseases. BOS, not being independent nosological unit can occur in various diseases of the heart and lungs, causing airway obstruction. The main clinical manifestations of BOS are paroxysmal cough, expiratory dyspnea and sudden asthma attacks. Clinical manifestations of BOS are divided into latent and flowing with severe clinical picture. Adrift BOS divided into acute (sudden arisen) and chronic (ongoing).Functional changes associated with reduced BOS main spirometric indices reflecting the degree of bronchial obstruction and the character of «air trapping», namely: forced expiratory volume in 1 second (FEV1); the ratio of FEV1 / FVC < 70 % (forced vital capacity (FVC)).

These indicators are the diagnostic criteria of bronchial obstruction and are used to determine the severity of BOS.

According to the severity of clinical and functional manifestations of BOS divided into mild, moderate and severe.

The main clinical manifestations of BOS are shortness of breath, dyspnea (applies to life-threatening situations), paroxysmal cough, wheezing, noisy breathing. Symptoms are more noticeable during exertion. Other manifestations of BOS — excessive sweating, sleep disturbances, headache, confusion, convulsions — are found in severe symptom complex.

Variant forms of BS:

Spastic — the most popular variant of BOS (> 70 % of all cases), which lies in the development of bronchospasm due to dysfunction in the control systems of bronchial tone.

Inflammation — the mechanism is due to edema, infiltration of the airways, hyperemia membrane of the bronchi.

Dyscrinic — occurs when excessive stimulation of enzymes goblet cells and glands layer of the bronchi leading to the deterioration of sputum, disruption of the mucus and mucociliary transport.

Dyskinetic — bronchial patency compromised due to congenital underdevelopment membranous portion of the trachea and bronchi to facilitate the closure of the lumen during inspiration.

Emphysematous — accompanied collapse of the small airways due to the reduction and loss of elasticity of the lungs.

Hemodynamic — occurs secondary to disorders of the pulmonary hemodynamics: hypertension pre- and postcapillaries, stagnation in the bronchial veins and hypertensive crisis in the pulmonary circulation.

Hyperosmolar — observed with decreasing water content of the mucous membranes of the bronchi (breathing in the cold air), when the high osmotic concentration of cell-surface receptors causes irritation and bronchospasm.

The basis of bronchial obstruction are reversible (functional) and irreversible (organic) changes.

• reversible (functional): bronchospasm, inflammatory infiltration, edema, mucociliary insufficiency, viscous mucus hypersecretion;

• irreversible congenital stenosis of the bronchi, their obliteration and sclerotic changes.

Bronchial obstruction often is an infectious-allergic in nature. Some of the viruses most frequently causing bronchial obstruction, include respiratory syncytial virus, parainfluenza, at least — flu viruses and adenovirus; greater role for intracellular pathogens (Chlamydia and Mycoplasma infections) .Reported an association of bronchial obstruction with certain types of pathogenic organisms released from sputum or bronchial secretions, such as Moraxella catarrhalis, fungi Candida.

Of particular importance among environmental factors, which can lead to the development of an obstructive syndrome (especially in children first three years of life), is given:

• passive smoking in the family (tobacco smoke triggers hypertrophy of bronchial mucous glands, violation of mucociliary clearance, slow promotion mucus destruction of bronchial epithelium);

• pollution of the atmosphere by industrial gases, organic and inorganic dust. The following groups of diseases associated with BOS syndrome;

• respiratory diseases: bronchitis, bronchiolitis, pneumonia, obstructive bronchitis, bronchial asthma, bronchopulmonary dysplasia, malformations of respiratory system, tumors of the trachea and bronchi; foreign body trachea, bronchus, esophagus;

• disease aspiration genesis (or aspiration obstructive bronchitis): gastroesophageal reflux, tracheoesophageal fistula, malformation of the gastrointestinal tract, diaphragmatic hernia;

• diseases of the cardiovascular system of innate and adaptive nature of congenital heart disease with hypertension, pulmonary circulation, vascular anomalies, congenital Non-rheumatic carditis, and others.);

• diseases of the central and peripheral nervous system: birth trauma, myopathy, etc.;

• hereditary anomaly exchange: cystic fibrosis, deficit Alpha-1 trypsin, mucopolysaccharidoses;

• congenital and acquired immunodeficiency states;

• rare hereditary diseases;

• other conditions: injuries and burns, poisoning, exposure to various physical and chemical factors of the environment; compression of the trachea and bronchi of extrapulmonary origin (tumor, Hodgkin's disease).

Conventional studies at Bronchial syndrome:

• Sputum culture tests.

If the patient cough with sputum should be sent to the laboratory for testing and culture, including Mycobacterium tuberculosis:

• Peak expiratory flow volume (FEV 1 ").

Determination of the maximum flow rate at the beginning of forced expiratory volume in the patient directly to the Chamber assists in the diagnosis of asthma (low peak expiratory flow volume):

• leukocytosis in the blood count.

Leukocytosis is an indirect indicator of inflammation, it can also be detected in patients with pulmonary embolism:

• Rg-scopy chest.

You can use it to identify the seal in the lung inflammatory nature. Bronchiectasis in areas of prolonged inflammation indicates the presence of the patient bronchiectasis. Seal in the apex with areas of calcification and enlarged lymph nodes root of the lung — a characteristic feature of pulmonary tuberculosis. For radiographic pulmonary edema characteristic of heterogeneity, darkening of both lungs .In this case, can be detected radiological signs of heart failure, including cardiomegaly, pulmonary veins verhnedolevyh deviation, bilateral exudative pleurisy and Skittles line (thin horizontal lines 1–2 cm., Located in the peripheral regions of the lungs). Lung cancer appears as a tumor formation in the root, sealing in the peripheral regions of the lungs or atelectasis site and its seal due to violations of bronchial obstruction.

• Functional respiratory tests.

Used to diagnose bronchial obstruction (asthma, chronic bronchitis, bronchiectasis), and in monitoring the tidal volume loops can be detected fixed airway obstruction.

Special studies:

Ventilation-perfusion scan.

Diagnostic test for pulmonary embolism is performed in patients with suspected pulmonary embolism.

• Angiography of pulmonary vessels Studies performed in critically ill with suspected pulmonary embolism in deciding whether surgery or an thromboembolic therapy.

• Computed tomography (CT) of the chest.

Used to diagnose and refinement stage lung cancer, as well as recognition of bronchiectasis.

Bronchial asthma. Classification. Plan examination of a patient with asthma. Indications for hospitalization. General principles of treatment of bronchial asthma in the outpatient setting.

Clinical examination

In 2002, the International Covenant has been determined that asthma — a chronic inflammatory disease of the airways in which many cells play a role and cellular elements. Chronic inflammation causes a concomitant increase in hyperactive airways leading to recurrent episodes of wheezing, shortness of breath, feeling of tightness in the chest and coughing, particularly at night or early in the morning.

These episodes are usually associated with widespread but differ in the severity of airflow obstruction that is often reversible either spontaneously or under the influence of treatment.

Airway hyperreactivity and bronchial obstruction — the two main manifestations of ventilation disorders in asthma .An important component of asthma is an increased response of the bronchi in the exogenous and endogenous stimuli.

There are four components of bronchial obstruction, each of which is associated with inflammation of the bronchi:

1) acute obstruction — due to spasm of smooth muscle;

2) subacute — swelling of the mucous membranes of the respiratory tract;

3) chronic — bronchial obstruction, especially their terminal parts, viscous secretion;

4) irreversible — sclerotic process in the airway wall, occurs against a background of inadequate treatment.

Acute bronchoconstriction developed as a result of bronchial hyperreactivity to various stimuli (a consequence of airway inflammation). Acute bronchospasm quickly removed B2-agonists short action.

Swelling of the bronchial wall and leads to bronchial obstruction and can be combined or not combined with bronchospasm. Bronchodilators affect certain component of such obstruction, but are more effective in this situation, anti-inflammatory drugs.

Chronic bronchial obstruction viscous secretion more difficult to treat, require 1.5–2 months of treatment basic anti-inflammatory drugs (usually corticosteroids) to produce the effect.

When sclerosis bronchial wall bronchoobstruction little treat anti-inflammatory drugs. In this regard, the actual remains timely appointment of anti-inflammatory therapy.

Risk factors:There are risk factors that: are relevant to the development of asthma and cause worsening of the disease (triggers).

Risk Factors leading to the development of asthma:

I. Predisposing factors:

• Atopy.

• Heredity.

II. Causal factors:

• Pet allergens: house dust; allergens animals; cockroach allergen; mushrooms.

• External allergens: pollen; mushrooms; aspirin; occupational allergens.

III. Factors that contribute to asthma:

• Respiratory infections.

• Children's age.

• Food allergens.

• Air pollutants: external pollutants; internal pollutants.

• Smoking: passive smoking; active smoking.

IV. Factors aggravating for asthma (trigerr):

• Allergens.

• Respiratory infections.

• Exercise and hyperventilation.

• Weather conditions.

• Sulphur dioxide.

• Food, food additives, drugs.

The pathogenesis of asthma due to both reversible and irreversible pathological physiological mechanisms . By reversible include bronchospasm, inflammation, swelling and infiltration of the bronchial mucosa, mucus obstruction due to violation of cough. The most difficult runs obturation mucus. With the progression of bronchial lumen obstruction decreases, and increases the amount of mucus accumulating. This is based on the transformation of epithelial cells in the mucus-producing goblet cells. By irreversible mechanisms include a number of pathophysiological changes, which are based on morphological violations: stenosis, deformation and obliteration of the lumen of the bronchi, fibrosis wall expiratory collapse of small airways, emphysema. These changes result in irreversible tend to frequent exacerbations, progression of disease and disability of patients. In this regard it is important to diagnose AD at an earlier stage, in the step of reversible changes.

Classification of asthma

Classification of asthma severity:

Intermittent asthma — symptoms of at least 1 time per week, night — not more than 2 times per month, acute intermittent, FEV1 greater than or equal to 80 % of the variability of PEF less than 20 %.

Mild persistent asthma — symptoms more than 1 time a week night — more than 2 times per month, exacerbation violate physical activity and sleep, OFV1bolshe or equal to 80 % of the variability of PEF less than 30 %.

Moderate persistent asthma — symptoms daily, night — more than 1 time per week, exacerbation violate physical activity and sleep, OFV1 – 60–80 % variability in PEF-30 %, required daily intake of ß2-agonists short-acting; ***severe persistent asthma*** — symptoms daily, frequent nocturnal symptoms, exacerbations violate physical activity and sleep, FEV1 less than 60 % variability in PEF of more than 30 %, limit physical activity, daily intake of ß2-agonists short action.

Thus, for the first time rendered the diagnosis of asthma indicates the severity of the disease and prescribe appropriate severity level of health events that form the basis of basic therapy. Later in medical practice is recommended to use a classification based on an assessment of the degree of asthma control: a controlled, partly controlled and uncontrolled. This reflects the understanding that the severity of asthma depends not only on the severity of the disease itself, but also on the degree of susceptibility to the prescribed treatment, which is a specific feature of asthma in an individual patient and can vary within a month or a year.

Controlled asthma is characterized by the complete absence of all symptoms of the disease and a normal level of spirometry. In patients with long-lasting controlled asthma (not menee 3 months) could be considered to reduce the amount of basic therapy.

Partially controlled asthma is characterized by a limited number of symptoms. Therapeutic tactics in this level of control is ambiguous and depends on the choice of doctor: it is possible to either increase the amount of therapy in anticipation of better control of the disease, or stay on the same basic therapy.

Uncontrolled asthma: the presence of 3 or more symptoms partially controlled asthma and / or 1 episode of exacerbation during any week (according to the new GINA, blow-week — this week uncontrolled asthma). In turn, uncontrolled asthma during the week is considered as aggravation and require treatment by the rules of treatment of acute exacerbations of asthma.

The main symptoms of asthma — inflammation of the airways. The most common symptom is detected at physical examination, — wheezing on auscultation. At the heart of the pathophysiology of asthma is often episodic reversible airflow obstruction, which is characterized by airflow limitation on the exhale. Leading morphological feature — inflammation of structural changes to the stage of the remodeling of the bronchial passages.

Indications for hospitalization:

- severe asthma exacerbation;
- moderate severity in the acute phase;
- patients with newly diagnosed;
- status asthmaticus.

General principles of treatment of asthma in the outpatient setting

For the treatment of asthma medications are used basic therapy, which have control over the disease, as well as use of symptomatic drugs that relieve symptoms. Preparations for the so-called ***basic therapy***: These drugs do not

work with the already developed asthma attacks, their task is to "strategic" — to prevent the occurrence of attacks by suppressing specific inflammation in the bronchi, which are the cause of hyperactivity. This is a non-hormonal preparations of basic therapy, which include groups:

1) non-steroidal anti-inflammatory drugs: *Intal* (cromolyn sodium), *tayled* (nedocromil). These assets are shown as basic therapy with intermittent asthma and mild. Cromones less powerful in its effectiveness of inhaled corticosteroids. Since there are indications for the purpose of inhaled corticosteroids, even at mild asthma, cromones gradually replaced by more convenient to use inhaled glucocorticosteroids. Is not justified as the transition to cromones with inhaled corticosteroids and in full control of the symptoms of minimal doses of inhaled corticosteroids. Unlike Intal tayled has the following advantages:

1. A higher (approximately 10-fold) anti-inflammatory activity.

2. Nedocromil sodium are effective for the treatment of allergic and nonallergic asthma patients not only young, but also older age groups.

3. The product has a more rapid action. Its therapeutic effects as manifested within 5–7 days after the start of the application. 4. Compared with Intaglio tayled has a distinct steroid-saving activity. Taking the drug significantly reduces the need for patients to inhaled glucocorticoids.

2) Inhibitors of leukotriene receptor antagonists include leukotriene receptor cysteine and drugs that inhibit the synthesis of leukotrienes [RB patented in prep. singular]. Clinical use of drugs in this group — montelukast sodium, zafirlukast, pranlukast show a definite therapeutic efficacy. They prevent bronchoconstriction (including a night), inhibit the development of inflammation, edema, decrease vascular permeability reducing mucus secretion, improving sleep quality, reduced use of beta-agonists.

3) Hormonal (glucocorticosteroid) preparations of basic therapy. Their antiasthmatic effect is stronger than the drugs mentioned above. Drugs in this group have very high anti-inflammatory activity and reliably protect against hardening of the bronchial tubes, that is, from irreversible narrowing. Glucocorticosteroids, is available as a pocket inhaler, subject dosing regimen act only on the bronchial tree, without significant effect on other organs and body systems. By Inhaled steroids are bekotid, fliksotid, ingakort, benacort. Long-term use of these drugs that prevent the occurrence of asthma attacks, does not lead to any significant side effects. Only occasionally observed oral candidiasis, hoarseness that can be easily eliminated antifungal agents and does not require discontinuation of treatment. Also, the use of a nebulizer (delay coarse particles causing side effects) and washing the mouth with water after treatment to reduce side effects. Glucocorticosteroid hormone drugs intended for oral administration (prednisolone, methylprednisolone, triamcinolone, dexamethasone), appointed only in severe exacerbation of asthma. Typically, hormone therapy is not durable, which makes it possible at the end of treatment to abolish these drugs.

Bronchodilators, mainly affects only the symptoms of the disease. These include:

1) B-2 agonists (berotek, salbutamol, Ventolin, astmopent), commonly used as a handheld inhalers, can quickly eliminate choking at the first signs of its appearance (remember the basic preparations for this are not intended). Also available in the form of inhalers.

2) M-holinoblokatory (Atrovent) liquidating bronchospasm in some patients.

3) Symptomatic bronchodilator — extended form of theophylline (teotard, teopek, teolong).

Step 1: rescue medication as needed, this step is only for patients who did not receive maintenance therapy and occasionally experiencing short-term (up to several hours) asthma symptoms. For most patients, the recommended drugs emergency stage 1 are inhaled β2-agonists short action-fenoterol (DAI) 100 mcg (1–2 breaths) or salbutamol (DAI) 100 mcg (1–2 breaths). Alternative agents are inhaled anticholinergics, oral β2-agonists short-acting or short-acting theophylline, although these drugs is characterized by a slow onset of action and a higher risk of side effects, situational or salbutmol / beclomethasone 100 / 50 mkg 1–2 breaths during episodes of respiratory discomfort before loading.

In the case of more frequent occurrence of symptoms shows regular maintenance therapy

Step 2: Reliever medication — fenoterol (GAI) 100 mcg (1–2 breaths) or salbutamol (DAI) 100 mcg (1–2 breaths) — situationally plus one medication to control the disease.

As an initial maintenance treatment of asthma in patients of any age in the stage 2 of inhaled glucocorticoid is recommended in a low dose.

The initial maintenance therapy: inhaled glucocorticoids in low daily dose: 200–500 mcg of beclomethasone dipropionate or equivalent budesonide — 200–400 mcg, or fluticasone — 100–250 mcg, or ciclesonide 80–160 micrograms per day. Alternative means to control asthma are leukotriene receptor antagonists. Theophylline sustained release has only weak anti-inflammatory action and low efficiency as a maintenance treatment, moreover, it is often accompanied by the development of side effects of varying severity — from mild to severe. Cromones (nedocromil sodium and sodium cromoglycate) have relatively low efficiency, although characterized by high security. Symptomatic therapy. During exacerbation — hospitalization.

Step 3: Reliever medication plus one or two drugs to control the disease. At stage 3 adults and teenagers are encouraged to nominate a combination of inhaled glucocorticoids with an inhaled β2-agonist long-acting: salmeterol / fluticasone 25/50 mg / dose of 2 inhalations 2 times daily or salmeterol / fluticasone 50/100 multidisk mcg / dose of 1 inhalation 2 times per day, or budesonide / formoterol 80 / 4.5 mg / dose inhalation 2 1-2 times a day, or beclomethasone / 100 mcg formoterol / 6 2.1 mg 2 inhalations twice a day; after failure of monotherapy with optimal doses of inhaled corticosteroids (beclomethasone, budesonide, fluticasone, ciclesonide).

Step 4: Reliever medication plus two or more drugs to control the disease. The choice of drugs depends on the level of 4 from previous appointments at levels 2 and 3. In step 4 is preferable to use a combination of inhaled glucocorticoids in the medium or high dose inhaled a β2-agonist long-acting.

Step 5: Reliever medication plus additional options for the use of means to control the disease (as in step 4 + glucocorticoids orally or in / in, or IgE + blockers in severe atopic asthma with giper IgE-emiey).

Standard therapy for acute asthma — a high dose of β2-agonist with a modification of the delivery system (nebulizer therapy) and a short intensive course of high-dose systemic corticosteroids (oral or intravenous). After the relief of asthma exacerbations are usually given supportive therapy in the same volume.

Emergency medical care in a fit of asthma and developing status asthmaticus in an outpatient setting. Exacerbations of asthma (asthma attacks or acute asthma) are episodes of increasing shortness of breath, coughing, wheezing, chest tightness, or some combination of these symptoms**.** The primary therapies for exacerbations include repeated inhalation of bronchodilators quick action, the early use of systemic corticosteroids, and oxygen. The goal of treatment is to as quickly as possible the elimination of bronchial obstruction and hypoxemia and prevent further recurrences. Severe exacerbations are life-threatening, treatment should be carried out under the direct supervision of a doctor in the hospital, most patients — in the intensive care unit.

Asthmatic status — heavy prolonged attack of bronchial asthma with severe acute and progressive respiratory failure due to airway obstruction with the formation of patient resistance to treatment.

Slow-paced speaker. Bronhoastmatichesky status — a condition complicating bronchial asthma attack and characterized by an increase in intensity and frequency of asthma attacks against the background of resistance to standard therapy, inflammation and edema of the mucous membrane of the bronchial tubes in violation of the drainage function of the accumulation of thick mucus.

Diagnostics. During the AS traditionally distinguish 3 stages:

Stage 1 (relative compensation). Consciousness is clear, but the majority of a sense of fear, may be euphoria, excitement. Body position — forced — the patient is sitting with a fixed shoulder girdle. Pronounced acrocyanosis, shortness of breath 26–40 in 1 min. Difficult to breath, painful cough without sputum. Auscultation breathing hard, held in all parts of the lungs is determined by a large number of dry wheezing. Heart sounds are muffled, tachycardia, accent 2 tones of the pulmonary artery hypertension.

2 step (decompensation). Consciousness is kept, may cause hypoxic encephalopathy (excitation periods followed by periods of apathy). Overall condition is severe or extremely severe. Patients exhausted, the slightest load dramatically affects consciousness. Skin and visible mucous cyanotic, wet, swollen neck veins. BH 40 in 1 minute, breathing shallow. Breath sounds are heard at a distance of several meters, however, when auscultation there is a

mismatch between the expected number of wheezing and their actual presence, there are areas of "silent" light. This feature is typical of AS 2 stages. Heart sounds are muffled sharply, hypotension, heart rate 110–120 in 1 min, develop signs of acute right heart failure.

Stage 3 (hypercapnic coma). Overall condition is very serious. The patient is unconscious, before losing consciousness possible convulsions. Diffuse Diffuse "red cyanosis" cold sweat. Pupils greatly expanded sluggish reaction to light. BH 60 in 1 minute, breathing shallow, arrhythmic, a transition to bradypnea. Auscultatory sounds over the lungs is not listening, the painting "silent" light. Heart sounds are muffled sharply, hypotension, tachycardia, blood pressure dramatically reduced or not is determined. Signs of dehydration total reach their maximum. Compounded by signs of right heart failure.

Urgent Care. Basic principles: oxygen therapy, infusion therapy, drug therapy.

Oxygen: insuflirovat continuously through a mask anesthesia machine or inhaler oxygen-air mixture with an oxygen content of not more than 30–40 %. Do not use high concentrations of oxygen (possibility of absorbitsionnyh atelectasis, drying of the bronchial mucosa and enhancement of bronchopulmonary obstruction, toxic effects of reactive oxygen species).

Infusion therapy — aims to meet the shortfall in BCC, dehydration and hemoconcentration elimination of hypertensive type;

— catheterization of peripheral or central vein;

— vnutrivennoe introduction 5–10 % glucose solution, reopoliglyukina — 1000 ml for 1 hour assistance.

Reopoliglyukina number (or other low molecular weight dextrans must be 30 % of the total volume of infusible solutions). Infusion solutions, sodium salt content can not be used due to the original hypernatremia and hypertonic dehydration. Sodium bicarbonate in the form of 4–5 % solution is used only when comatose rate of 2–3 ml / kg body weight.

Drug therapy — based on the total elimination of agonists and used as bronchodilators xanthine derivatives (aminophylline and its analogues) and glucocorticoid hormones. is the initial dose of aminophylline — 10–20 ml of 2.4 % solution intravenously slowly over 5–7 minutes;

— Supports dose aminophylline — 5 ml of 2.4 % solution of fractional drip to improve the clinical status of the patient;

— glyukokortikoid hormones

— in terms of methylprednisolone 120–180 mg intravenously;

— geparin — 5000–10000 IU intravenously with one of the plasma substitutes; possible low molecular weight heparins (fraxiparine, Clexane and DNR).

When conducting medical therapy are contraindicated:

— sedativnye and antihistamines (depress the cough reflex, increase bronchopulmonary obstruction);

— holinolitiki (dried mucosa thickened mucus);

— mukoliticheskie means to liquefy phlegm;

— antibiotiki, sulfonamides, procaine (highly sensitizing activity);

— preparaty calcium (deepen hypokalemia);

— diuretiki (initial increase dehydration and hemoconcentration).

When coma:

— srochnaya inbutatsiya trachea at the inadequacy of spontaneous breathing;

— iskusstvennaya ventilation;

— with necessary — cardiopulmonary resuscitation.

COPD. Classification. Plan examination of the patient with COPD. General principles of treatment of COPD in an outpatient setting. COPD — a chronic inflammatory disease of environmentally mediated respiratory system, mainly affecting the distal parts of the airways and lung parenchyma with the development of emphysema, shown partially reversible airflow obstruction, characterized by a progressive and growing phenomena of chronic respiratory failure.

Risk factors: Factors predisposing to the development of COPD:

— smoking (both active and passive);

— the impact of occupational hazards (dust, chemical pollutants, acids and alkalis couples) and industrial pollutants (S02, N02, black smoke, etc.);

— atmospheric and home (smoke from cooking and organicheskogo fuel) air pollution;

— genetic predisposition (alpha1-antitrypsin deficiency).

Factors that trigger an exacerbation of the disease: bronchopulmonary infection; increased exposure to exogenous damaging factors; inadequate physical activity.

Classification of COPD: COPD is classified by severity, in accordance with the recommendations of the experts of the international program «Global Initiative for Chronic Obstructive Lung Disease» (GOLD — Global Strategy for Chronic Obstructive Lung Disease, 2003). The classification used two criteria: clinical, taking into account the main clinical symptoms — cough, phlegm and shortness of breath; function that takes into account the degree of irreversibility of airway obstruction.

Stage 0. Increased risk of COPD: Chronic cough and sputum production. Exposure to risk factors. Lung function is not changed. This stage is seen as a pre-disease and not always realized in COPD.

Stage I. It is easy for COPD: The patient may not be aware of the fact that lung function had broken. Obstructive violations — FEV1 "/ FVC < 70 % FEV1" > 80 % of predicted values. Usually, but not always observed chronic cough and sputum production. Stage PA to moderate COPD:Patients seek medical attention because of dyspnea and exacerbation of disease. The increase in obstructive disorders (50 % < FEV1 "< 80 % of predicted values of FEV1" / FVC < 70 %). Noted increased symptoms with shortness of breath, appearing during physical exertion. The presence of repeated exacerbations affect the quality of life of patients.

Stage II. Average severe COPD: Patients seek medical attention because of dyspnea and exacerbation of disease. The increase in obstructive disorders (50 % < FEV1 " < 80 % of predicted values of FEV1" / FVC < 70 %). Noted increased symptoms with shortness of breath, appearing during physical exertion.

Stage III. Severe COPD: A further increase in airflow limitation (FEV1 "/ FVC < 70 %, 30 % < FEV1" < 50 % of predicted value), increase of dyspnea, exacerbations, which affects the quality of life of patients.

Stage IV. Very severe COPD: Quality of life suffers considerably, exacerbation can be life-threatening. The disease becomes debilitating for. Very severe airflow obstruction (FEV1 "/ FVC < 70 % FEV1" < 30 % predicted FEV1 values or "< 50 % in the presence of respiratory failure).

The severity of dyspnea was evaluated by questionnaire British Medical Research Committee (mMRS).

Table 1 — MMRS questionnaire to assess the severity of dyspnea

mMRC Degree 0	I feel short of breath only with strong physical load	
mMRC Grade 1	I gasp when I go fast on flat ground or climb the gentle slope	
mMRC Grade 2	Due to the shortness of breath I go roomed flat spine slower than people of the same age, and I stopped breathing when I walk on level ground in the usual change of pace	
mMRC Grade 3	I gasp after about 100m pass, or after a few minutes walk on level ground	
mMRC Grade 4	I have too severe shortness of breath to leave the house, or I choke, the pervasive nature of undress or dress	
Mark the statement that applies to you (only one statement)		

Table 2 — Symptoms and risk of progression to COPD

Groups	Characteristics	Spirometric classification	Exacerbations, in year	mMRC	CAT
A	Low Risk Few symptoms	GOLD 1-2	≤ 1	0–1	< 10
B	Low Risk More symptoms	GOLD 1-2	≤ 1	> 2	≥ 10
C	High risk Few symptoms	GOLD 3-4	> 2	0–1	< 10
D	High risk More symptoms	GOLD 3-4	> 2	> 2	≥ 10

Phase of COPD: By clinical signs distinguish two main phases COPD: a phase controlled or stable course of the disease and uncontrolled.

Considered a stable state when the progression of the disease can be detected only after prolonged dynamic monitoring of patients, and the severity of symptoms does not change for a few weeks and even months.

Uncontrolled flow is characterized by frequent exacerbations (more than 3–4 times per year), the progression *o*f clinical symptoms and disorders ERF. Aggravation — deterioration of the patient manifested increase of symptoms and functional disorders and lasting at least 5 days. Exacerbations may begin gradually, little by little, and can be characterized and the rapid deterioration of the patient with the development of acute respiratory and right ventricular failure.

Diagnostic criteria for COPD:

• A diagnosis of COPD should be considered in any man who has a cough, excessive sputum production, and / or shortness of breath. It is necessary to take into account each patient's risk factors for the disease.

• Chronic cough and excess sputum production often precede the long vent frustration, resulting in breathlessness.

• If you have any of these symptoms should conduct a study of respiratory function.

• The signs are not diagnostically significant individually, but there are several of them increases the likelihood of disease.

Table 3 — To quantify the severity of dyspnea scale is used dyspnea Medical Research Council Dyspnea Scale (MRS)

Degree	The severity	Characteristic
0	*No*	Dyspnea does not bother with the exception of very intense Loading the district
1	*Easy*	Shortness of breath when walking fast or climbing a small hill
2	*Average*	Dyspnea leads to a slow walking as compared with others of the same age or causes to make stops walking at its own pace on the level
3	*Heavy*	Dyspnea makes make stops at walking distance of 100 meters or a few minutes walk on level ground
4	*Very heavy*	Dyspnea makes it impossible to go beyond the home or shortness of breath occurs when dressing and undressing

Continuation of the table 3.

Action risk factors in history: if the patient smokes, it is necessary to calculate the *index smoker* "pack / years." IR is calculated as follows: the number of cigarettes smoked per day multiplied by the common experience of smoking (years) and divide by 20. IR more than 10 packs / year is a significant risk factor for COPD.

Plan examination of patients: ***ERF study***: registered a decrease VC reserves of inhalation and exhalation, to increase OOL, reducing values of FEV1, FVC, FEV1 decline in relation to the FVC less than 70 % (the early and sensitive indicator of airflow limitation). Bronchodilation test in patients with

suspected COPD should be made to exclude the group of patients with a significant increase in FEV1, "in response to a bronchodilator, which can be suspected asthma. Evaluate the test as follows:

• increase in FEV1, "more than 15 % (or 200 ml), indicating the reversibility of airflow obstruction;

• FEV1 "after using a bronchodilator has prognostic value and indicates standby treatment options;

• In the absence of increase in FEV1, "the patient can celebrate subjective dyspnea and increased distance walk, which indicates the advisability of administering bronchodilator him despite the negative functional response.

Chest X-ray in patients with COPD primarily used for differential diagnosis and detection of comorbidities in COPD.

CT scan can reveal the extent and prevalence of emphysema.

Arterial blood gas measurement can detect hypoxia and hypercapnia.

Electrocardiography: we can estimate the state of the myocardium and signs of hypertrophy and overload of the right ventricle and atrium.

Sputum. Sputum examination can reveal the cellular composition of bronchial secretions. Bacteriological examination of sputum is useful to identify the causative agent if there are signs of purulent process in the bronchial tree, as well as its sensitivity to antibiotics.

Treatment of patients with COPD:

Main directions of treatment:

I. Reducing the influence of risk factors: Smoking - Cessation and Prevention.

II. Educational programs: smoking cessation; basic information about COPD; general approach to therapy and specific aspects of medical treatment; samovyvedeniyu skills and decision-making during an exacerbation.

