Lecture complex

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№1

1. Theme: Lobar pneumonia. Bronchopneumonia. Bronchiectasis. Emphysema. Bronchial asthma.

2.Purpose: to know the etiology, pathogenesis of acute lung disease, their classification, morphological characteristics, complications, outcomes, diagnose them by macroscopic and microscopic picture.

3. Theses of the lecture: Lobar pneumonia — an acute infectious-allergic disease that affects one or more lobes of the lung (lobar, lobar pneumonia), alveoli appears fibrinous exudate (fibrinous, or croupous, pneumonia) and pleura fibrinous overlay (pleuropneumonia). Etiology and pathogenesis. The causative organism is the pneumococcus I, II, III and IV; in rare cases lobar pneumonia is caused by diplobacillus Friedlander. The acute onset of large-scale pneumonia among complete health and in the absence of contacts with patients, as well as the carriage of pneumococci by healthy people, allows to link its development with auto-infection. Morphogenesis, pathological anatomy. Existing for over 100 years, the classic conception, croupous pneumonia, which should be viewed as parenchymatous, in its development goes through 4 stages: tide, red obucheniya, gray obucheniya. All stages take 9-11 days. The tide stage lasts 24 hours and is characterized by sharp hyperemia and microbial edema of the affected lobe; a large number of pathogens are found in the edematous fluid. There is an increase in capillary permeability, the beginning of diapedesis of red blood cells in the lumen of the alveoli. A few light compacted, dramatically fulfilling.

Stage red obucheniya occurs on the 2nd day of illness. Against the background of full blood and microbial edema increases diapedesis of red blood cells that accumulate in the lumen of the alveoli. Neutrophils are added to them, fibrin threads fall out between the cells. Stage gray obucheniya occurs on the 4-6-th day of illness. In the lumen of the alveoli accumulate fibrin and neutrophils, which together with macrophages phagocytic disintegrating pneumococci. You can see how the fibrin threads through the interalveolar pores penetrate from one alveolus to another. The number of red blood cells subjected to hemolysis, decreases, and the intensity of hyperemia. The stage of approval comes on the 9-11-th day of illness. Fibrinous exudate under the influence of proteolytic enzymes of neutrophils and macrophages undergoes melting and resorption. There is a purification of the lung from fibrin and pneumococci: exudate is eliminated by lymphatic drainage of the lung and sputum. Fibrinous overlays on the pleura dissolve. Complications. Distinguish between pulmonary and extrapulmonary complications of the croupous pneumonia.

Pulmonary complications develop due to a violation of the fibrinolytic function of neutrophils. In case of insufficiency of this function, the mass of fibrin in the alveoli is subjected to organization, i.e., germinate by granulation tissue, which, when ripe, turns into a Mature fibrous connective tissue.

This process of organization is called carnification (from lat.sagpo-meat). Extrapulmonary complications are observed during the generalization of the infection. With lymphogenic generalization, purulent mediastinitis and pericarditis occur, with hematogenous — peritonitis, metastatic ulcers in the brain, purulent meningitis, acute ulcerative or polypous ulcerative endocarditis, more often than the right heart, purulent arthritis, etc.

Death with croup pneumonia comes from heart failure (especially often in the elderly, as well as chronic alcoholism) or from complications (brain abscess, meningitis, etc.). Bronchopneumonia is an inflammation of the lungs developing in connection with bronchitis or bronchiolitis (bronchoalveolar). Etiology. The disease has a diverse etiology. It can cause a variety of microbial agents-pneumococci, Staphylococcus, streptococci, enterobacteria, viruses, Mycoplasma, fungi and other Pathogenesis. Development of bronchopneumonia is associated with the acute bronchitis or bronchiolitis, the inflammation often spreads to lung tissue intrabronhialno (downward way, usually in catarrhal bronchitis or bronchiolitis), less peribronchial (usually in destructive bronchitis or bronchiolitis). Pathological anatomy. The walls of the bronchi and bronchioles thicken due to edema and cellular infiltration. In the distal bronchi often arises unbranched and panbronchiolitis, and in the proximal — endometrosis. Edema and cellular infiltration of the bronchial wall violate the drainage function of the bronchi, which contributes to the aspiration of infected mucus in the distal bronchial tree; cough tremors may appear transient bronchial lumen expansion — transient bronchiectasis.

Bronchopneumonia has some features in different age periods. In newborns with pneumonia on the surface of the alveoli, the so-called hyaline membranes consisting of compacted fibrin are often formed. In weakened children up to 1-2 years, the foci of inflammation are localized mainly in the posterior, adjacent to the spine and not completely straightened after birth lung segments (11, VI and X segments). This pneumonia is called paravertebral.

Complications. To a large extent, complications of bronchopneumonia depend on the characteristics of their etiology, age and General condition of the patient. Foci of pneumonia can be subjected to carnification or suppuration with the formation of abscesses; if the focus is located under the pleura, pleurisy is possible.

The death of patients may be due to suppuration of the lung, purulent pleurisy. Especially life-threatening bronchopneumonia in early childhood and old age.

Bronchiectasis-bronchial expansion in the form of a cylinder or bag, which can be congenital and acquired. Congenital bronchiectases occur relatively rarely (2-3% in relation to the total number of cnsl) and develop due to violations of the formation of the bronchial tree. Sometimes cysts are formed (the so-called cystic lung), as in the parenchyma of the lung blindly end small bronchi. Histological sign of congenital bronchiectasis is a disorderly location in the wall of the structural elements of the bronchus. Congenital bronchiectases are usually detected when suppuration of their contents. Acquired bronchiectasis is a consequence of chronic bronchitis.

The cavity of bronchiectasis is lined with prismatic epithelium, but often multi-layer flat, resulting from metaplasia. In the wall of bronchiectasis there is chronic inflammation, elastic and muscle fibers are significantly destroyed and replaced by connective tissue. In the cavity bronhoektaza a purulent content.

Emphysema of the lungs (from Greek.emphysao-swell) called the disease, which is characterized by excessive air content in the lungs and increase their size. There are the following types of emphysema: chronic diffuse obstructive; chronic focal (perifocal, scar); vicar (compensatory); primary (idiopathic) panacinar; senile (emphysema in the elderly); inter-daily.

Chronic diffuse obstructive pulmonary emphysema. This type of emphysema is particularly common.

Etiology and pathogenesis. The development of this type of emphysema is associated with previous chronic bronchitis and bronchiolitis and their consequences —multiple bronchiectasis, pneumosclerosis. Emphysema affects the elastic and collagen framework of the lung due to the activation of leukocyte proteases, elastase and collagenase. Pathological anatomy. The lungs are enlarged in size, cover the front mediastinum with their edges, swollen, pale, soft, do not fall off, cut with a crunch. From the lumen of the bronchi, the walls of which are thickened, squeezed mucopurulent exudate. The bronchial mucosa is full-blooded, with an inflammatory infiltrate, a large number of goblet cells; there is an uneven hypertrophy of the muscle layer, especially in small bronchi. Stretching the walls of the acinus leads to stretching and thinning of elastic fibers, expansion of alveolar passages, alveolar septum changes. The walls of the alveoli become thinner and straightened, mezhalveolyarnyh pores dilate capillaries zapustevayut.

Chronic focal emphysema. This emphysema develops around old tuberculous foci, postinfarction scars, more often in the I-II segments. Therefore, it is called perifocal, or scar. Chronic focal emphysema usually panacinary: in dilated acini, there is complete smoothing of the walls, formed smooth-walled cavity, which mistakenly can be taken when screening for tuberculous cavities. In the presence of several cavities (bubbles) speak of bullous emphysema. Located under the pleura bubbles can break into the pleural cavity, developing spontaneous pneumothorax.

Vicar (compensatory) emphysema of one lung is observed after removal of a part of his or her lung or another. This type of emphysema is accompanied by hypertrophy and hyperplasia of the structural elements of the remaining lung tissue.

Primary (idiopathic) panacinar emphysema is very rare, its etiology is unknown. Morphologically, it is manifested by atrophy of the alveolar wall, reduction of the capillary wall and severe hypertension of the small circle of blood circulation.

Senile emphysema is considered as obstructive, but developing due to age-related lung involution. Therefore, it is correct to call emphysema in the elderly.

Inter-daily emphysema is fundamentally different from all other species. It is characterized by the flow of air into the inter-daily lung tissue through the ruptures of the alveoli in patients with enhanced cough movements. Air bubbles can spread into the mediastinal tissue and subcutaneous tissue of the neck and face (subcutaneous emphysema). When pressing on the inflated air areas of the skin heard a characteristic crunch (crepitation).

Asthma (from the Greek.asthma-suffocation) - a disease in which there are attacks of expiratory dyspnea caused by an allergic reaction in the bronchial tree with a violation of bronchial patency.

Etiology, pathogenesis, classification. Factors that cause bronchial asthma, consider mainly exogenous allergens with the undoubted role of heredity. Among the reasons for the repeated attacks of asthma, secrete infectious diseases, especially upper respiratory tract, allergic rinosinusopatii, the external environment, exposure to substances suspended in the air (dust room and production, smoke, various smells, etc.), meteorological (humidity of atmospheric air, mists) and psychogenic (psychogenic stimulation) factors, the use of certain food products and medicines. On the basis of the leading participation of a causal factor talk about infectious, allergic, occupational, psychogenic (psychological), bronchial asthma, due to the influence of the environment, and its other forms.

Infectious-allergic bronchial asthma is observed under the influence of allergens on patients with acute or chronic bronchopulmonary diseases caused by infectious agents.

Pathological anatomy. Changes in the bronchi and lungs in bronchial asthma can be acute, developing at the time of the attack, and chronic, which is a consequence of repeated attacks and a long course of the disease.

In the acute period (during the attack) of bronchial asthma in the bronchial wall there is a sharp fullness of blood vessels of the microcirculatory bed and an increase in their permeability. Develop swelling of the mucous membrane and submucosal layer, infiltration of their labrocytes, basophils, eosinophils, lymphoid, plasma cells. With repeated attacks of bronchial asthma over time, diffuse chronic inflammation, thickening and hyalinosis of the basal membrane, sclerosis of the interalveolar septum, chronic obstructive emphysema of the lungs develop in the bronchial wall. There is a desolation of the capillary bed, there is a secondary hypertension of the small circle of blood circulation, leading to hypertrophy of the right heart and ultimately — to cardiopulmonary failure.

4. Illustrative material:

1. Presentation of lecture material;
2. Posters on the topic of classes;
3. Tables.
4. Macropreparations on the topic.

5. Literature: "Annex 1."

6. Control question. (feedback):

1. What is bronchopneumonia? This definition.
2. What is the classification of respiratory diseases?
3. What is lobar pneumonia? This definition.
4. List the stages of lobar pneumonia?
5. What is primary pneumonia?
6. What is secondary pneumonia? .
7. What are pulmonary complications of lobar pneumonia?
8. What is bronchiectasis of the lung? This definition.
9. What are the etiology and pathogenesis of bronchiectasis of the lung?
10. What are the macroscopic and microscopic characteristics of bronchiectasis.
11. What are the complications, outcomes and causes of death in bronchiectasis?
12. What are the etiology, pathogenesis and classification of lung emphysema.
13. What are the macroscopic and microscopic characteristics of emphysema.
14. What are the complications, outcomes and causes of death in emphysema?

№2

1. Theme: Rheumatism.

2. Objective: To learn how to determine the etiology, pathogenesis, pathological anatomy, morphogenesis of rheumatic diseases, as well as to be able to distinguish between clinical and morphological forms of rheumatic diseases and their most frequent complications.

3.Thesis of the lecture: Rheumatism (Sokolsky — Buyo disease) is an infectious and allergic disease with a predominant lesion of the heart and blood vessels, wave-like course, periods of exacerbation (attack) and subsiding (remission). Alternating attacks and remissions may continue for many months and even years; sometimes the rheumatism is taking latent period.

Etiology. In the emergence and development of the disease proved the role of b-hemolytic Streptococcus group A, as well as the sensitization of the body Streptococcus (recurrent angina). The importance of age and genetic factors (rheumatism — polygenic inherited disease) is given.

Pathogenesis. In rheumatism there is a complex and diverse immune response (hypersensitivity reactions of immediate and delayed types) to numerous antigens of Streptococcus. The main importance is attached to antibodies that cross-react with Streptococcus antigens and antigens of heart tissues, as well as cellular immune reactions. Morphogenesis. Structural basis of rheumatic fever are systemic prog russiausa disorganization of connective tissue, blood vessels damage, particularly to the microvasculature, and immunopathologic processes. To the greatest extent, all these processes are expressed in the connective tissue of the heart (the main substance of the valvular and parietal endocardium and, to a lesser extent, the leaves of the heart shirt), where all phases of its disorganization can be traced: mucoid swelling, fibrinoid changes, inflammatory cell reactions and sclerosis.

Mucoid swelling is a surface and reversible phase of connective tissue disorganization and is characterized by increased metachromatic reaction to glycosaminoglycans (mainly hyaluronic acid), as well as hydration of the main substance.

Fibrinoid changes (swelling and necrosis) are a phase of deep and irreversible disorganization: layering on mucoid swelling, they are accompanied by homogenization of collagen fibers and their impregnation with plasma proteins, including fibrin.

Cellular inflammatory reactions are expressed by the formation of a specific rheumatic granuloma. The formation of granulomas begins with the moment of fibrinoid changes and is characterized initially by the accumulation of macrophages in the lesion of the connective tissue, which are transformed into large cells with hyperchromic nuclei.

Pathological anatomy. The most characteristic changes in rheumatism develop in the heart and blood vessels.

Pronounced dystrophic and inflammatory changes in the heart develop in the connective tissue of all its layers, as well as in the contractile myocardium.They mainly determine the clinical and morphological picture of the disease.

