Orenburg state medical university

**ASSESSMENT FUND**

**FOR ONGOING MONITORING OF STUDENTS` PERFORMANCE AND MIDTERM ATESTATION** **in the subject**

«PATHOLOGICAL ANATOMY,

CLINICAL PATHOLOGICAL ANATOMY»

for

*31.05.01 General Medicine, Faculty of Foreign Students*

It is part of the main professional educational program of higher education *31.05.01. General Medicine, Faculty of Foreign Students* approved by the Academic Council of the Orenburg state medical university

record No. 8 dated March 25th, 2016

Orenburg

1. **Assessment fund passport**

Assessment fund for practice contains standard control and assessment materials for monitoring learning performance formed in the process of passing the practice at intermediate attestation in the form of final test.

Control and assessment materials for intermediate attestation correspond to the form of intermediate certification for this type of practice, defined in the major professional academic program curriculum and are aimed at checking skills and experience in practical activities for each competence established in the practice program.

As a result of undertaking practical training, a student develops **the following expertises**:

Common cultural expertise-1 the ability for abstract thinking, analysis, synthesis.

Common professional expertise-9 the ability to assess morphofunctional, physiological conditions and pathological processes in the human body for solving professional problems.

Professional expertise – 5 the readiness to collect and analyze the patient's complaints, data from his anamnesis, examination results, laboratory, instrumental, pathological and other studies in order to recognize a condition or establish the presence or absence of a disease.

**2. Assessment materials for students` midterm attestation.**

Evaluation materials across the discipline.

Evaluation materials within the discipline module

Module 1 General pathological anatomy.

Evaluation materials for each topic of the discipline

**Module 1 General pathological anatomy.**

**theme 1: Pathological anatomy: substance, objectives, subjects and methods. Necrosis. Apoptosis. Death AND POSTMORTEM changes.**

**The Lesson Content**

**Prepare answers to these questions:**

1. Pathological anatomy. The substance, objectives, materiaks, methods and levels of study. Historical data: stages of disease, the role of the Russian school of pathological anatomy. Communication pathological anatomy with basic and clinical disciplines. Pathologic service and it importance in the health system. Ethics and professional standards of pathological anatomy.

2. Necrosis. Causes, mechanisms of development, morphological characteristics. Clinical and morphological forms of necrosis (coagulation, liquefactive, gangrene, sequestration, infarction): pathogenesis and morphogenesis, clinical and morphological characteristics, diagnostic methods, outcomes and functional significance.

3. Apoptosis as a programmed cell death. Definition, pathogenesis, morphological characteristics and methods of diagnosis. Stage of apoptosis. Value of apoptosis in physiological and pathological processes.

4. Death. Definition. Natural death. The concept of gerontology and geriatrics. Characteristics of the violent death and the death from disease. The sudden death. The concept of fetal and perinatal death. The essence of the clinical and biological death. The concept of resuscitation. Basic mechanisms of death. Signs of biological death and postmortem changes: the sequence of occurrence and morphological characteristics.

**Test tasks**

**Select one or more correct answers**

**1. Necrosis- it is**

a) programmed cell death

b) the blood flowstop in the vessels of the ICR

c) cells and tissues death in a living organism

d) transition of one type of tissue in to the other

**2. Liquefactive necrosis is developing in the tissues of:**

a) protein-rich

b) proteins-poor

c) low water content

d) high water content

**3. The characteristic signs of dry gangrene**

a) tissue mummification

b) edema and swelling of tissues

c) expresseddemarcation zone

d) demarcation zone is not expressed

**4. Hemorrhagic infarction is typical for**

a) spleen

b) myocardium

c) lungs

d) kidneys

**5. Apoptosis is**

a) necrosis of cells and tissues in vivo

b) blood flowstop in the vessels of the ICR

c) programmed cell death

d) termination of life of the organism as a whole

**6. Types of coagulation necrosis**

a) waxy necrosis

b) caseation

c) fibrinoid necrosis

d) necrosis of the brain

**7. Infarction is**

a) trofonevrotic necrosis in critically ill patients

b) necrosis tissue contacting of with the external environment

c) portion of the dead tissue not affected to autolysis

d) ischemic necrosis

**8. Types of gangrene**

a) dry

b) black

c) decubital

d) wet

**9. Correct successive stages of necrosis**

a) autolysis (4)

b) paranecrosis (1)

c) necrobiosis (2)

d) cell death (3)

**10. Types of liquefactive necrosis**

a) waxy necrosis

b) caseation

c) fibrinoid necrosis

d) necrosis of the brain

**11. Gangrene it is**

a) trofonevrotic necrosis in critically ill patients

b) necrosis of tissues contacting with the external environment

c) zone of the dead tissue not affected to autolysis

d) ischemic necrosis

**12. Coagulation necrosis develops in what tissues**

a) protein-rich

b) proteins-poor

c) low water content

d) high water content

**13. Sequester is**

a) trofonevrotic necrosis in critically ill patients

b) necrosis of tissues contacting with the external environment

c) portion of the dead tissue is not affected by autolysis

d) ischemic necrosis

**14. Characteristic signs of wet gangrene**

a) tissue mummification

b) edema and swelling of tissues

c) demarcation zone is expressed

d) demarcation zone is not expressed

**15. Signs of clinical death**

a) stop of breathing

b) absence of reflexes

c) circulatory arrest

d) lowering the temperature

**16. The cause of infarction**

a) long spasm

d) hemorrhage

c) embolism

d) functional stress in low circulation

e) thrombosis

**Answers to tests**

|  |
| --- |
| 1.c 5.c 9.bcda 13.c  2.bd 6.abc 10.d 14.bd  3.ac 7.d 11.b 15.ac  4.c 8.ad 12.ac 16.ace |

**theme 2: the Morphology of pathological accumulation of the of endogenous and exogenous product.**

**Lesson Content**

**Prepare the answers the following questions**

1. Dystrophy as a kind of tissue damage. Functional and morphological essence of dystrophy. Etiological factors, basic pathogenetic dystrophy links , morphogenesis. Principles of dystrophies classification.

2. Intracellular accumulation: definition, mechanisms of development. Lipid accumulation (lipidoses): causes, patho- and morphogenesis, clinical and morphological characteristics, diagnostic methods and outcomes. Steatosis. Fatty changes in the myocardium, liver, kidneys. Cholesterol and its esters. Acquired and congenital disorders of lipid metabolism, morphological characteristics.

3. The accumulation of proteins (disproteinoses): Causes, patho- and morphogenesis, morphological characteristics and methods of diagnosis, clinical symptoms and syndromes, consequences.

4. The accumulation of glycogen: causes, patho- and morphogenesis, morphological characteristics and methods of diagnosis, clinical manifestations, outcomes. Acquired and congenital glycogen storage.

5. Stromal vascular degeneration. Determination the basic conditions and mechanisms of development, classification. Protein stromal-vascular dystrophies: mucoid swelling, fibrinoid swelling, determination, morphological manifestations, outcomes.

6. Fat vascular-stromal dystrophies, definition, principles of classification. The causes of obesity, morphological changes in the organs, the clinical significance, outcomes of local and general obesity.

7. Hyaline changes. Intracellular and extracellular hyaline: morphogenesis, morphological characteristics. Hyaline changes in various pathological conditions

8. Metabolic pigments (chromoproteids). Exogenous pigments. Endogenous pigments: types, formation mechanism, morphological characteristics and methods of diagnosis.

9. Metabolic hemoglobinogenic pigments. The main causes and morphological changes in the exchange of oraganisme in violation of hemoglobin. Hemosiderosis (local, systemic), hemochromatosis. Bilirubin metabolic, morphological characteristics. Jaundice. Classification, causes and mechanisms of development of jaundice.

10. Disorders of lipofuscin and melanin. Classification, clinical and morphological characteristics, outcomes.

11. Disorders of nucleic acids. Classification, clinical and morphological characteristics, clinical manifestations, outcomes.

12. Pathological calcification (calcifications). Types of calcifications: dystrophic, metastatic. Reasons of patho- and morphogenesis, morphological characteristics, diagnosis, clinical manifestations, outcomes.

**The practical part**

Micropreparations: To study, sketch and label the listed morphological signs.

**1. fatty dystrophia of myocardium**. Stain: Sudan III.

Near the venules and veins (a) in cardiomyocytes accumulation of fat droplets is observed (b), there is no transverse striations cell nucleus lysed; (c) other fatty cardiomyocytes are free from fatty inclusions.

**2. FATTY DYSTROPHIA OF LIVER.** Stain: Sudan III.

(a) Hepatocytes are increased, filled with fat droplets, the core is shifted to the periphery (b).

3 **Adiposity of** **myocardium**. Stain: hematoxylin and eosin.

a) layer of fatty tissue, (b) thinned muscle fibers.

**4. Hyalinosis of vessels of the spleen**. Stain: hematoxylin and eosin.

(a) The artery lumen is narrowed sharply, (b), the arterial wall is significantly thickened due to the deposits in the intima of the homogeneous mass, muscle fibers of tunica are atrophied, (c) the number of cells in lymphoid follicles is decreased.

**5. Liver with obstructive jaundice**. Stain: hematoxylin and eosin.

(a) The bile ducts are dilated, (b) fatty degeneration of hepatocytes in the centers of bile location.

**6. pigmented nevi**. Stain: hematoxylin and eosin.

(a) The accumulation of melanocytes in the dermis.

**7. Brown atrophy of the liver**. Stain: hematoxylin-eosin. A) Hepatic cells and their nuclei are reduced; B) The space between the beams is expanded by thinned liver; C) hepatocytes cytoplasm center contain lots of small brown granules (lipofuscin).

**8. Brown atrophy of the myocardium.** Stain: hematoxylin-eosin. A) cardiomyocyte and their nuclei are reduced; B) close to the nuclei of cardiomyocytes cytoplasm contains clusters of brown granules (lipofuscin).

**9. Brown induration of the lung**. Perl's reaction. In the lung tissue on a background a) hyperemia and edema, b) deposition of hemosiderin, which gives a positive reaction on iron and bluish-green color, the proliferation of connective tissue in the alveolar septa, around the bronchi and blood vessels is observed .