III. Treatment of COPD in stable condition:

• Stepwise increase in treatment, depending on the severity.

• Patient education, elimination of risk factors.

• Bronchodilators are central to the symptomatic treatment of COPD.

• Inhaled corticosteroids are indicated in patients with FEV < 50 % predicted and repeated exacerbations.

• At all stages of the process flow of the high efficacy of physical coaching programs that improve exercise tolerance and reduces breathlessness and fatigue.

• Patients with severe respiratory failure is a long-oxygen therapy (more than 15 hours a day).

— ***Bronchodilators*** with stable COPD:

• Bronchodilators are central to the symptomatic treatment of COPD.

• inhalation therapy is preferred.

• Bronchodilators are appointed "on demand" or on a regular basis to prevent or reduce the symptoms of COPD. In order to prevent the rate of progression of bronchial obstruction priority is long and regular treatment.

• M-anticholinergics are the first-line drugs in the treatment of COPD and their purpose is obligatory at all degrees of disease severity. Parasympathetic tone is a leading component of airflow obstruction in COPD.

• bronchodilators combination enhances bronchodilator effect and reduces the risk of side effects compared with increasing the dose of one drug.

• Regular treatment of long-acting bronchodilators more effective and convenient than treatment with short-acting bronchodilators, but more expensive.

• Regular treatment of long-acting bronchodilators (tiotropium bromide, salmoterol, formoterol) is recommended for moderate, severe, very severe COPD.

• Patients with moderate, severe or very severe COPD an inhaled AHP, beta2-agonists, long-acting as monotherapy or in combination with long-acting theophylline.

• Xanthines effective in COPD, but in view of their potential toxicity of drugs are "second line". Xanthines can be added to regular inhaled bronchodilator therapy in more severe disease.

— ***Corticosteroids with stable COPD***: The therapeutic effect of corticosteroids in COPD are much less pronounced than in BA. Regular treatment prescribed corticosteroids in addition to bronchodilator therapy in patients with severe and very severe COPD at an annual or more frequent exacerbations in the past 3 years. The effectiveness of treatment is estimated at 6–12 weeks of inhaled corticosteroids by acting bronchodilator test. Response to treatment is considered to be positive for growth in FEV1 greater than 15 % or 200 mL or more to the initial value in the bronchodilator test.

— ***vaccines***: In order to prevent COPD exacerbations during outbreaks of influenza flu vaccines are recommended, once appointed in October and November each year. Also applies pneumococcal vaccine.

— ***Antibiotics***: Because of the risk of adverse drug events in patients and the emergence of bacterial resistance Antimicrobial Chemotherapy use as a prophylactic measure in patients with COPD should not be a daily practice**.**

— ***Mucolytic means***: Mucolytics shown limited contingent of patients with stable COPD in the presence of viscous mucus.For the prevention of exacerbations is a promising long-term use of N-acetylcysteine (ACS), which has both antioxidant activity. Admission NAS for 3–6 months at a dose of 600 mg / day, accompanied by a decrease in the frequency and duration of exacerbations of COPD.

— ***Oxygen***: The main cause of death in patients HBOL is Nam. Correction of hypoxemia with oxygen — the most reasonable method of therapy pathophysiologically Nam. The use of oxygen in patients should be permanent, durable and usually held at home.

— ***Surgical treatment***: Perhaps a bullectomy that leads to a reduction of dyspnea and improved lung function in COPD patients.

Table 4 — Pharmacotherapy with stable COPD treatment

Groups patients	Preparations first-line	Preparations second line	Alternative selection
A	Short-actinganticholinergic or ß2 - agonist short-acting	A long-actinganticholinergic or ß2 - agonist long-acting, or anticholinergic and short-acting ß2 - agonist short-acting	Theophylline
B	A long-actinganticholinergic or ß2 - agonist short-acting	A long-actinganticholinergic and ß2 - agonist long-acting	ß2 - agonist short actions and / or short-acting anticholinergic Theophylline
C	Inhalation GCS + ß2 agonist long-acting or long-acting anticholinergic	A long-actinganticholinergic and ß2 - agonist long-acting	PDE - 4 inhibitor ß2 - agonist short-acting and / or short acting anticholinergic. Theophylline
D	Inhalation GCS + ß2 agonist long-acting or a long-acting anticholinergic	Inhalation GCS + ß2 agonist or inhaled long-acting GCS and ß2 agonist long-acting and long-acting anticholinergic or inhaled GCS and ß2 agonist \ long-acting and PDE - 4 or anticholinergic long-acting and ß2 - agonist or long-acting anticholinergic long-acting and PDE - 4	Karbotsistein ß2 - agonist short-acting and / or short-acting anticholinergic Theophylline

Table 5 — The dosage, administration route, duration and brand names of drugs used in COPD

Preparations: international (business) name)	Inhalation, g	A solution for a nebulizer, mg / ml	Orally	The duration of the action, watch
ß2 agonists				
Short-acting				
Fenoterol (Berotek, Berotek H)	100–200 (MDI)		0.05 % (syrup)	4–6
Fenoterol (Berotek H)	100–200 (MDI)		0.05 % (syrup)	4–6
Salbutamol (Ventolin, Salbutamol)	100–200 (MDI, DPI)	5	5 mg (Table)	
0.024 % (syrup)	4–6			
Terbutaline	400, 500 (DPI)			4–6
Long-acting				
Formoterol (Faraday)	4.5–12 (MDI, DPI)	0.01		12

Preparations: international (business) name)	Inhalation, g	A solution for a nebulizer, mg / ml	Orally	The duration of the action, watch
Indakaterol (Onbrez Brizhayler)	75–300 (DPI)			24
Salmeterol (Serevent)	25–50 (MDI, DPI)	0.0075		12
Tulobuterol			2 mg (Trans-dermally)	24
Anticholinergic				
Short-acting				
Ipratropiumbromide (Atrovent, Atrovent H)	20, 40 (MDI)	0.25–0.5		6–8
Long-acting				
Tiotropiumbromide (Spiriva)	18 (DPI), 5 (SMI)			24
The combination of short-acting ß2-agonist and anticholinergic in a single inhaler				
Fenoterol /ipratropium bromide (Flomax, Flomax H)	200/80 (MDI)	1.25 / 0.5		6–8
Salbutamol / ipratropium bromide (Iperamol Ster-Neb)	75/15 (MDI)	0.75 / 0.5		6–8
Methylxanthines				
Aminophylline (Eufillin)			200–600 mg (Table)	24 hour
Theophylline (SR) (Teotard, teopeka)			100–600 mg (Table)	24 hour
Inhaled corticosteroids				
Beclomethasone (bekotid, Beklazon Eco Maple)	50–400 MDI,DPI)	0.2–0.4		
Budesonide (Pulmicort, benacort, budesonide Iziheyler)	100, 200, 400 (DPI)	0.2; 0.25; 0.5		
Fluticasone (Flixonase, Flixotide)	50–500 (MDI, DPI)			
The combination of long-acting ß2 agonist and corticosteroids in one inhaler				
Formoterol / budesonide (Symbicort, Foradil Combi)	4.5 / 160 (MDI); 9/320 (DPI)			
Salmeterol / fluticasone (Seretide, Seretide Multidisk)	50/100, 250, 500 (DPI); 25/50, 125, 250 (MDI)			
Beclomethasone / formoterol (Foster)				
Systemic corticosteroids				
Perdnizon			5–60 mg (Table)	
Metilprednizon			4, 8, 16 mg (Table)	
Phosphodiesterase inhibitors - 4				
Roflumilast (Daxas)			500 g (Table)	24

Continuation of the table 5

Treatment of COPD exacerbations: Typically, during the year the patient suffers from one to four or more exacerbations of COPD.

— *Bronchodilators:* One of the basic principles of treatment of COPD exacerbations is the intensification of bronchodilator therapy by increasing the dose and modification methods of drug delivery. First-line drugs are ß-agonists, short-acting (salbutamol, fenoterol) and ipratropium bromide.

— *Corticosteroids*: During exacerbation of COPD, accompanied by a decrease in FEV1 less than 50 % of predicted values, SCS appointed along with bronchodilator therapy.

— *Antibiotic therapy*: Indications for antibiotic therapy in patients undergoing acute exacerbation of COPD are increased dyspnea, increased sputum and purulent her character, fever and leukocytosis.

— *Oxygen*: Oxygen is one of the key areas of complex treatment of patients with acute exacerbation of COPD in the hospital. After the start of oxygen therapy via nasal catheters.

— *Non-invasive ventilation*: If after 30–45 minutes of inhalation of the patient with ARF effectiveness of oxygen therapy is not available, should make a decision on ventilation.

Clinical management of patients with acute exacerbation of COPD in an outpatient setting:

I. Standard laboratory control and monitoring tool.

1. Complete blood count.
2. Chest X-ray.
3. General analysis of sputum.
4. Sputum smears.
5. Bacteriological examination of sputum (indication).
6. ECG.
7. Spirometry.
8. The peak flow.

II. Algorithm therapy.

1. Bronchodilators. Increasing the dose and / or frequency of administration. If not applied earlier, it added anticholinergic drugs to improve symptoms. Preferably combined bronchodilators (Flomax). With lack of effective bronchodilators and corticosteroids may be prescribing theophylline.

2. Corticosteroids. If the initial FEV1 < 50 % predicted, then added into 40 mg of prednisolone daily for 10 days to receive a bronchodilator.

3. Antibiotics. In strengthening the symptoms of shortness of breath and cough, purulent sputum and increasing its volume used antibiotics, taking into account local characteristics and individual tolerance.

Prevention of asthma and COPD. Primary prevention of asthma and COPD should be undertaken to people (especially children) to high-risk groups.

These include the following people:

• with a hereditary predisposition to allergic reactions and diseases, especially allergic respiratory diseases;

• with signs of atopic dermatitis;

• with repeated episodes of croup;

• with signs of bronchial obstruction during SARS.

Measures for primary prevention of asthma and COPD are:

• elimination of occupational hazards;

• smoking cessation;

• a balanced diet with restriction of products with high allergenic and gistaminliberatornoy activity;

• prevention of acute respiratory viral infections;

• Reducing exposure to aeroallergens dwellings;

• termination of passive smoking;

• use of physical rehabilitation, hardening;

• Reducing exposure to chemical agents in the home;

• good ecological environment.

Secondary prevention of exacerbations of asthma and COPD is based on:

• combat chronic lung infection, sinusitis et al.;

• eliminate contact with the allergen.

TOPIC 2: Differential diagnosis of chest pain. Noncoronary heart disease: patient diagnosis, treatment guidelines, medical tactics, clinical examination, the prevention.

Main diseases and pathological conditions manifested by pain in the chest, like the heart (cardialgia). Chest pain has many causes, including requiring attention. Causes pain can be divided into two broad categories — "cardiac" and "non-cardiac".

Cardiac causes

• Myocardial infarction — a blood clot that blocks blood flow in the arteries of the heart can be the cause of crushing, the contraction of chest pain lasting more than a few minutes. The pain may be given (radiate) to the back, neck, jaw, shoulders and arms (especially the left). Other symptoms may include shortness of breath, cold sweat, nausea.

• Angina. Restriction of blood flow through the arteries of the heart is the cause attacks of chest pain — angina. Angina is often described as a feeling of people of compression or compression of the chest. It usually occurs during exercise or stress. The pain usually lasts for about a minute and ends in peace.

• Inflammation of the heart shirts (pericarditis), most often occurs due to a viral infection. Pain in pericarditis are often acute, stabbing character. There may also be fever and malaise.

• Aortic dissection. The inner layer of the artery can be separated under the pressure of the blood and the result is a acute and sudden severe pain in the

chest. Aortic dissection can be a result of chest injuries or complications of uncontrolled hypertension.

"Non-cardiac" causes

• Heartburn. The acidic gastric juice gets from the stomach into the esophagus, may be the cause of heartburn — painful burning sensation in the chest. Often it is combined with a sour taste and belching. Chest pain heartburn is usually associated with eating and can last for hours. This symptom usually occurs when bending or lying down. Eases heartburn antacids.

• Panic attacks. If a patient experiences bouts of irrational fear, combined with chest pain, heart palpitations, hyperventilation (rapid breathing), and profuse sweating, it could be "panic attacks" — a peculiar form of dysfunction of the autonomic nervous system.

• Pleurisy. The acute localized pain in the chest, worse during inspiration or coughing can be a symptom of pleurisy. The pain occurs due to inflammation of the membrane that lines the chest cavity from the inside and covering the lungs. pleurisy can occur in various diseases, but most of all — with pneumonia.

• Osteochondrosis of the cervical and thoracic spine leads to the so-called vertebral cardialgia that resembles angina. In this condition there is an intensive and prolonged pain in the chest, in the left side of the chest. Can be observed in the irradiation of the hands, interscapular region.

• Tietze syndrome. Under certain conditions, the cartilage of the ribs, especially cartilage that attach to the breastbone can become inflamed. The pain in this disease may occur suddenly and be quite intense, mimicking angina.

• Pulmonary embolism. This type of embolism occurs when an embolus gets into the pulmonary artery, blocking blood flow to the heart. The symptoms of this life-threatening conditions may include sudden, sharp chest pain that occurs or increasing deep breathing or coughing. Other symptoms — shortness of breath, palpitations, anxiety, loss of consciousness, pronounced cyanosis.

• Other diseases of the lungs. Pneumothorax (collapsed lung), high blood pressure in the blood vessels that supply the lungs (pulmonary hypertension) and severe asthma also may be manifested by pain in the chest.

• Diseases of the muscles. The pain caused by muscle disease usually begins to disturb the body during cornering or when a show of hands.

• Damage to edges and pinched nerves.

• Diseases of the esophagus. Certain diseases may cause esophageal swallowing disorder, and hence, discomfort in the chest. Esophageal spasm may be the cause of chest pain.

• Shingles. It is an infection caused by the herpes virus and affects the nerves, may be a cause severe pain in the chest. The pain may be localized in the left side of the chest or wear shingles character.

• Diseases of the gallbladder and pancreas. Gallstones or inflammation of the gallbladder (cholecystitis) and the pancreas (pancreatitis) may cause pain in the upper abdomen, smack in the heart area.

• If the patient less than 30 years and he was concerned aching pain in the apex of the heart, lasting for hours, from which it is possible to escape during a conversation or daily activities, then it is likely a manifestation of vegetative-vascular dystonia. Such pain, as well as piercing in the apex of the heart, is not dangerous.

Features pain in heart disease (CHD, dry pericarditis, myocardial dystrophy, myocarditis, dilated and hypertrophic cardiomyopathies), with somatoform autonomic nervous system dysfunction, mitral valve prolapse. The algorithm of diagnostic search .Differential diagnosis.

When **angina** often worried feeling of pressure in the chest with a typical radiating to the left arm; usually during physical exertion, often after a meal or in connection with emotional stress. Diagnostically significant effect of nitroglycerin and recreation.

Acute myocardial infarction. Feeling close to those described in myocardial ischemia, but the intensity and duration (about 30 minutes), rest or nitroglycerin does not get rid of them.

Noncoronary heart disease.Myocarditis. Breast pain occurs in 75–90 % of patients with myocarditis. As a rule, pressing, aching, or stabbing pain, usually in the region of the heart. Communication with physical activity can not be traced, sometimes there is a growing pain in the subsequent days after exercise. Nitrates pain is not cropped.

Pericarditis. Most often, the pain of pericarditis occurs only at the beginning of the disease, when there is friction sheets pericardium. When large amounts of fluid in the pericardial cavity or cavities fusion of pain disappears, and therefore, short pain. By the nature of the pain may be dull, aching, or, on the contrary, acute cutting. A characteristic feature of the pain of pericarditis is dependent on breathing and body position.

Cardiomyopathy. Pain syndrome occurs in all patients with cardiomyopathy, but it is most typical in hypertrophic cardiomyopathy.

Acquired heart defects. Severe myocardial hypertrophy contributes to the development of the relative failure of the coronary circulation disorders and metabolic processes in the myocardium.

Mitral valve prolapse. The chest pain in this pathology long, aching, stabbing or poignant, often on inspiration, not cropped nitroglycerin.

Myocardiodystrophy. Clinical manifestations of myocardial dystrophy characterized by little and at the same time are quite diverse. Pain in the precordium there often is diverse.

Hypertension and symptomatic arterial hypertension is often accompanied by a variety of pain in the precordium. caused by excessive voltage wall of the aorta and the stimulation of mechanoreceptors in the left ventricular myocardium.

Neurocirculatory dystonia. Paroxysmal, relatively brief, but often repeated during the day, compressing or squeezing pain of various localization, passing mostly on their own, but often ameliorated nitroglycerin.

The algorithm of diagnostic search. Differential diagnosis.

On questioning the patient especially isolated complaints of pain in the heart, heartbeat, feeling disruptions of the heart, pulsation in various parts of the body, shortness of breath, presence of edema, nocturia.

On examination can reveal typical symptoms of heart and vascular diseases: characteristic changes in a person with mitral heart disease (facies mitralis), its expression at the time of angina attack, forced sitting position of the patient with heart failure, cyanosis, pallor and marble pattern skin with vascular insufficiency, pulsation various vessels, heart hump, especially the apical impulse, presence of edema.

Auscultation allows to evaluate heart sounds and heart sounds, identify vascular noise, which is important in the diagnosis of congenital and acquired heart disease. With *ECG* determine the position of the heart, signs of hypertrophy of its departments, metabolic abnormalities in the myocardium. Important information about the state of the walls of the heart and its valves and the walls of the aorta gives *echocardiography*. With its help detect various heart defects, cardiomyopathy, aneurysm of the heart and aorta, scarring wall infarction, pericarditis. To assess the state of vessels and local blood flow is used angiography, including *coronary angiography*. *Nuclear medicine* use in myocardial infarction, for the study of central and peripheral hemodynamics. In the diagnosis of myocardial infarction is widely used definition of the content of certain *enzymes in the blood* (creatine phosphokinase, lactate dehydrogenase). Atherosclerosis for diagnosis and management of treatment investigated *fats, fat-protein complexes* in the serum. *Bacteriological and immunological studies*, determination of proteins and protein-carbohydrate complexes is carried out in septic endocarditis, myocarditis.

Features pain in the chest caused by diseases of the abdominal cavity, lungs, pleura, peripheral nervous system and the muscles of the shoulder girdle (intercostal neuralgia, osteochondrosis of the cervical-thoracic spine, shingles). Differential diagnosis of diseases of the heart.

Chest pain may be a manifestation pleuritis, pneumonia, malignant neoplasm of the lung. Diseases of the intestine (duodenal ulcer, pancreatitis or cancer pancreatic cancer, cholecystitis) , subphrenic abscess.

Respiratory diseases. Pain in diseases of the lungs:

1. The emergence or worsening of pain with deep breathing or coughing.
2. Acute intermittent pain, usually limited, with no tendency to irradiation.
3. The presence of other pulmonary symptoms (cough, sputum, shortness of breath or dyspnea of different kinds).
4. Acute or chronic lung disease history, pleural rub, dry or moist rales, percussion evidence of emphysema, cavernous formations or seal lung tissue.

Abdominal disease. When *esophagitis* is marked a burning sensation behind the breastbone, pain along the esophagus, worse when swallowing, associated with the intake of cold or hot, solid food. With *Achalasia* (cardiospasm, idiopathic enlargement of the esophagus) localized pain behind

the breastbone, is clearly associated with dysphagia and regurgitation of food. Pain episode may be triggered by eating. Pain during hiatal *hernia* is most often localized in the lower part of the sternum. Characteristic of its appearance or worsening after a meal, in a horizontal position, decreases pain when rapid change in body position. Pain in *gastric ulcer* and *duodenal ulcer*, *chronic cholecystitis* can sometimes radiate to the left side of the chest.

Chest pain associated with neurological diseases

The syndrome of musculo-fascial or rib-vertebral pain (not visceral):

1. Fairly constant localization of pain.

2. Unconditional link pain with a voltage corresponding muscle groups and the position of the body.

3. The low intensity of pain, lack of common symptoms associated with chronic course or clear conditionality beginning in acute injury.

4. Clear data palpation, enabling the identification of pathology: local pain (limited) palpation relevant muscle groups, the presence of trigger zones.

5. Reduction or disappearance of pain in various local impacts.

Radicular pain syndrome (including intercostal neuralgia):

1. Acute onset or exacerbation clear the chronic course.

2. The preferential localization of pain in the area of the corresponding nerve root.

3. A clear connection with the movements of the spine (with radicular pain) or trunk (neuralgia).

4. Neurological symptoms of cervical or breast sciatica.

5. A sharp local pain at the exit of the intercostal nerves.

Osteochondrosis. This degenerative-dystrophic lesions of the intervertebral disc, in which the process, from the nucleus pulposus in more progressively extended to all elements of the disk followed by the involvement of the entire segment (adjacent vertebral bodies, intervertebral joints, ligamentous apparatus).

Musculo-fascial syndrome occurs in 7–35 % of cases. When *frozen shoulder* pain is associated with the movements of the shoulder joint, marked trophic changes hands.

The main signs and symptoms of **shingles** are there is a feeling of pain for a couple of days before the appearance of skin rash. With shingles pain is worse at night, as well as under the influence of different stimuli (touch, cold). With shingles pain is localized along the nerves.

Myocarditis. Plan examination of the patient in an outpatient setting, diagnostic criteria. Principles of treatment on an outpatient basis. Clinical examination.

Myocarditis — is damage to the heart muscle, predominantly inflammatory nature caused by immune mechanisms mediated by the action of infections, parasitic or protozoal infestations, chemical and physical factors, and occurs as a result of allergic and immune diseases.

Classification

The most frequent causes of myocarditis are factors of infection and allergy. For any infectious disease is probable development of myocarditis.

***The etiology*:**

1. *Infectious* myocarditis is represented by various microorganisms:

• Viruses: Enterovirus (Coxsackie A and B, ECHO, polio), adenoviruses, arboviruses, cytomegalovirus, HIV, hepatitis, influenza, mumps, rubella, measles, chicken pox, herpes, infectious mononucleosis (Epstein-Barr virus), and others.

• Bacteria: streptococci, staphylococci, pneumococci, meningococci, gonococci, bacteria, diphtheria, tuberculosis, brucellosis, and others.

• Spirochetes (syphilis, leptospirosis, relapsing fever, Lyme disease).

• Rickettsiae (typhus fever, Q).

• Simple and helminths (toxoplasmosis, trypanosomiasis (Chagas disease), trichinosis, cysticercosis,

• Fungi (actinomycosis, candidiasis, coccidioidomycosis, aspergillosis, and others).

2. *Non Infectious:*

— Allergic — when serum drug and disease.

— Caused by physical and chemical effects — radiation, toxic; electrolyte imbalance, etc.

3. *Idiopathic* — in cases of unknown etiology.

The cause of allergic myocarditis are immuno allergic reaction to form antigen-antibody complexes. Similar complexes are formed in infectious and allergic, serum, nutritional, burns and some other myocarditis. Allergic myocardial damage is defined in systemic connective tissue diseases, asthma, Lyell's syndrome. Often the cause of myocarditis are *drugs*. Among the drugs that cause myocarditis leading role for antibiotics (doxorubicin, anthracycline, streptomycin, chloramphenicol).

By severity:

1. Mild — without increasing the size of the heart and heart failure.
2. Moderate — with an increase in heart size without HF.
3. Severe — cardiomegaly, heart failure and severe arrhythmias.

For the duration of myocarditis are divided into:

• acute;
• subacute;
• recurrent;
• chronic;
• latent.

Pathogenetic basis for myocarditis is divided into infectious and infectious-toxic, allergic (immunologic), toxic and allergic. In the presence of an infectious agent isolated certain pathogenic phases: an infectious-toxic, immunoallergic, dystrophic, cardiosclerosis. The process can stop at one of the phases is not going to follow. When myocarditis allergic genesis marked the development of the first phase of the disease.

As the prevalence of pathological process in the heart muscle myocarditis divided (rather arbitrary) on *focal and diffuse*. By the nature of the clinical course of the inflammatory process is isolated *acute,subacute, chronic myocarditis*. Chronic course of myocarditis confirmed by histological examination, however, in the majority of cases are diagnosed dilated cardiomyopathy.

In bacterial myocarditis predominant humoral immune response, antibodies. When myocarditis caused by Mycobacterium tuberculosis or fungi prevalent cellular immune responses, but play an important role and humoral responses. The role of inflammation in a *viral infection* of the myocardium can be traced only in the acute stage of the process. Infiltration into the heart muscle, they are fixed on the surface receptors of muscle cells, and then penetrate into the cells of the myocardium. It should be braking function of host cells, biosynthesis and the multiplication of viruses — replication and damaged myocyte becomes autoantigen. Microvascular changes appear sharp expansion of arterioles, capillaries, venules, swelling of the endothelium, the changes of the vascular walls — homogenization, fibrinous swelling, less necrosis.

The clinical picture. The clinical picture of myocarditis depends on the extent of myocardial injury and varies from asymptomatic course to severe heart failure and sudden death. Pathognomonic symptoms of myocarditis does not exist. Infectious and infectious-toxic myocarditis developed in the early days, an infectious-allergic — after 2–3 weeks of infection. The first manifestations of myocarditis may be fatigue, excessive sweating, low-grade fever, false angina, palpitations, and disruption of the heart, shortness of breath with exercise, and at rest, arthralgia. Pain intensity does not change during exercise does not depend on the time and emotional stress. May resemble angina, but receiving antianginal drugs does not eliminate it. On physical examination, pay attention to the increase in heart size — from small shift to the left border of cardiomegaly. Apical impulse is usually shifted to the left, down and relaxed. Muted tones (mostly I tone at the top of the heart in 80–90 % of patients). Physical findings are variable in myocarditis. Almost 50% of patients registered tachycardia, 10 % — bradycardia. Can be observed extrasystoles, tachycardia, atrial fibrillation and ventricular fibrillation.

Clinical variants of myocarditis:

1) oligosymptomatic option. It is characterized by minimal clinical manifestations, in particular, kardialgichesky symptom is weak, there is no pronounced hemodynamic disturbances, changes in ECG unstable, transient.

2) Pain option. Distinguished by intense pain in the heart (sometimes reminiscent of anginal pain severity status); ECG changes resembling myocardial infarction or ischemic focal changes; there may be problems left ventricular failure of varying severity (from mild stagnation in the lungs to the attacks of cardiac asthma).

3) Pseudo valve option. It is characterized by symptoms of sound from the heart, it is very reminiscent of the sound picture of heart disease, most often

mitral insufficiency (intense systolic murmur is heard in the apex of the heart), or combined mitral heart disease (intense systolic murmur, atrial fibrillation), with the possible polyarthralgia, circulatory failure. To eliminate heart disease requires careful ultrasound examination of the heart, it is also required to differentiate this version of the disease to rheumatism.

4) Arrhythmic option. The clinical picture is dominated by a variety of cardiac arrhythmias, while false angina, circulatory failure expressed little or even absent.

5) Thromboembolic option. It starts with thromboembolic complications, often with pulmonary embolism, at least — with thromboembolism in the artery of the systemic circulation, usually in patients with marked cardiomegaly and severity of clinical symptoms of congestive heart failure circulation.

6) Decompensated option. It is characterized by severe, total resistant to the treatment of heart failure, cardiomegaly, relative mitral and tricuspid insufficiency, severe, often combined cardiac arrhythmia.

7) Mixed option. The most common variant is characterized by a combination of symptoms of different options, that is, in fact, expanded clinical myocarditis.

Criteria for the diagnosis of myocarditis New York Heart Association (1973)

I. Major criterion.

1. Abnormal ECG changes — changes in repolarization, arrhythmias and conduction; in cases of doubt, this study adds to the sample with orthostatic or physical activity, drugs potassium (Board-blockers).
2. Increase in the activity of enzymes (AST, LDH 1-2 UFC).
3. Cardiomegaly, identified by X-ray or echocardiography study.
4. Congestive heart failure.

II. Minor criteria.

1. Tachycardia.
2. Weakening 1 tone.
3. Gallop rhythm.

Typically diagnosis: diagnosis is justified in the case of indications of previous infection + 2 large or 1 large and 2 minor criteria.

Instrumental and laboratory diagnostics

Complete blood count. In patients with mild myocarditis blood count does not change. When myocarditis moderate and severe marked increase in ESR, a moderate increase in the number of white blood cells with neutrophilic and stab shift, monocytosis.

Urinalysis unchanged, but with the development of severe heart failure found in the urine protein cylinders (mostly hyaline).

Biochemical analysis of blood. It is known that in case of damage of any etiology cardiomyocytes (ischemic, inflammatory or toxic) there is an increase of concentration of cardiac enzymes and proteins — creatine phosphokinase (CPK), its cardiac enzyme (CK-MB), lactate dehydrogenase (LDH), troponin T and troponin I.

The degree of damage and destruction of cardiomyocytes under myocarditis has not so massive nature, such as in myocardial so cardioselective enzyme concentration usually increases only in 1.5–2. When myocarditis with severe clinical symptoms characterized by "a biochemical syndrome of inflammation": an increase in the blood levels of fibrin, haptoglobin, seromucoid, A2 and γ-globulins at lower levels of albumin, the appearance of C-reactive protein.

By the value and volume of information received leading role in the diagnosis of myocarditis include **ECG**. ECG changes are non-specific, but they accompany myocarditis in 100 % of cases. ST segment shifts up or down from the isoelectric line with a simultaneous decrease in the amplitude of the wave flattening or T. These changes are characterized by phasic evolution. The first stage — acute — observed in the first days of the disease and is characterized by ST-segment depression and flattening teeth T. In the second stage (2–3 week disease) are formed negative, often symmetrical, pointed teeth T. In the third stage of the normalization of the ECG. With a moderate course of myocarditis these changes are observed 6–8 weeks.

Biopsy of the myocardium. It is believed that the subendocardial myocardial biopsy is the most accurate method of diagnosis of myocarditis, many researchers call it the "gold standard" diagnosis of this disease.

Recommendations for histological diagnosis of myocarditis, called "Dalass criteria".

Table 6 — "Dalass criteria"

Definedmyocarditis	Inflammatory infiltration of myocardial necrosis and degeneration of adjacent cardiomyocytes, not typical of the ischemic lesions of coronary artery disease
The probable myocarditis	Inflammatory infiltrates are rare or cardiomyocytes infiltrated leukocytes. No portions of cardiomyocyte necrosis. Myocarditis can not be diagnosed in the absence of inflammation
The absence of myocarditis	Normal or pathological changes in the myocardium biopsy noninflammatory nature

By using the ***ultrasonic*** method reveal signs of contractile function of the heart muscle. In patients with diffuse myocardial damage observed dilatation of the heart cavities and decreased hemodynamic parameters. ***X-ray*** method is used in patients with myocarditis to assess heart size and signs of pulmonary congestion. Apply other non-invasive methods of diagnosis of myocarditis: ***scintigraphy*** with radioisotopes (67Ga, 99mTc-pyrophosphate), the definition of monoclonal antibodies to aktinomiozinu labeled with 111In; magnetic resonance imaging. Improving ***enzymes*** AST, LDH (LDG1 and LDG2), CK and its fractions with an increase in muscle activity indicates necrosis of cardiomyocytes under pain version of myocarditis. Hyperenzymemia may persist for 2–3 weeks.

