OUTPATIENT THERAPY
Handbook
for 4th year foreign students

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ПОЛИКЛИНИЧЕСКАЯ ТЕРАПИЯ
Пособие
для студентов 4 курса
факультета иностранных учащихся

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Yankovskaya, L.V.


The manual consists of two lectures in outpatient therapy for 4th year students, about the prevention work of district physicians and clinic treatment strategy in hypertensive crises at preadmission stage. The first lecture examines the primary prevention of non-communicable diseases, the identification of risk factors for hypertension, ischemic heart disease, COPD, digestive diseases, kidney and other, forming groups of people to the presence of certain risk factors, how to resolve them, as well as secondary prevention of non-communicable diseases – issues medical examination. Lists of active counselling groups, the frequency of inspections, plan examination and treatment, rehabilitation activities. The second lecture is devoted to the diagnosis and emergency treatment in hypertensive crises at preadmission stage. In the practical lessons detailing the thematic curriculum that describes the content of each parsed questions for each topic, the basic and additional sources for each lesson.
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INTRODUCTION

Healthcare system is now being reformed in Belarus, and as result more attention is paid to outpatient medical care, as 75% of patients starts and finishes their therapeutic treatment in polyclinics. Therefore, demand for outpatient training of future physicians is increasing.

Department of outpatient (polyclinic) therapy aims at theoretical training of future doctors for working in outpatient settings as a district physician and a general physician (GP).

_Curriculum for outpatient therapy is as follows:_

- study of the organization of health care and medical education in a particular organization;
- laying the foundations of clinical thinking and medical ethics on the diagnosis and treatment of diseases of the internal organs, familiarization with the organization of medical and preventive care;
- development of clinical methods of diagnosis, treatment and prevention of acute bronchitis and pneumonia, acute respiratory infections, tonsillitis, primary and secondary hypertension, ischemic heart disease, chronic gastritis and peptic ulcer disease, study of issues of health and social assessment, emergency assistance with hypertensive crisis, onset of angina pectoris;
- getting communication skills: experience of social, organizational and educational work,
- introduction to medical records and reports.

_The student should know:_

- Basics of general therapeutic disciplines in the amounts necessary for diagnosis and treatment of major diseases at preadmission stage.
- The district principle of medical care of population.
- Risk factors of major internal diseases.
- Groups of people going to medical examination and its procedure.
- Assessment of the effectiveness of primary and secondary prevention of major internal diseases.
- Principles of diagnosis and treatment of major internal diseases.
- Principles of rehabilitation of patients and disabled people on an outpatient basis.
- Principles of work of rehabilitation department (office).
- Ability to use physical agents, physical therapy in complex treatment and rehabilitation, principles of medical diagnostic equipment work.
Indications for patient hospitalization in the hospital and referral to day care, the organization of hospital at home.

- Principles of work of prevention department.
- Communication and continuity in the work of health care institutions.
- Criteria for temporary (TD) and permanent disability, timing of TD, procedure and indications for referral to the Medical rehabilitation expert board (MREB), timing of re-examination, indicated and contraindicated types and working conditions for patients and disabled people.

The student should be able to:

- Perform general examination of the patient.
- Make a preliminary diagnosis, assign survey plan of patient care.
- Estimate total blood count, urinalysis and biochemical blood test of patient.
- Assess the patient's ECG, chest X-ray.
- Set a final diagnosis after the examination and perform dynamic monitoring of the disease.
- Monitor during the required period of temporary disability, indications for hospitalization.
- Refer promptly the disabled patient to the MREB.
- Conduct a routine inspection and consultation of the population.
- Organize and conduct public education of the area.
- Write a journal of ambulatory examination of the patient in the patient card.

**LECTURES**

for outpatient therapy, content and volume in hours

<table>
<thead>
<tr>
<th>№ p/n</th>
<th>Topic and content</th>
<th>Volume in hours</th>
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<td>Prophylactic work of district physician. Dispanserization (controlled independent work (CIW)) -0.2 h.</td>
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<td>2.</td>
<td>Diagnosis and treatment of hypertensive crises at preadmission stage (CIW) – 0,2h.</td>
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## PRACTICAL CLASSES TOPICS

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<td>Contents of the district physician’s work. Basic outpatient medical records. Dispanserization</td>
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<td>1.2</td>
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<td>The medical social expertise in medical institutions. MCC functions. The concept of MREB.</td>
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<td>Ischemic (coronary) heart disease: outpatient aspects of diagnosis of various forms of ischemic heart disease, medical tactics, medical-social examination, dispanserization, primary prevention. Treatment of angina pectoris. Emergency care for angina attack on an outpatient basis</td>
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<td>Chronic gastritis, peptic ulcer and duodenal ulcer. Outpatient aspects of diagnosis and treatment, medical tactics, medical and social examination, dispanserization, primary prevention. First aid for acute abdominal pain and suspected gastrointestinal bleeding</td>
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**TOTAL:** 36,0
LECTURES

Topic 1. Prophylactic work of district physician. Primary and secondary prevention (dispanserization)

XX – beginning of XXI century is the era of non-infectious diseases, which reduce life expectancy and worsen demographic situation in a great extent. Their share in the structure of the total mortality of the Republic of Belarus in 2007 amounted to 77.3%. The causes of most diseases are well known: smoking, lack of exercise, unbalanced diet, alcohol, stress, ecological trouble.

Experience in developed countries shows that the increase in life expectancy of the population was not reached by improving the quality of treatment, but by reducing the incidence by primary prevention.

PRIMARY PROPHYLAXIS of non-infectious diseases (PPNID) is a set of measures aimed to reduce the adverse health effects and causes of the disease, increased positively influencing factors. Success can only be a comprehensive medical, social and environmental prevention, providing for the implementation of the health system, by society and the State. PPNID technologies consist of the formation of risk groups, screening, consulting on lifestyle, integrated measures to prevent diseases, integration of the various services and agencies involved in PPNID, vaccination and chemoprophylaxis, health monitoring, health promotion, responsibility for the health of the individual.

In the activities of doctors of any specialty PPNID is a very important factor.

Doctor's preventive work includes primary prevention, aimed at the prevention of disease and early diagnosis, in which a special role is played by preventive examinations, as well as secondary prevention of relapse of present chronic diseases (dispanserization).

In PPNID medical check-ups are also important; the main purpose of this activity is to assess the state of health, regarding the possible work in certain industries. These preventive medical examinations are important for the further dynamic surveillance of workers, as well as to reveal connection of the disease emerged with the profession.
The main objective of prophylactic examinations:
- Assessment of health status.
- Deciding on the possibility to work in certain industries.
- The dynamic monitoring of workers.
- The question of connection of the with the profession.

The main purpose of prophylactic examinations:
- Early detection of early signs of alleged diseases.
- The dynamic monitoring of the health of persons exposed to the influence of unfavorable factors of production.
- Identification of disease, adverse occurring under the influence of adverse factors of production.
- Development of recommendations aimed at improving the working conditions.
- Elimination or significant reduction in the impact of unfavorable factors of production.
- Conducting individual therapeutic measures.

The role of district physician in PPNID: district physician (workshop physician) is actively engaged in primary prevention, which involves a set of measures aimed at the prevention of the fact of the disease, as well as secondary prevention, ie early detection and treatment of an existing illness.

Objectives of outpatient service
• Identification of risk factors in the population served, to register annually.
  • Implementation of measures for the prevention and correction of risk factors, especially among those with high levels of its development.
  • Actively identify patients with clinical manifestations of the disease, and their follow-up, long-term treatment, including correction of risk factors.

Risk Factors
• RF – characteristics of the subject and the environment which can be associated with the probability of coronary heart disease, cardiovascular and other non-infectious diseases in this person. Using the concept of risk factors, doctor can predict the development of the disease.
  • Primary risk factors – factors directly reflecting the adverse effects on human health (unbalanced diet, smoking, alcohol, lack of exercise).
• **Secondary risk factors** – diseases and pathological syndromes, leading to the development of major non-infectious diseases (e.g. hypercholesterolemia, hypertension, diabetes).

  **Smoking is a risk factor for several diseases**

  "After a nuclear war, famine and plague, the greatest threat to human health is smoking."

  On average, a heavy smoker shortens his life by 7 years. Each cigarette consumption is estimated to reduce life by 14 minutes. In Belarus mortality from diseases caused by smoking rose, life expectancy in the age group of 35-69 years was reduced by 21 years.

  In all age groups, the risk of death from coronary heart disease (ischemic heart disease) is directly dependent on the number of cigarettes smoked daily. Mortality from coronary heart disease in men younger than 45 years with more than 25 cigarettes smoked daily is 15 times higher than among non-smoking men of the same age. Particularly high risk with regard to morbidity and mortality from cardiovascular disease is common for smoking young women, using oral contraceptives containing estrogen for a prolonged period. IHD occurs in them 10 times more often.

  Comparison of the results of coronary angiography with the intensity of smoking revealed that:

  - Smoking 1.5 cigarettes per day has no visible changes in the coronary vessels.
  - Smoking 2 cigarettes striking one coronary artery.
  - Smoking 2.5 cigarettes affects two arteries.
  - Smoking more than 4 cigarettes affects three arteries.

  Smoking is an obligate risk factor in the occurrence of chronic obstructive pulmonary disease (COPD). Smoking intensity is of particular importance in COPD diagnosing. It is calculated with the following formula: number of cigarettes smoked per day divided by 20 and multiplied by the number of years of smoking. If the result is more than 10 there is a reliable risk factor for COPD.

  When combined with other risk factors adverse effects of cigarettes are significantly enhanced. Hypercholesterolemia or arterial hypertension increases risk of IHD about two-fold in male smokers.

  Accumulated data currently indicate that cigarette consumption is accompanied by certain changes in the lipid composition of the blood,
which are regarded as atherogenic (increasing concentrations of all three major lipid components: total cholesterol, LDL, triglycerides).

The effect of smoking on the functional activity of platelets and the coagulation and fibrinolytic state of the blood system is established. Cigarette smoking increases platelet aggregation and thereby contributes to the formation of microaggregants in the bloodstream; stimulates the release a number of biologically active substances from platelet (thromboxane A2, serotonin, platelet-derived growth factor). Platelet derived growth factor is particularly important in atherogenesis, which has the ability to stimulate the migration of smooth muscle cells in the subendothelial space of the vascular wall, the capture of lipoproteins, intracellular synthesis of cholesterol and its esterification processes.

Nicotine is a powerful stimulant of sympathetic ganglia and adrenal medulla, it increases the release of catecholamines in the bloodstream, which, along with the hemodynamic shifts, can activate platelets and blood coagulation. Nicotine stimulates the secretion of corticosteroids, anti-diuretic hormone; it has arrhythmogenic properties. To some extent this may explain the increased risk of sudden death and the development of acute myocardial infarction in chronic smokers.

It should be kept in mind that smoking can distort pharmacological effect of many drugs, e.g. propranolol while smoking does not reduce, but, on the contrary, increase diastolic pressure.

Effect of passive smoking on the frequency of angina attacks has been proven. In women who have a spouse-smoker, the risk of getting lung cancer is 30% higher than that in women whose husbands do not smoke.

Besides the fact that smoking is the most common cardiovascular risk factor among the working population, in smokers, according to WHO, along with heart diseases there is a risk of developing cancer (cancer of the lung, bladder), bronchopulmonary disease, and peptic ulcer disease.

According to WHO, if people on the Earth stop smoking, deaths from disease can be reduced by 19%.

Thus, reduction of smoking rate is one of the priorities of district physician.

**Lack of exercise**

The rapid development of technology, automation, transportation, television has reduced physical activity, and resulted not only in people mental, but also in physical labor. Physical activity has decreased not only at work but also at home, with negative impact on the cardiovascular, respiratory, metabolic, etc.

According to the Framingham prospective study of the death risk from coronary heart disease in physically inactive is 3 times higher than for people with an active lifestyle. The risk of sudden death in patients who have regularly physical exercises is 59% less than the risk for those with sedentary life style.

*Physical inactivity is characterized by 5 hours in a sitting position per day and in less than 10 hours of exercise per week.*

Physical activity is an important measure to prevent the adverse effects of a sedentary lifestyle (obesity, hypertension, cardio-vascular diseases, metabolic disorders).

*Prevention of inactivity*

- Increased physical activity in the form of graduated exercise at least twice a week for 30 minutes at a fast pace (120 steps per minute).
- Physical exercise, aerobic cyclical nature (walking, jogging, swimming, morning exercise).

District doctor – therapist should indicate motion activity for each patient based on their professional activity (at work, at home, at weekends and on public holidays, etc.).

**Obesity**

Obesity is recognized by WHO as ‘epidemic’ of our time because of its high prevalence in the population at high risk of cardio-vascular disease, early disability and premature mortality.

Over the past 20 years, the prevalence of obesity has increased by almost 3 times. The number of people suffering from obesity increases progressively every 10 years by 10%.

According to WHO, more than 1 billion people worldwide are overweight. Obesity has become a social problem in countries with high economic standards of living. Approximately one in five people in these countries is obese.
Obesity is associated with coronary heart disease, hypertension, diabetes mellitus, cholelithiasis, osteoarthritis and gout.

It is known that excess body weight increases the risk of coronary heart disease by 1.3-3.2 times (with an increase in body weight by 30% to twofold).

Mortality from coronary heart disease in people with overweight is 1.7 times higher than in those with normal weight.

District physician, singles out individuals with excess weight by comparing their body mass index (BMI) with its normal values. BMI can be calculated with Quetelet index (QI – body weight in kg/height in m²).

According to the WHO classification (1997), in the whole range of QI variations adults can be of 4 levels:

**Body mass index (Quetelet index)**

- less than 18.5 kg/m² are underweight;
- 18.5-24.9 kg/m² – normal weight;
- 25-29.9 kg/m² – overweight;
- 30 kg/m² or more – obesity.

Especially dangerous is the central type of obesity with predominant deposition of fat in the abdominal region. Frequent combination of visceral obesity, disorders of carbohydrate and lipid metabolism, breathing disorders during sleep and hypertension, and there is a close connection between the pathogenic was the basis for selection of an independent syndrome – "metabolic". Isolation of MS is of great clinical importance, because on the one hand, this condition is reversible, ie with appropriate treatment can achieve extinction, or at least reduce the severity of its main manifestations, and on the other hand, it is preceded by the emergence of diseases such as type 2 diabetes and atherosclerosis – the diseases that are the major causes of increased mortality.

Abdominal type of obesity can be identified by the characteristic redistribution of body fat. This android type of obesity, with a primary deposition of fat in the abdomen and upper body (type "apple"), as opposed to gynoid (type "pear") with the deposition of fat in the hips and buttocks. A number of studies have found a direct proportional relationship between body weight and total mortality.
Type of obesity can be determined by using an index waist/hip (IWH)

- waist circumference (WC) is the smallest circumference measured below the rib cage on the navel;
- hip circumference (HC) is measured at widest point of hips

\[ IWH = \frac{WC}{HC} \]

- 0.8-0.9 – intermediate type of fat distribution;
- less than 0.8 – gynoid type (thigh buttock);
- more than 0.9 – android type (abdominal).

Treatment of Obesity
- A set of dietary recommendations (compliance to energy daily caloric intake).
  - Balanced diet (protein – 15% of total calories – 90-95 g, fat 35% – 80-100 g, carbohydrates – 50% 300-350 g).
  - Food intakes at least 4-5 times a day, the last one should be 2-3 hours before bedtime.
  - Limitation of energy intake to 1800-1900 kcal/day.
  - Fasting days 1-2 times a week.

*Obesity with hypercholesterolemia*

- Do not eat more than 3 egg yolks a week, including egg yolks, used for cooking.
  - Eat less offal (liver, kidney).
  - Limit consumption of all types of sausages, fatty ham, butter and ghee, oil-rich milk and milk products.
  - When cooking, replace roasting and grilling by boiling and steaming.
  - Prefer fish dishes, seafood, prepared with vegetable oil.
  - Use low-fat varieties of dairy products, eat more vegetables and fruits.

*Alcohol as risk factor for somatic diseases*

Chronic alcohol abuse can cause serious damage to the hormonal regulation of the system of human body. Alcohol impairs the function of hormonal regulation of blood pressure and fluid and electrolyte balance, affecting the blood levels of two hormones - vasopressin and atrial natriuretic peptide. These violations persist during periods of abstinence for at least 9 months after giving up alcohol.

It is now established that the heart disease from alcoholism – alcoholic cardiomyopathy, alcoholic heart disease is an independent
nosological form in WHO classification. It has been demonstrated that the frequent intake of significant amounts of alcohol affects both increase in the frequency of lesions of the cardiovascular system, and their severity.

Alcohol affects the liver (liver cirrhosis, alcoholic hepatitis), nervous system (toxic encephalopathy), pancreas (acute and chronic pancreatitis) increases blood pressure and disturbance of water and electrolyte balance, affects the heart (alcoholic cardiomyopathy), kidneys (glomerulonephritis alcoholic), leads to the development of cancer of esophagus, pharynx and larynx.

Chronic alcohol intoxication is accompanied by deficiency of antioxidants (vitamins A, E, folic acid, zinc, selenium), which is also a complex mechanism of carcinogenesis. Even small doses of alcoholic beverages increase the risk of developing prostate cancer by 87%.

From 60 to 80% of cancer cases can be prevented by healthy lifestyle, adequate diet, regular consumption of fruit and vegetables.

Pay attention to markers of chronic alcohol intoxication, which can be determined objectively. These markers include the characteristic appearance with facial flushing, skin capillary network expansion, coated tongue, liver enlargement, tremor of fingers, polyneuropathy, parotid glands, muscle atrophy, venous engorgement of the conjunctiva, Dupuytren's contracture, gynecomastia, transient hypertension, and finally, change of weight, either its deficit or obesity. As markers of alcohol intoxication the following laboratory parameters are also used: increase in blood gamma-glutamyl transpeptidase, aspartate aminotransferase, and alkaline phosphatase.

### Arterial Hypertension

Arterial hypertension (AH) in its socio-medical significance is one of the major health challenges.

In industrialized countries, hypertension affects about 15% of adult population. According to the Ministry of Health of the Republic of Belarus in 2010 in Belarus, there were 1.5 million patients with hypertension, and in the structure of mortality rank it is the first complication of cardiovascular disease (myocardial infarction, cerebral infarction, heart failure).

The problem is of great social significance, since AH is one of the causes of persistent disability. Successful control of hypertension
and prognosis of the disease depends largely on early and correct diagnosis, and on organization and effectiveness of patients’ treatment in the clinic.

PREVENTION OF ARTERIAL HYPERTENSION

Identifying people with risk factors for hypertension:

- Exogenous:
  - Long-term and frequent psycho-emotional stress;
  - cranial trauma and concussions;
  - kidney disease in the past;
  - smoking;
  - alcohol abuse;
  - high salt intake;
  - over-nutrition, especially for those prone to obesity;
  - lack of exercise;
  - different industrial RF’s.

- Endogenous:
  - genetic predisposition;
  - personal traits that appear in childhood and adolescence;
  - psycho-emotional instability with marked sympathoadrenal responses to small stimuli;
  - vegetative dystonia;
  - pregnancy;
  - menopause;
  - reduced gonadal function in men;
  - endocrine system disorders;
  - age.

Diagnosis of hypertension

According to the WHO/ISH guidelines in adults of both sexes of all ages AH is characterized by increased blood pressure > 140/90 mm Hg. AH is revealed by repeated rates during 2-3 visits to a therapist.

Lower blood pressure 130-139 and 85-89 mm Hg is considered normal.

Requirements for treatment of patients with hypertension are set out in the order No. 225 of 03.09.2001.

The goal of primary prevention is elimination or minimizing the negative effects of risk factors for hypertension.
**Risk factors for ischemic (coronary) heart disease**

Ischemic (coronary) heart disease (IHD) remains dominant both in prevalence and mortality in our country and in most developed countries.

Success in reducing morbidity and mortality from cardiovascular disease is largely dependent on the effectiveness of primary prevention of coronary heart disease, in particular, correct approach to the identifying individuals who require urgent preventive actions. Modern preventive cardiology is based on the concept of risk factors.

To assess the overall risk of system it is recommended to use SCORE (Systematic Coronary Risk Evaluation – a systematic assessment of coronary risk) (Tabl.1). It is based on numerous prospective European studies and it predicts the risk of death from atherosclerosis in 10 years. Risk assessment involves the examination of the following risk factors: gender, age, smoking, systolic blood pressure and total cholesterol or the ratio of cholesterol/HDL cholesterol. The criterion of high risk is the probability of death from cardiovascular disease ≥5% (instead of ≥20% for the total risk of coronary events, as before).

Table 1 – SCORE assessment.
Russia in SCORE has the following risks: low risk <5% corresponds to the probability of dying within the next 10 years, average risk – 5-9%, high risk – 10-14% and a very high risk of ≥15%. Special attention is required to patients with a high risk of cardiovascular disease.

Patients with high and very high risk:
- SBP ≥ 180 mm Hg and/or diastolic blood pressure 110 mm Hg
- SBP > 160 mm Hg, low DBP (<70 mm Hg)
- Diabetes
- Metabolic syndrome
- ≥3 risk factors
- Target organ damage (TOD):
  - Left ventricular hypertrophy (LVH) on ECG or Echocardiography
  - Ultrasonic signs of carotid thickening (IMT> 0.9 mm or atherosclerotic plaque)
  - Increase of the arterial wall stiffness
  - Moderate increase in serum creatinine
  - Reduction of SFK or creatinine clearance
  - Microalbuminuria or proteinuria
- Associated clinical conditions

Two groups of risk factors for coronary heart disease are the most important (K.S.Ternovoy et al.):
1. *Socio-cultural (exogenous)*
2. *Internal (endogenous).*

*Socio-cultural risk factors include:*
1. Excessive consumption of food rich in calories, carbohydrate, saturated fat and cholesterol.
2. Lack of exercise.
3. Psycho-emotional overload.
4. Smoking, alcoholism.
5. Prolonged use of oral contraceptives (for women).

*The internal (endogenous) risk factors are:*
1. Hypertension.
2. Increased lipids.
3. Breach of tolerance to carbohydrates.
4. Obesity.
5. Hyperuricemia.
6. Metabolism of electrolytes and trace elements.
7. Hypothyroidism.
8. Cholelithiasis.
11. Age, gender factors.

Success in reducing morbidity and mortality from cardiovascular disease is largely dependent on the effectiveness of primary prevention of IHD.

Risk factors for coronary artery disease are also increasing LDL, triglycerides, hereditary (genetic) predisposition to disease.

Hypertension increases the risk of coronary heart disease in 1-6 times, particularly dangerous, as the cardiovascular risk factors, is a hypertension in young adults.

District physician must carefully collect family history to clarify who of the relatives had angina, myocardial infarction, hypertension, diabetes, to find out whether there was a sudden death at the age of 50-60 years in a family history.

It is known that men with cholesterol levels in blood plasma of 6.5-7.0 mmol have increased risk of IHD, and more than that of men with cholesterol levels up to 4.4 mmol/L.

Cholesterol in the range 5.2-6.2 is the level when physician should indicate the tactics of further treatment. If there are no signs of coronary artery disease, other risk factors should be taken into consideration. If besides this level of cholesterol there are two other factors, hypolipidemic agents should be considered (male + hypertension, male + smoking). If there are signs of IHD, only one risk factor is enough to start active treatment with hypocholesterolemic drugs.

Risk factors for non-specific lung disease (NLD). Basic principles of primary prevention of nonspecific pulmonary diseases

Risk factors for non-specific lung diseases

Exogenous factors:
- Smoking.
- Injuries and deformities of the chest.
- Adverse weather conditions (temperature fluctuation of the environment, humidity, etc.).
• Adverse operating conditions (dust, fumes, exposure to harmful gases and toxic chemicals).

*Endogenous factors:*
• Frequent or long flowing respiratory infections (ARI three or more times a year, or 14-20 days 2-3 times a year).
• The presence of foci of chronic infection in the body (especially in the paranasal sinuses, nasopharynx, and upper respiratory tract).
• Transferred pulmonary tuberculosis.
• Moved acute pneumonia (at least 2 times).
• Hereditary predisposition (the presence of the patient or his relatives of pulmonary tuberculosis, asthma, cystic fibrosis, etc.).

The high degree of danger NLD determined when a person has five or more risk factors, or 3 significant risk factors such as smoking, the presence of foci of chronic infection, myocardial tuberculosis.

Average degree of danger when there are 2-3 RF.
Minimum degree of danger when there is one RF.

It is necessary to carry out a comprehensive examination for everyone with risk factors for early detection of the active form of the disease. Most important influence on the emergence and development of NLD belongs to age, gender, disease, acute pneumonia, acute bronchitis, acute respiratory infections and flu, duration and intensity of smoking, hard physical labor, chronic comorbidities of throat and meteorological factors.

Thus, the main elements of primary prevention of NLD are combating dust and gas jobs in industry and agriculture, and the promotion of healthy lifestyles, hardening of the body and physical therapy, management of employment and vocational guidance, proper treatment of acute respiratory illness (ARI, acute bronchitis and acute pneumonia). These measures should be implemented comprehensively.

**Risk factors for diseases of the digestive system**

The prevention of diseases of the digestive system provides active detection of healthy individuals at increased risk of disorders of the digestive system

*Risk factors:*
1. Family history.
2. Functional disorders of the mucous membrane of the stomach
and duodenum in the bull and acid-secretory function.

3. Frequent stressful situations.
4. Poor nutrition, excessive spices, coffee and alcohol.

For the primary prevention population screening is important, with good methodological approach to the survey.

Predisease can be defined as the unrealized risk of disease which is caused by external factors and lower adaptive capacity of the organism.

**Secondary prevention (dispanserization)**

It is a set of health interventions aimed at early detection and treatment of existing diseases. It includes specific measures taken by the medical institutions in the prevention of progression of existing diseases, their early detection, their effective treatment and rehabilitation.

The goal of secondary prevention is to prevent the possibility of exacerbation or complications of emerging diseases, i.e. to slow down the progression of diseases.

Secondary prevention provides for medical examination of the adult population of the Republic of Belarus. Organizations for examination are set by the Ministry of Health of Belarus decree № 51 dated June 1, 2011.

Clinical supervision of the adult population of Belarus is a system of medical interventions aimed at detecting diseases or factors that affect their appearance, at assessing health of every citizen of the Republic of Belarus, which includes:

- clinical examination;
- dynamic monitoring of health during dispanserization;
- promoting healthy lifestyles and responsibility for health.

Citizens of the Republic of Belarus have right to pass examination when medically remain under medical supervision in a dynamic health care organizations that provide outpatient care for adults.

Dispensary examination is carried out with some tests determined for each age group of adults. Results of examination are recorded in the medical outpatient (Form 025/y) card.

Considering results of laboratory, clinical and instrumental investigations and planning necessary medical measures physician determines what group of dynamic dispensary observation a patient belongs to:

D (I) – healthy citizens, with no complaints about the state of
health, who during examination do not reveal any acute or chronic dysfunction of individual organs and body systems, as well as those having slight variations in health status (no tendency to progression) which have no effect on the ability to work.

D (II) – almost healthy citizens with a history of risk factors for chronic diseases, acute illnesses, which can lead to chronic disease process (including frequent or long-term ill persons (hereinafter - FLIP). FLIP are citizens who often (6 or more times a year) or for a long period (more than 40 days a year in total) suffer from acute diseases, as well as individuals with chronic diseases in remission without dysfunction of organs and body systems).

D (III) – citizens with chronic diseases with impaired function of organs and systems of the body and (or) periodic exacerbations.

D (IV) – citizens with a disability.

In each group, the dynamic dispensary observation should be carried out taking into account risk factors for certain diseases or exacerbation of existing chronic disease.

People from group D (I) should have clinical examination at least once in two years.

Dispensary dynamic monitoring of people from groups D (II-III) should be carried out primarily taking into account state of the disease.

Multiple outpatient examination and the volume of the survey, including appointment of additional medical tests, examinations, diagnostic tools are determined by physician performing outpatient follow-up, taking into account the degree of functional impairment, the frequency of relapses (exacerbations), according to the Decree of the Ministry of Health of Belarus № 51 dated June 1, 2011.

**Topic 2: Diagnostics and relief of hypertensive crises at preadmission stage**

According to the Russian National Scientific-Practical Society ambulance for the last 3 years the number of medical emergencies at the hypertensive crises (HC) and the number of admissions in Russian Federation has increased by 1.5 on average. Not more than 33% patients with arterial hypertension (AH) express their willingness to
carry on continuous therapy (according to survey of patients), so we can predict the increase of HC. HC is the most predictive of adverse AH: 25-40% of patients undergoing complicated HC die within the next 3 years from kidney failure (Grade A) or stroke (grade B). This risk increases with age (grade A), with essential hypertension (grade A), with increased serum creatinine (A level), and serum urea above 10 mmol/l (grade B), with a longer duration of AH (grade B), the presence of 2 and 4-degree hypertensive retinopathy (grade C), in 3.2% of patients renal failure develops, requiring dialysis (grade B). Therefore, HC refers to AH manifestations determining mortality from its complications. All the points mentioned above need to be detected timely to eliminate HC at preadmission stage and for doctors to form a clear view of the use of drugs for emergency aid to the sudden increase in blood pressure (BP).

Defining HC: a sudden rise in BP by 20-25% and 10-15% of systolic/diastolic individually to high values at the minimum of subjective and objective symptoms is regarded to be uncomplicated HC. Complicated HC is characterized by dangerous or violent manifestations with subjective and objective evidence of cerebral, cardiovascular and autonomic disorders.

Predisposing factors of HC include frequent stressful situations, alcohol, abolition of antihypertensive therapy, intense exercise, receiving large amounts of water and salty foods, drinking lots of coffee, heavy smoking, excessive mental strain from lack of sleep, eating cheeses containing tyramine (cheddar, etc.), fluctuations in the atmospheric pressure, treatment with glucocorticoids, nonsteroidal anti-inflammatory drugs, etc. These factors are the background for the development of AH, they exacerbate its course. Some of them, as well as several others, may be the direct cause of the HC, including acute psycho-emotional trauma, physical stress, alcohol loads, use of sympathomimetics (cocaine and etc.), severe allergic reaction, intercurrent respiratory tract infection, acute urinary retention, severe head trauma, extensive burns of the skin, surgical stress, and others. In response to the irregular antihypertensive therapy (often with promiscuous receive β-blockers and clonidine) so-called "rebound crisis" can occur and it can be accompanied by complications.
Both predisposing and precipitating factors of HC lead to pathogenetic development of HC: there is hyperactivation of the sympathetic nervous system, which is accompanied by release of norepinephrine, which raises the tone of the arteries and arterioles. The endothelium is a large auto-and paracrine organ, responsible for increase in production of thromboxane and endothelin (potent vasoconstrictor factors), as well as for decrease in production of nitric oxide and prostacyclin (vasodilators). Hypothalamus increases the secretion of vasopressin, which causes spasm of the arteries and arterioles. The body is an acute sodium and water retention, increasing the tone of the arterioles, which leads to an increase in total peripheral resistance. Activation of the renin-angiotensin-II-aldosterone system and calcium mechanism of smooth muscle cells of arteries and arterioles leads to spasm, as well as to an increase in total peripheral resistance.

Currently, there are several classifications of the HC, but the most commonly used is based on the clinical classification of A.L. Myasnikov, N.A. Ratner, 1968, with the crises I and II (Tabl. 1).

**Table 1 – Clinical HC classification by A.L. Myasnikov, N.A. Ratner**

<table>
<thead>
<tr>
<th>Crisis of type I  (hyperkinetic or adrenal)</th>
<th>Crisis of type II (hypokinetic or norepinephrine)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• headache, dizziness, nausea;</td>
<td>• intense headaches, often with vomiting, dizziness;</td>
</tr>
<tr>
<td>• excitement, hand tremor, throbber and shaking all over the body;</td>
<td>• visual disturbances (blur, fog, flashing &quot;flies&quot;);</td>
</tr>
<tr>
<td>• palpitations;</td>
<td>• condition of stupor;</td>
</tr>
<tr>
<td>• red spots on skin, sweating, excessive urination at the end of the crisis;</td>
<td>• parastezia throughout the body;</td>
</tr>
<tr>
<td>• Increased blood coagulation;</td>
<td>• pain in the heart;</td>
</tr>
<tr>
<td>• leukocytosis in peripheral blood;</td>
<td>• possible transient paresis;</td>
</tr>
<tr>
<td>• increase in blood glucose;</td>
<td>• Increased blood coagulation;</td>
</tr>
<tr>
<td>• increase mainly SBP, increased heart rate, increased SI, MO, ND;</td>
<td>• leukocytosis in peripheral blood;</td>
</tr>
<tr>
<td>• duration of a crisis from several minutes to several hours;</td>
<td>• normal blood glucose;</td>
</tr>
<tr>
<td>• no complications.</td>
<td>• increase mainly DBP, bradycardia, increased SVR;</td>
</tr>
<tr>
<td></td>
<td>• duration of a crisis from a few hours to several days;</td>
</tr>
<tr>
<td></td>
<td>• complications.</td>
</tr>
</tbody>
</table>
**Hypertensive emergencies** can be defined as severe elevations of BP in the presence of acute target organ damage. Acute coronary syndromes, dissecting aortic aneurisms, acute pulmonary oedema, hypertensive encephalopathy, acute cerebral infarction, intracerebral haemorrhage, or acute arterial bleeding or eclampsia represent clinical conditions in which an immediate blood pressure reduction is needed to prevent the progression of target-organ damage (TOD) (Tabl. 2). Hypertensive urgencies are characterised by severe elevations in BP (>180/120 mm Hg) without evidence of acute TOD. In hypertensive urgencies BP can usually be reduced in the emergency department (ED) by orally administered drugs without hospital admission and with ambulatory follow-up [1].

**Table 2 – Hypertensive emergencies**

<table>
<thead>
<tr>
<th>Hypertensive encephalopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe hypertension associated with acute target organ damage:</td>
</tr>
<tr>
<td>• acute coronary syndromes</td>
</tr>
<tr>
<td>• pulmonary oedema</td>
</tr>
<tr>
<td>• acute aortic dissection</td>
</tr>
<tr>
<td>• intracerebral haemorrhage, subarachnoid haemorrhage acute brain infarction</td>
</tr>
<tr>
<td>• acute or rapidly progressing renal failure</td>
</tr>
<tr>
<td>Severe hypertension after thrombolysis for ischaemic stroke</td>
</tr>
<tr>
<td><em>Pheochromocytoma</em> crisis</td>
</tr>
<tr>
<td>Guillain-Barré syndrome</td>
</tr>
<tr>
<td>Spinal cord injury</td>
</tr>
<tr>
<td>Drugs related hypertension (sympathomimetics. cocaine, phencyclidine, phenylpropanolamine, lysergic acid diethylamide, cyclosporine, antihypertensive treatment withdrawal, interaction with MAO inhibitors)</td>
</tr>
<tr>
<td>Eclampsia</td>
</tr>
<tr>
<td>Postoperative bleeding</td>
</tr>
<tr>
<td>Post coronary artery bypass hypertension</td>
</tr>
</tbody>
</table>

**Initial evaluation**

Appropriate triage of patients is a crucial part of the initial evaluation. After a complete history (with particular attention paid to pre-existing hypertension and TOD) and an accurate physical examination (including fundoscopic examination), selected laboratory studies such as urinalysis, creatinine, urea, electrolytes, and a full blood count should be performed. When a secondary form of hypertension is
suspected a sample for plasma renin activity, aldosterone, and catecholamines should also be drawn. Each patient is advised to carry on an electrocardiogram and a chest radiogram (Tabl. 3).

Table 3 – Diagnostic workup

<table>
<thead>
<tr>
<th>Repeated blood pressure measurements (first measurements on both arms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical history and physical examination:</td>
</tr>
<tr>
<td>● cardiovascular</td>
</tr>
<tr>
<td>● CNS</td>
</tr>
<tr>
<td>● fundus oculi</td>
</tr>
<tr>
<td>Selected laboratory studies:</td>
</tr>
<tr>
<td>● urinalysis, creatinine, urea, electrolytes, and a full blood count</td>
</tr>
<tr>
<td>● when a secondary form of hypertension is suspected, a sample for plasma renin activity, aldosterone, and eventually catecholamines should also be drawn</td>
</tr>
<tr>
<td>Electrocardiography</td>
</tr>
<tr>
<td>Chest X rays</td>
</tr>
<tr>
<td>Further investigations (according to the clinical presentation):</td>
</tr>
<tr>
<td>echocardiography (N, TE)</td>
</tr>
<tr>
<td>● brain CT scan or MRI</td>
</tr>
<tr>
<td>● abdominal ultrasonography</td>
</tr>
<tr>
<td>● thoraco-abdominal CT scan or MRI</td>
</tr>
<tr>
<td>● vascular ultrasound</td>
</tr>
</tbody>
</table>

In elderly patients the clinical course of HC has its own characteristics: gradual development of clinical symptoms (rise for a few hours), prolonged or recurrent, persistent headache often accompanied by severe dizziness, nausea, and vomiting, drowsiness, confusion, transient paresis and sensory disturbances. Older people have a high risk of complications, including fatal (stroke, myocardial infarction, aortic dissection, etc.).