Endocarditis-inflammation of the endocardium-one of the clearest manifestations of rheumatism. On localization distinguish endocarditis valve, chordally and parietal. The most pronounced changes develop in the mitral or aortic valve valves. Isolated lesion of the right heart valves is very rare and in the presence of endocarditis of the left heart valves.

In rheumatic endocarditis, there are dystrophic and necrobitic changes in the endothelium, mucoid, fibrinoid swelling and necrosis of the connective basis of the endocardium, cell proliferation (granulomatosis) in the thickness of the endocardium and thrombosis on its surface. The combination of these processes can be different, allowing you to select several types of endocarditis.

Diffuse endocarditis, or valvulitis [according to V. T. Talalaev], is characterized by diffuse lesions of valve flaps, but without changes in the endothelium and thrombotic overlays. Acute warty endocarditis is accompanied by damage to the endothelium and the formation of thrombotic overlays in the form of warts along the closing edge of the valves (in places of endothelial damage).

Fibroplastic endocarditis develops as a consequence of the two previous forms of endocarditis with a special tendency of the process to fibrosis and scarring. Recurrent warty endocarditis is characterized by repeated disorganization of the connective tissue of the valves, changes in their endothelium and thrombotic deposition on the background of sclerosis and thickening of the valve valves. In the outcome of endocarditis, sclerosis and endocardial hyalinosis develop, which leads to its thickening and deformation of the valve valves, i.e. to the development of heart disease.

Myocarditis-inflammation of the myocardium, constantly observed in rheumatism. There are 3 forms of it: 1) nodular productive (granulomatous);

2) diffuse exudative interstitial; 3) focal exudative interstitial.

Nodular productive (granulomatous) myocarditis is characterized by the formation of rheumatic granules in the perivascular connective tissue of the myocardium. Granulomas, which are recognized only by microscopic examination, are scattered throughout the myocardium, the largest number of them occurs in the left atrium ear, in the interventricular septum and the posterior wall of the left ventricle. Granulomas are in different phases of development. "Blooming ("Mature") granulomas are observed during the attack of rheumatism," fading "or" scarring " — during remission. In the outcome of nodular myocarditis, perivascular sclerosis develops, which increases with the progression of rheumatism and can lead to severe cardiosclerosis.
Diffuse interstitial exudative myocarditis described by M. A. Skvortsov, characterized by edema, hyperemia of interstitium of the myocardium and a significant infiltration of it by lymphocytes, histiocytes, neutrophils and eosinophils. Rheumatic granulomas are extremely rare, and therefore talk about nonspecific diffuse myocarditis. The heart becomes very flabby, expanding its space, the contractile force of the myocardium in connection with developing it degenerative changes dramatically disturbed. This form of rheumatic myocarditis occurs in childhood and can quickly end with decompensation and death of the patient. With a favorable outcome, diffuse cardio-sclerosis develops in the myocardium.
Focal daily exudative myocarditis is characterized by a slight focal infiltration of myocardium by lymphocytes, histiocytes and neutrophils. Granulomas are rarely formed. This form of myocarditis is observed in the latent course of rheumatism.
Complications of rheumatism are more often associated with heart damage. In the outcome of endocarditis there are heart defects. Verrucous endocarditis can become a source of embolism of the vessels of the systemic circulation, raises the heart attacks the kidneys, spleen, retina, foci of softening in the brain, gangrene of limbs, etc. Rheumatic disorganization of connective tissue leading to sclerosis, especially expressed in the heart. Complication of rheumatism can be adhesions in the cavities (obliteration of the pleural cavity, pericardium, etc.).
Death from rheumatism can occur during an attack from thromboembolic complications, but more often patients die from decompensated heart disease.

4. Illustrative material:

1.presentation of lecture material;
2.posters on the topic of classes;
3.tables.
4. macropreparations on the topic.

5. Literature: "Annex 1."

6. Control question. (feedback):

1. What is rheumatic disease? This definition.

2.What are the etiology and pathogenesis of rheumatic disease?
3.What are the macroscopic and mikroskopicheskaya characteristic of rheumatic fever?
4.What are the complications, outcomes and causes of death in rheumatic disease?
5.What are the morphogenesis and pathological anatomy of heart defects?
6.Explain the outcome and complications, clinical and morphological types of heart defects?
7.Explain the causes of heart defects?

№3

1. Theme: Ischemic dystrophy of myocardium. Myocardial infarction. Coronary heart disease.

2. Objective: Learn to determine the etiology, pathogenesis, pathological anatomy, morphogenesis of heart disease, as well as to be able to distinguish between clinical and morphological forms of coronary heart disease and their most frequent complications.
3.Thesis of the lecture: Ischemic myocardial dystrophy, or acute focal myocardial dystrophy, develops in relatively short-term episodes of coronary crisis, when there are characteristic changes in the electrocardiogram, but enzymemia (increased activity of transaminases, lactate dehydrogenase, etc.) is absent, which is one of the proofs of the absence of myocardial necrosis.

The myocardium is flabby and pale, in the areas of ischemia are sometimes mottled and swollen. A fresh blood clot is often found in the coronary artery.

Macroscopic diagnosis of foci of ischemic dystrophy is possible with the help of tetrazolium salts, potassium tellurite. In areas of ischemia, where the activity of redox enzymes is sharply weakened, formazan grains and reduced tellurium do not fall out, so the areas of ischemia look bright on a dark background of unchanged myocardium.

Microscopically find paretic expansion of capillaries, stasis of erythrocytes, edema of interstitial tissue. You can join timezonename hemorrhages and leukodepleted, accumulations of leukocytes in the peripheral zone of ischemia. Muscle fibers lose their streak, devoid of glycogen, they are intensely stained with eosin, fuchsin, pyronine and Schiff reagent, indicating necrobiotic changes. Painted with acridine orange, they give a luminescent microscope is not orange, and green glow, which allows you to distinguish the ischemic area from the intact myocardium.

Complication of ischemic myocardial dystrophy is most often acute heart failure, it also becomes a direct cause of death. This is probably why clinicians usually refer to this form of coronary heart disease as "acute heart failure".

Myocardial infarction is an ischemic necrosis of the heart muscle, so clinically, in addition to changes in the electrocardiogram, it is characterized by enzymemia. As a rule, this is an ischemic (white) heart attack with a hemorrhagic Corolla.

Classification and pathological anatomy. Myocardial infarction is classified according to a number of features: 1) by the time of its occurrence; 2) by localization in different parts of the heart and heart muscle; 3) prevalence; 4) downstream.

Myocardial infarction is a temporary concept. It takes about 8 weeks from the moment of attack of myocardial ischemia-primary (acute) myocardial infarction.

If myocardial infarction develops after 8 weeks after the primary (acute), then it is called a repeated heart attack. A heart attack that developed within 8 weeks of the primary (acute) existence is called a recurrent myocardial infarction. Myocardial infarction is localized most often in the region of the apex, anterior and lateral walls of the left ventricle and anterior sections of the interventricular septum, i.e. in the pool of the anterior interventricular branchial coronary artery, which is functionally more burdened and more strongly affected by atherosclerosis than other branches. Less often, a heart attack occurs in the region of the posterior wall of the left ventricle and the posterior sections of the interventricular septum, i.e. in the pool of the enveloping branch of the left coronary

artery. When the main trunk of the left coronary artery and both its branches are subjected to atherosclerotic occlusion, extensive myocardial infarction develops. In the right ventricle and especially in the Atria, a heart attack develops rarely.

Complications of heart attack are cardiogenic shock, ventricular fibrillation, asystole, acute heart failure, myomalacia, acute aneurysm and cardiac rupture, parietal thrombosis, pericarditis.

Myomalacia, or melting of necrotic myocardium, occurs in cases of predominance of autolysis of dead tissue. Myomalacia leads to rupture of the heart and hemorrhage into the cavity of the heart shirt (hemopericardium and tamponade of its cavity).

Acute aneurysm of the heart, i.e. swelling of the necrotic wall of it, is formed with extensive heart attacks. The cavity of the aneurysm usually trombicula in the wall appear the tears of endocardium, blood enters these tears, detaches and destroys the endocardium necrotizing myocardium. There is a rupture of the heart and hemopericardium.

Parietal blood clots are formed in subendocardial and transmural heart attacks, they are associated with the risk of thromboembolic complications. Pericarditis, usually fibrinous, is often found in subepicardial and transmural heart attacks.

Death in myocardial infarction can be associated with both the myocardial infarction and its complications. The immediate cause of death in the early period of heart attack are ventricular fibrillation, asystole, cardiogenic shock, acute heart failure. Fatal complications of myocardial infarction in the later period are a rupture of the heart or its acute aneurysm with hemorrhage into the pericardial cavity, as well as thromboembolism (for example, cerebral vessels) from the heart cavities, when thromboembolism becomes a source of thromboembolism on the endocardium in the infarction, in the acute aneurysm, in the ears of the heart.

4. Illustrative material:

1. Presentation of lecture material;
2. Posters on the topic of classes;
3.Tables.
4. Macropreparations on the topic.

5. Literature: "Annex 1."

6.Control questions (feedback)

1. What is ischemic dystrophy of myocardium? This definition.
2. What is myocardial infarction? This definition.
3. How does the heart change in coronary heart disease?
4. Explain the clinical and morphological forms, complications of myocardial infarction?
5. What are the forms, stage acute ischemic heart disease (myocardial infarction)?
6. Describe the macroscopic, microscopic and ultrastructural characteristics of myocardial infarction at various stages of its development?
7. Describe the macroscopic and microscopic characteristics of chronic IHD?
8. Explain the complications and causes of death in chronic IHD?

№4

1. Theme: Atherosclerosis. Hypertensive disease.

2. Objective: To learn how to determine the etiology, pathogenesis, pathological anatomy, morphogenesis of cardiovascular disease, as well as to be able to distinguish between clinical and morphological forms of atherosclerosis, hypertension and their most frequent complications.

3.Thesis of the lecture: Atherosclerosis is a chronic disease affecting primarily the intima of large and medium-sized arteries and is characterized by fibrolipid plaques or atheromas. Atherosclerosis is the commonest arterial diseases.

Epidemiology. It is known that the most common cause of death (40%) is cardiovascular diseases, most of witch are related to atherosclerosis. Epidemiologic investigations on living populations have revealed a number of risk factors, which are associated with increased risk of developing clinical atherosclerosis.

The risk factors are often acting in combination rather than singly.

The first of them is age. Early atherosclerotic lesions may be present in childhood, but clinical manifestations will appear later. In scientific research, which have been performed at our department, morphological signs of atherosclerosis were found in the fetus and new-born aorta.

The second of risk factors is sex. The incidence and severity of atherosclerosis are higher in men than in women.

Genetic factors play a significant role in atherosclerosis. There is familial predisposition to atherosclerosis which may be related to other risk factors like diabetes, hypertension and hyperlipoproteinemia.

Geographic factors also play a role. There is a high incidence of atherosclerosis in the Europe, Australia, and US.

Many studies have demonstrated specific effects of diet including the amount of dietary cholesterol ingested on lipid and lipoprotein levels.

Hypertension is a major risk factor in atherosclerosis development. It acts probably by mechanical injury to the arterial wall due to increased blood pressure.

Other factors are diabetes mellitus, smoking, obesity, etc.

Pathogenesis. There are several patho-genesis theories of atherosclerosis: Virchow's, Rokitansky's, Anychkow's, Benditt's. Historically, two hypotheses for atherogenesis were dominant: one emphasized cellular proliferation in the intima as a reaction to insudation of plasma proteins and lipids from the blood, whereas the other postulated that organization and repetitive growth of thrombi resulted in plaque formation. The contemporary view of the pathogenesis of atherosclerosis incorporates elements of both older theories and is called the response to injury hypothesis. Formulated in 1973 and modified in 1986 and 1993, it states that atherosclerotic lesions are initiated as are sponse to some form of injury to arterial endothelium. The injury is a form of endothelial dysfunction without necessary denudation, which increases permeability to plasma constituents, including lipids, and permits blood monocytes and eventually platelets to adhere to endothelium. Monocytes adhere and subsequently enter the intima, transform into macrophages, and accumulate lipid to become foam cells, contributing to the evolution of the lesion. Factors released from activated platelets at the surface or monocytes then cause migration of smooth muscle cells from media into the intima, followed by proliferation and synthesis of extracellular matrix components by smooth muscle cells, leading to the accumulation of collagen and proteoglycans. Single or short-lived injurious events can be followed by restoration of endothelial function and regression of the lesion. Repeated or chronic injury, however, results in the development of an atheromatous plaque, probably by permitting continuing increased permeability, ingress of monocytes, or perhaps platelet interactions.

Morphology. Although the fibrous and complicated plaques are the atherosclerotic lesions associated with disturbances in blood front that cause clinical disease states, the morphologic changes of diffuse intimal thickening and fatty streaks may be either precursors of the basic atherosclerotic lesion or stages in its development.

1. Intimal thickening. Fibromuscular thickening of the intima of arteries may be a part of atherosclerotic process, particularly rapid during the first two decades of life. Macroscopically, the lesions may appear as small white areas of intimal cushion at the bifurcation and branching of arteries, or many be appear as «diffuse intimal thickening». Microscopically, the lesions consist of smooth muscle cells, fibrous tissue, some collagen but no lipid.

2. Fatty streaks especially prominent in the aorta and major arteries, more often on the posterior wall than the anterior wall. Macroscopically, the lesions may appear as flat or slightly elevated and yellow. They may be either in the form of small (about lmm in size) or in the form of elongated, beaded streaks. Microscopically, fatty streaks lying under the endothelium are composed of closely-packed foam cells, lipid-containing elongated smooth muscle cells and a few lymphoid cells. Small amount of extracellular lipid, collagen and proteoglycans are also present. The cholesterol esters in foam cells of fatty streaks differ chemically from those in the lipid core of atheromatous fibrous plague. These dissimilarities might be explained on the basis of two types of fatty streaks, which differ in morphology, cellular genotype, and lipid content, with one type related to the fibrous plague and the other type not related.