**macropreparations:**

1. fatty dystrophia of MYOCARDIUM («tiger heart").

Enlarged heart, flabby consistence, chambers of the heart are stretched resemble clay in the trabeculae and papillary muscles under the endocardium yellow and white striations are marked.

Causes: occurs due to hypoxia (in diseases of the blood circulatory insufficiency), intoxication (alcoholism, infectious diseases, poisoning by phosphorus, arsenic).

Exodus: a reversible process, chronic heart failure.

**2. fatty dystrophia of liver.**

The liver is enlarged in size of loose consistency, yellow color, the cut has a greasy look.

Cause: vitamin deficiency, diabetes, general obesity, alcohol intoxication, toxins.

Exodus: a reversible process; hepatic failure.

**3. Adiposity of MYOCARDIUM**

Heart is increased in size, under the epicardium accumulation of large amounts of fat is determined, fatty tissue grows into the stroma of the myocardium.

Causes: alimentary, cerebral, endocrine, hereditary obesity.

Exodus: reversible changes, heart failure, cardiac rupture.

**4. Hyalinosis of Tht splenic capsule**.

Spleen capsule thickened, dense whitish color, pulp body is not changed.

Reasons: inflammation,

Outcome: not favorable.

**5. Skin with Addison's disease**.

Skin is dark brown.

Reasons: Tumor of adrenal glands, tuberculosis of adrenal glands.

**6. The Birthmark (pigmented nevus**).

The drug portion of the skin, which has drain-teaching irregular brown color, not bulging above the surface.

Causes: congenital hypermelanosis.

Exodus: unchanged malignancy.

**7. lung Silicosis:** lobe of the lung is sealed, the cut-views are blackened scars dense in the center of some scars visible cavities arising at the site of the local ischemic necrosis.

Reason: aspiration of quartz dust particles.

Complications and outcomes: pulmonary fibrosis, pulmonary heart development.

**Tests**

**Select one or more correct answers**

**1. Decomposition is**

a) excessive penetration of metabolic products from blood (lymph) in to cells or intercellular substance

b) collapse of cell ultrastructure and intercellular substances

c) synthesis in a cell or tissue of substances not normally found in them

d) formation of products of one kind exchange from the other

**2. Mucoid swelling of connective tissue it is**

a) reversible process

b) irreversible process

c) transient process

**3. The cause of death in obesity can be**

a) cardiac rupture

b) renal insufficiency

c) adrenal insufficiency

d) hepatic impairment

**4. Infiltration- it is**

a) excessive penetration of metabolic products from the blood (lymph) in cells or intercellular substance

b) the collapse of cell ultrastructure and intercellular substance

c) synthesis in cells or tissues of substances, which normally does not occur

d) the formation of one kind of exchange of products for the expense of the other.

**5. Development mechanism of fatty degeneration - it is**

a) decomposition

b) transformation

c) infiltration

d) warped synthesis

**6. Systemic hyalinosis can develop in**

a) hypertension

b) chronic gastric ulcer

c) cholecystitis

d) diabetes

e) rheumatic diseases

**7. Warped synthesis – it is**

a) excessive penetration of metabolic products from the blood (lymph) in to cells or intercellular substance

b) collapse of cell ultrastructure and intercellular substance

c) synthesis in cells or tissues of substances, which normally does not occur

d) formation of the products of one kind exchange of other

**8.What is revealed by sudan-3 in the tissues?**

a) proteins

b) fats

c) glycogen

d) calcium

**9. What is the outcome of the hyaline-droplets dystrophia?**

a) transition to turbid swelling

b) transition to granular dystrophy

c) transition to coagulation necrosis

d) transition to liquefactive necrosis

**10. "Tiger heart" is a reflection of**

a) hyaline droplet degeneration

b) fatty degeneration

c) glycogenosis

d) dropsical dystrophy

**11. Basis of the fibrinoid swelling is**

a) surface disorganization of connective tissue

b) deep connective tissue denaturation

c) mucous tissue degeneration

d) increase in vascular permeability

e) appearance of fibrin in tissues

f) emergence of lipids in tissue

**12. Transformation – it is**

a) excessive penetration of metabolic products from the blood (lymph) in to cells or intercellular substance

b) the collapse of cell ultrastructure and intercellular substance

c) synthesis in cells or tissues of substances not normally found in them

d) formation of the products of one kind exchange from the other

**13. Morphological characteristics of "tiger heart" are**

a) reduced heart size

b) stretched heart chambers

c) significant increase in fatty tissue under the epicardium

d) brown myocardium section

e) under the endocardium on the papillary muscles there are visible yellow and white striations

**14. To the mechanisms of the development of dystrophies refer:**

a) infiltration

b) proliferation

c) aggregation

d) phanerosis

e) transformation.

**15. To the protein parenchymatous dystrophies refer:**

a) hydropic

b) fat

c) horn

d) hyalinedroplet

e) metabolic

**16. Mallory’s bodies formed by**

a) horny dystrophy

b) balloon dystrophy

c) leukoplakia

d) hyaline droplet degeneration

e) fatty degeneration

**17. Horn dystrophy falls into:**

a) carbohydrate dystrophy

b) protein dystrophy

c) fatty degeneration

d) mineral dystrophy

e) cancer variety

**18. The characteristic signs of mucoid swellin**

a) glycosaminoglycans are accumulate

b) hydrochloric acid is accumulated

c) hyaluronic acid is accumulated

d) processes of hydration, swelling are being developed

e) phenomenon metachromasy disappears

**19. hyalinosis vessels - it is typical for:**

a) hyaline is accumulated in the lumen of blood vessels

b) hyaline is accumulated in the subendothelial space

c) elastic plate is pushed inwards and stored

d) elastic plate is pushed outwards and destroyed

e) the lumen vessel is sharply narrowed

**20. The characteristic signs of fibrinoid swelling**

a) it is a superficial and reversible disorganization

b) it is deep and irreversible disorganization

c)it is characterized by the destruction of the basic substance and the fibers

d) it is characterized by the phenomenon of metachromatia

e) it is characterized by a sharp increase in vascular tissue permeability

**21. Options for general adiposity**

a) hypertonic

b) hyperlipidemic

c) hypertrophic

d) hyperplastic

e) hypersthenic

**22. What is referd to the stromal-vascular disproteinosis**

a) glycogenoses

b) mucoid swelling

c) hyalinosis

d) lipidosis

**23. At hyalinosis the connective tissue is**

a) loose

b) thick

c) whitish

d) black

d) translucent

**24. Mucoid swelling is**

a) surface disorganization of connective tissue

b) deep disorganization of connective tissue

c) reversible disruption of connective tissue

d) irreversible disruption of connective tissue

e) characterized by the emergence of the phenomenon of metachromatia

**25. Developing of a disseminated hemosiderosis leads to**

a) vasculitis

b) necrosis of the vascular wall

c) intravascular hemolysis

d) the formation of a hematoma

e) rupture of aortic aneurysm

**26. What leads to the development of wide-spread hemosiderosis?**

a) vasculitis

b) necrosis of the vascular wall

c) intravascular hemolysis

d) the formation of a hematoma

e) rupture of aortic aneurysm

**27. The cause of the albinism**

a) increase the amount of tyrosinase

b) failure of the adrenal glands

c) reducing of tyrosinase amount

d) lack of vitamin D

**28. In brown colored atrophy the color of the organism depends on the** accumulated of

a) hemosiderin

b) gemofustsina

c) lipofuscin

d) free iron

e) protein

**29. The pigment formed in the erosion of ulcers and acute gastric mucosa**

a) gemomelanin

b) hydrochloric acid hematin

c) porphyrin

d) bilirubin

**30. The cause of congenital hemochromatosis**

a) increased absorption of exogenous iron

b) increased breakdown of red blood cells

c) erythremia

d) lack of exogenous iron

e) diapedetic hemorrhage

**31. the general melanosis applies to**

a) leucoderma

b) pigmentosum xeroderma

c) nevus

d) Addison's disease

e) albinism

**32. Hematoidin is formed in**

a) fresh hemorrhages

b) in the old hematomas

c) in the scar of infarction

d) in the caseous necrosis

e) in tumor

**33. Petrifikation can be formed in**

a) pulmonary tuberculosis foci

b) hematomas

c) in gumma

d) the foci of purulent inflammation

e) in atherosclerotic plaques

**34. The pigment formed under physiological conditions,**

a) melanin

b) hemosiderin

c) bilirubin

d) porphyrin

e) adrenochrome

**35. Lipofuscin formation is increased in the cells at**

a) body atrophy

b) hypertrophy of the body

c) the aging of the organism

d) malignancy

e) beriberi

**36. Causes of hepatocellular jaundice are**

a) compression of the duct tumor

b) viral hepatitis

c) hemolysis

d) parasitic infestation of the bile ducts

**37. The diseases that lead to hypercalcemia:**

a) parathyroid adenoma

b) atrophy of the heart

c) necrotizing colitis

d) hypervitaminosis D

e) hyperthyroidism

**38. Pigments gaving positive reaction to iron**

a) bilirubin

b) hematoidin

c) ferritin

d) hemosiderin

e) gemomelanin

**39. Proteinogenic pigments include**

a) melanin

b) gemomelanin

c) adrenochrome

d) epinephrine

e) pigment granules of enterochromaffin cells

**40. mechanical jaundice can be caused by**

a) acute hepatitis

b) cholelithiasis

c) biliary atresia

d) hypoplasia of bile ducts

e) hemolytic disease

**41. The hereditary systemic melanosis**

a) gemomelanoses

b) carcinoid syndrome

c) melanoma

d) vitiligo

e) pigmentosum xeroderma

**42. Process, reflecting a sharing violation of proteinogenic pigments, include**

a) melanosis

b) calcification

c) leukoplakia

d) albinism

e) leukoderma

**43. The pigments being the varieties of hematin**

a) formalin pigment

b) Melanin

c) malarial pigment

d) hemin

e) gemomelanin

**Answers to tests**

|  |
| --- |
| 1.b 5.ab 9.c 13.be 17.b 21.cd 25.c 29.b 33.ace  2.a 6.ade 10.b 14.ade 18.acd 22.bc 26.c 30.a 34.ac  3.a 7.c 11.bde 15.acd 19.bde 23.bcd 27.c 31.bd 35.ace  4.a 8.b 12.d 16.d 20.bce 24.acd 28.c 32.bc 36.b  37.acd 38.cd 39.bcd 40.bde 41.e 42.ade 43.acde |

**theme 3: DISORDERS of blood and lymph circulation. Arterial and venous engorgement. Bleeding and hemorrhage. Ischemia. Thrombosis. Embolism. Disseminated intravascular coagulation. SHOCK.**

**Lesson Contents**

**Prepare answers to these questions:**

1. Arterial hyperemia (redness). Causes, types, morphology.

2. Venous hyperemia: general and local, acute and chronic. Local venous hyperemia, causes of morphological manifestations, outcomes.