Treatment. Acute focal myocarditis that develops because of a viral infection often occurs oligosimptomatic and has a favorable prognosis. Treatment of patients is supportive in nature, aimed at arresting the main symptoms. Bed rest is given to all patients with acute myocarditis, its duration (1 to 6 weeks) depends on the severity of myocardial injury. Physical activity is limited to the disappearance of clinical symptoms and normalization of heart size and function indicators. Health food restriction of salt and fluid is indicated for symptoms of circulatory failure. Focus is on the causal treatment, which takes into account the identified or suspected factors. Bacterial myocarditis prescribe antibiotics in usual therapeutic doses for up to 3 weeks. Treatment of viral myocarditis should be carried out taking into account the phase of the pathological process: Phase 1 — virus replication; Phase 2 — autoimmune damage; Phase 3 — dilated cardiomyopathy.

In the treatment of myocarditis in allergic diseases and systemic antibiotics do not play a significant role, their application in some cases may even be unsafe. Rheumatic diseases using NSAIDs (aspirin, diclofenac, indomethacin, et al.) and glucocorticosteroids. Treatment of congestive heart failure is generally accepted means: diuretics, ACE inhibitors, cardiac glycosides, β-blockers. Anticoagulant therapy (heparin, low molecular weight heparins) is assigned when there is a risk of thromboembolic syndrome. The duration of treatment of myocarditis (1–6 months or more) depends on the severity of disease and the effectiveness of the therapy. Heart transplantation is planned if the treatment is not accompanied by improvement of clinical and functional parameters. Outcomes of myocarditis can be a full recovery, death in the early period of the disease, progressive dilatation of the heart cavities with development of chronic heart failure, further defining prognosis and survival ability to work, the formation of cardiosclerosis with persistent arrhythmias and conduction.

Pericarditis. Plan examination of the patient in an outpatient setting, diagnostic criteria. Principles of treatment on an outpatient basis.

Pericarditis — an inflammation of serous pericardium, the serous membrane of the heart.

I. Clinical classification

A. Acute pericarditis (less than 6 weeks).

1. Fibrinous.
2. exudative (or hemorrhagic).

B. Subacute pericarditis (from 6 weeks to 6 months).

1. Constrictive.
2. constrictive-exudative.

C. Chronic pericarditis (more than 6 months).

1. Constrictive.
2. Exudative.
3. Adhesive.

II. Etiologic classification

A. Infectious pericarditis.

1. Viral.
2. Festering.
3. Tuberculosis.
4. Fungal.
5. Other infections (syphilitic, parasitic).

B. Non-infectious pericarditis.

1. In acute myocardial infarction.
2. When uremia.
3. If a malignancy in primary tumors (benign or malignant) b) metastases of tumors in the pericardium.
4. Miksedeme.
5. Cholesterol.
6. In sarcoidosis.
7. In infectious mononucleosis.

C. Pericarditis probably associated with hypersensitivity or autoimmune.

1. Rheumatoid.
2. Collagen vascular diseases a) systemic lupus erythematosus, b) in rheumatoid arthritis, scleroderma.
3. Caused by drugs a) novokainamidom, b) hydralazine, c) other.
4. After an injury or damage to the heart a) after myocardial infarction (Dressler's syndrome).

Examination:

Laboratory studies — conducted to establish the cause and nature of pericarditis. For this purpose, a total analysis — may be signs of inflammation in the peripheral blood, specific immunological and biochemical studies of blood following indicators: total protein and its fractions, sialic acid, fibrinogen, urea.

ECG — with pericarditis reduces the overall electrical activity of the heart and recorded a sharp decrease in voltage.

Chest X-ray — chest radiograph is determined by the increase in size and change in the normal configuration of the heart, which can be configured as "bottles of water." If the shell heart deposited calcium salts, they form a "crustacean heart", which is clearly visible on the radiograph, in the form of lime on the pericardium.

Computed tomography (CT) — This method determines the thickening and calcification (deposition of calcium salts) in the pericardium.

Pericardiocentesis — with the pericardial introduced needle and take part of the accumulated liquid to research and determine the nature of the fluid (pus, tumor cells, fungi, tuberculosis and others.)

Echocardiography — the most effective and affordable method of diagnosis. In the presence of a small pericardial effusion detected relatively free

from the echo back of the space between the pericardium and the posterior part of the left ventricular epicardium.

Diagnostic criteria for the various forms of pericarditis

1. Acute fibrinous (dry), the initial phase of exudative clinically:
• Pain in the heart and / or abdomen.
• Pericardial rub. In some cases, there are no.
2. Acute exudative (exudative):
• Dull pain in the heart, shortness of breath.
• Tachycardia.
• Forced position of the patient.
3. Cardiac tamponade:
• Anxiety, fear of the patient.
• Strengthening of dyspnea and tachycardia.
• Acrocyanosis, cold sweat.
• Fainting.
• Clinical death.
4. Chronic adhesive, without compression of the heart:
• Weakness, fatigue.
• Pain in the heart with a load.
• Pericardial rub.
5. Chronic adhesive, with compression of the heart (constrictive):
• Acrocyanosis.
• Weakness, fatigue.
• Poor tolerance to physical and emotional stress.
• Pain in the right upper quadrant.
• Puffy face.
• Swelling of the neck veins.
• Increase in liver.
• Focus II tone of the pulmonary artery.
• Abnormal tone III.

Treatment

1. Basic principles of treatment hospitalization and follow-up (blood pressure, central venous pressure, heart rate), echocardiographic monitoring of hemodynamic parameters):

— Limitation of physical activity, bed rest;

— NSAIDs;

— Glucocorticoids mainly with intensive pain syndrome (dry pericarditis), allergic and autoimmune pericarditis and pericarditis, developing on the background of diffuse connective tissue diseases;

— Antibiotics only when purulent pericarditis and pericarditis, developed on the background of severe bacterial infections (sepsis, pneumonia, etc.).

2. When purulent pericardial effusion, in addition to parenteral administration of antibiotics is shown pericardiocentesis with maximum removal of fluid, washing the cavity and the re-introduction of antibiotics in the pericardial cavity (through the catheter).

3. cardiac tamponade shows emergency (life-saving) pericardiocentesis with the removal of exudate.

4. In constrictive pericarditis — subtotal perikardektomiya.

Myocardial dystrophy. Plan examination of the patient in an outpatient setting, diagnostic criteria. Principles of treatment on an outpatient basis.

Myocardial dystrophy — inflammation of the myocardium characterized by degeneration of the contractile cells of the heart muscle, cardiac conduction system structures and manifested symptoms of the main functions of the heart (automaticity, contractility, conductivity, excitability).

Myocardial dystrophy causes are exposure to bacterial toxins (chronic tonsillitis), industrial poisons, ionizing radiation; overdose of some drugs (alupenta, astmopenta et al.); chronic alcoholism; Prolonged deficiency in the diet necessary for the body of a number of substances (proteins, vitamins). Excess or deficiency of any biologically active substances underlies myocardiodystrophy developing in many non-communicable diseases. The so-called thyrotoxic heart — a consequence of severe myocardial dystrophy, arising under the influence of excess blood thyroid hormone, and myocardial hypothyroidism due to the lack of these hormones. Hormone also occurs when myocardial pathological climax. After elimination of the causative factor of traffic affected cardiomyocytes can fully recover, but if it works for a long time, some of these cells are killed and replaced by connective tissue, formed kardiosklerosis. Clinically manifested symptoms of myocardial contractility (shortness of breath, tachycardia, often mild and occur only during physical exertion), automaticity, conduction and excitability of the heart, which is reflected in a variety of arrhythmias and cardiac conduction disturbances (especially frequently found arrythmia), detected on ECG or auscultation.

Plan examination of the patient:

X-ray examination of the patient reveals the expansion of the boundaries of the heart to the left, congestion in the lungs.

ECG: ST-segment lower contour at least 1 mm in any two leads, U wave is distorted, wrong orientation and deformation of the T wave in at least two leads.

Echocardiography reveals the expansion of the ventricles and atria, changing the structure of the muscle tissue of the heart.

Biochemical blood analysis reveals a high level of lactate, pyruvate.

To assess the degree of dystrophic changes in the cardiac muscle cells determine the enzyme activity of mitochondria cells.

Treatment regimen necessarily implies the exclusion of physical activity, occupational exposures, exposure to household chemicals, alcohol and smoking.

Apply vitamin, electrolyte metabolism violations are corrected, restored trophic processes in the heart muscle.

Drugs used intravenously potassium 5 % glucose solution or physiological saline. 1 g potassium chloride per 500 ml of solution. To reduce the excitability of the heart muscle is used β-blockers. Used sedatives. To stimulate metabolism in the myocardium is used Riboxinum, preduktal, inosine, anabolic steroids, vitamins of group B. If necessary, use drugs, eliminating the signs of heart failure and rhythm regulation. Cardiac glycosides are used in dosages that are slightly smaller than standard full therapeutic to reduce their toxic effects on the cells of the heart. Dosages standard anti-arrhythmic drugs. Cordarone IV 5 mg / kg, per os, or 6 mg per day for the first seven days at 4 mg per day, the next week, and 2 mg per day thereafter.

Acute rheumatic fever. Chronic rheumatic disease. Plan examination of the patient in an outpatient setting, diagnostic criteria. Principles of treatment on an outpatient basis.

Acute rheumatic fever (ARF) — Post-complication of tonsillitis (angina) and pharyngitis caused by b-hemolytic streptococcus group A (BGSA), a systemic inflammatory disease of connective tissue with predominant localization in the cardiovascular system (carditis), joints (arthritis migrating), brain (chorea), and skin (erythema annulare, rheumatic nodules) that develops in susceptible individuals, mostly young children (7–15 years old), due to an autoimmune response to antigens of streptococcus organism and similar cross-reactivity with the targeted tissue autoantigens person (the phenomenon of molecular mimicry).

Five main *diagnostic criteria*: migratory polyarthritis, carditis, chorea, annular erythema, rheumatic nodules.

Carditis — leading syndrome of acute rheumatic fever (90–95 % of cases), which determines the severity and outcome of the disease. Carditis is considered a fundamental component of valvulitis. The most important feature valvulita at the first attack of rheumatic fever — a clear positive trend under the influence of active antirheumatic therapy. ARF as a problem of great social significance determined acquired rheumatic heart disease who progression lead to permanent disability and premature death.

Rheumatoid arthritis (60–100 % of cases) — migratory polyarthritis mainly large and medium-sized joints (knee, ankle, at least — elbow, shoulder, wrist). As a rule, combined with carditis and less (10–15 % of cases) occurs in isolation. The predominant form of defeat in recent years — oligoarthritis, at least — monoartrit. Has a rapid complete regression of inflammatory changes in the joints under the influence of anti-inflammatory therapy. In 10–15 % of cases detected only ***arthralgia*** (migratory pain in large joints of varying intensity), which, unlike the arthritis is not accompanied by tenderness and other symptoms of inflammation.

Rheumatic chorea (chorea, Sydenham's chorea) is diagnosed in 6–30 % of cases, mainly in children, rarely in adolescents. More common in girls and

young women. The main clinical manifestations — this pentad syndromes observed in different combinations:

•·Chorea hyperkinesis.

• Muscular hypotonia (up to sagging muscles with simulated paralysis).

• Disorders of statics and coordination.

•·Dystonia.

• Psycho-emotional violations.

Annular (of annular) erythema observed in 4–17 % of sick children at a height of attack of rheumatic fever. Characterized by pale pink ringed rash diameter from a few millimeters to 5–10 cm preferentially localized on the trunk and proximal extremities (but not on the face!). It has a transient migratory character, does not rise above the level of the skin, is not accompanied by itching or induration, pales when pressed quickly disappears without residual effects (pigmentation, peeling, atrophic changes).

Subcutaneous rheumatic nodules in recent years are very rare (1–3 %). This rounded dense sedentary painless formation of various sizes on the extensor surfaces of the joints in the ankles, Achilles tendons, the spinous processes of the vertebrae, the occipital region gallea aponeurotica with reverse cycle development from 2 weeks to 1 month. Chronic nonspecific (arthralgia and fever) and laboratory syndromes (increased protein island. Inflammation, increased erythrocyte sedimentation rate and C-reactive protein, as well as PQ prolongation in the *ECG*), relate to "***small" diagnostic criteria***. The presence of two big criteria or one large and two small, combined with the data, document confirming previous BGSA infection (culture from the throat or strep antigen test, or increased streptococcal antibodies), indicates a high probability of rheumatic fever. Repeated attacks in patients with a history of rheumatic considered as a new episode of rheumatic fever, but is not the first relapse. Under these conditions (especially against the background formed heart disease when diagnosis of carditis largely difficult), a presumptive diagnosis of rheumatic fever can be re-made on the basis of the "big" or just a "small" criteria in combination with elevated or rising titers of antristreptococcal antibodies.

Classification

Two variants of the disease outcome.

When it comes to the ***convalescence*** of the full regression of clinical symptoms of rheumatic fever with normalization of laboratory values and the absence of any permanent change.

Under ***chronic rheumatic heart disease*** refers to a disease characterized by lesions of the heart valves in the form of post-inflammatory fibrosis edge of the valve leaflets or heart disease (insufficiency and / or stenosis), formed after undergoing ARF. Chronic heart failure (CHF) is estimated in accordance with the classifications Strazhesko – Vasilenko (degree) and the New York Heart Association (functional class).

Plan evaluation of patients:

When ARF:

1. Complete blood count.
2. Urinalysis.
3. Biochemical blood tests: determination of bilirubin, urea, C-reactive protein, antistreptolisin-(hereinafter ASL-O, potassium aspartate aminotransferase activity (hereinafter AST), alanine aminotransferase (ALT hereinafter).
4. Echocardiogram.
5. ECG.
6. X-ray of the chest cavity.
7. Consultation otolaryngologist.

In chronic rheumatic heart disease:

1. Complete blood count.
2. Urinalysis.
3. Biochemical blood tests: determination of the concentration of total protein, bilirubin, CRP; potassium, urea, AST, ALT, ASL-O.
4. Study of hemostasis: the definition of the calculation PTV INR (when using anticoagulants).
5. ECG.
6. X-ray of the chest cavity.
7. Test 6-minute walk.
8. Measuring blood pressure (profile); Heart Rate.
9. Control of body weight.

Treatment and prevention

The goals of treatment:

• Eradication BGSA.
• Inhibition of the inflammatory process.
• Caution in patients with carditis carry-forward formation of heart defect.
• Compensation for congestive heart failure.

I. Causal treatment.

Benzylpenicillin is used for 10 days in adults 500000–1000000 units 4 times a day intramuscular. Subsequently moving to penicillin depot secondary prevention mode. Intolerance to penicillins or macrolides used lincosamides.

II. Anti-inflammatory therapy.

Glucocorticoids are used in FRA flowing with severe carditis and / or polyserositis. Prednisolone is prescribed to adults at a dose of 20 mg / day in 1 reception in the morning after a meal to achieve a therapeutic effect (on average within 2 weeks). Then gradually reduce the dose (2.5 mg every 5–7 days) until the total abolition. The total duration of the course is 1.5–2 months.

NSAIDs (diclofenac usually) prescribed for weakly expressed valvulitis, rheumatoid arthritis without valvulita, minimal activity of the process (ESR < 39 mm / h). Diclofenac prescribed for adults 25–50 mg 3 times a day, 3 times a

day before the normalization of inflammatory activity (average for 1.5–2 months). If necessary, treatment with diclofenac can be lengthened up to 3–5 months.

Therapy for congestive heart failure:

• Diuretics: loop — furosemide; thiazide and thiazide — hydrochlorothiazide, indapamide; sparing — Spironolactone, triamterene.

• Calcium channel group of a long-acting dihydropyridines (Amlodipine). B-blockers (carvedilol, metoprolol, bisoprolol).

• Cardiac glycosides (digoxin).

PRIMARY PREVENTION ARF: Basis of primary prevention — antimicrobial therapy of acute and chronic recurrent BGSA — upper respiratory tract infections (tonsillitis and pharyngitis).

Secondary prevention ARF: The goal — prevention of repeated attacks and disease progression in patients who have had ARF. Secondary prevention starts back in the hospital immediately after antistreptokokkovoy causal therapy. for patients with healed carditis without heart defect — at least 10 years after the attack, or up to 25 years of age (according to the principle "that the longer"); for patients with heart disease formed (including the operated) — for life.

TOPIC 3: Articular syndrome: differential diagnosis. Diagnosis and treatment of inflammatory diseases (rheumatoid arthritis, reactive arthritis) and degenerative (primary osteoarthritis) joint disease in an outpatient setting, medical tactics, medico-social examination, clinical examination, primary prevention.

Articular syndrome: the concept of arthritis, arthrosis, arthropathy. Methods of general physical and technical study of the joints and spine. Major diseases accompanied by articular syndrome. Diagnostic algorithm for searching the articular syndrome.

Articular syndrome — a characteristic symptom is manifested by pain in the joints, their deformation and deformation limitation of joint movement, changes in tendon and ligaments of the joints surrounding muscles. The pathogenesis of articular syndrome are inflammatory or degenerative changes in the joints and o apparatus, in mild cases of arthralgia syndrome is manifested only.

Joint pain are constant symptom in rheumatic diseases. In the event of pain, their initiation factors play a role of mechanical overload of the joint, tendon-stretching ligaments, synovial membrane irritation; microcirculatory disorders; metabolic disorders in the skeleton, the joint development of inflammatory and degenerative changes. As a consequence of these processes in the tissues of the joints accumulate substance analgeics (tissue proteases, kinins, prostaglandins, histamine, serotonin), which irritate pain receptors and give rise to pain reflex arc. Nociceptors are in adventitial microvessels, fibrous joint capsule, periosteum of bones, ligaments and tendons. They are not in the synovium, cartilage and meniscus. Required parameters are found out joint pain, exact location, nature, duration, intensity, time of occurrence during the day.

Second subjective symptom — *restriction of joint movement*. Degree of expression of this trait is usually directly proportional to the severity of the organic and functional changes in the joints.

The objective indicators of joint damage include defiguration and deformation of the joints, swelling, redness of the skin over the joints, dysfunction of joints.

Defiguration joint (or joints) — a change in the shape of the joint due to the inflammatory swelling of the synovial membrane and periarticular tissues, joint effusion, synovial hypertrophy and fibrotic and sclerotic changes in periarticular tissues.

Deformation of joints — a persistent change in the shape of the joints due to bone changes, development of ankylosis, subluxations.

Swelling in the joint can be both in these states. Redness of the skin over the affected joints due to a local increase of skin temperature and evidence of active inflammation in the joint. On examination and palpation of the affected joints tentatively set limit range of motion specific to the joint. Estimated restriction of active and passive movements of the joints.

Osteoarthritis — a disease of the joints, is very widespread in the world. More correct name of osteoarthritis — Osteoarthritis. The main symptoms of osteoarthritis: severe pain in the joints, decreased mobility of the joint. In case there is a neglect of osteoarthritis joint stiffness. The main difference between osteoarthritis arthritis: osteoarthritis at the main destructive activities performed not inflammatory and degenerative processes in the articular cartilage.

Arthritis — an inflammation of the joint. Observed in arthritis pain when moving or lifting heavy objects, the joint loses mobility, swelling, changes shape, the skin over the joint is red, there is a fever. Arthritis o one joint known as monoarthritis, many — polyarthritis. Arthritis can start sharply (acute arthritis) or develop gradually (chronic arthritis). Causes of Arthritis — infections, allergies, malnutrition. Arthritis can be a separate disease or a manifestation of another disease.

Arthropathy — is a secondary joint damage against various pathological processes. Can be both inflammatory and degenerative-dystrophic. General *features arthropathy* are:

• asymmetry of destruction;

• dependence of the clinical picture of the joints syndrome clinic underlying disease;

• a positive trend during the treatment of the underlying disease;

• scarcity of X-ray pictures (joint space narrowing, signs of ankylosis).

The reasons are:

I. Birth:

— Achondroplasia.

— Ehlers.

— Danlos syndrome.

— Marfan syndrome.
— Osteogenesis imperfecta.

II. Acquired:

1. Infectious.
2. Inflammatory: ex. Rheumatoid arthritis.
3. Degenerative: Osteoarthritis.
4. Enteropathic: Acute gastrointestinal infections.
5. Endocrine: Acromegaly.
6. Metabolic: Gout.
7. Neuropathic: Joints Charcot (diabetic arthropathy).
8. Hematologic: Hemophilia.
9. Medication: - Anticoagulants – Hormones.
10. Neoplastic.

When **Ehlers Danlos-marked** congenital developmental disorder of connective tissue (collagen structures), which manifests itself hyperelastic vulnerability and skin laxity of the joints.

In patients with **Marfan syndrome** develop laxity of joints. They complain of pain in the joints.

In patients with **osteogenesis imperfecta** (brittle bones) easily occur dislocations of joints.

In **septic arthritis** usually affects one joint, there is flushing of the skin, swelling, joint pain and limited mobility. Joint disease is caused by hematogenous spread of infection, but can spread to the adjacent focus of osteomyelitis. Etiological factors are Staphylococcus aureus, gonococcus, agents of brucellosis and typhoid. In a patient with **tuberculous arthritis** is usually observed swelling and limitation of movement in the joint. Unlike septic arthritis, tuberculous not marked flushing of the skin and the local temperature rise in the joint. The cause of **rheumatoid arthritis** is a streptococcal infection, such as scarlet fever and tonsillitis. The patient observed migrating joint pain, along with symptoms of heart disease, erythematous rash on the skin and the formation of subcutaneous nodules.

Rheumatoid arthritis. Morning stiffness in the joints for more than 30 minutes. Periarthritis. Less frequently oligo- and monoartrit. Affects the small joints of the hands and feet — metacarpophalangeal, proximal interphalangeal. During an exacerbation, and as the disease progresses pronounced deformation of joints, the violation of their functions. Typically, due to the absence of the infection.

Rheumatic fever. Articular manifestations appear in 2.5–3 weeks after a sore throat, pharyngitis. Affects the large joints, characterized by volatility, symmetrical lesions remarkably rapid effect of aspirin and other NSAIDs. No joint disability.

Reactive arthritis. There is a clear link with the infection — urogenous, enterogenous, tonzilogenous. Joint damage by type of mono- or oligoarthritis, often signs of sacroiliitis. Defeat is often the nature of the ascending (upward). Severe joint deformity not. A marked beneficial effect of antibiotics and NSAIDs.

Reiter's syndrome. Triad signs — poly-oligoarthritis, conjunctivitis, urethritis.

Ankylosing spondylitis. The defeat of the axial skeleton with Ankylosing spondylitis predominates over the involvement of the peripheral joints, and the joints are affected predominantly "cartilage" type — the sacroiliac joint, small intervertebral joints, sternoclavicular and edge-sternal articulation. Inflammation of the joints caused by immunological mechanisms.

Gout. Recurrent arthritis, especially the defeat of 1 metatarsophalangeal joint (often, but not always) caused by a metabolic disorder of purine metabolism.

Infectious arthritis specific. In the anamnesis for tuberculosis, gonorrhea. Predominantly asymmetric mono — oligoarthritis.

Psoriasis. The defeat of the distal interphalangeal joints mainly hands (fingers in the form of sausage or radishes). There are signs of sacroiliitis.

Deforming osteoarthrosis. Affects mainly the large joints, pain aggravated by stress. Can be expressed deformation of joints, re - signs of synovitis.

When ascertaining signs of articular syndrome as a primary diagnostic hypotheses (PDH) can be hypothesized three groups of diseases:

1) actual joint disease, when the clinical picture, there are signs only articular syndrome;

2) diffuse connective tissue disease, when along with articular manifestations are signs polysystemic destruction;

3) systemic vasculitis.

Research methods at the articular syndrome include functional methods — imaging and histomorphological methods.

Goniometer — an objective assessment of motor function of the joints, which is made by measuring the angles of various directions of movement in the joint.

Rheumatoid factor — this immunoglobulins specific to the site Fs- IgG, an indicator of rheumatoid arthritis. Of the various isotypes rhematoid predominate factor isotype IgM. The main disease in which the rheumatoid factor detected is rheumatoid arthritis (but there are also so-called seronegative variant of the disease, in which the rheumatoid factor is not determined). In addition, increased levels of rheumatoid faktor may be present in systemic lupus erythematosus and some other connective tissue, hepatitis, infectious mononucleosis.

CCPA (antibodies to cyclic peptide tsitrullinovomu) — a marker for early diagnosis of rheumatoid arthritis. Citrulline is not a standard amino acids include proteins during their synthesis, it is formed by the subsequent modification of arginine. Determination of antibodies to cyclic tsitrullinovomu peptide (anti-CCP) has the highest value among the new immunological methods. This method has now become the second standard immunological tests after determining factor for the diagnosis of rheumatoid revmotoidnogo arthritis (RA). antibodies to cyclic peptide tsitrullinovomu appear in serum for 1 year prior to disease onset (its sensitivity at the early stage of the disease up to 75 %).

LE-cells (SLE) is most often found in the blood of patients with systemic lupus erythematosus (SLE) and less at other connective tissue. Typical LE-cell —

is segmented neutrophilic granulocyte (less eosinophilic granulocyte or monocyte), the core of which sidelined as a crescent on the periphery of the cell, and its center takes phagocytosed, homogeneous mass of a round shape. LE-cell is much larger than normal white blood cells.

Antinuclear antibodies. One common screening tests used in the diagnosis of systemic lesions of connective tissue.

In the differential diagnosis of articular syndrome **presence HLA-B27** is a characteristic feature spondyloarthritis: This allele is present in 90–95 % of patients with ankylosing spondylitis have 60–90 % — with reactive arthritis, 50 % — with psoriatic arthropathy and 80–90 % — with juvenile ankylosing spondylitis.

The study of **synovial fluid** allows to differentiate degenerative and inflammatory diseases of the joints, in some cases allocate a specific disease entity. Synovial fluid obtained by arthrocentesis. It is estimated by several parameters: color, viscosity, transparency, the nature of the mucin clot and cytological structure.

X-rays of the joints — one of the most informative methods of imaging studies in patients with articular pathology. Thus it is necessary to take into account the stage of development process.

Table 7 — The most important and typical radiographic changes at the articular syndrome

Radiographic signs	Disease
Marginal bone erosion epiphyses	Rheumatoid arthritis
Osteolysis of the distal phalanges	Psoriatic arthropathy
Subchondral osteosclerosis, osteofity	Deforming arthrosis
Destructive arthrosis, "punches"	Gout
Sacroiliitis, calcification of ligaments of the spine	Ankylosing spondylitis

Ultrasound examination of the joints allows you to visualize soft tissues such as muscles, tendons, ligaments, cartilage, which is not available on conventional imaging X-ray, to see only the bone structure.

Radionuclide scintigraphy joints performed using radiopharmaceuticals labeled osteotropic 99 mTc. These drugs are taken up at sites of active bone and collagen metabolism.

Arthroscopy — a direct visual examination of the joint cavity. It allows you to install inflammatory, traumatic or degenerative menisci, ligaments, cartilage lesions, synovial membrane.

Synovium biopsy carried out in two ways — by puncture of the joint or during arthroscopy.

Rheumatoid arthritis: survey design, diagnostic criteria. Medical tactics, the general principles of treatment on an outpatient basis.

Rheumatoid arthritis — a chronic, systemic inflammatory autoimmune disease of the connective tissues, mainly affecting the joints of the type of erosive-destructive progressive polyarthritis. The disease affects 0.5–1 % of the population.

Etiology:

1. Genetic factors. Proved a close correlation between the development of RA and antigens of histocompatibility HLA DR, DR4.

2. Infectious agents. This virus Epstein-Barr virus, retroviruses, rubella, herpes, cytomegalovirus, Mycoplasma, and others.

Risk factors for RA

— female — age 45 years and older;

— genetic predisposition;

— presence aforementioned HLA-antigens;

— comorbidities (nasopharyngeal infection, congenital defects of bones and joints).

Pathogenesis

The pathogenesis of RA are genetically determined autoimmune processes, which contributes to the emergence of a deficit of T-suppressor function of lymphocytes. Unknown etiological factor causes the development of an immune response. Damage to the joint begins with inflammation of the synovium (synovitis), then acquiring proliferative in nature (pannus) with the damage of cartilage and bone. The intensity and type of clinical inflammatory immune response genes is determined. The synovium is infiltrated by T lymphocytes DM + (helper), plasma cells, macrophages. The interaction of macrophages and T lymphocytes CD + (helper) launches an immune response. Macrophages in conjunction with Class II molecules, HLA-system - DR. are hypothetical antigen to T-helper T lymphocytes, leading to their activation. Activated T-helper cells stimulate B-lymphocyte proliferation, and their differentiation into plasma cells. Plasma cells produce a modified aggregate synovitis G Ig. In turn, it is recognized by the immune system as a foreign antigen, plasma and synovial cells, lymph nodes, spleen begin to produce antibodies thereto — Rheumatoid factors (RF). The most important is the Russian class IgM, which is found in 70–80 % of patients with RA. Also proved the existence of other types of RF - IgG and IgA. In determining the levels of the classical RF IgM say seropositive RA version.

RF can be detected in healthy individuals (in the credits, not exceeding 1:64), SLE, Sjogren's syndrome, hematological malignancies, tumors.

The clinical picture

The defeat of the hand joints. Most often it is the second or third metacarpophalangeal, proximal interphalangeal joints. Affected joints swell, movement in them painful. Later formed various subluxations, in particular ulnar deviation of the hand (the deviation of the fingers in the direction of the ulna). ***Rheumatoid nodules*** are observed in 20–25 %. This dense connective tissue rounded education to 1.5–2 cm, painless, mobile, in rare cases soldered to the aponeurosis.

The defeat of the lungs and pleura. The most frequently detected or fibrinous pleural effusion.

Heart disease. Pericarditis most adhesive, high activity of the process — exudative. Features of the flow of rheumatoid myocarditis are torpid course, a positive trend with a decrease in the activity of RA under the influence of glucocorticoids. Endocarditis can lead to the formation of heart defects.

Kidney damage in the form of glomerulonephritis or amyloidosis — is the most severe visceral manifestation of RA.

Nervous system: peripheral ischemic neuropathy, polyneuritis, compression neuropathy, functional disorders of the autonomic nervous system, encephalopathy.

Seronegative rheumatoid arthritis disease called variant in which the rheumatoid factor is not detected in any serum or synovial fluid. This option RA occurs in 20 % of patients and has the following features: Begins more acute than seropositive RA, Pathological process begins with the destruction of wrist and knee joints, but within 6 months occurs generalization of the disease and others involved joints. The defeat of the large joints in the form of monoarthritis in the early asymmetric, symmetric polyarthritis in the future. Characterized by early and heavy defeat of the hip joints; ischemic necrosis of the femoral head occurs 10 times more frequently than in seropositive variant of the disease. Develop early contractures of the elbow and knee joints due to severe fibrotic changes in them.

Clinical stage:

• Very early stage: the duration of the disease for at least 6 months.

• Early stage: the duration of the disease from 6 months to 1 year.

• The developed stage: the duration of the disease more than 1 year in the presence oftypical symptoms of RA.

• Late stage: the duration of the disease 2 years or more severe degradation of the small + (III-IV stage of the X-ray) and large joints.