Treatment of uncomplicated HC crisis should start with the drugs under the tongue or internally. Antihypertensive drugs for oral use in HC are presented in Table 4.

As an aid in the HC to improve regional hemodynamics dibazol can be used at a dose of 30-40 mg (3-4 ml of a 1% solution intramuscularly (i/m)). Its hypotensive effect is due to a decrease in cardiac output and peripheral vascular expansion due to its antispasmodic action. With intravenous (i/v) administration the onset occurs in 10-15 minutes, with i/m – 30-40 minutes. The duration of
action comprises up to 12 hours. Side effects may be paradoxical increases in blood pressure, and sometimes excessive sweating, hot flashes, dizziness, headache, nausea, and allergic reactions. Contraindications: severe heart failure, hypersensitivity to the drug.

Table 4 – Antihypertensive drugs for oral use in the HC

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage</th>
<th>Start and duration of effect</th>
<th>Side effects and contraindications</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaprilin (propranolol, Inderal)</td>
<td>10, 20, 40 mg sublingually</td>
<td>5-30 min, 4-6 hours</td>
<td>Side effects: bronchospasm, bradycardia. Contraindications: AV-block II and III degree., SA block, bradycardia (heart rate less than 55 bpm / Min), SSS, chronic heart failure IIB-III stages, acute heart failure</td>
<td>The dosage depends on the severity of tachycardia and BP levels</td>
</tr>
<tr>
<td>Captopril</td>
<td>25, 50 mg sublingually, if necessary, again on 25 mg every 30-60 minutes</td>
<td>5-10 min, 4-6 hours</td>
<td>Side effects: angioneurotic edema, dry cough. Contraindications: bilateral renal artery stenosis, the state after a kidney transplant, hemodynamically significant aortic stenosis, left AV holes, hypertrophic cardiomyopathy</td>
<td>Not shown in pregnancy, including eclampsia during pregnancy</td>
</tr>
<tr>
<td>Moxonidine</td>
<td>0.4 mg</td>
<td>20-30 min</td>
<td>Contraindications: AV-block II and III degree., SA block, bradycardia (heart rate less than 55 bpm / Min), SSS, chronic heart failure IIB-III stages, ACS</td>
<td></td>
</tr>
<tr>
<td>Drug</td>
<td>Dosage</td>
<td>Start and duration of effect</td>
<td>Side effects and contraindications</td>
<td>Note</td>
</tr>
<tr>
<td>-----------</td>
<td>---------------------------------</td>
<td>-----------------------------</td>
<td>-----------------------------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------</td>
</tr>
<tr>
<td>Nifedipine</td>
<td>10, 20 mg sublingually, if necessary, again after 30 minutes</td>
<td>5-30 min, 4-6 hours</td>
<td>Side effects: flushing of the skin of face and neck, tachycardia, drowsiness, headache, dizziness.</td>
<td>Contraindications: the syndrome of &quot;tachy-brady&quot;, acute coronary insufficiency, severe heart failure, hemodynamically significant aortic stenosis, hypertrophic cardiomyopathy, acute ischemic stroke</td>
</tr>
</tbody>
</table>

**Treatment of hypertensive emergencies**

Patients should be admitted to an intensive care unit for clinical surveillance and continuous BP monitoring. Aggressive treatment with parenteral drugs is the preferred approach; in the majority of cases, however, the initial goal should be a partial reduction (not normalisation) of BP, with a reduction in BP of no more than 20-25% within the first minutes and up to one or two hours, with possible cautious further decreases in subsequent hours. In most hypertensive emergencies a rapid lowering of BP is beneficial, with the exception of cerebrovascular accidents, in which it is advisable to take a more cautious approach. An excessive reduction of BP values is potentially dangerous, possibly leading to ischaemic complications such as acute myocardial infarction and stroke.

Several parenteral agents are available for the treatment of hypertensive emergencies (Tabl. 5); the choice of first-line antihypertensive agents should be tailored to the patient's clinical status. Nitroprusside is a highly effective short-acting arteriolar and venous dilator, which can be used in most hypertensive emergencies. In patients with primary intracerebral haemorrhage caution is needed
because of the potential antiplatelet effect and intracranial pressure increase. The risk of cyanate toxicity is greater when the drug is used for long periods (days) or in patients with hepatic or renal dysfunction. With nitroprusside, BP should be continuously monitored intraarterially; hypotension can, however, be managed in most cases by discontinuing the infusion.

Table 5 – Drugs for hypertensive emergencies

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Onset</th>
<th>Duration</th>
<th>Adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium nitroprussiate</td>
<td>0.25-10 μg/kg/min</td>
<td>Immediate</td>
<td>1-2 min</td>
<td>Hypotension, vomiting, cyanate toxicity</td>
</tr>
<tr>
<td>Labetalol</td>
<td>20-80 mg bolus</td>
<td>5-10 min</td>
<td>2-6 h</td>
<td>Nausea, vomiting, heart block, bronchospasm</td>
</tr>
<tr>
<td></td>
<td>1-2 mg/min infusion</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glyceryl trinitrate</td>
<td>5-100 μg/min</td>
<td>1-3 min</td>
<td>5-15 min</td>
<td>Headache, vomiting</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Enalaprilat</td>
<td>1.25-5.00 mg bolus</td>
<td>15 min</td>
<td>4-6 h</td>
<td>Hypotension, renal failure, angioedema</td>
</tr>
<tr>
<td></td>
<td>0.1-0.6 mg/μg/kg/min</td>
<td>5 min</td>
<td>2 h</td>
<td>Hypotension</td>
</tr>
<tr>
<td>Furosemide</td>
<td>40-60 mg</td>
<td>5 min</td>
<td>2 h</td>
<td></td>
</tr>
<tr>
<td>Fenoldopam</td>
<td>10 mg bolus</td>
<td>10 min</td>
<td>2-6 h</td>
<td>Reflex tachycardia, flushing</td>
</tr>
<tr>
<td>Nicardipine</td>
<td>2-10 mg/h</td>
<td>5-10 min</td>
<td>2-4 h</td>
<td>Reflex tachycardia</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>10-20 mg bolus</td>
<td>10 min</td>
<td>2-6 h</td>
<td>Reflex tachycardia</td>
</tr>
<tr>
<td>Phentolamine</td>
<td>5-10 mg/min</td>
<td>1-2 min</td>
<td>3-5 min</td>
<td>Reflex tachycardia</td>
</tr>
<tr>
<td>Urapidil</td>
<td>25-50 mg bolus</td>
<td>3-4 min</td>
<td>8-12 h</td>
<td>Sedation</td>
</tr>
</tbody>
</table>

**Nitroglycerin** is a venous and, to a lesser extent, arteriolar dilator, particularly indicated in acute coronary syndromes and pulmonary oedema. **Labetalol** is an alpha- and beta-adrenergic blocker, which can be given as an intravenous bolus or infusion; it is highly effective and is indicated in most hypertensive emergencies, in particular in aortic dissection and in acute coronary syndromes. It may be given also after cocaine or amphetamine use, which may induce transient but significant hypertension leading to stroke and/or serious cardiac damage. **Urapidil**, an alpha-blocker with additional actions in the central nervous system (it activates 5-HT1 A receptors), has also been found effective since it induces vasodilatation without tachycardia. Finally, it must be remembered that **furosemide** can be
particularly indicated when volume overload is present, as in left ventricular failure. In the presence of volume depletion, in contrast, diuretics can cause additional reflex vasoconstriction and should therefore be avoided.

**Specific hypertensive emergencies**

In patients with **acute coronary syndromes** a severe elevation of BP values is not uncommon; on the other hand, myocardial ischaemia may also be induced by acute elevations in BP in patients without haemodynamically relevant coronary artery disease through an increase in left ventricular wall stress and myocardial oxygen consumption. In this setting intravenous vasodilators, such as nitroglycerin and nitroprusside, should be the initial drugs, in combination with a beta-blocker (labetalol, metoprolol, esmolol, or atenolol), which may further decrease Bp and reduce heart rate and, consequently, myocardial oxygen consumption. In the presence of **acute left ventricular failure** BP should be rapidly controlled. The preferred drugs are intravenous nitroglycerin or nitroprusside in combination with loops diuretics for volume overload control. In patients with **aortic dissection** and hypertension BP control is crucial. The treatment should be started immediately and systolic BP rapidly reduced to less than 100 mm Hg; the ideal drug should not only allow the reduction of BP but also reduce heart rate and cardiac contractility with the aim of reducing stress on the aortic wall. This can be achieved with a combination of a beta-blocker and a vasodilator, such as nitroprusside or nitroglycerin, administered intravenously. **Pheochromocytoma crises** can be managed with an intravenous alpha-bijocker such as phentolamine, followed by concomitant infusion of a beta-blocker; nitroprusside may also be added. Beta-blockers should always be associated with alpha-blockers in patients with pheochromocytoma since inhibition of beta-receptor-induced vasodilation may lead to a further increase in BP values in the presence of alpha-adrenergic vasoconstriction. Simultaneous alpha- and beta-blockade may also be achieved with monotherapy with labetalol. In patients with **acute stroke** the use of antihypertensive therapy is still controversial. Autoregulation of blood flow is impaired in ischaemic areas of the brain, and BP reduction may further reduce flow in the ischaemic penumbra and further expand the size of the
infarction. It seems reasonable to recommend the institution of antihypertensive treatment only in the presence of BP values above 220/120 mm Hg (or mean BP > 140 mm Hg) in ischaemic stroke and to obtain an initial reduction of BP values of about 10-15%. Treatment may be initiated with intravenous labetalol, and, if needed, with nitroprusside or nitroglycerin. In patients with acute stroke treated with thrombolysis BP should be kept below 185/110 mm Hg. In primary **intracerebral haemorrhage**, treatment should be started if BP values are greater than 180/105 mm Hg. For less marked elevations of blood pressure the available data do not support the initiation of antihypertensive treatment in the early phases of stroke. In fact, after the promising results of ACCESS study (342 patients with acute stroke), tore recently, SCAST study showed no evidence of a beneficial effect of careful blood pressure lowering treatment with an angiotensinn-receptor blocker in more than 2000 patients with acute ischaemic (85%) or haemorrhagic (14%) stroke and a mean blood pressure of 171/90 mm Hg. These results are further reinforced by those of a meta-analysis performed by the same authors, including more than 3600 patients, which confirmed the lack of benefit of BP lowering in acute stroke and mild to moderate elevations in BP. For haemorrhagic stroke, in the recently published INTERACT study, in which 404 patients with intracerebral haemorrhage and systolic BP between 150 and 220 mm Hg, underwent early intensive BP-lowering treatment, a significant reduction in haematoma growth over 72 hours was observed in actively treated patients. The ongoing main study (INTERACT2) will assess the effect of early intensive BP-lowering on functional outcome on a larger sample of patients (2800). Therefore, while awaiting the results of the ongoing studies, routine BP lowering in the acute phase of stroke in patients with mild to moderate elevations in blood pressure does not appear advisable. **Acute postoperative hypertension** is not uncommon, particularly after cardiothoracic, vascular, head and neck, and neurosurgical procedures. For most non-cardiac types of surgery there is no agreement on BP thresholds for treatment, and the patient's baseline BP, type of surgical procedure, and associated clinical conditions should be taken into account in patient management. It seems reasonable to maintain blood pressure within 20% of preoperative
arterial pressure. For cardiothoracic surgery there is more evidence of an increased risk associated with a postoperative increase in BP values, which should be kept below 140/90 mm Hg. Labetalol (and other beta-blockers), nitroprusside, nitroglycerin, or fenoldopam should be the preferred intravenous drugs for BP control.

**Treatment of hypertensive urgencies**

In the majority of patients with severe hypertension no signs of acute TOD are usually observed. In these patients BP should be lowered gradually over a period of 24-48 hours; this can often be achieved by orally administered drugs without hospital admission and with close ambulatory follow-up. Clinical surveillance is advisable during the first few hours after drug administration. Blood pressure lowering should be gradual: there is no proven benefit from a fast drop in BP in asymptomatic patients who have no evidence of acute TOD, and a precipitous fall in BP could do more harm than good. In Table 4 recommended oral agents for hypertensive urgencies are reported. An initial approach with a combination of antihypertensive drugs increases the likelihood of effective BP reduction. The degree of BP reduction induced by sublingual nifedipine can neither be predicted nor controlled and this preparation is not recommended (14).

Magnesium sulfate at a dose of 1000-2500 mg (5-20 ml of a 20% solution) / slow (for 7-10 minutes or more), and if you cannot provide the in / in a product is acceptable (as an exception) in intramuscular administration of the drug in the form of heat, followed by heating of the injection site, as / m introduction painful and fraught with the development of complications, the most unpleasant of which the formation of infiltrates buttocks. Magnesium sulfate has a vasodilator, sedative and anticonvulsant effect, lowers blood pressure and reduces the intracerebral brain swelling. Its use is particularly indicated for CC, followed by the development of seizures (eg, eclampsia of pregnancy), as well as the appearance of ventricular arrhythmias in the background of an increase in blood pressure. Hypotensive effect develops within 15-25 minutes after administration. Side effects: respiratory depression (eliminated on / in the 5.10 mL of 10% calcium chloride solution), bradycardia, atrioventricular block II degree. Contraindications: hypersensitivity, male, bradycardia, AV block II degree.
In HC with severe autonomic and emotional coloring (optional panic attack) better sublingual use propranolol (20 mg), or captopril (25-50 mg) in conjunction with sedatives – diazepam 0.5% – 1-2 ml/m.

**Tactics of district physician for further case management.**
- Correction of predisposing factors or directly cause of HC.
- Correction of antihypertensive therapy.
- Self-monitoring of patient’s blood pressure at least three times a day in the next few days to stabilize the blood pressure indices.
- Patient examination.

**PRACTICAL LESSONS (topic’s plan)**


**Aim of lesson:**
1) to get acquainted with organization of preadmission stage medical care, structure of polyclinic, modern abilities of diagnostic and treatment of diseases at preadmission stage, organization of dispensarization of adult citizens of the Republic of Belarus;
2) to find out main principles of medico-social examination of temporal and stable disability, order of direction to MREB.

**Questions:**
1. Polyclinic as the main link of primal medical care. Structure of the polyclinic, its main departments and functions.
2. District general medicine care organization. District physician’s rights and responsibilities.
3. Coordination of district general physician’s work with other doctors of hospitals and ambulance care.
4. Documentation of district physician. Main criteria of district general physician work.
5. Modern abilities of diagnostics and treatment in polyclinic.
6. Main principles of medico-social examination (MSE).
7. Disability and its types.
8. Main principles of temporal disability (TD) examination.
9. Organization of TD examination, TD terms and documentation.
10. Patients with prolonged or often sickness, peculiarity of examination.
11. MCC (medical-counselling committee): staff and functions.
13. Assingment of patients to MREB. Documents needed for assignment to MREB.
15. Main paragraphs of the law of the Republic of Belarus "About prevention and rehabilitation of the disabled".

References:

2. Documentations forms for lesson topic registered in Belarus (in the department)

LESSON № 2. Acute respiratory infections (flu and other acute respiratory viral infections (ARVI)), acute tonsillitis. Diagnostics, treatment, tactics, medico-social examination of ARVI and acute tonsillitis. Dispanserization in case of acute tonsillitis

Questions:

1. Acute respiratory infections (influenza, ARVI).
   1.1. Etiology, pathophysiology, epidemiology of acute respiratory infections.
   1.2. Clinical features of different ARVI and flu. Complications of flu.
   1.3. Diagnosis and differential diagnosis ARVI and flu. Indications for hospitalization.
   1.4. Treatment and prophylaxis of ARVI and flu.
2. Acute tonsillitis in district general physician practice.
   2.1. Etiology, pathophysiology, classification of acute tonsillitis.
   2.2. Clinical features, different types of acute tonsillitis.
   2.3. Instrumental and laboratory methods of examination of patient.
   2.4. Differential diagnosis of acute tonsillitis.
   2.5. Treatment of acute tonsillitis.
   2.6. Temporal disability to work, dispensarization, prophylaxis of acute tonsillitis.

References:

LESSON № 3. Acute bronchitis and pneumonia. Diagnostics, treatment, tactics, medico-social examination, dispensarization, primal prophylaxis at preadmission stage

Aim of lesson: to teach students to diagnose and treat acute bronchitis and pneumonia at preadmission stage, to know indications for hospitalization.

Questions:
1. Definitions of terms “acute bronchitis” and “pneumonia”, etiology and definitions of these diseases.
2. Plan of examination of patient with acute bronchitis or pneumonia at preadmission stage.
3. Diagnosis formulating, indications for hospitalization.
4. Treatment at preadmission stage.
5. Indications for acute bronchitis therapy with antibiotics.
6. Complications of acute bronchitis and pneumonia.
7. Medico-social examination of acute bronchitis and pneumonia.
8. Dispanserization and rehabilitation of patients after acute bronchitis and pneumonia.
REFERENCES:

LESSON № 4. Ischemic heart disease (IHD): diagnostics, treatment, tactics, medico-social examination, dispensserization, primal prevention of different types of IHD at preadmission stage. Treatment of angina pectoris. Urgent treatment of unstable angina at preadmission stage

Aim of lesson: to teach students to diagnose and treat IHD at preadmission stage, to know indications for hospitalization.

Questions:
1. Classification of IHD.
2. Classification and clinical features of angina pectoris. ECG diagnosis, functional tests. Laboratory diagnosis.
3. Differential diagnosis of diseases with chest pain (diseases of heart, lung and chest pain)
4. Treatment of angina pectoris.
5. Urgent treatment of unstable angina at preadmission stage.
6. Dispanserization of patients with angina pectoris.

REFERENCES:
LESSON № 5. Primary (essential) and secondary hypertension, somatoform dysfunction of autonomic nervous system (neurovegetative dystonia). Diagnostics, treatment, tactics, medico-social examination, dispanserization, primary prevention at preadmission stage

Aim of lesson: to teach students to diagnose, to study aspects of treatment, dispanserization, examination of disability to work, prevention in patients with arterial hypertension at preadmission stage.

Questions:

1. Classification of arterial hypertension (ESH 2011); stratification of risk factors.
2. Differential diagnosis of arterial hypertension.
3. District general physician tactics in case of revealing of patient with arterial hypertension.
4. Aspects of treatment of patients with arterial hypertension at preadmission stage.
5. Algorithm of treatment of arterial hypertension in patients with additional pathology, in pregnant women.
6. Classification of symptomatic hypertensions.
7. Conception somatoform dysfunction of autonomic nervous system (neurovegetative dystonia).
8. Aspects of medico-social examination, dispanserization, rehabilitation, treatment in sanatoriums and resorts, primary prevention of arterial hypertension at preadmission stage.

References:

LESSON № 6. Chronic gastritis, peptic ulcer diseases of stomach and duodenum. Diagnosis, treatment, tactics, medico-social examination, dispanserization, primary prevention at preadmission stage. Urgent care in case of acute abdominal pain and probable gastrointestinal bleeding

Aim of lesson: to teach students to diagnose with differential diagnosis and tactics in case of functional dyspepsia, chronic gastritis and peptic ulcer diseases of stomach and duodenum.

Questions

1. Classification and clinical features of chronic gastritis.
2. Classification and clinical features of peptic ulcer diseases of stomach and duodenum.
3. Aspects of diagnosis, treatment, tactics, medico-social examination, dispanserization, primal prevention of the above-mentioned diseases at preadmission stage.
4. Differential diagnosis of diseases with acute abdominal pain (onset of cholecystolithiasis or pancreatitis).
5. Urgent care in case of acute abdominal pain and probable gastrointestinal bleeding.

References:

Lesson № 1. The content of district physician’s work.

The content of district physician’s work. Basic outpatient medical records. Dispanserization

Currently, the leading element of our health care is outpatient care. It is known that most of patients begin and end their treatment in polyclinics. The central figures are physicians, endowed with certain knowledge on related subjects, that is, they can function as general practitioners.

The arrangement of the entire polyclinic is based on territorial principle, i.e. district medical care to people living on a fixed area or shop principle in industry, construction companies, etc. This principle ensures the continuity of observation of patients by the same physician, enables early detect diseases in patients, provides comprehensive preventive measures, effective clinical examination, promotes to getting acquainted with conditions of life, work, etc. It is widely believed that general practitioner and family physician is not the same. General physician is a specialist in wide-oriented basic medical specialties and with the ability to provide a multi-disciplinary outpatient care for common diseases and emergency conditions. Family doctor is a doctor specially trained to provide a multi-disciplinary primary health care to family members, regardless of gender and age. International documents consider GP and family therapist as identical concepts. Experts from different countries have agreed to write a general practitioner (GP) in fraction with family doctor (GP/FD), as long as their functions can not be divided.

The main aspects of the district physician’s work:
- providing skilled therapeutic care to outpatient and home care;
- organization and implementation of preventive measures in the population area, clinical examination;
- medical-social examination, participation in the MCC, registration of referrals to MREB;
- referrals to health care institutions, the sanatorium-treated chickens;
- sanitary and anti-epidemic work;
- public education;
- analysis of the incidence area, activity analysis, reporting.

District physician in his work is directly subordinate to the head of therapy department, and in his absence to the deputy chief physician of polyclinic. District physician is directly reported by nurse.

The district physician must provide:
- Timely therapeutic assistance to the population of the district in polyclinic and at home.
- Emergency medical care to patients, regardless of their place of residence in case of their appeal in acute states, injuries, poisoning.
- Timely hospitalization of medical patients with a mandatory preliminary examination, routine hospitalization.
- Dispanserization of patients when necessary in clinic and other health institutions.
- application of modern methods of prevention, diagnosis and treatment, including the treatment and rehabilitation treatment (medications, diet, exercise therapy, massage, physical therapy, etc.).
- Examination of temporary disability of patients.
- Organization and necessary activities for clinical examination of adult population of the district (identification, registration, dynamic monitoring, health and recreational activities) in accordance with the list of entities that are subject to dispensary observation by district physician, the analysis of the effectiveness and quality of clinical examination.
- Making diagnostic decisions for patients carrying on medical examination and departing abroad.
- Organization of vaccinations and deworming of the population.
- Early detection, immediate notification of head of therapeutic department (and in his absence of the head of the institution) and the doctor-infectionist on all cases of infectious diseases or infection-suspicion patients, on food and professional poisoning, and on all cases of violation of regime and of antiepidemic requirements infectious patients, leaves for home treatment. Notification of the appropriate center of hygiene and epidemiology.
- Systematic improvement of their own and nurse’s skills and knowledge;
- Active and systematic health education among the public area, the fight against bad habits and training of public active persons of district;
- Identification of people with risk factors for AIDS, their registration and examination in accordance with orders and instructions;
- Identification of people suspected of drug addiction, toxicomania and alcoholism;
- Keeping to ethic principles in their work.
Profile register or medical passport section is the document, which helps district physician purposefully organize and estimate the entire treatment and preventive work. Medical passport may contain the following information:

• plan of the district indicating the number of storeys of buildings, number of apartments, the location of schools, kindergartens, etc.;
• list of a variety of industrial and other facilities with the number of employees;
• demographic data, description of the age and sex composition of the population in the area, the number of teenagers taken from the children's clinics;
• main quarterly performance (a measure of outpatient admission, patient care at home, the percentage of active visits, etc.);
  • information on infectious diseases;
  • information on the incidence (measured at reported cases of diseases, quarterly, on separate nosological groups specified in the form № 271 IU/y, from the acute disease accounted for influenza, tonsillitis, acute pneumonia, acute respiratory infections;
  • size and movement of dispensary patients;
• list of examinations of disabled World War II veterans;
• list of disabled workers;
• list of personal pensioners;
• list of frequently and chronically ill persons;
• list of patients with acute myocardial infarction;
• list of cancer patients;
• list of patients who are registered at tuberculosis dispensary;
• information on training physicians, health nurses. Very
important is data on the development of immune segment of the population (data on vaccination against specific diseases).

Thus, the passport section contains the information necessary for work organization of district physician. This information shall be entered in the passport systematically and analyzed quarterly.

To identify shortcomings in work of GP reports of local doctors to the population are carried out.

**Analysis of the district physician work**

The work of physician should be evaluated integrated, good performance in the program of the territorial state guarantees:

- outpatient care;
- home visits;
- day care at polyclinic;
- home care.

According to these indicators targets for each district physician are set. In addition, the activities of the district physician are evaluated with results model outcomes (RMO). Model outcomes include indicators of health, activity, as well as indicators of defects such as mortality, morbidity laborable population, primary disability, including working-age population, rehabilitation of patients with full and partial disabilities, incidence of acute intestinal infections, identification of patients with active tuberculosis, hospitalization rates, uptake on the ambulance calls to patients with chronic diseases in the hours of the clinic, hospital admissions, extrabudgetary activities (provision of paid services). Defects indicators included in RMO are identification of patients in terminal stages of cancer (IV degr.), identification of patients with visible forms of breast cancer (III-IV degr.), identification of patients in advanced stages of tuberculosis.

Evaluation of work of district physician is held by head of therapy department each quarter on the basis of accounting quality and quantity of his work, compliance with main requirements to official documents keeping, with rules of labor discipline, moral, ethical, social behavior.

District physicians are responsible both for poor quality of work and missteps and for inaction and failure to take decisions that are in the scope of their responsibilities and competence, in accordance with law.
Patient reception is an important part of the physician’s work. 12 min is assigned for one patient, i.e. 5 people per hour, and for home visiting this time equals to 30 minutes per patient. This time requires a doctor's high professional competence, ability to understand the particular clinical situation quickly, to determine therapeutic tactics, to decide on the need for hospitalization.

In many cases, patients need urgent or planned hospitalization. When hospitalization is planned, lab tests should be carried out. Having decided the question on need in patient’s hospitalization, physician gives referral to hospital, where aim of hospitalization and results of conducted laboratory and instrumental tests are indicated. Patients’s "health card" is also directed to the hospital, as it has detailed statement on the diagnosis with all laboratory and functional tests. This will lead to more rapid and precise diagnostics and earlier start of therapy, thus reducing time of staying at hospital. At the end of hospitalization patient card with discharge from a hospital is forwarded to polyclinic to physician. Such continuity in the work of the district physician allows him to organize patient care in the future.

Seriously ill patients and patients with fever are attended by physicians at home. The visit is required to be made on the day of the call. Physician decides on the need for hospitalization or treatment at home taking into account patient’s clinical condition. Primary care physician assigns therapy and decides on the working ability. Follow-up visits are made on the initiative of the patient and the doctor, depending on the state of patient.

During outpatient visits or home visits physician can diagnose or suspect infection, which requires prompt and timely diagnosis due to the fact that it represents possible risks to the patient and for others. Such infectious diseases as dysentery, acute viral hepatitis, diphtheria, influenza, SARS, AIDS, etc may occur. Therefore, primary care physician must not only know exactly clinic of these diseases, but also must be able to collect detailed epidemiological history. This will determine the right tactics and appropriate early therapy. Primary care physician should know under what infections in hospitalized patient profile department of infectious hospital and under what can be treated outside the hospital (influenza, SARS). Suspected infectious disease,
primary care physician reported by telephone to the Center for Hygiene and Epidemiology, fills emergency notification (Form 058/y) and provides information on detection of infectious disease head of therapeutic department and infectious diseases physicians. If the patient is hospitalized, it is isolated from the others, which explains the primary care physician measures prevention of disease and establishes the dynamic monitoring of contacting persons, during the incubation period.

Outpatient medical record (Form 025 – 4/y) should contain:
- Passport data with address, date of the verification of registration with a list registrar, if the patient has any category of benefits/discounts, job and career, labeling of dispensary patient.
- Detailed patient’s anamnesis, which is most important for the War veterans – military anamnesis, anamnesis at primary examination and annual addition to medical history at every development of new nosology or disability.
- Information about the annual X-ray examinations, gynecological, cancer examinations, blood pressure, vaccination.
- List of diagnosis with the introduction of all cases.
- Clearly defined records of outpatient visits (if necessary substantiating the diagnosis, indications for hospitalization, etc.).
- Medical prescriptions based on objective status and functional diagnosis.
- Results of additional tests.
- Plans for the dispensary examination of patients and annual epicrisis marked clinical examination of the effectiveness of the signed heads of departments, as well as fulfillment of individual program of rehabilitation.
- Notes on preferential discharge prescriptions with details.
- Case records (in case of in-patient treatment).
- Records should include the date of the examination and signature of doctor and signed by the head of department, according to the approved quality system.
- List of risk factors.

In the practice of district physicians are guided by the provisions of the Order № 3 of MHC from 8.01.2003. "On approval of the testimony of hospitalization in health care organizations."
Indications for hospitalization:
• grave condition and threat of deterioration;
• need for intensive care and constant supervision of medical personnel;
• complexity of the diagnostics in outpatient setting.

If a patient does not require immediate hospitalization, which is diagnostically uncertain, head of therapeutic department should carry on consultation at home. If necessary, primary care physician arranges laboratory examination at home, ECG, consultation of specialists.

Examination of disability is one of the main parts of district physician’s work. The physician assesses temporary disability and, if necessary, determines its symptoms. In practice the doctor uses resolution of MHC and Ministry of Labor and Social Protection of Belarus № 89/84 of 29 August 2011 "On Approval of the Procedure for Issuing sick leave certificates of temporary disability" as well as MHC resolution No. 61 of 12 August 2002 "On approval of the instruction by the definition of disability and instructions for determining the cause of disability."

Primary care physician should strictly follow the basic regulations of these documents, as wrong actions can lead to chronic course of the disease and premature disability. To determine severity of disability referral to MREB is issued.

"Referral to MREB" (form № 088/y) is filled after direct consultation of the patient to the local office of the MCC and the MCC decision corresponding solutions.

Examination of temporary disability
Temporary disability (TD) is the inability of the employee to perform their duties; or it is contraindications to work due to medical reasons (acute illness (trauma), crises, exacerbation, chronic diseases, examination, spa treatment and medical rehabilitation), and social factors as established by the applicable legislation (care for a sick family member, a child up to three years and a disabled child, in case of quarantine in the hospital, maternity leave).

Control over the status of work on the examination of the temporary incapacity, accuracy and validity of issuing leaflets disability in health care organizations, is, above all, the main doctors (leaders) of health organizations.
In case of issuance, processing and storage of sick leaves and certificates workers of Health Organization attract to disciplinary and administrative responsibility, and if their actions showed signs of crime, they are subject to criminal responsibility in accordance with applicable law.

**Documents certifying TD and the rules for their registration**

Main document certifying temporary disability is a sick leave (SL), which has several important functions:

- **Medical** - SL certifies that the person to whom it is issued has disease or injury, which led to the TD
- **Legal** – SL provides the right (complete or partial) to be absent at work for the period specified in the SL;
- **Financial** – SL is the basis for the payment of TD benefits
- **Statistical** – the primary statistical document for registration and study of morbidity.

The procedure for issuing and registration of sick leave (SL) and certificates of temporary disability, BH identity, pregnancy and childbirth, and supporting a temporary exemption from the civil work, study, service and other work due to medical reasons, and social factors stipulated by the current legislation → property governed by the Decree of Ministry of Health of Belarus and the Ministry of Labor and Social Protection of the Republic of Belarus from 29.08.2011 No. 89/84 "On amendments and additions to the Regulation on the procedure of issuing SL and disability certificates of temporary disability No. 52/97 of 07/09/2002."

In outpatient treatment physician gives a piece of disability after a personal examination of the patient on the day of the establishment of temporary disability (including weekends and public holidays, the day of discharge, during the holiday season - labor, social) alone within 6 days a lump sum or in installments if you want more frequent monitoring of patients.

Extension of certificate of incapacity for more than six calendar days, held on the last day of leave from work by the attending physician with department head or chairman of the MCC deputy chief medical officer, chief physician after a personal examination of the patient by these persons.

Remember that in obscure in diagnostic or treatment plan, as
well as in conflict cases, the doctor has the right to represent the patients head of department in any term TD.

Leaf extended disability lump together these doctors for a period of not more than 10 calendar days, for a total of no more than 30 calendar days.

The further extension of certificate of incapacity (over 30 days) is solved MCC Health Organization.

A general practitioner, physician district hospital (ambulance) if he works alone, has the right to grant sick leave for outpatient and inpatient treatment alone for the duration of the temporary loss of disability, but not more than 14 consecutive days in one case of temporary disability.

Extension of sick leave for up to 30 calendar days is decided after consultation with the specialist health of the parent organization, and for more than 30 days – MCC of Health Organization.

You should know!

In the treatment in terms of anonymity and confidentiality SL is issued.

We remind you that the issue of the identity BH, must be justified by medical reasons, so the medical card doctor to specify the criteria for TD, determine the optimal location for further examination and treatment of the patient (specialized or general therapeutic department of the hospital, hospital at home, day care clinics), The possibility and feasibility of outpatient treatment, issue a document certifying the TD (medical certificate or a certificate) for the required period of time and set the date for the next appearance of the patient at the reception.

To assess the patient's ability to work, timing of treatment and rehabilitation are important nature and conditions of professional activity.

For example, in similar clinical situations period of TD of patient with ARVI will be more for a builder than than e.g. for a teacher (seller, cashier) with symptoms of acute pharyngitis who require more intensive and prolonged rehabilitation, especially given the profession and the need for voice mode than working with the worst conditions. SL will be issued upon presentation of a passport or foreign identity document patient in health institution on residence place, and in the presence of departmental organizations of Health
also at the place of work, study, service, or at the place of fixing the patient for medical care.

**Important!** SL number and series, with period of TD and date of the next appearance should always be recorded in the patient's medical card.

*When a patient with TD appeals for medical help* to a healthcare organization which district he/she does not belong to, SL should be issued only with the permission of the head physician (head) or a person performing his duties.

SL is issued to:
- Workers who are citizens of the Republic of Belarus, foreign citizens and stateless persons who work in organizations, regardless forms of properties.
- People involved in business and other activities, subject to the payment of insurance contributions to the Social Security Fund of Ministry of Labour and Social Protection of the Republic of Belarus;
- Unemployed, registered in the State employment centre, at the time of public works.
- Workers, citizens of the countries of Commonwealth of Independent States, in case they have temporary disability while staying in the territory of the Republic of Belarus.

SL is issued to people with temporary disability occurred during the work (of the business and other activities) and lasted after its abandonment.

Republican health institutions (clinics, hospitals, and others) have the right to grant SL to citizens residing on the territory of the Republic of Belarus, regional health institutions – to citizens living in the region, district health institutions – to the citizens of the district, – the area, departmental – to the employees of relevant public authorities or organizations. Sick leaves of these health organizations are issued without resolution of chief doctor (head) of health institution.

**The following doctors are not eligible to issue SL:** doctors of regional consulting clinics, diagnostic centers, departments of medical rehabilitation sanatoriums, sanatoriums, medical and rehabilitation expert boards (hereinafter – MREB) and other medical advisory committees, emergency stations, stations blood transfusion, hospital admissions department, the State service of medical forensic
examinations, hygiene and epidemiology centers, balneomud clinics, balneary and urban resort, recreation, tourist centers, medical exercises clinics, nursing homes.

If you lose SL medical counseling committee (MCC) of health institution issues a duplicate. When making a duplicate of SL in the upper part there are words "Duplicate Series., N ... (lost SD)", in the section "Relief from work," one line indicates the period of TD, the signature of the attending physician, and head of the department Deputy Chief of Medical Rehabilitation and expertise. In the medical card must specify the number and series of newly issued SL.

SL are issued and renewed by state healthcare organizations regardless of affiliation and public health organizations with a special permit (license) for the TD examination and SL issuing. The license is issued with joint decision of Ministry of Healthcare and Fund of Social Protection of population of Belarus for a period of not more than five years, according to the law.

Senior students of higher medical educational institutions and health centers paramedics can be granted the right to issue SL during flu epidemic with the special order of the Ministry of Healthcare, agreed with the Fund, and with the Healthcare committee of Minsk Executive Committee, Regional departments of healthcare.

SL forms are documents with a certain degree of protection, recorded and stored as registered forms. SL forms consist of two parts: the upper part - a receipt for SL, lower – SL.

Front side of SL is filled by a physician or MCC of medical institution, medical personnel with rights to issue SL in accordance with the Regulations on the Issuance of sick leaves and certificates of temporary disability. Individual rows and columns front sick leave fill medical rehabilitation expert board (MREB), sanatorium’s doctors, rehabilitation departments of medical health centers and other healthcare organizations.

Number of forms and sick leave certificates, the date of their issuance, renewal or discharge to work recorded in primary care documentation of outpatient or inpatient (adult, child), in the book (log) Registered sick leave (Form 036 / y) and information – in the log of issued certificates and primary medical documents patient.