3. Venous stagnation in the pulmonary circulation: pathogenesis and morphogenesis, clinical and morphological characteristics, outcomes.

4. Venous stagnation in the systemic circulation: patho- and morphogenesis, clinical and morphological characteristics, outcomes. Venous hyperemia in the portal vein (portal hypertension): pathogenesis and clinical and morphological manifestations.

5. Bleeding: external and internal hemorrhage. Causes, types, clinical and morphological characteristics. Hemorrhagic diathesis.

6. Stasis. Mechanisms of development, reasons, value.

7. Thrombosis. Definition, local and general factors of thrombosis. Thrombus, its types, morphological characteristics. Vein thrombosis. Arterial thrombosis. Thrombosis in the cavities of the heart. Value and outcomes of thrombosis.

8. Embolism: definition, types, causes and morphological characteristics. Orthogradic, retrograde and paradoxical embolism. Thromboembolism: reasons for the development, clinical significance. Pulmonary embolism, acute pulmonary heart. Thromboembolism: clinical and morphological characteristics.

9. Ischemia (anemia). Definition, classification, causes, mechanisms of development, morphological characteristics and methods of diagnosis, clinical significance. The role of collateral circulation. Acute and chronic ischemia. Heart attack: definition, causes, classification, morphological characteristics of different types of heart attacks, complications, outcomes.

10. DIC. Definition, mechanisms of development, stage, morphological characteristics, clinical manifestations.

11. Shock. Definition, types, mechanisms of development, stage, morphological characteristics, clinical manifestations.

**The practical part**

**Micropreparations.** To study, to learn to sketch and identify morphological features listed.

* **Acute venous hyperemia (edema) of lungs**. Stain: Hematoxylin and eosin.

a) enhanced, plethoric blood vessels of interalveolar partitions, b) eosinophilic content (protein transudate) with a dash of macrophages and desquamated epithelium in the lumen of the alveoli.

* **Cerebral hemorrhage**. Stain: Hematoxylin and eosin. a) in the brain tissue - accumulation of hemolytic and preserved red blood cells, b) no substance of the brain in the center of hemorrhage (bundles of brain tissue with blood), c) pericellular and perivascular edema.
* **Lung brown induration**. Perls reaction. Against the backgrounds of a) hyperemia and edema, b) deposits of hemosiderin, which gives a positive reaction to iron while its grains are colored by blue-green color, in the lung tissue proliferation of connective tissue in the alveolar septa, around the bronchi and blood vessels are observed.
* **Chronic hepatic congestion ("muscat liver")**. Stain: Hematoxylin and eosin. In the center of the lobules there are found: a) expansion and congestion of the veins and sinusoids. b) necrosis and atrophy of hepatocytes. c) On the periphery of the lobules sinusoidal perfusion is normal, the structure of hepatic beams is preserved, hepatocytes in the state fatty dystrophy.
* **Myocardial infarction**. Hematoxylin and eosin stain. a) the infarct area stands out for its homogeneous pink color, the contours of the muscle fibers are retained, but they are completely devoid of nuclei and striated striation. b) Around the infarct zone there is visible demarcation inflammation: advanced thin-walled vessels with a full-blooded boundary standing leukocytes. c) expressed leukocyte infiltration and foci of perivascular hemorrhages, d)  over the demarcation zone normal muscle tissue is visible.
* **Metastatic abscesses in the lung**. Hematoxylin and eosin stain. In the preparation of the lung tissue numerous foci of purulent inflammation are visible, presented as an accumulation of polymorphonuclear leukocytes (a) with the melting of the lung tissue in the center of the foci (b), around foci of inflammation the vessels are dilated and congested (c), sometimes with areas of perivascular hemorrhages (d ).
* **Metastatic abscesses in the kidneys** (embolic nephritis abscess). Hematoxylin and eosin stain . In preparation of cortex and medulla of kidney numerous foci of purulent inflammation are visible presented as an accumulation of polymorphonuclear leukocytes (a). In the center of the foci kidney tissue is melted (b) visible microbial emboli (c), around the foci of inflammation the vessels are dilated and congested (d), sometimes with areas of perivascular hemorrhage (d).

**Macropreparations.**

**1. Acute hyperemia of meninges in the flu**. In the preparation there is brain. Soft meninges are edematous, with advanced plethoric blood vessels gyri are smoothed.

Reasons: the flu.

Complications: brain edema on the background of serous meningitis. Outcomes: As a rule, a full recovery.

**2. Muscat liver**. In preparation - liver, increased in size, dense consistency, with a smooth surface and rounded front edge. Cut surface of the organ is motley, gray-yellow (fatty dystrophia of hepatocytes at the periphery of the lobules) with dark red specks (central stagnant parts of lobules) and resembles nutmeg.

Causes: Chronic heart failure with the development of venous stasis in the systemic circulation: cardio sclerosis of various origins, tricuspid valve failure. Hypertension in the pulmonary circulation, chronic lung disease with the outcome in pulmonary fibrosis.

Complications and outcomes: the transition to the stagnant (cardiac) fibrosis (cirrhosis) of the liver, development of portal hypertension, ascites, splenomegaly, varicose portocaval anastomosis, bleeding, anemia.

**3. Brown induration of lungs**. In the preparation there are lungs, increased in size, of brown ("rusty") color, dense consistency. Around the bronchi, vessels and diffusely in the lung tissue visible layers of thick white tissue (fibrosis) are presented. The changes are more pronounced in the lower and posterior portions of the lung.

Causes: Chronic heart failure.

Complications and outcomes: respiratory insufficiency exacerbates chronic heart failure - pulmonary heart disease is progressing.

**4. Hemopericardium with cardiac tamponade.**

In preparation there is heart with heart shirt in cross section. Pericardial accumulation of coagulated blood. On the back wall of the left ventricle there is a portion of necrosis with violation of myocardium integrity, measuring about 2.0 cm.

The reasons: the rhexis of acute or chronic heart aneurysm, rupture of the heart wall by transmural infarction, rupture of the wall by the heart in obesity.

Outcome: death.

**5. Brain hematoma.**

In the parietal-temporal region of the right hemisphere - accumulation of coagulated blood of brownish-red color. The matter of the brain is destroyed in the area of hemorrhage.

Reason: aneurysm rupture, rupture of hyalinized vascular microcirculation in hypertensive crisis, necrosis of the wall in the atherosclerotic plaque ulceration.

Complications: paralysis, paresis, a break – through of blood into the ventricles of the brain.

Outcome: death or the formation of cysts on the site of the hematoma with a "rusty" walls.

**6. Myocardial infarction.** In the lateral wall of the left ventricle, there is a pathological site of irregularly shaped lesions presented by foci grayish yellow color (coagulative necrosis). Around there is the area of plethora and hemorrhages (the demarcation zone). In the lumen of the descending branch of the left coronary artery occlusive thrombus is visible. Coronary heart arteries are sclerotic with fibrous plaques. On the part of endocarditis there are visible thrombotic overlays.

Causes: thrombosis, long spasm, thromboembolism, myocardial functional overstrain in the presence of atherosclerotic occlusion.

Complications: pulmonary edema, cardiogenic shock, arrhythmias and conduction, myocardial rupture (3-10 days with transmural infarction) or acute rupture of the aneurysm (4-14 days), thromboembolism. Complications of later period: chronic cardiac aneurysm.

Outcome: death or transition in to macrofocal cardiosclerosis.

**7. Pulmonary embolism**. In preparation organocomplex the heart and lungs. The lumens of the pulmonary trunk and both pulmonary arteries are obturated by vermiform dryish thromboembolic masses of gray-red color with a corrugated surface.

Reasons: deep vein thrombosis of the lower limbs, followed by a separation of blood clots. Flutter and atrial fibrillation. Cavities thrombosis of the right heart (most often - right atrial appendage).

Complications: heart attack, pneumonia, metastatic abscesses (abscesses) of lung.

Outcome: death or the formation of hemorrhagic infarction lung followed by focal pneumosclerosis of infarcted area.