3. Fibrous plaque (atheroma). The lesions are raised, pearly white to grey, smooth-surfaced, plaque-like structures in the intima but may extend into the media, may encroach considerably on the vascular lumen, especially in muscular arteries such as coronary arteries. On cross section a typical lesion has a soft, yellow central zone with gruel-like material covered on the luminal aspect by a layer of dense fibromuscular tissue (fibrous cap).

In general, plaques are found most often in the abdominal aorta, large arteries of the lower limbs, carotid arteries, proximal portions of the coronary arteries and circle of Willis.

The histologic appearance of lesions varies considerably depending on the relative amounts of the different components. The central atheroma of an uncomplicated fibrous plague consist of acellular, amorphous, electron-dense material that contains lipids, cellular debris, fibrin and other plasma proteins. The fibrous cap is composed of avascular connective tissues and elongated smooth muscle cells covered by endothelium. Inflammatory cells, including macrophages, may be present. In older and more advanced lesions the collagen in the fibrous cap may be dense and hyalinised, smooth muscle cells may be atrophic and foam cells are fewer.

4. Complicated plaques develop from preexisting fibrous plaques as a result of one of a combination of several pathologic changes that include calcification, ulceration, thrombosis and haemorrhage. The complicated lesion is the most common type of atherosclerotic lesion that produces significant circulatory change and clinical disease.

a) Calcification the intima is brittle and cracks like an eggshell when the vessel is opened. Microscopically the dystrophic calcific process involves both the fibrous cap and the atheromatous portion of the plaque.

b) Ulceration and thrombosis. Advanced fibrous plaques with soft pultaceous atheromas, especially those with calcification may ulcerate as a result of mechanical or hemodynamic forces. With ulceration, cholesterol or lipid debris from the atheroma may be discharged and embolize. Murul thrombi may form on the ulcer or at sites of endothelial damage or mural hemorrhage. Such thrombi also may become organised and incorporated within the intimal plaque. Mural thrombi in medium-sized arteries may progress to occlusive thrombi that may reconalize.

c) Hemorrhage into atherosclerotic plaque is a common finding in advanced lesions, especially in the coronary arteries. The blood may reach the lesion from the vascular lumen through surface ulcerations or from rupture of capillaries that vascularized the atheroma from adventitial vasa vasorum.
d) With secondary changes in the media: 1) the internal elastic lamina is attenuated and fragmented; 2) smooth muscle cells and elastic lamina atrophy is connected a form of aneurysmal dilatation, which may follow, especially in elastic arteries.
Microscopically following states of atherosclerosis are distinguished:
1) pre-lipidosis, 2) lipidosis, 3) liposclerosis, 4) atheromatosis, 5) ulceration, 6) atherocalcification.
There are several clinico-morphological types of atherosclerosis according to localization. There are:
1) Atherosclerosis of aorta; 2) Atherosclerosis of coronary arteries; 3) Atherosclerosis of cerebral artery;
4) Atherosclerosis of arteries of lower extremities;
5) Atherosclerosis of arteries of small intestine;
6) Atherosclerosis of renal arteries.
The clinical effects result from the following:
1. Slow luminal narrowing causing ischemia and atrophy.
2. Suddenly luminal occlusion causing infarction necrosis.
3. Propagation of plaque by formation of thrombi and emboli.
4. Formation of aneurysmal dilation and eventual rupture.
The symptomatic atherosclerotic disease involves most often the heart, brain, kidneys, small intestine and lower extremities.
Some of the important effects are listed below: Aorta aneurysm formation, thrombosis and embolization to other organs; Heart — myocardial infarction, ischemic heart disease; Brain — chronic ischemic brain damage, cerebral infarction; Small intestine — ischemic bowel disease, infarction; Lower extremities — intermittent claudication, gangrene. Kidney — chronic ischemic renal damage, sclerosis, atrophy.
Most of them may be as a cause of death.

Hypertensive heart disease is an important and common form of heart disease in all worlds. Macroscopically, the most significant finding is marked hypertrophy of the heart, especially of the left ventricle. The weight of the heart increases to 500— 700 gm (may be 1000 gr) (normal weight is about 300 gr). The weight of the heart is directly related to the severity of hypertension but there is no correlation between the weight of the heart and duration of hypertension. The left ventricular wall is thickened (up to 20 mm or more), the papillary muscles are rounded and prominent, and the cardiac chamber is small (concentric hypertrophy). However, when decompensation and cardiac failure develop, there is eccentric hypertrophy with thinning of the ventricular wall and dilation of the left ventricular and atrial cavities. There may be dilation and hypertrophy of right heart as well.
Changes in the blood vessels involve arterioles and arteries. There are 2 types of these changes:
a) hyaline arteriolosclerosis that results in homogeneous and eosinophilic thickening of the wall of small blood vessels,
b) intimal thickening due to proliferation of smooth muscle cells in the intima.
In the large blood vessels atherosclerosis usually develops.
Renal type of hypertension may be benign and malignant. Benign nephrosclerosis is the term used to describe the kidney of benign phase of hypertension. Macroscopically, both kidneys are affected

equally and are reduced in size and weight, often weighing about 100 gm or less. The capsule is often adherent to the cortical surface. The surface of the kidney is finely granular and shows V-shaped areas of scarring («small contracted kidney»). The cut surface shows firm kidney and narrowed cortex. Microscopically there are primarily diffuse vascular changes, which produce parenchymal changes secondarily as a result of ischaemia. Parenchymal changes. There is variable degree of atrophy of parenchyma. These include glomerular shrinkage, deposition of collagen in Bowman's space, periglomerular fibrosis. Clinical features is variable: elevation of the blood pressure with headache, dizziness, palpitation. Renal failure and uremia may occur.

Cerebrovascular diseases (cerebral type).

Hypertension can result in two main types of parenchymal diseases of the brain:

1) ischemic brain damage (hypoxic encephalopathy and cerebral infarction);

2) intracranial hemorrhage (intracerebral and subarachnoid hemorrhage).

The pathologic appearance of the brain in hypoxic encephalopathy varies depending on the duration and severity of hypoxic episode and the length of survival. Macroscopically, there is focal softening. The area supplied by distal branches of the cerebral arteries suffers from the most severe ischemic damage and may develop border zone or watershed infarcts in the adjacent zones between the territories supplied by major arteries. Microscopically, the nerve cells die and disappear and are replaced by reactive fibrillary glia.

Cerebral infarction is a localized area of tissue necrosis caused by local vascular occlusion. Clinically, the signs and symptoms associated with cerebral infarction depend on the region infracted. Cerebral infarcts may be anemic or hemorrhagic.

Macroscopically, an anemic infarct becomes evident 6—12 hours after its occurrence. The affected area is soft and swollen and there is blurry of junction between grey and white matter. Within 2—3 days, the infarct undergoes softening and disintegration. A hemorrhagic infarct is red and superficially resembles a hematoma. It is usually the result of fragmentation of occlusive arterial emboli or venous thrombosis. Hemorrhage into the brain of patient with hypertension, is intracerebral hemorrhage, which is usually of hypertensive origin.

Most hypertensives over middle age have microaneurism in very small cerebral arteries in the brain tissue. The common sites of hypertensive intracerebral hemorrhage are the region of the basal ganglia, pons and cerebella's cortex. About 40% of patients die during the first 3—4 days of hemorrhage, mostly from hemorrhage into the ventricles. The outcome of intracerebral hemorrhage is cyst formation.

The causes of death among hypertensive patients were the following:

• congestive heart failure;
• coronary artery disease;
• cerebrovascular accidents;
• uremia;
• causes unrelated to hypertension. The cardiac complications therefore account for 36% of the death.

Secondary hypertensionmay be classified as follows:

1. Renal:

a) vascular diseases (atherosclerosis, arteritis, mechanical obstruction attributable to thrombosis, embolism, tumors);

b) parenchymal renal diseases (glomerulonephritis, pyelonephritis, hydronephrosis, amyloidosis, tumors);

c) perinephric diseases (perinephritis, tumors, hematoma).
2. Cerebral:
a) increased intracranial pressure (trauma, inflammation, tumors);
b) anxiety states;
c) lesions of brainstem (poliomyelitis).
3. Cardiovascular: coarctation of aorta (aorta has narrow).
4. Endocrine:
a) pheochromocytoma;
b) adrenocortical adenomas;
c) pituitary adenomas;
d) hyperthyroidism.
5. Preeclampsia and eclampsia.
If these causes of secondary hypertension are eliminated, hypertension disease can cured.
4. Illustrative material:
1. Presentation of lecture material;
2. Posters on the topic of classes;
3. Tables.
4. Macropreparations on the topic.
5. Literature: "Annex 1."
6. Control question. (feedback):
1. What is atherosclerosis? This definition.
2. What is hypertensive disease? This definition.
3. Hypertension, its difference from symptomatic hypertension.
4. What are the stages of morphogenesis of atherosclerosis?
5. What are the stages of hypertensive (hypertensive) disease with predominant heart disease?
6. As izmenenyaya kidney in atherosclerosis and hypertensive disease?
7. The difference between the morphological manifestations on the background of hypertension and atherosclerosis.
8. Factors that are most important in the development of atherosclerosis.
9. Clinical and morphological forms, complications of atherosclerosis.

№5

1. Theme: Acute heart failure. Acute carditis. Chronic heart disease. Pulmonary heart. Cardiomiopatia-primary and secondary. Cardiosclerosis.

2. Objective: to define heart disease. Explain the principles of diagnosis. To reveal the causes, mechanisms of development and classification of cardiovascular disease.
3.Thesis of the lecture: Cardiomyopathy is a group of diseases characterized by primary degenerative changes of the myocardium. This group includes various diseases of non-coronary (non-coronary cardiomyopathy) and non-rheumatic (non-rheumatogenic cardiomyopathy) origin, different in etiology and pathogenesis, but similar clinically. The main clinical manifestation of cardiomyopathy is the insufficiency of myocardial contractile function due to its dystrophy.
Among the primary (idiopathic) cardiomyopathies are: 1) hypertrophic (constrictive); 2) dilated (congestive); 3) restrictive (endomyocardial fibrosis). Secondary cardiomyopathies occur in: 1) intoxication (alcohol, ethylene glycol, heavy metal salts, uremia, etc.); 2) infections (viral infections, typhoid, trypanosomiasis-Chagas disease, trichinosis, etc.); 3) diseases of the exchange of hereditary (thesaurismosis-cardiopathic amyloidosis, glycogenosis) and acquired (gout, thyrotoxicosis, hyperparathyroidism, primary amyloidosis, beriberi, disorders of electrolyte-steroid metabolism) character; 4) diseases of the digestive system (malabsorption syndrome, pancreatitis, cirrhosis, etc.).PRIMARY (IDIOPATHIC) CARDIOMYOPATHY. 1. Hypertrophic (constrictive) cardiomyopathy is hereditary. Among the morphogenetic hypotheses, the following are discussed:

1) increased contractility, possibly due to increased sensitivity to catecholamines, leads to damage to cardiomyocytes, fibrosis and hypertrophy of the contractile myocardium; 2) increased myocardial contractility in the embryonic period (prenatal phase) leads to the development of hyperplasia of cardiomyocytes in certain parts of the myocardium, mainly in the interventricular septum. Hyperplasia of cardiomyocytes is replaced in the postnatal phase by progressive myocardial hypertrophy; 3) the primary pathology of collagen with a violation of the fibrous skeleton of the myocardium leads to disorganization of myofibrils.SECONDARY CARDIOMYOPATHY. Morphological manifestations of secondary cardiomyopathies quite polymorphic in connection with a variety of causing their causes. But at the heart of secondary cardiomyopathies, regardless of etiological factors, is cardiomyocyte dystrophy. Alcoholic cardiomyopathy is the most important among secondary cardiomyopathies.

The pathogenesis of alcoholic cardiomyopathy is primarily associated with the biological properties of ethanol — its direct toxic effect on cardiomyocytes, as well as the influence of the metabolite of ethanol — acetaldehyde. Of course the importance of vascular disorders and associated hypoxia, damaging effects on the myocardium of catecholamines.

Morphological changes in the heart are reduced to moderate myocardial hypertrophy, expansion of the heart cavities with parietal thrombi. Myocardium flabby, clay species, sometimes with small scars. Coronary arteries are intact, there are possible lipid spots and strips in the intima, there are no marked atherosclerotic changes. Microscopic examination showed a combination of dystrophy (hydropic and fatty), atrophy and hypertrophy of cardiomyocytes, there are foci of lysis of cardiomyocytes and sclerosis. Damaged areas of the myocardium alternate with unchanged. In electron microscopic examination of heart biopsies are cystic expansion of the sarcoplasmic reticulum and t-system of cardiomyocytes, which is considered characteristic of alcoholic cardiomyopathy.

Complications of alcoholic cardiomyopathy-sudden death (ventricular fibrillation) or chronic heart failure, thromboembolic syndrome.

Complications of cardiomyopathy are usually expressed by chronic cardiovascular insufficiency or thromboembolic syndrome due to the presence of blood clots in the heart cavities.

Myomalacia, or melting of necrotic myocardium, occurs in cases of predominance of autolysis of dead tissue. Myomalacia leads to rupture of the heart and hemorrhage into the cavity of the heart shirt (hemopericardium and tamponade of its cavity).

Acute aneurysm of the heart, i.e. swelling of the necrotic wall of it, is formed with extensive heart attacks. The cavity of the aneurysm usually trombicula in the wall appear the tears of endocardium, blood enters these tears, detaches and destroys the endocardium necrotizing myocardium. There is a rupture of the heart and hemopericardium.