Criteria for the classification of rheumatoid arthritis (RA), established in 2010 by the American College of Rheumatology / European League Against Rheumatism. Patients to be examined by the algorithm:

1. Patients who have at least one affected joint, with clinically established synovitis.

2. If no other diagnosis explaining diagnosis synovitis.

RA classification criteria are based on the calculation of points (scoring algorithm).

A. Affected joints

Derived factor for RA factors:

1 — large	0
2–10 — large joints	1
1–3 — small joints (regardless of large joints)	2
4–10 — small joints (regardless of large joints)	3
> 10, including at least one small	5

B. Serological tests (for classification requires that at least 1 study

Negative test results: RF and anti-CCP	0
Weak positive test result: RF or anti-CCP	2
High positive test result: RF or anti-CCP	3

C. Acute-phase response (for classification requires that at least 1 study)

Test results: CRP and ESR (normal)	0
Test results: CRP or ESR (above normal)	1

D. Duration of symptoms

< 6 weeks	0
> 6 weeks	1

Rheumatoid arthritis is confirmed, if the sum of the coefficients of the factors RA ≥ 6.

Categories joints in RA criteria ACR / EULAR 2010.

Joints exceptions:

— Does not take into account changes of the distal interphalangeal joints, I carpometacarpal joints and I metatarsophalangeal joints.

Large joints:

— Shoulder, elbow, hip, knee, ankle.

Small joints:

— Metacarpophalangeal, proximal interphalangeal, II–V metatarsophalangeal, interphalangeal joints of the thumb, wrist joint.

Other joints:

— Joints that may be affected in RA, but not included in any of the above groups (eg, temporomandibular, acromioclavicular, sternoclavicular, and others).

Step rheumatoid arthritis by chest X-ray:

Stage I — osteoporosis in destructive X-ray changes.

Stage II — a minor destruction of cartilage and bone, a slight narrowing of the joint space, single Uzury bones.

Stage III — extensive destruction of cartilage and bone, marked joint space narrowing, multiple Uzury, subluxations.

Stage IV — the symptoms of stage III, accompanied by ankylosis.

The degree of activity of rheumatoid arthritis

a) 0 degree — inactive rheumatoid arthritis. The absence of pain in the joints. The absence of exudative phenomena. Temperature, ESR, C-reactive protein, sialic acid, fibrinogen is normal.

b) The degree of I — minimal activity. Small joint pain and stiffness in the morning a little. Minor exudative phenomena in the joints. Body temperature above normal joints. Slightly elevated ESR (within 20 mm / h), the amount of normal leukocytes. Indicators of C-reactive protein and fibrinogen, sialic acid slightly increased.

c) Grade II average activity. Pain in the joints, not only during movement, but also alone. Stable exudative phenomena in the joints (swelling, effusion, bursitis).

Expressed painful restriction of mobility in joints. The defeat of the internal organs are not clearly expressed. Low-grade fever. ESR of 30–40 mm / h, leukocytes — 000 8000-10 1 mm3. A marked increase in C-reactive protein, fibrinogen.

d) Construction III — high activity. Severe morning stiffness. Severe pain at rest. Expressed exudative phenomena in the joints (significant swelling, redness and increased temperature of the skin over the joints). Marked limitation of mobility. Symptoms of active inflammatory process in the internal organs (pleurisy, pericarditis, myocarditis). Body temperature is high. A significant increase in ESR (40–60 mm / h). Leukocytes of 15 000–20 000 in 1 mm3. A significant increase in C-reactive protein, fibrinogen.

The activity of the disease:

• 0 — remission (DAS28 < 2,6);
• 1 — low (2,6 < DAS28 < 3,2);
• 2 — average (DAS28 = 3,2–5,1);
• 3 — high (DAS28 > 5,1).

index DAS28:

DAS28 = 0,56VNPJ+0,28VNSJ+0,701n ESR +0,014 OAH,

where NPJ — the number of painful joints; NSJ — the number of swollen joints of the following 28: shoulder, elbow, wrist, metacarpophalangeal, proximal interphalangeal, knee; ESR — erythrocyte sedimentation rate by Westergren method; OAH — an overall assessment of the health of patients in millimeters on a 100-mm visual analog scale.

You can use other methods of calculating the activity, which proved a good compatibility with the DAS28.

Functional disorders of the musculoskeletal system:

I — completely self-maintained, non-professional and professional activities;

II — maintained self-service, professional work, unprofessional activity is limited;

III — maintained self-service, limited to non-professional and professional activities;

IV — limited self-care, non-professional and professional activities.

Felty's syndrome — an option seropositive RA, more frequently in women, a measure of high activity. It can be explained by the appearance of antibodies to peripheral neutrophils with the secondary response in the spleen, there is a circulating inhibitor of granulocytes, which leads to neutropenia (leukopenia). High titer RF, IgG, IgM much. Often accompanied by necrotizing vasculitis. A characteristic symptom — necrotic leg ulcers. Always there splegomegaliya, mozhert be hepatomegaly. Maybe polyneuropathy, episcleritis, articular syndrome with rapid progression. It requires aggressive therapy. It is necessary to differentiate with drug agranulocytosis.

Adult Still's disease — a disease in which joints are involved, along with the skin on the type of vasculitis, there is high fever with chills, marked leukocytosis.

Plan evaluation of patients:

Required:

1. Complete blood count — signs of moderate normochromic anemia, leucopenia. The most important and regularly changing indicator — increased ESR.

2. Urinalysis.

3. Biochemical blood tests: determination of bilirubin, urea, glucose, total protein, potassium, CRP; AST, ALT.

4. Immunological study of blood: determination of the concentration of RF; CCPA.

5. Investigation of serum markers of viral hepatitis (HBsAg; HBc-core; and / HCV-once before the appointment of cytotoxic immunosuppressants).

6. X-ray joints of the hands, feet; other affected joints.

7. X-ray of the chest cavity.

8. ECG.

Supplementary

1. Biochemical blood tests: determination of the concentration of creatinine, alkaline phosphatase, ferritin in serum; cholesterol, lipid profile.

2. Identification of markers of bone remodeling: osteocalcin, ionized calcium.

3. Investigation of the synovial fluid.

4. Determination of DNA fragments Chl. trachomatis in the synovial fluid by PCR.

5. Echocardiogram.

6. Ultrasound of the abdomen.

7. EGD.

8. X-rays of the cervical spine (for suspected subluxation of the atlanto-occipital joint).

9. MRI of the cervical spine.

Treatment

I. Preparations of a standard basic treatment as recommended by the European League Against Rheumatism against 2010:

1. Methotrexate.

2. Sulfasalazine.

3. Leflyunamid ("Arava").

4. Gold salts (Tauredon).

5. Hydroxychloroquine (Plaquenil).

6. Azathioprine.

II. Preparations biological Genn - Engineering therapy:

1. Infliximab, adalimumab.

2. Rituximab.

3. Tocilizumab.

1. Pathogenic drugs

1.1. Antimetabolites ***methotrexate*** 7,5–25 mg / week. inside, n / a; / m -is the gold standard in the treatment of RA or

1.2. Anti-inflammatory agents, acting on gut (aminosalicylic acid): ***sulfasalazine*** 1.5–3 g / day orally (if any side effects or intolerance to methotrexate) or

1.3. Selective immunosuppressants: ***leflunomide*** 20 mg / day orally (in ineffectiveness / intolerance to methotrexate, sulfasalazine) or

1.4. Antiprotozoal drugs ***hydroxychloroquine*** 200 mg / day orally (in case of intolerance to methotrexate, sulfasalazine, leflunomide activity combined with low RA).

2. ***Folic acid*** 1–3 mg / sut.vnutr (when methotrexate is days of his administration). Methotrexate inhibits the production of the active form of folic acid — tetrahydrofolate, so for the prevention of anemia folievodefetsitnoy necessarily need its reception.

3. ***NSAIDs***: aceclofenac 200 mg / day orally or diclofenac 100–150 mg / day orally or / m, or ibuprofen 1200–2400 mg / day orally, or meloxicam 15 mg / day PO / m, or nimesulide 200 400 mg / day orally or celecoxib 400 mg / day orally or etodolac 600–1200 mg / day orally.

4. ***Ingibitory proton pump*** (risk factors for GI complications): omeprazole 20–40 mg / day or rabeprazole 20–40 mg / day or lansoprazole 30–60 mg / day or pantoprazole 20–40 mg / day or esomeprazole 20–40 mg / day.

4.1. Inhibitory proton pump (risk factors for GI complications): omeprazole 20–40 mg / day or rabeprazole 20–40 mg / day or lansoprazole 30–60 mg / day or pantoprazole 20–40 mg / day or esomeprazole 20–40 mg / day.

5***. Corticosteroids*** for systemic use:

5.1. Prednisolone 5–10 mg / day (methylprednisolone 4–8 mg / day) inside — with the development of systemic manifestations of the disease; to the effect of the use of drugs of pathogenic therapy for 3 months (extinction / reduce pain, stiffness, normalization of CRP, ESR), followed by a gradual decrease to total abolition — on 0,125–0,5 mg / day of prednisone (1.2 mg / day methylprednisolone) per week.

5.2. Prednisolone — 10–40 mg / day (8–32 mg methylprednisolone / day) orally for systemic manifestations, special syndrome as a continuation of inpatient therapy 1–1.5 months before the onset of the effect of the use of drugs in the pathogenetic therapy within 3 months (extinction / reduce pain, stiffness, normalization of CRP, ESR), followed by a gradual reduction in dose 0,125–0,5 mg / day of prednisone (1–2 mg / day Medrol) per week to support (5–-10 mg prednisolone / day (methylprednisolone 4–8 mg / day) for a long time.

5.3. Betamethasone 2.1 ml (5.10 mg) or triamtsinalon 40–80 mg or 40–80 mg methylprednisolone (intra no more 2–3 times / year in one joint, periarticular or / m on the testimony of a single dose after consulting rheumatologist).

6. Preparations for the treatment and prevention of ***osteoporosis***.

7. Massage.

Infliximab is a specially synthesized compound based on mouse and human antibodies. Once in the body, the antibodies bind to tumor necrosis factor alpha (TNF - α), which is assigned to one of the leading roles in the mechanisms of disease progression. Infliximab is administered to patients at an early stage and deploy RA. It is used in combination with methotrexate. The greatest effect was observed in the early stage RA. Indications: treatment failure with standard DMARDs, severe progressive course of RA. Lack of effect of the drug after

12 weeks is an indication to cancel it. Infliximab is administered in / veno drip using a filter at a dose of 3 mg / kg, then the drug is administered at the same dose 2 weeks and 6 weeks after the first injection, and then — every 8 weeks.

Rituximab (MabThera) is an antibody to a component B-lymphocytes (immune cells) — CD — 20 antigen. Rituximab inhibits the activity of B-lymphocytes. It is prescribed for patients with early and advanced stage in the case of RA drugs inefficiency standard basic therapy and tumor necrosis factor blockers, in severe RA. The drug is given intravenously at a dose of 1000 mg 2 infusion. The first infusion (500 mg) was carried out on the first day of treatment for 4–6 hours, and the second — on the 15-day treatment for 4 hours. Before each infusion intravenous 125–250 mg methylprednisolone / veno drip infusion to prevent these reactions, fever, itching, runny nose, cough.

Tocilizumab (ACTEMRA) — recombinant humanized monoclonal antibody against human interleukin-6 receptor (IL-6) from the immunoglobulin subclass IgG1. Tocilizumab selectively binds and inhibits both soluble and membrane IL-6 receptor (sIL-6R and mIL-6R). IL-6 is a multifunctional cytokine produced by various cell types involved in a paracrine regulation of systemic physiological and pathological processes, such as the stimulation of the secretion of Ig, T cell activation, stimulation of acute phase proteins in liver and stimulation of hematopoiesis. IL-6 is involved in the pathogenesis of various diseases, including inflammation, osteoporosis and tumors. The clinical effect of 20, 50 and 70 % according to the criteria of the American College of Rheumatology (ACR) at 6 months was observed more frequently in the treatment of tocilizumab 8 mg / kg than in the therapy with the comparison, regardless of the presence or absence of rheumatoid factor, age, sex, race toiletries, number of prior treatments or disease stage. Response to therapy arose quickly (already on the 2nd week), Intensified during the course of treatment and was maintained over 18 months. In patients treated with tocilizumab 8 mg / kg, significant improvements were noted in respect of all criteria AKP (number of tender and swollen joints, improving the overall evaluation of the effectiveness of treatment according to the doctor and the patient, the degree of functional disorders according to the questionnaire HAQ, assessment of the severity of pain, indicators of C-reactive protein) compared with patients receiving placebo plus methotrexate / basic anti-inflammatory drugs. Tocilizumab not only has a positive effect on joint inflammation and destruction, but also affect the extra-articular manifestations of RA. It is indicated for moderate to high activity of RA. It may be administered in combination with methotrexate or other DMARDs, or without them. It may be administered as a first-line drug. Tocilizumab is one of the best and most effective drugs for the treatment of RA. The effect develops in 2 weeks, growing throughout the course and lasts more than 18 months. According to studies of RA remission during treatment with tocilizumab 6 months achieved more than 30 % of patients a year — every second patient, and after 2 years — remission achieved two of the three patients.

The drug is administered at a dose of 8 mg / kg body massya (minimum 480 mg) was intravenously drip for one hour. Repeated infusions are held every 4 weeks.

In remission:

1) continuation of basic drugs;

2) spa treatment on the testimony;

3) vitamin (B vitamins and vitamins C, A, E);

4) massage, sauna, sanitation radical foci of infection, as a temporary measure (physiotherapy);

5) rational employment — to recommend the profession with limited physical activity, work outdoors;

6) diet, the regulation of body weight;

7) periodic unloading joints (walking with additional support, special tires to prevent deformation of the brush, contractures).

Reactive arthritis. Survey plan. Differential diagnosis. General principles of treatment on an outpatient basis.

Reactive arthritis (ReA) — "sterile" purulent arthritis developing in response to extra-articular infection, in which the alleged causative infectious agent does not stand out from the joint using conventional culture media. It is now proposed to refer to only ReA arthritis associated with intestinal and urinary infection and associated with HLA B27 antigen, wherein the rheumatoid factor in the serum is detected. etiology: Depending on the etiology of ReA distinguish two groups:

1. Post enterocolitis (pathogens: Yersinia, Salmonella, Shigella — dysentery bacillus, Campylobacter, Clostridium); yersiniosis at an acute ReA develops in 20 % salmonellosis — in 2–7.5 %, shigellosis — 1.5 %, with campylobacteriosis — less than 1 %.

2. Urogenital (agent: chlamydia, ureaplasma, as well as associated with HIV infection).

Pathogenesis: According to the theory of immuno ReA occurs in individuals with a genetic predisposition to result from excessive immune response to microbial antigens in the blood which circulate and persist in synovial fluid and articular tissues. When post enterocolitis ReA penetration of antigens in the blood, and then in the joints, helps to increase the permeability of the inflamed intestinal wall for antigens and microbes. When the microorganism Chlamydia ReA and thus its spread throughout the body antigens blood phagocytes. An important factor is also the pathogenic microbial mimicry (i.e. similarity microorganism antigens and HLA B27), whereby the immune response is directed against not only the microorganism but also against its own joint tissues.

Common diagnostic features of reactive arthritis:

1. Prior or simultaneously evolved urethritis or diarrhea.

2. The defeat of the eye (conjunctivitis, iritis).

3. Asymmetric arthritis of the lower limbs (usually affects the knees, ankles and joints of toes, and the process is mono- or oligoarticular character). The first clinical manifestation, as a rule, is urethritis. Somewhat later joined conjunctivitis, arthritis, skin and mucous membranes — this symptom triad of symptoms is called Reiter.

4. Frequent loss of the Achilles tendon and plantar fascia with severe pain.

5. Arthritis of the big toe, pain and swelling in the heel.

Clinical signs of osteoarthritis:

1. Pain in the joint / joints.

2. Stiffness — subjective feeling of obstacles movement, which is usually most pronounced immediately after waking or rest period of inactivity. Stiffness caused by violation of the outflow of fluid from the inflamed joint at rest, decreased or held with the resumption of joint movement.

3. Swelling — transient increase in size and change in the contour of the joint, caused by the accumulation of fluid in the joint cavity and edema periarticular tissues.

4. Increase the temperature of the joints is also a sign of inflammation. Determined by carrying out the back of his hand across the surface of the joint.

5. Painful joints palpation confirms that joint pain is caused by his defeat, and is not reflected.

6 The absence of RF in the blood.

ReA likely to complete regression for 4–6 months, but can recur and even acquire a chronic course with the involvement of an increasing number of joints.

Differential diagnosis of Re A. Differential diagnosis often have to spend with gonococcal arthritis, in which there is no eye disease, keratoderma, gonorrhea detected in the synovial fluid.

As a rule, it is easy to differentiate between Reiter's syndrome with rheumatoid arthritis in which arthritis is not preceded by urethritis, joint damage symmetric, positive tests for rheumatoid factor or anti-CCP. Psoriatic arthritis, Reiter's syndrome is characterized by a more gradual and less acute joint disease, the chronic nature of skin lesions, conjunctivitis and urethritis absence of rare and mucous membranes. Sometimes ulcerative lesions of the mucous membranes of the mouth and genitals Reiter's syndrome should be differentiated from Behcet's syndrome, in which ulcers are extremely painful. Diagnostic criteria of Yersinia infection — allocation stool culture and increasing titer of antibodies to Yersinia defined by PHA (diagnostic titer of 1: 160 and above); dysentery — stool culture isolation of Shigella, indirect hemagglutination reaction with standard erythrocyte diagnostic a titer of 1: 200 and above. Salmonella and kampilobacteria ReA — determination of antibody titers in the blood, at least — the study stool culture (at the time of ReA it can be negative).

ReA treatment carried out in two directions:

1. Antibiotic therapy.

2. Treatment of articular syndrome:

• Duration of treatment is 3–4 weeks.

• Patients with Chlamydia infection should be evaluated for the presence of other infections, sexually transmitted diseases.

• It is recommended to abstain from sexual intercourse for 7 days after completion of treatment, and as long as all the sexual partners of patients will not pass an appropriate course of treatment.

Recommended scheme:

1. *Anti-infective medicines* (tetracyclines, macrolides, fluoroquinolones) in the case of reactive arthritis urogential etiology.

1.1. Macrolides: azithromycin 0.5–1 g / day or roxithromycin 0.3 g / day, or spiramycin by 4.5–9 mln.ED / day.

1.2. Tetracyclines: doxycycline 0.2 g / day.

1.3. Fluoroquinolones inside: ofloxacin 0.4–0.8 g / day; lomefloxacin 0.8 g / day; moxifloxacin into the 0.4 g / day; levofloxacin 0.5 g / day. The treatment duration is one of 1 month, the above drugs (available antimicrobials serial reception from each group for 10 days in case of mixed infection).

2. *NSAIDs:* aceclofenac 200 mg / day orally or diclofenac 100–150 mg / day orally or / m, or ibuprofen 1200–2400 mg / day orally, or meloxicam 15 mg / day PO / m, or nimesulide 200 400 mg / day orally or celecoxib 400 mg / day orally or etodolac 600–1200 mg / day orally.

3. *In the presence of risk factors for GI complications* — proton pump inhibitors: omeprazole 20–40 mg / day or rabeprazole 20–40 mg / day, or lansoprazole 30–60 mg / day, or pantoprazole 20–40 mg / day, or esomeprazole 20–40 mg / day, and others.

4. *Corticosteroids for systemic use.*

4.1. Prednisolone 0.25–0.5 mg / kg / day (methylprednisolone 0.2–0.4 mg / kg / day) into the presence of systemic manifestations (carditis, arthritis), followed by a gradual decrease (2.5 / 2 mg / day per week) to complete abolition.

4.2. Betamethasone 2.1 ml (5.10 mg) or 40–80 mg methylprednisolone (intra no more than 2–3 times / year in one joint.

5. Anti-inflammatory agents, acting on gut (aminosalicylic acid): sulfasalazine 1.5–2 g / day orally with the ineffectiveness of symptomatic therapy.

The primary osteoarthritis. Diagnostic criteria. Outpatient treatment.

Primary osteoarthritis (PO) — a chronic degenerative joint disease, based on the degeneration of articular cartilage with subsequent changes in bone articular surfaces, the development of marginal osteophytes, deformation of joints, as well as the development of moderate synovitis. Causes of primary osteoarthritis (OA) has not been known. The main factors in the development of prospective primary OA are: a mismatch between the mechanical stress on the articular cartilage and its ability to resist the effects; genetic predisposition, as expressed in particular in reducing the ability of cartilage to withstand mechanical stress.

Clinical picture. Pain in the joints of the mechanical type, occur when the load on the joint, more in the evening, calm down alone and at night. Crepitus on movement in the joint. Resistant strain of joints due to bone changes. Relatively minor limitation of joint mobility, with the exception of the hip. joint disease is usually bilateral, with the first to suffer knee, hip, distal interphalangeal joints, proximal interphalangeal joints.

Classification

Pathogenetic variants: Primary (idiopathic).

Secondary (due to injuries, disorders of statics, hyperplastic joints, arthritis, and others).

Clinical forms: polyosteoarthrosis: nodular, oligoosteoarthroisis; monoarthrosis; In conjunction with spinal osteochondrosis, spondyloarthrosis.

The preferential localization: interphalangeal joints (Heberden's nodes, Bouchard); hip joints (coxarthrosis); knee joints (gonarthrosis); other joints.

Radiological step;- I, II, III, IV.

Synovitis: present or absent.

The functional ability of the patient

Ability to work is limited in time (functional disorders):

1) Lost the ability to work (functional disorders).

2) Needsconstant care (functional disorders).

3) **Diagnostic criteria:** Clinical criteria Pain in the joints, resulting in the end of the day and / or in the morning. Joint pain, occurring after mechanical load and decreasing alone.

Deformation of joints due to growths.

Radiological criteria: joint space narrowing, osteosclerosis, osteophytes.

Laboratory data: complete blood count without significant changes. When reactive synovitis may be increased ESR to 20–25 mm / h. LHC without significant changes. In the case of synovitis in the blood increases the content of fibrin seromucoid, sialic acids, haptoglobin. Analysis of urine — standard.

CTX-II — a new marker in the diagnosis of rheumatoid arthritis and osteoarthritis. CartiLaps are degradation products of type II collagen — the main structural component of articular cartilage.

COMP — is a calcium-binding protein with a high molecular weight, present in the extracellular matrix of articular, nasal and tracheal cartilage. studies describe the correlation of serum levels of COMP cartilage degradation and its potential value as a prognostic marker of inflammatory joint diseases such as osteoarthritis and rheumatoid arthritis. The results showed elevated levels of communication COMP with progressive destruction of articular cartilage observed by radiography.

Stage osteoarthritis by Kellgren and Lawrence:

0 — absence of radiographic signs;

I — cystoid restructuring bone structure, linear osteosclerosis in subchondral departments, the appearance of small marginal osteophytes;

II — symptoms of stage I + more pronounced osteosclerosis + joint space narrowing;

III — pronounced subchondral osteosclerosis, large marginal osteophytes, significant joint space narrowing;

IV — coarse massive osteophytes. joint space can be traced with difficulty, the epiphyses of bones forming the joint, twisted sharply condensed.

Treatment:

1. Symptomatic medicines quick steps: 1.1 NSAIDs: aceclofenac 200 mg / day orally or diclofenac 100–150 mg / day orally or / m, or ibuprofen 1200–2400 mg / day orally.

2. Opioid analgesic: Tramadol 50–200 mg / day PO / m (with no effect on the use of NSAIDs)

3. When the presence of risk factors for GI complications, proton pump inhibitors: omeprazole 20–40 mg / day or rabeprazole 20–40 mg / day, or lansoprazole 30–60 mg / day, or pantoprazole 20–40 mg / day, or esomeprazole 20–40 mg / day, and others.

4. Symptomatic slow-acting drugs (with stage I–II osteoarthritis):

4.1. Chondroitin sulfate is 1000–1500 mg / day - treatment 1,5–3 months 1–2 times a year or chondroitin sulfate 200 mg / m in a day - 25 injections of 1–2 times a year.

4.2. Glucosamine sulfate 1500 mg / day orally or in 2 ml / m in a day (6–8 injections of 2 to 3 times a year).

5. Symptomatic therapy:

5.1. Central muscle relaxants.

5.2. Tolperizon 150 mg 3 times daily by mouth and others.

5.3. Antiplatelet agents: pentoxifylline (600–800 mg / day).

5.4. Antidepressants (within treatment of chronic pain syndrome):

Selective serotonin reuptake inhibitors: fluoxetine 20 mg.

5.5. intraarticular corticosteroids (no more than 1–2 times a year, only in the presence of synovitis) or periarticular: Betamethasone 1.2 ml (5–10 mg) or methylprednisolone 40–80 mg or 40–80.

TOPIC 4: Dyspeptic syndrome and abdominal pain, differential diagnosis and tactics of local therapist on an outpatient basis. Irritable bowel syndrome, patient diagnosis, treatment and rehabilitation of patients. Acute abdomen, medical tactics.

Dyspeptic syndrome as a collective term used to describe a variety of motor dysfunction of digestive tract: stomach, intestine, bile ducts (symptoms, pathogenesis). Circle of diseases associated with these symptoms, survey patients on an outpatient basis.

At a meeting of the International Working Group for the Study of functional gastrointestinal disorders, known as the Rome Consensus III (2006), Functional Dyspepsia is defined as a set of clinical symptoms (pain or burning sensation in the epigastric region, or a feeling of early satiety and fullness in the epigastric region) arising during the period of at least 6 months prior to diagnosis and notes for the past 3 months. On the basis of these complaints at the prevailing clinic are two main syndromes: epigastric pain syndrome (ulcerative form of Rome criteria II) and postprandial distress syndrome

(dyskinetic form of Rome criteria II). Diagnosis of Functional Dyspepsia is placed by an exception in cases where the result of the survey (EGD and morphological research) does not have any organic, systemic or metabolic disease that can explain these symptoms. The prevalence of Functional Dyspepsia hovers around 30–50 %, almost the same as in Western countries and the eastern region of the globe. Women PD occurs twice as often than men, and the peak uptake with dyspeptic complaints between the ages of 35–45 years. Analyzing the data of International Conference on Gastroenterology and Hepatology in 2010, held in Thailand, it should be noted that patients with Functional Dyspepsia, along with the above symptoms occurs symptoms characteristic of other diseases of the gastrointestinal tract. Thus, the symptoms of gastroesophageal reflux disease (GERD) occur in 7–20 % of patients Functional Dyspepsia, and irritable bowel syndrome (IBS) — in 8–50 % of patients. The presence of this pathology does not exclude Functional Dyspepsia, and serves as a pretext for staging several independent diagnoses and the use of appropriate diagnostic and therapeutic tactics.

Table 8 — Classification of functional dyspepsia (Rome III)

Postprandial distress syndrome	Disturbing feeling of fullness after eating the normal amount, at least several times a week. Early satiety, preventing completion of the regular meal, at least once a week
Epigastric pain	Pain or burning sensation in the epigastric, at least moderate severity, ≥ 1 times a week, alternating, not generalized and not in other parts of the abdomen and chest

Continuation of the table 8.

To date, the PD is regarded as a heterogeneous disorder with diverse etiopathogenetic mechanisms. According to Talley NJ, Choung RS, 2009, are the following etiological factors of PD:

• heredity;
• availability of violations of motor function of the stomach and intestines;
• infection of H. pylori;
• psychosocial factors.

Etiology and pathogenesis

pathogenetic role has not hypersecretion of HCI, and the increase of acid contact time with the mucosa of the stomach and duodenum, as well as hypersensitivity to the formation of its chemoreceptors inadequate response**.** About 50 % of patients with FD are H. pylori-positive. H. pylori infection leads to chronic helicobacter gastritis, accompanied by some patients dysfunction of pacemaker gastric fundus insufficient relaxation, expansion of the antrum with the weakening of its postprandial motility. This eventually leads to violation of the motor-evacuation function of the stomach with the development of gastroparesis. H. pylori therapy can lead to the normalization of gastric motility.

However, complete elimination of dyspeptic complaints noted in only 20–25 % of patients with FD. A role in the occurrence of dyspeptic disorders, especially in patients with nonspecific dyspepsia option plays decrease the sensitivity of the stomach wall to stretch (violation of the afferent link), which occurs in 50–70 % of patients.

Dyspepsia, depending on the affected organ is divided: gastric, intestinal, hepatoduodenal, pancreatogenic.

Gastric dyspepsia is caused by insufficient or excessive secretion of hydrochloric acid, as well as overly fast or dramatically slow gastric emptying. The symptoms of gastric dyspepsia:

1. Eructation — a sudden, involuntary release of stomach contents into the mouth.
2. Heartburn — burning sensation along the esophagus or in the epigastric region, due to hit the gastric contents into the esophagus. It is often a manifestation of increased acidity in the stomach and the cardiac sphincter insufficiency.
3. Nausea — painful pressure in the epigastric region, accompanied by an unpleasant sensation in the mouth and hypersalivation.
4. Vomiting — a complex reflex act caused irritation of the vomiting center, during which there is an involuntary, jerky ejection of gastric contents through the mouth.

Gastric indigestion often manifests itself in diseases such as chronic gastritis, duodenitis, gastric ulcer and 12 duodenal ulcer.

Intestinal dyspepsia — a violation of the process of food digestion in the intestine as a result of the enzyme deficiency of the small intestine, pancreas, liver or due to the accelerated movement of food through the intestines dysbacteriosis or changing the morphology of the intestine.

Types of intestinal dyspepsia: flatulence, rumbling, defecation disorder (diarrhea, constipation, and their sequencing).

Types of diarrhea: enteral, colitis.

Enteral diarrhea observed in the pathology of the small intestine. It noted a relatively small number of bowel movements 4–6 times a day, malabsorption during defecation, there is polyfeacalia.

Colitis diarrhea observed in the pathology of the colon. They are characterized by frequent bowel movements more than 10 times a day, scanty stool, tenesmus, pain during bowel movements, rectal symptom of spitting.

Reasons and types of stomach pains, their pathogenesis. The concepts of acute and chronic abdominal pain, "acute abdomen", a circle of disease causing the pain.

There are abdominal pain:

1. Intraabdiminal:

— peritonitis (primary and secondary);

— recurrent disease;

— inflammatory diseases of the abdominal cavity (appendicitis, cholecystitis, peptic ulcer, pancreatitis and others);

— pelvic inflammatory disease (cystitis, adnexitis, etc.);

— obstruction of the hollow body (intestinal, biliary, genitourinary);

— ischemia of the abdominal cavity;

— irritable bowel syndrome;

— hysteria;

— narcotic withdrawal, etc.

2. Extraabdominal:

— diseases of the oral cavity pulmonary embolism, pneumothorax, pleural effusion;

— polyneuritis;

— diseases of the spine;

— metabolic disorders (diabetes, uremia, porphyria, etc.);

— exposure to toxins (insect bites, poison poisoning).

Mechanisms of abdominal pain:

a) visceral (with pathological stimuli in the internal organs) with increasing pressure, tension, poor circulation;

b) parietal (with the involvement of abdominal cover) usually is acute, clearly localized, accompanied by muscle tension of the abdominal wall, is enhanced by changing the position of the body, coughing;

c) of referred (or reflected) — a reflection of the intense pain when the visceral momentum as areas of increased skin sensitivity;

g) psychogenic (without somatic causes failure due serotonergic mechanisms).