**Test tasks**

**Select one or more correct answers**

**1. Changes developing by venous stasis in the portal vein system**

a) "nutmeg" liver

b) stagnation in the kidneys

c) congestive spleen induration

d) anasarca

e) ascites

f) the esophageal varices

**2. Morphological characteristic of "nutmeg"liver**

a) congestion of the central parts of the liver lobules

b) congestion on the periphery of the lobules

c) atrophy and death of centrolobular hepatocytes

d) fatty degeneration of the peripheral hepatocytes

**3. the basic Morphological manifestations of acute left ventricular failure**

a) "nutmeg" liver

b) pulmonary edema

c) pulmonary hemosiderosis

d) body induration

**4. Brown induration mild develops in**

a) acute left ventricular failure

b) chronic decompensated right heart ventricle

c) chronic insufficiency of the left ventricle

d) chronic lung abscess

**5. The accumulation of clotted blood in tissue under the preservation of tissue elements**

a) hematoma

b) hemorrhagic impregnation

c) hemorrhagic infiltration

d) petechiae

e) ecchymosis

**6. The liver in chronic venous engorgement**

a) increased

b) reduced

c) fabric brown

d) fabric bluish color with white specks

e) fabric gray-yellow in color with dark red specks

**7. to the Internal bleeding apply**

a) melena

b) hemothorax

c) hemopericardium

d) hematuria

e) hemoperitonium

**8. The main causes of bleeding**

a) exsicosis

b) corrosion of the vessel wall

c) rupture of the vessel wall

d) blood stasis in the vessels

e) thrombosis

**9. chronic venous plethora in lungs results in**

a) hemomelanoz

b) hemosiderosis and sclerosis

c) cyanotic induration

d) hemochromatosis and sclerosis

e) brown induration

**10. there are The following types of haemorrhage:**

a) hematoma

b) exsicosis

c) ecchymosis

d) chylothorax

e) bruise

**11. Outcome of hemorrhage can be:**

a) suppuration

b) encapsulation

c) chylothorax

d) the formation of cysts

e) melena

**12. In skin bya total of chronic venous engorgement are observed:**

a) raising of the temperature

b) lowering of the temperature

c) cyanosis

d) multiple sclerosis

e) hemochromatosis

**13. Small petechial hemorrhages in the skin:**

a) bruise

b) lymphoedema

c) petechiae

d) ecchymosis

e) hematoma

**14. Closure of the artery thrombus can lead to:**

a) atherosclerosis

b) collateral hyperemia

c) vacant hyperemia

d) anemia

e) ischemia

**15. The plane hemorrhages in the skin are called:**

a) petechiae

b) ecchymosis

c) purpura

d) hematoma

e) hemorrhagic impregnation

**16. Choose the most frequent complication of deep vein thrombophlebitis in** **lower limb:**

a) ischemic cerebral infarction

b) renal infarction

c) myocardial infarction

d) hemorrhagic pulmonary infarction

e) bowel gangrene

**17. Specify the reasons of shock:**

a) a decrease in cardiac output

b) an increase in cardiac output

c) the common peripheral vasodilation

**18. Which of these ways of embol moving is said to be paradoxic:**

a) moving along blood flow

b) moving against blood flow

c) moving from the veins of the systemic circulation in the arteries of the systemic circulation

**19. «Canalisation» of thrombus is:**

a) the restoration of blood flow in the thrombosed vessel

b) the germination of vessels clot

c) septic of thrombus melting

**20. Favorable outcome of thrombosis includes:**

a) thrombus abruption

b) aseptic autolysis of thrombus

c) the organization of the thrombus

d) septic thrombus melting

e) calcification of thrombus

**21. Main source of pulmonary embolism:**

a) the pulmonary veins

b) mesenteric vein

c) the veins of the lower extremities

d) mitral valve cusps

**22. In prolonged ischemia there are develops:**

a) atrophy of the parenchyma of the organ

b) hyperplasia of the parenchyma

c) stroma atrophy

d) hyperplasia of fibroblasts

e) sclerosis

**23. Thromboembolus from crus vein with blood flow usually falls into:**

a) inferior vena cava

b) jugular vein

c) portal vein

d) right atrium

e) pulmonary artery

**24. Infarction of the spleen is characterized by:**

a) white color

b) red color

c) red with white crown

d) irregular shape

e) triangular shape

**25. Infarction - is:**

a) ischemic necrosis

b) trophoneurotic necrosis

c) toxic necrosis

d) vascular necrosis

e) the angiogenic necrosis

**26. In developing pulmonary infarction:**

a) gemomelanoses

b) hemosiderosis

c) organization

d) miomalacia

e) lipofuscinosis

**27. Hemorrhagic infarction typical for:**

a) Heart

b) liver

c) Spleen

d) Kidney

e) lungs

**28. Pulmonary fat embolism usually develops under:**

a) fatty liver

b) fracture of long bones

с) crushing of the subcutaneous tissue

d) atherosclerotic plaque ulceration

e) the alimentary obesity

**Answers** **to tests**

|  |
| --- |
| 1.cef 5.bc 9.be 13.cd 17.ac 21.c 25.a  2.acd 6.ae 10.ace 14.be 18.c 22.ade 26.bc  3.b 7.bce 11.abd 15.c 19.a 23.ade 27.e  4.c 8.bc 12.bcd 16.d 20.bce 24.ae 28.bc |

**Theme 4: the MORPHOLOGY EXUDATIVE inflammation. Productive inflammation. Granulomatous Diseases.**

**Lesson Content**

**Answer to these questions:**

1. Inflammation: definition, nature and biological significance. The problem of local and general understanding of inflammation. Clinical signs and symptoms of inflammation (local and systemic). Common manifestations of inflammation, etiological features.

2. Acute inflammation. Etiology and pathogenesis. The reaction of the blood vessels in acute inflammation. Transudate, exudate, edema, stasis. The emigration of leukocytes, pus formation. Outcomes of acute inflammation.

3. Morphological manifestations of acute inflammation. Exudative inflammation: serous, fibrinous, purulent, catarrhal, hemorrhagic, mixed.

4. Chronic inflammation. Definition, etiopathogenesis, cell cooperation (macrophages, lymphocytes, plasma cells, eosinophils, fibroblasts and others.). Morphological features (the nature of the infiltrate, persistent degradation of connective tissue) outcomes.

5. Productive inflammation. Species. Etiology, pathogenesis, clinical and morphological characteristics and methods of diagnosis, outcomes. Interstitial inflammation with the formation of polyps and warts.

6. Productive inflammation around the animals parasites (echinococcosis, cysticercosis, trichinosis). Actinomycosis, clinical and morphological characteristics.

7. Granulomatous diseases (acute and chronic). Cell kinetics of granulomas. Pathogenetic types of granulomas.

8. Specific granulomas (tuberculosis, syphilis, leprosy, malleus, rinoskleroma) and nonspecific granuloma.

**The practical part**

**Slides: To study, sketch and label listed morphological features.**

**1. lobar pneumonia**. Stain: Hematoxylin and eosin.

All the alveoli are filled with exudate containing a) fibrin strands; b) leukocytes.

**2. Diphtheritic colitis**. Stain: Hematoxylin and eosin.

a) mucosal necrosis b) fibrinous exudate leukocytes in) survived gland d) submucosa is hydropic, full-blooded.

3**. Purulent nephritis.** Stain: Hematoxylin and eosin.

a) abscesses with colonies of microbes and PMN, b) vessels around the abscess dramatically expanded, full-blooded.

4**. Miliary tuberculosis**: Stain: Hematoxylin and eosin.Typical tuberculous granuloma showing an area of central necrosis (а) surrounded by multiple epithelioid cells (b), lymphocytes (c), and Langhans-type giant cells (d).

**5. Actinomycosis of liver**. Stain: Hematoxylin and eosin.

Find ulcers which determine A) Druses, surrounded by connective tissue and B)by macrophages.

***Macropreparations***

**1. Croupous pneumonia** (the stage of gray hepatization).

The affected lobe is increased in size, heavy, dense, airless, grain surface on the cut, from the cut surface turbid liquid flows. The pleura is thickened, with fibrinous deposits.

Causes: The disease is provoked by pneumococci of types 1-3, rarely by Klebsiella and other pathogens.

Outcomes favorable: fibrinous exudate resorption by proteolytic enzymes PMN and removing it through the lymphatic drainage and with phlegm when coughing. Unfavorable: 1) carnification - fibrinous exudate organization due to insufficient activity of proteolytic enzymes of neutrophils. 2) abscess formation - the formation of abscess cavity as a result of excessive activity of proteolytic enzymes of neutrophils. 3) gangrene – by joining the anaerobic flora.

**2. Fibrinous pericarditis** ("hairy heart").

Dull epicardium, covered with grayish-yellow roughs imposed in the form of threads and resemble hair ("hairy heart"). Overlays can be easily removed.

Causes: uremia, rheumatic fever, transmural myocardial infarction, lobar pneumonia.

Outcomes: obliteration of the pericardial cavity, petrification and ossification of organized fibrinous exudate - "armored heart."

**3. Diphtheritic colitis:**

Colon wall thickened, hydropic. The mucous membrane is covered with a grayish-yellow rough pellicle, tightly linked to the underlying tissues.

Causes: severe forms of dysentery.

Outcomes: in the place of deep ulcers arising from the rejection of the pellicle scars are formed.

**4.Purulent nephritis.**

Kidneys are symmetrically enlarged, of flabby consistency. On the cut in the cortex and medulla of kidney lesions seen numerous gray-yellow color of 1-2 mm. in diameter. Vessels are dilated, congested.

Causes: hematogenous metastasis of purulent emboli in sepsis.

Outcomes: scars are formed at the site of abscesses, the process of chronicity with the formation of pyogenic membrane.

**5. Milliarny pulmonary** tuberculosis. Lungs are swollen, tight. Along all pulmonary field numerous small (D = 1-2 mm.), round, gray-yellow in color, with clear boundaries pockets are seen.

The reasons: the development of widespread hematogenous infection of Mycobacterium tuberculosis screenings foci in various organs after a primary tuberculosis suffering.

Complications: caseous pneumonia, tuberculous meningitis, tuberculous sepsis.

Outcomes: the lethal outcome of tuberculous leptomeningita, caseous tuberculous pneumonia or sepsis.

**6. Brain gumma.** In brain tissue rounded formation gray-white color, round, with clear boundaries D = 5-7 cm. is determined, compressing the lumen of the lateral ventricles.

Causes: development of gumma in tertiary syphilis. The causative agent is a pale treponema.

Complications: Gumma compression of the lateral ventricles leads to the disruption of cerebral fluid outflow, with the development of hydrocephalus, with the development of neurological symptoms. Brain swelling

Outcomes: Death due to the swelling of brain.

**7. Syphilitic mesoaortitis and aortic aneurysm***.* Ascending department of aorta and aortic arch are deformed, uneven sclerotic, its inner shell is rough, wrinkled and bumpy. In place of the thinning of the aortic wall aneurysm is being formed.

Causes: The causative agent is pale treponema, through vasa vasorum penetrates into the tunica media of the aortic wall. Necrosis in the wall of the aorta causes destruction of elastic and proliferation of granulative tissue.