Parietal blood clots are formed in subendocardial and transmural heart attacks, they are associated with the risk of thromboembolic complications. Pericarditis, usually fibrinous, is often found in subepicardial and transmural heart attacks.

Death in myocardial infarction can be associated with both the myocardial infarction and its complications. The immediate cause of death in the early period of heart attack are ventricular fibrillation, asystole, cardiogenic shock, acute heart failure. Fatal complications of myocardial infarction in the later period are a rupture of the heart or its acute aneurysm with hemorrhage into the pericardial cavity, as well as thromboembolism (for example, cerebral vessels) from the heart cavities, when thromboembolism becomes a source of thromboembolism on the endocardium in the infarction, in the acute aneurysm, in the ears of the heart.

Cardiosclerosis as a manifestation of chronic ischemic disease can be atherosclerotic diffuse small focal or postinfarction large focal, on the basis of which a chronic heart aneurysm (postinfarction changes) is formed.

A chronic heart aneurysm is usually formed in the outcome of a transmural extensive heart attack, when the scar connective tissue that replaced the heart attack becomes the wall of the heart. It thins and under blood pressure swells-formed aneurysmal SAC filled with layered thrombotic masses. Chronic aneurysm is associated with the development of chronic heart failure (the heart is constantly "residual" blood), thromboembolic complications and rupture of the aneurysm wall with the pericardial cavity tamponade. These complications are also more frequent causes of death in chronic coronary heart disease. It should be remembered, however, that a patient with chronic coronary heart disease is constantly in danger of developing a second heart attack with all possible complications in such cases.

4. Illustrative material:

1. Presentation of lecture material;
2. Posters on the topic of classes;

3. Tables.
4. Macropreparations on the topic.
5.Literature: "Annex 1."
6. Control question. (feedback):
1. What is the pulmonary heart? This definition.
2. What is cardio? This definition.
3. How does the heart change in cardiomyopathy?
4. Explain the clinical and morphological forms, complications of myocardial infarction?
5. Explain the causes of death in acute heart failure?
6. Give explanations of chronic aneurysm of the heart?

№6

1. Theme: Diverticulum of the esophagus. Esophagus cancer. Acute and chronic gastritis. Gastric ulcer. Enteritis. Colitis.

2. Objective: to Teach to determine the etiology, pathogenesis, classification and pathological anatomy of the digestive tract disease, as well as to distinguish them, guided by the morphological characteristics

3. Lecture notes: esophageal Diverticulum is a limited blind protrusion of its wall, which may consist of all layers of the esophagus (true diverticulum) or only the mucous and submucosal layer protruding through the cracks of the muscle layer (muscle diverticulum). The reasons for the formation of diverticula may be congenital (incompleteness of connective and muscular tissues of the esophagus wall, pharynx) and acquired (inflammation, sclerosis, scarring, increased pressure inside the esophagus).

Esophageal cancer most often occurs at the border of the middle and lower third of it, which corresponds to the level of bifurcation of the trachea. Much less often it occurs in the initial part of the esophagus and at the entrance to the stomach. Esophageal cancer is 2-5% of all cancers. Etiology. Predispose to the development of esophageal cancer chronic irritation of its mucous membrane (hot rough food, alcohol, Smoking), scarring after burn, chronic gastrointestinal infections, anatomical disorders (diverticula, ectopia of the cylindrical epithelium and gastric glands, etc.). Pathological anatomy. Following macroscopic forms of cancer of the esophagus: a ring-shaped thick, the papillary and wounded. Ring-shaped dense cancer is a tumor formation that circularly covers the wall of the esophagus in a certain area. The esophageal lumen is narrowed. With the collapse and ulceration of the tumor, the patency of the esophagus is restored. Papillary cancer of the esophagus is similar to the mushroom-like stomach cancer. It is easily decomposed, resulting in formation of ulcers penetrating into adjacent organs and tissues. Ulcerated cancer is a cancer ulcer that has an oval shape and is elongated along the esophagus.

Among the microscopic forms of esophageal cancer are: carcinoma in situ, squamous cell carcinoma, adenocarcinoma, glandular-squamous, glandular-cystic, mucoepidermoid and undifferentiated carcinoma Metastasis of esophageal cancer is mainly lymphokine.

Complications associated with invasion into adjacent organs — the trachea, stomach, mediastinum, pleura. Esophageal-tracheal fistulas are formed, aspiration pneumonia, abscess and gangrene of the lung, pleural empyema, purulent mediastinitis develop. Esophageal cancer early there is cachexia.

Gastritis (from Greek.gaster — stomach) — an inflammatory disease of the mucous membrane of the stomach. There are acute and chronic gastritis.

Etiology and pathogenesis. In the development of acute gastritis, the role of mucous membrane irritation a rich, indigestible, spicy, cold or hot food, alcohol, drugs (Sali cility, sulfonamides,

corticosteroids, biomitsin, digitalis, etc.), chemical substances (occupational hazards). A significant role is also played by microbes (Staphylococcus aureus, Salmonella) and toxins, products of disturbed metabolism. In some cases, such as alcohol poisoning, substandard food, pathogenic factors directly affect the mucous membrane of the stomach-exogenous gastritis, in others - this action is mediated and is carried out with the help of vascular, nervous, humoral and immune mechanisms-endogenous gastritis, which include infectious hematogenous gastritis, elimination gastritis in uremia, allergic, stagnant gastritis, etc.

Pathological anatomy. Inflammation of the mucous membrane can cover the entire stomach (diffuse gastritis) or certain parts of it (focal gastritis). In this regard, there are fundal, antral, pyloroantral and pyloroduodenal gastritis.

Depending on the features of morphological changes in the gastric mucosa, the following forms of acute gastritis are distinguished: 1) catarrhal (simple); 2) fibrinous; 3) purulent (phlegm); 4) necrotic (corrosive).

The outcome of acute gastritis depends on the depth of the mucous membrane (wall) of the stomach. Catarrhal gastritis can result in a complete restoration of the mucous membrane. With frequent relapses, it can lead to the development of chronic gastritis. After significant destructive changes characteristic of phlegmonous and necrotic gastritis, mucosal atrophy and sclerotic deformation of the stomach wall — cirrhosis of the stomach develops.

Chronic gastritis. In some cases, it is associated with acute gastritis, its relapses, but more often this connection is absent.

Etiology. Chronic gastritis develops under gastric mucosa primarily exogenous factors: violation of the diet and meal timing, alcohol abuse, the effect of chemical, thermal and mechanical agents, the impact of occupational hazards, etc. Pathogenesis. Chronic gastritis can be autoimmune (gastritis type a) and nonimmune (gastritis type B).

Autoimmune gastritis is characterized by the presence of antibodies to parietal cells, and therefore the defeat of the fundal stomach, where many lining cells (fundal gastritis). Morphological type. Chronic gastritis is characterized by long-existing dystrophic and necrobiotic changes in the epithelium of the mucous membrane, resulting in a violation of its regeneration and structural restructuring of the mucous membrane, ending with its atrophy and sclerosis; cellular mucosal reactions reflect the activity of the process.

The importance of chronic gastritis is extremely high. It takes the second place in the structure of gastroenterological diseases. It is important to note that chronic atrophic gastritis with severe epithelial dysplasia is a precancerous stomach disease.

Ulcer disease is a chronic, cyclically current disease, the main clinical and morphological expression of which is a recurrent ulcer of the stomach or duodenum. Pathological anatomy. The morphological substrate of peptic ulcer is a chronic recurrent ulcer. During the formation, it passes the stages of erosion and acute ulcers, which allows us to consider erosion, acute and chronic ulcers stages of morphogenesis of peptic ulcer. These stages are especially well traced in gastric ulcer.

Microscopic picture of chronic gastric ulcer in different periods of peptic ulcer disease is different. During remission, scar tissue is found in the edges of the ulcer. The mucous membrane around the edges is thickened, hyperplastic. In the area of the bottom, the destroyed muscle layer and the scar tissue replacing it are visible, and the bottom of the ulcer can be covered with a thin layer of epithelium.

Acute enteritis-acute inflammation of the small intestine. Etiology. Often occurs in many infectious diseases (cholera, typhoid, colibacillar, staphylococcal and viral infections, sepsis, giardiasis, opisthorchiasis, etc.), especially in food poisoning (salmonellosis, botulism), poisoning (chemical poisons, poisonous mushrooms, etc.). Pathological anatomy. Acute enteritis can be catarrhal, fibrinous, purulent, necrotic-ulcerative.

In catarrhal enteritis, which is most common, the full-blooded and edematous mucosa of the intestine is abundantly covered with serous, serous-mucous or serous-purulent exudate. Chronic enteritis is a chronic inflammation of the small intestine. It can be an independent disease or manifestation of other chronic diseases (hepatitis, liver cirrhosis, rheumatic diseases, etc.).

Etiology. Chronic enteritis can cause numerous exogenous and endogenous factors that can disrupt the physiological regeneration of the small intestine mucosa with prolonged exposure and damage to enterocytes.

Pathological anatomy. Changes in chronic enteritis recently well studied on the material of enterobiasis.

There are two forms of chronic enteritis - without mucosal atrophy and atrophic enteritis.

4. Illustrative material:

1. Presentation of lecture material;

2. Posters on the topic of classes;

3. Tables.

4. Macropreparations on the topic.

5. Literature: "Annex 1."

6. Control question. (feedback):

1.What is gastritis? This definition.

2.What is the classification of diseases of the digestive system?

3.What is a diverticulum? This definition.

4.What is Crohn's disease? This definition.

5.What is appendicitis? This definition.

6. What is the cause of congenital bowel abnormality?

№7

1. Theme: Hepatitis. Alcoholic steatosis of the liver. Cirrhosis. Liver cancer. The Pancreatitis. Pancreatic cancer.

2. Objective: to Explain the variety of clinical and morphological manifestations, etiological factors, diseases of the digestive system. to Give definition of disease of the pancreas. Explain the principles of diagnosis. To reveal the causes, mechanisms of development and classification of pancreatic disease.

3. Thesis of the lecture: Hepatitis is a liver disease, which is based on its inflammation expressed in both dystrophic and necrobiotic changes of the parenchyma, and in inflammatory infiltration of the stroma. Hepatitis can be primary, i.e. develop as an independent disease, or secondary as a

manifestation of another disease. Depending on the nature of the course distinguish acute and chronic hepatitis.
The pathological anatomy of acute and chronic hepatitis is different. Acute hepatitis can be exudative and productive. In exudative hepatitis in some cases (e.g., thyrotoxicosis) the exudate is serous in nature, and impregnates the stroma of the liver (serous hepatitis), in others the exudate is purulent (suppurative hepatitis) can diffuse infiltrating portal tracts (for example, in purulent cholangitis and cholangiolitis) or to form ulcers (pylephlebitis liver abscesses in purulent appendicitis, amoebiasis; metastatic abscesses in septicopyemia).
Productive of acute hepatitis is characterized by degeneration and necrosis of the GE - patcito various divisions of the segments and the reaction of the reticuloendothelial system of the liver. Formed as a result of alopecia or spilled infiltrates of proliferating stellate retikuloendoteliotsity (copperhouse cells), endothelial, joined by hematogenous elements.
The appearance of the liver in acute hepatitis depends on the nature of the developing inflammation.
Chronic hepatitis is characterized by destruction of parenchymal elements, cell infiltration of the stroma, sclerosis and regeneration of liver tissue. These changes can be presented in different combinations, which allows to distinguish three morphological types of chronic hepatitis: active (aggressive), persistent and cholestatic. In chronic active hepatitis, sharp dystrophy and necrosis of hepatocytes (destructive hepatitis) are combined with severe cell infiltration, which not only covers the sclerosed portal and periportal fields, but also penetrates the lobules.
Etiology and pathogenesis. The emergence of primary hepatitis, i.e. hepatitis as an independent disease, is most often associated with the impact of the hepatotropic virus (viral hepatitis), alcohol (alcoholic hepatitis) or drugs (drug, or drug, hepatitis). The etiology of secondary hepatitis, i.e. hepatitis as a manifestation of another disease (nonspecific reactive hepatitis), is extremely diverse. These are infection (yellow fever, cytomegaly, typhoid fever, dysentery, malaria, tuberculosis, sepsis), intoxication (thyrotoxicosis, hepatotoxic poisons), gastrointestinal tract lesions, systemic connective tissue diseases, etc.
The outcome of hepatitis depends on the nature and course, from the prevalence of the process, the degree of liver damage and its restorative possibilities. In mild cases, a complete restoration of the structure of the hepatic tissue is possible. In acute massive damage to the liver, as well as in the chronic course of hepatitis, cirrhosis may develop.
Alcoholic hepatitis. Allowed to participate and autoimmune mechanisms, and in the role of autoantigen, probably acts alcoholic hyaline.
Pathological anatomy. Liver changes in acute and chronic alcoholic hepatitis are different.
Acute alcoholic hepatitis has a well-defined macroscopic (laparoscopy) and microscopic (liver biopsy) characteristics. The liver looks dense and pale, with reddish areas and often with scarring.
The microscopic picture of acute alcoholic hepatitis is reduced to necrosis of hepatocytes, infiltration of necrosis zones and portal tracts by neutrophils, to the appearance of a large amount of alcoholic hyaline (Mallory cells) in the cytoplasm of hepatocytes and extracellularly.
Cirrhosis is a chronic disease characterized by increasing hepatic insufficiency due to scarring and structural restructuring of the liver. The term "cirrhosis" was introduced by R. Laennec (1819), referring to the features of morphological changes in the liver (dense bumpy liver of red color).
Classification. Modern classifications of liver cirrhosis take into account the etiological, morphological, morphogenetic and clinical functional criteria.
Etiology. Depending on the reasons leading to the development of cirrhosis, and can distinguish between: 1) infectious (viral hepatitis, parasitic liver diseases, infections of the biliary tract); 2) toxic and toxic-allergic (alcoholic beverages, industrial and food poisons, drugs, allergens); 3) biliary