SL should be filled in legibly, in Belarusian or Russian with
violet, blue or black ink. In this case, erasures and not specified corrections are forbidden. Any correction should be made clear, as is written down in the fields of SL (no more than two corrections).

In the certificate of incapacity allowed common and do not cause double interpretation reduction when you make a row "MCC", "Special labels ", "Report on disability" and others, with the exception of reduced indicate the type of disability and the date of release from work and coming back to work.

"Special Notes" contains information on specific situations specified by the Regulations on the Issuance of SL and certificates of TD, as well as information, which in some cases affect duration of payment of benefits for TD.

*In case of violation of the prescribed regime* physician includes: date and nature of violations, the signature and stamp of the physician or chairman of the MCC;

*If the patient does not visit the doctor or MCC at the appointed time* – "Regime violation – was not at reception from .... – to ... (dates)". In case of recovery the line "Report on the ability to work" states "(date) capable of working" (day and month written in words). If patient has not recovered, extension of SL can be made only from the date of patient examination by the doctor. Passed patient missed days of disability-section "Release from work" is performed.

"MCC" contains examination dates and the duration of the case of temporary disability (TD) in calendar days continuous or intermittent over the past 12 months), the name, signature and personal seal Chairman MCC.

When SL is issued simultaneously for the duration of TD (e.g., during inpatient treatment) or pregnancy and childbirth in the "assertion of work" in the first and second columns in one line indicates the entire period absence from work due to temporary disability, pregnancy and childbirth, for example: 22.03-23 June. The third column indicates the name of the post and the attending physician, in the fourth and the signature of the doctor's personal seal. Lower in the third column indicates – "Managing branch", the fourth of his signature and seal, in the next line below (when applicable) in the third column "Chairman of MCC", his last name, and signature and personal seal in the fourth column.

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After medical and social expertise MREB Chairman in the line "MREB" indicates the start and end of the expertise and encrypts "MREB conclusion," which is sealed MREB.

Conclusion of patient disability shall be made only by the attending physicians on the last day of his release from work or in the event of the end of the blank certificate of incapacity, or after examination of the patient to MREB. If a disabled patient recovered, the doctor in the "Report on disability" writes "to work" and (pointing separately signed by the day and month date from which the patient has to go to work), or "to continue to hurt" if disabled patient not recovered, or "continues to care for the sick", if there is need in care and given a statement.

People who after MREB examination were set disability working group have in their resolution statement "To work" and (in words the day and month date when the patient has to go to work – the date following the date of the beginning MREB examination) "disabled of group I, II, III" and the date of disability.

If a disabled person at a survey team established in MREB invalidity without working recommendations "Report on the disability" states "Disability, disabled of group I, II" and the date on the examination on the MREB, the date of disability.

The last line of SL specifies title and last doctor that covers SL, which should contain signature and personal seal.

For verification of records in SL the following seals approved by Ministry of Healthcare are used: personal seal of attending physician, seal of healthcare organization"For SL and certificates" and official seal, MREB seal.

The line "Special Notes" includes:
- Permission of doctor to continue treatment in healthcare institution at the place of residence or work – "Permit the extension of treatment" (name of the organization of health care), signature, date and personal seal of physician.
- Permission to leave MCC in another city, country for treatment (dispanserization) – "permission to travel for medical treatment (the name of the city (country), Chairman MCC", signature, date and personal seal.
- Permission of the head physician of health organization to issue
SL to non-residents (foreigners) – "The issuance of SL permission, the chief doctor" signature, date and seal of healthcare organizations.

- Referral of patient for treatment to sanatorium, in centres of medical rehabilitation, for speleotreatment – validity period of the tickets are indicated, as well as the name of healthcare organization and at discharge - dates of arrival and departure for treatment.

- MCC decision when referring the patient to MREB – "Referred to MREB and the dates of the referral (MCC extends SL up to the proposed date of examination of the patient to MREB). Further clearance certificate of incapacity is made after receipt of the survey results of patient in MREB.

- The issuance of SL to continue (with the issuance of a certificate in the case of patient care) states – "The continuation sheet (certificate) disability" and his (her) N.

"Special Notes" may include as well:

- In case of changing the regime – a new type of regime and date of the change.

- Notes on regime violation – in the "Special Notes".

- The issuance of SL to replace the certificate on TD which occurred in a patient during their stay abroad, a duplicate of SL – in the "Release from work"; "Signature, personal seal" line should contain signature and seal of doctor.

Signature of the chief physician in all cases is authenticated by the official seal of the healthcare organization – in line "Special Notes".

Stump of Health Organization "for sick leave and certificates", placed in a special place for it – left bottom of blank certificate of incapacity:

- Closing certificate of disability (disability in the blank sheet of rough case).

- The exchange of documents certifying temporary incapacity issued abroad.

- The issuance of a duplicate certificate of disability.

- The issuance of certificate of disability to replace references.

- In the case of certificates specified patches to form sheet of disability.

Seal of MREB is placed after medical and social examination in MREB in a special place "Stamp of MREB."
One case of temporary disability SL issued in an amount necessary to permit the entire event of temporary disability. In this first piece of disability is encrypted as "Primary" (1), the other issued in the course of the primary as "Continuation" (O).

Temporary disability is considered complete if the patient was released to work or in the primary direction of the MREB he established degree of disability, and for re-examination in the time period of disability of disabled workers – group disability increased or has not made a labor recommendation.

If you close a SL and the patient experiences TD again, the case is suspended, regardless of whether it is new disease or the same TD. If you close SL and the patient falls ill again before returning to work, ou should issue a new SL, which is encrypted as a primary.

**Certificates are issued to:**
- Unemployed, registered at departments of employment and social protection.
- Military personnel, rank and files, officers of law enforcement bodies, financial investigation officers, authorities and units of Emergency in case of appeal to the territorial healthcare organization for medical assistance and determine their temporary disability.
- Discharged from work, having long-term (more than one month) TD due to disease or injury occurred within one month after discharge.
- Students of secondary, vocational and higher education institutions, graduate school, clinical residency, including the discharge of agricultural work.
- Entrepreneurs, not registered in the departments of the Fund and the premium paid to the Fund.
- Persons who take care of a sick child under 14 years with helping him on an outpatient basis, if he needs care after a 14-day period, certified by a certificate of disability.
- People to care for a sick child under 14 years, with helping him on an outpatient basis, if the decision of the MCC child needs care after a 7-day period, certified in the SL.

To care for a child under 10 years in quarantine period in preschool or secondary education institution.

For patients with basic career with additional, sheet of disability
issued in the amount required to be submitted for each workplace. In the upper right corner of the additional SL the mark "additional" is made.

*SL may be issued post factum to:*

1) people who work in shifts, appealing for medical help after hours for outpatient clinics during the ambulance copper assistance to the emergency department of the hospital or health center to the doctor. At their subsequent treatment in health care, the attending physician, establishing a temporary disability, with the approval of the MCC (MCC’s resolution is stated in "Special Notes") of SL on the basis:
   - data obtained from ambulance station (date time of call ambulance crews, diagnosis and character of medical care);
   - certificates issued by the admission room in hospitals defining the diagnosis and the nature of medical care if it turns out);
   - certificates issued by the medical assistant health center, with the date, time, treatment, diagnosis, nature rendered medical care.

   If the doctor admits a patient for work, SL is issued only for the day (hours) specified in the certificate.

2) for hospital treatment given SL doctor with head of the department on the day of discharge for the entire period of stationary treatment of the day hospital and day of discharge, inclusive.

   In case of temporary disability for more than 30 consecutive days, at the request of the patient, it may be issued and closed SL during this period, to present at the place of work for temporary disability benefits, what is the corresponding mark in the medical hospital patient. At discharge, the patient is issued to SL leaving period of treatment in a hospital as a continuation of the previous SL.

   If at the time of discharge disabled patient recovered, SL is issued by the day of discharge and is closed. If necessary, due to the remoteness of the medical facility from the residence patient SL separately extended by one day.

   If at the time of discharge disabled patient has not recovered, SL is extended for a period of 3 days, and during prolonged temporary disability in the absence of evidence to the part of the medical examination, SL may be renewed once for up to 10 calendar days. Further extension and closing SL is making by converges Health Organization, which will continue treatment of patient.
If, after discharge from the hospital with the patient's appearance outpatient physician finds his rehabilitation before of the period of leave from work by hospital, he sends the patient to the medical-advisory committee (MCC) for the issue of decide competitiveness. If MCC in its resolution recognizes a patient for work, SL is closes. In line of SL "Report on disability" MCC makes the appropriate corrections.

Remember!

In the day care of outpatient clinics SL is issued and renewed as in outpatient treatment, and in hospital inpatient facilities – as in inpatient treatment.

Cardiological and oncological patients diagnosed with sick leave may be granted and renewed in health organizations at the place of service.

During quarantine SL is issued by infectionist, and in his absence – by another doctor, for the entire period detunings of the work, if the employee has been in contact with infected patients and was off from work by decision of sanitary-epidemiological service. "Type of disability" line states "Quarantine" and the line "Regime" states "Home".

If possible (at the conclusion of the MCC and the agreement with the sanitary-epidemiological service) temporary employment for a period of quarantine SL is issued.

When the direction of the patient for a consultation or treatment to another city of the Republic of Belarus, for that there is the main conclusion of the expert (the deputy chief physician of the medical work, head of the organization of health care), or abroad if the direction of the Ministry of Health for consultation (treatment), MCC Health Organization issues a SL at the required time, but not more than 10 calendar days. MCC on the line "Special Notes" indicates "Referral to the consultation (treatment) (specify the name of the organization of health care and address)." In SL "Release from work" in the field "From date" the date of start of temporary disability is stated. Renewal and closure of SL is made by MCC on return of the patient after treatment based on the certificate (case record) of consultation (treatment).

Patients who have a temporary disability occurred during the sanatorium-resort treatment, are issued SL by the attending physician
with the head of the department, and in his absence – the deputy head
doctor or health center with the chief doctor.

Persons sent in accordance with legislation on the work after
graduation, graduate studies, clinical studies, SL is issued on the day
appointed for the coming back to work.

In the case of alcohol, drug or toxic coma, and in the treatment
of chronic alcoholism (addiction, substance abuse), not complicated
by other disorders and diseases, the patient is given SL, which in the
"Special Notes" states "The disease is associated with use of alcohol,
drugs or toxic substances".

Complications in the treatment of alcoholism, drug abuse, toxins
addiction SL is issued to a patient without this mark for the entire
period of temporary disability.

In carrying out cosmetic surgery or plastic surgery, starvation
treatment SL shall not be issued except for their conduct on medical
occurrence in a patient or complications.

SL for sanatorium treatment is issued for:
- ill and undergoing radiation sickness caused by consequences
  of the Chernobyl disaster;
- the disabled, for which a causal relationship with the disabled
  of the Chernobyl disaster;
- tuberculosis patients for treatment in a tuberculosis sanatorium.

SL in these cases is issued by MCC (in its absence – the
attending physician with the chief physician) of health organization,
refer the patient to a sanatorium treatment on presentation of
vouchers, to attending to a sanatorium: when you make a SL in the
line "Type of disability "write "Sanatorium treatment"in the"Regime"-
"Sanatorium"in the"Special Notes" – the name of the nursing home, 
start date, and final of sanatorium vouchers.

SL is closed by MCC of health organization places of service
patient with the deadline of its actual stay in the sanatorium and time
spent on travel. In case of arrival of the patient in a sanatorium late or
early it out MCC Health.

During outpatient medical rehabilitation for patients with
temporary disability SL is issued as in outpatient treatment. After
recovery rehabilitation of the patient is carried out in their free time.
Patients with temporary disability, sent to hospitals or medical rehabilitation centers, SL is issued by hospital admission room.

When sending patients for medical speleotherapy SL is closed and issued by MCC of health organization at the place of residence of the patient. SL issued for the entire period speleotherapy against time to get to the place of treatment. The line "Type of disability" states "Disease", "Regime" – "Stationary".

SL for carer’s is issued if lack of care threatens health of the patient, and hospitalization is not possible due to the severity of the patient and non-transportable or temporary lack of places in the hospital. SL for carer’s issued from date on which there is a need for additional (individual) care. If a person busy caring for the sick, do not need to have leave from work (weekends, holidays, during the holiday season), SL issued from the day when the need arises.

SL for carer’s to patients in need may be given alternately to different carers.

One should know that SL for carer’s of patients is permanent patient care or off-site residence of a person caring for a sick, Health Organization issued at the location of the patient with the permission of the head physician.

SL for carers of patients over 14 is issued by the physician only in case of outpatient treatment to a family member or other person caring for a patient, for up to 3 days. In exceptional cases, by decision of MCC, considering severity of the patient SL may be extended up to 7 calendar days.

**Major medical indications for referral to MREB are:**
- Long-term, not less than 4 months of continuous TD.
- TD duration of five months with breaks for the same or related diseases.
- Signs of disability, regardless the period of TD.

**The concept of disability**
- Persistent disabilities – a condition always disruption of the body caused by disease or injury, is irreversible or partially reversible, although long-term complex treatment, and forced the employee to cease all work in a production environment, or go to sheltered employment, often with leads to loss of primary occupation, lower skills or a significant reduction in the volume of work or norms.
Disabled person is a person who due to limited life due to physical or mental disability, in need of social protection and assistance.

The criterion is to limit the life of disability, caused by diseases (injuries, defects), leading to social dezadaptation due to a limitation or inability to learning, communication, guidance, control your behavior, movement, self-service, participation in the labor force.

Criteria for establishing the disability:

The basis for determining I degree of disability is dependent on the continued unregulated care parties owing to the pronounced disability due to illness, the effects of trauma, severe combined anatomical defects and leads to social failure.

I degree of disability defined as in diseases with absolutely unfavorable prognosis regarding life for the near future independent of the degree of disability at the time of examination at MREB.

The basis for determining II degree of disability is significantly expressed disability, diseases caused by consequences of injuries, combined anatomical defects and leading to social failure.

Significant disability appear: in pathology of visceral, nervous, endocrine, cardiovascular, musculoskeletal, visual system when the violation of their functions reach far-severe (FC 3), the combination of moderate dysfunctions (FC 2) two or more systems, create syndrome giving mutual aggravation within the same category of vitality and lead to a significant limitation of it, in the expression of mental disorders.

II degree of disability is defined as independent of the severity of disability under questionable (despite of treatment and rehabilitation) labor forecast and contraindications to labor in connection with the possible health disorders.

The basis for determining III degree of disability is moderate restriction of life due to diseases, traumas and severe anatomical defects and leads to social failure.

- To limit the scope of professional activities in the trade for 4 or more digits (over 25%) job title specialist or the level of control of the head.

- Time limit of labor time (to work only part-time).

- The need to provide a workplace adapted auxiliary mechanisms and other means. Moderate restrictions of vital activity (FC-2) arise: the pathology of visceral, nervous, endocrine, musculoskeletal,
cardiovascular, visual systems, psychic activity, when the malfunction of any of them reaches moderate or mild dysfunction (FC-l) more of the one of systems that create mutual aggravation syndrome within the same category of life, resulting in moderate to limit it, with mild abuse professional but significant function (FC -l), the guiding it impossible to continue the work what reduce his qualifications, as well as a pronounced hearing loss (deafness III-IV degree).

**Structure, functions, organization of MREB**

Medical rehabilitation expert board (MREB) conducts medical and social expertise of disability based on a comprehensive assessment of health status, the degree of functional disability and disabilities including mental status and motivations of patient, social and environmental professional factors. MREB under circumstances (medical indications) establishes the degree of restriction of life of patients, the cause – the time of disability, determines the extent of the loss of professional disability (as a percentage), work out individual rehabilitation program (IRP). Issuance (extension) of SL for longer periods of temporary disability is made by MCC, based on the opinion of the MREB in case of lack patient disability and deciding to aftercare rehabilitation.

**Main tasks of MREB:**

- Conducting quality medical and social assessment of patients with determines degree of patients’ disability;
- Determination of the cause (work accident, occupational disease, wounded at the front, etc.) and time of onset of disability to decide on a pension or compensation to family members of the deceased invalid;
- The percentage of disability in occupational diseases and for occupational injuries;
- Prevention of disability, making individual rehabilitation programs with the volume of the order and sequence of the medical, social and professional rehabilitation, control of implementation;
- Analysis of performance on a quarterly basis to report to the higher standing MREB;
- Conferences, meetings, seminars on the prevention of disability, medical and social assessment and rehabilitation in various healthcare organisations;
- Advice to healthcare organisations and control of medical and social expertise of temporary disability and medical rehabilitation.

*Remember*, identifying the signs of disability is the basis for sending the patient to MREB at any period of TD!

Survey citizens in MREB held at home (on constant residence) or attaching them to the healthcare organisations toward MCC direction of healthcare organisations. MREB hold session in the healthcare organisations on a scheduled basis, and go to your home or in a hospital, if the patient can not come to the meeting for health reasons.

MREB conducts examinations of patients, study medical (Form 088/u) and other documents as required and after discussing make decision.

With a favorable clinical and labor prognosis of patients with 4 months of continuous TD are directed to MREB for sanctions to further extend the SL up to 4 months in addition to the recovery ability to work.

If MREB recognizes sick for work, MCC extends SL for the duration of the examination, including the day of its completion, and forward the patient to work the next day.

If MREB does not reveal the basis of disability in the patient and recommends further treatment, the MCC puts down in the "MCC" field of SL "Permit extension of TD", which is to be signed and sealed by the chairman of MCC.

Disabled workers with labor recommendation of MREB, in case they have illness or injury TD, SL is given in accordance with regulations. When issuing a SL in the "Special Notes" must be stated the following: "The disease (trauma) is not linked or connected with the cause of disability".

Degree I of disability is set for two years, II and III degree – for one year. Disabled in connection with the accident at the Chernobyl nuclear power plant persons have done next examination in five years. Disabled in connection with participation in military operations in Afghanistan and in other states in the primary examination of disability is set to 5 years, with next examination – on the same basis, ie depending on the degree of disability.

Reexamine of persons with disabilities is held after the expiration of the period of constant disability, which ends the first day
of the month following the one when disability was diagnosed.

There is a detailed list of anatomical defects, which constantly assigned a degree of disability.

For children up to 18 years if there are medical grounds MREB determines the degree of disability and loss of health of the child. The degree and cause of disability are not set, and conclusion is made – "disabled child" and establishes the degree of loss of health. A month before achievement 18 years of child healthcare organisation admit "disabled child" at MREB, where he, under circumstances which establish is one of the disability and its cause – "disabled in childhood" ("disabled in childhood in connection with the Chernobyl Nuclear Power Plant disaster").

MREB decision shall enter into force after the child turns 18. Prolonging disability if there are grounds to persons who are 18 years old, based on the conclusions of MREB interdepartmental expert advice about disease (injury) causation with the accident at the Chernobyl nuclear power plant.

Lesson № 2. Acute respiratory infections (influenza and other viral respiratory infections), acute tonsillitis (angina). Diagnosis, treatment, medical tactics, medical-social examination. Dispanserization in acute tonsillitis (angina)

Acute respiratory disease – a disease that is defined by the sudden appearance of at least one of the four respiratory symptoms (cough, sore throat, shortness of breath, acute rhinitis) in the presence of clinical evidence that the disease is caused by infection (may show a increased temperature or without fever).

Influenza – an acute infectious disease with a droplet mechanism of transmission, occurring with symptoms of intoxication, high fever (above 38°C), sore throat, and, often, the phenomena of tracheitis.

The spectrum of clinical manifestations of disease caused by influenza viruses, including pandemic influenza A (H1N1), includes light, moderate and severe forms of the disease. Symptoms of pandemic influenza A (H1N1), in general, has no significant clinical differences from the normal seasonal flu. However, it had a
participation in the process of all age groups, including young people, more frequent complications of the lungs, especially in those related to the risk group.

**Risk group of patients with influenza**
1. Patients with immunodeficiency (congenital immunodeficiency, HIV infection or AIDS).
2. Patients with chronic obstructive pulmonary disease and asthma.
3. Patients with cachexia.
4. Patients with decompensated or subcompensated diabetes as well as obesity.
5. Patients with chronic cardiovascular disease (except isolated hypertension).
6. Pregnant.
7. Children up to two years.

**Clinical manifestations and complications of influenza**
Clinical manifestations of uncomplicated influenza:
- acute onset, fever (38-39°C), weakness, headache, myalgia, ocular symptoms (photophobia, tearing, pain in the eyeballs), dry cough, runny nose, stomach pain, vomiting and diarrhea in adults with pandemic A grippe (H1N1).

For most patients, the disease ends in complete recovery without complications within 7-10 days, although cough and weakness can persist for two weeks or more.

Complications of the respiratory organs
- In mild flu inflammation limited to the mucosa of the nose, pharynx, larynx and trachea. In moderate flu affects the trachea, bronchi, leading to complications from the respiratory tract in the form of acute laringotracheobronchitis, in severe influenza virus develops pneumonia with acute respiratory distress syndrome (ARDS hereinafter), the appearance of multiple organ failure. A serious complication of severe influenza is the development of encephalopathy.

Primary viral (influenza) pneumonia develops in the first 24-72 hours of onset (1-3 day runs as toxic acute hemorrhagic pulmonary edema, with 4-6 days as polysegmental often bilateral pneumonia).

**Risk groups:** the elderly, pregnant women and children, patients
with a history of premorbid background (chronic lung disease, cardiovascular, diabetes, overweight). However, in some patients the risk factors are not detected.

**Clinic:** acute onset with chills, a rapid rise in body temperature, increase of intoxication and breathlessness. The cough is usually unproductive, sometimes with scanty sputum streaked with blood, the appearance of dyspnea, cyanosis increases. Auscultatory pattern changes with the progression of the disease: in the early stages - a crackling, scattered dry rales, wheezing then extend to all parts of the lungs, breathing weakened, in the terminal stages – wheezing little heeded, breathing is much weaker in patients with severe tachycpnea.

**Characterized by:** leukopenia peripheral blood, sputum basic cellular elements – mononuclear cells, complications of primary viral pneumonia – ARDS, acute renal failure, disseminated intravascular coagulation.

Severity due to the development of acute respiratory failure (number of respiratory movements and more than 30 in a moment, in the act of breathing involved supporting muscles of the chest and abdomen, oxygen saturation below 90%), the growth of the intoxication syndrome and multiple organ failure. The most frequent and serious complication – acute hemorrhagic pulmonary edema and cerebral edema.

**Radiographic features:** in the early stages has been increasing pulmonary pattern without signs of focal infiltrative changes characteristic of bacterial pneumonia in the progression of the disease for 3-4 days can be seen on the radiograph bilateral confluent infiltrates.

**Morphologically:** hyperemia and edema of the lung tissue, pockets of red seal, the mucous membrane of the trachea and bronchi, bright red, covered with semi hemorrhagic mucus. A microscopic examination – desquamation of epithelial cells of the mucous membrane of the trachea and bronchi, and swelling of loose connective tissue of the submucosa, in the lumen of the bronchi – hemorrhagic exudate, with further progression of the disease – reduced lung airiness by distelektazov and accumulation in the lumen of the alveoli amorphous mass of protein, red blood cells and desquamated alveolocytes ("flu-like cells") that ends with the formation of hyaline membranes.
Example of diagnosis:
Influenza. Primary viral pneumonia complicated by acute respiratory distress syndrome, hemorrhagic pulmonary edema. Multiple organ dysfunction. III degree respiratory failure (specify type of respiratory support).

Secondary (later) a bacterial or viral and bacterial pneumonia. The interval between the first symptoms and signs of respiratory involvement in the lung parenchyma is 4-6 days. The main pathogens – Str. pneumonieae, Staph. aureus (50%), H. influenza.

Clinic of such patients have a progressive course of influenza, the body temperature returns to normal, remaining signs of intoxication, to 4-6 days cough increased, becomes painful, scanty sputum, sometimes mixed with blood, shortness of breath may be pleural pain. When auscultation listened scattered like dry and moist rales. Pronounced tachycardia, cyanosis.

Changes in peripheral blood can be multi-directional and have no diagnostic value. In the sputum of patients show as viruses (polymerase chain reaction – PCR) and bacteria. Cellular composition of sputum – polinuklearnye leukocytes with more bacteria.

Rise due to the severity of respiratory failure, multiple organ failure connection.

Radiographic features of the data: the absence of signs of focal infiltrative changes in the early stages, and the emergence of consolidating pneumonic lesions to 4-6th day of disease.

Morphologically: with viral and bacterial pneumonia increased hemorrhagic nature of inflammation – lung tissue and mucous membrane of the trachea and bronchi, the bright red color in the lumina of the alveoli – a large number of fresh and hemolyzed red blood cells microscopically bronchi filled with desquamated epithelial cells.

Example of diagnosis:
Influenza. Viral and bacterial pneumonia complicated by ARDS. Multiple organ failure. Respiratory failure 3 degree (specify type of respiratory support).

Secondary bacterial pneumonia develops in patients later than the fifth day from the beginning of the disease, with the regression against the symptoms of influenza joins secondary bacterial microflora, usually Str. pneumonieae, Staph. aureus, H.influenza, Kl. pneumoniae.
Clinic: increases cough, which becomes painful, there is pain in the chest associated with the act of breathing, resurged general intoxication symptoms (increased body temperature – the second wave of fever, sweating, decreased appetite). Becomes purulent sputum. Physical examination revealed signs of local parenchymal process. Staphylococcal pneumonia runs particularly difficult. Clinical experience shows that this kind of pneumonia is often complicated by the development of degradation with the formation of lung abscess.

Characterized by leukocytosis or leucopenia in peripheral blood, sputum Gram stain reveals a large number of bacteria, and polymorphonuclear leukocytes.

Severity due to the nature and severity of developing complications: local (external respiration) and system (from other organs and systems).

Radiographically: infiltrative changes characteristic of bacterial pneumonia, an inhomogeneous structure, fuzzy outer contours, segmental or polysegmental length, up to abscess formation.

Morphologically: focal or confluent infiltrates, microscopic features of which are due to respiratory pathogen.

Acute laryngotracheitis with stenosis of larynx is also the name of a competing croup syndrome and implies emerged quickly (within hours or days), shortness of breath associated with the narrowing of the airways. Characterized by three major symptoms:
- Changes in voice;
- Rude "barking cough";
- Sonorous breathing difficulties (stenotic breathing).

There are four degrees of stenosis of the larynx, with the leading criterion for assessing the severity of stenosis is respiratory failure.

Example wording of diagnosis:
Influenza, complicated with acute laringotracheobronchitis and bronhospastic syndrome, prolonged duration, respiratory failure

Extrapulmonary complications of influenza:
- Cardiac complications – myocarditis, pericarditis in previously healthy young patients or cardiac arrhythmias and congestive heart failure in patients with chronic cardiac disease;
- Acute toxic encephalopathy (often referred to as encephalitis less
valid because of the lack of influenza virus tropism to neurocytes and glia) – is very rare, only in children is the result of microcirculation in the brain to form multiple ischemic and hemorrhagic lesions. Characterized by a high mortality rate and the formation of severe neurological deficits in survivors;

**Landry-Guillain-Barre syndrome** – a form of acute inflammatory poliradikuloneuropatia manifested flaccid paresis, sensory symptoms, autonomic disorders. At the heart complication is the development of autoimmunity, although corticosteroids therapy usually does not lead to stabilization and rapid regression of symptoms;

**Reye's syndrome** – rarely encountered pathology of the liver and central nervous system with a high mortality rate. Reye's syndrome most often occurs in children and adolescents in the background of a number of viral infections, including influenza, treatment with aspirin. In this regard, for the relief of fever in this age group should not be given medications containing aspirin;

**myositis** relatively more common in children with influenza type B, rare cases of rhabdomyolysis can lead to acute renal failure;

**acute otitis media, sinusitis, bronchitis**, occurring against the backdrop of a portable acute respiratory infection. Etiological factors are not only viruses, but many bacterial agents – Streptococcus pneumoniae, Haemophilus influenzae, β-hemolytic streptococcus gr. A, Moraxella katarralis, staphylococcus, chlamydia and mycoplasma;

**exacerbation or decompensation of any underlying chronic disease.**

Danger signs of more severe disease.
- Shortness of breath during physical activity or at rest;
- Shortness of breath;
- Cyanosis;
- Bloody or colored sputum
- Pain in the chest;
- Altered mental status, high body temperature for more than 3 days, bad stoped standard doses of antipyretic drugs;
- Low blood pressure.
Clinical features of pandemic influenza A (H1N1):

- appearance of pain on swallowing, presence dyspeptic symptoms in 10-12% of patients;
- more frequent development of viral pneumonia with the appearance of respiratory failure on average 5-6 days before the date of the occurrence of the first clinical signs of disease;
- development of viral pneumonia in patients who are overweight (body mass index of 30 or more);
- the rapid development of ARDS, which is preceded by severe shortness of breath and persistent, difficult to treat cough and hemoptysis, and in some cases, the development of multiple organ failure;
- in laboratory research in the general analysis of blood often prevails leukopenia, leukocytosis, and less common neutrophilia;
- changes in the X-ray examination of the lungs appear later in the development of clinical signs of respiratory distress, often interpreted as "stagnation in the small circle", "increased vascular pattern", "pulmonary fibrosis";
- tendency to hypercoagulability of blood that determines the need for coagulation monitoring with compulsory prevention of thrombosis in patients in severe cases;
- development of severe disease in patients younger and middle-aged.

Provision of care for patients with influenza and acute respiratory infections

Care for patient with risk from mild to moderate with uncomplicated influenza carried out in an outpatient setting, and includes:

- home regime, drinking a lot of fluid, nutrition;
- paracetamol and other antipyretics in standard therapeutic doses (those under 18 years of drugs, including acetylsalicylic acid, not appointed);
- symptomatic treatment of rhinitis, pharyngitis, tracheitis.

Indications for hospitalization of adults are:
- severe flu flow: severe weakness, lethargy, impaired consciousness, convulsions, dyspnea (respiratory rate over 20 per
minute for those over 13 years), dehydration, inability to drink (for example, because of repeated vomiting), fever above 38.5°C not stopped the usual dose of fever-reducing medicines, stable persistent cough accompanied by shortness of breath, coughing up blood;

suspected pneumonia or other complications;

patients at risk, including pregnant women, in the absence of the effect of medical care provided for 3 days;

the signs of involvement in the central nervous system;

When treating any flu in patients who are at risk in the outpatient setting (if the decision was made by the attending physician or patient refusal of admission), it must be assigned antiviral therapy within 48 hours of the disease and, if indicated, antibiotic therapy. Treatment of complications of influenza, bronchitis, otitis media, sinusitis, according to local clinical protocols.

Principles of causal treatment of influenza and its complications

The basis for the treatment of severe and moderate forms of influenza, caused or allegedly caused by the pandemic strain of H1N1, all cases occurring in contingent risk of viral or bacterial and viral pneumonia

all severe forms of the disease, is an antiviral drug oseltamivir or zanamivir in the relevant age doses (Tabl. 1). It is essential that the most effective antiviral drugs noted in the first 48-72 hours after the onset of the disease, but in severe and complicated course of their useful purpose in any stage of the onset of the disease. Oseltamivir dose may be increased to 150 mg two times per day and extended to 10 days. Increasing the dose and duration of therapy is especially warranted in patients who are in intensive care and receiving respiratory support, as well as in patients with a body weight of over 110 kg, especially the introduction of the drug through the probe specified in section nutritional support.

It is antiviral drugs are the basis of causal treatment of influenza, especially in the early stages of the disease and its complications.

Antibacterial drugs used in the treatment of pneumonia, prescribed depending on the severity and timing of the disease and are used in conjunction with this antiviral therapy. Their role is to increase with the terms of the disease.
Opening arrangements may be protected designation aminopenicillins cephalosporins or 2-3rd generation, in more severe cases, protected cephalosporin or carbapenem non-antipseudomonal effect, combined with a macrolide or respiratory fluoroquinololones.

When you are in the intensive care unit, patients who are on a mode of mechanical ventilation for more than 5 days, antibiotic therapy is assigned with the sensitivity of nosocomial flora: antibiotics with antipseudomonal activity (especially carbapenems) in combination with respiratory fluoroquinololones or macrolides or aminoglycosides (gain antipseudomonal effect). In cases of possible accession of methicillin-resistant Staphylococcus aureus, recommended the inclusion in the scheme of therapy glycopeptides oxazolidinones (preferred because of its good tissue penetration).

**Category of patients with acute respiratory viral infection and flu, to be appointed by the antiviral drugs oseltamivir and zanamivir:**

- pregnant;
- patients with severe and complicated influenza;
- patients admitted to hospital with symptoms of involvement in the lower respiratory tract (tachypnea, dyspnea: reduced blood oxygen saturation), regardless of the length of the disease;
- children younger than 5 years;
- persons with severe chronic diseases (especially with COPD, asthma, diabetes) and/or immunodeficiency (HIV immunosuppressive therapy, post-splenectomy);
- obese patients;
- patients undergoing outpatient treatment, with persistent fever 38.5°C and above for one or two days or more, cough and other symptoms of tracheitis.

Drug oseltamivir or zanamivir can be used for post-exposure prophylaxis of health care workers for medical care for patients with pandemic influenza A H1N1.

Oseltamivir is taken two times a day for treatment up to 5 days. However, in severe cases, adults often need a higher dose – 150 mg 2 times a day, and a long course (7-10 days).

In the absence of significant clinical symptoms and complications, your doctor may refrain from administration to a patient etiotropic drugs.
Table 1 – Doses of antiviral drugs used for the prevention and treatment of influenza

<table>
<thead>
<tr>
<th>Medication (name, dose, dosage forms)</th>
<th>The destination</th>
<th>Doses for different age groups</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1-12 years</td>
<td>13-64 years</td>
</tr>
<tr>
<td>Ozeltamivir*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>prevention</td>
<td>Not used</td>
<td>75 mg/day</td>
</tr>
<tr>
<td>Treatment</td>
<td>dose varies depending on the weight **</td>
<td>75 mg 2 times a day</td>
</tr>
<tr>
<td>Zanamivir (in blister for inhalation)</td>
<td>prevention</td>
<td>10 mg 1 times a day until 7 years</td>
</tr>
<tr>
<td>Treatment</td>
<td>10 mg 2 times a day until 7 years</td>
<td>10 mg 2 times a day</td>
</tr>
</tbody>
</table>

* Reduce the dose of oseltamivir in patients with creatinine clearance less than 30 ml/min.
** Weight <15 kg – 30 mg 2 times a day,.> 15-23 kg – 45 mg 2 times a day,.>23-40 kg – 60 mg 2 times a day,.> 40 kg – 75 mg 2 times a day.

Indications for antibiotics is the severity of the patient and the presence of bacterial complications. If there are indications for antibiotics in ambulatory practice is starting amoxicillin or amoxicillin/klavulonat. In the case of allergy in the patient to ß-lactam antibiotics, or the presence of clinical manifestations in favor of atypical microorganisms justified macrolide antibiotics or respiratory fluoroquinolones.

Algorithm of antibiotic treatment

Outpatient practice

If there are indications for antibiotic therapy starting antibiotics are beta-lactam antibiotics: amoxicillin inside 0.5-1 g three times daily, or amoxicillin / clavulanate into 0.625 g 3 times a day or 1 g, 2 times a day, or intramuscular ceftriaxone 1.0-2.0 g 2 times a day in combination with macrolide antibiotics: clarithromycin into 0.5 g 2 times a day, or azithromycin into the 0.25-0.5 g 1 per day (course dose of 1.5 g), or respiratory fluoroquinolones, levofloxacin into 0.5-0.75 g 1-2 times a day, moxifloxacin into 0.4 1 g once a day. In the
case of allergy patient to beta lactam antibiotics are prescribed macrolide antibiotics clarithromycin into 0.5 g 2 times a day, or azithromycin into 0.2-0.5 g 1 per day (course dose of 1.5 g), or respiratory fluoroquinolones – levofloxacin inside 0.5-0.75 g 1-2 times a day, and moxifloxacin – into 0.4 1 g once a day.

Antipyretic therapy provides for the appointment of antipyretics in acute respiratory infection. Typically antipyretic drugs for acute respiratory infection using:
- adults – at temperatures above >38,5°C;
- malignant hyperthermia in violation of microcirculation;
- the presence of severe subjective symptoms associated with fever (headache and muscle pain).

Immunomodulating therapy

The absolute majority of patients with severe pneumonia caused by influenza A H1N1 in the general analysis of blood revealed leukopenia (<4.0 h109 / l) and / or lymphopenia (absolute lymphocyte count <1.2 h109 / l), indicating the presence of secondary immunodeficiency reducing the effectiveness of the anti-viral and antibiotic therapy, and requires a corresponding correction.

In the presence of leukopenia (<4.0 h109 / l) and lymphopenia (<1.2 h109 / l) is assigned to intravenous immunoglobulin (venoimmun rate of 200mg/kg, an average of 15 g/day, 1-2 introductions), and 2 ml cycloferon in / muscle or intravenously on days 1, 2, 4, 6, 8, 10, 12, 14, 16, 18 day (total of 10 injections). In the presence of an isolated lymphopenia – cycloferon by the above scheme.