Complications: the formation of the aneurysm of the ascending part and aortic arch and the transition of the inflammatory process in the aortic valve followed by the formation of heart disease

Outcomes: aneurysm rupture leads to sudden death.

**8. Single chamber Echinococcus of various organs** - spleen, liver, lungs, heart. In liver, lungs, heart one or more bubbles are formed. They are covered by a chitinous sheath and are filled with a clear, colorless liquid containing succinic acid. From the inner sheath layer branch bubbles with scolex are growing. On the border with echinococcosis - a chronic productive inflammation, the gradual formation of a fibrous capsule.

Causes: The causative agent - Echinococcus granulosus.

**Tests**

**select one or more correct answers**

**1. What are the main cells in the focus of exudative inflammation:**

a) neutrophils

b) lymphocytes

c) the monocytes

d) mast cells

e) histiocytes

**2. What cells predominate in the purulent exudate composition:**

a) fibroblasts

b) erythrocytes

c) leukocytes

d) macrophages

e) plasma cells

**3. Select types of purulent inflammation:**

a) catarrhal

b) croupous

c) abscess

d) spilled

e) diphtheritic

**4 What is the composition of exudate in fibrinous inflammation:**

a) fibrin

b) neutrophils

c) mast cells

d) Viruses

e) transudate

**5. Select types of fibrinous inflammation:**

a) abscess

b) flegmona

c) catarrhal

d) diphtheritic

e) croupous

**6. Localization of catarrhal inflammation:**

a) stroma of organ

b) serous membranes

c) liver parenchyma

d) mucosal

e) capsule of bodies

**7. Select the correct determination of inflammation:**

a) a vascular mesenchymal tissue response to the injury

b) a restoration of lost structures

c) a unchecked growth of cellular elements

d) an ultrastructure hyperplasia

e) a circulation of foreign bodies in the blood stream

**8. Morphological manifestation of alteration:**

a) multiple sclerosis

b) atrophy

c) necrosis

d) dystrophy

e) fibrosis

**9. to Exudative inflammation are include:**

a) fibrinous inflammation

b) putrid inflammation

c) interstitial inflammation

d) granulomatous inflammation

e) purulent inflammation

**10. Phlegmon is characterized by:**

a) the presence of catarrh

b) the presence of fibrinous inflammation

c) delineation from the adjacent tissues by the shaft granular tissue

d) the presence of pyogenic membrane

e) the presence of diffuse purulent inflammation

**11. The initial phase of inflammation is:**

a) exudation

b) proliferation

c) alteration

d) phagocytosis

**12. in Purulent exudate, unlike serous one prevail:**

a) exfoliated cells of the surface epithelium

b) exfoliated cells of the mesothelium

c) neutrophils

d) mucus

e) pus corpuscles

**13. Proliferative phase characterizes by:**

a) tissue damage

b) the blood circulation violation

c) formation of exudate

d) phagocytosis

e) the reproduction of cells in the area of inflammation

**14. Caseous necrosis is encountered in:**

a) diphtheria

b) gas gangrene

c) tuberculosis

d) cerebral infarction

e) renal infarcts

**15. Granuloma is the:**

a) accumulation of polymorphonuclear leukocytes

b) presence of mucus in the exudate

c) limited productive inflammatory reaction

d) formation of fibrin

**16. in the formation of tuberculous granuloma all the** cells **participate**:

a) epithelioid ones

b) lymphocytes

c) neutrophil leukocytes

d) giant cells of Langhans-type

**17. prevaling reaction of tissue in granulomatous inflammation:**

a) exudative

b) productive

c) alterative

**18. Granulomatous inflammation is a variaty of:**

a) productive inflammation

b) exudative inflammation

c) interstitial inflammation

**19. Tuberculous granuloma is characterized by:**

a) neutrophils

b) lymphocytes

c) epithelioid cells

d) eosinophils

**20. Tuberculous granuloma is characterized by:**

a) Virchow cells

b) Langhans cells

c) Mikulic cells

d) coagulative necrosis

**21. Indicate the cell whick are the most typical for tuberculous granuloma:**

a) lymphocyte

b) monocyte

c) eosinophil

d) epithelioid cell

**22. in The outcome of tuberculous inflammation may arise:**

a) encapsulation

b) petrification

c) leproma

d) gummas

e) sclerosis

**Answers** **to tests**

1.a 5.de 9.abe 13.e 17.b 21.d

2.c 6.d 10.e 14.c 18.a 22.b

3.cd 7.a 11.c 15.c 19.c

4.ab 8.cd 12.ce 16.c 20.bd

**Theme 6 :ADAPTATION. HYPERPLASIA. HYPERTROPHY. ATROPHY. METAPLASIA. REPAIRATION. CUTANEOUS WOUND HEALING**.

**CONTENTS OF THE LESSON**

QUESTIONS TO ANSWER:

* **Adaptation.** Physiological and pathological. Types of adaptation processes.
* **Hyperplasia.** Definition, causes, mechanisms, types, morphological features. Physiological and pathological hyperplasia.
* **Hypertrophy.** Definition, causes, mechanisms, types, morphological features. Hypertrophy of myocardium.
* **Atrophy.** Definition, causes, mechanisms, types, morphological features.
* **Metaplasia.** Definition, causes, mechanisms, types, morphological features. Displasia.
* **Regeneration.** Definition, causes, mechanisms, types, morphological features.
* **Granular tissue.** Angiogenesis. Wound healing. Morphogenesis of the scar.
* **Pathologic aspects of repair.** Deficient scar formation, excessive formation of the repair components and the formation of contractures.

**PRACTICE**

**MICROPREPARATIONS.** To study, to draw and to identify morphological features.

1. **Granular tissue.** Hematoxylin and eosin stain. A) angiogenesis; B) soft tissue cells C) extracellular matrix.

**2**. **Myocardial hypertrophy**. Hematoxylin and eosin stain. A) enlarged cardiomyocytes; B) enlarged hyperchromic nuclei.

**3**. **Hydronephrosis.** Hematoxylin and eosin stain. A) thinning of the renal cortex; B) glomerular atrophy and sclerosis; C) atrophy and dilatation of tubules; D) interstitial fibrosis.

**5**. **Lung emphysema.** Hematoxylin and eosin stain. A) dilatation of bronchioles and alveoli; B)thinning of the alveolar walls; C) thickening and sclerosing of vessel walls.

**6. Endometrial hyperplasia.** Hematoxylin and eosin stain. A) glands are disorderly placed and not particularly crowded; B) glands usually round but may be irregular with cystic dilation; C) lining epithelium is pseudo stratified or mildly stratified; D) cellular stroma with variable mitotic activity is rich in cells.

**MACROPREPARATIONS:**

**1. Myocardial hypertrophy** – heart is enlarged. Atrial and ventricular walls are thickened. Chambers are dilated. Myocardium is of flabby consistency.

*Causes*: increased organ workload due to arterial hypertension, valve disease, physical activity increased.

*Outcomes*: decomposition.

**2. Splenomegaly** – spleen is enlarged, capsule is tense and has clay forminacut, pulp is plethoric.

*Causes*: infection diseases, hemoblastoses, anemia, sepsis.

*Outcomes*: according to the main desease.

**3. Bladder hypertrophy** – cavity of bladder is enlarged, muscle layer is thickened. Mucosa - with petechial hemorrhages. Prostate gland is enlarged, urethra is contracted.

*Causes:* nodular hyperplasia of prostate gland.

*Outcomes:* urolithiasis, pyelonephritis, urinary retention.

**5. Hydronephrosis –** kidney is enlarged, renal pelvis is dilated, thinning of the renal cortex.

*Causes*: urolithiasis, urinary retention.

*Outcomes*: renal failure.

**Tests**

**select one or more correct answers**

**1. Choose the morphological features of hypertrophy:**

а) lipofuscin granules in cytoplasm

b) hyperchromic nuclei

c) vacuolization of cytoplasm

d) increased size of the cells

**2. Distinctive sign of atrophy is intracellular accumulation of:**

a) hemosiderin

b) lipoproteins

c) melanin

d) lipofuscin

**3. Match the stages of hypertrophy (1, 2) with electron microscopic changes of cardiomyosites:**

1. compensation stage

2. decompensation stage

а) increased myofylament number

b) mitochondrial distraction

c) increased mitochondrial size

d) cytoplasmic lipids appearance

e) increased mitochondrial size and number

**4. Choose organs subject to pathological hypertrophy:**

а) brain

b) heart

c) lung

d) spleen

e) bladder

**5. Choose the process that cause decreasing cell size, tissue size followed by dysfunction of organs:**

а) hypoplasia

b) atrophy

c) agenesis

**6. The reasons of atrophy are:**

а) endocrine stimulation

b) loss of endocrine stimulation

c) pressure

d) diminished blood supply

e) decreased functional load

f) aging

**7. The type of uterus hypertrophy during the pregnancy is:**

a) work hypertrophy

b) neurohumoral hypertrophy

c) vicar hypertrophy

d) hypertrophicproliferation

**8. The reasons of pathological atrophy are development:**

а) stomach cancer

b) aging

c) hemorrhage to pituitary body

d) pituitary body adenoma

e) inadequate production

**9. myocardial hypertrophy is in hypertension refers to:**

a) increased workload

b) vicar

c) neurohumoral

d) developmental anomalies

**10. Match the type of atrophy (1, 2, 3, 4) with the changes in organs (a, b, c, d):**

1. Disfunctional

2. Of diminished blood supply

3. Of pressure

4. Under physical and chemical factors

a) muscle atrophy by the bone fracture

b) interstitial kidney sclerosis

c) insolational skin atrophy

d) hydrocephaly

**11. Match the condition (1, 2, 3, 4) with the process (a, b, c, d):**

1. Increase of mammae size by lactation

2. Myocardial hypertrophy by hypertension

3. Hydronephrosis

4. Endometrial hyperplasia

a) hypertrophy

b) hyperplasia

c) atrophy

d) hypoplasia

**12. The outcomes of glandular endometrial hyperplasia are:**

а) endometrial atrophy

b) involution

c) endometrial metaplasia

d) malignancy

**13. Cell regeneration prevails in all listed tissues except:**

а) heart muscle

b) epithelium of urothelial system

c) endothelium

d) neurons of CNS

e) mesothelium

**14. Granulation tissue growth is a sign of:**

а) physiological regeneration

b) regeneration hypertrophy

c) repair regeneration

d) pathological regeneration

**Answers to the tests**

|  |  |  |  |
| --- | --- | --- | --- |
| 1. b, d | 5. b | 9. a | 13. a, e |
| 2. d | 6. b, c, d, e, g | 10. 1-a; 2- b; 3- d; 4 –c | 14. c |
| 3. 1 –a,c,e; 2 – b, d | 7. b | 11. 1- b; 2 – a; 3 – c; 4 – b |  |
| 4. b, e | 8. a, b, e | 12. b, d |  |

Theme 6 : **DISEASES OF THE IMMUNE SYSTEM**

**CONTENTS OF THE LESSON**

BE RAEDY TO ANSWER THE QUESTIONS:

**1. The morphology of immune system.** Relations with the immunity. The conception of normal and adaptive immune response.