(cholangitis, cholestasis of different nature); 4) metabolic and nutritional (deficiency of protein, vitamins, lipotropic factors, cirrhosis accumulation in hereditary metabolic disorders); 5) circulatory (chronic venous congestion in liver); 6) cryptogenic cirrhosis.
The main clinical significance currently have viral, alcohol and biliary cirrhosis liver. Viral liver cirrhosis usually develops after hepatitis type B, and alcohol, as a rule, after multiple attacks of alcoholic hepatitis. Among exchange-nutritional cirrhosis special group of cirrhosis accumulation, or thesaurismosis that occur in hemochromatosis and HEPA - terebralia degeneration (Wilson disease — Konovalova).
Pathological anatomy. Characteristic changes in the liver in cirrhosis are dystrophy and necrosis of hepatocytes, perverted regeneration, diffuse sclerosis, structural rearrangement and deformation of the organ.
Liver with cirrhosis dense and bumpy, its size is often reduced, rarely-increased.
Among the microscopic types of cirrhosis, on the basis of the construction features of the node regenerates allot monotonously cirrhosis, if the node regenerates capture one hepatic lobule, multilobular if they are based on several hepatic lobules, and nanomultilayers — the combination of the first two types of cirrhosis.
Liver cancer is a relatively rare tumor. It usually develops against the background of liver cirrhosis, which is considered a precancerous condition; among precancerous liver changes the most important is hepatocyte dysplasia. Morphological classification of liver cancer provides macroscopic form, nature and features of tumor growth, histogenesis, histological types.
Pathological anatomy. Among the macroscopic forms of liver cancer are distinguished: nodular cancer-the tumor is represented by one or more nodes; massive cancer — the tumor occupies a massive part of the liver and diffuse cancer — the whole liver is occupied by numerous merging tumor nodes. The special forms include small and peduncularis cancer.
Metastatic cancer of the liver how lymphokine (okulomotornyi lymph nodes,
Pancreatitis, inflammation of the pancreas, has an acute or chronic course. Acute pancreatitis develops when the violation of the outflow of pancreatic juice (dyskinesia ducts), the penetration of bile in the excretory duct of the gland (biliopankreaticheskoe reflux), alcohol poisoning, alimentary disorders (overeating) etc. Changes of the glands are reduced to edema, the appearance of white-yellow areas of necrosis (fat necrosis), hemorrhages, foci of suppuration, false cysts, sequestrations. With the prevalence of hemorrhagic changes become diffuse, talking about haemorrhagic pancreatitis, suppurative inflammation, acute suppurative pancreatitis, necrotic changes of pancreatic necrosis.
Chronic pancreatitis can be a consequence of relapses of acute pancreatitis. The cause of it are also infections and intoxication, metabolic disorders, malnutrition, liver disease, gallbladder, stomach, duodenum. In chronic pancreatitis is not dominated by destructive and inflammatory, sclerotic and atrophic processes in conjunction with regeneration Acinonyx cells and the formation of regenerative adenomas. Sclerotic changes lead to a violation of the patency of the ducts, the formation of cysts. Scar deformation of the gland is combined with calcification of its tissue. Death of patients with acute pancreatitis comes from shock, peritonitis.
Pancreatic cancer. It can develop in any part of it (head, body, tail), but more often found in the head, which has the form of a dense gray-white node. The node compresses, and then sprouts the pancreatic ducts and the common bile duct, which causes disorders of the function of both the pancreas (pancreatitis) and the liver (cholangitis, jaundice). Tumors of the body and tail of the pancreas often reach significant sizes, since they do not cause serious disorders of the gland and liver for a long time.

Pancreatic cancer develops from the epithelium of the ducts (adenocarcinoma) or from the acinuses of the parenchyma (acinar or alveolar cancer). The first metastases are found in the lymph nodes located directly near the head of the pancreas; hematogenous metastases occur in the liver and other organs.
Death comes from exhaustion, cancer metastases or joined pneumonia.

4. Illustrative material:

1. Presentation of lecture material;
2. Posters on the topic of classes;
3. Tables.
4. Macropreparations on the topic.

5. Literature: "Annex 1."

6.Control questions (feedback)

1. What is hepatitis? This definition.
2.What is the classification of liver disease?
3.What's the definition of cirrhosis?
4.What are the types of viral hepatitis?
5.How does the liver change in cancer?
6.Can you explain the acute alcoholic hepatitis?
7.What are the complications and outcomes of acute viral hepatitis B and C?
8. What is pancreatitis? This definition.
9.What's the definition of pancreatic cancer?
10. Give the definition of acinar cancer?
11.What are the complications of pancreonecrosis?
12.As izmenenyaya pancreas in pancreatic cancer?
13.Explain the morphological characteristics of pancreatitis?
14.Explain the significance and outcome of pancreatitis?
15. Can you give me a hug for a congenital pancreatic abnormality?

№ 8

1. Theme: Glomerulopathy.

2. Objective: To explain the clinical and morphological manifestations, etiological factors, diseases of the urinary system.

3. Thesis of the lecture: Glomerulonephritis - a disease of infectious-allergic or unstabilized nature, which is based on bilateral diffuse or ocular non-suppurative inflammation of the glomerular apparatus of the kidneys (glomerulitis) with typical renal and extra renal symptoms. Oliguria, proteinuria, hematuria, cylindruria, and renal symptoms include arterial hypertension, left heart hypertrophy, dysproteinaemia, edema, hyperasotemia and uremia. Combinations of these symptoms with glomerulonephritis can be expressed differently, and therefore the clinic distinguishes between hematuria, nephrotic (nephrotic syndrome), hypertensive and mixed forms of glomerulonephritis.
The classification of glomerulonephritis takes into account:
1) its nosological affiliation (primary - as an independent disease and secondary - as a manifestation of another disease);
2) etiology (established etiology - usually these are bacteria, viruses, protozoa and of unknown etiology); 3) pathogenesis (immunologically determined and not immunologically caused);
4) current (acute, subacute, chronic);

5) morphology (topography, nature and prevalence of the inflammatory process).
Etiology of primary glomerulonephritis. In some cases, the development of glomerular nephritis is associated with infection, usually bacterial (bacterial glome neuronephritis), in others there is no such link (abacterial glomerulo nephritis).
Among the possible causative agents of glomerulonephritis, the main role is played by P-hemolytic streptococcus (its nephritogenic types). Staphylococcus, pneumococcus, viruses, plasmodium malaria are of less importance.
Pathological anatomy. According to the topography of the process, intra- and extracapillary forms are distinguished, according to the nature of the inflammation - exudative, proliferative (productive) and mixed.
The morphological changes of the kidneys during glomerulonephritis concern not only the glomerular apparatus, although glomerulitis is the leading process evaluation, but also other structural elements - the tubules, stroma , and vessels. In this regard, isolated glomerulonephritis with a tubular, tubulo-interstitial or tubulo-interstitial-vascular component.
Depending on the nature of the course, as already mentioned, acute, subacute and chronic glomerulonephritis are distinguished. Pathological anatomy of each of them has its own characteristics.
Complications. Complication of glomerulonephritis in its acute and subacute course may be acute renal failure. Chronic course of glomerulonephritis is characterized by chronic renal failure with manifestations of azotemic uremia. Cardiovascular insufficiency, hemorrhage in the brain, which cause death, are also possible.
The outcome of acute glomerulonephritis is usually favorable - recovery, subacute and chronic - unfavorable. Chronic glomeruli nephritis is the most common cause of chronic renal failure, kidney transplantation and chronic hemodialysis are the only possible ways for patients to live in such cases.

4. Illustrative material:

1. Presentation of the lecture material;
2. Posters on the topic of employment;
3. Tables.
4. Macro preparation on the topic.

5. Literature: “Attachment №1”.

6. Control Test questions. (Feedback):

1. What is glomerulopathy? Give a definition.
2. What are the classification of diseases of the genitourinary system?
3. What does a kidney look like in acute glomerulonephritis?
4. What are the complications and outcomes of the genitourinary system?

№ 9

1. Theme: Tubulopathy. Nephrotic syndrome.

2. Objective: To explain the clinical and morphological manifestations, etiological factors, diseases of the urinary system.

3. Thesis of the lecture:

The nephrotic syndrome is characterized by high proteinuria, dysproteinemia, hypoproteinemia, hyperlipidemia (hypercholesterolemia) and edema.

Classification. There are primary, or idiopathic, nephrotic syndrome, which is an independent disease, and secondary nephrotic syndrome as an expression of a renal disease, for example, glomerulonephritis, amyloidosis.
Primary nephrotic syndrome. This syndrome can be represented by three diseases: lipoid nephrosis (nephropathy with minimal changes), membranous nephropathy (membranous glomerulonephritis) and focal segmental sclerosis (hyalinosis).
Lipoid nephrosis (nephropathy with minimal changes) occurs in both children and adults.
Etiology and pathogenesis. The cause of the development of lipid nephrosis is unknown; It is possible that we are talking about podocyte dysplasia. The pathogenesis of lipoid nephrosis does not differ from the nephrotic syndrome of any etiology. Dystrophy and necrobiosis of the tubule epithelium arising for the second time due to changes in the glomerular filter becomes the leading and largely determines all clinical manifestations characteristic of the nephrotic syndrome.
Pathological anatomy. For membranous nephropathy characteristic diffuse thickening of the walls of the capillaries of the glomeruli in the absence or extremely weak proliferation of mesangiocytes. Thickening of the capillary walls occurs due to the neoplasm of the substance of the basement membrane by the podocytes in response to deposits in the capillary wall of the subepithelial immune complexes. In the outcome of focal segmental sclerosis (hyalinosis) usually develops chronic renal failure.
Acute renal failure is a syndrome morphologically characterized by necrosis of the tubule epithelium and profound impairment of renal blood and lymph circulation. Acute renal failure is identified with necrotizing nephrosis (necronephrosis).
Pathogenesis. The development of acute renal failure is closely related to the mechanisms of shock of any etiology - traumatic, toxic, hemolytic, bacterial. Pathological anatomy. In various stages of cyclic flow, the pathological anatomy of acute renal failure is ambiguous. There are different initial (shock), oligoanuric and diuresis recovery stages. The appearance of the kidneys, regardless of the stage of the disease is the same: they are enlarged, swollen, swollen, fibrous capsule is tense, easily removed. The wide pale gray cortical layer is sharply delimited from the dark red pyramid, in the intermediary zone of the kidney and the pelvis often hemorrhages occur.
Histological examination at various stages of the disease in the kidneys find various changes. The dynamics of these changes can be traced using puncture biopsies of the kidney.
Complications. Severe complication of acute renal failure is segmental or total necrosis of the renal cortex. In its occurrence, the main role is played by the duration of renal ischemia and the depth of circulatory disorders, although the importance of allergic mechanisms cannot be excluded.
The outcome. Recovery from hemodialysis is common today. However, in some cases, acute renal failure will lead to death from uremia, which often occurs in a shock or oligoanuric stage. In the overwhelming majority of cases, necrosis of the cortical substance of the kidneys ends in death, although the life of patients can be prolonged due to the use of hemodialysis.

4. Illustrative material:

1. Presentation of the lecture material;
2. Posters on the topic of employment;
3. Tables.
4. Macro preparation on the topic.

5. Literature: “Attachment №1”.

6. Control Test questions. (Feedback):

1. What is tubulopathy? Give a definition.
3. What are the classification of diseases of the genitourinary system?

4. What are the complications and outcomes of the genitourinary system?
5. Name the primary nephrotic syndrome?

№10

1. Theme: Anemia. Acute and chronic anemia.
2. Objective: To define acute and chronic anemia. Explain the principles of the construction of the diagnosis. Disclose the causes, mechanisms of development and classification of anemia.
3. Thesis of the lecture: Diseases of the system, blood make up the content of clinical hematology, the founders of which in our country are I.I. Mechnikov, S.P. Botkin, M I. Arinkin, A.I Kryukov, I.A.Kassirsky. These diseases develop as a result of dysregulation of blood formation and blood destruction, which affects the composition of peripheral blood. Therefore, based on data from a study of the composition of peripheral blood, one can tentatively judge the state of the hematopoietic system as a whole. You can talk about changes in red and white sprouts, as well as blood plasma - both quantitative and qualitative.
Changes of red sprout blood system can be represented by a decrease in hemoglobin and red blood cell count (anemia) or increase (polycythemia Vera, or erythema) violation of the form of red blood cells - eritrotsitopatiyami (micro spherocytosis, elliptocytosis) or sinte¬za hemoglobin - hemoglobinopathies, or haemoglobinopathies (thalassemia, sickle cell anemia).
Changes in the white germ of the blood system can affect both leukocytes and platelets. The number of leukocytes in the peripheral blood can increase (leukocytosis) or decrease (leukopenia), they can acquire the quality of a tumor cell (hemoblastosis). Anemia (grech.ap - negative prefix and haima - blood), or little blood, - a group of diseases and conditions characterized by a decrease in the total amount of hemoglobin; it usually manifests itself in a decrease in its content per unit volume of blood.
Based on the study of punctate of the sternum, one can judge the state (hyper- or hypo regeneration) and the type of erythropoiesis (erythroblastic, normoblastic, megaloblastic) characteristic of one or another form of anemia.
Etiology and pathogenesis. The causes of the development of anemia can be blood loss, insufficient hermtropoetic function of the bone marrow, increased blood destruction.
With blood loss, anemia occurs when the loss of red blood cells in the blood exceeds the regenerative capacity of the bone marrow. The same should be said about blood destruction and, ie, hemolysis, which may be associated with exogenous and endogenous factors. The lack of erythro-poetic function of the bone marrow depends on the deficiency of substances necessary for normal blood formation: iron, vitamin B12, folic acid (the so-called deficient anemia), or the bone marrow's inability to digest these substances (the so-called acrostic anemia).
Classification. Depending on the etiology and mainly pathogenesis, there are three main groups of anemia:
1) due to blood loss (post hemorrhagic anemia);
2) due to impaired blood circulation;
3) due to increased blood destruction (hemolytic anemia). In each group, forms of anemia are highlighted.
Anemia due to blood loss may have an acute or chronic course.
Acute post-hemorrhagic anemia is observed after massive hemorrhages from the gastric vessels in peptic ulcer disease, from small intestine ulcers in typhoid fever, in the uterine tube rupture in case of ectopic pregnancy, in pulmonary tuberculosis, in aortic aneurysm rupture or injury of its wall and large branches extending from the aorta.