Venoimmun an immunologically active protein fraction Ig G. Contains antibodies against a variety of pathogens – bacteria and viruses, including herpes, influenza, Staphylococcus aureus, Escherichia coli, and other pneumococcal infections. The number of transfusions, intravenous immunoglobulin depends on the severity of the process. In severe bacterial and viral infections that are difficult to care, amid persistent leukopenia venoimmuna permissible dose increase to 0.4 g/kg or increasing the dose frequency to 3-4 times.

Cycloferon an inducer of endogenous interferon, which defines a wide range of its antiviral, antibacterial and anti-inflammatory immunomodulatory activity. It activates the bone marrow stem cells, stimulating the formation of granulocytes, and activates T cells and
natural killer cells, normalizes the balance between subpopulations of T-helper and T-suppressor cells.

When transferring patients who had leukopenia and/or lymphopenia and suffered respiratory failure 2-3 degrees of OITR in pulmonology department to continue the work started by the introduction tskloferona diagram above.

**Corticosteroids**

Appointment of glucocorticosteroids is conducted only in hospital with a diagnosis of severe acute respiratory distress syndrome.

**Angina** (ICD-10 – acute tonsillitis, J03) – an acute infectious disease of mainly streptococcal etiology, characterized by symptoms of intoxication, fever, inflammatory changes in the lymphoid formations throat ring / more often in the tonsils/and regional lymphatic nodes. Although the term "angina" (from Latin ango – Compress, choke) is not exact (acute inflammation of the tonsils is rarely accompanied by suffocation), it is widely distributed to health professionals and the public and can be used on an equal footing with the more accurate term – "acute tonsillitis." For inflammatory tissue oropharynx (mostly almonds) encountered in many infectious (scarlet fever, diphtheria, infectious mononucleosis, etc.) and non-infectious (leukemia, agranulocytosis, etc.) diseases should use the term "tonsillitis", or rather "secondary tonsillitis."

**Epidemiology.** The prevalence of angina is highest in countries with low levels of socio-economic development and poor material conditions. Urban population suffers significantly more rural in one and the same climatic zone, due to the greater density of the urbanized population.

The incidence of angina during the year is subject to large fluctuations, it is minimal – in the summer, the maximum – in the autumn-winter period (October – January). Increasing incidence has an impact, as a drop in temperature and increase the humidity. Increased incidence in the fall and winter due to the increasing number of patients with acute respiratory illness by coughing and sneezing much more intense isolated streptococci into the environment, creating favorable conditions for the rapid contamination of surrounding people. Angina is a certain contagious. Sore outbreaks occur most often in large groups
(children, adolescents, production, etc.). This is due to the fact that streptococci mainly infected people in the immediate vicinity of the source of infection. These conditions are often created by the presence of people in organized groups, when using public transport.

Infection of angina occurs mainly by airborne droplets. In cold weather and high humidity, the conditions under which lasts longer aerosol droplet phase in which bacteria are virulent state. In accommodation, the sources of infection are contaminated with germs bedding and household items. Infection occurs during physical activity (lifting, dressing, making the bed, etc.). However, in these conditions, bacteria rapidly lose their virulence properties and can cause clinical forms of disease. Infection with angina can occur when using an infected tableware, ie in violation of sanitary rules, the conditions for the spread of streptococcal nutritional means. It should be noted that the $\beta$-hemolytic streptococci, getting on the meat, dairy and containing sugar, the products may actively multiply and create the conditions to infect large numbers of people, united by a single point of supply.

The source of infection are patients manifest forms of streptococcal disease and healthy carriers of germs. The greatest danger is posed by the epidemic patients with angina. The frequency of healthy carriers of group A streptococci is characterized by large fluctuations. It rises sharply shortly after the update, or groups, in epidemic outbreaks of streptococcal disease and decreases after the identification and isolation of patients. Of healthy carriers of strep is 1-2 months to 1 year or more. However, the role of healthy carriers of $\beta$-hemolytic streptococci in the epidemic process is much less than in patients open forms of streptococcal disease – sore throat and scarlet fever.

**Etiology.** The causative agent of angina may be bacteria, viruses, spirochetes, fungi. In most cases (31-80%), sore throat caused $\beta$-hemolytic streptococcus group A (BHSA) or other groups, at least - staphylococci, or both. In organized groups bacterial pathogens angina can be pneumococcus, meningococcus, influenza bacillus, typhoid, Klebsiella. Very rarely can cause a sore throat anthrax bacillus, anaerobes like Clostridium. The reason most often viral sore throats are adenoviruses (1-9 types), enterovirus Koxakie, herpes virus, and spirochetal – oral spirochete in conjunction with the fusiform bacteria.

**Pathogenesis.** Infection atriums are lymphoid ring formation
Pirogov. Contained in the hull β-hemolytic streptococcus lipoteichoic acid has an affinity to the epithelium of the oropharynx and lymphoid apparatus thus provides fixation of these microorganisms in the tonsils or other clusters of lymphoid tissue.

Streptococcal M protein and streptococcal toxins inhibit the ability of phagocytes to engulf and digest bacteria, which promotes long-streptococcal bacteremia and antigenemia.

Reproduction of streptococci in the body is accompanied by production of toxins that cause inflammation of tissues tonsils. With the penetration of streptococci and their metabolic products on lymphatic routes in lymph nodes occurs regional lymphadenitis.

With a favorable course of the disease is limited to the spread of microorganisms lymphoid formations oropharynx and regional lymph nodes. When failure of the barrier function of the tissues surrounding the tonsils, streptococci can invade okolomindalikovuyu fiber and cause its inflammation (peritonzillit, peritonsillar abscess).

In the pathogenesis of angina are important both exogenous and endogenous factors. Among the exogenous factors determine paramount infectious agent (its virulence) penetrating through the epithelium of the inner surface of the tonsils, their gaps. In the mechanism of angina are also important such harmful environmental factors like dust, pollution, changes in air temperature. Plays an important role general and local hypothermia. A certain role in the pathogenesis of angina is given the power factor – monotonous protein foods deficient in vitamins C and B predisposes to the development of angina. Contributing factor may be a minor injury tonsils foreign body (hair toothbrush, small fish bone), spatula in determining the contents of the lacunae of tonsils. A very important role in the pathogenesis of angina is a violation of nasal breathing. Sometimes, surgery of the nose is a provocative moment for its occurrence.

**Clinical classification of angina** (By Y. Liashenko, 1985)

*Etiology:*
- Streptococcal;
- Strepto-staphylococcal;
- Staphylococcal;
- Fungal;
- Fuzospirohetal symbiosis etc.
Localization of the pathological process:
- Tonsils;
- The side bolsters of the throat;
- Nasopharyngeal tonsil;
- Lingual tonsil;
- Lymphoid formations posterior pharyngeal wall;
- Lymphoid formations larynx.

By the nature of inflammatory changes:
- Catarrhal;
- Follicular;
- Lacunar;
- Necrotic (necrotizing, fusospirochetal).

By severity:
- Easy;
- Moderate;
- Heavy.

By form:
- Primary;
- Repeat;
- Secondary.

By the presence of complications:
- Uncomplicated;
- Complicated.

In addition, as noted above, acute tonsillitis is divided into primary and secondary. Primary acute tonsillitis - ostrovospalitelnye disease, the clinical picture is that the leading element are symptoms of tonsillitis. Acute tonsillitis is a secondary lesion in the tonsils of acute infectious diseases (mainly in diphtheria, scarlet fever, tularemia, typhoid fever), and diseases of the blood system (primarily for agranulocytosis, leukemia).

The clinical picture

The incubation period for a. tonsillitis is 1-2 days. Acute onset. Almost within a day developing a complete picture of the disease – fever, fatigue, headache, aching joints, pain in the throat when swallowing. Often in the onset of the disease – fever, which lasts for 15 minutes – 1 hour, and then followed by a feeling of heat, in severe forms – chills repeated. Body temperature usually reaches a maximum
level by the end of 1st day from the start of the disease and a 37.5-40°C. Fluctuations between morning and evening performance of body temperature greater than 1°C, with a decrease in it – marked sweating. Duration of febrile period 3-7 days, and the treatment of effective drugs – no more than 2-3 days. More prolonged fever indicates complication. Dull headache, no localization and lasts for 1-2 days. Disturbed appetite and sleep. Symptoms of intoxication are generally correlated with the level of fever.

On examination, patients have facial flushing and neck, pale nasolabial triangle. The rash of angina is not typical, but in some cases it may be herpes rash on his face.

Symptoms of angina, the nature of pathological changes in lymphoid ring Pirogov nature of the disease depends on the clinical form of acute tonsillitis.

Catarrhal angina is characterized mainly superficial lesions tonsils and often precedes the deeper their defeat. Prodromal period lasts from a few hours to 2-4 days. The disease begins suddenly with a feeling of dryness, sore throat, general malaise, headache, pain in joints and muscles. Soon there is a pain in the throat, worse when swallowing. Can its irradiation in the ear. The body temperature rises slightly, but there may be chills. On examination tonsils hyperemic, edematous. Lymph nodes in the mandibular angle increased slightly painful on palpation. Changes in the blood are absent or negligible. The disease lasts for 1-2 days, after which the effects of inflammation in the throat or sore throat subsides goes to another form. Weather favorable.

Tonsillitis is characterized by a primary lesion of tonsillar parenchyma, their follicular apparatus. As well as lacunar, tonsillitis fever begins suddenly with fever up to 40°C, severe pain in the throat. Pronounced symptoms of intoxication, patients worried sudden weakness, headache, pain in the heart, joints and muscles. Sometimes there is the phenomenon of dyspepsia, oliguria. Hyperemic tonsils, swollen dramatically. Through the epithelium of follicles shine fester as whitish-yellowish formations size of a pinhead. The surface of the tonsils, the figurative expression of N.P. Simanovsky, takes the form of "star sky." Pronounced regionar limfadenitis. Changes in blood and urine are similar to those of lacunar tonsillitis.
These forms of angina essentially represent different stages of the same process. Allocation is carried out according to the predominance of certain symptoms.

In addition to the tonsils in acute inflammation may be involved, and other clusters limfadenoid tissue, in particular those in the nasopharynx (retronazal angina tubaric angina), on the tongue (lingual tonsillitis or sore throat IV\textsuperscript{th} tonsils), on the side walls of the pharynx (tonsillitis lateral ridges), in larynx (laryngeal angina). Sometimes inflammation is poured, spread throughout limfadenoidnomu throat ring.

For acute inflammation characterized by pharyngeal tonsils sore throat, radiating to the deep divisions of the nose, nasal breathing is labored.

The defeat of the lingual tonsil with pain on swallowing and protruding tongue. Inflammation spreads to the connective tissue and intermuscular, can lead to interstitial purulent inflammation of the tongue.

In acute inflammation limfadenoid tissue lateral ridges of the pharynx is usually a slight pain on swallowing, a slight increase in body temperature, Faringoskopy – redness and swelling of the lateral ridges, there can be seen festering follicles. Sometimes, the disease becomes rapid course, causing purulent mediastinitis.

Acute inflammation limfadenoid tissue located at the entrance to the larynx and its ventricles – laryngeal angina, characterized by sharp pain when swallowing, swelling of the epiglottis, arytenoid cartilage area, erythema and swelling folds vestibule and vocal folds, constriction of the glottis. On palpation the larynx (the front and sides of the middle portion of the neck), there is pain. The disease may be associated with the phenomenon of choking (mechanical asphyxia), and in some cases require tracheostomy.

Lacunar angina begins with a sharp rise in temperature to 39-40\textdegree C, with chills, significant malaise, sore throat, heart, joints, headache. May be a delay of a chair. Often there is increased salivation.

In pharyngoscopy – sharp hyperemia tonsillar swelling and infiltration. Gaps widened in them – a yellowish-white fibropurulent content, form on the surface of the tonsils soft plaque in the form of small lesions or film. Plaque can cover the whole amygdala, but does
not go beyond it, dull, and rises above the surface of the tonsils. He porous, friable and relatively easily removed from the surface of the tonsils, leaving a bleeding defect. Regional lymph nodes are enlarged, palpation are sharply painful. In the blood - leukocytosis to 12-20x10³ in 1ml, neutrophilic left shift, ESR 40-50 mm / h. The urine may appear traces of protein, sometimes – erythrocytes.

Lacunar tonsillitis usually lasts 5-7 days. During her stormy. Symptoms grow fast and also quickly subside. In stihaniya clinical manifestations of angina starts cleaning the tonsils of raids. Temperature falls politically. Swelling of the regional (submandibular) lymph nodes of other symptoms lasts longer – up to 10-12 days.

A special place among the primary acute tonsillitis is pseudomembranous angina, a clinical picture which was first described in 1890 by N.P. Simanovsky. A few years later published data Plaut and Vincent about the pathogen of the disease – the symbiosis of the fusiform bacteria and spirochetes of the oral cavity.

Vincent's angina usually occurs in people with a sharp decline in the body's defenses, suffering hypovitaminosis C and B, immunodeficiency, cachexia, some intoxication, and is characterized by the predominance of inflammation phenomena necrosis. The defeat of the tonsils, usually unilateral. On their free surface appear Easy removable grayish-yellowish-ups, which are formed by rejection maloboleznennye superficial ulcers with gray bottom. Ulceration may spread to the tonsils of the soft palate, gums, back of the throat, down into the vestibular larynx. In some cases, the necrotic process can cover and underlying tissues down to the periostem. The disease occurs at a relatively good general condition, accompanied by bad breath, drooling, pain when chewing, swallowing. On the affected side is developing regional lymphadenitis. The body temperature rises to subfebrile digits. Changing the composition of the blood usually comes down to a moderate leukocytosis, elevated erythrocyte sedimentation rate.

The diagnosis is confirmed when in the discharge of ulcers or film symbiosis fusiform bacteria and spirochetes of the oral cavity. Collection of material for laboratory studies should be made a loop on a glass slide. When taking a cotton swab can be negative due to the hygroscopic properties of cotton swab and drying on the swab.
Detection fuzospirohet symbiosis is not always possible to regard necrotizing process in the throat as pseudomembranous angina. Please keep in mind the possibility of joining fuzospirohet symbiosis with other ulcerative processes, such as ulceration cancer, angina in leukemia, agranulocytosis, chlamydia, radiation pharyngitis. In 10% of cases of pseudomembranous angina occurs in combination with diphtheria.

Severity and nature of the pathological changes in the organs and systems of angina depends on the severity of the disease and complications.

Angina always, to some extent, in the pathological process involves the cardio-vascular system. In patients observed tachycardia, arrhythmia, weakening tone and function of the heart sounds on auscultation. Almost all patients have declined in ECG voltage teeth, ST-segment above contour, rhythm and conduction disturbances. Many patients have a perverse reaction to physical stress: the mild exertion stroke and cardiac output does not increase, as is observed in healthy, but rather decreases. It is important to emphasize that all of these changes in the cardiovascular system, often do not occur in the acute stage of the disease and during convalescence and persist from a few weeks to several months.

Trachea and bronchi with angina are not affected.

Liver disease with angina is not typical. However, in the acute stage in severe in some patients it is possible to identify a moderate increase, pain on palpation of the xiphoid process (Lyakhovitskii symptom), muscle tension in the anterior abdominal wall of the projection area of the gallbladder (Glinchikov’s symptom), pain in the same area on inspiration from pre-fixing her right thumb (symptom Murphy) and some other symptoms indicating mild expressed cholangiohepatitis. All of these changes are due to infectious and toxic factors, the duration – short-term. Splenomegaly with angina is very rare, and mostly severe. When resistant (2-3 days), and a significant increase in the spleen, to examination of the patient in terms of exception conditions for which, along with other symptoms characteristic of tonsillitis and splenomegaly (a blood disease, infectious mononucleosis, etc.).

Renal disease with angina is often the case. Revealed certain
changes in urine – oliguria, nocturia, moderate proteinuria, leukocyturia, microhematuria, cylindruria. Observed in acute tonsillitis urinary syndrome is not a complication, but one of the manifestations of the disease and is caused by an infectious-toxic influence.

Pronounced changes in the gut with angina does not happen. However, the proportion of patients in the acute stage momentary delay chair (within 1-3 days). Violation of motor function of the colon due to the impact of microbial toxins. 1% of patients with angina may develop acute appendicitis, probably due to a streptococcal etiology of hematogenous drift from the oropharynx.

When laboratory blood of patients with angina observed neutrophilic leukocytosis (up to 9-20*10⁹ / L, relative lymphopenia, monocytosis accelerated ESR. Tendency to normalize blood counts and ESR appears only on 5-7 days of normal body temperature, but most of convalescents ESR remains elevated in a later period. the biochemical blood analysis disproteinemia (reducing the concentration of albumin and albumin-globulin ratio), increased CRP levels of sialic acids and seromucoid, increased fibrinogen. Changing these indicators due to inflammatory and destructive processes in the body and, to some extent, reflects their severity, as well as observed in the development of such complications of angina, as myocarditis and acute rheumatic fever. In practice, the definition of these parameters is important not only for the diagnosis of angina and assess the severity of disease, but rather to monitor the full recovery and early detection of these diseases metatonsillar.

Bacteriological examination of the surface microflora of tonsils in streptococcal angina revealed continuous growth of beta-hemolytic streptococci on 5% blood agar.

For serological study of paired sera collected in the first and 10-12 days, found an increase in antibody titer to streptolysin-O, streptokinase, streptococcal polysaccharide.

Determine the severity of angina to the general and local changes in the body, and are critical systemic toxicity violations – the height of fever, disorders of the nervous, cardiovascular, and other vital organs and systems. The degree of local changes in the throat usually correlates with the severity of the general intoxication.
Mild form of angina is characterized by low-grade fever in the body for 2-3 days, mild fatigue, mild pain in the throat when swallowing, catarrhal or follicular tonsillitis character, an increase of up to 1 cm in diameter of lymph nodes and moderate their pain. Symptoms of kidney disease are low (poorly defined urinary syndrome). Indicators of inflammation in blood tests are in the upper normal range.

Moderate form of angina is manifested by increased body temperature to 38.1-39°C during the first 4-6 days, significant intoxication (fever, fatigue, headache, muscle and joint pain, impaired appetite and sleep), severe pain in the throat swallowing, severe local changes (hyperemia of the palatine arches, uvula and tonsils, a large number of purulent follicles on the surface of the tonsils or pus in the gaps), an increase in diameter and the severity of their tenderness. May be short-term increase in liver size and tenderness to palpation its edge, impaired protein and carbohydrate its function. The kidneys – more severe and persistent symptoms of urinary symptoms. In the analysis of the blood – a moderate leukocytosis (up to 10-18 * 10^9 / L, increased erythrocyte sedimentation rate of 20 mm/h, a moderate increase in the sialic acid, CRP, seromucoid. Clinical signs persist for 5 to 6 days.

For severe angina is characterized by sudden weakness, severe headache, dizziness, loss of appetite, insomnia, periodic fever, succeeded by a feeling of heat, sweating. Temperature exceeds 39°C. Tonsillitis is characterized by constant pain in the throat, worse when swallowing, severe hyperemia tonsils, extending not only to the palatal arch and tongue, and the soft palate, a large amount of pus in the gaps. In some cases, tonsillitis is necrotic nature. Submandibular lymph nodes are enlarged to 2.5-3 cm in diameter and sharply painful. In 25% of patients have a short-term increase in the liver and the violation of its functions, in some patients, increased spleen. The absolute majority of patients show signs of kidney damage. Leukocytosis in the blood is in the range 16-24*10^9/l, ESR increased to over 30 mm/h.

From a theoretical and practical point of view it is important to allocate the primary, relapsing and secondary angina. These forms
of the disease have similar clinical manifestations, but differ in etiology, pathogenesis and outcome.

Immune responses after undergoing angina fade gradually and disappear completely in just 2 years. This period of time and should be a criterion that allows diagnosis to first and second form of the disease. The primary is angina that occurred for the first time or develop after 2 years after previously postponed. Relapsing angina in people who suffer from it every year or at least once every 2 years.

Strep throat is accompanied by the formation of autoimmune and immune-complex processes, which play an important role in the occurrence of diseases metatonsillar. When re-streptococcal tonsillitis newly formed immune responses superimposed on the medical history and increase their. Therefore, repeated angina more often and much more intensive autoimmune and immunopathological reactions, and much more likely to have diseases such as myocarditis, nephritis, etc., than the primary.

Secondary angina – angina, which is a symptom of another infection (scarlet fever, diphtheria, infectious mononucleosis, tularemia, etc.) or non-infectious diseases (acute leukemia, agranulocytosis).

Complications and adverse outcomes

The most important in a practical complications of angina are tonsillar abscess, and paratonsillitis paratonsillar abscess, cervical lymphadenitis and neck abscess, meningitis, mediastinitis, sepsis. Adverse outcomes, the so-called metatonsillar (tonzillogenic) diseases developing after undergoing angina include chronic tonsillitis, acute rheumatic fever, myocarditis, tonsillogenic myocardiodistrophy, nephritis, holangiocholecystitis. Doctors, especially district internists need to know the clinical features of the disease, to be able to foresee the possibility of their development in order to conduct timely preventive measures.

Abscess tonsils (tonsillar abscess) – are relatively rare, but quite threatening complication. Usually occurs at 3-4th day of the disease with moderate or severe sore throat, accompanied by a pronounced inflammation of the tonsils, joined by a staphylococcal infection causing purulent fusion of the affected tissues within the amygdala. Against the background of perceived at that time improvements in the patient's condition suddenly the body
temperature rises to 39°C or more. Often repeated shivering, followed by a feeling of heat. Rapidly increase the toxic symptoms of central nervous system – general weakness, headache, pains in the back, muscle pain, anorexia, insomnia. Simultaneously, there is a sharp increase or that existed before, sore throat with one hand. Unlike pain in uncomplicated angina, she worried sick and alone. When viewed from the oropharynx than typical angina changes, there is a significant increase in the size of the tonsil on one side and the corresponding location of the emerging abscess. The infected tonsil almost covers half the throat, has smoothed the gaps, more sharply hyperemia compared with the second tonsil, often acts anteriorly or posteriorly (depending on the location of the abscess) and sharply painful when touched. On the same side dramatically enlarged and painful lymph nodes uglochelyustnye. In peripheral blood characterized by leukocytosis of 18-25*10^9/L, neutrophilia to 78-85%, ESR increase to 30-40 and over mm/h.

Described similar clinical symptoms characteristic for paratonsillitis and paratonsillar abscess, but there are differences. When tonsil abscess no lockjaw masticatory muscles, mouth opening is free, there is no inflammatory infiltration of the anterior arch and the soft palate on the same side. For 3-5 days of the onset of signs of an abscess in a limited area of the affected tonsil superficial abscess appears as a yellowish-whitish protrusion. When you do not surgery, the abscess in the next 2-3 days revealed themselves. An autopsy is carried out through a gap tonsils, usually during sleep, unnoticed by the patient. After opening the abscess relatively quickly, within 2-3 days, normal body temperature, the symptoms of intoxication, reduces the size of the affected tonsil. However, changes in the blood (leukocytosis, high ESR) saves up to 2-3 weeks.

**Paratonsillitis, paratonsillar abscess** are stages of the same inflammatory process that develops as a result of the penetration of pathogens in paratonsillar tissues. Initially, there are inflammatory edema and cellular infiltration of the affected tissue – paratonsillitis, then it is purulent melting and forming paratonsillar abscess. Paratonsillitis and paratonsillar abscess may develop as the mild and severe disease. This generally occurs in people with frequent episodes of angina. They sharply reduced local (tonsillar) immunity, and will
break the barriers in the capsule of the tonsils, thus creating favorable conditions for the spread of disease pathogens in paratonsillar tissues and its involvement in the inflammatory process with subsequent formation of an abscess.

Tonsillar and paratonsillar abscesses have similar symptoms: the background of relatively satisfactory condition, the patient re-angina, sudden increase in body heat to high numbers with a fever, with symptoms of severe general intoxication, unilateral severe pain in the throat when swallowing, and even at rest radiating to the ear or teeth on the affected side. Pain also increases with spitting and moving head to the side. Often develop increased salivation. Patients are forced to take the position with tilt head forward, to promote self-draining of saliva from the mouth. Because of the spread of the inflammatory process with paratonsillar tissues the soft palate tissue violated its mobility, which leads to a change in voice, he becomes not loud with a nasal tone. When abscess of paratonsillar fiber is forming it often cause a lockjaw with masticatory muscles, which manifests the difficulty and limited mouth opening. Spread of the inflammatory process in the lymph nodes and tissues of the neck is accompanied by pain in them, growing moving neck. Because of this, patients have to tilt your head to the affected side and keep it there motionless (state torticollis).

In patients with paratonsillitis and paratonsillar abscess from mouth spreads nasty putrid smell. When there is a one-way pharyngoscope hyperemia, infiltration, edema and swelling in paratonsillar of the possible spread of the soft palate and uvula, and the offset to the center of the affected tonsil asymmetry and uvula.

Peripheral blood - similar as in tonsillar abscess.

**Parafaringeal flegmona, parafaringeal abscess** develop in patients with paratonsillar abscess. The symptoms characteristic of paratonsillar abscess toothache joins a hearing loss due to damage and twigs n.alveolaris inferior pharyngeal part of the auditory tube. When viewed from the side of the throat swelling of the affected side is detected tissues, often extending into hypopharynx. There is swelling and tenderness of the affected soft tissues of the neck, mainly in the upper part of the sternocleidomastoid muscle and submandibular area. Often these changes in the cervical lymph nodes located along the neurovascular bundle from the mastoid process to the clavicle.
**Purulent inflammation of the neck lymphatic nodes** – an extremely rare complication of tonsillitis. It is preceded by a significant increase of nodes (up to 4 cm in diameter), accompanied by severe morbidity, and then there is their softening and appears symptom fluctuation. Without prompt surgical treatment – formed fistula.

Spread of inflammation from parafaringeal space into the base of the skull into the mediastinum or in the blood leads to the generalization of infection and causes meningitis, mediastinitis and sepsis.

On the development of **meningitis** may indicate the appearance of the background of high fever and severe intoxication, arching nature of headaches and symptoms of lesions of the meninges – photophobia, hyperesthesia, hyperacusic (hypersensitivity to auditory stimuli), nausea, vomiting, neck stiffness, Kernig and Brudzinsky symptoms – upper, middle and lower.

**Mediastinitis** is the result of the spread of pus from parafaringeal space along the neurovascular bundle in the mediastinal tissue. In this case, the patient's condition deteriorates, increased toxicity (severe fatigue, headache, dizziness, insomnia), significantly increased swelling and tenderness of soft tissues across the side of the neck. There are signs of compression of the mediastinum (esophagus, larynx, trachea).

**Tonsillar sepsis** occurs in a sharp decline in the local (tonsillar) and total body resistance and is accompanied by a rough generalization of agents with the formation of secondary foci of their localization, reproduction and re-generalization.

The dramatic suppression of local and general immunity most often caused by long-term recurrent illness (diabetes, asthma, systemic connective tissue disease, etc.), prolonged use of corticosteroids and other immunosuppressive drugs, or exposure to macroorganism – by microbes themselves.

Streptococcal sepsis have an acute onset. A patient with angina, often complicated paratonsillar abscess, several times a day marked a stunning chill, accompanied by an even more significant increase in body temperature, the appearance of sharp weakness until adynamia, thirst, severe headache, pain in the large joints, myalgia. Body temperature during the day exposed to large fluctuations. The temperature profile is incorrect, hectic and sometimes permanent.
Lowering it is accompanied by profuse sweating, disappearance of artalgy and myalgias.

The patient's face is pained expression. Ashy-gray skin with cyanotic or yellowish tint. Sometimes there is a rash of hemorrhagic or type of hives.

Paramandibular lymphatic nodes are enlarged and tender. Can be affected other anterior neck nodes. Revealed swelling and tenderness of tissues adjacent to the front surface of the sternocleidomastoid muscle.

Lips are dry, cracked, mouth – unpleasant putrid odor. Tongue is dry, covered with white or brown tinge. Changes in the oropharynx depends on the phase of the disease with angina. Sepsis can develop to the stage of recovery, then it will be observed only diffuse redness of the mucous membrane oropharynx.

Almost always reveals a heavy defeat of the cardiovascular system (thready pulse, tachycardia, arrhythmia, hypotension, expanding the boundaries of the heart, ECG changes typical for myocarditis) and respiratory system (shallow rapid breathing, secondary screenings pus in the lungs).

Revealed liver enlargement, the edge of her tight and painful. Palpable spleen at the costal arch, soft, painless.

In the analysis of peripheral blood is anemia, neutrophilic leukocytosis with a shift to the left, significantly accelerated ESR.

**Chronic tonsillitis** is a consequence of repeated sore throats, especially when they occur in short intervals of time, during which the body does not have time to completely eliminate the local inflammatory changes in the tonsils and disorders in other organs. However, under certain conditions, chronic tonsillitis can emerge as the primary slowly progressive disease. In the development of chronic tonsillitis leading role played by reduced local (tonsils) and the total resistance of the body, the effects of cold and other adverse factors, not germs as angina.

Chronic tonsillitis is fundamentally different from angina in pathogenesis of local and systemic disorders, clinical symptoms, outcomes, principles and methods of treatment and prevention.

Chronic tonsillitis long flowing inflammation of the tonsils leads to the replacement of the parenchyma by connective tissue, the formation of adhesions of these bodies to palatal temples, as well as
the gaps between the walls. This violates the evacuation of the gaps microbial saprophytes ottorgnuvshihsyap epithelial cells, tissue fluid and other substances. Accumulation of the products of inflammation in the gaps accompanied their cone-shaped extension, thinning or complete disappearance of the surface epithelium. In this connection, facilitated absorption of content gaps and keep germs inside the tonsils. When complete obliteration of the mouths of lacunas form a closed cavity (retention cysts), which accumulate pathological substrates. The above changes in the tonsils promote a low-intensity of the inflammatory process, which under certain conditions may worsen. Accumulated in the gaps of the deformed products of microbial and inflammatory origin are able to provide not only local, but the overall effect on the body. Being absorbed into the blood, they can determine the appearance of symptoms of intoxication – general weakness, decreased performance, artalgii, subfebrile body temperature, etc. (decompensated form of chronic tonsillitis).

Microbial penetration into the parenchyma of the tonsils leads to the fact that in different parts of the fabric of these bodies meet single or multiple microinfiltrats, the center of which may appear microabscesses. Periodic formation in different parts of the tonsils leads to the gradual disappearance of the lymphoid follicles and their replacement with scar tissue. Penetration of microbes and their metabolic products in the lymph path leads to the defeat of the regional (angular) for tonsil glands. They become enlarged, and an exacerbation of the process and painful. In the long lymphadenitis or part of a lymph node recurrence germinate connective tissue, which gives them a dense texture.

Symptoms of chronic tonsillitis polymorphic. Along with symptomatic forms of the disease, often virtually asymptomatic. Patients may complain of discomfort or recurrent moderate pain in the throat when swallowing, blurred pain submandibular nodes, palpitations, irregular heart function, false angina, hot flashes, etc. The objective changes may also have a different picture. Tonsil size in patients with chronic tonsillitis have no diagnostic value. Lumpy nature of the surface of tonsils also very relative. Relative diagnostic importance is fusion of the palatine arches with almonds. Reliable sign of the disease is the combination of these changes with edema and
congestive hyperemia palatine arches, due to a violation of local blood and lymph circulation in the tonsils and surrounding tissues, and the presence of caseous-purulent (yellowish-white) masses in the gaps of scar-modified tonsils. Changes in chronic tonsillitis tonsils resemble lacunar tonsillitis with lacunar angina. However, unlike angina, chronic tonsillitis missing all her other symptoms (fever, severe intoxication, inflammatory tissue changes tonsils - redness, swelling, abundant on the surface of fluid). Content gaps in chronic tonsillitis has a relatively dense consistency and stored for an unlimited time.

Blood picture in chronic tonsillitis in most cases does not change.

In the pathogenesis of chronic tonsillitis play a significant role of neuro-reflex mechanisms. Pathological process in the tonsils accompanied by severe degenerative changes in the vagus nerve and the sympathetic ganglia, which is reflected in the functional status of innervated organs and systems. First of all, it concerns the cardio-vascular system. Significant changes in the tonsils in chronic tonsillitis rarely correlate with common manifestations of the disease. Even when there is scarring of the tonsils and the formation of adhesions to palatal temples, constant presence of pus or cheesy masses of dense fluid in the gaps, expressed lymphadenitis patients usually do not experience any trouble.

There are two clinical forms of chronic nonspecific tonsillitis (by I. Soldatov) – compensated and decompensated. *Compensated chronic tonsillitis* has only local (on the tonsils) manifestations. *Decompensated chronic tonsillitis* is characterized not only by changes of the tonsils, but expressed general symptoms (low-grade fever, fatigue, decreased performance, artalgii etc.), frequent exacerbations of the disease, in many cases – the presence of conjugated diseases.

*Myocarditis* develops in primary angina in the early days of the period of convalescence, and when re-from the first days of illness. When it is caused by streptococcal angina allergic and infectious-toxic factors. The typical picture of myocarditis occurs in only 1/3 of patients: general weakness, fatigue, discomfort or pain in the heart, casual low-grade temperature of the body (mainly in the evening and at night), the lability of the pulse with a tendency to tachycardia, muted tones of the heart, systolic murmur at the apex. Marked ECG
changes: extension segment PQ, changing the shape and direction of the T wave, offset segment ST. In the analysis of the blood is neutrophilic leukocytosis, accelerated erythrocyte sedimentation rate, increased C-reactive protein (CRP), sialic acid aminotransferase, lactate dehydrogenase (LDG).

However, most often the only signs of myocarditis are persistent ECG changes indicating a focal disease of the heart muscle and inconsistent changes in blood tests: the upper limit of normal or slight increase in white blood cell count and ESR, increased CRP, sialic acid, 1-2\textsuperscript{nd} fractions LDH.

**Tonsillogenic myocardiodistrophy** developed in patients with recurrent and chronic tonsillitis, is one of non-inflammatory disease of the heart muscle. Factors causing myocardial dystrophy include:
- Cardiotoxicity streptolysin O produced by Streptococcus.
- Allergy to streptococcus antigens and autoimmune reactions associated with the appearance of autoantibodies antikardialnyh.
- Tonsillitis-cardiac reflex, leading to changes in the blood vessels and muscle fibers of dystrophic myocardium.

Schematically pathogenesis of tonsillogenic myocardiodystrophy can be represented as follows:

```
recurrent angina (chronic tonsillitis)  ↓
abnormal afferent signals in the higher parts of the CNS  ↓
violation of the autonomic regulation of the heart  ↓
change in the balance of neurotransmitters in the myocardium (epinephrine, norepinephrine, acetylcholine)  ↓
violation of oxidative phosphorylation and the development of myocardial hypoxia  ↓
disorders of energy  ↓
transition to glycolytic pathway exchange  ↓
anxiety disorder and electromechanical coupling in the myocardium  ↓
disorder and rhythmic contractile function of the heart.
```
Biochemical and structural changes in the heart muscle during tonsillogenic myocardiodystrophy are reversible.

Clinically, there is pain in the apex of the heart, stabbing or aching nature, long-term, often intense, sometimes radiating to the left shoulder girdle, shoulder blade, shoulder. The pain associated with lesions of the cervical and upper thoracic sympathetic nerve plexus. In addition, patients complain of fatigue, general weakness, growing in the second half of the day, a sense of dissatisfaction with breath that occurs most often at rest and decreases markedly during physical stress or distraction. Rarely observed shortness of breath, palpitations, feeling of disruption of the heart.

Objectively in patients with moderate tonsillogenic myocardiodystrophy are expanding the boundaries of the heart to the left, I weakening tone at the top, soft tone, blowing, weak or moderate systolic murmur at the apex of the heart, often disappearing after exercise and does not conduct in the left armpit. At the base of the heart often auscultated systolic murmur, which is often a wire from the top. Of the pulmonary artery can focus listens and splitting II tone.

On the ECG recorded dysfunction automaticity and conduction. Marked decrease in the amplitude of P wave and T wave less R, – segment ST. ECG test with potassium and obsidan – is positive.

Changes in blood tests are not usual for myocardiodystrophy, rather they reflect the activity of the inflammatory process.

**Nephritis** occurs mostly after a sore throat caused by β-hemolytic streptococcus group A, the so-called nefritogenic strains. Development of nephritis is the time of formation of autoimmune (against kidney tissue) and immunopathological factors – 5-6 days of normal body temperature (8-10 days of the disease. Most nephritis after undergoing angina occurs without extrarenal symptoms. Sole manifestation of it may be urinary syndrome: proteinuria, hematuria, leukocyturia and often cylindruria.

It should be noted that small changes in the urine are observed in the recovery period in patients who have had a sore throat, quite often the result of infection and renal toxicity during the acute period of the disease. An important feature of these changes is that they are short-term and repeated examination of the urine for 10-12 days of normal
body temperature are no longer registered. In the case of jade changes in urine are persistent.