**2. The morphology of normal immune response.** The main machanisms of humoral and cell immunity. Morphological features of immune response.

**3. The diseases of immune system.** Hypersensitivity reactions. Autoimmune disorders. Immunodeficiency sindroms.

**4. The type I hypersensitivity.** Mechanisms, phases, clinical and morphological features. Systemic and localized immediate hypersensitivity reactions.

**5. The type II hypersensitivity.** Phagocytosis or lysis of target cell by activated complement or Fc receptors. Functional derangements without cell or tissue injury. Clinical and morphological features.

**6. The type III hypersensitivity.** Deposition of antigen-antibody complexes. Clinical and morphological features.

**7. The type IV hypersensitivity.** T cell–mediated cytotoxicity. Rejection of tissue transplants. Mechanisms. Clinical and morphological features.

**8. Autoimmune disorders.** General features of hypersensitivity disorders. The development of hypersensitivity diseases. Clinical and morphological features.

**9. Immune complex - mediated hypersensitivity.** Exogenous and endogenous antigens. Clinical and morphological features.

**10. Immunodeficiency syndromes.** Conception, etiology, classification, diagnostic methods. Primary immunodeficiencies. Definition, classification, diagnostic methods. Secondary immunodeficiencies. Definition, classification, . Definition, classification, diagnostic methods. AIDS.

**11. Amyloidosis.** Amyloid: structure, physical and chemical nature, diagnostic methods. Classification. Systemic and localized amyloidosis. Amyloidosis of aging.

**PRACTICE**

**MICROPREPARATIONS.** To study, to draw and to label morphological features.

**1. Antigen stimulation of spleen and lymph nodes.**

Hematoxylin and eosin stain. a) germinal centers; b) macrophage reaction; c) reticular cells and lymphocytes hyperplasia.

**2. Acute thymic involution.** Hematoxylin and eosin stain. a) marked lymphocyte depletion; b) Hassall’s corpuscles; c) fibrohyaline changes of basement membranes of vessels and thymic epithelium.

**3. Hyperplasia of Peyer's patches due to Typhoid fever of the colon.** Hematoxylin and eosin stain. a) lymphocyte, reticular cells and plasma cells hyperplasia; b) mucosal necrosis; c) Macrophage aggregates are called typhoid nodules.

**4. Rheumatic heart disease.** Hematoxylin and eosin stain. a) Aschoff nodules; b) central necrosis.

**5. Spleen amyloidosis.** Congo red stain. a) amyloid deposits in blood vessel walls, interstitium; in red pulp there are brown and red masses of it.

**6. Renal amyloidosis.** Congo red stain. Amyloid is displayed in a) extracellular deposition in mesangium; b) interstitium and around tubules c) in subendothelium that obliterate glomeruli; d) blood vessel walls.

**MACROPREPARATIONS:**

**1. Spleen hyperplasia.** Spleen is enlarged, loose.

*Causes*: any antigen stimulation (infectious disease, autoimmune disease).

*Outcomes*: according to the disease.

**2. Hyperplasia of Peyer's patches due to Typhoid fever.**

The solitary lymphoid follicles of the cecum are enlarged, elevated and have a central hemorrhagic dimple.

*Causes*: Salmonella typhi.

*Outcomes*: perforation or toxic megacolon, hemorrhage.

**3. Thymic hyperplasia.** Thymus is enlarged.

*Causes*: status thymico-lymphaticus.

*Outcomes*: favorable – recovery; unfavorable - sudden infant death syndrome.

**4. Spleen amyloidosis.**  Spleen is enlarged, firm and of waxy consistency, capsule is tense.

*Causes*: chronic infections, myeloma, hemodialysis etc.

*Outcomes*: unfavorable.

**5. Kidney amyloidosis.** Kidney are enlarged, firm and of waxy consistency. Cortex is wide and pale. Medulla is pink.

*Causes*: chronic infections, myeloma, hemodialysis etc.

*Outcomes*: unfavorable – acute or chronic renal failure.

**TESTS**

**1. Amyloidoblasts are:**

a) normal cells

b) trasformated parenchymatous cells

c) trasformated mesenchymal cells

d) the result of involution of hyaline producing cells

**2. Population of T-lymphocytes is characterized by:**

a) synthesis of immunoglobulins

b) part of type I hypersensitivity

c) part of types II-IV hypersensitivity

**3. Cells with affinity to HIV:**

a) CD8+ T-cells

b) CD4+ T-cells

c) NK – cells

d) dendritic cells

**4. Reactive changes in lymph nodes are characterized by the features exept:**

a) sinuses macrophage activity

b) inflammation reaction

c) paracortex reaction

d) follicular hyperplasia

**5. B-zones of lymphatic node are:**

a) cortex

b) sinuses

c) paracortex

d) germinal centers

e) medulla

**6. General lymphoid organs are:**

a) spleen

b) thymus

c) lymph nodes

d) tonsils

e) red bone marrow

**7. Non-specific immunity factors are:**

a) complement system

b) lymphocytes

c) macrophages

**8. Choose the mechanisms of immediate hypersensitivity reactions:**

a) Ig E - mediated

b) antibody - mediated cytotoxic reactions

c) immune complex - mediated cytotoxic reactions

d) T cell-killers mediated cytotoxicity

e) granulomatosis

**9. The autoimmune diseases are:**

a) tuberculosis

b) Hashimoto’s thyroiditis

c) stomach ulcer

d) systemic lupus erythematosus

**10. for IV hypersensitivity are typical all except:**

a) 12-24 hours development

b) predominance of lymphocytes in reaction zone

c) predominance of granulocytes in reaction zone

**11. B – cells population of lymphocytes take part in:**

a) Ig synthesis

b) I-III type hypersensitivity reactions

c) IV type hypersensitivity reactions

**12. Germinal center consists of the following cells exept of:**

a) prolymphocytes

b) macrophages

c) plasma cells

d) reticular cells

e) lymphoblasts

**13. T-zone of lymph node is:**

a) cortex

b) paracortex

c) medulla

d) germinal center

**14. The specific immunity cells are:**

a) neutrophils

b) lymphocytes

c) macrophages

d) basophils

e) reticular cells

**15. Amyloidosis is:**

a) parenchymatous dysproteinosis

b) mesenchymal glycogenosis

c) mesenchymal lipidosis

d) mesenchymal dysproteinosis

**16. The result of humoral mediated response is:**

a) proliferation of B-cells

b) suppression of B-cells

c) proliferation of T-cells

**17. The morphological signs of II-IV hypersensitivity reactions are:**

a) alterative inflammation

b) granulomatosis

c) vascular reaction and exudation

**18. Amyloidoisis is based on:**

a) damage of vascular wall

b) damage of connective tissue

c) anomaly protein synthesis

d) anomaly lipoprotein synthesis

**19. Secondary immunodeficiencies are:**

a) Bruton's agammaglobulinemia

b) AIDS

c) bone marrow myeloma

d) angioneurotic edema

**20. The causes of amyloidosis are:**

a) tuberculosis

b) chronic osteomyelitis

c) typhoid fever

d) influenza

e) rheumatoid arthritis

f) bronchiectasis

**Answers to the tests**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| * C | * a, d, e | * b, d | * b | * b |
| * c | * b, e | * c | * b, d | * c |
| * b | * a, c | * a, b | * d | * b, c |
| * b | * a, b, c | * a | * a | * a, b, e, f |

**theme 7: Tumors. General problems of Oncology. Tumors of epithelial origin. Precancerous processes. Mesenchymal tumor. Tumors of the melanin synthesizing tissue. Tumours of the nervous system and the meninges .**

**Lesson Content**

**Prepare answers to these questions:**

1. Definition of the term "tumor". Epidemiology of cancer. Morphological oncology as one of the leading branches of science studying the tumor. Value biopsy in oncology. Nomenclature and classification principles of tumors (clinical and morphological, by levels of histological differentiation, by TNM system.

2. Benign and malignant tumors: species, comparative characteristics. Histogenesis (cytogenesis) and tumor differentiation. Biological and clinical and morphological characteristics of the tumor distinguishing it from normal tissue. The structure of the tumor in relation to severity of parenchyma and stroma in it, of the source, the of degree of differentiation. Structural and functional features of tumor cells and stroma. Types of anaplasia. Morphological anaplasia (tissue and cell). Types of tumor growth: an expansive, infiltrative and apposition; exophytic and endophytic.

3. The most important clinical and pathological manifestations of tumor growth. Characteristics of the tumor process. Local and general effect of tumors in the body. Local tumor effect. Violation of homeostasis. Secondary changes in the tumor. Metastases and systemic non-metastatic effects. Cancer cachexia, paraneoplastic syndromes.

4. Etiology of tumors. Risk factors for the development of tumors. The main causes of tumors in humans. The role of environmental and genetic factors in the development of tumors. Carcinogenic agents and their interaction with cells. Chemical carcinogenesis. The most important group of chemical carcinogens. Radiation carcinogenesis. Viral carcinogenesis. Steps mechanisms. Premalignant (precancerous) status and change, their essence, morphology.