Characteristics of pain:

Persistent abdominal pain (chronic) are more common in progressive inflammatory lesions are observed in granulomatous and ulcerative colitis, irritable bowel syndrome, bowel tumor perifocal inflammation, diverticulosis and diverticulitis with the formation of inflammatory infiltrate or peritonitis.

Persistent abdominal pain does not stop for 2 hours, abdominal tenderness to the touch, accession to vomiting, diarrhea, fever should seriously alerted.

Colic — cramping abdominal pain.

Stabbing pain in the abdomen can be a manifestation of the disaster in the abdomen — perforation of a hollow organ.

Possible causes of abdominal painand the localization of abdominal pain. Some people have an intolerance to a certain type of product, such as milk, milk sugar, or lactose. Use them in a food leads to spastic abdominal pain, bloating and diarrhea. Depression, spinal disorders, thyroid disease, anemia, urinary tract infection may be accompanied by abdominal pain. The reason may be the use of alcohol, drugs, antibiotics, hormonal and non-hormonal agents, iron supplementation.

Peritonitis (acute, chronic) — due to perforation of inflammatory, chronic inflammatory foci and trophic (including ischemic) disorders of the

gastrointestinal tract: the appendix, gallbladder, colon diverticulum (appendix cysts), cysts of the pancreas, sexual of women. Diagnosis is based on detection of fluid and free gas in the abdominal cavity (ultrasound and X-ray Review) due to acute destructive pancreatitis. The most important diagnostic techniques are ultrasonography (especially dynamics) and laparoscopy, which can simultaneously be and treatments.

The pain is localizedbelow the waist (abdomen):

• Men are possible diseases of the urinary system, you must monitor the urination and urine.

• Women may be the disease of the urinary system, pregnancy, painful menstruation, inflammation of internal genitals.

Painover the pubis (lower abdomen, "sore abdomen") in women — pathological processes in the bladder, uterus and appendages, may indicate any problems with the reproductive system. Pelvic pain that occurs each month before menstruation, can talk about endometriosis — a condition in which tissue from the uterus particles move through the fallopian tubes; get on the ovaries, pelvis, bladder and other organs. Soreness in the lower abdomen can mean pelvic inflammatory disease (infection of the uterus, ovaries or fallopian tubes). In women of childbearing age ectopic pregnancy can also cause acute or sharp stabbing pain in the peritoneum.

The pain is localized in the projection of the stomach, diseases of the esophagus, stomach. In myocardial infarction, pneumonia and pyelonephritis may be similar localization. Soreness in the umbilical region is observed in diseases of the small intestine. Acute pain at the top of the abdomen may be due to a perforated ulcer and 12 duodenal ulcer.

Pain in the right iliac region (about the right wing of the ilium) — inflammation of the appendix in the cecum.

Abdominal pain started ***in the lower back*** and moved in the groin: possible pathologiesurinary system, urolithiasis. Sudden pain in the lumbar — renal colic.

Abdominal pain is distributed in the ***right hypochondrium*** in the abdomen on the right, can issue under the right shoulder blade, can be liver disease, biliary tract or gall bladder; watch the color, the color of urine and feces. The emergence of pain in the upper right abdomen usually confirms biliary colic, which usually cause gallstones or bile ducts, preventing the free flow of bile from the liver and gall bladder.

Pain in the left lower abdomen can be a symptom of diverticulitis. Diverticulitis occurs when the walls of the colon produces small spherical capsule called diverticula, which subsequently become infected and inflamed. Among other symptoms of diverticulitis include fever, nausea, vomiting, chills, cramps and constipation.

Pain in the hernia — a sign of strangulated hernia. There has been an increase in the seal herniation. Often, the skin over the hernia bluish color.

Girdle pain in the epigastric area radiating to the shoulder blades, and is characterized for acute pancreatitis. The pain is accompanied by nausea and vomiting. The patient usually lies motionless on his side. Belly swollen and tense. Connection of jaundice.

Chronic enteritis — a disease characterized by inflammatory and degenerative changes in the mucosa of the small intestine .Exacerbation of ***chronic colitis*** .This inflammation of the colon mucosa . Abdominal pain minor wear or localized or diffuse nature of the lower abdomen; marked feeling of heaviness, burning, itching in the rectum, rumbling stomach pain along the colon.

Pseudoabdominal syndrome. The mechanism of formation of PAS: The generality of the innervation of the chest and the anterior abdominal wall (spinal nerves to the parietal peritoneum of the upper 2/3 of the abdominal cavity) Irritation of the phrenic, sympathetic and vagus nerves involved in the formation of the solar plexus, in the initial part of his run in the chest, and acute heart disease, lung and pleura in the early hours, in the absence of physical and auscultatory data It can come to an agreement, both acute abdominal disease. Diseases forming PAS:

- Acute pneumonia, basal pleurisy.
- Acute pericarditis, rheumatic carditis.
- thyrotoxic crisis.
- Periodic disease.
- Pyelonephritis.
- Acute myocardial infarction.
- Dissecting aneurysm of the abdominal aorta.
- Acute adrenal insufficiency, etc.

In pneumonia, resulting in toxic effects on the nervous system can occur paralysis of the gastrointestinal tract of varying intensity. Acutely which developed congestive heart failure can lead to stretching Glisson capsule. When kidney disease develops as a result of community PAS innervation and reflex connections between nerve plexus and gastro-intestinal.

"Acute abdomen":

Acute abdomen — a term adopted to describe the supposed catastrophe in the abdomen (peritonitis, bleeding), requiring emergency surgery. This term is used only in the preliminary diagnosis in the direction of the hospital

Classification:

Surgical reasons:

- Acute appendicitis (one of the most common causes of peritonitis). Abdominal pain appear suddenly, usually radiating to the umbilical region, capture the entire abdomen and only a few hours are localized in a certain place, usually on the right lower abdomen. The pain is constant, aching in nature. The body temperature rises. It may be nausea and vomiting. If the inflamed appendix is located high in the liver, the pain is localized in the right upper abdomen. If

the inflamed appendix is located behind the cecum, the pain is localized in the right lumbar region and spreads throughout the abdomen. If the inflamed appendix is located in the pelvis, pain in the right iliac region joined signs of inflammation of adjacent organs: cystitis (bladder inflammation adnexitis, inflammation of the right of the uterus). People elderly mild signs of appendicitis in mind the reduced reactivity of the organism, atherosclerosis and speed of change in the process.

- Intestinal obstruction (in history — hernia surgery on abdominal organs, adhesive disease, recurrent pain symptoms; when viewed from the abdomen — the visible peristalsis and contours inflated intestinal loops). In 70 % of patients with obstruction due to postoperative abdominal adhesions .. painless form of acute intestinal obstruction does not happen. On this basis one can assume this disease. In later stages of the disease the pain subside and tested. Another symptom is vomiting, marked dry tongue, rapid pulse, blood pressure reduction at later stages, bloating.
- Perforation of peptic ulcers (acute onset, signs of peritoneal irritation, and sometimes can be the first manifestation of peptic ulcer disease).
- Acute cholecystitis (pain and tenderness in the right upper quadrant, can be palpated enlarged gall bladder, symptoms of irritation of the peritoneum in the right half of the abdomen).
- Acute pancreatitis (in history — alcohol; etiological factor may be gallstones, characterized by severe epigastric pain radiating to the back, repeated vomiting, increased amylase concentration in serum and urine — indicative, but not obligatory symptom).
- Mesenteric vascular thrombosis (diagnosis based on clinical manifestations, may be difficult, patients often show heart disease, accompanied by atrial fibrillation: mitral, post-infarction left ventricular aneurysm).
- Diverticulitis or perforation of colon diverticulum (frequent localization — sigmoid colon).

Urologic causes:

- Testicular torsion. Egg sharply painful on palpation, often localized pain in the lower abdomen.
- Acute urinary retention (arching pain in the lower abdomen, where palpable painful distended bladder, small amounts of urine may deteriorate.

Gynecological causes:

- Ectopic pregnancy — the pain is localized in the abdomen radiating to the shoulder area of hypochondria, clavicle, or scapula (frenikus-symptom), the symptoms of anemia; pregnancy test can be negative serological test is more sensitive.
- Diseases of the ovaries — the gap cysts, ovarian cyst torsion legs, salpingo.
- Fibroids — torsion legs subserous myoma node, necrosis, bleeding into the abdominal cavity. Nonsurgical causes of acute abdomen.

- Metabolic disorders — diabetic ketoacidosis, porphyria, hypertriglyceridemia, hemochromatosis, tetany (develops when the level of calcium).
- Infectious causes — gastroenteritis, diverticulitis, hepatitis, perihepatitis, infectious mononucleosis, herpes zoster, pyelonephritis, epididymitis, orchitis, sepsis.
- Radiating pain — myocardial infarction, pericarditis, pleurisy, lung infarction, heart failure (stagnation in the liver), kidney stones.
- Immunological disorders — angioedema, periarteritis nodosa, Henoch purpura Shenlyayn-hypersensitivity reactions.

Gastrointestinal bleeding occurs in diseases of the liver (of the veins of the esophagus); stomach ulcers; erosive gastritis; gastric cancer in the last stage; peptic ulcer 12 duodenal ulcer; Ulcerative colitis (disease of the colon); rectal hemorrhoids and other diseases of the gastrointestinal tract: infectious diseases, diathesis, trauma.

Symptoms:

1. Onset is usually acute.
2. When bleeding from the upper gastrointestinal tract (stomach, esophageal varices) is bloody vomiting fresh blood or blood color of coffee grounds. The remainder of the blood passing through the intestines, is released during defecation as tarry stools (a liquid or semi-liquid stool of black color with a pungent smell).
3. When bleeding 12 duodenum in peptic ulcer hematemesis is less than bleeding from the esophagus or stomach. In this case, the blood passing through the intestines, is released during a bowel movement in the form of tarry stool.
4. When bleeding from the colon appearance of blood changes slightly.
5. Hemorrhoidal veins of the rectum bleed red blood (hemorrhoids).
6. When gastrointestinal bleeding there is a general weakness, rapid and weak pulse, blood pressure reduction, copious cold sweat, pallor, dizziness, fainting.
7. In severe bleeding sudden drop in blood pressure.

Malabsorption, maldigestion and malnutrition: the concept of pathogenesis. Differential diagnosis of the level of destruction of the intestine. Malabsorption (malabsorption in the intestines) — a syndrome characterized by a set of clinical manifestations (diarrhea, steatorrhea, polyhypovitaminosis, weight loss), developing as a result of violations of the digestive and transport functions of the small intestine, which in turn leads to pathological changes in metabolism**.**

Classification of malabsorption. Malabsorption syndrome is classified by severity: the first (easy) stepen- (weight loss up to 10 kilograms, weakness, decreased performance, and some signs of hypovitaminosis), second (moderate) stepen- (weight loss of more than 10 kilograms, expressed polyhypovitaminosis, disruption of water and electrolyte homeostasis, anemia, reduced levels of sex hormones), third (heavy) stepen- (significant deficit of body weight, heavy multivitamin and electrolyte deficiency, osteoporosis, severe anemia, edema,

convulsions, severe endocrine disorders). The symptoms of malabsorption Patients with malabsorption syndrome marked changes in the bowel diarrhea, steatorrhea, bloating and rumbling, sometimes pain in the stomach. The pain is usually localized in the upper abdomen, may radiate to the lower back or have herpes character, if there is a chronic pancreatitis. In patients with lactase deficiency pain cramping in nature. When malabsorption amount of feces, usually increased, a mushy stool or watery consistency, fetid. From the nervous system manifested asthenovegetative syndrome — weakness, fatigue, apathy. This is due to a violation of water-electrolyte homeostasis and deficiency of necessary materials for the nervous system. Pathological changes of the skin: dry, dark spots, dermatitis, eczema, hair loss, brittle nails and blurred, ecchymosis — relate to vitamin and mineral deficiencies. For the same reasons often noted glossitis (inflammation of the tongue). Lack of vitamin K is shown the formation of petechiae (red spots on the skin) and subcutaneous hemorrhage. Complications malabsorption. Major complications of malabsorption syndrome due to a lack of nutrients into the blood: anemia (iron deficiency and megaloblastic anemia), impaired fertility, neurovegetative disorders, degeneration, multiple organ pathology associated with polyhypovitaminosis and micronutrient deficiencies.

Syndrome of insufficient digestion (maldigestion) is a series of features that characterize a violation of the recessed or wall digestion in the gastrointestinal tract.

Etiology and pathogenesis. When advantageous abuse cavity digestion is important etiological factors of a number. Above all, this failure of gastric secretion.

Insufficient digestion in the stomach diseasesn Pathology of the secretory function of the stomach can develop at achylii and achlorhydria, decompensated pyloric stenosis, as well as atrophic gastritis and cancer.

Insufficient digestion in the intestine can be characterized as a disease of the recessed and wall digestion, as well as mixed. Patients with intestinal insufficiency digest food complained of feeling rumbling and transfusion in the intestines, bloating, flatulence expressed, diarrhea. Usually accompanied by maldigestion syndrome malabsorption syndrome, as it is not digested food is not absorbed.

The concept of "malnutrition» (malnu-trition) appeared in medicine 40–50-ies. The twentieth century. in describing the states resulting from malnutrition in children. This power state in which the lack, excess or imbalance of energy, protein and other nutritional ingredients leads to severe negative effects in tissues and organs disrupts the normal functioning of their. Malnutrition caused by a number of mechanisms: 1) physiological or anatomical impairment food intake, 2) disorders of digestion or absorption of nutrients, 3) metabolic disorders and 4) catabolic processes in some diseases. At the heart of the nutritional status of the patient is based on three main components: 1) energy and protein balance, 2) the degree of metabolic stress, 3) functional state authorities. Patients with this syndrome have an increased risk of infection and other complications, longer length of stay in the hospital treatment.

General principles for the diagnosis and differential diagnosis of diseases accompanied by abdominal pain and dyspeptic syndromes.

Intestinal indigestion occurs when insufficient exocrine function of the pancreas, chronic inflammatory diseases of the small intestine. For coprological studies characterized by: steatorrhea, amylorrhea, creatorrhea. When X-ray observed rapid passage of barium suspension through the small intestine. Research exocrine pancreatic function, enterobiopsy aspiration, determination enterokinase and alkaline phosphatase in the intestinal juice allow to specify the cause of intestinal dyspepsia. Research glycemic curve with a load of oral starch and radioisotope research allow us to estimate the degree of violation of the digestive cavity. It is important to study the intestinal microflora.

Dyspepsia alimentary results from prolonged malnutrition. Distinguish the fermentation, putrid fat and dyspepsia. Fermentation dyspepsia associated with excessive ingestion of carbohydrates, resulting in the gut, the conditions for the development of the fermentation flora. Putrid neuralgia occurs when the predominant eating protein foods that are slowly digested in the gut. Adipose indigestion caused by excessive consumption of slowly digested, especially refractory, fat. When scatological study identifies a large number of starch grains, crystals, organic acids, fiber, iodophilic microorganisms. Reaction sharply acidic feces. Dyspepsia putrid manifested as diarrhea, but the color of bowel movements very dark, the smell — a putrid. When fat dyspepsia feces bright, abundant with bold luster, neutral or alkaline reaction. Coprological study reveals a large number of them in a neutral undigested fat (in the form of droplets) of the crystals of fatty acids and their insoluble salts. Diagnosis is based on the questioning of the patient (the nature of power), clinical manifestations of dyspepsia, data coprological research. When rektoromano- colonoscopy and signs of inflammation of the mucous membrane of the colon is not detected.

Helminthiasis. Opisthorchiasis. Pathogen — cat fluke which infests the bile ducts of the liver, gallbladder and pancreatic ducts man, cats, dogs. In the early period may be fever, muscle aches and joint pain, vomiting, diarrhea, pain and enlargement of the liver, rarely spleen, leukocytosis, and high eosinophilia, allergic skin rash. The most common phenomenon at opistorchose developed cholecystitis, biliary dyskinesia, chronic hepatitis and pancreatitis. Hookworm (hookworm and necatoriasis). Etiology, pathogenesis. Pathogens — American hookworm and hookworm, parasites in the human small intestine, most often in the duodenum. When fixing to the intestinal mucosa, they injure tissue, leading to the formation of hemorrhages, erosions, induce bleeding anemisation support allergy condition, dyskinesia gastrointestinal tract and dyspepsia. Shortly after infection occurs itching and burning, urticaria rash, wheeze phenomenon, fever, eosinophilia. Characterized by the development of hypochromic iron deficiency anemia.

Dyspepsia due to exocrine ***pancreatic*** insufficiency of the pancreas (from chronic pancreatitis, tumors, etc.). Insufficient intake in the intestine pancreatic

lipase, amylase and trypsin digestion breaks nutrients. There is a feeling of rumbling and transfusion in the abdomen, bloating, impaired appetite, may be colicky abdominal pain, characterized by a rich «pancreatogenic» diarrhea (fatty stools), steatorrhea, amylorrhea, kreatoreya.

Enteritis

Acute enteritis. In acute enteritis often in the pathological process involved at the same time as the stomach (gastroenteritis) and colon (gastroenterocolitis). There are acute enteritis: infectious and viral origin (with the clinical picture of severe enteritis occur cholera, typhoid fever, salmonellosis; alimentary- (due to overeating with taking a large number of acute or too rough foods that irritate the mucous membrane of spices, alcoholic beverages); toxic — (for poisoning by arsenic compounds, mercuric chloride and other poisons; mushroom poisoning — a pale toadstool, amanita, false mushrooms and other non-bacterial nature of the toxic substances that may be present in food products — stone fruit, some fish products — liver burbot, pike, caviar mackerel, etc.).

Allergic (with idiosyncrascy to certain foods — strawberries, eggs, crab or an allergic reaction to medication — iodine, bromine, some sulfonamides, antibiotics, etc.).

Ischemic Colitis — segmental lesions of the colon caused by a violation of its blood supply. Most affected area of splenic curvature, at least — the transverse colon, the descending and sigmoid colon. The diagnosis is confirmed irrigo-, rektoromano- and colonoscopy. When barium enema in the affected area found painting "pseudotumora" defective filling a "thumbprint". Endoscopic examination reveals swelling of the mucous membrane lesion, submucosal hemorrhage, in chronic cases — inflammatory infiltration of the mucosa, ulceration and scarring due to ulcers — stricture of the affected part of the colon. Selective mesenteric angiography is used to confirm a violation of mesenteric artery patency.

Acute colitis is usually common, often combined with simultaneous acute inflammation of the mucous membrane of the small intestine (acute enterocolitis), and sometimes the stomach (gastroenterocolitis). The causative agents of acute colitis — shigella (dysentery bacterial), salmonella, rarely other pathogenic bacterial flora, viruses. Its cause may be the non-bacterial food poisoning, rude errors in the diet. When sigmoidoscopy determined hyperemia and mucosal edema distal colon, on the walls of the intestine can be seen a large amount of mucus, and in more severe cases — pus; may be erosion, ulceration and hemorrhage. Blood test reveals moderate leukocytosis with stab shift, increased ESR.

Chronic colitis — one of the most common diseases of the digestive system. Often combined with inflammatory lesions of the small intestine (enterocolitis) and stomach. Colitis of infectious origin can be caused by pathogens of intestinal infections, particularly Shigella and Salmonella pathogens other infectious diseases (tuberculosis mycobacteria, etc.), Opportunistic and saprophytic flora of the human intestine (due to dysbiosis).

Colitis allergic nature are observed in food allergy, intolerance to certain drugs and chemicals, increased individual sensitivity to some types of bacterial flora of the intestine and the decay products of microorganisms.

The symptoms for colitis. The main symptoms are a violation of the chair (chronic diarrhea or constipation), pain in various parts of the stomach, sometimes painful tenesmus, flatulence, dyspepsia. Dominated by diarrhea — defecation occurs 10–15 times or more per day, is frequently observed alternating diarrhea and constipation. Characteristic symptom of inadequate bowel movement: after a bowel movement in a patient there is a sensation of incomplete emptying. Sometimes the pain gets spastic character (with spastic colitis), subsides by the application of heat (heating pad, compress), after taking choline and antispasmodics; twinge may be accompanied by flatus, or the emergence of the urge to defecate. Flatulence in colitis is due to violation of the digestion of food in the small intestine and dysbiosis. When the superficial palpation often reveals areas of soreness of the abdominal wall, located along the colon. With deep palpation of the affected areas of the colon is usually painful and spastic reduced, there may be an alternation of spastic contraction and expansion of areas filled with dense or liquid contents, strong rumble and even splashing in the appropriate department intestine. In exacerbations of colitis can be mild leukocytosis with a left shift, increased ESR, low-grade fever. The aggravation of allergic colitis, in addition to pain attack, often accompanied by fever, eosinophilia, appearance of Charcot-Leyden crystals in the faeces. Purulent, fibrinous and necrotic ulcerative colitis occur with a much more severe clinical picture. A special form of colitis is ulcerative colitis.

Crohn's disease — non-specific inflammation of the gastrointestinal tract at any level with the formation of his inflammatory infiltrates deep longitudinal ulcers complicated by perforation, external or internal fistulas, bleeding and other serious complications. The etiology is unknown. In the colon, Crohn's disease manifested as granulomatous colitis. The emergence of foci of granulomatous inflammation and ulceration of large areas of the colon leads to intoxication depleting diarrhea, intestinal bleeding, anemia, dysproteinemia, electrolyte imbalance and complications of septic character. More common many segmentary lesions of the colon, which is more severe and resemble the symptoms and course of ulcerative colitis, but granulomatous colitis rectum is significantly less affected than other parts of fat. The most common form internal and external fistulas, deep anal fissures. The importance of the differential diagnosis are X-ray examination, and colonoscopy rektoromano- (longitudinal ulcers, fissures) and biopsy (deep plasma-lymphocytic infiltration, sarcoid granulomas).

Ulcerative colitis is characterized by necrotizing inflammation of the mucous membrane of the large intestine with the formation of ulcers, hemorrhage and pus. Presumably etiological factors are infections (viruses, bacteria), poor diet, often a predisposing factor to the disease.

The pathogenesis of ulcerative colitis:

1) intestinal bacteria overgrowth associated with violation of the normal microflora;

2) violation of neurohumoral regulation of bowel function due to the dysfunction of the autonomic and endocrine gastrointestinal tract;

3) increased permeability of the mucosa of the colon to the protein molecules,

4) damage to the intestinal wall and the formation to form AT autoantigens in the intestinal wall;

5) formation of immune complexes in the wall of the colon.

At UC develops a pronounced inflammatory process in the wall of the colon in 80 % of patients microabscesses crypts of the colon. The most commonly affected areas of the distal colon and rectum.

The main symptoms of ulcerative colitis:

1. Diarrhea with blood, mucus, pus. In severe intestinal disease characterized by painting a chair up to 20 times a day, the number of patients per day lost by 100–300 ml of blood. Stool with a lot of pus and smelly odor.

2. Pain in the abdomen. The pain is constant, cramping in nature. Localized in the projection of the sigmoid, transverse colon. Perhaps increased pain after eating and its weakening after stool.

3. Intoxication syndrome, characteristic of severe and acute fulminant forms of the disease. Intoxication syndrome manifests weakness, weakness, weight loss, lack of appetite.

4. Syndrome of systemic manifestations:

— polyarthritis — affects the ankle, knee, interphalangeal joints. Some patients develop spondylitis and sacroiliitis;

— erythema nodosum — often manifested by multiple nodes on the extensor surface of the tibia, skin lesions — may develop gangrenous pyoderma, skin ulceration, focal dermatitis of the eyes develop iritis, uveitis, keratitis;

— liver and extrahepatic bile ducts: ventricular liver, portal fibrosis, chronic active hepatitis, cirrhosis, cholangitis sclerosing-manifestation of extrahepatic biliary tract lesions damage mucous membranes of the mouth is characterized by the development of aphthous stomatitis, glossitis with pain, possible ulcerative stomatitis, autoimmune thyroiditis and autoimmune hemolytic anemia. Complications UC.

1. The perforation of the colon. The main symptoms of perforation: the appearance, sharp abdominal pain, symptoms of intoxication detection, detection of free gas in the abdominal cavity during fluoroscopy.

2. Intestinal bleeding. Blood in the stool permanent feature of UC. If you stand out from the rectum blood clots — is bleeding.

3. Strictures of the colon.

4. Inflammatory colon polyps, a biopsy, irigoscopy.

5. Colon cancer.

Laboratory and instrumental diagnostics: anemia, leukocytosis, increased ESR. proteinuria, microscopic hematuria. Coprogram: leukocytes, cells of the intestinal epithelium.

Bacteriological analysis of feces: goiter; Proteus, Escherichia, staphylococcus, fungi of the genus Candida. Microscopy feces: pasty or liquid blood, mucus, pus. Endoscopy, colonoscopy, sigmoidoscopy.

Plan evaluation of patients with suspected bowel disease. Irritable bowel syndrome: diagnostic criteria, the general principles of treatment and rehabilitation of patients, prevention.

Examination of the patient with suspected bowel disease:

1. Complete blood count, urinalysis.

2. Scatological study can be characterized as a method of integral evaluation processes of digestion and absorption in the intestine, as well as movement disorders. Estimated macroscopic changes feces, as well as the changes that can be detected by microscopic examination. The main symptoms of scatological following:

• Creatorrhea — the presence of muscle fibers in the stool due to a deficiency of enzymes involved in the hydrolysis of proteins. The muscle fibers are changed (without cross-striations) and unchanged (with striated).

• Fat in stool — steatorrhea. They found as neutral fat, fatty acids and soaps (salts of fatty acids). Steatorrhea can be detected with the lipase deficiency, as well as in violation of suction end products of hydrolysis of fats.

• Carbohydrates in feces — amylorrhea. It is defined as due to lack of extracellular starch pancreatic amylase or intestinal, as intracellular (in plant shell) starch because of irregularities bacterial fermentation in the cecum.

3. Analysis of fecal occult blood. His goal — to reveal the presence of bleeding in the overlying intestine. If you suspect a dysbiosis, irritable bowel syndrome, and others. Disease proctologist may appoint fecal seeding fecal flora with sensitivity to antibiotics and bacteriophages.

4. Irrigoscopy — X-rays of the colon during retrograde filling it radiopaque suspension. Barium enema is used for diagnosis of diseases of the colon (malformations, tumors, chronic colitis, diverticulosis, fistulas, scar contraction, etc.). Irrigoscopy allows information on the morphological changes of the colon, which in terms of diagnosis of clinical entities is more valuable. Barium enema is often a decisive method of diagnostics of tumors, diverticula of the colon. Increases diagnostic capabilities technique of double contrast barium enema. With respect to diseases such as colitis, tuberculosis can be obtained only indirect indications. Irrigoscopy contraindicated in severe condition of the patient and perforation of the colon wall.

5. Colonoscopy — a method of endoscopic diagnosis of diseases of the colon. Colonoscopy is the most informative method for the early diagnosis of benign and malignant tumors of the colon, ulcerative colitis, Crohn's disease, etc., and enables

80–90 % of the viewing colon throughout. During colonoscopy visually evaluate the state of the colonic mucosa. Colonoscopy is also possible to perform various therapeutic procedures — removal of benign tumors, stop the bleeding, removal of foreign bodies, recannalization bowel stenosis, and others. Study contraindicated in acute infectious diseases, peritonitis, and in the later stages of heart and lung failure, marked disturbances of blood coagulation. It is impossible to carry out a colonoscopy in patients with severe ulcerative and ischemic colitis.

6. A biopsy of the intestinal mucosa.

A biopsy of the colon makes it possible to carry out a study histomorphological. There are two ways to obtain fragments of intestinal mucosa — using blind aspiration biopsy and targeted biopsy, performed during endoscopy. Indications for both biopsy different — blind is used for suspected diffuse lesions ulcers, aiming can be carried out in these situations, but especially in cases of suspected tumor, Crohn's disease, tuberculosis. To assess the state of the intestinal mucosa can be used three ways: Light microscopy, where it is possible to hold a normal histological examination of biopsy material, also used histochemical methods of processing material. Stereoscopic microscope, which allows to study the drug in three dimensions, giving the impression of spaciousness mucosa. Electron microscopy for the study of ultrastructural changes in the mucosa. Morphological examination of the mucosa is decisive method of diagnostics of basic clinical entities intestinal lesions.

The functional disorders of the colon include disorders of its motor (motor), transport (absorptive and secretory) functions without irreversible structural changes in the bowel. There are many synonyms to refer to this pathology: irritable bowel syndrome, neurogenic spastic colon, mucous colic, dyskinesia of the colon and others. It is not difficult to observe that each name to some extent reflects the characteristics of functional disorders. This problem is easily solved if the disease denoted as a "functional disorder of the colon" and at the same time point in the diagnosis of the type of violation. Etiology and pathogenesis. The causes of functional disorders of the colon include, above all, psycho-emotional disorders associated with abnormal development of the type of obsessive-phobic, hypochondrical, or hysterical depressive syndromes. Observed in these patients a variety of neuro-vegetative disorders, inadequate response to stress and other external environment often lead to disorders of regulation of functions of the colon, as it becomes their critical organ of mental maladjustment. The cause of the loss of the normal regulation of defecation.

Among other factors that predispose to dysfunction of the intestine, are important sedentary lifestyle, poor diet deficient in the diet fiber and intestinal infections and other diseases of the digestive system. Classification: The colon has 3 main functions: motor, transport and excretory, thereby forming and evacuation of stool. The main options for functional disorders include:

1. Violations of the motor:

a) hypermotor (increased tone, express propulsion);

b) hypomotornomous (reduced tone, slow propulsion).

2. Violations of transport:

a) the hypersecretion of ions and water into the lumen;

b) the increased absorption of water and ions in the colon.

3. Violations of mucus secretion:

a) the excess secretion of mucus;

b) a reduced mucus secretion.

These features, and are reflected in the formulation of a diagnosis:

1. Hypermotornomous dyskinesia with episodes of watery diarrhea neurogenic origin; asthenic-neurotic syndrome.

2. Functional disorders of colon hypokinetic type syndrome atonic constipation.

3. Functional disorders of colon hypermotor type syndrome spastic constipation and overproduction of mucus.

Differential diagnosis. The diagnosis of irritable bowel syndrome is a diagnosis of exclusion, since the complaint to be met by patients, may just accompany prognostically unfavorable for organic diseases, but excluding that, the doctor can focus on the functional diagnosis of the disease.