The incidence of nephritis in angina is largely dependent on the type of etiotrop treatment, but even more it is associated with the etiology of the disease and the presence of frequent repeated sore throats in the past.

**Cholangiocholecystitis** is fairly common consequence of angina, especially re. Clinically it is manifested slight increase in liver size and the presence of gall bladder symptoms – Musso, Ortner, Murphy, Kerr, etc. In the study of duodenal contents found clumps of mucus and increased number of white blood cells.

Cholangiocholecystitis phenomenon in patients undergoing primary angina usually pass quickly, in people with recurrent angina, and frequent exacerbations of chronic tonsillitis may be permanent in nature.

**Diagnosis of angina.**

The diagnosis of angina is based on clinical, clinical and laboratory data.

Supporting differential diagnostic symptoms of streptococcal sore throat (on Lobzina Yu et al., 2000):
- Predominantly young age;
- Characteristic epidemic anamnesis;
- Acute sudden onset with chills, high fever, intoxication;
- Sharp pain when swallowing.

Proper planning of treatment strategy dictates the need for at least a minimum set of paraclinical examinations, which must include:
- Complete blood count (to exclude infectious mononucleosis and blood disorders) – changes are nonspecific: neutrophilic leukocytosis, elevated erythrocyte sedimentation rate;
- Urinalysis (to avoid jade) – short-term notes albuminuria and pyuria;
- Blood chemistry (often used to diagnose complications and adverse outcomes) – increase of CRP, sialic acid, seromucoid,asl-o, etc.);
- Swab of the oropharynx and nose with a border of inflamed and healthy tissues (for the differential diagnosis of diphtheria, to highlight the microflora and its sensitivity to antibiotics): isolation from the surface of the tonsils and throat mucous membranes of
microorganisms is of little diagnostic value, because healthy people often found a variety of representatives of both opportunistic and pathogenic organisms. The value of this study is increased by determining the number of pathogens at the site of abnormality. However, due to complexity of this research is rarely done.

- ECG.
- Consultation on the testimony of the otolaryngologist and other professionals.

Methods for specific diagnosis of \( \beta \)-hemolytic streptococcus group A (BHSA) include:
- Allocation of pathogen culture swabs from the surface of the tonsils;
- Rapid diagnosis of streptococcal antigen;
- Immunoserologicheskoe study.

Cultural method – collection of material with a cotton swab with immediate transferring it into the medium - as should be done with angina to prevent diphtheria, suspected specific infections, with the ineffectiveness of empirical antibiotic therapy. Sensitivity 90%, specificity \( \geq 96-99\% \).

The method of rapid diagnosis BHSA based on the identification of streptococcal antigen in smears by its enzymatic or acid extraction followed by agglutination, demonstrating the formation of a complex "antigen-antibody". The advantage of the method is the fastest results, efficiency, high specificity (95-100\%) and the ability to restrict the use of antibiotics to cases of the disease that requires eradication BHSA. However, the method has a wide sensitivity range – 50-95\%.

**Immunoserological diagnosis** is based on the detection of elevated antistreptolysin titer, anti-DNAse \( \beta \)-antistreptokinase and other antistreptococcal antibodies. This method is more specific for streptococcal infection, but sensitivity decreases sharply against antibiotics.

According to the recommendations of the American Academy of 2002, the indications for microbiological studies to identify BHSA acute tonsillopharyngitis based on the presence and severity of the four main characters: a raid on the tonsils, painful cervical lymph nodes, fever and no cough.
The differential diagnosis

Angina should be differentiated from flu and acute respiratory viral disease, acute pharyngitis and measles, as well as with secondary acute tonsillitis, that is, with a sore throat for infectious diseases such as diphtheria, scarlet fever, tularemia, typhoid fever, and in diseases of the blood system – infectious mononucleosis, agranulocytosis, leukemia. Differential diagnosis of primary acute tonsillitis, you should always be mindful of possible defeat in the throat early contagious syphilis and tuberculosis. Differentiation is done by comparing the clinical features of these diseases, and laboratory results.

**Influenza** is characterized by: a short incubation period (from a few hours to two days), acute onset, severe intoxication. Catarrhal symptoms runny nose – rhinitis, cough, sore throat or pain when swallowing, etc.) often delayed by 1-2 days, or do not occur. Characterized nasopharyngitis, laryngotraceitis, tracheitis, tracheobronchitis. Hyperemia throat varying degrees in all patients is often combined with granulosa pharyngitis on the back of the throat and fine-grained tongue and soft palate. In blood – leukopenia, ESR is normal, sometimes moderately increased.

**Parainfluenza infection** is characterized by symptoms of intoxication, and catarrhal syndrome, which occurs by type rinofaringolaringita. The incubation period – 3-4 days. Onset of the disease – a subacute, symptoms grow to 2-3 day of illness. Moderate intoxication syndrome, intensity increases by the 3rd day of illness, duration – 1-6 days or more. Catarrhal symptoms in the first hours of the disease and lasts 8-10 days. Observed moderately severe flushing handles, the tongue, dry and grainy mucosa of the pharynx. The mucous membrane of the tonsils, and the tonsils themselves rarely affected. In peripheral blood normocytosis with a tendency to lymphopenia. ESR is normal or slightly increased.

**Adenovirus infection** is characterized by a variety of clinical manifestations. Any of the clinical forms of its characteristic set of symptoms of acute respiratory illness and other losses – keratoconjunctivitis syndrome, angina, poliadenopatiya, hepatolienalny syndrome, etc.

Onset of the disease in most patients with acute, the body temperature to the 2-3rd day reaches 38-39°C, rare – 40°C. The disease
may begin gradually, then the heat only comes to the 4th day of illness. The rise in temperature is often accompanied by a slight fever, or a short-term chilling. Fever in typical cases it is long – about 6 days or more, sometimes up to 2-3 weeks. The temperature profile is constant or remittent character, may be two-wave or periodically subfebrile through consistent involvement in the pathology of other organs.

At the height of the disease the symptoms of intoxication are mild or moderate: slight weakness, headache, muscle and joint pain. Catarrhal symptoms of the upper respiratory tract develop in the first few days of illness. In most patients, there is congestion with abundant serous or sero-purulent discharge. In contrast to the banal angina at 1-3-day sickness appears conjunctivitis, which is more often catarrhal, at least – follicular and filmy. When viewed from the mouth marked diffuse hyperemia, granularity of the soft palate and the posterior pharyngeal wall. Characteristically catarrhal or follicular lesion lacunar tonsils with submandibular and cervical lymphadenitis. When adenovirus infection can be observed abdominal pain, diarrhea, liver enlargement, at least – the spleen.

In the blood was small leukocytosis, in some cases, leukopenia, neutrophilia, normal or elevated ESR. For the specific diagnosis of adenovirus infection are widely used RAC and HAI in paired sera.

Infection caused by respiratory syncytial virus, is characterized by mild symptoms of intoxication and a primary lesion of the lower respiratory tract. Moderate headache in the fronto-temporal or occipital region, fever, chills, nausea, vomiting, and tend to be in the early days of the disease. Syndrome of intoxication lasts from 1 to 7 days. Catarrhal symptoms rather scanty: rhinitis occurs in one third of patients, moderate hyperemia throat – in almost all patients. Duration catarrhal syndrome 4-6 days. In 10% of patients the effects of bronchitis with asthmatic component. In the blood – eosinophilia, neutrophilic shift to the left of the formula for the normal number of white blood cells.

In rhinovirus infection incubation period is 1-6 days, no prodromal symptoms, intoxication is weak: malaise, "chilling", dragging pain in the muscles, heaviness in the head, low-grade fever. Rhinitis in the first hours of the disease, first mucus, sometimes profuse, watery consistency, then thicker seromucous. Hyperemia of
the throat and the back of the throat is expressed slightly more often the process is limited bows. It is sometimes swelling of the mucous and "grain" of the soft palate. Often there is conjunctivitis. Hematologic abnormalities sometimes appear small leukocytosis.

**Coronavirus infection**, especially mild, like a clinic rhinovirus infection and is characterized by profuse watery discharge from the nose. Among other catarrhal symptoms observed intense sneezing, at least – a cough. An objective examination of observed redness and swelling of the mucous membranes of the nose, flushing of the oropharynx. Intoxication symptoms are mild, increase in body temperature, usually not observed or not higher subfebrile. May be affected by the lower respiratory tract by type of acute bronchitis.

**Mycoplasma infection** is characterized by polymorphism of clinical symptoms, moderate toxicity, moderate or mild catarrhal symptoms, which occur in two clinical variants: acute respiratory infections and pneumonia. The incubation period lasts from 1-8 up to 25 days or more. The gradual onset of the disease with symptoms of intoxication that peak at 2-7 day and keep from 3 to 10 days. Catarrhal syndrome manifested primarily rhinopharyngitis, faringobronhitom. In the acute phase is often determined by an increase in the submandibular, cervical, axillary and inguinal rarely, lymph nodes. Mycoplasma pneumonia often develop within the first three days of the disease, combined with symptoms of ARI. Pneumonias are often patchy, physical symptoms are poor and inconsistent.

Acute respiratory disease occurs with pronounced symptoms of rhinitis, rhinorrhea. Body temperature is usually not reach high numbers, absent or mild symptoms of intoxication.

In **acute** or in **exacerbation of chronic pharyngitis** the general condition does not suffer, do not bother the patient pain, and the feeling of irritation, sore throat or interference, which manifests itself in the "empty" throat and disappears when taking thick or solid food. The disease can appear suddenly and disappear just as suddenly, last a few minutes, hours or weeks or months, if not eliminate the causative stimulus. Relief comes usually after receiving a warm non-irritating foods. On examination of the pharynx revealed relatively inflammatory changes in the mucous membranes, but it is predominantly back and side walls, in the form of congestion and
Diphtheria of throat. The greatest difficulty for professionals of all skill levels, including infectious diseases, causes diagnosis of diphtheria throat. This is due to the severity of the disease and its complications, the timing of the early etiotrop treatment (serotherapy) that will often depend on the patient's life.

Depending on the extent of the local process and degree of general intoxication are three main forms of diphtheria throat: 1) localized (fibrinous coating is within the tonsils) subdivided into ostrovchatuyu, filmy (solid) and bluetongue, 2) common, in which attacks go on palatal arch, the tongue or the back of the throat, and 3) the development of a toxic edema of the throat and neck subcutaneous tissue.

Localized form of diphtheria throat unlike catarrhal angina begins gradually. Body temperature is usually less than 37.5-38°C. Patients concerned about the general weakness, loss of appetite, heaviness in the head, mild sore throat.

When island form of diphtheria during moderate intoxication tonsils are enlarged and hyperemic (congestive hyperemia with a bluish tint), on the surface there are islands of fibrinous films, which is slightly above the back of tonsils and can not be removed with a spatula.

In filmy form of diphtheria against congestive hyperemia of the mucous membranes of tonsils, palate, soft palate arches and the tonsils are found solid fibrinous raids white and grayish-white in color with a smooth surface and well-rounded edges. Plaque is removed with difficulty, and the subiculum bleeds. Previous film is pulverized between spatulas, insoluble in water and slowly settles to the bottom of the vessel. Regional lymph nodes are moderately enlarged and painful on palpation. The fever lasts for several days, but after normalization of temperature condition of the patients is not improved.

Catarrhal form of diphtheria at the throat, which should be differentiated from catarrhal angina, there is no characteristic symptom of the disease – fibrinous plaque. Leading symptoms – gradual onset, mild redness and some swelling of the tonsils. Body temperature is usually not increased, no toxic symptoms. Diagnosis in such cases is only possible on the basis of epidemiological data, and
detection of toxigenic diphtheria bacilli for bacteriological examination of mucus from the nose and throat.

Under the influence of serotherapy in 24 hours film as it rises above the mucosa, no further spreading, and disappears in 2-3 days. Without the introduction of diphtheria serum disease progresses – symptoms of intoxication are increasing, raids beyond the tonsils, developing common or toxic diphtheria throat.

Common form of diphtheria throat often develops from a localized and rarely alone. Disease begins acutely: the body temperature rises to 38.5-39°C, symptoms of intoxication – general weakness, fatigue, headache, drowsiness, and sometimes vomiting, pallor, tachycardia, cardiac muted tones. Against the background of congestive hyperemia and enlarged tonsils are found on the surface of solid fibrinous raids, which apply to the tongue, palatal arch, the back of the throat, which is not typical for primary angina. Color plaque may be whitish-gray, dirty yellow, depending on the duration of the disease. The regional lymph nodes are enlarged to the size of a large bean, painful on palpation, but the cervical tissue swelling does not happen.

The toxic form of diphtheria throat sometimes develop after localized, but most often occurs from the beginning as toxic. In most cases, it starts rapidly: the body temperature rises to 40°C or more, headache, severe fatigue, insomnia, anorexia, sore throat, vomiting, and can be a pain in the stomach. From the first hours of the disease marked diffuse redness and swelling of the mucous membranes of the oropharynx, which often precede attacks. Soft palate, uvula and arch swollen. With a pronounced swelling of the tonsils are touching. Especially swollen and enlarged tongue, he squeezed and disadvantaged enlarged tonsils, causing the back of the throat examination difficult. The raids are initially delicate websing the film, which is easy to remove, and then reappears. After 2-3 days of onset raids become dense, thick, dirty-gray color, fully cover the surface of the tonsils, moving to the bow, the tongue, soft and hard palate. Hyperemia of oropharynx at this time is reduced, they become bluish tint, swelling reaches its maximum development. Tongue coated, lips dry, cracked, mouth feel peculiar sweet, sickly smell. Nasal breathing is difficult, from the nose appear sukrovichnye allocation, macerate
the skin in front of the nose, some patients show a film on the nasal septum. Weak voice with a nasal tone.

Along with the development process in the throat occurs regional lymphadenitis – increase verhnesheynye lymph nodes, sometimes forming large conglomerate. On palpation it elastic and painful. There is swelling of subcutaneous tissue, the skin color of edema is not changed, pressure is painless and leaves no holes. The prevalence of cervical subcutaneous tissue edema corresponds severity of intoxication. Therefore, depending on the degree of swelling are three toxic diphtheria: I – spread to the middle of the neck swelling, II – to the clavicle, III – below the collarbone.

At subtoxic form throat diphtheria intoxication moderate, raids are located mainly on the tonsils and rarely applied to the tongue, soft palate, the back of the throat. Swelling of the cervical tissue are usually mild and mostly reaches the submandibular lymph nodes, swelling of the tonsils and soft palate at otdelnyx patients can be clearly expressed (edematous form). The raid on the tonsil tissue and swelling of the neck is often one-sided.

Objective picture in the throat with toxic diphtheria looks like paratonsillar abscess. And unlike these diseases with toxic diphtheria is no express lockjaw and the patient is able to open his mouth, which allows for inspection of the horny part of the pharynx. When paratonsillar abscess patient fails. In addition, when quinsy in contrast toxic edema pronounced inflammatory response, a significant increase in regional lymph nodes and extreme soreness.

During the demonstration, and diphtheria in various age groups is largely determined by the amount and intensity of prevention efforts to eliminate the infection. Therefore, in recent years, the frequency of disease disappeared and seasonal variations, suffer mostly the elderly, the increased proportion of cases among rural residents. The clinic diphtheria were recorded more often light and blurred form of the disease with an increase in the proportion of toxic diphtheria throat disappears diphtheria rare localizations and diphtheria throat, accompanied by croup, decreased rate of complications and mortality.

Manifestations of local process in diphtheria vaccinated correspond to those of bluetongue lacunar angina or with the presence
of loose, easy to shoot strikes, does not apply even if the toxic form of diphtheria (S.D. Nosov, 1980).

The course and the manifestation of diphtheria in adults characterized by the fact that the initial period resembles that with lacunar angina (body temperature to 38-39°C, headache, weakness, fainting, pain in the throat). Local changes little consistent with the typical signs of diphtheria: bright hyperemia, the presence of loose, easy to remove plaque in the gaps, moderate swelling (L.A. Favorova et al., 1988). Pathological process in these cases may have a favorable outcome, which is the cause of delayed diagnosis.

Scarlet fever. Angina occurs when the disease to the syndrome of acute tonsillitis in combination with fever and intoxication. Disease begins acutely: the body temperature rises from subfebril to 40°C and more, there is pain in the throat, picking up signs of intoxication (general weakness, vomiting, headache). The most common manifestation of intoxication – vomiting of central origin without anticipatory nausea.

In contrast to the banal sore throats for the presence of scarlet fever typically exanthema. The rash appears on the end of first or on the second day of onset. For scarlet fever is characterized by a rash on the dotted background hyperemic skin, which thickens in the natural folds (axillary, inguinal, popliteal region). On the face as a result of the merger of separate elements abundant rash observed bright with pale cheeks flushing nasolabial triangle. In the folds of the skin, especially in the elbow, there are petechiae, the merger of which folds become saturated color with brown or purple hue (a symptom of pasta). The rash usually lasts 3-7 days, depending on the severity of the disease and disappears, leaving pigmentation. In the second week of illness appears peeling, most pronounced in the toes and hands.

For scarlet fever is typical bright hyperemia tonsils, uvula, handles, not passing on the hard palate. The defeat of the tonsils may occur as a catarrhal, follicular lacunary, necrotic and fibrinous false angina. Catarrhal and follicular lacunar angina of scarlet fever developed from the first day of illness, are more vivid oropharyngeal hyperemia compared to those with normal (banal) angina and disappear after 4-5 days. Necrotic angina appears on the 2-4th day of illness. Depending on the severity of the disease can be superficial
necrosis (in the form of small individual plots) or deep, ranging across the surface of the tonsils. Necrosis has a dirty gray or greenish color, fade slowly – within 7-10 days. Pseudofibrinous angina develops most often on the first week of the disease, accompanied by a high temperature (up to 39-40°C) and the formation of a thick fibrinous film, tightly knit with the underlying tissue. The film covers the surface of the tonsils and can be distributed on the buccal mucosa, as well as nasopharyngeal space. After removal of the film ulcerated mucosa and bleeding. When a significant distribution process is violated not only swallow, but breathing.

Language in the disease densely coated gray-white coating, but with 2-3rd day begins to clear from the edges and the tip turns bright red with prominent papillae ("raspberry" language).

Accordingly, the degree of destruction of tonsils in the process involved regional lymph nodes. It should be noted that cervical lymphadenitis in scarlet fever occurs earlier and less prominent than the usual angina.

**Infectious mononucleosis.** Typically, the syndrome presents with fever, intoxication, poliadenopatiey, hepatosplenomegaly and characteristic hematological data. The disease usually begins acutely: the body temperature rises to 38-39°C and is accompanied by a fever, appear sharp weakness, headache, myalgia, arthralgia, later – sore throat MRI swallowing.

Fever – is often the first and most common symptom of the disease. At the height of the disease the body temperature rises to 38-40°C, the duration of fever – 2-4 weeks. Temperature curve basically wrong type, but it can be a constant, remittent, hectic.

Angina appears from the first day of illness or develops later against fever and other symptoms of the disease (5-7-day). It may be catarrhal, follicular, lacunary, filmy and necrotizing. Hyperemia tonsils, uvula and arches moderate, slight sore throat. In most cases, loosening the tonsils, swollen, can link up to the middle line and obstruct breathing. Often on the surface of the tonsils appear loose, rough-ups, which are easily removed and pounded. These come in the form of islands, stripes or solid films whitish-yellow or dull gray. The raids are located mainly in the gaps (88%), but sometimes applied to the surface of the tonsils. In 10% of patients have filmy angina
resembling diphtheria throat, and even more rarely – follicular and necrotic. On average raids held about a week after their disappearance mucosa hyperemic and loosened.

Often affects the nose throat and nasopharyngeal tonsil, which manifests the difficulty of nasal breathing, voice changes ("muffled" voice, as in paratonsillitis). Typically, patients breathe through the mouth, while the nasal passages are free and no nasal discharge, due to lesions of the mucous membrane of the inferior turbinate and the entrance to the nasal part of the pharynx (back rhinitis).

Lymphadenopathy – a characteristic and the most constant symptom of infectious mononucleosis. From the first days of illness revealed generalized lymph nodes – cervical, axillary, inguinal, neck, chin, BTE, subclavian, mesenteric, although it noted ¬ denotes not always and in all patients. Tend to increase in the lymph nodes, especially the posterolateral neck, arranged in a chain on the falling edge of the sternocleidomastoid muscle. Their diameter is 2-4 cm nodes are not soldered to each other and to the surrounding tissues, dense little painful and not suppurate. After 1.5-2 weeks glands begin to decrease, being enlarged and sensitive to palpation for a few more weeks or even months.

Sometimes, the main manifestation of the disease is the loss of mesenteric (ileocecal) lymph nodes. It is accompanied by pain in the right iliac region, simulating an attack of acute appendicitis.

Hepatosplenomegaly is observed in almost all patients. Liver begins to increase with the onset of the disease, reaching a maximum 4-10th day, in some cases accompanied by jaundice of the skin and sclera. Enlargement of the spleen – also one of the early symptoms of the disease. Normalization of liver and spleen size comes at the end of the month of onset.

Some patients may be rash (roseolous, blotchy, erythematous), which is stored from 1-2 days to several weeks.

Crucial for the diagnosis of infectious mononucleosis are indicators hemogram and results of specific serological tests. At the height of the disease in the blood was leukocytosis (15-20*10^9/L), the increase in the number of mononuclear cells (lymphocytes and monocytes) with the appearance of atypical mononuclear cells, and a moderate increase in erythrocyte sedimentation rate (20-30 mm/h).
Serological diagnosis of infectious mononucleosis is based on the detection in serum of patients with heterophilic antibodies to red blood cells of animals (sheep, ox, horse, etc.).

**Acute leukemia.** Accompanied by high temperature (39-40°C) with chills and then a sharp general weakness, headache and dizziness. Often have nosebleeds, against pale skin and mucous marked hemorrhagic rash. Characterized by lymphadenopathy and hepatosplenomegaly.

Angina that develops because of a satisfactory condition at the beginning of the disease is the catarrhal and the harbingers of septic flow leukemia. Later, the disease becomes septicemic form, and the local process has necrotizing character. Plaque that forms on the surface of the necrotic tissue, becomes a dirty gray color. When removing it opens bleeding tonsil tissue defect with an uneven surface. Necrosis may extend to the oral mucosa, gums, throat.

The diagnosis is usually confirmed hematologic data. Leukocyte count varies from normal to hyperleukocytosis (100-200*10^9 / L). In hemogram dominated youngest power hematopoietic cells – hemocytoblasts, myeloblasts, lymphoblasts in the absence of transitional forms from young to mature. Characterized by severe anemia (red blood cell count drops to 1-2*10^12 / L, hemoglobin level – up to 25-40 g/l) and thrombocytopenia.

**Agranulocytosis.** Is not seen as separate entities that as a clinical and hematological syndrome characterized by the almost complete disappearance of granulocytes in the peripheral blood. Often cause agranulocytosis is receiving different drugs – dipyrone, phenylbutazone, sulfonamides, chloramphenicol, and others, it is important to consider in the differential diagnosis of angina with another origin.

Clinic agranulocytosis made by symptoms of acute sepsis and necrotizing tonsillitis. Disease begins acutely, the body temperature rises to 39-40°C, general weakness, pain in the throat. Necrotizing process is localized mainly in the tonsils, occasionally grabbing mucous gums, tongue, pharynx, larynx. Often on the tonsils, the bow, the tongue appears grayish dirty hard detachable thick filmy coating. Necrotic areas, purifying, form an extensive ulcer surface. Characterized by
sharp leukopenia (below $2 \times 10^9$/L), a significant reduction in the number of neutrophils, relative lymphocytosis (90% or more).

Differential diagnosis of angina, you should remember about the possibility of **tuberculous lesions of tonsils**, which amid the pale mucous membrane has its defect, ulcerate infiltrates with saped edges. Swallowing sharply painful. Tuberculous lesions of the pharynx usually arises against pulmonary tuberculosis, urinary tract, and the diagnosis is facilitated by specific bacteriological, serological and radiological investigations.

The differential diagnosis of angina should take into account the possibility of initiating tumor (cancer limfoepitelioma, clasmocytoma tonsils), especially with unilateral enlarged tonsils, presence of enlarged slightly painful lymph nodes around the angle of the lower jaw and the long process. The diagnosis is made after a biopsy and subsequent histological examination.

**Treatment**

In mild angina current medical therapies are carried out in an outpatient setting district physician (general practitioner), and for moderate and severe degrees of severity, patients should be hospitalized.

Indications for hospitalization:

a) **The general indications:**
   - Severity of the patient, suspected complications;
   - Recurrent angina;
   - Doubts about the diagnosis;

b) **epidemiological evidence:**
   - The inability of constant care and monitoring of patients;
   - The inability to isolate patients from closed and organized groups;

c) **The provisional indication:**
   - The development of angina in patients not vaccinated against diphtheria.

**General principles for the treatment of angina:**

- Sanitary measures;
- Diet N2 – in the acute period, with the transition to a diet N15, medical treatment;
- Causal treatment (systemic antibiotic therapy, topical administration of antimicrobial drugs);
- Pathogenetic therapy (detoxification, vitamins); - Symptomatic therapy (non-steroidal anti-inflammatory agents, topical anesthetics, antipyretics);
- Physiotherapy.

In the early days of the disease the patient is in need of bed rest, with the improvement in the state – in the ward. To avoid contamination of patient isolation is in a separate room or enclosure by his bed curtain or sheet, allocated a separate utensils, towels, etc.

The diet should be gentle, rich in vitamins C and B, containing sufficient amount of liquid in the form of tea, fruit infusions, jelly, mineral water, etc.

Systemic antibiotic therapy is aimed primarily at the main pathogen eradication angina – BHSA the following objectives:
- Reducing the risk of rheumatic fever;
- Preventing the spread of streptococcal infection;
- Reducing the severity of symptoms and their duration;
- Prevention of suppurative complications.

Empirical antibiotic therapy is indicated in cases when the following clinical criteria:
- Fever,
- Purulent exudate or raids in the gaps,
- Cervical lymphadenitis
- No cough.

In the absence of symptoms of systemic antibiotic therapy is prescribed only if positive blood culture or rapid strep swab on the antigen. In the case of clinical failure of empirical antibiotic therapy should also microbiological examination of smears from the surface of the palatine arches with the determination of the sensitivity of the selected agent. Drug of choice is antibiotics, active primarily against BHSA. Typically, antimicrobial drugs administered orally, however, with clinical symptoms of intoxication and shown parenteral antibiotics (Tabl. 2).

Benzatinpenitsillin i/m appropriate to appoint at:
- Questionable diligence patient compliance to the use of antibiotics;
- A history of rheumatic fever in the patient or next of kin;
- Adverse social conditions;
BHSA infection outbreaks in day care centers, schools, boarding schools, military units, etc.

Dentists in, given the dosage form in the form of a suspension, it is recommended mainly in young children in appropriate doses (Tabl. 2).

**Table 2 – Drugs of choice**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage Form</th>
<th>Schedule</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amoxicillin/clavulanate</td>
<td>per os for 30 min before a meal</td>
<td>375-625 mg 3 p/day or 1.0 g 2 p/day for 10 days;</td>
</tr>
<tr>
<td>Phenoxyethylpenicillin</td>
<td>per os for 30 min before a meal</td>
<td>0.25-0.5 g 4 p / day for 10 days;</td>
</tr>
<tr>
<td>Cefotaxime</td>
<td>i/m 1-2 g 3 p/day</td>
<td>3-5 days;</td>
</tr>
<tr>
<td>Benzatipenicillin</td>
<td>i/m once 2.4 million units;</td>
<td></td>
</tr>
<tr>
<td>Amoxicillin</td>
<td>0.5 g 3 p / day;</td>
<td></td>
</tr>
<tr>
<td>Cefadroxil</td>
<td>0.5 g 2 p / day.</td>
<td></td>
</tr>
</tbody>
</table>

**Alternative medicines:**
Intolerance to beta-lactam antibiotics (Tabl. 3).

**Table 3 – Non-beta-lactam antibiotics**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage Form</th>
<th>Schedule</th>
</tr>
</thead>
<tbody>
<tr>
<td>Azithromycin</td>
<td>0.5 g 1 p/day on day 1, then 0.25 g 1 p/day 1 hour before meals for 4 days;</td>
<td></td>
</tr>
<tr>
<td>Clarithromycin</td>
<td>0.25 g 2 p/day or 0.5 g of 1-2 p/day for 7-10 days;</td>
<td></td>
</tr>
<tr>
<td>Midekamycin</td>
<td>0.4 g 3 p/day for 1 hour before meals for 7-10 days;</td>
<td></td>
</tr>
<tr>
<td>Roxithromycin</td>
<td>to 0.15 g 2 p/day or 0.3 g 1 p/day for 7 to 10 days;</td>
<td></td>
</tr>
<tr>
<td>Spiramycin</td>
<td>by 3 million IU 2 p/day for 7-10 days;</td>
<td></td>
</tr>
<tr>
<td>Erythromycin</td>
<td>0.5 g 3 p/day 1 hour before meals for 10 days.</td>
<td></td>
</tr>
</tbody>
</table>

Erythromycin with oral macrolides are more likely to cause side effects, particularly on the part of the gastrointestinal tract.

**In recurrent tonsillitis BHSA**-drugs of choice are:
- Amoxicillin / clavulanate – 0.625 g 3 p / day;
- Cefuroxime axetil of 0.25 g 2 p / day (after meals), intolerance to beta-lactam antibiotics, alternative drugs are clindamycin or lincomycin for 10 days (in the above doses).

With frequent recurrences of sore throats, with adequate treatment of exacerbations of treatment showed an lincomycin 0.3-0.6 2 times per day i/m.

Systemic antibiotic therapy should be combined with a local appointment of antimicrobial drugs with a wide spectrum of action. Local prescribing necessary due to the viral etiology of some forms of...
tonsillitis, there are more resistant strains of bacteria, as well as unwanted side effects of antibiotics. Topical administration of drugs with a wide spectrum of antimicrobial activity, in some cases, it may be an alternative to traditional antibiotics. Active ingredients of topical preparations are usually one or more antimicrobial agents (antiseptics, antibiotics, sulfonamides), essential oils, local anesthetics, non-steroidal anti-inflammatory drugs. This may also include natural antiseptic (plant extracts, bee products) synthesized factors of nonspecific protection of the mucous membranes, which have antiviral, vitamins (ascorbic acid), and other drugs for the local treatment must meet the following requirements:

- A wide range of antimicrobial activity, ideally combining antibacterial, antifungal and antiviral activity;
- The low absorption rate of mucus;
- Low allergenicity;
- No irritating to the mucous membranes (Tabl. 4).

Table 4 – Drugs for topical application

<table>
<thead>
<tr>
<th>Drug</th>
<th>Application</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ambazon tablets</td>
<td>kept in the mouth to complete resorption of 30-50 mg 3 times per day, 7 days a course of treatment;</td>
</tr>
<tr>
<td>Benzydamine</td>
<td>gargling with 15 ml of 3 times per day for 7 days;</td>
</tr>
<tr>
<td>Biklotimol</td>
<td>keep the mouth to complete resorption of 1 lozenge 3 times per day, 7 days a course of treatment;</td>
</tr>
<tr>
<td>Hexetidine</td>
<td>gargling with 20-30 ml of 3-4 times per day for 7 days;</td>
</tr>
<tr>
<td>Gramicidin</td>
<td>tablets kept in the mouth to complete resorption of 3 g 4 times per day, 7 days a course of treatment;</td>
</tr>
<tr>
<td>Dioxidine</td>
<td>0.5% solution, gargling 4 times per day for 7 days;</td>
</tr>
<tr>
<td>Carbamide peroxide</td>
<td>0.25% solution, gargling 3 times per day for 7 days;</td>
</tr>
<tr>
<td>Miramistin</td>
<td>0.01% solution, gargling with 6-8 times per day for 7 days;</td>
</tr>
<tr>
<td>Nitrofurazone</td>
<td>0.02% solution, gargling every 2-3 hours for 7 days;</td>
</tr>
<tr>
<td>Polyvidone-iodine</td>
<td>Polyvidone-iodine</td>
</tr>
<tr>
<td>Fuzafungin</td>
<td>inhaled for 4 breaths 4 times per day for 7 days;</td>
</tr>
<tr>
<td>Chlorhexidine,</td>
<td>1% aqueous solution, gargling every 2-3 hours 1-2 days, then 3-4 times per day 5-6 days</td>
</tr>
</tbody>
</table>

In the angina treatment antibiotic fuzafungin (Bioparox) is widely used with a broad spectrum of antibacterial action and local
anti-inflammatory effect. Inhalation is done every 4 hours for 4-5 days.

Surface treatment of the tonsils with an antiseptic solution is indicated for Simanovskiy–Vincent’s angina.

Locally applied rinse with warm emollient decoction of sage and chamomile, as well as sodium chloride, sodium bicarbonate, potassium permanganate, boric acid, Frc, ethacridine lactate and hydrogen peroxide. Rinsing is performed after a meal. Good effect observed when used to rinse anti phytoncidic fees. For example, in the following recipe: St. John's wort (herb) – 2 parts, the common oak (bark) – 2 parts nettle (leaf) – 1 part, tansy (flowers) – 1 part pine (bud) – Part 1 , Licorice (root) – 2 parts buckwheat (grass) – Part 1, 2 tablespoons of the mixture boil in 1 cup of boiling water for 15-20 minutes, stirring occasionally contents, strain through a double layer of cheesecloth, lightly squeeze, cool 30-45 minutes. Apply as a warm gargle for 1/2-1/3 cup several times a day, 1/3 cup can be taken orally in the form of heat in the morning and evening.

To reduce the severity of pain shown topical anesthetics:
Diklonin keep the mouth to complete resorption of 8 1 tablet once a day;
Lidocaine 10% spray, 1-2 irrigation throat and tonsils 3 times a day;
Menthol, 2% alcohol solution, 2-3 drops to 1 liter of water, two inhalations twice daily.

To mobilize the body's defenses used vitamin therapy:
Ascorbic acid into 1 g 2 times per day 1-3 days, then 500 mg/day;
Multivitamin complexes into 1 capsule. (tablet) per day.

In severe intoxication, pain and fever prescribe non-steroidal anti-inflammatory drugs:
Diclofenac inside 50-100 mg/day;
Paracetamol inside of 1-4 g/day for no more than 3 days.

Physiotherapy. Applied to the neck cotton-gauze bandage or a hot compress. With a pronounced regional lymphadenitis appoint microwave therapy or current UHF SoLux.

Treatment of the patient is carried out under the control of blood counts, urinalysis, febrile reaction of the heart, etc., and the question
of discharge convalescent decided to work in the normalization of all these indicators.

**Evaluation of results of treatment.** Criteria for clinical cure:

- A normalization of body temperature
- Improvement in general well-being
- Disappearance of pain in the throat
- Regression of regional lymphadenitis

Repeated microbiological examination after antibiotic therapy is indicated:

- Patients with a history of rheumatic fever
- Outbreaks of tonsillitis BHSA in organized groups
- In the period of high incidence of rheumatic fever in the region.

Under the ineffectiveness of antibiotics, acute tonsillitis BHSA should understand conservation of clinical symptoms of the disease and the positive results of microbiological tests after treatment drug of choice, most often – penicillin. Failures in therapy may partly be due to a lack of executive patient adherence to the treatment (taking the drug immediately after meals, reducing the daily dose, early termination of treatment, and so on). In situations like this shows once i/m introduction benzatinpenicillin (2.4 million units). In other cases, we recommend a second course of treatment with one of the drugs used for recurrent tonsillitis, BHSA (amoxicillin/clavulanate, cefuroxime, axetil, lincosamides).

**Errors and unreasonable use**

Errors in the treatment of acute tonsillitis-BHSA:

- Undue preference to local treatment (including antimicrobials) at the expense of systemic antibiotics;
- Underestimation of clinical and bacteriological efficacy and safety of penicillins;
- The use of macrolides and lincosamides as means I have a number of patients with good tolerability of beta-lactam antibiotics;
- Reduction of antibiotics in clinical improvement;
- Wrong choice of antibiotics;

When BHSA-tonsillitis not shown application:

- Sulfonamides and co-trimoxazole (BHSA resistance, toxicity);
- Tetracycline (resistance BHSA);
- Quinolones and fluoroquinolones early (low natural activity).
Medical-social examination
To address the issue of health person who has had a sore throat must adhere to the following criteria:
- The complete disappearance of symptoms of tonsillitis;
- The lack of a regional lymphadenitis;
- No symptoms of the syndrome common infectious intoxication;
- Stable normalization of body temperature;
- Absence of pathological changes in the internal organs;
- Normalization of controlling blood count and urinalysis.

Period of temporary disability for those heavy physical labor or working in difficult conditions, with catarrhal angina – 7-8 days, and lacunar tonsillitis – 9-12 days, for other patients with catarrhal angina – 5-7 days, with lacunar and follicular – 9-10 days.