5. Benign tumors of the epithelium:

a) papilloma - the main sources of development and localization of the tumor. Structural features of the clinical course. Prognosis.

b) adenoma - sources of development, localization. Types of adenomas depending on the structure. Clinical features of adenomas of endocrine organs. Adenomatous polyps, their localization, prognosis.

6. Malignant tumors of epithelial (cancer). Definition. The main sources of development. Histological cancer variants, depending on the degree of tumor differentiation. Clinico-anatomical features of growth, metastasis, effects on the organism.

7 Mesenchymal tumors. Sources of their origin, classification and nomenclature according to their histogenesis and differentiation. Features of mesenchymal tumors.

8. Benign mesenchymal tumors: fibroma, lipoma, uterine, angioma, chondroma, osteoma, and others. The main places of their localization, growth characteristics, structure, clinical manifestations.

9. Malignant tumors (sarcomas): structure, clinical and morphological manifestations, peculiarities of various histological types, the role of the previous benign tumors.

10. Tumors of Melanocytes. The source of development and localization:

a) pigmented nevi. Morphology of different types of nevi. The clinical significance. Pigmented lesions with a high probability of malignancy,

b) melanoma, clinical and morphological classification and characteristics of the main options, prognosis.

11.Tumors of the nervous system and meninges.

**The practical part**

**MACROPREPARATIONS. To learn, to sketch and to identify morphological characteristics:**

**1. Skin papillomas**: Hematoxylin and eosin stain. a) hyperplastic proliferation of stratified squamous epithelium with an increase in the number of layers, but with the preservation of polarity and complexity of the arrangement of cells, b) basal membrane is preserved c) papillary proliferation of uneven nature of the surface epithelium and stroma, hyperkeratosis, d) excessive formation of small blood vessels.

**2. Fibroadenoma of breast**: Hematoxylin and eosin stain. a) glandular structures of various shapes and sizes are randomly located, b) the proliferation of alveoli and intralobular ducts, c) expansion of intralobular connective tissue.

**3. Squamous cell carcinoma of the lower lip with keratinization**: Hematoxylin and eosin stain. a) atypical squamous cells with polymorphism, hyperchromia nuclei, single mitosis, and b) the formation of cancer "pearls", c) cords and nests of atypical cells infiltrating the underlying tissue.

**4. Gastric Adenocarcinoma**. Hematoxylin and eosin stain. a) tumor tissue shows glands of various shapes and sizes with multilane atypical epithelium, b) polymorphic cells with nuclei hyperchromia, the presence of mitosis c) infiltration of the entire thickness of the gastric wall by all tumor tissue.

**5. Fibroma**: Hematoxylin and eosin stain. a) chaotic arrangement of connective tissue fibers, b) thickened fiber) fibroblasts.

**6. Angioma** (cavernous hemangioma): Hematoxylin and eosin stain. a) thin-walled vascular cavities such as the sinusoids of various size are enlarged, b) the cavities are lined by flattened endothelial cells and separated by connective tissue layers of different thickness.

**7. Leiomyoma**: Hematoxylin and eosin stain. a) a longitudinally and transversely spaced beams spindle-shaped smooth muscle cells, b) stroma consists of the layers of the connective tissue, c) blood and lymph vessels.

**8. Polymorphocellular fibrosarcoma:** Hematoxylin and eosin stain, a) tumor tissue differs by a pronounced polymorphism cells, b) nuclei hyperchromatosis, abnormal mitosis, c) the predominance of cells over fibers.

**Macropreparations**

1. **Skin papillomas**. On the surface of the skin there is spherical tumor formation size of 1 cm.: on a narrow stalk, of soft consistency, with papillary surface species. On the cut the tumor is well vascularized .

Localization: skin, mucous membranes, lined with transitional or squamous and flat epithelium, oral mucosa, the true vocal cords, pelvis of the kidneys, ureters, urinary bladder. Exodus: favorable; but papilloma of the bladder and the vocal cords can recur and become malignant.

**2. Gastric carcinoma**. (Polypoid cancer) a mucosal surface has a growing tumor formation in the lumen of the stomach as a villous node of 3 cm in diameter on the crus. Tumor tissue is of gray-pink color, the cut is rich in blood vessels; grows into the thickness of the wall of the stomach.

Causes: food (smoked food, pickled vegetables, pepper), biliary reflux (after operations on the stomach, especially by Billroth II), Helicobacter pylori (promotes mucosal atrophy, intestinal metaplasia, dysplasia). Precancerous lesions: adenoma, peptic ulcer, chronic atrophic gastritis.

Metastatic spreading: 1. Orthogradic nodal metastases into regional nodes on the small and large curvature,

2. Retrograde nodal metastases into the left supraclavicular lymph node – Virchov’s metastasis;

into ovary – Krukenberg’s metastases;

into pararectal fat – Shnitzler’s metastases.

3. Hematogenous metastases into the liver, lungs, brain, bones, kidneys, adrenal glands and less frequently in the pancreas.

  4. Implantational metastases - carcinomatosis of the pleura, pericardium, diaphragm, peritoneum, omentum.

**3. Fibroma of the skin**: skin flap in the slide, on the surface there is a round tumor formation on a broad basis, of 6 cm in diameter. On the cut whitish, of fibrous structure and tightly-elastic consistency. Localization: skin, uterus, mammary gland, spinal canal, eye socket and other organs. Exodus: favorable; complications are defined by localization.

**4. Lipoma**: tumor-node with a pronounced thin fibrous capsule of irregularly rounded shapes, sizes 5 by 7 cm. The section shows a non-uniform fat slices of yellow color, soft consistency, of various sizes, separated by thin layers of connective tissue. Localization wherever there adipose tissue. Exodus: favorable

**4. Liver angioma (cavernous hemangioma)** in the preparation is the liver; the pathological site with sharp edges, of irregular shapes, sizes 2 to 3 cm., of red-bluish color, of sponge sight is determined in the thickness of parenchyma. On the section it is presented as a clotted blood. Types: capillary, venous, cavernous, benign hemangiopericytoma. Exodus: favorable.

**5. Fibrosarcoma**: on the lower limb between the hip muscle arrays there is a pathological formation of size-15 by 10 cm, of nodular sight, on the cut is a fabric of gray-pink like a "fish flesh." The boundaries of the tumor node are not clear, with invasion into the surrounding tissue, with foci of necrosis and hemorrhages. Exodus: it frequently recurs, but rarely metastasizes.

**Tests**

**select one or more correct answers**

**1. Select signs of anaplasia:**

a) polymorphism

b) reduction of nuclear-cytoplasmic index

c) hyperchroma

d) abnormal mitosis

e) increase in nuclear-cytoplasmic index

f) reduction in the number of mitoses

**2. Signs of infiltrative tumor growth:**

a) germination of tumor cells in the vessel wall of the capsule and

b) the penetration of tumor cells into the surrounding tissue and destruction

c) encapsulating the tumor

d) lymphohistiocytic infiltration

**3. Adenoma is:**

a) a benign tumor of squamous epithelium

b) a malignant tumor of squamous epithelium

c) a benign tumor of glandular epithelium

d) teratomatous tumor

e) a malignant tumor of glandular epithelium

**4. Select the characteristic features of the tumor process:**

a) have a compensatory-adaptive character

b) ends with the maturation of cells

c) acquires new properties

d) unlimited growth

e) autonomous growth

**5. Signs of malignant tumors:**

a) infiltrative growth

b) the ability to metastasize

c) expansive growth

d) tissue and cell anaplasia

e) causes cachexia

**6. The source of the development of epithelial tumors are:**

a) vessels

b) skin epithelium

c) endothelium

d) the bladder epithelium

e) mucosal epithelium

f) endocrine organs

**7. Scirrhus - is:**

a) a benign tumor of epithelial

b) sarcoma

c) a malignant tumor of epithelial

d) a benign tumor of mesenchymal

**8. Adenocarcinoma - is:**

a) a benign tumor from epithelium

b) sarcoma

c) a malignant tumor from glandular epithelium

d) a benign tumor from mesenchyma

**9. Epithelial tumors are:**

a) papilloma

b) sarcoma

c) cancer

d) all answers are correct

e) all of the answers are wrong

**10. Papilloma this is:**

a) a benign tumor from the squamous epithelium.

b) a malignant tumor from squamous epithelium.

c) a benign tumor from glandular epithelium.

d) terathoid tumor.

e) a malignant tumor from glandular epithelium.

**11. Malignant tumors from the epithelium are:**

a) adenoma.

b) melanoma.

c) teratoma.

d) scirrhus.

e) angiosarcoma.

f) chondrosarcoma.

g) mucosal cancer.

**12. The term "dysplasia" means:**

a) violation of regeneration

b) increase of morphological atypism

c) poor circulation

d) all answers are correct

e) all of the answers are wrong

**13. Malignant epithelial gastric tumor is:**

a) adenocarcinoma

c) mucosal cancer

d) all of the above mentioned is true

**14. Malignant epithelial tumors of the lung:**

a) adenocarcinoma

b) lymphoma

c) fibrosarcoma

d) leukemia

**15. the External carcinogen having a decisive significance in lung cancer’s origin:**

a) chlorinated water

b) nicotine

c) carotenes

d) cholesterol

e) nitrates

**16. The most common primary malignant tumors of the esophagus:**

a) adenocarcinoma

b) squamous cell carcinoma

c) non differentiated carcinoma

d) melanoma

e) leiomyosarcoma

**17. Benign tumors occuring in the liver:**

a) hepatocellular adenoma

b) angioma

c) hepatocellular carcinoma

d) bile duct cystadenoma

e) all of the above mentioned is true

**18. Malignant tumor of kidneys in children:**

a) Wilms' tumor

b) medulloblastoma

c) hypernephroid cancer of granular cell variant

d) Ewing's sarcoma

e) the correct variants are a) and d)

**19. Predominant way of cancer metastasis:**

a) hematogenous

b) lymphogenous

c) implantational

**20. Predominant way of sarcoma metastazing:**

a) hematogenous

b) lymphogenous

c) implantational

d) perineural

**21. Select benign tumors developing from connective tissue**

a) fibrosarcoma

b) fibroma

c) fibrous histiocytoma

d) glomus tumor

**22. Ewing's sarcoma is attributable to:**

a) benign tumors of connective tissue

b) benign tumors of bone

c) malignant tumors of connective tissue

d) malignant tumors of bone

**23. Malignant tumors of blood vessels are:**

a) hemangioma

b) lymphangioma

c) cavernous angioma

d) hemangiosarcoma

e) Kaposhi's sarcoma.