Pathological anatomy. If the bleeding turned out to be non-lethal, then the blood loss is reimbursed due to regenerative processes in the bone marrow. The bone marrow cells of the flat and epiphyses of the tubular bones proliferate vigorously, the bone marrow becomes juicy and bright. The fatty (yellow) bone marrow of the tubular bones also becomes red, rich in cells of the erythropoietin and myeloid series. In addition, there appear ouch gi extra costal cerebral (extra medullary) hematopoiesis in the spleen, lymphatic nodes, thymus, in perivascular tissue, the fiber of the gates of the kidneys, slimy and serous membranes, skin.

Chronic post-hemorrhagic anemia develops in cases where there is a slow but prolonged loss of blood. This is observed with minor bleeding from a decaying tumor of the gastrointestinal tract, bleeding gastric ulcer, hemorrhoid veins of the intestine, from the uterus, with hemorrhagic syndrome, hemophilia, etc.

Pathological anatomy. The skin and internal organs are pale. Bone marrow flat bones of the usual type; in the bone marrow tubular bones, there are observed in varying degrees, the phenomenon of regeneration and transformation of the fatty bone marrow into red. Multiple foci of extracellular cerebral hematopoiesis are often noted. Anemia due to impaired blood formation are represented by the so-called deficient anemia that occur with the intake of iron, vitamin B12, folic acid, hypo- and aplastic anemia.

Anemia due to lack of vitamin B12 and / or folic acid. They are characterized by the perversion of erythropoiesis. These are megaloblastic hyper chromic anemia.

Pernicious anemia was first described in 1855 by Addison, in 1868 it was described by Birmer (Addison anemia - Birmer anemia). The disease usually develops in adulthood (after 40 years). For a long time, before establishing the role of vitamin B12, folic acid and gastromucoprotein in the pathogenesis of pernicious anemia, it was malignant (malignant anemia) and, as a rule, ended with the death of patients.

Pathological anatomy. On external examination of the corpse, paleness of the skin (skin with a lemon-yellow tinge) and yellowness of the sclera are determined. The subcutaneous fat layer is usually well developed. Cadaveric hypostasis is not expressed. The amount of blood in the heart and large vessels is reduced, the blood is watery. Point hemorrhages are visible in the skin, mucous membranes and serous membranes. Internal organs, especially the spleen, liver, kidneys, on the section of a rusty type (hemosiderosis). Changes are most pronounced in the gastrointestinal tract, bone marrow and spinal cord.

In the gastrointestinal tract there are atrophic changes. The tongue is smooth, shiny, as if polished, covered with red spots. Microscopic examination finds sharp atrophy of the epithelium and lymphoid follicles, diffuse infiltration of the subepithelial tissue with lymphoid and plasma cells. These changes are referred to as Gunter glossier (after the name Gunter described for the first time these changes). The mucous membrane of the stomach, especially the fundus, thinned, smooth, devoid of folds. The liver is enlarged, dense, the cut has a brown-rusty tint (ge ¬ osiderosis). Bone marrow flat bones crimson-red, juicy; in tubular bones it has the appearance of raspberry jelly. In hyperplastic bone marrow, immature forms of erythropoiesis prevail - erythroblasts, normoblasts, and especially megaloblasts, which are also in peripheral blood. These blood elements undergo phagocytosis by macrophages (erythrophagia) not only of the bone marrow, but also of the spleen, liver, lymph nodes, which leads to the development of general hemosiderosis.

Hypo-and aplastic anemia. These anemia are the result of deep depression of blood formation, especially the young elements of hemopoiesis.

The cause of the development of such anemia can be both endogenous and exogenous factors. Among endogenous factors, a large place is occupied by hereditary factors, with which the development of familial aplastic anemia (Fanconi) and hypo plastic anemia (Ehrlich) is associated.

4. Illustrative material:

1. Presentation of the lecture material;
2. Posters on the topic of employment;
3. Tables.
4. Macro preparation on the topic.

5. Literature: “Attachments №1”.

6. Control questions. (Feedback):

1. What is anemia? Give a definition.
2. What are the types of anemia?
3. What are the complications of anemia?
4. Describe the macro and microscopic characteristics of organs in anemia?
5. What are the complications and causes of death in anemia?
6. Explain the causes of megaloblastic anemia?
7. Give an explanation of the clinical and morphological characteristics of post-hemorrhagic anemia?

№11

1. Theme: Chronic myeloid leukemia. Chronic lymphatic leukemia. Lymphosarcoma, Hodgkin's disease (Hodgkin's disease). Pathological anatomy.

2. Objective: To define hemoblastosis. Expand morpho-histogenesis and the main properties of the tumor process. Explain the essence and modern theory of the tumor process.

3. Thesis of the lecture: Chronic leukemia of myelocytic origin. These leukemia are diverse, but the main place among them is occupied by chronic ¬ myeloid leukemia, chronic erythromyelosis, erythremia, and true polycythemia.

Chronic myeloid leukemia (chronic myelitis). This leukemia proceeds in two stages: monoclonal benign and polyclonal malignant. The first stage, which takes several years, is characterized by increasing neutrophil leukocytosis with a shift to myelocytes and promyelocytes, an enlarged spleen. At necropsy of those who died from chronic myeloid leukemia in the thermal stage, especially pronounced changes are found in the bone marrow, blood, spleen, liver, lymph nodes. The bone marrow of the flat bones, epiphyses and diaphysis of the tubular bones is juicy, gray-red or greyish-yellow pussy (pioid bone marrow). Histological examination of the bone marrow reveals promyelocytes and myelocytes, as well as power cells. There are cells with changes in the nucleus (ugly nuclei) and cytoplasm, pycnosis or karyolitic. Signs of reactive osteosclerosis are sometimes noted in the bone tissue. The blood is gray-red, the organs are small.

The spleen is sharply enlarged, sometimes occupies almost the entire abdominal cavity; its mass reaches 6-8 kg. On a section it is dark red, ischemic heart attacks are sometimes found. The spleen tissue displaces the leukemic infiltrate mainly from cells of the myeloid series, among which blasts are visible; follicles are atrophic. Often, sclerosis and hemo-siderosis of the pulp are found. Leukemic blood clots are found in the vessels.

The liver is significantly enlarged (its weight reaches 5-6 kg). Its surface is smooth, the fabric on the cut is gray-brown. Leukemic infiltration is usually observed along the course of sinusoids, much less often it is visible in the portal tracts and capsule. Hepatocytes in a state of fatty degeneration;

hemosiderosis of the liver is sometimes noted. Chronic lymphocytic leukemia. It usually occurs in persons of middle and old age, in some cases in members of the same family, it develops from B-lymphocytes and is distinguished by a long benign course. The content of leukocytes in the blood increases dramatically (up to 100 • 109 / l), among them lymphocytes predominate. At autopsy, the main changes are found in the bone marrow, lymph nodes, spleen, liver and kidneys.
The bone marrow of the flat and tubular bones is red, but unlike myeloid leukemia, in the diaphysis of the tubular bones, among the red bone marrow there are patches of yellow color. Histological examination of bone marrow tissue revealed foci of tumor cell proliferation. In extreme cases, the entire myeloid tissue of the bone marrow is ousted by leukemic lymphocytic infiltration and only small islands of myeloid hematopoiesis remain intact.
Lymph nodes of all areas of the body are sharply enlarged, merging into huge soft or dense packages. On the cut, they are juicy, white and pink. The size of the tonsils, group and solitary lymphatic follicles of the intestine, which also constitute a juicy white-pink tissue, increases.
Lymphosarcoma is a malignant tumor arising from cells of the lymphocyte series. When this tumor affects the lymph nodes, and more often - mediastina and retroperitoneal, less often - inguinal and axillary. A tumor may develop in the lymphatic tissue of the gastrointestinal tract, spleen and other organs. Initially, the tumor is local, limited in nature.
Burkitt's tumor is an endemic disease found in the population of Equatorial Africa (Uganda, Guinea-Bissau, Nigeria), sporadic cases occur in different countries. Children usually get sick at the age of 4-8 years. Most often the tumor is localized in the upper or lower jaw, as well as the ovaries. Less commonly, the process involves the kidneys, adrenal glands, lymph nodes. Quite often there is generalization of the tumor with the expression of many organs. Lymphogranulomatosis (Hodgkin's disease) is a chronic recurrent, less commonly acute disease in which the growth of a tumor occurs predominantly in the lymph nodes.
Morphologically distinguished isolated and generalized lymphogranulomatosis. In an isolated (local) lymphogranulomatosis, one group of lymph nodes is affected. Most often it is the cervical, mediastina ¬ or retroperitoneal, less often - the axillary, inguinal lymph nodes, which increase in size and are soldered together. Microscopic examination of both the foci of primary tumor localization (most commonly in the lymph nodes) and its metastatic screenings reveal proliferation of lymphocytes, histiocytes, reticular cells, among which are giant cells, eosinophil, plasma cells, neutrophil leukocytes. Proliferating polymorphic cellular elements form nodules that undergo sclerosis and necrosis, often caseous.
Clinical and morphological classification. There are 4 variants (stages) of the disease:
1) a variant with a predominance of lymphoid tissue (lymphohistiocytic);
2) nodular (knotty) sclerosis;
3) mixed cell variant;
4) variant with suppression of lymphoid tissue.
Thus, the progression of Hodgkin's lymphoma is morphologically expressed in the successive change of its three variants: with a predominance of lymphoid tissue, mixed cell tissue and with suppression of lymphoid tissue. These clinical and morphological variants can be considered as stages of Hodgkin's disease.

4. Illustrative material:

1. Presentation of the lecture material;
2. Posters on the topic of employment;
3. Tables.
4. Macro preparation on the topic.

5. Literature: “Attachment №1”.
6. Control questions. (Feedback):
1. What is hemoblastosis? Give a definition.
2. What is the classification of hemoblastosis?
3. List the main types of leukemia?
4. Give the definition of Hodgkin's disease?
5. What are the complications and causes of death for leukemia?
6. What is lymphosarcoma? Give a definition.
7. Give an explanation of the clinical and morphological manifestations of myelodysplastic syndrome?
8. Explain the stage of blast crisis in chronic myeloid leukemia?

№12

1. Theme: Diabetes.
2. Objective: To reveal the main causes and mechanisms of the development of the disease of the endocrine system. Explain the morphological changes of organs in diabetes mellitus.
3. Thesis of the lecture: Diabetes mellitus (sugar disease) is a disease caused by the relative or absolute insufficiency of insulin.
Classification. The following types of diabetes are distinguished: spontaneous, secondary, pregnant diabetes and latent (subclinical). Among the spontaneous diabetes are distinguished diabetes type I (insulin-dependent) and type II diabetes (insulin-independent). Secondary diabetes is called diabetes in diseases of the pancreas (pancreatic diabetes), diseases of the endocrine system (acromegaly, Itsenko-Cushing syndrome, pheochromocytoma), complex genetic syndromes (ataxia-telangiectasia Louis-Bar, myotonic degeneration etc.). a number of drugs (drug diabetes). Pregnant women are told about diabetes in cases of impaired glucose tolerance during pregnancy, and the so-called latent (subclinical) diabetes — in cases of impaired glucose tolerance in seemingly healthy people. Only spontaneous diabetes is considered as an independent disease.
Among the etiological and pathogenic factors — risk factors — in diabetes mellitus, the following are distinguished:
1) genetically determined dysfunctions of function and number of B-cells (reduced insulin synthesis, impaired proinsulin conversion to insulin, synthesis of abnormal insulin);
2) environmental factors that violate the integrity and functioning of B-cells (viruses, autoimmune reactions; nutrition, leading to obesity, increased activity of the adrenergic nervous system).
Insular insufficiency determines the violation of glycogen synthesis, an increase in the sugar content in the blood (hyperglycemia), its appearance in the urine (glycosuria). Under these conditions, a significant part of the sugar, glucose) is formed due to the transformations of fats and proteins, hyperlipidemia, acetone and ketonemia occur, under-oxidized “ballast” substances accumulate in the blood, and acidosis develops.
Pathological anatomy. In diabetes mellitus, changes in the insular apparatus of the pancreas, changes in the liver, the vascular bed and kidneys are observed first of all. The pancreas is often reduced in size, there is its lipomatosis (see Fig. 36) and sclerosis. Most of the islands undergo atrophy and hyalinosis, some islands compensate hypertrophically. However, in some cases, iron appears unchanged, and only with the help of special methods of histochemical research is it possible to

detect the degranulation of B cells. The liver is usually somewhat enlarged, glycogen in hepatocytes is not detected, there is obesity of liver cells.
The blood vessels changes due to its reaction to latent and pronounced metabolic disorders, as well as to immune complexes circulating in the blood. Diabetic macro- and microangiopathy develops. Diabetic macroangiopathy is manifested by atherosclerosis of arteries of the elastic and muscular-elastic types. Changes in diabetic microangiopathy are reduced to plasmorrhagic damage to the basement membrane of the microvasculature with a friendly endothelial and perithelial reaction, resulting in sclerosis and hyalinosis, and lipogalin, which is characteristic of diabetes, appears. In some cases, a pronounced proliferation of the endothelium and perithelium is combined with lymphohistiocytic infiltration of the micro vessel wall, which suggests vasculitis. Microangiopathy in diabetes has a generalized character. Stereotypic changes in micro vessels are found in the kidneys, retina, skeletal muscles, skin, mucous membrane of the gastrointestinal tract, pancreas, brain, peripheral nervous system and other organs.
The so-called exudative manifestations of diabetic nephropathy are possible - the formation of "fibrin caps" on the capillary loops of the glomeruli and the "capsular drop". These changes in the glomeruli are complemented by a peculiar change in the epithelium of a narrow segment of the nephron, where glucose is polymerized into glycogen, the so-called glycogen infiltration of the epithelium. The epithelium becomes high, with a light translucent cytoplasm, in which glycogen is detected using special staining methods. Diabetic angiopathy has a peculiar morphology in the lungs: in the wall of the arteries, especially of the muscular type, lipogranulomas appear, consisting of macrophages, lipophages and giant cells of foreign bodies. diabetes is characterized by lipid infiltration of cells of the histomacrophagic system (spleen, liver, lymph nodes) and skin (skin xanthoma).
Complications. Diabetes complications are varied. Possible development of diabetic coma. Often there are complications caused by macro- and microangiopathy (gangrene of the limb, myocardial infarction, blindness), especially diabetic nephropathy (renal failure - acute with papilla-necrosis, chronic with glomerulosclerosis). In patients with diabetes easily develop infections, especially purulent (pyoderma, furunculosis, sepsis), often exacerbation of tuberculosis with generalization of the process and the prevalence of exudative changes.
Death in diabetes comes from complications. Diabetic coma is currently rare. Most often, patients die from limb gangrene, myocardial infarction, uremia, and infectious complications.