Rehabilitation
Rehabilitation of patients with angina is conducted in the rehabilitation clinic or directly district physician (general practitioner). Testimony to the direction in the rehabilitation are worsening comorbidities, complications.

Dispanserization and prevention
Persons who are ill with uncomplicated angina, are exempt from hard labor for 7 days, from the sport for 1 month and have to visit doctor 1 time every 10 days for 1 month.

Lesson 3. Acute bronchitis and pneumonia. Outpatient aspects of diagnosis and treatment, therapeutical approach, social medical examination, dispanserization, primary prevention

Acute Bronchitis
Acute bronchitis (AB) is inflammation of the trachea, bronchi, bronchioles, and mostly viral, less bacterial origin, with acute course and diffuse reversible lesion of mucosa mostly.

Epidemiology
Acute bronchitis is one of the most common respiratory diseases. Frequency of acute bronchitis among bronchopulmonary diseases is about 34.5%, which indicates an important socio-economic role of the prevention of the disease.
Etiology and pathogenesis
The most common causes of acute bronchitis are viral agents: influenza viruses (A, B), parainfluenza, respiratory syncytial virus infection, rhinovirus infection and other. Bacterial agents cause acute bronchitis more rare: Streptococcus pneumoniae, Hemophilus influenzae, Staphylococcus aureus, Moraxella (Branhamella) catarrhalis, more often in people with weakened immune systems and children. A common cause of AB are: Mycoplasma pneumoniae, Chlamydia pneumoniae, Bordetella pertussis (VartJett J., 1999).

Common pathogenic mechanisms:
• introduction of the infectious agent through the upper respiratory tract;
• fixation on the surface of the mucous membrane;
• reproduction and further spread of infectious agents;
• infectious and toxin mechanism;
• development in response to the introduction of infectious agents and local and total reaction of the body;
• suppression of local factors bronchopulmonary protection and suppression of general resistance of the body;
• restoration of disturbed functions, recovery.

Classification of acute bronchitis:
On the etiological reasons:
viral, bacterial, viral and bacterial, caused by chemical and physical influences, not the proximate.

On pathogenesis: primary, secondary.
The level of bronchial lesions: tracheobronchitis, bronchitis, bronchial lesions with medium caliber, bronchiolitis.

By the nature of inflammation: catarrhal, purulent.
For the ventilation: nonobstructive, obstructive.

By the nature of the flow: acute, prolonged, recurrent.
By the presence of complications: uncomplicated, complicated with the development of emphysema, respiratory failure, hemoptysis.
According to severity: mild, moderate, severe.

Common clinical manifestations of acute bronchitis:
1) clinic of acute respiratory viral infection with secondary acute bronchitis;
2) syndrome of common infectious intoxication of varying severity;
3) respiratory syndrome, which is characterized by shortness of breath;
4) soreness in the throat and in the chest, hoarseness and/or hoarseness;
5) occurrence of dry cough in the first 1-2 days of the disease, which can be and with sticky sputum;
6) cough can be rough, paroxysmal, "barking";
7) at 2–3rd day character of the cough is changing, becomes more soft and moist, improved sputum release (mucous character);
2) accession superinfection sputum is muco-purulent character, picking common infectious intoxication symptoms, bronchial obstruction of varying severity

Characteristics of clinical manifestations of acute bronchitis depending on the etiological factor causing it.

*Influenza*
In the first 1-2 days for symptoms of bronchitis otsytsstvuyut. Then comes the cough, in the discharge of sputum may be streaked with blood. Characterized nekrotic and degenerative changes in the mucous membrane of the trachea and bronchi, the large and medium-sized, often bronchitis proceeding like panbronchitis with severe peribronchitis. In the development of bronchitis has important value joining secondary microflora (superinfection). Body temperature - 38°C and above. The degree of severity – moderate and severe. TBC can reveal leukopenia with a left shift, and sometimes – monocytosis.

*Parainfluenza*
Poorly defined syndrome of intoxication, fever to 380S, respiratory tract damage – laryngitis, no hemoptysis, no conjunctivitis, mild or moderate severity, normocytosis.

*Adenovirus infection*
Adenoviral diseases peculiar to the presence of a pronounced exudative component with involvement in the pathological process of mucosa of respiratory tract, eyes, and involvement of the lymphoid tissue. General state suffers a little, higher temperatures can be long-term (7-10 days).
Respiratory syncytial virus infection
Most commonly affects the lower respiratory tract, upper suffer a little. Characteristic bronchial tubes lesions with the development of wheezing, shortness of breath that lasts 3-4 days. Syndrome is a common infectious intoxication is moderately expressed, often low-grade temperature. Characteristic part of joining the superinfection.

Respiratory mycoplasmosis
Characterized by lesions of all respiratory tract with predominantly involving small bronchi. No differences from the classical flow bronchitis, but very often complicated by pneumonia.

Respiratory chlamydiosis
Intoxication syndrome moderate, low-grade temperature, damage of respiratory tract – trachiobronchitis, no hemoptysis, no conjunctivitis, mild or moderate severity, normocytosis, increased ESR.

Whooping cough
First period - catarrhal, manifested rise bronchitis symptoms and has no distinguishing features. The second period is called convulsive and characterized – coughing fits, which occur suddenly as paroxisms and accompanied by reprises. Third period – terminal, bronchitis symptoms disappear.

Treatment
Bed rest, well ventilated area to avoid hypothermia, excessive drinking, antibacterial drugs in accordance with the type of pathogen, antipyretics, exectorals, anticough drugs (canceled in sputum presence), aminophylline, vitamines, physiotherapy (Solux, UHF).

Prophylaxis
hardening, preventing acute respiratory infections, rehabilitation of chronic infection foci, smoking.

Examination of disability
Easy – 5-7 days, medium-heavy and heavy cases – 8-14 days.

Dispanserization
AB recoverers without obstruction with respiratory failure II (6 months from the observation 1-3-6 months).
With obstruction with respiratory failure II – examination 3-6 times per year.

Community-acquired pneumonia
Pneumonia – an acute infectious disease, occurring with the
formation of the inflammatory exudate in the lung parenchyma and burning in X-ray examination, which was previously missing (there is no other known cause darkening by the appearance by X-ray examination of the lungs).

Diagnostically significant criteria accepted to pneumonia are following clinical signs and symptoms: the appearance on patient’s X-ray-grams new infiltration (progression or already had) in the first two days of the onset of clinical symptoms (all or several):

- Fever.
- Leukocytosis.
- Department of purulent sputum.
- Presence in the sputum, stained by Gram, more than 25 polymorphonuclear leukocytes in the field of view and less than 10 epithelial cells in a field of view (with microscopia with low magnification).
- Identification of the etiologically significant pathogen during microbiological research.

**Classification**

According to the existing classification of diseases and causes of death (ICD-10) with the infectious origin of the disease is distributed by type of pneumonia pathogens. These principles underlie the classification used in the past by N.S. Molchanov, supplemented in 1983 and by O.V. Korovina E.V. Gembitski that difficult to draw diagnosis to complete the survey and does not allow the practitioner to determine the tactics.

Consensus on pneumonia in 1995, and standards for diagnosis and treatment of patients with non-specific lung diseases provides physicians an opportunity to formulate a diagnosis even when the primary treatment for the patient's medical care, providing that:

- Community-acquired pneumonia;
- Hospital pneumonia;
- Pneumonia in immunocompromised.

The European Society of Pulmonology offered stratify patients into two main groups, each one includes several subdivisions.

1. *Ambulatory patients over pneumonia*
   - disease emerged in a region with high prevalence of resistant microorganisms;
• non-severe pneumonia in young people during the epidemia of M. pneumoniae;
• pneumonia in patients with COPD, in patients recently treated aminopenicillins, and in areas with high prevalence of H. influenzae producing beta lactamase.

2. Patients requiring hospitalization:
• pneumonia in the area with low presence of H. influenzae, producing of beta-lactamase;
• aspiration pneumonia, abscesses;
• Severe pneumonia (observation in the ICU).

The volume of lung tissue damage is usually (but not always) determines the severity of the disease. By volume of lesions are distinguished:
- Lobar pneumonia (takes a whole lobe);
- Segmental (occupying a separate segment);
- Bronchopneumonia (limited alveoli adjacent to the bronchus);
- Interstitial pneumonia (mainly affecting the interstitial tissue).

In practice is not always possible to accurately distinguish individual representation of types of pneumonia, so this classification is primarily aims past mortem examinations and, in addition, is used in radiology.

Most clinical practice guidelines distinguish the severity of pneumonia, as required hospitalization of patient:
1) does not require hospitalization (mild course);
2) required hospitalization (Severe)
3) must be treated in the intensive care unit (ICU) (Very Severe).

European Respiratory Society has proposed a set of clinical criteria, making hospitalization necessary:
- The presence of chest pain.
- The number of heart rate over 125 beats / min.
- Body temperature <35.0C or ≥40.0C.
- The number of breaths over 30 breaths/min.
- Cyanosis.
- Blood pressure <90/60 mm Hg.
- Suspicion of the presence of pleural effusion, and abscess formation.

In addition, hospitalization is necessary in cases when it is
impossible to conduct adequate treatment at home (the presence of the patient vomiting, poverty and low social protection, impaired memory and intelligence, in which it is impossible to implement medical recommendations).

*Hospitalization and treatment is indicated in patients in the ICU, with the following signs:*

**Severe respiratory failure:**
- The number of more than 30 breaths per minute.
- The need for mechanical ventilation.
- The rapid spread of pneumonia on X-ray data (increase in infiltration by 50% in 48 hours).

**Hemodynamic instability:**
- Blood pressure <90/60 mm Hg.
- The need for vasopressor drugs for more than 4 hours.
- Urine output <20 mL / h (in the absence of hypovolemia).

**Metabolic and hematological criteria:**
- Acidosis (pH <7, W);
- DIC.

**Acute renal failure, the need for dialysis. Severe failure of other organs and systems.**

At the same time experts of the European Respiratory Society allocate several groups of patients with relative indications for hospitalization:

1. Patients who had originally appointed antibiotic ineffective.
2. Patients at high risk of infection caused by gram-negative organisms or resistant strains of pneumococcus:
   - Over 65 years;
   - Patients from orphanages and homes for the elderly;
   - Persons strayushie alcoholism;
   - Patients have COPD, heart disease, neurological disorders, diabetes, kidney and liver failure;
   - Patients recently undergone acute viral infection of the respiratory tract;
   - Patients with oropharyngeal aspiration;
   - Patients who have recently hospitalized and given antibiotics.
3. Patients with risk factors for severe pneumonia.

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4. On laboratory and radiographic criteria: leukopenia ($<4\times10^9/l$) or leukocytosis ($>20\times10^9/l$), the symptoms of kidney failure (increased urea and creatinine blood), acidosis (pH $<7.3$), disseminated intravascular coagulation syndrome and also defeat more than one lobe of the lung on chest radiograph, the presence of pleural effusion or abscess formation.

**Differential diagnosis**

For practitioner is noteworthy differential diagnosis between pneumonia and cardio-vascular system, with forming stagnant changes in pulmonary circulation and the development of secondary pneumonia (heart failure, pulmonary embolism, pulmonary edema). Relevance of a correct diagnosis in these cases is determined by the need to make decisions quickly for urgent measures. At last, in 2-4% of cases in the presence of a light later infiltration identified is tuberculosis, which accounts for increased vigilance against the disease.

**The choice of drugs**

The first rule of use of antibiotics in patients with pneumonia is the earliest possible start of treatment. It is proved that the delay in the prescription of the first dose of antibiotics for pneumonia over 8 h led to increased mortality.

*Antibiotic treatment of pneumonia* are usually subdivided into **two main phases**:

1. *Empirical* - to identify the pathogen.
2. *After identifying the causative agent* – according to the etiological diagnosis and to the study of sensitivity to antibiotics.

However, so far the treatment of pneumonia remains largely empirical, since the start of treatment until the data from the lab runs 2-3 days and, in addition, about 50% of the cases reveal the true agent of inflammation ¬ inflammatory process in the lung fails.

Empirical antibiotic therapy should be based on clinical and epidemiological data (allows to suspect the presence of an impact ¬ arouser), the idea of the most common pathogens for the country pneumonia. A significant role is played by prior antibiotic therapy. If the previous treatment was ineffective (despite adequate doses and dosing regimens of antibiotics and the belief that the patient did receive a prescribed drug), can be oriented to "gaps" in the antibacterial spectrum previously used antibakterial drugs.
In recent years, antibiotic treatment choice for empirical therapy is increasingly dependent on regional spread of antibiotic-resistant strains of respiratory infections, the greatest importance is the prevalence penicillin-resistant strains of S. pneumoniae. When you select a product has to bear in mind, many antibiotics (such as cephalosporins III generation) cause induction of beta-lactamase production and the rise of antibiotic resistance.

In the treatment of the patient (in the clinical efficacy of the therapy) is possible to transfer a patient from parenteral to oral (sequential therapy) antibiotics. If the patient received an antibiotic, no analog for oral administration, it can be replaced with a similar drug spectrum.

Most clinical practice guidelines indicate that antibacterial treatment of pneumococcal pneumonia should last about 3 days after normalization of temperature.

At least 2 weeks should continue treatment for Mycoplasma, Chlamidia or Legionella pneumonia.

**Complications of pneumonia**

Complications include pneumonia, shock, respiratory distress syndrome of adults (ARDS), pleurisy, abscess formation, prolonged duration of pneumonia. Pneumonia can also be accompanied by the development of respiratory distress syndrome, and systemic inflammatory response syndrome (SIRS).

**Shock**

In patients with severe pneumonia tscheniem there are *two types of shock*:

- true hypovolemic shock with insufficient consumption of liquid, with increased its loss manifests a decrease in cardiac output and a compensatory increase in total peripheral vascular resistance (TPVR);
- Septic shock (dilation occurs when resistance vessels exposed to endotoxin of gram-negative organisms), in this case a reduction of TPVR and compensatory increase in cardiac output.

*Septic shock* is often diagnosed only when a violation of consciousness. Therefore, patients with severe pneumonia need frequent (every 1-2 hours) a tonometry, diuresis control. Reduction of blood pressure (systolic blood pressure <90, and diastolic blood...
pressure <60 mm Hg.) with decrease of urine output (<20 ml/h) show the development of shock.

The volume of fluid therapy is determined by the severity of hypovolemic shock. At a septic shock can not expect a quick positive effect of infusion therapy (as in hypovolemic).

With the lack of effectiveness of infusion therapy can be recommended the appointment of dopamine or dobutamine. The use of corticosteroids in septic shock is not currently recommended.

*Respiratory distress syndrome of adults.*

Basic pathogenetic mechanism is massive propotevanie plasma and blood cells in the interstitial and alveolar spaces, which leads to hypoxia not corrected by inhalation of oxygen (exudate occupies almost the entire surface of the lungs), and the subsequent development of inflammation and outcome in severe interstitial fibrosis with the development of severe restrictive respiratory failure. Clinically indicated pronounced shortness of breath, which develops in 1 to 2 days after the initial manifestation of the disease (severe pneumonia, aspiration of gastric contents, trauma of the chest, shock, burns).

*Lung abscess*

The most common pathogens cause destruction of lung tissue, are Staphylococcus, Streptococcus, *K. pneumoniae*, anaerobic flora. Average period of abscess formation in pneumonia is 5-7 days, and with pneumonia, caused by *K. pneumoniae*, – 3-4 days. Maintaining a high temperature and a massive phlegm allow suspect abscess.

When abscess formation is necessary to increase the dose of antibiotics, improve the drainage function of the bronchi, through postural drainage and bronchoscopic redevelopment. In cases when it is impossible to achieve drainage of abscess, surgical treatment. Wherever abscess formation should be carried out a series of sputum for TB.

*Pleurisy*

Treatment of patients with non-infected pleural effusion with no tendency to increase its volume is usually conservative. It is a condition bacteriological and cytological examination of fluid after the diagnosis of pleural puncture with a fine needle.

*Systemic inflammatory response syndrome*

Pneumonia may be accompanied by severe sepsis, which in this case should be considered as its complications. According to the
consensus of the American College of Physicians, and the Society of Critical of Medicine, the term sepsis is systemic response to infection. It is believed that in addition to bacteremia in base of septic states is a systemic inflammatory response, and the presence in the blood of microorganisms no longer plays a key role.

A key feature for the diagnosis of sepsis is fever, training conditionality presence of inflammatory mediators in the blood (especially prostaglandin E2). In sepsis (and less frequently in SIRS) in elderly patients with violation thermoregulatory mechanisms may experience hypothermia. During the early stages of sepsis is usually accompanied by hyperventilation with the development of respiratory alkalosis and fatigue of respiratory muscles.

On the evidence of multiple organ failure: – rady-and tachypnea (more than 30 min.)
- Shock, bradycardia, ventricular arrhythmias, acute myocardial infarction;
- Oliguria, increased serum creatinine and urea;
- Hyperbilirubinemia (>50 mmol/l), increased AST and ALT 2 times or more;
- ICE;
- Stress ulcers, bleeding from the gastrointestinal tract, intestinal perforation;
- Impaired consciousness.

Treatment of sepsis is a complex task.

**Medical and social examination of pneumonia**

Acute pneumonia causes about 40% of all days of temporary disability (TD) with nonspecific lung diseases.

It is preferable in treatment of acute pneumonia in hospital to continue rehabilitation in polyclinic.

*Mild form of acute pneumonia* observed with focal lesions. Subjective and objective signs are moderate, slight intoxikation, the temperature is not above 38 degrees, slight tachycardia, a trend to some reduction in blood pressure, changes in the peripheral blood of an inflammatory nature are not very pronounced, the focal inflammatory infiltration at X-raygram. TD is 17-20 days, including 15 days of hospital treatment.

*The average severity of pneumonia* is determined by large pockets
or share with severe lung lesions and fever and intoxication syndromes, significant reaction from the cardiovascular system as tachycardia and lower blood pressure, a sharp increase in white blood cell count, erythrocyte sedimentation rate and acute phase indicators in serum. TD is at least 28 days, followed by work arrange by MCC with the exception of adverse factors at work for a period of 3-4 weeks.

*Severe form of acute pneumonia* observed in polysegmental, bilateral pneumonia complicated by suppuration, etc., is grounds for immediate hospitalization and intensive comprehensive treatment respectfully in specialized hospitals or departments intensive therapy unit (ITU). TD duration ranges from 40-45 days with favorable course up to several months if necessary surgery. Patients working in adverse weather conditions associated with dust, exposure to irritant gases, with a significant, even of a nonconstant physical strain should be temporarily transferred to conclusion of MCC to other work, excluding the impact of these adverse factors.

Lesson 4. Ischemic heart disease: outpatient aspects of diagnosis of various forms of ischemic heart disease, therapeutic approach, medical-social examination, dispanserization, primary prophylaxis. Treatment of angina pectoris. First aid in angina attack on an outpatient basis

**Ischemic (coronary) artery disease**

*Ischemic (coronary) heart disease (IHD)* remains dominant both in prevalence and in mortality in our country as well as in the most economically developed countries.

**Diagnosing, classification and clinical forms of angina**

For the diagnosing of chronic ischemic heart disease, a district physician needs to know:

- Features of pain in angina pectoris;
- "Atypical" versions of angina pectoris;
- Asymptomatic clinical forms of coronary artery disease;
- Risk factors;
- How to eliminate disease, simulating IHD (non-coronary cardialgia).
District physician receives information in favor of a presumptive diagnosis on the basis of physical examination.

Laboratory and imaging studies can confirm the assumption of the presence or absence of IHD and its complications.

**Complaints:** most often retrosternal pain "during a physical exertion", often pain irradiation in the left shoulder, left arm. If angina occurs during the examination, you can see orthopnea (in this position venous flow can be reduced as well as volume and tension of ischemic ventricular wall of heart).

Diagnosing of angina is primarily clinical, based on a thorough qualitative description of pain. In order to avoid subjectivity in the interpretation of pain, WHO Expert Committee on Cardiovascular proposed the following diagnostic criteria for pain, typical for angina:

1. *The nature of pain* – compressing or crushing.
2. *Localization of pain* – retrosternal precordium to the left edge of the sternum.
4. *Duration of pain* – no more than 10 minutes.
5. *Rapid and complete effect* of nitroglycerin.

Therapist must remember that pain which is not corresponding to the criteria, especially the last three, can not be regarded as angina, and requires further examination to determine the cause of the pain.

Bear in mind that when variant angina attacks occur only at night during sleep, which is associated with the occurrence of vasospasm and ischemia during an attack of parietal thrombosis. Recorded during angina attack ECG demonstrates the presence ST-segment elevation, sometimes in the form of single-phase curve.

**Examination of the skin:** during angina attacks, resulting in peripheral vasoconstriction integument becomes usually grayish. Skin examination in the interictal period reveals features typical for coronary heart disease: xanthisms and xanthelasma, located in the inner corner of the eye, yellowish bumps or growths tuberous xanthisms on the elbows or knees, flat xanthisms on the skin of chest and neck.

In young people early symptoms of angina, as well as atherosclerosis is presence of corneal arcus (arcus senilis).

Examination and palpation of vessels, heart rate, determination of boundaries of the vascular bundle and heart, listening heart and
great vessels, evaluation of sonority tone and heart rhythm are very important.

It should be kept in mind that there are atypical forms of angina. There are the following equivalents of angina: arrhythmic, asthma, peripheral. Arrhythmic manifestation of angina can be the equivalent of ventricular arrhythmias, atrial fibrillation, paroxysmal supraventricular tachycardia. At an altitude of ischemia transient slowing down of atrioventricular and intraventricular conduction is observed. Usually these rhythm and conduction disturbances are temporary, and after disappearance of ischemia they pass. In arrhythmic equivalent angina transient arrhythmias are often associated with physical activity, may be successfully arrested not by antiarrhythmic drugs, but by nitroglycerine and other antianginal drugs.

With extensive areas of ischemia of transient nature, especially following myocardium, it may cause asthma equivalent of angina with developing acute congestion in lungs and symptoms of cardiac asthma. Clinical manifestations of peripheral angina equivalent are of different intensity not at the normal irradiation with typical angina. Peripheral angina equivalent can be characterized by a sensation of heartburn when walking fast and it can simulate gastric and duodenal ulcer, chronic gastritis. Angina in some patients is manifested by sudden bouts of muscle weakness and numbness of the left arm, 4 or 5 fingers of the left hand. Shortness of breath may often be the equivalent of angina, as well as coughing and coughing when walking fast.

Great practical importance is now given to asymptomatic myocardial ischemia. It includes episodes of transient, reversible myocardial ischemia, which does not manifest clinically. Since in most cases asymptomatic IHD is not detected in vivo, it often can be complicated by myocardial infarction and it can cause untimely and sudden cardiac death. Asymptomatic ischemia can occur without other forms of angina, and along with either stable or unstable angina. Diagnostics of asymptomatic myocardial ischemia is based on carrying out daily electrocardiographic monitoring, as well as on bicycle exercise, and other functional tests.

"Syndrome X". Angina with intact coronary arteries was first described in 1973 by H. Kemp. This syndrome can be found in patients with typical retrosternal pain, positive exercise test and
angiographically with no signs of coronary spasm. Pathogenesis of ischemia-induced functional or structural abnormalities of the coronary microcirculation is important as well as metabolic disorders, provoking disturbances in consumption of energy substrates myocardium. ECG results during stress testing are significant as well. Almost always there are ischemic ST-changes, which do not differ from those with angina pectoris due to stenotic coronary atherosclerosis. However, patients with Syndrome X have higher exercise capacity. Patients with "Syndrome X" have a very favorable outlook in life. Treatment is mostly medicamental. The same drugs as in classic angina are used. Along with xanthine drugs are widely used, e.g. aminophylline (euphylline).

There are some specific features of IHD in the young and in the elderly. More often they have asymptomatic or oligosymptomatic IHD. Specific features of coronary artery disease in the elderly include a gradual onset, less clear emotional coloring of angina pain longer in comparison with the middle-aged. Short-term pain should be considered to be angina, when it occurs after emotional stress or going alone after receiving antianginal drugs. The elderly are observed painless forms of IHD, accompanied by various equivalents of pain significantly more commonly. Sometimes not pain bothers patients, but difficulties in swallowing, feeling of food pressure and stoppage in the esophagus and in the chest.

In some cases, pain occurs in the supine position, the so-called angina de cubitas, when pain stops in the sitting position.

Not all kinds of chest pain should be related to IHD. It requires careful differential diagnosis.

Diagnostics of angina in young patients can be complicated by the fact that doctors are psychologically unprepared for the fact that angina and heart attack may occur in 20-30 year-old patient.

There is a wrong assumption that young women cannot have angina. The physician should make questioning keeping in mind the following risk factors: hereditary hyperlipidaemia, diabetes mellitus, hypertension, hormonal contraceptives, spontaneous dysovarian disorders, smoking, professional activity increases the risk of heart disease in women. Complaints and a history of women may be masked by cardialgias.
Spontaneous (singular) angina is known as Prinzmetal’s angina. During spontaneous angina anginal attacks usually occur at the same time, often at night or early in the morning. They are connected to a local spasm of the coronary artery, accompanied by ST-segment elevation on the ECG. Spontaneous angina is often associated with angina.

**Instrumental diagnostising.**

**Electrocardiography.** The diagnostic value of ECG in patients with angina is not significant, as for most of them it was recorded at rest and out of pain attack. ECG should be evaluated carefully taking into account the clinical picture and the possible dynamics. For signs of coronary insufficiency velo ergometer test is carried out (VET).

**Indications for VET:**
1. Detection of latent forms of IHD.
2. Atypical chest pain
3. Presence of non-specific ECG changes in the absence of pain or in its atypical character.
4. Detection of exercise tolerance in patients with established coronary heart disease
5. Diagnostics of coronary artery disease with noECG changes.
7. Professional selection.

**Contraindications for VET:**
1. Aortic stenosis and congenital heart disease.
2. Atrial fibrillation and other conditions associated with embolism threat.
3. High arterial hypertension (200/120 mm Hg).
4. Stroke, disorders of the vestibular system.
5. Severe heart failure.
6. Early ventricular arrhythmias, such as R-on-T
7. Frequent polytopic extrasystole.
8. Bundle-branch block.
9. II-III degree atroventricular block
10. Unstable angina.
11. Acute myocardial infarction (at least 2-3 months).
Relatively contraindicated for VET:
1. Asthma and chronic non-specific lung diseases along with another respiratory failure.
2. WPW syndrome.
3. Venous thrombosis of the lower limbs, retinal detachment, high myopia.
5. Blindness, deafness, and concomitant difficulties in contact with the patient.
6. Orthopedic disorders.
7. History of complex cardiac arrhythmias or syncope.
8. Fever.

Temporary contraindications for VET:
1. Acute exacerbation of chronic infectious diseases.
2. Sleepless night, heavy fatigue.

Interpretation of the VET results.
Exercise tolerance:
- High threshold load >900 kgm/min;
- Medium threshold load 600-900 kgm/min;
- Low threshold load <600 kgm/min.

VET is positive if during the test itself or during the recovery there could be observed one or more of these criteria. Angina attack is a clinical sign specific for IHD, ischemic movement of ST-segment up or down by 1 mm or more is an electrocardiographic sign. If there are only positive ECG signs, it indicates asymptomatic myocardial ischemia.

VET is negative if the age submaximal heart rate is reached and there are no signs of positive test.

Indications for ECG Holter monitoring:
1. Identification of atypical anginal attacks.
2. Complaints of patients on cardiac arrhythmias, undocumented by ECG.
3. Detection of asymptomatic arrhythmias.
4. Syncope of unknown etiology
**Pharmacological tests** with administration of the sample with drug-controlled transient myocardial ischemia (with dipyridamole, isadrin) is used in conjunction with other research methods to assess the coronary circulation and the functional state of the myocardium, especially in patients who, for one reason or another cannot be done electrocardiographic exercise test (disease joints, physiotherapists, orthopedic defects, etc.). Nowadays these tests are not performed frequently.

**Other ECG stress testing:**
- Psycho-emotional test;
- Hyperventilation;
- Cold sample.

**Echocardiography** identifies regions of hypo/ akinesia, respectively region of myocardial ischemia, or focal cardiosclerosis, sealing structures of aortic and mitral valve.

**Transesophageal atrial electrical stimulation (TAES)** reveals evidence of myocardial ischemia during exercise. The criterion for a positive test is the appearance of horizontal or ischemic obliquely descending (2 mm) ST-segment deviation at the height of stimulation. Samples are indicated for patients who can not be tested with exercise due to comorbidities and detraining.

**Radionuclide myocardial imaging** in diagnostics of ischemic heart disease was the most practical method for determination of myocardial perfusion with 201 Tl; radionuclide myocardial scintigraphy with 99m TL-pyrophosphate to detect focal myocardial changes.

**X-ray studies** include computed tomography, selective coronary angiography, left ventricular angiography.

**Selective coronary angiography** is carried out in typical stenocardiac pains, but with negative or questionable electrocardiographic stress tests results to confirm the diagnosis of coronary artery disease and to assess the location and extent of coronary artery disease.

Angiography helps to determine a method of treatment (conservative or surgical), to assess the severity of the patient’s state, the patient's ability to work, and the prognosis. Diagnostically significant for angina is narrowing of artery lumen more than 75% and occlusion of blood vessels.
Classification of coronary artery disease (WHO):
1. Primary cardiac arrest.
2. Angina.
   2.1. Angina:
      2.1.1. first effort angina;
      2.1.2. stable;
      2.1.3. progressive;
   2.2. Angina at rest (spontaneous angina)
      2.2.1. specific form of angina (Prinzmetal’s).
   3.1. Acute myocardial infarction:
      3.1.1. definite:
      3.1.2. possible:
   3.2. Myocardial infarction.
4. Heart failure.
5. Arrhythmia.
6. Postinfarction cardiosclerosis

To assess patients with stable angina (SA) there is Canadian classification that divides patients into 4 functional classes:

I class. Regular physical activity does not cause angina. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea, or anginal pain. In these patients angina is latent.

II class. Minor limitation of usual activity. Angina occur when walking and climbing stairs, walking fast uphill, walking or climbing stairs after meals, during emotional stress or in the first hours after waking up. Mild angina.

III class. Marked limitation of ordinary physical activity. Angina occurs on walking 1 to 2 blocks on level ground or climbing 1 flight of stairs at a normal pace in normal conditions. Moderate angina.

IV class. Inability to carry on any physical activity without discomfort. Symptoms of heart failure or the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased. Severe angina.

Indications for hospitalization of patients with stable angina

Patients with stable angina I functional class (FC) require hospitalization only when the disease is of unclear nature (stable or
unstable) and emerges for the first time. They need more precise diagnostics in atypical clinical presentations and when there is no possibility to carry out stress tests, test for lipid metabolism, microcirculation, etc. on an outpatient basis, as well as in combination of angina with hypertension to clarify its origin.

Patients with **stable angina II-IV FC** are hospitalized in the following cases:

- when the disease has atypical course and progression, for diagnostics of functional class of angina pectoris;
- for patients resistant to outpatient antianginal therapy, to define the individualized choice of antianginal drugs, to identify pathogenetic features of the treatment;
- when coronaryography study and surgical treatment is needed;
- when there are difficulties in administration of therapy in patients with angina pectoris in combination with hypertension, persistent arrhythmias, II-III degree heart failure;

**Non-drug treatments for IHD.**

**Physiotherapy.**

Physiotherapy treatment is ucarried out for the treatment of patients with coronary artery disease. Different types of physiotherapy may be useful: electric, medicine electrophoresis, electro-magnetic fields in the UHF band (UHF-therapy), low-frequency alternating magnetic field (AMF), sinusoidal modulated currents (SMC).

**Balneotherapy.** Bathing in natural mineral waters and their analogues prepared artificially: carbon dioxide, hydrogen sulfide, radon, oxygen and minerals. Bathing should be limited for patients with postinfarction cardiosclerosis, I-II FC, FC III angina (in CHF no more than Stage I, exercise capacity not less than 50 watts and in the absence of complex arrhythmias).

**Contraindications:** Stable angina of FC IV with frequent attacks of angina at rest, II degree heart failure, complex arrhythmias, cardiac aneurysm, consistently high numbers of blood pressure (over 180/100 mm Hg).

**Therapeutic physical exercises.** Physical training is indicated for patients with angina I-II FC. Patients with III FC can be indicated as well, but with certain restrictions. The same goes for patients receiving
antianginal drugs. Physical training for FC IV patients is contraindicated. They can be recommended easy exercises: morning gymnastics, walking slowly along with active antianginal therapy. Patients should be informed that if during exercise they have pain in chest, irregular heart function, fatigue, shortness of breath, load should not be continued. At the first angina attack nitroglycerin should be taken.

**Sanatorium treatment.** Indications: patients with I-II FC angina without severe cardiac rhythm, and I degree heart failure. Patients with FC III angina can be sent only to the local sanatorium cardiology. Spa treatment is contraindicated for FC IV patients.


Among other methods of non-drug effects on patients with IHD external laser irradiation of reflex zones on the skin is used in the polyclinic, as well as intravascular laser and ultraviolet blood irradiation, ultrasound, magnetic effect on the heart, etc.

**Drug therapy of chronic ischemic heart disease**

**Angina attack treatment**

Effective relief of angina attack can prevent premature death in patients with ischemic artery disease. Angina attack is resultant of reversible myocardial ischemia. In most cases the seizure lasts about 1 to 10 minutes. Longer reversible ischemia may be referred to as "prolonged attack of angina." If anginal attack lasts more than 20 minutes, there is a risk of irreversible, necrotic changes in the ischemic area. Even with its reversibility, clinical ischemia can more or less lead to microdamage of miocardioocytes and their organelles. In addition, the anginal attack which begins as light and harmless may become severe and result in irreversible ischemic necrotic changes, i.e. myocardial infarction, and sometimes adverse outcome, ending in sudden death. Therefore, the more quickly the attack is stopped, the better both the immediate and long-term prognosis is.

The doctor should clearly explain to the patient the importance of fast and complete relief of angina attack, explain that with this disease, patient may live for tens of seconds, when "the first episode is the last," to tens of years, and it depends not only (and not so much) on the doctor, but on the patient too. The physician must teach the patient how to arrest anginal attack on their own quickly and effectively.
The physician should explain:
- the purpose of nitroglycerin emphasizing that it is irreplaceable, so you should always have it with you;
- how to use nitroglycerin, explain to them that treatment should be started with lower dosages (1/4 or 1/5 or 1/2 tablet) as the possible side effects may frighten patients, and they will avoid taking this medicine. Tablets and capsules must dissolve for 10-20 seconds. Feeling of warmth or burning on site of absorption of the drug and the occurrence of tremors in the head indicate that the medicine has effected;
- that they can get used to nitroglycerin and that its side effects (headaches) are gradually reduced. To reduce this discomfort simultaneous validol intake can be recommended, and these side effects may be minimized by taking aspirin or acetaminophen;
- that regardless the fact angina attack is short, light or severe, patients need to take nitroglycerin. The sooner the medicine will be taken, the more relief will come;
- that since there is no effect for 5 minutes after taking nitroglycerin, one more dose should be taken, but not more than 2-3 times. If there is no effect within 5-10 minutes after the re-taking nitroglycerin should be resorted to calling emergency doctor for administration of analgesics;
- that absorption of oral antianginal drug starts in 40-50 seconds and lasts up to 20 minutes;
- that to relieve angina attack at home or at hospital, besides nitroglycerin tablets they can use nitroglycerin spray (isoket, nitromint) spraying the solution under the tongue, without inhaling it;
- that to relieve angina attack oil solutions in capsules may be used, which are of slower action and less convenient; for more rapid effect, they may crush with the teeth;
- when there are no forms of nitroglycerin of fast action, sublingual intake of nitroglycerin of prolonged action is allowed in angina attack. In this case, they should be chewed. For those with poor nitroglycerin tolerability or lack of its effect it is recommended to take sublingual isosorbide dinitrate (nitrosorbidi) of 5 or 10 mg, molsidomine (dilasidom) 2 or 4 mg. This antianginal effect begins in about 3-3.5 minutes, i.e. later than nitroglycerin, but for a longer period;
- clinical observations show that sublingual isosorbide or
dilasidom is better tolerated by patients of older age groups. But even in these cases it is necessary to bear in mind that the dose of nitroglycerin in prolonged forms is much more, therefore complete and fast absorption into the oral cavity in patients sensitive to nitroglycerin may result in adverse consequences to sudden drop in blood pressure and collapse. In case of drop in blood pressure, the patient should quickly swallow the drug. Sublingual nitroglycerin should not be used, as it is only to prevent strokes.