First of all, the differential diagnosis should exclude simple reason bowel irritation: chronic exposure to dietary factors and medications. A role in the genesis of irritable bowel syndrome can play some of the physiological changes in the body of women: menstrual period, pregnancy, menopause may occur with symptoms of IBS. Long-term psycho-emotional and intellectual overexertion, fear, excitement may lead to the development of IBS symptoms that quickly disappear after rest and resolve a stressful situation. The most common pathology, accompanied by manifestations of IBS, is a congenital fermentopathy — and disaccharidase lactase deficiency, which is the simplest way to diagnose a diet exclusion that does not contain milk and its products, sorbitol (chewing gum), which requires the assimilation of lactase or disaccharidase. Organic diseases of the bowel — colorectal cancer, polyps, diverticulosis, Crohn's disease and ulcerative colitis, microscopic colitis, mastocytosis, intestinal infections, parasitic infections, malabsorption syndrome, short small intestine, dolichosigma, celiac disease, tuberculosis — must fall within the scope of differential diagnosis search. Neuroendocrine tumors of the gastrointestinal tract, primarily gastrinoma, carcinoid syndrome and Vipom in the early stages may take place under the guise of diarrheal form of IBS or pain. Among endocrine diseases often occur on the type of diarrheal form of IBS hyperthyroidism and diabetes mellitus with autonomic diabetic enteropathy.

Currently, based on the principles of evidence-based medicine has developed a strategy for managing patients who first approached with dyspeptic complaints, predetermining that the following points:

• the need to obtain evidence of an association with symptoms of upper gastrointestinal tract;

• eliminate the "symptoms of anxiety" that require further examination of patients in good faith, in order to identify a more severe underlying pathology;

• exclude acetylsalicylic acid or other NSAIDs;

• the presence of typical reflux symptoms should make a preliminary diagnosis of GERD and treated;

• expedient is non-invasive determination of H. pylori, and a positive response is necessary to eradication of Helicobacter pyloric — Strategy «test and treat»;

• patients with "alarm symptoms" or over the age of 40–45 years is required EGD.

Symptomatic therapy dyspeptic syndrome. Indications for prokinetic, antispasmodics, laxatives and obstipiruyuschih funds.

Treatment

The basis of a medical complex in the syndrome is considered a functional food that contains pectin, dietary fiber — ***probiotics.*** Dietary fibers not only absorb water, increasing fecal weight, but also stimulate the motility of the colon. The traditional source of dietary fiber and pectin considered bran. Taking into account the property of bran to bind water, it is advisable to recommend drinking 1.5–2 liters. The ineffectiveness of a diet high in dietary fiber in the treatment of constipation associated with the fact that some patients are not disturbed defecation due to changes in the consistency of stool or rectal function, and as a result of disorders of coordination of muscle contractions of the pelvic floor: the lack of relaxation of the striated muscles of the pelvic floor during bowel movement. Dysbiotic changes at EBS is usually characterized by a deficiency of bifidobacteria and an increase in opportunistic microorganisms population level, the emergence of E. colic altered enzymatic properties, so after the end of a three-course treatment of intestinal antiseptics recommended intake ***probiotic*** — bifidumbacterin, Bifikol, Bifidok, atsilikta, atsinola etc. at 10 dose per day for 3 weeks. Probiotics eliminate opportunistic and pathogenic microflora and normalize the content of the genus Lactobacillus and Bifidobacterium, which play a role in the regulation of normobiotsenoza and stability. Diarrhea recommended binders, coating agents, sorbents: bismuth salts Polyphepanum, Bilignin, Almagelum, Aluminium phosphate gel and other aluminum containing 1 dose at 1 hour after eating and antidiarrheal drugs that slow gut motility — loperamide (imodaum) 2–4 mg on reception (16 mg daily) prior to termination of diarrhea. Flatulence effective Espumizan which lowers the surface, the tension of the gas bubbles in the chyme, envelops and protects the intestinal wall, preventing the accumulation of gases. Assign it 1–2 capsules orally 3 times a day. Tribuks contains trimebutin that stimulates bowel movements. The mechanism of action is direct action on the smooth muscle of gastrointestinal tract disorders and regulation of motor function without affecting the central nervous system. It characterized as a stimulating effect and a retarding motility of the gastrointestinal tract. The process of normalization of motor activity starts 30 minutes after taking the medicine. Appointed on 1 tab. 3

times per day. To reduce the increased excitability of the nervous system used tranquilizers — elenium (hlozepida) 0.005 g, 0.005 g diazepam Phase 2 a day, bromides. Because psychotropic drugs are most commonly used tricyclic antidepressants (amitriptyline, imipramine, doxepin). or selective serotonin reuptake inhibitors (fluoxetine, sertraline, paroxetine): 1 table. 2–3 times a day.

First of all at check patient dyspeptic complaints is necessary to exclude the so-called symptoms of anxiety, which include unmotivated weight loss, recurrent vomiting, bleeding (vomiting blood or "coffee grounds", melena, gematoheziya), dysphagia, fever. generally administered antisecretory agents (proton pump inhibitors, H2-blockers less histamine receptors), antacids, prokinetics, or combinations thereof. It should be noted that a positive response to receiving PPIs is observed more frequently in patients with symptoms of epigastric pain. Given that the main pathogenetic mechanism of occurrence of PD symptoms is dyskinesia of the stomach and duodenum, mandatory component of treatment for any clinical forms of dyspepsia are modern ***prokinetics***. widely used dopamine receptor antagonists (metoclopramide, domperidone, mosapride and new prokinetic dual mechanism of action combined itopride hydrochloride (Ganaton). Ganaton — the drug activates the release of acetylcholine at the same time preventing it degradation.Itoprida hydrochloride has also expressed antiemetic effect, increases the propulsive motility of the stomach and accelerates the emptying. Of course, the use of itopride when PD is preferable because of the minimal range of side effects in the therapeutic dosage range (50 mg, 3 times / day.). All of this makes itopride hydrochloride drug of choice for PD, that is confirmed by numerous multi-center study the effectiveness of this drug in patients with FD.

Therapy constipation (constipation) stages:

1. The process of collecting anamnesis attention is paid to the duration of the disease, the frequency of bowel movements and stool quality. Taking into account the act of defecation, presence or absence of pain, feeling of incomplete bowel movements, impurities in the stool. Furthermore, it is established which accompany the disease can occur in the body.

2. Diagnosing the causes of the phenomenon of constipation:

— bowel cancer;

— the formation of polyps (growths) in the colon;

— narrowing (stenosis) of the lumen of the colon;

— the stones in the stool, or the so-called debris;

— protrusion in the passage of the colon;

— varicose in the colon;

— adhesions in the abdominal cavity;

— intestinal volvulus or herniation.

Confirmation or refutation of neurological, psychogenic reasons.

Confirmation or refutation of disorders of the endocrine system, which sets the endocrinologist in laboratory studies.

3. Assign a specific individual treatment that may be of medical or surgical in nature.

Emergency medical care and medical tactics in an outpatient setting under "acute abdomen" and abdominal pain.

Physical examination:

• It is necessary to find out whether any symptoms of acute abdomen suddenly for the first time developed a long while exacerbating abdominal pain. Acute onset may indicate perforation of the organ or colic.

• Localization of secondary pain may indicate the spread of the pathological process. Pancreatic pain, for example, is localized in the epigastric; often radiating to the left upper quadrant, can carry herpes character.

• Aversion to food and the connection of pain with food can help in establishing the diagnosis. Pain after a meal typical of a stomach ulcer, but it can be associated with other diseases of the upper gastrointestinal tract.

• Vomiting involves a violation of the passage of food through the stomach or bowel obstruction.

• Vomiting eaten food is typical for pyloric stenosis.

Cholemesis suggests intestinal obstruction at the proximal small intestine.

Fecal vomiting — a late sign of severe intestinal obstruction. Obstruction of the proximal small intestine manifested indomitable copious vomiting. When obstruction of the distal portions of vomiting is not so pronounced. Reflex vomiting may be associated with intense pain with colic (kidney, liver).

• Constipation are usually chronic. Changes in the nature of a chair — an important feature, allowing suspected organic pathology of the intestine, which can cause obstruction.

In all cases, you should find out whether the patient diarrhea, impurities of blood or mucus in the stool, and pain during bowel movements.

Physical examination in the general examination: examination of the heart and lungs; BP measurement.

• Inspection of the abdomen: flat belly or bloated: is there any scars after surgery; whether the hernia (visible or palpable).

• Abdominal palpation: pain and the most painful area; the state of the anterior abdominal wall — the abdomen is soft or tense (with the inflammation of the peritoneum); palpable education; localization of hernias; ascites; palpation of the external genitalia, hernias, pain or swelling of the testicles.

• Rectal examination: the overhang of the front wall and tenderness of the rectum in the presence of abdominal purulent exudate; swelling, bleeding; the condition of the prostate gland; the presence of stool in the rectum, the color of feces.

• Auscultation abdomen — informative study. Increased noise peristalsis and splashing talk about mechanical obstruction; no noise — a paralytic ileus. Voiced numerous bowel sounds can only listen to the first stages of mechanical intestinal obstruction, bowel sounds then disappear.

Instrumental research:

- Panoramic radiography of the abdomen: the air in the abdominal cavity (perforation of a hollow organ); stretched intestinal loops filled with liquid and gas; fluid levels (ileus).
- US. The fluid in the abdomen: in acute cholecystitis, abscesses, gynecological diseases, detection of aortic aneurysms, which can cause acute abdomen at break.
- Chest X-ray: the identification of fluid in the pleural cavity, signs of pericarditis or congestive heart failure.
- ECG is always displayed with suspected heart disease.
- Diagnostic laparoscopy.

Emergency laparoscopy: The method allows for the differential diagnosis of acute appendicitis, acute cholecystitis, perforated gastroduodenal ulcer, acute pancreatitis, intestinal infarction, acute diseases of the pelvic organs. At the same time if there are indications at the same time it can be carried out drainage of the abdominal cavity, packing bags, cholecystectomy.

Treatment goals of therapy: In outpatient — urgently resolve the issue of hospitalization in a hospital surgical for diagnosis and surgical treatment.

Drug-free treatment: In acute abdomen should be carried out by local hypothermia (ice pack on his stomach), thermal procedures are contraindicated. Repeated vomiting is an indication for the introduction of a nasogastric tube.

Drug therapy: Contraindicated administration of narcotic and non-narcotic analgesics, hypnotics and antipsychotics to establish a definitive diagnosis because they can change, "gloss over" the clinical symptoms. When cramping pain is permissible to use spasmolytics.

Infusion therapy should be started prior to transport to the hospital. It should start measuring the volume of urine. The most severe disorders of water and electrolyte balance must be corrected quickly before the operation, rather than delaying it.

Surgical treatment is carried out immediately after the final diagnosis, sometimes carried out by peritonitis or intra-abdominal bleeding, the cause of which is set only during the operation.

TOPIC 5: Differential diagnosis of jaundice and hepatosplenomegaly. Diagnosis and treatment of diseases of the liver, gallbladder and biliary tract in an outpatient setting, medical tactics, medico-social examination, clinical examination. Emergency medical care in hepatic colic.

Jaundice: Concept. Classification. Differential diagnosis of jaundice.

Jaundice is clinically defined when serum bilirubin levels above 50 mmol / L.

Causes of jaundice:

1. ***Hemolytic***: neonatal, hemolysis, Gilbert's syndrome, syndrome Crigler-najjar.

2. ***Hepatic:*** Viral hepatitis, alcoholic, hepatitis cirrhosis, Liver metastases, Drug jaundice, Autoimmune, hepatitis, liver abscess, Hepatoma, lymphoma Leptospirosis, Budd-Chiari syndrome, Heart failure, pregnancy, Heart surgery, disease Vilsona – Konovalov, syndrome Dubina – Johnson,Disease "graft versus host".

3. ***Cholestatic:*** Stones in the ducts, Pancreatic cancer, Primary biliary cirrhosis, cholangiocarcinoma, Sclerosing cholangitis Benign strictures, pancreatitis, Portal lymphadenopathy, Chronic pancreatitis, Cyst of the common bile duct, pregnancy, Biliary atresia Holangiopatiya AIDS, parenteral nutrition, hepatic granulomas.

Survey. *Medical history*:

• occupation (alcohol consumption, contact with animals, exposure to industrial hazards);

• travel abroad in the past or recent (endemic regions of hepatitis, malaria);

• contact with patients with jaundice;

• injection (abuse of drugs, blood and plasma transfusions, tattoos);

• medicine (alternative medicine — herbal teas, Chinese medicine);

• sexual intercourse;

• consumption of oysters, crabs (hepatitis A).

To determine jaundice need good daylight. Initially, there ikterichnost sclera (usually preceded by the change in color of urine). Jaundice is determined, and when viewed from the soft palate, especially in patients with discolored sclera (elderly patients). The intensity of jaundice is not a reliable indicator of its causes. Need search symptoms of acute or chronic liver disease or cholestasis.

Other important symptoms are:

• in younger patients may be hepatitis, caused by Epstein – Barr virus;

• surgery on the bile ducts in the past — possibly jaundice;

• fever implies cholangitis, although hepatitis may also be a slight increase in temperature;

• gall bladder is usually palpable when obstructive jaundice due to tumor. Every fourth patient is caused by obstruction of the common bile duct stones;

• portosystemic encephalopathy;

• liver breath in patients with hepatic impairment;

• asterixis — flapping tremor.

Rarely arterial auscultated noise over the liver in hepatomas or acute alcoholic hepatitis. Discolored feces during rectal examination observed in cholestatic jaundice.

Diagnostics

Blood tests. Liver enzymes: aspartic transaminase (AST) and alkaline phosphatase (ALP) are markers of liver function abnormalities. Alanine transaminase (ALT) is more specific than AST. Increased AST 700 IU / L is very rare with alcoholic hepatitis and usually indicates a connection of a viral infection or toxic effects of drugs (eg, acetaminophen). Level

gammaglyutamiltranspeptidazy (GGT), increased alkaline phosphatase at a high level of liver, not the bone of origin. It is not a very reliable test for determining alcohol abuse, however, the level of GGT changes after his admission. Reliable marker of liver function are the serum albumin level, prothrombin time and international index. Serum albumin levels can vary during the redistribution of body fluids.

Urinalysis. When nadpechenochnoy jaundice bilirubin is absent (the urine is clear, not orange). Urobilinogen not at full cholestasis.

Other tests. Ultrasound is used to evaluate the expansion of bile ducts or liver metastases. It also helps to determine the size of the liver, spleen, pancreas, portal circulation, to reveal lymphadenopathy and ascites. For the diagnosis of cirrhosis of the method is not reliable.

CT is performed in obese patients, or if difficulties arise ultrasound (usually due to flatulence).

Chest radiography is performed to exclude metastases or tumor bronchial ERPHG - in pathology in pancreatic ducts or biliary ducts.

Cholestatic **jaundice**: Ultrasound examination→ dilated bile ducts → percutaneous cholangiography → liver biopsy→ Endoscopic retrograde cholangiopancreatography (ERCP).

Hemolytic jaundice:

• blood smear;

• counting the number of reticulocytes;

• coombs, determination haptoglobins serum (when no hemolysis);

• consultation of a hematologist (a bone marrow examination, a test to exclude the Hema paroxysmal nocturnal hemoglobinuria).

Hepatic jaundice. Viral markers: the surface antigen of hepatitis B virus (HBsAg), antibodies to hepatitis A (anti-HAV IgM), antibodies to hepatitis C. If you suspect that the toxic effects of drugs determine the level of paracetamol for admission of the patient to the hospital. In the absence of viral markers detect antibodies to smooth muscle, antinuclear and mitochondrial antibodies for the diagnosis of autoimmune hepatitis and primary biliary cirrhosis. The reaction of Paul-Byunnelya conducted for the diagnosis of infectious mononucleosis, a blood serum test — to diagnose atypical infections (eg, Leptospira, Mycoplasma) if viral markers were not identified. Determination of serum iron and total iron binding capacity and serum ferritin (hemochromatosis), α-antitrypsin, serum copper and ceruloplasmin, and daily excretion of copper in the urine (Wilson's disease) is carried out if the test results are negative for markers of viral hepatitis, and no autoantibodies.

Medicinal liver damage. Hepatotoxic effects are divided into dose-related (occur at a sufficiently high dose of the drug) and idiosyncratic (dozonezavisimye). The diagnosis is likely if abnormal liver function or jaundice develops within 3 months from the start of a new drug. A slight

increase in AST (up to three standards) after receiving potentially hepatotoxic drugs (especially isoniazid, rifampicin) is not an indication of their abolition, as usually occurs improvement.

Postoperative jaundice. Possible causes:

• anesthetics — re-use of halothane for 4–6 weeks. To re enflurane anesthesia is preferable;

• septicemia — the basis of cholestasis;

• pancreatitis — swelling of the pancreas can cause obturation of the common bile duct;

• benign postoperative cholestasis — self-limiting, 1–2 weeks, especially after heart surgery;

• large hematomas resorption — jaundice occurs only if there is a metabolic disorder, such as Gilbert's syndrome.

Jaundice in pregnancy: first trimester: uncontrollable vomiting pregnant women — 10 % of jaundice, especially in patients with Gilbert's syndrome; samorazreshaetsya, liver failure never occurs.

Third trimester: intrahepatic cholestasis of pregnancy that usually precedes itching; recur in subsequent pregnancies, disappearing within 2 weeks after birth; samorazreshayuschiysya. Acute fatty liver during pregnancy: nausea, abdominal pain, encephalopathy; potentially fatal without proper delivery. HELLP-syndrome — a potentially serious liver disease characterized by pre-eclampsia, hemolysis and thrombocytopenia.

Gilbert's syndrome: Usually determined small increase in unconjugated bilirubin (up to 5 % of the population). This is not grounds for an ultrasound or other studies in the absence of symptoms. Liver enzymes within the normal range, the bilirubin level rises during fasting. Jaundice is rare, except for accompanying anorexia. He requires no treatment.

Hepatomegaly, splenomegaly, hypersplenism: concept, the reasons; diseases associated with hepato- and / or splenomegaly, differential diagnosis.

Hepatomegaly — abnormal enlargement of the liver. Hepatomegaly characteristic of many liver diseases, particularly for viral hepatitis, alcoholic fatty liver for and others. Also hepatomegaly often observed in congestive heart failure. In this case, hepatomegaly occurs as a result of stagnation of blood in the portal vein of the liver and blood overflow hepatic venous sinuses. Hepatomegaly may also be observed in chronic infections, intoxications, in the removal of the liver which plays an important role, and these infections, which can parasitize pathogens in the liver (eg, malaria). It is hepatomegaly and liver tumor infiltration — both primary liver carcinoma and metastases of malignant tumors of other organs to the liver. Particularly severe hepatomegaly seen in leukemia and other hematological malignancies, as a result of a massive infiltration of the liver tissue malignant leukemia or lymphoblastic cells, or as a result of the formation in the liver foci vnekostnomozgovogo (extramedullary)

hematopoiesis. In these cases, the liver sometimes reaches enormous proportions, occupying more than half of the abdominal cavity and sometimes reaching the weight of 10–20 kg. Dimensions of the liver is best determined through CT or ultrasound. It is important to assess the contours of the body fabric and pattern; The increase in those or other tissue sites; "Rocky" texture suggests a tumor; pain on palpation indicates inflammation (hepatitis) or a rapid increase in the size of the body (right heart failure, Budd-Chiari disease (Budd-Chiari syndrome), fatty infiltration).

The most important causes of hepatomegaly. Vascular congestion: right heart failure (including loss tricuspid valve), Budd – Chiari disease.

Infiltrative processes: accumulation of fat ("fatty" liver, for example, ethanol, diabetes, excess parenteral nutrition, pregnancy), lymphoma or leukemia, extramedullary hematopoiesis, amyloidosis, granulomatous hepatitis (caused by tuberculosis, atypical mycobacteria, cytomegalovirus, sarcoidosis), hemochromatosis, a disease Gaucher glycogen. Inflammatory diseases: viral or drug-induced hepatitis, cirrhosis.

Tumors: Primary carcinoma, metastatic cancer, focal nodular hyperplasia, hepatic adenoma.

Cysts: Polycystic.

Splenomegaly: is almost always secondary to other diseases, which is a lot, as well as possible ways to classify them. Myeloproliferative and lymphoproliferative diseases, storage diseases (eg Gaucher's disease), and connective tissue disorders are the most common causes of splenomegaly in temperate climates, while infectious diseases (such as malaria, kala-azar) predominate in the tropics.

The cause pronounced splenomegaly (spleen palpable 8 cm below the costal arch) are usually the following diseases: chronic lymphocytic leukemia, non-Hodgkin's lymphoma, chronic myeloid leukemia, polycythemia vera, myelofibrosis with myeloid metaplasia, and hairy cell leukemia.

Splenomegaly may lead to cytopenias.

Common causes of splenomegaly: congestive splenomegaly (Banti's disease).

Cirrhosis. External compression or thrombosis of portal or splenic vein. Some vascular malformations. Infectious or inflammatory disease. Acute infections (eg, infectious mononucleosis, infectious hepatitis, subacute bacterial endocarditis, psittacosis). Chronic infection (eg, miliary tuberculosis, malaria, brucellosis, Indian visceral leishmaniasis, syphilis). Sarcoidosis. Amyloidosis. Connective tissue diseases (eg, SLE, Felty's syndrome). Myeloproliferative and lymphoproliferative diseases. Leukemias, particularly chronic lymphocytic leukemia and chronic myeloid leukemia. Inspection. Normally, up to 3 % of people have palpable spleen. Furthermore, palpable in the upper left quadrant of the mass can be caused by another reason, not enlarged spleen.

Other additional symptoms include friction noise spleen, suggesting the presence of infarction of the spleen, and splenic and epigastric sounds that indicate

congestive splenomegaly. In generalized adenopathy can assume the presence of myeloproliferative, lymphoproliferative, infectious or autoimmune disease.

Diagnostics. ultrasound is the method of choice because of its high accuracy and low cost. CT and MRI are able to provide a more detailed image of the body. MRI is particularly effective in determining the portal thrombosis or splenic vein thrombosis. Radioisotope study is a precision method of diagnostics capable of identifying additional details splenic tissue, but the method is very expensive and difficult to perform. If examination has not revealed any abnormalities except splenomegaly, the patient should be evaluated again in the range of 6 to 12 months, or if new symptoms.

Symptoms of splenomegaly

Splenomegaly hypersplenism is the criterion; spleen size correlated with the degree of anemia. You can expect an increase in the size of the spleen by approximately 2 cm below the costal arch for every 1 g of hemoglobin decline. Hypersplenism is a syndrome characterized by a decrease in the number of blood cells (leukopenia, thrombocytopenia, anemia) in patients with liver disease, manifested by hepatosplenomegaly.

What triggers Hypersplenism

Hypersplenism more common in patients with chronic hepatitis, cirrhosis, diseases of accumulation, granulomatosis with an enlarged spleen (sarcoidosis, Hodgkin's disease), proceeding with portal hypertension.

The pathogenesis of hypersplenism: increasing the pressure in the portal vein blood stagnation in the spleen. Prolonged congestion promotes connective tissue in the spleen with increase in the number of cells it mononuclear phagocyte system. The leading mechanism "hypersplenism" is the destruction of the peripheral blood cells. Because the pathogenesis of chronic hepatitis and liver cirrhosis essential belongs immune disorders, the incidence of immune cytopenias when cirrhotic stage is large enough. The questions splenogennogo braking medullary hematopoiesis, formed elements of destruction in the spleen.

Symptoms Hypersplenism. When this syndrome often develops leukopenia, which can reach a considerable degree (less than 2,000 in 1 ml of blood), neutropenia and lymphocytopenia or moderate thrombocytopenia. Anemia of Hypersplenism usually regenerative type, with anisocytosis of erythrocytes (prevalence macrocytes with cirrhosis and hepatitis). Reducing the number of cells in the peripheral blood is combined with bone marrow cellularity.

Diagnosis Hypersplenism. An important role in the diagnosis of hypersplenism assigned instrumental methods. Clinical analysis of blood, bone marrow, liver biopsy, radioisotope and immunological research.

Clinical and laboratory syndromes, liver disease (cytolysis mesenchymal inflammation, cholestasis, hepatocellular failure), the clinical significance.

Clinical and laboratory syndrome.

Cytolysis syndrome clinically jaundiced, hepatalgia. Biochemical markers — an increase in ALT, AST, aldolase, LDH, sorbitdegidrogenazy, glutamate,

hyperbilirubinemia with the increase primarily the direct fraction, increased serum concentrations of iron and vitamin B12.

Syndrome of mesenchymal inflammation clinically manifested by fever, hepatalgia, arthralgia, erythema nodosum, polyserositis, iridocyclitis. Biochemical markers: an increase in immunoglobulin G, M, A, globulin, change belkovoosadochnyh samples (thymol, sublimate, formolovoy) RBTL, changes in the amount and ratio of T-lymphocytes.

Cholestatic jaundice syndrome, persistent pruritus, xanthelasma and osteoporosis and bone pain. Biochemical markers of cholestasis — increasing the level of bilirubin, cholesterol, lipoproteins, bile acids, alkaline phosphatase, 5-nucleotidase, GGT.

The syndrome of hepatic cell deficiency manifests astenovegetative syndrome and dyspepsia; high activity — jaundice, hepatomegaly, the presence of spider veins, gynecomastia, alopecia due to violations of estrogen inactivation. Biochemical markers of the syndrome are: reduction of serum albumin, clotting factors 2, 5, 7, prothrombin, cholesterol, decreased clearance of antipyrine, delayed excretion bromsulfaleina during the corresponding sample, increasing the number of nitrogenous bases.

Edematous-ascitic syndrome is associated with hypoalbuminemia, secondary hyperaldosteronism, portal hypertension.

Portal hypertension is manifested ascites, varicose veins of the anterior abdominal wall (Head of Medusa), esophagus, stomach and upper third of the rectum.

Anemic, hemorrhagic syndrome associated with:

a) bleeding from varicose veins — due to the syndrome of portal hypertension; with bleeding from upper gastrointestinal tract when Zollinger – Ellison and hemorrhagic gastritis; at insufficiency of blood coagulation factors;

b) with iron and B12-deficiency anemia, hypoalbuminemia;

c) with increased degradation of red blood cells in the spleen when splenomegaly;

d) ethanol has direct toxic effects on the bone marrow.

Diagnostic algorithm. Once: blood group Rh-factor, blood cholesterol, blood amylase, coagulation, proteinogramma, coprogram, feces on a dysbacteriosis and worms, fecal occult blood, histological examination of biopsy, markers of viral hepatitis B, C and D.

Two-time: full blood count, reticulocytes, platelets, urinalysis, bile pigments, AST, ALT, bilirubin and its fractions, alkaline phosphatase, GGT, immunograma.

Virological diagnosis. Markers of viral infections: HBsAg — marker of infection.

Anti-HBs — a marker of past infection or successful vaccination.

anti-HBcor IgG — a marker of past infection a marker of infection.

HVcor anti-IgM — marker of acute or chronic infection replicative phase.

HBeAg — a marker of acute or chronic infection replicative phase.

anti-HBe — a marker cease replication HBV.

Markers of viral hepatitis D: anti-HDV IgM, HDV RNA.

Markers of hepatitis C: anti-HCV, HCV RNA. Anti-HCV detected in 40 % of patients after 2 months after infection, 80 % in 4 months and 95 % after 5 months.

Biochemical diagnosis (ALT, AST). Normalny or subnormal levels of ALT does not always correlate with histological hepatitis activity. ALT > 10 norms usually corresponds to bridging necrosis (to rule out other causes of liver disease!). AST > ALT: severe fibrosis, alcoholic and viral infection, other etiological factors.

Conducted by indications depending on the intended disease: esophagogastroduodenoscopy, chrespechënochnaya percutaneous liver biopsy, endoscopic retrograde cholangiopancreatography (in the differential diagnosis of cholestasis syndrome), computed tomography, laparoscopy (a biopsy of the liver).

Chronic hepatitis and cirrhosis of the liver: differential diagnosis of chronic hepatitis with cirrhosis, and primary liver cancer. Plan of inspection of the patient, the general principles of outpatient treatment, medical tactic.

CLASSIFICATION. ***The etiological*** (IWP-WCOG, Los Angeles, 1994):

1. Chronic viral hepatitis (B, C, D).
2. Chronic viral hepatitis (not otherwise characterized by a).
3. Chronic autoimmune hepatitis.
4. Chronic hepatitis not classified as viral or autoimmune.
5. Chronic drug-induced hepatitis.
6. Primary biliary cirrhosis.
7. Primary sclerosing cholangitis.
8. Wilson's disease.
9. Liver disease caused by lack of a1-antitrypsin.

As compensation:

1. Compensated.
2. Decompensated (Class B or C on the Child-Pugh or the presence of one or more complications: ascites, variceal bleeding, hepatic encephalopathy, hepatic hydrothorax).

By activity:

1. Active (available biochemical (ACT, ALT) or histologically active).
2. Inactive.

Diagnostic criteria for nonalcoholic fatty liver disease (AGA, AASLD, American College of gastroenterology, 2012):

1. Existence of steatosis according imaging techniques or morphological study of liver.
2. The absence of secondary causes of the accumulation of fat (alcohol, taking drugs , congenital metabolic disorders).

Diagnostic criteria for primary biliary cirrhosis (EASL, 2009):

1. ALP more than 2 standards or GGT greater than 5 norms.

2. Mitochondrial antibodies > 1: 40.

3. Liver biopsy: flourishing damaged bile ducts (biopsy is not necessary if the first two criteria, but allows you to evaluate the activity and the stage of disease, the need for diagnosis in the absence of specific autoantibodies).

The diagnostic criteria of alcoholic liver disease

1. The exclusion of other etiological factors

2. Alcohol at doses exceeding mode low-risk (10 g of ethanol per day for women and 20 g — for men) in combination with the features of alcoholic liver disease: increase in mean corpuscular volume (MCV) increased activity of GGT, the ratio of AST / ALT > 2 Index ANI > 0) or

3. Drinking alcohol in doses endangering health (more than 2 standard drinks per day for women and 4 doses — for men) in combination with signs of chronic hepatitis.

Diagnostic criteria of hemochromatosis (AASLD, 2010).

Genotype C282Y / C282Y in conjunction with the manifestations of iron overload In identifying genotype H63D / H63D and C282Y / H63D requires further examination to clarify the secondary iron overload.

Diagnostic criteria for disease Vilsona-Konovalovam (EASL, 2012)

1. Positive results of biochemical tests of copper metabolism:

— Reduction of ceruloplasmin.

— Free copper serum levels> 1.6 mmol / l.

— Daily urinary copper excretion> 100 mg / day.

2. Identification of genotypes H1069Q / H1069Q or H1069Q / (absence of these mutations does not rule out the disease). The main method of diagnosis of Wilson's disease is a biochemical study copper metabolism — the presence of characteristic abnormalities of biochemical tests copper metabolism conducting molecular genetic testing is not mandatory.

Classification of esophageal varices (OMED, N. Soehendra, K. Binmoeller, 1997):

1. Degree: small, uncrimped varicose veins of the esophagus (esophageal varices), which smoothed the air insufflation; vein diameter is less than 5 mm, elongated veins are located only in the lower third of the esophagus

2. Degree: esophageal varices crimped occupying less than 50 % of the lumen of the distal esophagus; vein diameter from 5 to 10 mm, veins distributed on the middle third of the esophagus

3. Degree: large and convoluted esophageal varices, which occupy more than 50 % of the lumen of the distal esophagus; vein diameter greater than 10 mm, tense, with thin wall, are located close to each other on the surface veins "red marks" (red color signs).

The term "chronic hepatitis" (hCG) indicate inflammation of the liver that lasts more than 6 months. The notion of the "cirrhosis" corresponds to a chronic diffuse

liver disease with total or nodular proliferation of connective tissue in it, a deep restructuring of its structure with symptoms of functional impairment of the liver and involvement in the pathological process of a number of organs and systems.