4. Illustrative material:

1. Presentation of the lecture material;
2. Posters on the topic of employment;
3. Tables.
4. Macro preparation on the topic.

5. Literature: “Attachment №1”.

6. Control questions (feedback):

1. What is diabetes? Give a definition.
2. What is the definition of diabetic macroangiopathy?
3. Describe the macro and microscopic characteristics of diabetic nephropathy?
4. What are the complications of diabetic coma?
5. What are the causes of death in diabetes?
6. Give insulin resistance?
7. Give the concept of clinical and morphological characteristics of diabetes mellitus type 1 and 2?

№13

1. Theme: Colloid goiter. Parenchymal goiter. Endemic goiter. Sporadic goiter. Diffuse toxic goiter.
2. Objective: To reveal the main causes and mechanisms of the development of thyroid disease. Explain the morphological changes of organs in thyroid disease.
3. Thesis of the lecture: Goiter (struma) is a pathological enlargement of the thyroid gland.
The goiter classification takes into account, on the one hand, morphological signs, on the other - epidemiology, causes, functional and clinical features.
Guided by morphological features, diffuse, nodular and diffuse nodular (mixed) goiter are distinguished in appearance, and colloid and parenchymal in their histological structure.
The colloid goiter is constructed from different sizes of follicles filled with colloid. In some cases, the follicles are large cyst-like, the epithelium is flattened in them (macro follicular colloid goiter), in others - small (micro follicular colloid goiter), in the third - small follicles (macro-micro follicular colloid goiter) are found along with the large ones. In a colloid goiter, the epithelium grows in the form of papillae (proliferating colloid goiter). Over time, circulatory disorders, foci of necrosis and calcification, proliferation of connective tissue, sometimes with bone formation, appear in the goiter tissue. Colloid goiter is usually nodular, dense in incision.
Parenchymal goiter is characterized by proliferation of the epithelium of follicles, which grows in the form of solid structures with the formation of small follicle-like structures without or with a very small amount of colloid. It is often diffuse, has the appearance of a homogeneous, fleshy tissue of a gray-pink color. Possible combinations of colloidal and parenchymal goiter.
Depending on the epidemiology, cause, functional and clinical features, endemic goiter, sporadic goiter and diffuse toxic (thyrotoxic) goiter (Bazedov’s disease, Graves' disease) are distinguished.
Endemic goiter develops in individuals living in certain, usually mountainous, localities (some areas of the Urals, Siberia, Central Asia, in Europe - Switzerland and other countries). The reason for the development of goiter is a lack of iodine in drinking water. The thyroid gland increases significantly, has the structure of a colloidal or parenchymal goiter. The function of the gland is usually reduced. If the goiter appears in early childhood, then a general physical and mental underdevelopment is noted - endemic cretinism.
Sporadic goiter appears in adolescence or adulthood. It may have a diffuse, nodular or mixed colloidal or parenchymal structure. Goiter does not have a noticeable general effect on the organism, however, with a significant proliferation, it squeezes the neighboring organs (esophagus, trachea, pharynx), impairs their function (retro esophageal goiter, retro tracheal goiter, etc.).
Diffuse toxic goiter (Basedov's disease, Graves' disease) is the most vivid manifestation of hyperthyroid syndrome, therefore it is also called thyrotoxic goiter. The reason for its development is the autoimmune enzyme: autoantibodies stimulate thyroid cell receptors. This allows us to attribute diffuse toxic goiter to "receptor antibody diseases".
Morphological features of diffuse toxic goiter are detected only by microscopic examination. These include the transformation of the prismatic epithelium of the follicles into cylindrical; epithelial proliferation with the formation of papillae branching inside follicles; vacuolization and change in the tinctorial properties of a colloid (it does not perceive dyes poorly) due to its liquefaction and iodine depletion; lymphoplasmacytic stroma infiltration, formation of lymphatic follicles with germinal centers.
When Bazedova\’s disease find a number of visceral manifestations. In the heart, whose myocardium is hypertrophied (especially of the left ventricle), serous edema and lymphoid

infiltration of the interstitial tissue, as well as intracellular edema of the muscle fibers, the thyrotoxic heart, are observed in connection with thyrotoxicosis. In its outcome, diffuse interstitial sclerosis develops. Serous edema is also observed in the liver with a rare outcome to fibrosis (thyrotoxic liver fibrosis). Dystrophic changes in nerve cells, perivascular cell infiltrates are found in the intermediate and medulla brain. Often, there is an increase in the thymus gland, hyperplasia of the lymphoid tissue and atrophy of the adrenal cortex.

Death with diffuse toxic goiter can occur from heart failure, exhaustion. Acute adrenal insufficiency may develop during a goiter removal operation.

Thyroiditis. This is a group of diseases, among which Hashimoto thyroiditis, or Hashimoto disease, is a true autoimmune disease. Autoimmunization is associated with the appearance of autoantibodies to microsomal antigen and thyrocyte surface antigens, as well as thyroglobulin. An autoimmune process determined by DR histocompatibility antigens leads to diffuse infiltration of gland tissue by lymphocytes and plasma cells, and the formation of lymphoid follicles in it. The parenchyma of the gland as a result of exposure to predominantly immune effector cells dies, is replaced by connective tissue. In advanced cases, the morphological picture may resemble Riedel's thyroiditis (goiter).

4. Illustrative material:

1. Presentation of the lecture material;
2. Posters on the topic of employment;
3. Tables.
4. Macro preparation on the topic.

5. Literature: “Attachment №1”.

6. Control questions (feedback):

1. What is goiter? Give a definition.
2. What are the causes of goiter?
3. What are the types of goiter?
4. Describe the macro and microscopic characteristics of diffuse toxic goiter?
5. What are the complications of diffuse toxic goiter?
6. What are the causes of death in diffuse toxic goiter?
7. Give an explanation of euthyroid and hypothyroid goiter?
8. Explain the causes of death in Grave’s disease?

№14

1.Theme: Parathyroid osteodystrophy (Recklinghausen's disease). Osteopetrosis. Osteoarthritis. Osteomyelitis. Osteosarcoma. Progressive muscular dystrophy. Myasthenia. Muscular dystrophy.

2. Objective: The main causes and mechanisms of development, especially the musculoskeletal system. Explain morphological changes in diseases of the support system.

3. Thesis of the lecture: Parathyroid osteodystrophy (Recklinghausen's disease, generalized osteodystrophy) is a disease caused by the hyperfunction of the parathyroid glands and is accompanied by a generalized lesion of the skeletal system. The disease occurs mainly in women 40-50 years, rarely in childhood.

Etiology. Parathyroid osteodystrophy is associated with primary hyperparathyroidism, which is caused by the adenoma of the parathyroid glands or hyperplasia of their cells (cancer is very rare). Primary hyperparathyroidism should be distinguished from the secondary one that develops in chronic renal failure, multiple cancer metastases in the bone, etc.

Pathogenesis. Increased synthesis of parathyroid hormone causes increased mobilization of phosphorus and calcium from the bones, which leads to hypercalcemia and progressive

demineralization of the entire skeleton. Osteoclasts are activated in the bone tissue, and foci of lacunar bone resorption appear.
Changes in the skeleton in parathyroid osteodystrophy depend on the stage and duration of the disease. In the initial stage of the disease and with low parathyroid hormone activity, external bone changes may be absent. In the far advanced stage, the deformity of the bones is detected, especially those that are subjected to physical exertion, limbs, spine, ribs. They become soft, porous, easily cut with a knife.
Microscopic examination of bone tissue determines foci of lacunar resorption, neoplasms of fibrous tissue, and sometimes osteoid beams. In the foci of tumor-like formations, giant cell granulomas, clusters of red blood cells and hemosiderin, cysts are found.
The death of patients often occurs from cachexia or uremia due to wrinkling of the kidneys.
Under osteomyelitis (from the Greek osteon - bone, myelos - the brain) is understood as inflammation of the bone marrow, extending to the compact and spongy substance of the bone and periosteum.
Pathological anatomy. In acute hematogenous osteomyelitis, inflammation has the character of phlegmonous (sometimes serous) and captures the bone marrow, gaversovy channels and periosteum; foci of necrosis appear in the bone marrow and compact plate. A pronounced bone resorption in the vicinity of the epiphyseal cartilage can cause the separation of the metaphysis from the epiphysis (epiphysis), mobility and deformation of the periarticular zone appear. Nephrotic infiltration of the tissue by neutrophils is determined around the necrosis foci, and blood clots are detected in the vessels of the compact plate. Abscesses are often found under the periosteum, and in the adjacent soft tissues - phlegmonous inflammation.
Chronic hematogenous osteomyelitis is associated with chronization of the suppurative process, the formation of bone sequesters. A granulation tissue and a capsule are formed around the sequestration. Sometimes the sequester floats in a cavity filled with pus, from which fistulous passages go to the surface or body cavities, to the cavity of the joints. Along with this, bone formation is observed in the periosteum and the bone marrow canal. The bones become thick and deformed.
Complications. Bleeding from fistulas, spontaneous bone fractures, formation of false joints, pathological dislocations, development of sepsis; in chronic osteomyelitis, secondary amyloidosis is possible.
Osteopetrosis (marble disease, congenital osteosclerosis, Albers-Schoenberg disease) is a rare hereditary disease in which generalized excessive bone formation is noted, leading to bone thickening, narrowing and even complete disappearance of bone marrow spaces. Therefore, a triad is characteristic of osteopetrosis: an increased density of bones, their fragility and anemia.
Pathological anatomy. In osteopetrosis, the entire skeleton can be affected, but especially the tubular bones, the bones of the skull base, pelvis, spine, ribs. In the early form of osteopetrosis, the face has a characteristic appearance: it is wide, with eyes wide apart, the root of the nose is depressed, the nostrils are unfolded, and the lips are thick. In this form, hydrocephalus, increased hair growth, hemorrhagic diathesis, and multiple bone lesions are noted, whereas in the late form of osteopetrosis, bone lesions are usually limited.
The microscopic picture is extremely peculiar: pathological bone formation occurs throughout the whole bone, the mass of the bone substance is sharply increased, the bone substance itself is randomly piled up in the inner parts of the bone along with this there are beams of the embryonic coarse-fibrous bone. The complications. Often there are fractures of bones, especially femoral. In

the places of fractures often purulent osteomyelitis develops, which is sometimes the source of sepsis.

Causes of death. Patients with osteopetrosis often die in early childhood from anemia, pneumonia, sepsis. Progressive muscular dystrophy (progressive myopathy) includes various primary hereditary chronic diseases of the striated musculature (they are called primary because there is no damage to the spinal cord and peripheral nerves). Diseases are characterized by increasing, usually symmetrical, muscle atrophy, accompanied by progressive muscle weakness, up to complete immobility.

Pathological anatomy. Usually, the muscles are atrophic, thinned, eaten by myoglobin, therefore they resemble fish meat in the section. However, the volume of the muscles can be increased due to the vacant growth of the fatty cellular tissue and connective tissue, which is especially characteristic of muscular dystrophy

Microscopic examination of muscle fibers have different sizes: along with atrophic, there are sharply enlarged, the nuclei are usually located in the center of the fibers. Dystrophic changes in muscle fibers (accumulation of lipids, reduction of glycogen content, disappearance of transverse striation), their necrosis and phagocytosis are expressed.

Myasthenia (from the Greek. Myos - muscle, asthenia - weakness) is a chronic disease, the main symptom of which is weakness and pathological fatigue of striated muscles. Normal muscle contraction after their vigorous activity decreases in strength and volume and may completely cease. After resting, the muscle function is restored. In the far advanced stage of the disease, the rest time is increased, an impression of muscle paralysis is created.

Etiology and pathogenesis. The etiology is unknown. There is a correlation between anomalies of the thymus gland and myasthenia.