**Adverse effects of nitrates:**
Headache, hypotension, and tachycardia.

**General contraindications for the use of nitrates:**

**Absolute contraindications:**
- allergic reaction to nitrates, hypersensitivity to them;
- marked hypotension (systolic blood pressure of 95-100 mm Hg, diastolic blood pressure below 50-60 mm Hg), collapse, shock;
- myocardial infarction, flowing with severe hypotension, collapse, shock;
- noncurable hypovolemia;
- pericardial constriction and cardiac tamponade (intravenous);
- bleeding in the brain, severe brain injury, cerebral ischemia (intravenous);
- toxic pulmonary edema;
- angle-closure glaucoma with high intraocular pressure (with open-angle glaucoma there are no contraindications).

**Relative contraindications:**
- increased intracranial pressure;
- asymmetric hypertrophic cardiomyopathy;
- severe aortic or mitral stenosis (when it is necessary to maintain filling pressure or volume of the left ventricle);
- poor individual tolerance due to increased sensitivity to nitrates (acute headache):
  - severe hypotension and tachycardia;
  - severe anemia;
  - marked aortic stenosis (decreased left ventricular filling pressure, extreme hypotension);
  - first 3 months of pregnancy and during lactation.

Currently, **nitrates** are presented mainly by three drugs:
nitroglycerin, isosorbide dinitrate and isosorbide-5-mononitrate. There are no fundamental differences in their pharmacological effects. More important is classification of nitrate according to their action: short-acting (<1 h), moderately long-acting (<6 hours) and significant long-acting (6-16 hours, sometimes up to 24 hours).

Physicians should be aware that during the use of long-acting nitrates in patients for 6-8 weeks weakening antianginal effect may occur because of addiction. To reduce this effect nitrates are administered intermittently in 6-8 hours, using drugs with significant prolonged effect. When antianginal effect is weakened, a break of 3-7 days is needed, after which the sensitivity is usually restored. At the break, the patient should be prescribed one of the drugs from the group of calcium channel blockers if the patient tolerates monotherapy nitrates or molsidomine.

**β-blockers**

The second group of drugs that are widely used for the treatment of various forms of IHD include beta-blockers (β-AB). These drugs solve three problems:

- Improve prognosis of patients with previous myocardial infarction.
- Have marked anti-anginal effect.
- Have antiarrhythmic effect.

Therefore, β-AB should be prescribed to all patients with stable angina, and especially to people with a history of MI. Preference should be given to cardioselective β-AB. They have fewer side effects than non-selective β-AB and their effectiveness has been proven clinically.

District physician has to remember:

1. All β-AB, regardless of selectivity, intrinsic sympathomimetic activity, pharmacokinetics, have a relatively long clinical effect in patients with angina in adequately selected dose.

2. The optimal dose of β-AB is individual and it is based on clinical effect: reducing the number of anginal attacks or their complete disappearance and improving exercise tolerance.

3. Dosage of β-AB requires monitoring of heart rate and blood pressure levels, but moderate bradycardia may be observed, as often
only after deceleration of heart rate to 60-55, sometimes up to 50-52 beats per minute anginal effect of the drug becomes evident. Not the degree of bradycardia, but its tolerance is important. Consequently, moderate sinus bradycardia is not a contraindication for use β-AB, as decreasing of heart rate in response to their reception is not marked in all patients. The same applies to the decrease in blood pressure, although the overall β-AB have little effect on its normal index.

4. In case of bradycardia atropine may be used. Vagal tone will fall, without affecting β-AB, accompanied by heart rate acceleration.

5. Treatment of β-AB should be started with small doses. Metoprolol 12.5-25 mg twice a day, 15-20 minutes before eating. After assessing effect of the drug on the patient, dosage can be or must be increased every 3-5 days till clear improvement. If necessary, the daily dose may be increased. We must remember that the optimal dose of metoprolol is 100-150 mg daily. Maximum beneficial effect of the drug is in 1.5-2 weeks of treatment. For Bisoprolol (Concor) the initial dose may be 2.5 mg once a day, with a gradual increase of dose to 2.5-5 mg each 5 days to 10 mg per day.

Nebivolol (Nebilet) is a special β-AB as it has vasodilating cardioselective release with NO effects. Nebilet has a positive effect on metabolic parameters:
- reduces the level of glucose in hypertensive patients with diabetes and patients with impaired carbohydrate tolerance;
- increases glucose utilization and improves insulin sensitivity in patients with type 2 diabetes;
- normalizes levels of total cholesterol and HDL-cholesterol and LDL;
- Average daily dose is 2.5-5 mg.

6. The duration of the β-AB is determined individually according to the clinical course of the disease and tolerability. Treatment if effective may be long (months, years).

7. β-AB withdrawal should be done gradually, since fast withdrawal can lead to severe reactions in the form of increased frequency of angina, myocardial infarction, occurrence of cardiac arrhythmias and even sudden death.

8. β-AB can be combined with other drugs, including nitrates, calcium antagonists and diuretics.
9. β-AB should not be administered with calcium antagonists of the group verapamil (Isoptin, finoptin) due to the increased negative inotropic effect and a possible sharp oppression atrioventricular conduction.

10. We must remember that prolonged use of β-AB leads to changes in lipid metabolism (increased atherogenic lipoproteins).

**Calcium channel blockers**

The third group of antianginal agents widely used in the treatment of patients with coronary artery disease is calcium channel blockers.

Mechanism of their action is complex and mainly confined to the following:

1. They decrease preload by reducing venous inflow.
2. Decrease afterload due to peripheral arterial vasodilation.
3. Alter diastolic relaxation.
4. Increase coronary blood flow and coronary perfusion.
5. Prevent coronary vasospasm.
7. Protect myocardium from hypoxia.
8. Have anti-atherogenic effect.
9. Do not become addictive.

**Classification**

Two groups of calcium channel blockers (CCB):

dihydropyridines (nifedipine 10-20 mg orally or sublingually (nowadays it is used only for treatment of hypertensive crisis), amlodipine (Norvasc), felodipine 5-10 mg once a day, isradipine 2-5 mg twice a day, lerkanidipine 10 mg 1 tablet once a day).

Non-dihydropyridines: phenylalkylamine (verapamil 40-80 mg, Isoptin 40-80 mg 2-3 times a day, Isoptin-SR 240 mg 1-2 times a day, finoptin 40,80,120 mg 2-3 times a day);

benzodiazepines (diltiazem 30.60 mg 3 times daily, diltiazem – retard 180 mg 1-2 times a day, altiazem 120 mg 1-2 times a day).

The mechanism of CCB action varies considerably. In the properties of dihydropyridines peripheral vasodilation is dominated by effect of non-dihydropyridines: – negative chronotropic and inotropic effects. Non-dihydropyridines: CCB used instead of β-AB, in cases when the latter are contraindicated (COPD, marked peripheral artery disease of the lower limbs).
All CCB are appointed only in the form of second-generation long-acting formulations used once per day.

**Angiotensin-converting enzyme (ACE) inhibitors**

Symptoms of stable angina or myocardial infarction are indications for ACE inhibitors in patients with chronic coronary artery disease. For those with poor tolerability these drugs are replaced by angiotensin II receptor blockers (ARB). Studies have shown that the use of ramipril (tritatse) and perindopril (prestarium) reduces the occurrence of cardiovascular complications (CVC), including the risk of myocardial infarction and death from cardiovascular complications. ACE inhibitors should be administered by patients with angina pectoris in combination with arterial hypertension (AH), diabetes mellitus (DM), heart failure (HF), asymptomatic left ventricular (LV) dysfunction or myocardial infarction. Treatment starts with low doses (2.5 mg once a day), gradually increasing the dose each 5 days under the supervision of AH until a daily dose of 10 mg, as this dose was the most effective in terms of normalization of hemodynamic parameters of the cardiovascular system (CVS).

**I_f channel inhibitor**

Recently created a new class of antianginal drugs, I_f channel cells sinus inhibitor selectively slows sinus rhythm. Their first representative ivabradine (Procoralan, Coraxan) showed marked antianginal effect comparable to the effect of β-AB. Appointment of ivabradine for patients with stable angina, left ventricular dysfunction and heart rate > 70 beats/min reduces the risk of MI and improves revascularization. Procoralan is prescribed 5 mg 2 times a day, and a month – 7.5 mg two times a day.

**Activators of K^+ channels:** Ikorel (nicorandil) 10-20 mg twice a day; it has properties of three classes of medications: nitrates, calcium channel blockers and cardioprotectors – vasodilative effect without steal effect, reducing pre-and afterload by venous and arterial vasodilation, and cardioprotection (myocardial preconditioning). Can be used as monotherapy or in combination with other antianginal drugs.

**Antiplatelet drugs** are recommended for all patients with stable angina pectoris with no contraindications (peptic ulcer disease, gastritis, stomach or intestinal bleeding, blood system diseases, hypersensitivity to the drug).
The main anti-thrombotic drug for CVC is acetylsalicylic acid (ASA). The optimal dose of aspirin is 75-150 mg per day. Regular use of aspirin by patients especially with myocardial infarction reduces the risk of recurrent MI. It can provide additional security preparations ASA enteric-coated shell (polokard) or aspirin products with antacids (cardiomagnil).

Among other antithrombotics drugs Clopidogrel (Plavix) is used in the same dosage of 75-150 mg; it has a higher activity if compared with aspirin and ticlopidine (tiklid) and lower antiaggregant activity.

**Lipid-lowering agents**
The most important aspect of the medical treatment of patients with CVC is the use of drugs that lower blood lipids. Main drugs lowering cholesterol level (CL) and CL in low density lipoprotein (LDLP) in blood plasma are HMG-CoA reductase inhibitors – statins. These drugs reduce the risk of atherosclerotic MTR in both primary and secondary prevention. They have a marked hypocholesterolemic effect; they are safe, long-term and well-tolerated by most patients.

Simvastatin 40-80 mg after dinner, lovastatin 20-80 mg, atorvastatin 10-40 mg, fluvostatin 10-40 mg, rosuvastatin 5-40 mg.

Medicines should be administered in the afternoon, after dinner, taking into account possible increase of transaminases (AST, ALT) and creatine phosphokinase (CPK). When the enzyme levels increases three or more times the dose of drugs should be reduced.

With primary increase in plasma triglyceride levels statins should be supplemented by Trikor 145 mg or Omacor.

When there us no visible effect, you can add the drug, which violates the absorption of cholesterol in the intestine Ezetrol (ezetimib).

**Metabolic therapy**
**Preductal MR** (trimetazidine) 0.35 mg twice a day; it eliminates angina, improves survival for patients after myocardial infarction. Thiotriazolin of 0.1 mg twice a day has antianginal and anti-ischemic effect, improves metabolic processes in heart and liver.

**Surgical treatments for ischemic heart disease**
Along with drug therapy major IHD antianginal drugs (nitrates, beta-blockers and calcium channel blockers), and these drugs in combination with metabolic action drugs (trimetazidine), combined
with the use of antithrombotic drugs, lipid-lowering drugs in recent years, methods of revascularization infarction are widely used. They are carried out for treatment of patients with failure of medical therapy in oronary heart disease, and in some cases as an alternative to it.

What matters most, is the following methods of myocardial revascularization:

- Percutaneous transluminal coronary angioplasty (PTCA), most often balloon angioplasty without stenting or stenting (stent graft to prevent the inner walls of the arteries wears off after being removed from her bottle);
- Bypass the coronary artery (CA), in particular the most frequently used coronary artery bypass grafting (CABG), and imposing "mammarokoronarnyh anastomoses".

**Dispanserization of patients with IHD**

Local doctor should perform a dynamic observation of patients 2-4 times a year depending on the FC, once a year, patients should look about cardiologist, Department of rehabilitation doctor, neurologist, psychiatrist. On examination must be made one blood test once a year, to determine the spectrum of lipids and alpha-cholesterol, conducted ECG and functional tests, including VET. Especially careful of follow-demand patients with a high risk of complications. Patsieitov such group can be allocated on the basis of clinical signs and data load testing.

**Temporary disability in patients with angina**

First time emerged angina, aggravation of the disease, unstable angina, myocardial infarction can be reasons for TD.

**Approximate period for angina TD:**

- First emerged – 10-12 days or more for progressive course, class I patients are usually able to work, FC II – 10-14 days, FC III – 18-20 days, FC IV – 18-25 days prior to the registration of the direction to MREB , unstable angina 21-28 days (in the absence of myocardial infarction).

**Direction to MREB**

The direction to MREB with angina pectoris and myocardial infarction.
- With a poor employment outlook and the general prognosis.
- In need of employment (for example, a person driving jobs
with a favorable course of the disease, etc.).

- In need of continued treatment over a period of 4 months.

**Primary and secondary prevention of IHD**

The basis of primary prevention on the concept of the main risk factors for coronary heart disease was proved that for the elimination of risk factors incidence and severity of coronary artery disease was significantly reduced.

One of the urgent tasks of outpatient phase of rehabilitation, ischemic heart disease, including myocardial infarction, a secondary prevention. The aim is to prevent further progression of the disease, its prevention of exacerbations and complications. Measures for the secondary prevention of coronary heart disease is closely intertwined with the healing and rehabilitation. At the same time, in terms of secondary prevention special attention must be given to the active impact on the existing risk factors for IHD. They are causally related to disease factors.

Reduction of excess body weight in patients SS reduces breathlessness and reduce the frequency of angina attacks, improve physical performance.

Stopping smoking reduces the risk of a second heart attack by 20-50%.

Long-term exercise training helps slow the progression of coronary artery disease to reduce the doses of drugs.

**Lesson 5. Primary and secondary hypertension, somatoform autonomic dysfunction (cardiopsychoneurosis). Outpatient aspects of diagnosis and treatment, therapeutic approach, medical-social examination, dispensersation, primary prophylaxis**

**Arterial hypertension**

In industrialized countries, hypertension (AH) affects approximately 15% of the adult population. According to health authorities in Belarus in 2010 there are 1.5 million patients with hypertension.

The problem is of great social significance, since hypertension is one of the causes of persistent disability. Successful control of
hypertension and prognosis of the disease depends largely on how early and correct diagnosis was established, and on the necessary organization and the effectiveness of treatment of patients in the polyclinic.

Patients with uncomplicated hypertension obtain diagnostic and medical care in the clinic. Here are the basic principles of differential diagnosing of hypertension in the clinic.

**Criteria for diagnosing hypertension** are determined by the results of the daily blood pressure monitoring (DBPM), measurements of blood pressure at the doctor's (office BP) as well as by the patient at home. Hypertension is diagnosed if DBP is \( >130/80 \) mm Hg, if in two or more visits to a doctor blood pressure is \( >140/90 \) mm Hg, if blood pressure is \( >135/85 \) mm Hg measured at home by a patient.

**Analysis of patients with hypertension in a clinic.**

*Objectives of the survey:*
- Confirm the stability of the blood pressure increase;
- Exclude secondary character of high blood pressure;
- Detect removable and irremovable risk factors for cardiovascular disease;
- Evaluate the presence of target organ damage, heart disease and other related diseases;
- Assess individual risk of cardiovascular complications.

When analyzing the medical history following information should be obtained:
- Family history of hypertension, diabetes, efficacy and tolerability of previous antihypertensive therapy;
- Presence of ischemic artery disease or heart failure, cerebrovascular disease, peripheral vascular disease, diabetes, gout, dyslipidaemia, bronchospasm, sexual dysfunction, kidney disease, information about the drugs used to treat existing conditions;
- Symptoms suggestive of a secondary nature of AH;
- Patient's lifestyle, including diet (consumption of fat, salt, and alcohol), smoking, physical activity, presence of overweight or obesity (body mass index, waist/hips ratio for assessment of adipose tissue distribution);
- Administration of drugs that increase blood pressure (oral contraceptives, non-steroidal anti-inflammatory drugs, cocaine,
amphetamines, erythropoietin, cyclosporine, steroids);
- Personal, psychosocial and other factors (family environment, work, education), the ability to influence on the compliance to antihypertensive therapy.

**Complete physical examination includes:**
- Measurement of blood pressure 2-3 times in accordance with international standards,
- Height, weight, calculation of body mass index, waist circumference measurements and hip ratio calculation waist/hip;
- Investigation of the eye fundus to establish the degree of hypertensive retinopathy;
- Examination of the cardiovascular system: size of the heart, change of sounds, noises, signs of heart failure pathology of carotid, renal, and peripheral arteries, coarctation of aorta;
- Examination of the lungs (rales, signs of bronchospasm);
- Examination of the abdomen (vascular noise, increasing renal pathological pulsation of the aorta);
- Examination of peripheral arterial pulsation and the presence of edema in the extremities;
- Examination of the nervous system to clarify the presence of cerebrovascular disease.

Binding studies that should be done before treatment in order to identify target organ damage and risk factors:
- Analysis of the urine;
- Expanded TBC (total blood count);
- Blood chemistry (potassium, sodium, creatinine, glucose, total cholesterol and HDL);
- 12-lead ECG.

Special studies are conducted when the results may influence the treatment strategy of the patient:
- Advanced blood chemistry with the definition of low-density lipoprotein cholesterol, triglycerides, uric acid, calcium, glycated hemoglobin;
- Determination of creatinine clearance;
- Plasma renin activity, aldosterone levels, PH, T4;
- Study of daily urine (microalbuminuria, daily proteinuria, and urinary catecholamine excretion);
- Echocardiography, USI of kidney, ambulatory blood pressure monitoring;
  angiography, computed tomography.

Daily monitoring provides essential information on the mechanisms of cardiovascular regulation, in particular, reveals the daily variability of blood pressure, nocturnal hypotension, homogeneity of the hypotensive effect of antihypertensive drugs. To date, however, this method is not very common for a diagnosis of hypertension and has no standards of the results assessment.

The use of special examination techniques for investigation of high blood pressure causes is indicated in the following cases:
- Age, medical history, results of physical examination and routine laboratory tests, the severity of hypertension does not exclude its secondary character;
- Severe increase of blood pressure in previously moderate AH;
- The presence of crises with severe autonomic manifestations;
- AH III degree, and hypertension refractory to medical therapy;
- Sudden onset of hypertension

Diseases that can be detected in an outpatient’s clinic and can cause symptomatic hypertension:

**Renal hypertension:**
- Chronic glomerulonephritis
- Chronic pyelonephritis
- Diabetic nephropathy
- Polycystic kidney disease
- Kidney involvement in systemic vasculitis
- Renal amyloidosis
- Tuberculosis of kidney
- Tumor and kidney injury
- Nephropathy in pregnant
- Congenital anomalies of the positions and forms of kidney disease (hypoplasia, double, dystopia, hydronephrosis, horseshoe kidney).

**Renovascular hypertension:**
- Renal artery atherosclerosis
- Fibromuscular dysplasia of the renal artery
- Nonspecific aortoarteriitis
- Hematoma and tumor compressing the renal arteries
- Congenital atresia and hypoplasia of the renal arteries, angioamas and arteriovenous fistula, aneurysm.

**Endocrine hypertension:**
The damage of the adrenal cortex: hypersecretion of mineralocorticoids (primary aldosteronism and idiopathic aldosteronism, familial form of hyperaldosteronism type I).

Hypersecretion of glucocorticoids (Cushing's syndrome).

The damage of the adrenal medulla: hypersecretion of catecholamines (pheochromocytoma).

Dysfunction of the thyroid and parathyroid glands (hypothyroidism, hyperthyroidism, hyperparathyroidism) with the lesion of the pituitary gland (pituitary Cushing, acromegaly).

**Hypertension caused by arterial atherosclerosis,** aortic coarctation, stenosing lesions of the aorta and brachiocephalic arteries nonspecific aortoarteritis.

**Neurogenic or central AH**
In organic CNS lesions (tumors, trauma, encephalitis, polio, focal ischemic lesions), the syndrome of obstructive sleep apnea, lead poisoning, acute porphyria.

**Drugs and exogenous substances that can cause hypertension:** hormonal contraceptives, corticosteroids, sympathomimetics, mineralocorticoids, cocaine, foods containing tyramine or MAO inhibitors, NSAIDs, cyclosporine, erythropoietin.

**Somatoform autonomic dysfunction**
A group of disorders that manifest symptoms of internal organ or organ system, but do not have an objective basis recorded. Complaints such patients are presented as though they are caused by physical disorder of the system or organ, which mainly or entirely under the influence of the autonomic nervous system, i.e. cardiovascular, gastrointestinal or respiratory system. The most frequent and prominent examples include the cardiovascular system. Somatoform autonomic dysfunction of the heart and cardiovascular system (includes cardioneurosis, neuro-circulatory asthenia, Da Costa's syndrome).

Symptoms usually come in two types:
- The first type of symptoms, which is largely based on
diagnostics, characterized by signs of autonomic arousal (heart rate, sweating, flushing, and tremor).

- The second type is characterized by a sense of fleeting pain in the heart, burning, and heaviness.

One of the most frequent somatoform autonomic dysfunction of the cardiovascular system is cardialgyc syndrome, which is characterized by multiformity and variability, the lack of clear irradiation, the appearance alone with emotional stress, long hours – day, exercise do not provoke or relieve pain.

Cardialgia is often accompanied by anxiety, patients do not find a place for themselves, moan and groan. Palpitations in this type of disorder in only half of cases are accompanied by increased heart rate of 110-120 beats per minute which increase at rest, especially in the supine position. Unstable pressure increasing to 150-160/90-95 mm Hg, which appears due to stress, can also occur with somatoform disorders. It should be admitted that tranquilizers are more effective than antihypertensive drugs.

The main rules of diagnosing, screening, follow-up and treatment of patients with hypertension are given in the order of Ministry of Health of Belarus № 225 of 03.09.2001 y.

**Classification of AH** (Tabl. 1)


<table>
<thead>
<tr>
<th>Criteria BP</th>
<th>SBP</th>
<th>DBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optimal</td>
<td>&lt;120</td>
<td>&lt;80</td>
</tr>
<tr>
<td>Normal</td>
<td>120-129</td>
<td>80-84</td>
</tr>
<tr>
<td>High normal</td>
<td>130-139</td>
<td>85-89</td>
</tr>
<tr>
<td>AH stage I</td>
<td>140-159</td>
<td>90-99</td>
</tr>
<tr>
<td>AH stage II</td>
<td>160-179</td>
<td>100-109</td>
</tr>
<tr>
<td>AH stage III</td>
<td>&gt;180</td>
<td>&gt;110</td>
</tr>
<tr>
<td>Isolated systolic hypertension</td>
<td>&gt;140</td>
<td>&lt;90</td>
</tr>
<tr>
<td>(isolated systolic hypertension)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

If the systolic and diastolic blood pressure corresponds to different degrees of hypertension, blood pressure of the person should be referred to a higher category. For example, blood pressure to
165/95 mm Hg. should be considered as AH II stage, and blood pressure 212/108 mm Hg. – as AH III stage.

In accordance with WHO/ISH hypertension diagnosis should specify not only the rate of increase in blood pressure, but also the risk of an adverse outcome in view of all the factors influencing the prognosis. There are main risk factors that are taken into account when formulating a diagnosis, support, influence prognosis, but not taken into account when diagnosing.

**Criteria for risk stratification:**

**Risk Factors**

*value of pulse pressure* (in the elderly)

*age (men > 55 years: women > 65 years)*

*smoking*

Lipids: TCh > 5.0 mmol/l (190 mg/dL) or LDL cholesterol > 3.0 mmol/l (115 mg/dL) or HDL cholesterol < 1.0 mmol/L (40 mg/dL) for men < 1.2 mmol/L (46 mg/dL) for women or triglycerides > 1.7 mmol/l (150 mg/dL)

*fasting plasma glucose 5.6-6.9 mmol/L (102-125 mg/dL).*

*IGT.*

*Family history of early cardiovascular events* (men < 55 years, women < 65 years).

*Abdominal obesity* (from > 102 cm for men and > 88 cm for women) in the absence of metabolic syndrome (MS).

**Target organ damage (TOD)**

**LVH**

*ECG: a sign of the Sokolov-Lyons > 38mm Cornell product > 2440 mm x ms.*

*Echocardiography: LVMI > 125 g/m² in males and > 110 g/m² in females.*

**Vessels**

*ultrasound signs of arterial wall thickening (IMT > 0.9 mm) or plaques of great vessels;*

*the pulse wave velocity from the carotid to the femoral artery > 12 m/s;*

*ankle / brachial index < 0.9.*

**Kidneys**

*slight increase in serum creatinine: 115-133 mmol/l (1.3-1.5 mg/dL) for men;*
or 107-124 mmol/l (1.2-1.4 mg/dL) for women;
• low KFR <60 ml/min/1.73m2 (MDRD formula) or low creatinine clearance <60 mL/min (Cockcroft Gault formula);
• microalbuminuria 30-300 mg / day;
• albumin / creatinine ratio in the urine of >22 mg/g (2.5 mg/mmol) for males and >31 mg/g (3.5 mg/mmol) for females.

**Diabetes mellitus**
• fasting plasma glucose >7.0 mmol/l (126 mg/dL) with repeated measurements;
• plasma glucose after meals or 2 hours after a 75 g glucose >11.0 mmol/l (198 mg/dL).

**Metabolic syndrome**
The main criterion – AO (from >94 cm for men and >80 cm for women).

Additional criteria:
BP> 140/90 mm Hg,
LDL cholesterol >3.0 mmol/L, HDL cholesterol <1.0 mmol/L for men and <1.2 mmol/l for women, triglycerides >1.7 mmol/l.
fasting hyperglycemia >6.1 mmol/l,
IGT – plasma glucose 2 hours after taking 75 g glucose >7.8 and ≤11 mmol/l.

The combination of the main and 2 additional criteria indicates the presence of MS.

**Associated clinical conditions (ACC)**

**Cerebrovascular diseases:** ischemic brain infarction, hemorrhagic brain infarction, transient ischemic attack.

**Heart disease:** myocardial infarction, angina, coronary revascularization, heart failure.

**Hypertensive retinopathy:** hemorrhages or exudates, papilledema.

**Kidney disease:** diabetic nephropathy, renal failure: serum creatinine >133 mcmol/l (1.5 mg/dL) for men and >124 umol/l (1.4 mg/dL) for women.

**Peripheral arterial disease:** dissecting aortic aneurysm, peripheral artery disease.

Factors determining the prognosis of hypertensive target organ damage and associated clinical conditions that should be considered in
the risk assessment. Particularly increases the risk of combination of high blood pressure, obesity, hyperglycemia and hypercholesteremia known as "deadly quartet".

**Examples of diagnosis formulation:**
- Hypertension II degree, risk 2, hypercholesterolemia;
- Hypertension II degree, risk 3, hypertensive heart, ventricular premature beats;
- Hypertension III degree, risk 4, CHD: angina, PCF, postinfarction (1999r) infarction, left ventricular aneurysm;
- Hypertension I level, risk 1, hypertensive crisis one way (10/10/00) mild;
- Hypertension III degree, risk 4, hypertensive kidney nefroangiosclerosis, CRF II degree.

Table 2 – Clinical management and assessment of patients with recently diagnosed elevated blood pressure

<table>
<thead>
<tr>
<th>Systolic blood pressure</th>
<th>Diastolic blood pressure</th>
<th>Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>130-139</td>
<td>85-89</td>
<td>Healthy lifestyle, control after 1 year</td>
</tr>
<tr>
<td>140-159</td>
<td>90-99</td>
<td>Healthy lifestyle control during 3 months</td>
</tr>
<tr>
<td>160-179</td>
<td>100-109</td>
<td>Investigate and start treatment in 1 month</td>
</tr>
<tr>
<td>&gt;180</td>
<td>&gt;110</td>
<td>Investigate and start treatment immediately</td>
</tr>
</tbody>
</table>

*Indications for direction of the patients with hypertension to hospital*

Patients with hypertension should be treated continuously, throughout the life. In this regard, doctors of outpatient practice have to observe these patients regularly. Opening of diagnostic- and cardio centers in all regional cities of the country allow to expand the scope of diagnostic examination of hypertensive patients, performed in outpatient settings. In this case, the indications for hospitalization include:

1. Acute disorders of coronary or cerebral circulation.
2. Moderate and severe hypertensive crisis.
3. Refractory to antihypertensive therapy of hypertension.
4. Examination to identify the degree of target organ damage, if it can not be done on outpatient basis/
5. Worsening of arrhythmia, conduction of the heart, cardiac and/or renal failure.

When district physician or cardiologist refers a patient to hospital, he/she should give the following information:
1. The purpose of admission.
2. Outpatient examination results: clinical blood and urine tests, electrocardiogram, ophthalmologist consultation, blood chemistry (glucose, cholesterol, urea and creatinine), REG.
3. Character of antihypertensive treatment, its effectiveness, tolerance to individual drugs.

Indications for referral to Cardiology Center:
1. Need to clarify the extent of target organ damage in cases where this is not possible in the clinic (holding VEM, Holter ECG monitoring, echocardiography).
2. Refractory to antihypertensive therapy AH to clarify the causes of refractoriness.
3. Frequent hypertensive crises of type II or temporary incapacity due to prolonged hypertension within 6 months for treatment correction.

**Treatment of Hypertension**

The principles of non-drug treatment of hypertension
1. Lifestyle changes:
   - Weight reduction.
   - Giving up smoking.
   - Limiting alcohol consumption to 168 ml of 100% alcohol per week for men and up to 112 ml per week for women.
2. Diet therapy
   - Diet therapy should include:
     - Substantial reduction of dietary sodium intake to 2 g/day or 5 g of salt per day.
     - Reduction of the overall calorie count especially in individuals who are overweight.
     - Increase in the consumption of vegetable products in order to saturate the body with potassium ions, calcium and magnesium (tomatoes, oranges, potatoes, especially baked, raisins, dried apricots, etc.).
- Milk or milk products intake (if tolerated). Milk is rich in calcium salts, increasing the share of vegetable fats, rich in polyunsaturated fatty acids, while reducing the share of animal saturated fat.
- Add fish oils.
- Limiting the consumption of caffeine (tea, coffee, etc.).

3. Physical rehabilitation
Optimization of physical activity using exercise, exercise endurance (fast running, walking, skiing, and swimming). We recommend regular physical activity (exercise in the open air of moderate intensity and duration of at least 30-60 minutes 3-4 times a week).

4. Psychotherapy.
Training of appropriate response to stressful situations, as well as relaxation techniques.

5. Acupuncture.
Especially with the type of hyperkinetic circulation.

This therapy is increasingly used, especially in the elderly.

7. Phytotherapy.

8. Physiotherapy.

Drug treatment of patients with hypertension
Modern evidence-based medicine clearly shows that the main condition for the successful hypertension therapy is to achieve "target levels" of BP. According to national guidelines for the diagnosis and treatment of hypertension 2007, the "target level" is 140/90 mm Hg., and in persons with diabetes – less than 130/80 mmHg.
Achieving "target" with BP alone is possible only in 30-50% of patients. In patients with Stage II and Stage III in the presence of target organ damage (TOD), diabetes, metabolic syndrome, monotherapy can be effective only in a few cases, requiring the appointment of maximal doses of drugs that can cause several side effects. Therefore, in large clinical trials in these patients "target" BP achievement was possible only by a combination of two or more drugs. These data were obtained from the results of a series of multicenter studies: SHEP, MAPHY, ALLHAT, INVEST, LIFE, STOP, COOPE, the results of which are included in the Russian recommendations (third revision), "Diagnosis

**Group of antihypertensive drugs (AHD)**
- angiotensin-converting enzyme inhibitors (ACE inhibitors)
- angiotensin II receptor blockers, (ARBs)
- calcium antagonists (CA)
- β-blockers (β-AB)
- Diuretics

As additional classes of AHDs for the combination therapy α-blockers (α-AB) and imidazoline receptor agonists can be used.

Newly registered and approved for use direct renin inhibitor aliskiren – the first of a new class of AHD.

In recent years, as recommended by the European Society of Hypertension (ESH) in collaboration with the European Society of Cardiology (ESC) new, more selective aldosterone receptor antagonist – eplerenone was recommended.

The main criterion for the appointment of medical therapy is being in a risk group, and not the rate of increase in blood pressure. In high and extremely high risk along with non-pharmacological therapy drug therapy should be immediately introduced. In groups of patients with middle and lower risk of non-pharmacological treatment is prescribed together with the program, controlling blood pressure and risk factors. Duration of non-drug treatment in the group of average risk is 6 months, in the group of low-risk is 12 months. Particular attention should be paid to patients with high normal blood pressure (130-139/85-89), with diabetes and kidney failure. These patients should be given early antihypertensive treatment for prevention of cardiovascular complications.

**General principles of drug treatment of hypertension:**
- Patients should be informed that treatment of hypertension is essential;
- Patients should be aware that there is a course of hypertension treatment;
- The beginning of treatment with low doses of a single drug with a gradual increase in dose;
- Treatment of patients with hypertension should be done individually, based on the level of blood pressure, and other risk factors;
- Regulation of blood pressure is performed gradually, over several weeks and months, a doctor should find the optimal level of blood pressure for each patient;
  - Move to another class drugs with little effect of treatment after increasing the dose of the first drug or bad its portability;
  - The use of long-acting drugs, which are softer and provide constant hypotensive action, including morning hours;
  - Use of optimal drug combinations for maximal hypotensive effect and minimal adverse events.

The following factors influence on the choice of the drug:
- Risk factors of cardiovascular disease;
- TOD;
- Kidney damage, MS, DM;
- The presence of accompanying diseases, which may limit the use of antihypertensive drugs of this class;
- Variability in the individual response of patients to drugs of individual classes;
- Interaction with other drugs
- Social and economic factors determining the availability of a drug to a patient.

The goals of treatment (European guidelines on hypertension treatment, 2007.)
1. Reduction in the overall risk of cardiovascular morbidity and mortality:
   - Correction of reversible risk factors for cardiovascular disease and mortality (smoking, dyslipidemia, diabetes);
   - Treatment of associated medical conditions;
   - Decrease in blood pressure.

2. Reducing BP level:
   - <140/90 mm Hg. – In all hypertensive patients with good tolerability;
   - <130/80 mm Hg. – In patients with diabetes;
   - <125/75 mm Hg. – Diabetic patients with proteinuria >1 g/day;
   - Up to 150/90 mm Hg. – Persons older than 80 years.

Achieving "target level" of BP using monotherapy is possible only in 30-50% of patients. In patients with Stage II and Stage III in
the presence of target organ damage (TOD), diabetes, metabolic syndrome, monotherapy can be effective only in a few cases, requiring the appointment of maximal doses of drugs that can cause several side effects. Therefore, in large clinical trials the achievement of "target" BP in these patients was possible only with the use of combination of two or more drugs.

Recommended combinations of antihypertensive drugs.
Rational (effective):
ACEI + diuretic;
ARB + diuretic;
ACEI + CA;
ARB + CA;
dihydropyridine CA + β-AB;
CA + diuretic; β-AB + diuretic; β-AB + α-AB.

Possible:
dihydropyridine and non-dihydropyridine combination CA;
ACEI + β-AB;
ARB + β-AB;
ACEI + ARB;
α-AB with ACE inhibitors;
ARB + diuretic;
CA + diuretic.

Irrational
β-AB + non-dihydropyridine AK;
ACEI + potassium-sparing diuretic;
β-AB + medication central action.

In recent years, more and more patients use a fixed combination of drugs, especially in rational combinations, for example, equator (diroton + amlodipine), lizoretik (lisinopril + hydrochlorothiazide), Co-Sentor (losartan + hydrochlorothiazide), etc.

Medical-social expertise in hypertension
Temporary disability (TD) in hypertension can be observed in arrhythmias and disorders of cardial conduction, signs of heart or kidney failure, the appearance of clinical symptoms indicating a worsening of regional circulation (e.g., dizziness, increased frequency of angina attacks).

In patients during the crisis of type 2 cardiac (angina, heart
failure, arrhythmias, etc.) and brain complications (transient focal neurological symptoms) can develop. In this case the terms of TD are extended until elimination of these complications and stabilization of BP (average 2-3 days).

After the crisis the criterion of patients’ discharge is a stable compensation of pathological process. The doctor should be aware that patients with stage I or II without end-organ damage are not recommended to work if the work is associated with significant physical and mental stress, as well as with unfavorable weather and operating conditions (in hot, humid, cold, damp room with the production noise, vibration, night shifts, duties, etc.). These patients should employ through the MCC. The abovementioned working conditions are contraindicated in hypertensive patients with involvement of target organs and the presence of concomitant medical conditions.

The bases for referral of patients with hypertension to MREB are:
1. Prolonged TD (4 consecutive months or 5 months with breaks in the past 12 months) to detect disability signs or make a decision on the treatment extension. If MREB finds signs of disability and recommends the extension of treatment, TD will be extended by the decision of the MCC for more that the abovementioned terms.
2. Signs of disability.

Criteria for disability
Disability is defined for persons with disabilities of various aspects of life caused by the dysfunction of the organs and systems.

The following factors lead to disability in hypertension:
2. Evident damage of target-organs (heart, kidney, brain syndrome, impaired visual function).
3. Unfavorable course of hypertension with a worsening of its course in spite of treatment, increase of end-organ damage.
4. The syndrome of mutual aggravation of diseases (with concomitant ischemic artery disease, diabetes, chronic cerebral vascular insufficiency, chronic obstructive bronchitis, asthma, etc.) leads to the restriction of life occurring when less functional disability is caused by certain diseases. In patients with hypertension working disability occurs more often than, disability in independent movement

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and self service. Other aspects of life (orientation, communication, behavior control) in hypertension are rarely violated, mainly in brain complications.