Etiology. Hepatitis B virus (HBV), hepatitis C virus (HCV) virus, hepatitis D (HDV, delta factor), medicines, Wilson's disease, hemochromatosis, antitrypsin deficiency, idiopathic (autoimmune "lupus").

HCG and cirrhosis can be considered as a single serial links of the pathological process. Hence the justified recognition of the unity of their etiology and pathogenesis.

Cirrhosis of the liver can not be considered only as the final stage of chronic hepatitis. It can be formed as a result of liver damage in the midst of acute viral hepatitis, or may direct its sequel.

The development of cirrhosis of the liver may be associated with acute toxic and toxic-allergic effects on the liver, resulting in severe dystrophy and massive necrosis of liver cells — hepatocytes. Not every HCG passes into the stage of cirrhosis.

Viral infection is the cause of the disease is not less than 50 % of patients with chronic hepatitis.

Another cause of chronic hepatitis and cirrhosis of the liver is considered reasonably alcohol intoxication, and alcohol has a direct toxic effect on the liver tissue, causing the development of not only the degeneration of liver cells, but inflammation and necrosis of cell structures. Pathological process may progress to cirrhosis of the liver.

The cause of the so-called cholestatic liver disease is the most common long-term patency of the violation on the grounds of the biliary tract cholelithiasis, scar-inflammatory or innate restrictions of the common bile duct or faterova nipple.

The most frequent pathology Hepatitis is a viral liver disease, which provide two important factors: the viral replication and immune response of the patient. Effect of the immune response to antigens of HCV in liver tissue determine the outcome of infection and a broad range of viral liver disease from asymptomatic carriage, acute hepatitis, chronic hepatitis, liver cirrhosis to hepatocellular carcinoma.

The clinical picture of chronic hepatitis B: Chronic hepatitis — a group of disorders characterized by a chronic inflammatory response in the liver, which flows within 6 months.

The wide clinical spectrum, ranging from asymptomatic increase of aminotransferase activity in serum, to acute, even fulminant, gepatitopodobnogo disease. Common symptoms: fatigue, malaise, anorexia, low fever; jaundice noted in severe disease. Some patients have complications of cirrhosis: ascites, variceal bleeding, encephalopathy, coagulopathy, and hypersplenism.

Cirrhosis of the liver — a chronic progressive disease characterized by lesions of both parenchyma and stroma body with liver cell degeneration, nodular regeneration of liver tissue, the development of connective tissue restructuring diffuse lobular structure of the liver and cardiovascular system.

Etiology. In our country a leading role in the development of the disease plays a viral liver disease (especially hepatitis B), which is formed in the outcome of liver cirrhosis (according to various statistics — 17–70 % of the total number of patients with cirrhosis of the liver). Chronic alcohol intoxication in the development of this disease is of great importance is usually accompanied by background of malnutrition or deficiency in food proteins and vitamins. Nutritional factors — is one of the common causes of liver cirrhosis in a number of countries with tropical and subtropical climates. In tropical countries cirrhosis often occurs with chronic parasitic helminth and liver damage.

Biliary cirrhosis develops due to obstruction of intra- and extrahepatic bile ducts and inflammation, leading to stagnation of bile (cholestasis). At the heart of PBC are aseptic destructive cholangitis and autoimmune cholangioles that due to the formation of autoantibodies to intrahepatic bile paths (interlobular and septal bile ducts). As a result, bile duct cells are subject to influence of cytotoxic T-lymphocytes and antibodies. Immune complexes in large amounts circulate in the blood and deposited in the bile ducts, causing inflammation of the immune — non-bacterial cholangitis and autoimmune cholangioles. 20–30 % of patients the cause of cirrhosis remains unclear.

Pathogenesis. The development of connective tissue in the form of scars and strands that alter the normal architecture of the liver, leading to compression of its vessels, disrupting the normal blood supply to the liver cells; Hypoxia also contributes to further disrupt the normal flow of redox enzyme reactions in the liver tissue, degenerative changes and increases, contributing to the progression of the process, creating a vicious circle. Simultaneously with the compression of the hepatic vascular nodules of regenerating liver parenchyma and connective strands increases the number of anastomoses between the ramifications of the portal vein and liver and the hepatic artery, facilitating local intrahepatic circulation. However, in these blood anastomoses bypasses surviving hepatic parenchyma, which dramatically deteriorates its blood supply and can lead to new ischemic necrosis secondary collapses, that is, to the progression of cirrhosis.

Classification of Child-Pugh.

Table 9 — Function of liver cells in liver cirrhosis evaluated by Childe-Pugh

Parameter	Points		
	1	2	3
Ascites	No	Mild, easily treatable	Intense, poorly treatable
Encephalopathy	No	easy (I–II)	Severe (III–IV)
Serum bilirubin, mmol / L (mg%)	less 34 (2,0)	34–51 (2,0–3,0)	more 51 (3,0)
PTT (s) and PTI (%)	1–4 (more 70)	4–6 (40–70)	more 6 (less 40)
Serum bilirubin, mol / L, in primary biliary cirrhosis	17–67	68–169	more 170
Albumin, g	more 35	28–35	less 28

Class cirrhosis exhibit, depending on the amount of points for all parameters. The total score of 5–6 corresponds to class A, with the amount of 7–9 — the class B, but for a total of 10–15 points exhibiting class C.

System SAPS criteria. In recent years, to determine the prognosis of patients at the time of gastrointestinal bleeding, coma, sepsis and other complications, the system of criteria used by SAPS (Simplified Acute Physiology Score), which includes the basic physiological parameters: age, heart rate, respiratory rate, systolic blood pressure, temperature body, diuresis, hematocrit, white blood cells, urea, potassium, sodium and bicarbonates of plasma and hepatic coma stage.

Clinical picture: The main clinical signs of cirrhosis of the liver, to distinguish it from hepatitis and other disorders of the body, are:

1) the presence of an enlarged liver and spleen, dense (in advanced cases, the size of the liver may be reduced);

2) ascites and other signs of portal hypertension;

3) so called hepatic stigma (telangiectasia).

The cause of pain in the liver are usually enlarged liver and stretching of the capsule, the emergence of foci of necrosis, closely located to the capsule, and reactive involvement in the process of closely spaced sites hepatic capsule.

Cirrhosis of the liver, biliary and especially postnecrotic, often accompanied subfebrilnoi body temperature. Some authors regard the fever as a manifestation of progressive necrosis of hepatocytes and the process activity. Jaundice at postnecrotic cirrhosis and portal may be a manifestation of hepatocellular failure associated with degenerative processes and necrosis of liver cells. With long-term course of the disease due to the accumulation of bilirubin in the skin and move it into biliverdin skin of the patient acquires a greenish tint. In some cases, when biliary cirrhosis may be a brownish tinge to the skin, caused by the accumulation of melanin in it.

On examination, the patient in most cases you can identify ***liver symptoms***:

a) vascular "stars" — cutaneous arteriovenous anastomoses observed in patients. Vascular "star" located in the mucous membrane of the nose, are often the source of nasal bleeding. The appearance of spider veins is due to the increased amount of estrogen circulating in the blood, is not destroyed with the necessary speed in the affected liver;

b) palmar erythema (liver palms) — spilled bright red coloring of the palms diffuse or limited areas of thenar and hypothenar palm and fingertips; palm is usually warm. It is believed that the cause of erythema are multiple arteriovenous shunts develop in the skin with cirrhosis of the liver;

c) red glossy lips, red mucous membrane of the mouth, liver red, painted, magenta language detected in many patients with cirrhosis of the liver;

d) signs of hormonal disorders. In men, there are gynecomastia and other female sexual characteristics, which is caused by metabolic disorders of

estrogen due to liver disease, and excessive content of their blood. Women often have irregular menstruation (amenorrhea), impaired reproductive function;

e) xanthomatosis plaques on the skin: yellowish — brown patches, often settling for centuries (xanthelasma), sometimes on the palms and on the chest, back, knees (xanthomas), observed in patients with biliary cirrhosis. Their appearance is associated with increased levels of lipids and cholesterol in the blood and the local intradermal deposition of cholesterol.

Friendly enlarged spleen (**gepatolienal syndrome) is observed in most patients. The occurrence of this syndrome is caused by organic disorders of blood** circulation resulting in intrahepatic venous outflow obstruction-reclaimed sites and the formation of connective tissue septa with desolation most of the sine wave. Because of these reasons is prevented outflow of blood from the liver, is significantly increased portal pressure — up to 400–600 mm of water. Art. (Normally no more than 120–150 mm of water). For a long time portal circulatory disturbances can be compensated for the development of anastomoses. The most important natural ***portocaval anastomoses*** are:

a) governmental hemorrhoidal plexus, is the cause of bleeding from the rectum;

b) in the area of esophageal-gastric plexus generator of large varices;

c) When portal hypertension veins around the navel (up to 1 cm in diameter or more) in different directions, forming a kind of painting, called the head of Medusa — caput Medusae.

Esophageal varices (and gastric cardia), hemorrhoidal veins, caput Medusae up triad characteristic of portal hypertension. Fourth, the most characteristic feature of portal hypertension is ascites. The major importance in the development of ascites is portal hypertension. In addition, the matter is characteristic of cirrhosis hypoalbuminemia, leading to a decrease in plasma oncotic pressure, as well as secondary hyperaldosteronism.

Hemorrhagic syndrome occurs in approximately half of patients with liver cirrhosis. Massive bleeding from esophageal varices and stomach, as well as hemorrhoids are caused by increased pressure in these veins, thinning of their walls or injury to them. Repeated nosebleeds, uterine bleeding, skin hemorrhages caused by bleeding disorders as a result of violations of liver involvement in the development of certain clotting factors.

Current. Cirrhosis usually tend to progression. There are decompensated cirrhosis and compensated. In compensated cirrhosis, the complaint may be missing, or it may occur with minor symptoms and be detected during a random inspection on the basis of increase in liver and spleen, liver presence of "characters." Changes in laboratory parameters are also insignificant: marked hypergammaglobulinemia, moderate decrease in the absorption and excretory functions of the liver, increased erythrocyte sedimentation rate.

The most frequent ***complications*** of cirrhosis are profuse bleeding varices cardiac segment of esophagus and stomach, bleeding hemorrhoids (with

cirrhosis, portal hypertension from occurring). Gastrointestinal bleeding in the form of bloody vomiting and melena are the result of rupture of varicose governmental units of the lower third of the esophagus and gastric cardia. may develop liver cancer (cancer, cirrhosis), the incidence of which, according to some estimates, close to 20 %, as well as stomach ulcers, which are often accompanied by liver cirrhosis.

The terminal period of the disease, regardless of the form of cirrhosis is characterized by the progression of symptoms of functional failure of liver cells with the outcome of hepatic coma. Gastroesophageal bleeding and hepatic coma — the two most frequent immediate causes of death in patients with liver cirrhosis.

Other complications of liver cirrhosis (usually associated with a large specific complications):

• hepatorenal syndrome;
• spontaneous bacterial peritonitis;
• hepatic hydrothorax;
• the portal hypertensive gastropathy;
• gepatopulmonarny syndrome;

Associated Conditions

• malnutrition;
• bacterial infectious complications, including spontaneous.

CLASSIFICATION ascites in severity

1) degree. The fluid in the abdominal cavity is defined only by ultrasound;
2) (medium) degree. Ascites is moderate symmetrical distension of the abdomen;
3) (severe) degree. Ascites is marked distension of the abdomen (ascites tense).

Ascites CLASSIFICATION FOR presence of complications

Uncomplicated

Ascites, who is not infected and not accompanied by the development of hepatorenal syndrome (GRS) complicated:

• The infected ascites — defined term spontaneous bacterial peritonitis (SBP) as infected ascites in the absence of damage to the gastrointestinal tract.
• The presence of hepatorenal syndrome (GRS).

Hepatorenal syndrome

Progressive functional prerenal acute renal failure in patients with severe hepatic impairment and ascites.

Types (the International Ascites Club, 2007).

Type I (median survival of 2 weeks).

Rapidly progressive decrease in renal function: a doubling of the initial serum creatinine to > 2.5 mg / dL (226 mol / L) of less than 2 weeks.

Clinical pattern: acute renal failure.

Type II (median survival of 4–6 months).

Moderate renal dysfunction: an increase in serum creatinine with stable or slow progression.

The average life expectancy of patients — 3–6 years, in rare cases — up to 10 years or more. Death usually occurs from liver failure, often accompanied by bleeding. In secondary biliary cirrhosis prognosis depends largely on the reasons for the blockage of the gall of the prophet, and the possibilities of their elimination.

Diagnosis and differential diagnosis

Laboratory blood tests usually reveal anemia, leukopenia, thrombocytopenia, and increased ESR. Particularly severe hypochromic anemia were observed after bleeding. With moderate macrocytosis Anemia can also be the result of hypersplenism. In rare cases, develop megaloblastic anemia due to vitamin B12 deficiency.

The urine is found in large quantities urobilin, in severe jaundice — and bilirubin. Number stercobilin in feces decreases.

The defeat of the liver cell changes protein appears factors: a decrease in serum albumin concentration and hyperglobulinemia. When aggravation of cirrhosis decreased serum cholinesterase activity, increased activity of serum transaminases, with biliary cirrhosis observed high levels of serum alkaline phosphatase.

A manifestation of the human liver with cirrhosis is the reduction of serum fibrinogen and prothrombin, the synthesis of which the liver cells. Changing these parameters of coagulation and protivosverty-ing systems of blood reflects liver cirrhosis characteristic tendency to bleeding diathesis.

For the diagnosis of cirrhosis is widely used ultrasound also allows to determine the size of the liver and spleen, and their structure.

X-ray method of investigation reveals the veins of the esophagus. In lean people with flatulence sometimes during fluoroscopy can see the shadow of an enlarged liver and spleen.

Diagnosis is based on characteristic clinical picture of the disease. He confirmed in the first liver biopsy, ultrasound data, scan, computed tomography, angiography and other methods.

Cirrhosis is distinguished from chronic hepatitis, liver disease, its focal lesions in chronic infections, primary or secondary (metastatic) tumor lesions, secondary lesions in the liver syndrome Chiari helminth liver damage (primarily by hepatic echinococcosis), congestive hepatic fibrosis , aleukemic forms of leukemia. When steatosis (fatty liver) liver is usually enlarged, but its edge is not as sharp as in cirrhosis. Enlargement of the spleen is usually not observed.

When the tumor-induced liver injury is comparatively rapid increase in symptoms (several months — 1–1.5 years). inherent liver cancer: progressive weakness, weakness, cachexia, loss of appetite, vomiting, nausea, feeling of heaviness and continual pain in the right upper quadrant or epigastric pain, liver gradually increased, often bumpy, uneven edge, anemia, ascites, obstructive jaundice, fever, frequent nosebleeds, spleen is not enlarged. The clinic can manifest with distant metastases. The most common liver tumors metastasize to

the liver itself, regional lymph nodes (nodes or liver gate hepatoduodenal ligament), lungs, pleura, peritoneum, kidneys, pancreas, bones. Screening for hepatocellular carcinoma (early diagnosis) is based on measuring in at-risk groups (chronic hepatitis, cirrhosis) alpha - fetoprotein (AFP) every six months.

Treatment. GENERAL PRINCIPLES outpatient management of patients with compensated cirrhosis Good nutrition

Physical activity by limiting strenuous exercise activities for the conservation of functional liver tissue:

• Complete abstinence from alcohol.

• Limit use of drugs.

• Termination of contact with toxins.

• Prevention of parenteral hepatitis viruses (including reasonable limitation of invasive procedures).

Where there is evidence of active inflammatory process in the necrotic liver medicines:

• ademetionine;

• silymarin / silibinin;

• ursodeoxycholic acid;

• essential phospholipids.

Restricting the use of drugs. Failure to physiotherapy. Refusal of herbal medicine. Restricting dietary supplements. Refusal of mineral water with a high sodium content. Restriction intravenous solutions containing sodium.

It is known that the liver supine blood passes much more than in the vertical position of the patient, which improves the metabolic processes in the tissues. Physical activity adversely affects the disease and worsen its course. Bed rest helps to restore liver cells.

The second important trend in the treatment of patients with diet therapy is the principle under which the CG and cirrhosis of the liver is to appoint a diet rich in high-grade proteins, carbohydrates and vitamins. Recommended small in volume, but frequent meals. High-calorie food protects the liver from injuries and accelerates repair of hepatocytes.

Currently, alpha ***interferonterapiya*** is the primary means of causal treatment of viral hepatitis, the most studied and clinically meaningful results giving. The highest efficiency of a positive response to therapy - 3 million. IU of interferon 3 times a week for 12 months., for patients previously untreated with interferon. However, 38 % of patients are resistant to prolonged interferon. Treatment of patients with relapsed after a course of interferon or the answer to it is a difficult task. Some patients may be an effective increase in the dose up to 6 million. IU 3 times a week. In other cases it is possible to recommend a combination therapy with interferon and ribavirin (He received a dose of 1000–1200 mg per day in two divided doses). The dose of interferon is 3 million IU 3 times a week. Course duration 24 weeks. In the treatment of chronic liver

diseases of any etiology it is advisable to use metabolic, coenzyme, membranstabiliziruyuschih drugs, has a positive effect on the functional status of hepatocytes. Especially noticeable positive effect hepatoprotective therapy outpatient stage patients after acute Hepatitis disease.

Preventive drug therapy or prevention of esophageal gastric variceal bleeding: Non-selective beta-blockers (objective: reduction of heart rate at 25% or up to 55 beats / min):

• Propranolol — initial dose of 20 mg 2 times / day orally, to increase the maximum tolerated (maximum 160 mg / day, usually 20–40 mg four times a day) or

• nadolol initial dose of 40 mg 2 times / day by mouth, increasing to the maximum tolerated (maximum dose of 160 mg / day) or

• carvedilol: an initial dose of 6.25 mg 2 times / day by mouth, increase to maximum tolerated (maximum dose of 50 mg / day) Endoscopic ligation / hardening intolerance of beta-blockers and esophageal varices 2–3 tbsp.

To prevent a recurrence can be a combination of non-selective beta-blockers with isosorbide mononitrate 10–20 mg 2 times / day inside.

Vitamin E actively inhibits lipid peroxidation and the formation of free radicals involved in the syndrome of cytolysis of hepatocytes.

Pyridoxal — vitamin B6 coenzyme participates in the decarboxylation of amino acids, tryptophan, methionine, improves lipid metabolism.

Riboxinum — a precursor of ATP, stimulates the synthesis of nucleotides, increases the activity of enzymes of the Krebs cycle.

Essentiale — complex preparation in capsules contains phospholipids, linoleic acid, vitamins B1, B6, B12, E, PP, sodium pantothenate. For the basic *immunosuppressive*, antifibrotic therapy are different agents: methotrexate, ursodeoxycholic acid, D-penicillamine, cyclosporin, azathioprine, colchicine, chlorambucil, glucocorticoids.

Treatment with ursodeoxycholic acid. The mechanism of action of this drug: immunomodulatory effect of ursodeoxycholic acid; choleretic effect of ursodeoxycholic acid, which contributes to the elimination of the phenomena of cholestasis. Ursodeoxycholic acid is more effective in the treatment of PBC in pretsirroticheskoy step in this case is regarded as the drug of choice. The initial daily dose is 12–15 mg / kg, duration of treatment — a few months, sometimes a year or more. In the absence of effect may increase the daily dose of 18–20 mg. During decompensation treatment necessarily carried out in a hospital. Assign diet therapy, used ***corticosteroids*** (15–20 mg / day. Prednisolone or equivalent dose of triamcinolone), vitamins. Corticosteroids are contraindicated in liver cirrhosis complicated with esophageal varices, and the combination of cirrhosis with peptic ulcers of the stomach and duodenum, reflyuksezofagitom. When ascites prescribed diet with salt restriction and periodic use of diuretics. In order to prevent gastroesophageal bleeding in patients with portal hypertension is widely performed ***surgery*** — the imposition of additional portocaval anastomoses that can help reduce pressure in the portal, and, consequently, in the esophageal veins.

Treatment of ascites in different clinical situations

The first episode of moderate ascites

• Do not diuretic therapy (sodium Mode restrictions).

• If you are not effective: spironolactone 100 mg / day in the morning during the meal; with no effect (weight loss < 2 kg / week) — increasing the dose of 100 mg every 7 days up to a maximum dose of 400 mg per day; if not the effectiveness or the maximum dose of hyperkalemia — in addition to furosemide gradual increase in dose from 40 to 160 mg.

As the reduction of ascites dose diuretics reduce to the lowest possible, ensuring the absence of ascites *Recurrent ascites:*

• once in the morning inside spironolactone 100 mg with meals + furosemide 40 mg before meals;

• when there is insufficient effect after 3–5 days the dose doubled (spironolactone 200 mg / day + furosemide 80 mg) and then every 3–5 days increased in the same proportion to the maximum dose of spironolactone 400 mg, 160 mg of furosemide.

As the reduction of ascites dose diuretics reduce to the lowest possible, ensuring the absence of ascites *Severe ascites* (grade 3 ascites., Tense ascites).

Single paracentesis evacuating a large volume of fluid (5–10 L) was combined with infusion therapy at the end paracentesis (albumin 8–10 g per liter of the evacuated fluid, plasma expanders, crystalloids) followed by administration of a diuretic.

Refractory ascites

Serial large volume paracentesis one once every 2–3 weeks in conjunction with the end of the infusion therapy paracentesis.

When decompensated cirrhosis and beginning signs of hepatic encephalopathy and adsorbents used disaccharide and helps to eliminate toxins.

Lactulose is the "gold standard" in the treatment of PE. Such a high efficacy of therapy using lactulose associated with multidimensional effect in colon and ileum. Dose lactulose inwardly selected individually and ranges from 30 to 120 ml per day. The optimal dose is considered to be one in which the patient is observed while taking the drug soft consistency chair 2–3 times a day. To reduce the formation of ammonia in the colon using antibiotics currently is vancomycin, ciprofloxacin, metronidazole. Take the average dose for 5–7 days.

Chronic cholecystitis, gallbladder dysfunction and dysfunction of the sphincter of Oddi: the plan of patient examination, differential diagnosis. Treatment.

Chronic cholecystitis — an inflammatory disease of the gallbladder wall, combined with a motor-tonic biliary system disorders. Chronic cholecystitis is usually caused by opportunistic pathogens — Escherichia, streptococci, staphylococci, rarely — Proteus, Pseudomonas aeruginosa, enterococci. Germs enter the gallbladder hematogenous, lymphogenous and contact (mostly from the

gut) by. The cause of inflammation of the gallbladder may be an infestation of parasites. An important predisposing factor for the development of chronic cholecystitis is considered a violation of outflow of bile and its stagnation, the disease usually occurs on the background of cholelithiasis or biliary dyskinesia. On the other hand, chronic inflammation of the gallbladder is always accompanied by a violation of its motor-evacuation function and contributes to the formation of stones.

The clinical picture of chronic cholecystitis characterized by a long progressive course with periodic exacerbations. The pain is localized in the right upper quadrant, at least in the epigastric region, radiating to the right shoulder blade, collarbone, shoulder, at least — in the left upper quadrant. Intense paroxysmal pain is characteristic of the inflammatory process in the cervix duct and gall bladder, the constant — with the defeat of the body and the bottom of the bladder. In chronic cholecystitis, accompanied by hypotonic dyskinesia, pain less intense, but a constant, nagging , aching, almost does not stop the pain can occur when periholetsistite. In acute cholecystitis is characterized by fever. Atypical forms of chronic cholecystitis watch 1/3 patients. Kardialgicheskaya form is characterized by long dull pain in the heart that occur after a heavy meal, often in the supine position. There may be an arrhythmia, most types of arrythmia. ECG — flattening and sometimes inversion of T wave forms characteristic for ezofagalgicheskoy persistent heartburn, combined with a dull pain behind the breastbone. After a heavy meal may feel "stake" in the chest. The pain is prolonged. The intestinal form of low-intensity manifested, not clearly localized pain around the abdomen, bloating, tendency to constipation.

Dysfunction of the gallbladder and sphincter dysfunction Odie

Dysfunctional disorders of the biliary tract are represented by two states: dysfunction of the gallbladder and sphincter of Oddi dysfunction. The normal function of the gallbladder and sphincter of Oddi.

Biliary tract or biliary excretion system covers extrahepatic bile ducts, gall bladder and the sphincter of Oddi. The right and left hepatic ducts merge into the common hepatic, joined by the cystic duct and formed choledoch (common bile duct). At the junction of the common bile duct, pancreatic duct, and duodenum is sphincter of Oddi, which regulates the flow of bile and pancreatic juice in the intestine.

In interdigestive up to 90 % of hepatic bile enters the gallbladder and is accumulated in it. This process is due to the contraction of the sphincter of Oddi, which leads to an increase in pressure in the common bile duct. Increasing the pressure stimulates holedohopuzyrny reflex and relaxation of the gallbladder, causing bile flows into the bubble. The accumulation of bile in the gall bladder and the gradual concentration allows it to maintain a balance of pressure bile flow in the closed sphincter of Oddi. For the flow of bile into the gallbladder requires two conditions: the ability of the bladder to relax and traversed the cystic duct.

Biliary pain is described as an episode of severe pain, localized in the epigastric or right upper quadrant, at least 30 minutes. The pain typically

radiating to the right shoulder, may be accompanied by nausea and vomiting. Patients during an attack can not find a position that facilitates pain.

The mechanism of biliary pain, regardless of the reason, she was called, is associated with obstruction of the biliary tract, leading to increased pressure in it and expand it. When long-term obstruction, usually joined inflammatory changes. The most common cause is obstruction of the biliary tract cholelithiasis. However, disruption of the functioning of the gallbladder and / or sphincter of Oddi may also lead to transient obstruction and increased pressure in the biliary excretion.

The most frequent disease of the gall bladder — gallstones (cholelithiasis) — only 10–20 % of patients with clinical symptoms occurs (biliary pain), the disease is asymptomatic at rest. In the absence of cholelithiasis incidence of biliary pain is estimated to 2.4 % in the population, and its cause in most cases is a dysfunction of the gallbladder. Currently, the only objective characteristic of the formation of biliary pain dysfunction of the gallbladder emptying is considered to reduce it. Violation of contractile activity primarily associated with changes in the muscular layer of the wall of the gallbladder or in violation of its innervation that can be observed in autonomic neuropathy (diabetes).

Dysfunction of the sphincter of Oddi. The diagnosis of sphincter of Oddi dysfunction in most cases installed after cholecystectomy, and very rarely in patients with intact gallbladder.

Diagnostic criteria of sphincter of Oddi dysfunction. The main clinical criteria of sphincter of Oddi dysfunction — biliary pain; a change in the sphincter of the pancreatic pain in nature approaching Pancreatitis similar: radiate to the back and partly facilitated by the torso forward.

Classification. In accordance with the prevalence of biliary or pancreatic disorders of the sphincter of Oddi and allocate biliary similar and Pancreatitis similar options:

1. Biliary similar option. After cholecystectomy there are recurrent episodes of biliary pain, which may be accompanied by biochemical signs of transient obstruction of the biliary tract (increased transaminases, alkaline phosphatase and bilirubin conjugated) and changes during endoscopic retrograde cholangiopancreatography (ERCP).

2. Pancreatitis similar option. Clinically manifested classic symptoms of pancreatitis with epigastric pain that often radiates to the back and are accompanied by an increase in amylase and / or lipase.

Diagnosis of chronic cholecystitis. The main diagnostic symptom — biliary pain that develops in the absence of structural changes and the cause of which is impaired emptying of the gallbladder. Even a single but significant expression episode of biliary pain may require a whole complex of studies to establish its etiology. The pain may be accompanied by the following symptoms: irradiation under the right shoulder or in the back, combined with nausea and vomiting, the appearance of pain attack after eating and / or during the night.

In the analysis of the blood in the acute phase often find increased erythrocyte sedimentation rate, leukocytosis, leukocyte formula shift to the left, eosinophilia. When complicated forms in the blood can increase levels of bilirubin, cholesterol, transaminases. When contrast study gallbladder (cholecystography, cholangiography) may reveal a violation of concentrating ability (intravenous cholangiography cholecysto-accumulation of the drug lasts more than 90 minutes), a violation of the motor function (delay emptying), strain (uneven contours of the gallbladder) with periholetsistite. The main method of diagnosis of cholecystitis, ultrasound, not only to establish the lack of stones, but also to evaluate contractility and the state of the wall of the gallbladder (cholecystitis suggests a chronic thickening of its more than 4 mm). When the bilirubin level above 51 mmol / l and clinically apparent jaundice to clarify its reasons for endoscopic retrograde cholangiopancreatography spend.

The "gold standard" evaluation of gallbladder emptying is hepatobiliary scintigraphy.

The differential diagnosis is carried out primarily with duodenal ulcer, chronic duodenitis. It is necessary to take into account the features of occurrence of pain in these diseases, seasonality of exacerbations. The decisive role played by the results of endoscopic examination of the stomach and duodenum. It is sometimes difficult to distinguish between cholecystitis and biliary dyskinesia. However, dyskinesias are not characterized by fever, leukocytosis and increased ESR. It helps clarify the diagnosis ultrasound combined with duodenal intubation. Obstructive jaundice due to obstruction of the common bile duct stone, must be differentiated from jaundice caused by acute viral hepatitis, hemolytic anemia.

Treatment of chronic cholecystitis. The diet should help to prevent stagnation of bile in the gallbladder, reduce inflammation. Power fraction (5–6 times a day), is very useful vegetable fats (olive oil, sunflower oil), containing polyunsaturated fatty acids, vitamin E. Polyunsaturated fatty acids contribute to the normalization of cholesterol metabolism. Antibaktellnymi complex therapy, anti-inflammatory, normalizes motility zhelchevyvoschih tract drugs. Duration of antibiotic treatment of 7 days. If necessary, after the 3-day treatment interval can be repeated. Antibacterials desirable to combine with choleretic, anti-inflammatory effect and is called.

1 Drugs that increase the contractility of the gall bladder (prokinetics), usually applied as a course of treatment for a month and are assigned to food.

2. Drugs affecting the content of the gall bladder. Extract Fumaria officinalis (Fumaria officinalis) at giperholerezise slows, while gipoholerezise increases the production of bile. Another feature is an antispasmodic effect on the sphincter of Oddi. appoint 1–2 capsules 3–4 times a day for a month.

3. Reducing visceral hyperalgesia and inflammation. With this purpose a nonsteroidal anti-inflammatory drugs in conventional dosages.

It should be remembered that the degree of penetration into the bile antibacterial agents can be divided into three groups. Penetrating into bile at

very high concentrations: erythromycin (0.25 g 4 times a day), oleandomycin (0.5 g 4 times a day after meals), oxacillin (0,25–0,5 g 4–6 once a day orally or intramuscularly), ampioks (0.5 g 4 times a day orally or intramuscularly. In case of failure of medical treatment and persistent pain during solved the question of holding cholecystectomy, preference is given to laparoscopic cholecystectomy.

Treatment of dysfunction of the gallbladder and sphincter of Oddi

I. *Diet.* It is recommended that frequent meals at regular intervals throughout the day to prevent thickening of bile. You should also avoid foods that provoke pain (fried, greasy).