4. Illustrative material:

1. Presentation of the lecture material;
2. Posters on the topic of employment;
3. Tables.
4. Macro preparation on the topic.

5. Literature: “Attachment №1”.

6. Control questions (feedback):

1. What is parathyroid osteodystrophy (Recklinghausen's disease)? Give a definition.
2. What is progressive muscular dystrophy? Give a definition.
2. What are the reasons for the development of muscular dystrophy?
3. What are the causes of osteosarcoma?
4. Describe the macro and microscopic characteristics of Recklinghausen's disease?
5. What are the complications of diseases of the musculoskeletal system?
6. What are the causes of death for Recklinghausen's disease?

№15

1. Theme: Cerebrovascular diseases. Meningitis. Encephalitis.

2. Objective: To define the CVD disease. Explain the principles of the construction of the diagnosis. Disclose the causes, mechanisms of development and classification of CVD. To reveal the main causes and mechanisms of development, especially the inflammation of the membranes of the brain and spinal cord. Explain the morphological changes in organs with meningitis and encephalitis.

3. Thesis of the lecture: Cerebrovascular diseases are characterized by acute disorders of cerebral circulation, the background for the development of which are atherosclerosis and hypertension. In essence, these are cerebral manifestations of atherosclerosis and hypertension, less commonly symptomatic hypertension.

As an independent group of diseases, cerebrovascular diseases are distinguished, as is coronary heart disease, due to their social significance. These diseases in economically developed countries in terms of morbidity and mortality “caught up” with coronary heart disease.

Etiology and pathogenesis. All that has been said about the etiology and pathogenesis of coronary heart disease is applicable to cerebrovascular diseases. Among the immediate causes of acute disorders of cerebral circulation, the main place is occupied by spasm, thrombosis and thromboembolism of cerebral and pre-cerebral (carotid and vertebral) arteries. Of great importance is psycho-emotional overstrain, leading to angioedema.

Classification. Among the acute disorders of cerebral circulation that underlie cerebrovascular diseases, transient cerebral ischemia and stroke are distinguished. A stroke is an acutely (suddenly) developing local disorder of the cerebral circulation, accompanied by damage to the brain’s substance and impaired function. Distinguish:

1) hemorrhagic stroke, represented by hematoma or hemorrhagic impregnation of the brain substance; subarachnoid hemorrhage is also ranked;

2) ischemic stroke, the morphological expression of which is a heart attack (ischemic, hemorrhagic, mixed).

Pathological anatomy. The morphology of transient cerebral ischemia is represented by vascular disorders (spasm of arterioles, plasma soaking of their walls, perivascular edema, and isolated small hemorrhages) and focal changes in brain tissue (edema, dystrophic changes of cell groups). These changes are reversible; in place of the former small hemorrhages, perivascular hemosiderin deposits can be determined. During the formation of a hematoma of the brain, which occurs in 85% of hemorrhagic stroke, a pronounced alteration of the walls of arterioles and small arteries with the formation of micro aneurysms and the rupture of their walls is found. At the site of hemorrhage, the brain tissue is destroyed, a cavity is formed, filled with blood clots and softened brain tissue (red softening of the brain). Hemorrhage is localized most often in the subcortical nodes of the brain (optic tubercle, internal capsule) and the cerebellum. Its dimensions are different: sometimes it covers the whole mass of subcortical nodes, the blood breaks into the side, III and IV ventricles of the brain, seeps into the region of its base. Strokes with a breakthrough into the ventricles of the brain always end in death. During hemorrhagic impregnation of the brain substance as a type of hemorrhagic stroke, small confluent foci of hemorrhages are found. Among the blood-soaked medulla, nerve cells with necrobiotic changes are determined. Hemorrhages such as hemorrhagic soaking are usually found in the visual mounds and the brain bridge (pons) and, as a rule, do not occur in the cerebral cortex and cerebellum.

The ischemic cerebral infarction resulting from thrombosis of atherosclerosis ¬ ally precerebral or cerebral arteries, has a different localization. This is the most frequent (75% of cases) manifestation of ischemic stroke. Ischemic infarction looks like a center of gray softening of the brain.

Microscopic examination of the necrotic mass medium can reveal dead neurons.

Hemorrhagic cerebral infarction resembles the center of hemorrhagic soaking, but the mechanism of its development is different: ischemia of the brain tissue develops, secondary - hemorrhage into the ischemic tissue. More often hemorrhagic heart attack occurs in the cerebral cortex, at least - in the subcortical nodes.

With a mixed heart attack, which always occurs in the gray matter of the brain, you can find areas of both ischemic and hemorrhagic heart attack. At the site of cerebral infarction, as well as hematomas, cysts are formed, and
the cyst wall in place of a hemorrhagic infarction contains an accumulation of hemosiderin (“rusty cyst”).
Complications of strokes (hemorrhages and heart attacks of the brain), as well as their effects (brain cysts), are paralysis. Brain strokes are a common cause of death in patients with atherosclerosis and hypertensive disease.
Meningococcal infection is an acute infectious process, manifested in three main forms - nasopharyngitis, purulent meningitis and tningococcemia, characterized by periodic epidemic outbreaks. These flashes arise from. At intervals of 25–30 years, children under 5 years of age are more likely to be ill; people of any age can get sick.
Etiology and pathogenesis. The exciter is meningococcal (Neisseria meningitides), which has the form of coffee beans, located both outside and intracellular and defined in smears from the nasopharynx or cerebrospinal fluid. It is very sensitive to external influences (temperature, pH of the medium, humidity), therefore it dies quickly outside the body and in a corpse.
Infection occurs from the patient or carrier of the infection. Infection is spread through airborne droplets. The invasion of meningococcal into the mucous membrane of the nasopharynx in only 10-30% of cases causes the development of meningococcal nasopharyngitis. More rarely, mainly in young children, meningococcal is spread by the hematogenous route, overcomes the blood-brain barrier and is fixed in the pia mater, where it causes purulent meningitis. The predominant disease of children of the first 5 years of life is associated with the structural immaturity of this barrier.
Depending on the state of the body's immune reactivity, meningococcal can cause sepsis, called meningococcemia, which sometimes has a fulminant course. The basis of the lesion of blood vessels in meningococcemia is bacterial shock, coming from the intense collapse of phagocytosis bacteria with the release of their endotoxin. Observed paresis of small vessels with the development of stasis, thrombosis, hemorrhage and subsequent necrosis in the organs.
Pathological anatomy. Meningococcal nasopharyngitis is characterized by catarrhal inflammation of the mucous membranes with particularly severe hyperemia, edema of the posterior pharyngeal wall, and hyperplasia of the lymphatic follicles. This form has a large epidemiological value, since it is often not clinically diagnosed.
During meningococcal meningitis, the pie mater becomes sharply full-blooded on the first day of the onset of the disease, soaked with a slightly unclear serous exudate. By the end of the 2nd - the beginning of the 3rd day, the exudate gradually thickens, acquires a greenish-yellow color and purulent character. By the 5th – 6th day, it is further compacted from the addition of a fibrous effusion.
Microscopically, the vessels of the pie mater are sharply full of blood, the subarachnoid space is enlarged, saturated with leukocyte exudate, permeated with fibrin filaments. The process from the choroid can move to the brain tissue with the development of meningoencephalitis. Starting from the 3rd week of illness, the exudate is subjected to resorption.
Death can occur in the acute period from brain swelling with the cerebellar tonsils penetrating into the large occipital foramen and restraint of the medulla oblongata in it or in subsequent periods from meningoencephalitis, purulent ependymitis, and later from common cerebral cachexia due to hydrocephalus and atrophy big brain.
Meningococcemia is characterized by a generalized lesion of the microcirculatory bed, skin rash, changes in the joints, choroid, adrenal glands and kidneys.

Focal necrosis and hemorrhage or bilateral massive hemorrhages with the development of acute adrenal insufficiency - Waterhouse-Frederickson syndrome are noted in the adrenal glands. In the kidney there is necrosis of the tubule nephrothelium (necrotic nephrosis). Changes in the microvasculature are characterized by vasculitis, extravasation, necrosis.
The death of patients with the lightning current comes from bacterial shock, the severity of which is aggravated by hemorrhages in the adrenal glands, less frequently acute renal failure (in adults). With a longer course of death due to septicopyemia or purulent meningitis.
Encephalitis (from the Greek .encephalon - the brain) - inflammation of the brain associated with infection, intoxication or trauma. Infectious encephalitis can be caused by viruses, bacteria, fungi, but the most important among them are viral encephalitis.
Viral encephalitis occurs due to the effect on the brain of various viruses: arboviruses, enteroviruses, cytomegaloviruses, herpes viruses, rabies, viruses of many childhood infections, etc. The disease can have an acute, subacute and chronic course, vary in severity depending on the severity clinical manifestations (stupor, cerebral coma, delirium, paralysis, etc.). In favor of the viral etiology of encephalitis are evidenced by:
1) mononuclear inflammatory infiltrates from lymphocytes, plasma cells and macrophages;
2) diffuse proliferation of microglia and oligodendroglia with the formation of rod-shaped and amoeboid cells;
3) neuronophagy with the formation of neuronophagic nodules;
4) intranuclear and intracytoplasmic inclusions. A clinical pathologist (pathologist) can determine the etiology of viral encephalitis by determining the pathogen in the tissue (biopsy) of the brain using immunohistochemical methods and the in situ hybridization method. On the territory of the former USSR tick-borne encephalitis is most common.
The disease is characterized by seasonality: usually outbreaks occur in the spring-summer period (spring-summer encephalitis), less often in the fall. The incubation period is 7–20 days. The disease begins acutely, fever, severe headache, impaired consciousness, sometimes epileptiform primaging, meningeal symptoms, paresis and paralysis (in severe disease) develops. With a protracted course, a decrease in memory is noted. Muscles atrophy, movement is partially restored. Characterized by paresis and atrophy of the neck muscles (hanging head) and the muscles of the proximal parts of the upper limbs. In chronic course, Kozhevik epilepsy syndrome develops.
During the period of an epidemic outbreak, often erased forms of the disease without clear signs of damage to the nervous system, sometimes meningeal forms. With such forms there is a relatively complete recovery.
Pathological anatomy. Macroscopically note the hyperemia of the cerebral vessels, the swelling of its tissue, minor hemorrhages. The microscopic picture largely depends on the stage and nature of the course of the disease: in acute forms, circulatory disorders and an inflammatory exudative reaction predominate, and perivascular infiltrations and neuronophagia often occur. In case of a protracted course of the disease, the proliferative reaction of glia, including astrocytes, and focal destruction of the nervous system (areas of a spongy nature, clusters of grain balls) become the leading ones. The chronic course of encephalitis is characterized by fibrillatory gliosis, demyelination, and sometimes atrophy of certain parts of the brain.
Cause of death. In the early stages of the disease (2-3 days), death can - come back from bulbar disorders. The causes of death in the later stages of the disease are varied.
Infectious diseases are diseases caused by infectious agents - viruses, bacteria, fungi. When protozoa and worms are introduced into the body, they speak about invasive diseases.

Some infectious diseases are now eradicated, but many, especially viral, still pose a significant threat to the population. In addition, there are still endemic foci of a number of infectious diseases, which, at the speed inherent in modern means of movement, can easily be transferred to other countries.

The infection process is very complicated, and its development is determined by both the characteristics of the pathogen and the reactive state of the microorganism. The characteristics of a microorganism, the causative agent of an infectious disease, are determined not only by its structure, chemical structure, antigenic properties, but also by the nature of interaction with the host organism. The result of this interaction largely depends on the state of the protection systems of the body — phagocytic (neutrophils and monocytes phagocytes) and the immune system, especially the humoral immunity system.

4. Illustrative material:

1. Presentation of the lecture material;
2. Posters on the topic of employment;
3. Tables.
4. Macro preparation on the topic.

5. Literature: "Attachment №1".

6. Control questions (feedback):

1.What is cerebrovascular disease? Give a definition.
2. What are the reasons for the development of CVD?
3. Describe the macro and microscopic characterization of CVD?
4. What are the complications of CVD?
5. What are the causes of death in CVD?
5. What is viral encephalitis? Give a definition.
7. What is meningococcal infection? Give a definition.
8. What are the causes of meningitis?
9. Describe the macro and microscopic characteristics of meningitis?
10. What are the complications of viral encephalitis and meningococcal infection?
11. What are the causes of death for meningitis and encephalitis?

Literature: "Attachment №1".

In Russian

Main:

1. Pathological anatomy: a guide to practical exercises: studies. manual / [Zaratyants OV et al.] ed. O.V. Zayratyants, L.B. Tarasova. - 2nd ed., Corr. and add. - M .: GEOTAR-Media, 2015.additional:
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2. Strukov A.I. Pathological anatomy: a textbook / Ed. Strukova A.I., Serova V.V. - 5th ed. - M .: Litterra, 2011.

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5th ed. erased - Electron. text given. (51.9 MB) - M .: GEOTAR-Media, 2011. - El. wholesale disk (CD-ROM).
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Main:
1. Kumar V., Abbas A.K., Aster C. Robbins Basic Pathology. - Elsevier, 2017.
2. Kumar V. Robbins and Cotran. Pathologic Basic of Disease. - Elsevier, 2015.
3. Klatt, E.C., Kumar V. Robbins and Cotran. Review of Pathology. - Saunders, 2015.
4. Klatt, E.C., Robbins and Cotran. Atlas of Pathology. - Saunders, 2014.
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1. Litvitsky P.F., Pirozhkov S.V., Tezikov E.B. Pathophysiology. Concise lec-tures, tests, clinico-patophysiological situations: student manual. - M. GEOTAR-Media, 2012.
2. Robbins and Cotran. Review of Pathology // Klatt E.C., Kumar V. - Third Edition. - Saunders Elsevier, 2010.