**Dispencerization of patients with arterial hypertension**

All patients with hypertension are the third group of dispensary (D III). Depending on the severity of the disease, they are examined by a physician 2 to 4 times a year. At least 1 time a year, they should be assessed by a cardiologist, neurologist, neuropsychiatrist, ophthalmologist. Consultation at endocrinologist and urologist performed when indicated. Besides the following laboratory and other diagnostic tests should be taken in all hypertensive patients: urinalysis – 2-4 times a year, blood creatinine, cholesterol, triglycerides, alpha-cholesterol, fundus examination, ECG. Other examinations should be performed according to indications (REG, central hemodynamics, renal and heart ultrasound).

Active counseling of hypertensive patients should always include the main therapeutic measures listed below. District physician should explain the importance of these measures to a patient and recommend their implementation in the correct form:

Treatment in active counseling of patients with arterial hypertension:

1. Healthy lifestyle.
2. Correction of risk factors for hypertension, restriction of salt and saturated fats in food.
3. Psychotherapy.
4. The use of physical therapy techniques and exercise therapy.
5. Drug prevention and treatment, the use of antihypertensive drugs using step chart.
7. Guidelines on employment and monitoring of their implementation.

The criteria for the effectiveness of clinical examination of patients with hypertension are: reduction of primary disability, the incidence of stroke, myocardial infarction and sudden cardiac death, as well as increase of the number of individuals with controlled blood pressure (≤140/90 mm Hg.), reduced incidence of temporary disability.
Primary and secondary prevention

Measures for primary and secondary prevention of hypertension are aimed at improving the way of life in general. The most important is the fight against avoidable risk factors, especially in patients with uncorrected risk factors. It is necessary to reduce blood pressure in the general population, as the primary prevention of hypertension will prevent future development of the disease complications.

Secondary prevention of hypertension involves preventing the progression of the disease and associated complications. This can be achieved by assigning a patient a rational, adequate individualized antihypertensive therapy. A very important factor is a patient’s wish to be treated. Numerous and long-term clinical observations show that this tactic of antihypertensive therapy is very effective, able to delay the progression of the disease, reduce the incidence of complications such as stroke, myocardial infarction, and so on.

Secondary prevention measures are certainly not limited by the use of antihypertensive drugs. They involve the use of a set of measures directed to primary prevention of hypertension.

Thus, the active detection of persons with high risk of hypertension, as well as proper treatment of persons with already developed hypertension makes it possible not only to reduce the incidence of this form of heart disease, but also to decrease the number of severe complications and death due to them.

Lesson 6. Chronic gastritis, peptic ulcer and duodenal ulcer. Outpatient aspects of diagnosis and treatment, therapeutic approach, medical-social examination, clinical examination, primary prophylaxis. Emergency care for acute abdominal pain and suspected gastrointestinal bleeding

Chronic gastritis

Chronic gastritis (CG) is a chronic inflammation of the gastric mucosa, which is combined with the regeneration disorder of the epithelial cells which results in atrophy, impaired secretory, motor and endocrine function. So, first of all, CG – is a morphological concept.

CG is a collective concept that brings together different etiology
and pathogenesis of inflammatory or disregenerative (focal or diffuse) lesions of the mucous membranes of the stomach and submucosa with symptoms of progressive atrophy, functional and structural adjustment, with different clinical manifestations. All this makes gastritis diagnosis very difficult, especially at early stages of the disease course. Syndrome of gastric dyspepsia, combining clinical symptoms such as epigastric pain, appetite disorders, indigestion, nausea and vomiting were observed in many diseases of the stomach.

The most common cause of chronic gastritis is infection with Helicobacter pylori. The source of Helicobacter pylori infection is an infected person and pets. The main route of infection is oral-oral or fecal-oral.

**Classification**


**According to the mechanism of development:**
- gastritis A (autoimmune).
- gastritis type B.
- gastritis type C
- Mixed CG (atrophic pangastritis A – B).

**According to morphological features:**
- Surface
- Interstitial (with the defeat of glands without atrophy).
- Atrophic
- Atrophic with symptoms of pyloric metaplasia or intestinal type
- atrophic-hyperplastic (mixed)
- polypoid
- Hypertrophic, including giant hypertrophic (illness Menetries)
- Erosive

**According to localization:**
- Limited (antral, fundic)
- Common (diffuse)

**According to the acid-forming function of the stomach:**
- With enhanced and preserved secretion
- Secretory failure (moderate to severe, including achlorhydria)
According to phases:
- Aggravation
- Remission.

In a new classification system (modified "Sydney system," Philadelphia, 1994), there is no significant changes (Tabl. 1). International “Sydney” classification is more modern.

Table 1 – International “Sydney” classification

<table>
<thead>
<tr>
<th>Sections</th>
<th>Variants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphological (based on endoscopy),</td>
<td>erythematous (superficial) erosive haemorragic</td>
</tr>
<tr>
<td></td>
<td>Atrophic (moderate, severe)</td>
</tr>
<tr>
<td></td>
<td>Hyperplastic</td>
</tr>
<tr>
<td>Based on histological examination of mucosa</td>
<td>inflammation with erosions, bleeding</td>
</tr>
<tr>
<td></td>
<td>Atrophic (moderate, severe)</td>
</tr>
<tr>
<td></td>
<td>Violation of cell renewal, epithelial metaplasia</td>
</tr>
<tr>
<td>Etiology</td>
<td>associated with gastric HP (type B)</td>
</tr>
<tr>
<td></td>
<td>autoimmune (type A)</td>
</tr>
<tr>
<td></td>
<td>Reactive (type C)</td>
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<tr>
<td>Topography</td>
<td>antral</td>
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<tr>
<td></td>
<td>fundic</td>
</tr>
<tr>
<td></td>
<td>pangastritis</td>
</tr>
<tr>
<td>Activity</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>Moderately</td>
</tr>
<tr>
<td></td>
<td>Active</td>
</tr>
</tbody>
</table>

Gastritis (gastroduodenitis) is diagnosed considering the etiology, endoscopic and histopathological changes and the severity of the process. Predominant gastritis associated with Helicobacter pylori (HP) (85%) is atrophic, usually autoimmune (5%), it is often manifested as $\text{B}_{12}$-deficiency anemia. Allocated gastritis is associated with taking drugs, it is granulomatous and eosinophilic.

**Diagnostics**
The most common reason for treatment of patients by a local therapist is a syndrome of gastric dyspepsia.

Syndrome of "gastric dyspepsia":
- epigastric pain of varying intensity;
- feeling of heaviness and fullness in the upper abdomen (both usually associated with eating);
- feeling of early saturation, nausea and sometimes vomiting;
- loss of appetite;
- heartburn;
- belching;
- bitter taste in the mouth.
Patients with **antral gastritis (type B)**, associated with HP, suffer from fullness and slight epigastric pain. Pain occurs after taking spicy, fried and fatty foods at fast food restaurants. Patients feel better after belching (air, food). In 60% of patients hypersecretion is observed, 30% are with normal acid production and 10% have hyposecretion (formation of multifocal gastritis). The disease can also occur without symptoms. Antral gastritis is observed in all patients with gastroduodenal ulcers.

**Autoimmune gastritis (type A)** is caused by genetic factors associated with the HLA B8, DR4. It can be combined with autoimmune endocrinopathies: autoimmune thyroiditis, hyperthyroidism, type 1 diabetes, vitiligo, Addison's disease. In severe cases, deficiency of intrinsic factor leads to B12 deficiency anemia. Usually it is latent and is detected in the near relatives. Evident hypoacidy and high gastrinemia is characteristic.

**Reflux gastritis (type C)** causes permanent traumatization of the gastric mucosa by bile reflux throw-in, which is observed after gastrectomy, gastroenterostomy, cholecystectomy, in duodenal patency disorders and pyloric insufficiency. Epigastric pain, nausea and sometimes vomiting bile can be observed, there is a tendency to lose weight.

**Lymphocytic chronic erosive gastritis** is characterized by erosions on the mucous membrane of the stomach and evident lymphocytic infiltration of the epithelium.

**Examination**

Required lab tests:
- full blood count;
- total protein and protein fractions;
- histological and cytological examination of biopsy;
- Urinalysis;
- fecal occult blood test.

Required **instrumental** investigations:
- esophagogastroduodenoscopy with biopsy and brush histology;
- Ultrasound of the liver, biliary tract and pancreas.

Additional research and expert advice are implemented according to the main symptoms of the disease and the supposed concomitant diseases.
Diagnosis is based on a comprehensive assessment of the clinical picture and the results of laboratory and instrumental studies. Gastroscopy with mucous membrane of the antrum and body of the stomach biopsy is necessary. The gastroscopy results reflect the location and extent of the pathological process. Biopsy and the study of gastric secretion can finally verify chronic gastritis and its form (Tabl. 2, 3).

Confirmation of autoimmune gastritis is the detection of antibodies to parietal cells and intrinsic factor.

Table 2 – Normal values for key indicators of gastric secretion

<table>
<thead>
<tr>
<th>Key indicators of secretion</th>
<th>gastric secretion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Juice volume (ml)</td>
<td>Hunger</td>
</tr>
<tr>
<td></td>
<td>Basal</td>
</tr>
<tr>
<td>Not more than 50</td>
<td>50-100</td>
</tr>
<tr>
<td>Total acidity (titr.un.)</td>
<td>Not more than 40</td>
</tr>
<tr>
<td>40-60</td>
<td></td>
</tr>
<tr>
<td>Free HCL (titr. un.)</td>
<td>Not more than 20</td>
</tr>
<tr>
<td>20-40</td>
<td></td>
</tr>
<tr>
<td>Total acidity debit-hour/mili equivalent/debit-hour/mg /</td>
<td>debith-port to 2,0</td>
</tr>
<tr>
<td>1,5-5,5 (HLW) 55-200</td>
<td></td>
</tr>
<tr>
<td>Free HCL h/mEq/debit-hour/mg/</td>
<td>debith-port to 1,0</td>
</tr>
<tr>
<td>1-4,0</td>
<td></td>
</tr>
<tr>
<td>40-150</td>
<td></td>
</tr>
</tbody>
</table>

Table 3 – Criteria for chronic gastritis (CG) diagnosing

<table>
<thead>
<tr>
<th>Criteria</th>
<th>CG type A</th>
<th>CG type B</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Morphological</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>The preferential localization</td>
<td>Fundus, body</td>
<td>Antrum</td>
</tr>
<tr>
<td>Inflammatory response</td>
<td>Poorly expressed</td>
<td>Expressed</td>
</tr>
<tr>
<td>Atrophy</td>
<td>Primary</td>
<td>Secondary</td>
</tr>
<tr>
<td>The presence of erosions</td>
<td>Rare</td>
<td>Often</td>
</tr>
<tr>
<td><strong>Immunological</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HP</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Antibodies to HP</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Antibodies to parietal cells</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Antibodies to internal factor</td>
<td>+</td>
<td>-</td>
</tr>
</tbody>
</table>

158
<table>
<thead>
<tr>
<th>Criteria</th>
<th>CG type A</th>
<th>CG type B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Expressed gastrinemia</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Hypoacid secretion</td>
<td>Expressed</td>
<td>Any type of secretion</td>
</tr>
<tr>
<td>B12-deficiency anemia</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Combination with peptic ulcer disease</td>
<td>Rare</td>
<td>100%</td>
</tr>
<tr>
<td>Malignancy</td>
<td>Extremely rare</td>
<td>Often</td>
</tr>
</tbody>
</table>

- It is necessary to approve chronic gastritis diagnosis and determine its variant according to classification ("the Sydney System"), ie, try to find out the etiology, natural features, activity of the process.
  - Find out what other pathogenic processes are observed.
  - Specify if, chronic gastritis is complicated by accompanying diseases.

**Medical therapy**
Gastritis (gastroduodenitis) is associated with HP, with ulcer-like dyspepsia.
Drug treatment is carried out for one of these schemes.

**Seven-day scheme:**
1. Pilorid (ranitidine bismuth citrate) to 400 mg 2 times a day + clarithromycin (klacid) 250 mg 2 times a day, or tetracycline 500 mg 2 times a day or amoxicyclin 1 g 2 times daily + **metronidazole** (Trichopol) 500 mg 2 times a day.
2. **Omeprazole** (zerocid) 20 mg 2 times a day or pantoprazole (kontrolok) 40 mg 2 times a day +
   - **De-Nol** 240 mg two times a day or ventrisol 240 mg two times daily + **tetracycline** 500 mg 2 times a day or amoxicyclin 1 g 2 times a day.
3. **Famotidine** (Quamatel, famocid) 20 mg 2 times daily or ranitidine 150 mg 2 times a day +
   - **De-Nol** 240 mg two times a day or ventrisol 240 mg two times daily + **tetracycline** 500 mg 2 times a day or amoxicyclin 1 g 2 times a day. Instead of de-ethanol can be assigned **vikalin (Vikair)** 2 tablets 4 times a day.

**Ten-day scheme.**
**Ranitidine** 150 mg 2 times a day, or famotidine (Quamatel)
20 mg 2 times a day, or omeprazole 20 mg 2 times a day, or pantoprazole (kontrolok) 40 mg 2 times a day + **tetracycline** 250 mg 4 times a day with meals.

**Autoimmune (atrophic) gastritis** with megaloblastic anemia, confirmed by bone marrow examination.

Drug treatment includes vitamin B12 (or cyanocobalamin) 1000 mcg intramuscularly for 6 days, then – at the same dose 1 day a week for a month, and in the subsequent long-term (life) 1 every 2 months. You can assign replacement therapy achlorhydria, enzyme preparations and preparations of nicotinic acid.

**Other forms of gastritis (gastroduodenitis).**

Symptomatic treatment with the following drug combinations:

- **in non-ulcer dyspepsia** – gastrocepin to 25-50 mg 2 times a day + Maalox (Gastal, Aluminium phosphalugel, remagel, gelusil, etc.), 2 tablets or 15 ml (sachet) 3 times a day 1 hour after meals;
- **in case of hypomotoric dyspepsia** – domperidone (Motilium), or cisapride (koordinax) 10 mg 3-4 times before meals + Maalox (or other antacids) to 2 tablets or 15 ml (sachet) three times a day an hour after meals.

**Indications for hospitalization:**

Indications for planned hospitalization:
- expressed worsening;
- severe form of the disease;
- ineffective outpatient treatment;
- need for hospital examination and differential diagnosis:
- The need for surgical treatment.

Indications for emergency admission:
- bleeding.

Length of stay is 10 days. But considering the etiology and severity of morphological manifestations of the disease the period may be reduced. Most treatment is done on an outpatient basis with the patient (rational diet and lifestyle). Chronic H. pylori gastritis is now considered as a reversible process.

**A possible approach for the management of CG type B associated with HP**

With low HP dissemination and the absence of clinical symptoms the medical treatment is not necessary.
In moderate or significant colonization and absence of symptoms bismuth salts (De-Nol, bismuth nitrate) should be indicated.

Significant colonization, antral gastritis with presence of erosions or without them according to fibrogastrroduodenoscopy and moderate or severe clinical picture aquires "triple therapy": antibiotics (tetracycline or ampioks) + metronidazole + ranitidine or omeprazole.

Moderate or severe colonization of the gastric mucosa with HP with the spread of the inflammatory process throughout the stomach, moderate or severe atrophy with decreased secretory function, the presence of moderate to severe clinical symptoms acquires "triple therapy" - antibiotics metronidazole + + De-Nol and bismuth nitrate.

Gastritis associated with HP (antral), combined with duodenal ulcer and the presence of clinical symptoms acquires the "triple therapy" - antibiotics metronidazole + + ranitidine or omeprazole.

**Requirements to the treatment results:**
- No symptoms, endoscopic and histological evidence of active inflammation and infectious agent (complete remission).
- Cessation of pain and dyspeptic disorders, reduced histological evidence of inflammatory nrotsessa.

**Dispanserization**

Patients with active gastritis (gastroduodenitis) associated with autoimmune gastritis and HP should be subjected to dispensary observation. Clinical examination is necessary 1-3 times a year, gastroduodenoscopy is indicated by gastroenterologist.

It should be remembered that after HP eradication complete regression of gastritis, its activity and inflammation occurs. Atrophy is theoretically reversible, but its reverse development is observed in a small number of patients.

Chronic gastritis with prevalence of disregeratory processes, including epithelial metaplasia, can be considered as a precancerous condition. Manifestation of high alert to cancer should be considered as the presence of mucosal atrophy with signs of impaired epithelial regeneration and, as a consequence, significant reduction of the stomach secretory function. Such patients should undergo biopsy during fibrogastrroduodenoscopy twice a year.
Criteria for temporary disability

These criteria include: evident worsening of the disease with intense pain and dyspeptic symptoms, the presence of erosive changes in the mucous membrane of the stomach, complications, need for surgery (special forms of CG).

Tentative dates of temporary disability:
- In mild exacerbation of gastritis
  - Patients are able to work;
- CG Exacerbation of average severity – 6-7 days;
  - During the second (heavy) exacerbation CG – 12-14 days (inpatient treatment);
  - Exacerbation of erosive CG – 12-17 days (inpatient treatment).

Period of temporary disability in special forms of CG, the development of complications are determined by their character (anemia, bleeding), type of treatment (conservative and surgical), its efficacy. Longer temporary disability is possible with concomitant diseases and conditions (hypothyroidism, respiratory and heart failure, etc.), and adverse working conditions.

Contraindicated conditions for CG with frequent and severe exacerbations, severe, often relapsing erosive, atrophic diffuse, special forms of CG:
- work involving regular or occasional considerable physical and mental stress, body shaking, exposure to toxins and gastrotropic allergenic substances (vapours of acids and alkalis, chemicals), chemical carcinogens (polycyclic aromatic hydrocarbons, etc.);
- work involving frequent business trips and irregular working hours, inability to follow the diet.

Invalidity

CG is rarely the main cause of disability.

Rehabilitation for patients includes:
- balanced diet;
- avoidance of alcohol consumption and smoking;
- treatment is individually indicated depending on the disease nature considering the type of the disease, its activity and severity:
  - Identification and treatment of opportunistic diseases;
  - sanation of the oral cavity;
  - spa treatment;
• phytotherapy

**Peptic ulcer and duodenal ulcer**

**Peptic ulcer (PU),** is defined by WHO as a total chronic relapsing disease prone to progression, with polycyclic course, characteristic features of which are seasonal exacerbation accompanied by the appearance of the ulcer in the lining of the stomach or duodenum and the development of complications that threaten the patient's life. PU is a feature of the current involvement in the pathological process of all the digestive tract.

**Etiology**

In the etiology of ulcer important is the genetic predisposition (HLA antigens B5, B14, B15, increasing the number of parietal cells over-producing hydrochloric acid, with a deficit of alpha-2 macroglobulin and alpha-l-antitrypsin fukoglikoproteids in gastric mucus, which protects the mucosa from damaging action of proteolytic enzymes), neuro-emotional factors, impaired motor-evacuation function of the stomach and duodenum, persistence of helicobacter pylori in the mucosa, and the systematic violation of the food, bad habits (smoking, alcohol), adverse effects of certain drugs (a group of non-steroidal anti-inflammatory drugs, glucocorticoid hormones, etc.).

The task of local therapist in the initial examination of the patient with the syndrome of gastric dyspepsia and epigastric pain is early detection (or exclusion) of peptic ulcer. Therefore following measures should be held:

a) focused and detailed questioning of patient complaints,
b) identification of risk factors for peptic ulcer disease, including the evaluation of the "family" of history,
c) clinical examination of the patient, first of all, to avoid the possibility of surgical pathology
g) provide a patient (if necessary) with emergency medical care,
d) choice of terms and conditions (clinic or hospital) of following examination (primarily fibrogastroduodenoscopy for an objective assessment of gastric and duodenal ulcer, biopsy and HP detection and treatment of the patient,
e) assessment of patient disability,
g) choice of optimal treatment and preventive treatment.
Classification of peptic ulcer (PY Grigoriev, M. Boger 1986)

Localization of ulcer:
1. Stomach.
2. Duodenum.
3. Stomach and duodenum simultaneously.

Phase of peptic ulcer:
1. Aggravation.
2. Incomplete remission.
3. Remission.

Morphologic substrate of the disease:
1. Acute ulcer.
2. Active ulcer.
3. Ulcer scar (scar red phase, the phase of the white scar).
4. Chronic ulcer.
5. Postulcerous deformation and postulcerous scar.
6. Gastritis, indicating the location and morphological variant.
7. Duodenitis with morphological variant.
8. Duodenogastric reflux.
9. Gastroesophageal reflux, esophagitis.

Peptic ulcer course:
1. Latent.
3. Moderate.
4. Severe.

Complication:
1. Bleeding.
2. Perforation
3. Penetration
4. Perivisceritis.
5. Pyloric stenosis.
6. Malignancy
7. Reactive hepatitis.
8. Reactive pancreatitis.

E.S. Ryss offers classification, convenient in practical application.

The main characteristics:
1. According to ulcer localization: in the stomach, duodenum,
combined location, other departments of gastrointestinal tract.

2. According to form: a) acute (fresh), b) chronic (recurrent or persistent).

3. According to phase course: worsening, exacerbation subsides (subremission), remission.


Optional characteristic
1. More precise information on the clinical course: light, moderate and heavy.

2. Specification of ulcer localization: – in the stomach: cardial, subcardial, fundic (mediagastric), prepyloric (antral) and the pyloric channel; in the duodenum: bulbar, postbulbar – combined gastric and duodenal ulcers.

3. Information on the nature of gastric secretion (normal, increased, decreased).

Clinical picture

The classical clinical picture consists of ulcer pain, dyspeptic (belching, heartburn, loss of appetite or increased hunger, nausea, vomiting, etc.) and asthenic-vegetative syndrome (fatigue, weakness, sweating, irritability, sleep disturbances). It is necessary to give clinical features, depending on the ulcer location.

Cardiac and subcardial ulcers are located at the esophagogastric passage, mostly occur in men older than 45 years, it is accompanied by pain 15-20 minutes after meals. Pains are are located high in the epigastrium, they often irradiate to the precordium, and therefore require a differential diagnosis of coronary artery disease, left-sided pleurisy, often combined with hernia esophageal orifice and reflux esophagitis and in half the cases (according to the V.H. Vasilenko) lead to bleeding.

Ulcers of the lesser curvature of the stomach are characterized by quite intense pain 1-1,5 hours after a meal, are manifested by various dyspeptic phenomena with the retained gastric secretion.

Ulcers of the greater curvature of the stomach are rare, and characterized by subtle disease course. It should be considered as suspicious malignant – primary or secondary form of cancer of the
Antral ulcers – "prepyloric" is a favorite site of primary gastric cancers of the stomach, especially in elderly persons. It is clinically manifested as duodenal ulcers, although usually accompanied by a less evident hyperacidity, confirming pattern "regarding gastric secretion decreases as you get closer to the cardia ulcers".

Pyloric channel ulcers are manifested by intense late, "hunger" or nocturnal pain in the right side of the epigastric region, irradiating to the posterior or upper section of the back. They are manifested by repeated vomiting with sour liquid mass accompanied by evacuation disorders of chyme from the stomach due to pyloric spasm and expressed inflammation of the gastric mucosa, tend to formation of pyloric stenosis, as well as penetration, perforation and massive bleeding.

Postbulbar ulcers. According to Vasilenko V.H not more than 5-7% of all gastroduodenal events located in the upper curve in the initial segment of the descending part of the duodenum, they occur usually in men and are characterized by the intensity of pain, irradiating mostly under the right shoulder-blade or interscapulum, simulating cholecystitis, pancreatitis, enterocolitis; characteristic sign of postbulbar ulcer is an acute and recurrent gastro intestinal bleeding, manifested by chalking, increasing weakness, sweating, dizziness, nausea and other symptoms of acute hemorrhagic anemia.

Combined gastric and duodenal ulcers are detected in 5-10% of patients. Duodenal ulcers usually develop first, the simultaneous development occurs or there is a change in the localization in further exacerbations, is manifested by almost constant exacerbations.

Giant gastric and duodenal ulcers. The sizes of ulcer are over 2 (other sources – from 3 to 5) cm in diameter. This type takes about 5% of the total number of ulcers and occurs mainly in elderly and senile patients due to the severity of venous disorders. They are more often localized along the small curvature and require obligatory histological and oncological observation, prone to massive bleeding, penetration and perforation, prompting surgeons to surgery.

Nota bene! Clinical course of prepyloric ulcers is similar to the course of duodenal ones, but can possibly lead to malignancy.
Standard examination of patients with peptic ulcer:
- dynamics of body weight,
- Clinical blood and urine analysis, reticulocytes (in abnormality the test should be repeated in 10 days)
- Blood type, Rh factor,
- Blood sugar, serum iron,
- Coprocystoscopy,
- Fecal occult blood
- Fibrogastrroduodenoscopy (with biopsy and research on Helicobacter pylori – histology, cytology, urease test)
- X-ray of the stomach and duodenum (mainly for the assessment of motoevacuatory function)
- Study of gastric secretion (histamine or pentagastrin) and (or) – pH-metry,
- Ultrasound of the liver, biliary and pancreatic tracts.

Additional studies are conducted for suspected malignant degeneration (atypical cells), complications and co-morbidities.

Crucial to the diagnosis is endoscopic ulcer examination. But it is necessary to assess the clinical constellation of the clinical symptoms, as ulcer defect of gastrointestinal tract mucosa may be one of developing severe pathologies of internal organs.

**Medical therapy**

Local physician should give the patient detailed advice on diet and dietary characteristics (characteristics of diet, cooking, calorie), which is especially important in an aggravation of the process.

Bed rest is appropriate to comply with an exacerbation of the disease in the future – adherence to adequate physical activity, rational psychotherapy.

Important is the removal of harmful factors – smoking, alcohol, drugs, damaging the mucous membrane of the stomach.

Drug therapy depends on the phase of the process (worsening, remission), the nature of the disease, presence of complications and comorbidities, aims not only at rapid healing of the ulcer, but most important, at prevention of the disease recurrence and prevention of complications.

The main groups of drugs for the treatment of ulcer:

1. *Drugs suppressing H. pylori infection* (de-nol, metronidazole,
furazolidone, oxacillin, clarithromycin and other antibiotics).

2. *Antisecretory agents:*
   - M-selective anticholinergics (gastrocepin, pirencepin).
   - Blockers H2 histamine receptor (cimetidine, ranitidine – Zantac. Raniberl, ranisan, famotidine - kvamatel, ulfamid; roksatidin, nizatidine).
   - Blockers of H+, K+ ATPase (proton pump) – omeprazole, lansoprazole, pantoprazole, rabeprazole, esomeprazole.


4. *Gastrocytoprotectors:*
   - Stimulating mucosa formation (carbenoxolone, enprostil, Cytotec.).
   - Form a protective film (sucralfate – Venter, alsukral, colloidal bismuth – De-Nol, smectite – smectite dioptagidral).
   - Coating agents and astringents (bismuth preparations).

5. *Drugs normalizing the motor function of the stomach and duodenum* (Reglan, metoclopramide, domperidone, cisapride, eglonil, sulpiride), antispasmodics (no-spa, papaverine, Duspatalin).


According to the modern concept of treatment of *Helicobacter pylori* infection (the Maastricht 3-2006 consensus, Rome), absolute and relative indications for therapy of H. pylori are distinguished.

*Absolute indications* include:
- Duodenal ulcer or gastric ulcer (active or inactive, including peptic ulcer disease);
- Maltoma;
- Atrophic gastritis;
- Condition after gastrectomy for gastric cancer;
- First line relatives of the patients with gastric cancer;
- Patient’s wish (after thorough consultation with the doctor).

*Relative indications* include:
- Functional dyspepsia;
- Gastroesophageal reflux disease;
- Treatment with nonsteroidal anti-inflammatory drugs.

The following drug combinations and schemes for eradication of HP:
Seven-day scheme:

1) **omeprazole** (zerocid, omizak) 20 mg two times a day (morning and evening, every 12 h) or pantoprazol (kontrolok) 40 mg 2 times a day + **clarithromycin** (klacid) at 250 mg 2 times a day + **amoxicillin** (flemoksin-Solutab, ospamox, hikoncil) 1000 mg two times a day;

2) **omeprazole** (zerocid, omizak) 20 mg two times a day (morning and evening, every 12 h) or pantoprazol (kontrolok) 40 mg 2 times a day + **amoxicillin** (hikontsil) or ampicillin and 1 g 2 times a day at the end of a meal + **metronidazole** (Trichopol) 500 mg 2 times a day at the end of a meal;

3) **pilorid** (ranitidine) 400 mg to 2 times a day after meals + **clarithromycin** (klacid) 250-500 mg or 500 mg of tetracycline or amoxicillin 1 g 2 times daily + **metronidazole** (Trichopol) 500 mg 2 times a day at the end of a meal;

4) **omeprazole** (zerotsid, omizak) 20 mg 2 times a day (morning and evening, every 12 h) or pantoprazol (kontrolok) 40 mg 2 times a day + **colloidal bismuth subcitrate** (ventrisol, De-Nol), 120 mg + **metronidazole** or tinidazole 500 mg 2 times a day after meals + **tetracycline** or amoxicillin 500 mg 4 times a day.

Ten-day scheme:

1) **ranitidin** (Zantac), 300 mg two times daily or famotidine (kvamatel) 40 mg 2 times a day at intervals of 12 h + **bismuth drug** (De-Nol, ventrisol 120 mg 3 times a day 30 minutes before a meal and for the 4th time at night) + **metronidazole** 250 mg four times a day after meals + **tetracycline** 250 mg 4 times per day after meals. Eradication rate reaches 90%.

After the end of the combined 5-week eradication therapy for duodenal and 7 week therapy for gastric ulcer one of the following drugs should be used: ranitidin (Zantac) – 300 mg before bedtime, famotidine (kvamatel) – 40 mg at bedtime, 400 mg pilorid in the morning and evening.

For the prevention of gastric and duodenal ulcers relapse two types of treatment are recommended:

1) continuous (lasting months and even years) antisecretory maintenance therapy means a half of the daily dose, for example,
150 mg of ranitidine every evening or 20 mg or 40 mg of kvamatel, pantoprozol (kontrolok).

*Indications for this program:*
- Inefficient eradication therapy;
- Complications of pyloric ulcer (ulcer bleeding or perforation in history);
- Comorbidities, requiring the use of non-steroidal anti-inflammatory drugs;
- Accompanying erosive or ulcerative esophagitis;
- Annually recurrent peptic ulcers, despite adequate therapy coursework.

2) Preventive therapy "on demand", which provides the appearance of characteristic symptoms of acute peptic ulcers, administration of one of antisecretory drugs (ranitidine, famotidine, kvamatel, omeprazole kontroloka) in total daily dose for 2-3 days, and then in half – in for 2 weeks. If after such a course of exacerbation symptoms disappear completely, treatment can be stopped, but if symptoms persist or recur, it is necessary to conduct fibrogastroduodenoscopy and other surveys. This variant of treatment may be applied only by attentive and disciplined patients who are adequately assessed and promptly react to changes in their health.

**For the treatment of gastroduodenal ulcers not associated with HP** (negative morphological and urease tests taken from antrum and body of the stomach), use one of the following combinations and schemes:

1) **ranitidine** – 300 mg once a day preferably in the evening and antacid (Maalox, remagel, Aluminium phosphate gel) as a symptomatic treatment;

2) **famotidine** (kvamatel, famotsid) – 40 mg once a day preferably in the evening and antacid (Maalox, remagel, Aluminium phosphate gel) as a symptomatic treatment;

3) **sucralfate** (Venter, sukrat gel) – 4 grams per day (1 g for 30 minutes before eating and 4th dose at night 2 hours after a meal) for 4 weeks, then 2 grams per day for 8 weeks.

Antacids can be used as additional drugs (almagel, Aluminium phosphate gel, Maalox, Gustav, gelusil), prokinetic agents
(metoclopramide, domperidone, cisapride), gastroprotective drugs (sucralfate, colloidal bismuth), herbal medicine (flax seed, psyllium, mint and lemon etc.). Physiotherapy treatment is provided in the absence of contraindications.

Active counseling of patients with GU and DU is carried out by district physician and includes:

1) regular (at least twice a year, especially in spring and autumn) medical supervision and physical examination of patients with ulcerative process assesses the dynamics of the process. It is necessary to identify complications and comorbidities (total blood count, urinalysis, coprocytogram, fecal occult blood test, study of gastric secretion, fibrogastroduodenoscopy, ultrasound of the abdomen);

2) assessment of the patient's ability to work, in the presence of professional risk factors – timely consultation at MCC, in disability criteria are detected the patients should be referred to MREB;

3) active individualized rehabilitation treatment; recreational activities;
   a) different options of preventive treatment;
   b) sanatorium treatment.

Criteria for temporary disability in case of peptic ulcer:
   a) ulcer diagnosed for the first time;
   b) signs of acute peptic ulcer disease or its complications;
   c) need for surgical treatment.

Treatment in outpatient settings, particularly in the outpatient clinic, is provided for:
- Patients with moderate and severe, but not resistant pain,
- Patients with uncomplicated peptic ulcer disease,
- Patients with the absence of severe comorbidity.

Criteria for hospitalisation in case of peptic ulcer
1) Emergency admission:
   - Acute complications of peptic ulcer.
   - Patients with ulcers of different sizes with the threat of bleeding – the presence of thrombosed vessels and (or) dark-colored plaque on the bottom of the ulcer.

2) Planned admission:
   - First observed ulcer of any location (within 2 weeks of the
appearance of complaints).
- Stomach ulcer.
- Combined gastric and duodenal ulcer.
- Large ulcer.
- Severe illness.
- The lack of therapy effect on an outpatient basis.
- Suspicion for malignant ulcers.
- Exacerbation due to related diseases, aggravating the course of disease (respiratory, heart failure, severe hypertension, diabetes mellitus, receiving steroid therapy).
- Exacerbation in the elderly, somatically debilitated patients.
- The presence of the relative indications for surgical treatment (failure of medical treatment, repeated bleeding, callous gastric ulcer, recurrent ulcer after suturing of perforated ulcer, etc.).
- Decision of expert questions.

The criteria for evaluation of working ability in patients with ulcer are:
- Phase of the process (exacerbation or remission);
- The severity and course of acute illness;
- Characteristic features of ulcers (location, size, stage) and functional-morphological status of the gastroduodenal system;
- Developed complications;
- The effectiveness of previous treatment;
- Co-morbidities;
- Social factors (occupation, nature and conditions of work);
- Psychological characteristics (installation work).

Referral to MREB
If a patient is referred to MREB the appointment card should contain the most important information from the "ulcer" anamnesis, necessity of outpatient and inpatient care, the volume of treatment and its results, the effectiveness of preventive treatment. Additional research methods are following:
- Fibrogastroduodenoscopy with biopsy results (in case of inability to fulfill fibrogastroduodenoscopy-X-ray examination).
- Study of gastric secretion (using histamine or pentagastrin), pH-metry.
- Height, weight of the patient in the disease dynamics.
- Total blood count (dynamic indicators for anemia), urinalysis, fecal occult blood, coprocytogram.
- Biochemical serum (glucose, total protein, transaminase, cholesterol, lipoproteins, electrolytes, urea, amylase, alkaline phosphatase).
- Ultrasound of the abdomen.
- The decision of gastroenterologist, surgeon, oncologist (if necessary).

Surgical consultation is performed for the diagnosis of pyloric stenosis ulcer and duodenal ulcer, and in case of ineffectiveness of 3-4 courses of complex conservative treatment of deep callous gastric ulcer, continuous-recurrent course.

**Ulcer of the stomach and duodenum has the following diagnostic indications for Sanatorium Treatment:**

PU in remission, partial remission or exacerbation if no motor impairment of the stomach is present, tendency to bleeding, penetration and suspected malignant transformation – local sanatoriums and resorts with drinking mineral water and mud are recommended.

Operated stomach diseases with dumping syndrome and hypoglycemic syndrome (mild to moderate) require sanatorium treatment, but not earlier than 1 month after a surgery, and health resort treatment should be introduced not earlier than 2 months in case of satisfactory general condition and (resorts with drinking mineral water and mud).


**Contraindications for sanatorium treatment:**
- Gastric and duodenal ulcer in the acute phase.
- Gastric ulcer complicated by subcompensated stenosis, with re-bleeding noted in the last 8-10 months.
- Penetrating ulcer.
- Complications after gastric surgery/non-healing postoperative scar, fistula, afferent loop syndrome, dumping syndrome, severe hypoglycemic syndrome, atony of the gastric stump.

Single profuse bleeding in a year after a surgery is not a contraindication for sanatorium treatment.
Notes
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Пособие
dля студентов 4 курса факультета иностранных учащихся
(изд. на английском языке)

OUTPATIENT THERAPY
Handbook
for 4th year foreign students

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