II. *Pharmacotherapy.*

1. Medications that increase the contractility of the gall bladder (prokinetics) for a month and are assigned to food. Cisapride average doses are 10 mg three times a day. At the same time it is advisable to apply prokinetic drugs, which have antispasmodic effect on the sphincter of Oddi (mebeverin 100-300 mg per day or pinavreium 150–200 mg per day).

2. Drugs affecting the content of the gall bladder. Extract Fumaria officinalis (Fumaria officinalis): with giperholerezise slows, while gipoholerezise increases the production of bile.

3. Reducing visceral hyperalgesia and inflammation. For this purpose, used NSAIDs in standard dosages.

III. *Surgical treatment.* With the ineffectiveness of medical treatment and persistent pain during solved the question of holding cholecystectomy, preference is given to laparoscopic cholecystectomy.

When Pancreatitis similar option transduodenalnym sphincterotomy performed septoplasty or pancreatic duct, the clinical efficacy of about 70 %.

Emergency care about colic, medical tactic

Under biliary colic understand paroxysmal pain in the right upper quadrant, resulting in diseases of the biliary tract: cholelithiasis, cholecystitis, papillary stenosis, strictures, compression of the bile ducts, the presence of worms or foreign bodies in the biliary tract, hemobilia and biliary dyskinesia.

The pains are the result of the blockade of the outflow of bile. They are caused by spastic contraction of smooth muscles of the gall bladder and ducts, "seeking to overcome the" current of bile obstruction. This dramatically increases the pressure in the biliary system. The intensity and nature of pain are different. Generally strong pain, sometimes recurrent. There may be prodromal symptoms, such as feeling of heaviness and distension in the right upper quadrant. The pain is localized in the right upper quadrant, the epigastric region, sometimes in the left upper quadrant, radiating to the back, right side of the chest, the right shoulder girdle, shoulder and right arm. Frequent nausea and vomiting, not bringing relief, bloating and constipation. It can develop cholecysto-cardiac syndrome. Sometimes there is a short-term rise in temperature. The attack of biliary colic can last from several minutes to several

hours with wavy enhancement or suppression of pain. During the long and painful attack after nabyudayut sometimes transient pruritus, increased activity of alkaline phosphatase and bilirubin levels, the selection of dark urine and pale feces. The diagnosis may be confirmed by ultrasound.

Treatment. Not stoped patients with biliary colic should be hospitalized in the surgical ward. For relief antispasmodics used: nitroglycerine (sublingual), subcutaneously 1 ml of a 1 % solution of atropine sulfate, 1–2 ml 0.2 % solution platifillina gidrotartrata, 1–2 ml of p-ra papverina hydrochloride or shpy. You can use / vennoe introduction of 5–10 ml of 2.4 % solution of aminophylline. These drugs to enhance the effect can be combined with droperidol and analgin. If the attack is not stopped, atropine, analgin and droperidol is administered in / .venno infusion 200–300 ml of 5 % solution of glucose. An effective remedy is baralgin. If no effect promedol administered in combination with atropine.

Eliminate spasm promotes the use of heating pads (to the exclusion of an acute abdomen) Good effect on nabyudaetsya novocaine blockade (subksifoidnoy novocaine blockade or embargo the round ligament of the liver).

TOPIC 6: Urinary syndrome: differential diagnosis. Methods of diagnosis of kidney disease in an outpatient setting .Treatment of chronic pyelonephritis, chronic glomerulonephritis and chronic kidney disease in an outpatient setting; medical tactics, medico-social examination, clinical examination, primary prevention. Emergency medical care in renal colic.

The concept of the urinary syndrome, its features with pyelonephritis, glomerulonephritis, urolithiasis, urethritis, cystitis, bladder cancer, nephropathy.

Urinary syndrome — a combination of proteinuria, not exceeding 3.5 g / day, red blood cell in urine, and leykotsiturii, cylindruria. Chalice developed some combination of these components (proteinuria with leukocyturia, proteinuria, and red blood cell cylindruria et al.).

Pathological proteinuria — urinary protein excretion in inflammatory, degenerative and so-called urological diseases of the kidneys and urinary tract. Penetration of various proteins in urine depends on the state of the glomerular filter and renal proximal tubules and on the molecular weight of the protein, its shape and charge. Thus, myoglobin (muscle hemoglobin) 75 % filtered, and hemoglobin only 3 %. Depending on the underlying causes and mechanisms of development distinguish pre-renal, renal and postrenal proteinuria.

Pre-renal proteinuria is the result of the emergence and increasing blood concentrations of low molecular weight proteins, easy to be filtered in the glomeruli of the kidneys. This is observed in diseases of the blood, hemolysis, multiple myeloma (abnormal protein Bence – Jones), extensive muscle injuries (myoglobinuria), burns, after intense insolation.

Kidney, or renal, proteinuria always caused by the defeat of neurons (mainly glomeruli and tubules less), which leads to increased permeability of the glomerular capillary wall to plasma proteins and reduce reabsorbtsionnoy ability of the epithelium of the proximal tubules.

Postrenal proteinuria, usually associated with inflammatory or neoplastic processes in the urinary tract and is caused by the release of the protein from disintegrating leukocytes, epithelial and other cells.

Hematuria — excretion of red blood cells in the urine. Detection in urine 1–3 or more in view of erythrocytes is a pathology that hematuria. Depending on the intensity of erythrocyte in urine excretion distinguish microhematuria and gross hematuria. When microhematuria color of urine in evaluating the case does not change, and the number of red blood cells varies from unit to 100 in sight. Sometimes red blood cells coated with a thin layer of the entire field of view .If gross hematuria urine becomes the color of meat slops or become dark red, and red blood cells densely cover the entire field of view and can not be counting. Mechanisms of origin of hematuria vary and depend on the nature of renal disease and urinary tract.

Pyuria urinary excretion of leukocytes in an amount exceeding the normal, i.e. with an overall analysis of urine for more than 6–8 in sight, in a study on nechyporenko over $2{,}5 \times 10^6$ / L, Addis Kakovski more $4{,}0 \times 10^6$ / day. When the admixture of pus in the urine is so great that determined visually indicate pyuria. Thus leukocytes cover the entire field of view, clusters are located and served counting.

Cylindruria — urinary excretion, the so-called cylinder (protein or cell aggregates, having a cylindrical shape and a different length). They are hyaline, granular, waxy, erythrocyte, leukocyte, pigment.

Urinary syndrome in acute ***glomerulonephritis*** characterized by proteinuria, red blood cell, cylindruria, at least (1 / z cases) — leukocyturia. When an association between urinary syndrome and suffered an acute infectious disease (angina, acute respiratory illness, pneumonia, scarlet fever, etc.), or the aggravation of the process in the foci of chronic infection.

For ***pyelonephritis*** characterized by less severe proteinuria (0.5 g / day), the predominance of the red blood cell leykotsiturii the quantitative study of urinary sediment, the presence of epithelial, leukocyte and granular cylinders, often — bacteriuria.

Urolithiasis determined change in the reaction of urine, the presence of large noncellular sediment prevalence of red blood cell with very little proteinuria.

Urinary syndrome in ***cystitis and urethritis*** is characterized by neutrophilic leukocyturia (from 10–12 to the number of cells that covers all fields of view), red blood cell varying severity (usually the terminal, until gross hematuria), the presence of transitional epithelium and bacteriuria.

The earliest and characteristic symptom of ***bladder cancer*** is hematuria, or blood in the urine. Urine becomes alien to her "rusty" color, or the color of meat slops.

The main feature of the bladder syndrome with ***nephropathy*** is varying degrees of hematuria, usually intermittent nature. The amount of protein does not exceed the 0.5–1.5 g / day, and proteinuria similarly eritrotsiturii is detected may be inconstant or only symptom of the disease. Sometimes revealed leykotsituriya, which combined with bacteriuria indicates accession pyelonephritis.

Plan examination of the patient with chronic pyelonephritis, chronic glomerulonephritis, chronic kidney disease in an outpatient setting. Medical tactics in chronic renal failure.

Chronic pyelonephritis. Diagnostic:

— urinalysis;

— complete blood count;

— biochemical blood tests: determination of the concentration of urea, creatinine;

— renal ultrasound;

— urography Review;

— urography excretory.

Additional (indication):

— CT kidneys;

— renal scintigraphy,

— research filtration renal function;

— to conduct tests Rehberg-Tareeva (clearance by endogenous creatinine);

— RWG;

— urine culture.

Chronic glomerulonephritis.

Diagnostic:

— urinalysis;

— complete blood count;

— biochemical analysis of blood: determination of the concentration of total protein, potassium, urea, creatinine, cholesterol;

— renal ultrasound.

Additional (indication):

— definition of daily protein loss in the urine;

— research filtration renal function;

— to conduct tests Rehberg-Tareeva;

— lipidogram;

— determination of the concentration of cyclosporine in the blood;

— densitometry.

Chronic kidney disease. Diagnostic :

— urinalysis;

— complete blood count;

— biochemical blood test: determination of the concentration of total protein, potassium, total bilirubin, urea, uric acid, creatinine, cholesterol, activity of AST, ALT;

— renal ultrasound.

Additional (indication). Biochemical blood test: determination of the concentration of glucose, calcium, phosphorus, serum iron, ferritin.

— determination of markers of hepatitis B (HBsAg, anti-HBs, anti-HBcore) and hepatitis C virus (anti-HCV);

— detection of antibodies to HIV;

— definition of daily protein loss in the urine;

— research filtration renal function - to conduct tests Rehberg-Tareeva;

— lipidogram;

— acid-base status of blood;

— densitometry;

— determination of serum parathyroid hormone.

Table 10 — CKD stage, depending on the level of chronic kidney disease and blood pressure

Stage	Characteristic	SSKF ml / min per 1.75 m^2	AAG or labo-operator. deviations	Diagnostic and therapeutic measures
	The presence of risk factors	> 90	±	Observation, reducing the risk of renal disease
1.	Kidney damage with normal or elevated GFR	> 90 > 130	±	Diagnosis and treatment of nephropathy and comorbidity, nefroprotektsiya, reducing the risk of SS disease
2.	Kidney damage with mild decrease in GFR	60–89	±	Events of the previous stage, the assessment of progression
3.	Kidney damage with mild decrease in GFR	445–59 30–44	+ +	Events Events previous stage previous stage, treatment of complications
4.	Kidney damage with severe reduction in GFR	15–29	+	Events of the previous stage, preparation for renal replacement therapy
5.	ESRD	< 15	+	Renal replacement therapy

CKD codes designated by capital letters:

H — Standards albuminuria (up to 30 mg / day).

M — microalbuminuria (30 - 299 mg / day).

P — proteinuria (albumin 300 mg / day, or proteinuria over 150 mg / day).

D — dialysis (for the 5th stage CKD).

T — transplantation (5th stage CKD).

The clinical diagnosis of diabetes, type 2 diabetic nephropathy. CKD 4-P. In a patient with a long history of non-insulin diabetes developed nephrotoxicity angiosclerosis manifesting incomplete nephrotic syndrome with proteinuria (P index) and nitrogen violation renal excretory function with severe reduction in GFR (stage 4 CKD).

Treatment of chronic kidney disease: for right choice of adequate treatment is extremely important to consider the classification of CKD.

1. Conservative step with the fall in glomerular filtration up to 40–15 ml / min with great possibilities of conservative treatment. Treatment of chronic kidney disease in the conservative stage is to treat the underlying disease which led to the development of chronic kidney disease.

2. End-stage with a glomerular filtration rate of about 15 ml / min, when should discuss the issue of extrarenal purification (hemodialysis, peritoneal dialysis) or a kidney transplant.

Treatment program in chronic kidney disease in the conservative stage:

1. Treatment of the underlying disease, leading to uremia.
2. Mode.
3. Clinical Nutrition.
4. Adequate fluid intake (correction of the water balance).
5. Correction of electrolyte metabolism.
6. Reducing the delay of the final products of protein metabolism (control of azotemia).
7. Correction of acidosis.
8. Treatment of hypertension.
9. Treatment of anemia.
10. Treatment of uremic osteodystrophy.
11. Treatment of infectious complications.

Table 11 — Anti proteinuric strategy in CKD

Level 1 (CRD 2)	Level 2 (CKD 3 a)	Level 3 (CKD 36-4)
— Control of blood pressure. — ACE inhibitors. — ACE inhibitors + angiotensin receptor antagonists 2. — Beta-blockers. — Limiting the protein.	— Bed rest during exacerbation of the process. — Fluid restriction. — Control of lipids. — Reception Restriction of salt. — Aldosterone antagonists. — Smoking cessation. — Do Not apply oral contraceptives. — Eliminate heavy exercise. — The fight against obesity.	— Homocysteine and other antioxidants. — Compensation of acidosis. — Cancel NSAIDs. — Less caffeine. — Normalize the stock of iron. — Stimulation of erythropoiesis. — Allopurinol (gout). — Pentoxifylline. — Mycophenolate mofetil (in proliferative conditions).

Health food: Diet for CKD based on the following principles:

— Restriction of dietary intake of protein to 60–40–20 grams per day depending on the severity of renal failure.

— Ensuring sufficient caloric intake, the corresponding energy needs of the body, from fat, carbohydrates, full maintenance of the body in trace elements and vitamins; income limit phosphate intake; control over the receipt of sodium chloride, water and potassium.

In moderate hyperkalemia (6–6.5 mmol / l) should be limited in the diet foods rich in potassium, potassium-sparing diuretics avoided, taking ion exchange resins (rezonium 10 g 3 times a day for 100 ml of water).

If *hyperkalemia 6.5–7 mmol* / l is expedient to add an intravenous glucose vvdenie with insulin (8 U of insulin per 500 ml of 5 % glucose solution).

When *hyperkalemia above 7 mmol* / l there is a risk of complications from heart (beats, atrioventricular block, asystole). In this case, except for the intravenous administration of glucose to insulin is shown intravenous 20–30 ml of 10 % calcium gluconate solution or 200 ml of 5 % sodium hydrogen carbonate solution.

Used together with a diet of *adsorbents* is adsorbed onto itself ammonia and other toxic substances in the gut. As most commonly used sorbents or Enterodez carbol 5 g per 100 ml of water three times a day at 2 hours after a meal.

Gastric lavage (dialysis): It is known that the reduction of renal function azotovydelitelnoy urea and other nitrogenous metabolic products become prominent gastric mucosa. In this regard, gastric lavage can reduce azotemia. in the stomach is administered 1 liter 2 % sodium hydrogen carbonate solution, then aspirated. Washing the produce in the morning and evening. For 1 session can remove 3–4 g of urea.

Treatment of asthenia: have the ability to increase the excretion of urea. Hofitol and Lespenefril. Parenteral administration of *detoxification means*: Apply gemodez, 5 % glucose solution.

Treatment of hypertension. Should seek to optimize the BP as hypertension worsens the prognosis and reduces the life expectancy of patients with chronic renal failure. Blood pressure should be kept within the 130–150 / 80–90 mm Hg. Limiting the salt in the diet to 3–5 kg per day, severe hypertension — 1–2 g per day, as normal blood pressure, salt intake should be increased. Purpose natriyuretik — furosemide 80–140–160 mg per day uregita (ethacrynic acid) to 100 mg per day. Both drugs increase the number of glomerular filtration. These drugs are used in tablets, and swelling in the lungs and other urgent conditions — intravenously. ACE inhibitors are particularly indicated — Capoten (captopril) at 0.25–0.5 mg / kg two times per day. The advantage of the hood and its analogues is their normalizing effect on intraglomerular hemodynamics. Perhaps in some cases the use of beta-blockers (propranolol, obsidan, Inderal). These drugs reduce renin secretion, their pharmacokinetics in chronic renal failure is not broken. When refractory to treatment of hypertension ACE inhibitors in combination with saluretikami and beta-blockers. Dose regimens reduce the progression of chronic kidney disease.

Treatment of anemia. Unfortunately, the treatment of anemia in patients with chronic kidney disease is not always effective. It should be noted that the majority of patients with chronic kidney disease anemia satisfactorily transferred with reduced hemoglobin levels even up to 50–60 g / l, as the reaction develops adaptive improving the oxygen-transport function of blood. Iron supplements

are usually taken in and only with poor tolerance and gastrointestinal disorders are administered intravenously or intramuscularly. Dosed iron preparations should, based on the fact that the minimum effective daily dose for an adult of divalent iron is 100 mg and a maximum daily dose expedient — 300–400 mg.

Treatment NeoRecormon: Recombinant erythropoietin — Recormon is used to treat a lack of erythropoietin in patients with chronic renal failure. One ampoule preparation for injection contains 1000 ME. The drug is only administered subcutaneously, initial dose is 20 IU / kg three times a week, in the future with no effect on the number of injections increases three each month.

Treatment of uremic osteodystrophy:

1. Maintaining a close to normal levels of calcium and phosphorus in the blood. Typically, the calcium content in the blood is reduced, and increased phosphorus. It is recommended to take almagel 10 ml 4 times a day, it contains aluminum hydroxide, which forms insoluble compounds with phosphorus, is not absorbed in the intestine.

2. Inhibition of hyperactivity of the parathyroid glands. This principle of treatment is carried out taking into calcium.

3. Osteochin. In recent years, a drug osteohin (ipriflavone) for the treatment of osteoporosis of any origin. The proposed mechanism of its action — inhibition of bone resorption by increasing endogenous calcitonin and improved mineralization due to calcium retention. Prescribe drugs to 0.2 g 3 times a day for an average of 8–9 months.

Treatment of infectious complications. The emergence of infectious complications in patients with chronic kidney disease leads to a sharp decrease in renal function. The most nephrotoxic antibiotics, aminoglycosides (gentamicin, kanamycin, streptomycin, tobramycin, brulamitsin). The combination of these antibiotics with diuretics increases the possibility of toxic action. Moderately nephrotoxic tetracyclines. Nitrofuran compounds and drugs nalidixic acid can be administered in chronic renal failure only in a latent or compensated stages.

Basic principles of treatment of chronic kidney disease in the terminal stage

In ESRD glomerular filtration at a value of less than 10 ml / min (where the patient can not allocate more than 1 liter of urine per day) is necessary to adjust the fluid intake for diuresis (urine output to the quantity of the previous day was added 300–500 ml). In the later stages of CKD conservative methods of treatment are ineffective, so in ESRD held active treatments: continuous peritoneal dialysis, hemodialysis program, a kidney transplant.

Peritoneal Dialysis. This method of treatment for patients with chronic kidney disease is the introduction into the abdominal cavity of a special dialysate, which due to the concentration gradient across the peritoneal mesothelial cells diffuse various substances contained in the blood and body fluids. Peritoneal dialysis can be used as in earlier periods of the terminal stage, so ive its final period when dialysis is impossible. The efficacy of peritoneal

dialysis conducted three times per week for the duration of 9 hours for the removal of urea, creatinine, correction of electrolyte and acid-base status is comparable to hemodialysis conducted three times a week for 5 hours.

Hemodialysis — the main method of treatment of patients with acute renal failure and chronic kidney disease, based on the diffusion from the blood into the dialysis solution through a translucent membrane urea, creatinine, uric acid, electrolytes and other substances in the blood lingering in uremia. Hemodialysis is performed using the apparatus "artificial kidney", representing the hemodialyzer and a device with which prepared and served in a hemodialyzer dialysate. Hemodialyzers the process of diffusion from the blood to the dialysate various substances. Hemodialysis session usually lasts 5–6 hours, repeat it 2–3 times a week.

Chronic hemodialysis sessions begin with the following clinical and laboratory parameters:

• glomerular filtration rate of less than 5 ml / min; skrost effective renal blood flow less than 200 ml / min;
• the urea content in plasma over 35 mmol / l;
• creatinine in blood plasma over 1 mmol / L;
• contents of the "middle molecules" in the blood plasma of more than 1 U;
• the content of potassium in the blood plasma of more than 6 mmol / l;
• reduction of standard bicarbonate levels below 20 mg / dL;
• shortage of buffer bases more than 15 mmol / L;
• development of resistant oligoanuria (less than 500 ml per day);
• commencing pulmonary edema on the background of hydration;
• fibrinous or less pericardial effusion;
• increasing signs of peripheral neuropathy.

Absolute contraindications to chronic hemodialysis are:

• cardiac decompensation with stagnation in the large and small circles of blood circulation, regardless of kidney disease;
• infectious diseases of any localization with an active inflammatory process;
• cancer at any site;
• TB of internal organs;
• GI ulcer in acute phase;
• severe liver disease;
• mental illness with a negative attitude to hemodialysis;
• hemorrhagic syndrome of any origin;
• malignant hypertension and its consequences.

Kidney transplant — the best method of treatment of chronic renal failure, which consists in replacing the affected irreversible pathological process kidney unchanged by the kidney. Selection of donor kidneys produce system HLA-antigens.

General principles of treatment of patients with chronic pyelonephritis, chronic glomerulonephritis and chronic kidney disease in an outpatient setting.

1. ***Chronic pyelonephritis***:

— Treatment of the underlying disease: kidney stones, hydronephrosis, benign prostatic hyperplasia, neurogenic bladder, urethral strictures.

— Antibiotic therapy: fluoroquinolones: ciprofloxacin 500 mg inside — 10 days inside ofloxacin 400 mg 1 time per day — 10 days; cephalosporins: tsefuroksim1000 mg / m 2 twice a day or 1000 mg of ceftriaxone / m 2 twice daily 7–10 days; nitrofuran derivatives: furazidin inside 100 mg 3 times a day — 10 days (correction of antibiotic therapy based on the data on the microflora of urine culture).

— Nonsteroidal anti-inflammatory drugs: diclofenac 100 mg per day orally or rectally or / m — 5 days, or ketorolac 10–30 mg PO / m — 5 days, or nimesulide 100 mg orally — 5 days, or meloxicam 7.5 -15 mg orally — 5 days.

— Combination drugs of plant origin: extracts of herbal preparations, such as kanefron 50 drops orally 3 times a day.

2. ***Chronic glomerulonephritis***:

— ***Pathogenetic therapy***: ***corticosteroids***: oral prednisolone 1 mg / kg body weight (but not more than 80 mg / day) or methylprednisolone with the allocation coefficient for 2 months with a gradual reduction in dose to the complete abolition of a 6-month therapy and / or anti-tumor agents alkylating agents: cyclophosphamide into 2–3 mg / kg of chlorambucil or 0.2 mg / kg to about 3 months.

— ***Immunosuppressants*** (in the presence of contraindications to the use of glucocorticoids or antitumor agents, alkylating agents, the development of steroidrezistentnosti or steroidzavisimosti: cyclosporine orally 3–5 mg / kg per day (under the control of the concentration levels of the drug in the blood) to 12 months or mycophenolic acid into 2 g per day to 6 months or tacrolimus inwards 0.05–0.1 mg / kg daily for 6–12 months.

— Prevention of steroid ***osteoporosis*** preparations complex of calcium and vitamin D in a dose of 1500mg ionized calcium and vitamin D to 400–800 IU per day, and / or bisfosfanaty: alendronate orally at a dose of 35–1 70 mg once weekly or inside ibandronic acid 150 mg of 1 once a month.

— Prevention of secondary infection while receiving immunosuppressive therapy: co-trimoxazole 480 mg 1 time per day and intrakanozol 100 mg per day.

Prevention-steroid ulcer:

— *pratonnoy pump blockers*: omeprazole into 20 mg per day or 15 mg of lansoprazole, inside a day or antagonists of the histamine H2 receptor: inside ranitidine 150 mg 2 times a day;

— *antiemetic* for the prevention of nausea and vomiting associated with the treatment with cytotoxic drugs: inside ondansetron 8 mg 2 times a knock or tropisetron into 5 1 mg once a day for 5 days.

Nefroprotektsiya:

— *ACE inhibitors*: enalapril is 5–20 mg per day or fosinopril 5–20 mg per day or lisinopril 5–20 mg per day or ramipril 1.25–10 mg per day or perindopril 2–8 mg per day, and / or

— *angiotensin 2 receptor antagonists* inside: losartan 50–100 mg or 600 mg of eprosartan per day or day of valsartan 80–160 mg per day, or irbesartan 150–300 mg per day, or telmisartan 40–160 mg per day for at least 1 month — independently the level of blood pressure.

— ***Symptomatic hypertension***:

— inward *calcium channel antagonists*: nifedipine 5–40 mg per day of amlodipine, or 5–10 mg per day or verapamil 120–480 mg per day diltiazem or 180–480 mg per day, and / or

— *β-blockers inside*: Bisoprolol 5–20 mg of carvedilol per day or 6.25–100 mg a day metoprolol or 50–100 mg per day, and / or

— *blockers inside*: doxazosin 1–16 mg per day, or prazosin 0.5–20 mg per day, and / or

— *selective agonists imidozalinovyh* recipes moxonidine 0.2–0.6 mg per day.

Edema syndrome:

diuretics inside: Furosemide 40–400 mg daily, and / or hydrochlorothiazide 25–200 mg daily, and / or spironolactone 50–200 mg daily, and / or indapamide 2.5–5 mg per day.

Lipid metabolism disorders:

— *hypolipidemics* — *statins*: simvastatin is 5–40 mg per day of pravastatin, or 10–40 mg of lovastatin per day or 10 mg per day -80 or atorvastatin 10–80 mg per day.

— ***Hypertriglyceridemia***: *fibrates* — fenofibrate into 145 mg per day.

— ***Hypercoagulation:*** anticoagulants warfarin administration (under the control of INR greater than 3) and / or acetylsalicylic acid into 75–150 mg.

— With a proven link between ***hepatitis C*** and progressive decline in renal function: interferon alfa n / or / m 3 million units 3 times per week, or pegylated interferon alfa 1.5 mg / kg per week for 6–12 months and 800 inwardly ribavirin — 1200 mg per day for 6–12 months.

3. **Chronic kidney disease**

1. *Preparations improving renal hemodynamics:*

antiplatelet agents inside: dipyridamole 75–200 mg / day or pentoxifylline 300–600 mg / day.

2. *Symptomatic hypertension:* *ACE inhibitors* — inside: enalapril is 5–20 mg per day or fosinopril 5–20 mg per day or lisinopril 5–20 mg per day or ramipril 1.25–10 mg per day or perindopril 2–8 mg per day, and / or

— *angiotensin 2 receptor antagonists* inside: losartan 50–100 mg or 600 mg of eprosartan per day or day of valsartan 80–160 mg per day, or irbesartan 150–300 mg per day or 40–160 mg telmisartan per day, and / or

— inward *calcium channel antagonists*: nifedipine 5–40 mg per day of amlodipine, or 5–10 mg per day or verapamil 120–480 mg per day diltiazem or 180–480 mg per day, and / or

— *β-blockers* inside: Bisoprolol 5–20 mg of carvedilol per day or 6.25–100 mg a day metoprolol or 50–100 mg daily, and / or

— blockers inside: doxazosin 1–16 mg per day, or prazosin 0.5–20 mg per day, and / or

— selective agonists imidozalinovyh recipes moxonidine 0.2–0.6 mg per day.

3. Edema syndrome diuretics inside: Furosemide 40–400 mg daily, and / or hydrochlorothiazide 25–200 mg daily, and / or spironolactone 50–200 mg daily, and / or indapamide 2.5–5 mg per day.

4. Uremic syndrome intoxication: sorbents courses for 1 week per month: activated charcoal inside of 500 mg 2–3 times a day or enterosgel into 15 g 3 times a day for 2–3 weeks, and herbal drugs, has a detoxifying effect: tsinara 2 tablets 3 times day courses of 1 month 3–4 times a year.

5. ***Correction of acid-base balance*** with acidosis: sodium bicarbonate into the 0.5–1 g per day (under the control of blood gas).

6. ***Anemia syndrome*** — recombinant erythropoietin: Epoetin alfa n / 80–120 IU / kg 2–3 times per week or epoetin beta n / 80–120 IU / kg 2–3 times per week, or methoxypolyethylene glycol-epoetin beta 0.6–1, 2 mg / kg s / c 1–2 times a month;

— iron preparations inside: ferrous gluconate 300 mg of 2–3 times daily or iron sulfate -300 150 mg of 1–2 times a day, or 100 mg iron in karboksimaltoza / 1 time per week.

7. ***Lipid metabolism disorders*** hypolipidemics — statins: simvastatin is 5–40 mg per day of pravastatin, or 10–40 mg of lovastatin per day or 10 mg per day -80 or atorvastatin 10–80 mg per day.

8. ***Hypertriglyceridemia*** — fibrates — fenofibrate 145 mg per day.

9. ***Uremic gastroenteropathy***: — drugs used in violation of the secretory function of the stomach — blockers hydrogen pump: rabeprazole inside 10 mg daily or lansoprazole 15 mg inside a day or antagonists of histamine H2-receptor: ranitidine 150 mg inside 2 times a day;

— antiemetic — blockers dopaminovyhD2 — receptors: metoclopramide inside 20–30 mg per day;

— antidiarrheals: loperamide orally 4–16 mg per day.

10. ***Protein-energy malnutrition*** caused by changes in protein metabolism — ketoanalogi essential amino acids: Ketosteril inside 4–8 tablets 3 times a day.

11. ***Uremic dystrophy*** — anabolic hormones: nandrolone 50 mg / m 1 time per week № 3.

Renal colic: the clinical picture, emergency medical care on an outpatient basis, medical tactics.

Renal colic — an attack of acute pain that occurs in acute obstruction of the urinary tract.

Causes of renal colic:

— Urolithiasis.

— Pyelonephritis.

— Trauma — tumor of the kidney and bladder.

— The narrowing of the ureter.

Violation of the outflow of urine from the kidney due to obstruction of the urinary tract stone, blood clots, etc. leads to an increase in intraurethral pressure, it causes tensile renal capsule rich pain receptors.

Manifestations of renal colic

— Pain in the lumbar region, or the projection of the ureter. Distribution of pain depends on the level of obstruction. The upper third of the ureter — the average area of the abdomen. The middle third of the ureter — the groin and outer thighs. Lower third — genitals:

— Painful urination.

— Frequent urination.

— Chills, fever; nausea, vomiting.

Complications of renal colic:

— Pyelonephritis

— Hydronephrosis

— Uremia

A survey in renal colic:

— Complete blood count.

— Urinalysis.

— Biochemical analysis of blood.

— Renal ultrasound.

— Review radiography of the kidneys.

— Excretory urography — Retrogradnayaureteropielografiya.

— Determine the level of obstruction.

Emergency medical care in the outpatient setting, medical tactics.

Diagnostic measures.

— Assessment of general condition and vital signs: consciousness, respiratory, circulatory (heart rate, heart rate, blood pressure, BH).

— Inspection and palpation of the abdomen.

— Identify the symptoms of renal colic (sign of a beating, pain on palpation of the lumbar region on the affected side).

— The presence of associated symptoms (nausea, vomiting, fever, etc.).

Help.

— Place the patient on the couch- pain relief.

— Baralgin in / or / m 5ml.

— Ketorolac in / or / m.

— Drotaverine 2 % — 2 mL / or / m.

— If necessary — nitroglycerin 1 tab. sublingually.

Indications for hospitalization:

— No effect of treatment.

— The presence of clinical signs of complications.

— Bilateral renal colic or a kidney.

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