**24. Malignant tumors of melanocytes are:**

a) melanoma

b) freckles

c) pigmented nevus

d) lentigo

**25. Tumors of brain coverings:**

a) oligodendroglioma

b) meningioma

c) neurofibroma

d) neuroblastoma

**Answers to tests**

1.acde 5.abde 9.ac 13.d 17.abd 21.bc 25.b

2.ab 6.bdef 10.a 14.a 18.ca 22.d

3.c 7.c 11.dg 15.b 19.b 23.de

4.cde 8.c 12.b 16.b 20.a 24.a

**Theme 7: Final lesson on lecture and theoretical material on the module "General pathological anatomy".**

Form(s) of the current control of academic performance(testing, interview, diagnosis of macropreparation and micropreparation).

Evaluation materials of the current control of academic performance

Test tasks

Test tasks for the current classes.

Oral response

Consolidation of theoretical material (Oral analysis of the topics and a survey of students in the 1.Module. "General pathological anatomy»

1. Pathological anatomy. Content, tasks, objects, methods, and levels of research. Historical data: stages of pathology development, the role of the Russian school of pathological anatomy. The relationship of pathological anatomy with fundamental and clinical disciplines. The pathoanatomical service and its significance in the health care system.

2. Research methods in pathological anatomy. Autopsy. The importance of studying cadaveric material, substrates obtained from patients during their lifetime, and experimental material. Histological examination. Cytological examination, immunohistochemistry, electron microscopy. Biopsy-types, meaning in the clinic.

3. Morphology of cell damage and death. Reversible and irreversible damage. Necrosis. Causes, mechanism of development, morphological characteristics. Clinical and morphological forms of necrosis, pathogenesis and morphogenesis, clinical and morphological characteristics, diagnostic methods, outcomes.

4. Apoptosis as programmed cell death. Definition, mechanisms of development, morphological characteristics and diagnostic methods. Stages of apoptosis. The significance of apoptosis in physiological and pathological processes.

5. Signs of death and postmortem changes. Death. Definition. Sudden death. The concept of intrauterine, clinical, biological death. Signs of biological death.

6. Dystrophy as a type of tissue damage. Functional and morphological nature of dystrophy. Etiological factors, the main pathogenetic links of dystrophy, morphogenesis. Principles of classification of dystrophy.

7. Intracellular accumulation: definition, mechanisms of development. Accumulation of lipids( lipidosis): causes, pathogenesis and morphogenesis, clinical and morphological characteristics, diagnostic methods, outcomes. Steatosis. Fat changes in the myocardium, liver, and kidneys. Cholesterol and its esters. Acquired and congenital disorders of lipid metabolism, morphological characteristics.

8. Protein accumulation (dysproteinosis): causes, pathogenesis and morphogenesis, morphological characteristics and diagnostic methods, clinical symptoms and syndromes, outcomes.

9. Glycogen accumulation: causes, pathogenesis and morphogenesis, morphological characteristics and diagnostic methods, clinical manifestations, outcomes. Acquired and innate glycogen storage.

10. Stromal-vascular dystrophy. Definition, basic conditions and mechanisms of development, classification. Protein stromal-vascular dystrophy: mucoid swelling, fibrinoid swelling, definition, morphological manifestations, outcomes.

11. Fatty vascular-stromal dystrophy, definition, classification principles. Causes of obesity, morphological changes in organs, clinical significance, outcomes of local and general obesity.

12. Hyaline changes. Intracellular and extracellular hyaline: morphogenesis, morphological characteristics. Hyaline changes in various pathological conditions.

13. Disorders of the metabolism of pigments (chromoproteins). Exogenous pigments. Endogenous pigments: types, mechanism of formation, morphological characteristics and diagnostic methods.

14. Disorders of the exchange of hemoglobinogenic pigments. The main causes and morphofunctional changes in oraganism in violation of hemoglobin metabolism. Hemosiderosis (local, systemic), hemochromatosis.

Disorders of bilirubin metabolism, morphological characteristics. Jaundice. Classification, causes and mechanisms of jaundice development.

15. Disorders of lipofuscin and melanin metabolism: clinical and morphological characteristics.

16. Pathological calcification (calcinosis). Types of calcinosis: dystrophic, metastatic. Causes, pathogenesis and morphogenesis, morphological characteristics, diagnosis, clinical manifestations, outcomes.

17. Arterial hyperemia. Causes, types, morphology.

18. Venous congestion: general and local, acute and chronic. Local venous fullness, causes, morphological manifestations, outcomes.

19. Venous stasis in the small circulatory system: pato and morphogenesis, clinical and morphological characteristics, outcomes.

20. Venous stasis in the large circulatory system: pathogenesis and morphogenesis, clinical and morphological characteristics, outcomes. Venous fullness in the portal vein system (portal hypertension): pathogenesis and clinical and morphological manifestations.

21. Bleeding: external and internal, hemorrhage. Causes, types, clinical and morphological characteristics. Hemorrhagic diathesis. Disseminated intravascular coagulation syndrome.

22. Stasis. Mechanisms of development, causes, meaning.

23. Thrombosis. Definition, local and general factors of thrombosis. Blood clot, its types, morphological characteristics. Venous thrombosis. Arterial thrombosis. Thrombosis in the heart cavities. Significance and outcomes of thrombosis.

24. Embolism: definition, types, causes, morphological characteristics. Orthograde, retrograde, and paradoxical embolisms. Thromboembolism: causes of development, clinical significance. Pulmonary embolism, acute pulmonary heart. Thromboembolic syndrome: clinical and morphological characteristics.

25. Ischemia (anemia). Definition, classification, causes, mechanisms of development, morphological characteristics and diagnostic methods, clinical significance. The role of collateral circulation. Acute and chronic ischemia. Infarction: definition, causes, classification, morphological characteristics of different types of infarcts, complications, outcomes.

26. Shock. Definition, types, mechanisms of development, stages, morphological characteristics, clinical manifestations.

27. Inflammation: definition, essence, and biological significance. The problem of local and general is in understanding inflammation. History of the study of inflammation (Celsus. R. Virchow, D. F. Kongheim, P. Ehrlich, I. I. Mechnikov). Clinical signs and symptoms of inflammation (local and systemic). General manifestations of inflammation, pathogenesis, etiological features.

28. Acute inflammation. Etiology and pathogenesis. The reaction of blood vessels in acute inflammation. Transudate, exudate, edema, stasis. Emigration of white blood cells, formation of pus. Outcomes of acute inflammation.

29. Morphological manifestations of acute inflammation. Exudative inflammation: serous, fibrinous, purulent, catarrhal, hemorrhagic, mixed. Ulcerative-necrotic reactions in inflammation. Morphological characteristics, clinical significance.

30. Chronic inflammation. Causes, pathogenesis, cellular cooperation (macrophages, lymphocytes, plasma cells, eosinophils, fibroblasts, etc.).

31. Morphological features (nature of infiltration, persistent destruction of connective tissue), outcomes.

32 Granulomatous inflammation (acute and chronic). Etiology, developmental mechanisms, clinical and morphological characteristics and diagnostic methods, outcomes.

33. Cellular kinetics of granuloma. Pathogenetic types of granulomas. Granulomatous diseases.

34. The immune system: structure and functions. Humoral and cellular mechanisms of the immune response. Non-specific immune response. Mechanical protection. A specific immune response. Specificity. Memory. Autoimmune response. Immunological tolerance.

35. Pathological conditions of the immune system. Classification. Hypersensitivity reactions Mechanisms of development, morphological characteristics, clinical significance. Graft rejection. Cellular and antibody mechanisms of development, morphogenesis, morphological characteristics, clinical significance.

36. Autoimmunization and autoimmune diseases. Definition, mechanisms of development, clinical significance (role in the development of rheumatism, systemic lupus erythematosus, rheumatoid arthritis). Infectious agents in autoimmunity.

37. Immune deficiency syndromes. Immune deficiency: concept, etiology, classification. Primary immunodeficiency: definition, classification, diagnostic methods. Clinical and morphological characteristics of primary immunodeficiency. Causes of death. Secondary (acquired) immunodeficiency: definition, etiology, classification.

38. Acquired immunodeficiency syndrome (AIDS). Epidemiology, transmission routes, etiology. Biology of the human immunodeficiency virus. Patho - and morphogenesis. Clinical and morphological characteristics. AIDS-associated diseases: opportunistic infections, tumors. Complications. Causes of death.

39. Amyloidosis: structure, physical and chemical properties, methods of diagnosis of amyloidosis, theories of etiology and pathogenesis, principles of classification. Macro - and microscopic characteristics of organs in amyloidosis.

40. Adaptation processes. Physiological and pathological adaptation. Phase character of the course of the adaptation process. Types of adaptive changes.

41. Hyperplasia: definition, causes, mechanisms, types, stages, clinical and morphological characteristics. Physiological and pathological hyperplasia.

42. Hypertrophy: definition, causes, mechanisms, types, clinical and morphological characteristics. Morpho-functional features of myocardial hypertrophy.

43. Atrophy: definition, causes, mechanisms, types, clinical and morphological characteristics. Brown atrophy of the liver, myocardium, and skeletal muscles.

44. Regeneration: definition, essence and biological significance, association with inflammation, outcomes. Components of the healing process.

45. Metaplasia: definition, types. Metaplasia in epithelial and mesenchymal tissues: morphological characteristics, clinical significance, role in carcinogenesis.

46. Granulation tissue, angiogenesis: stages, morphological characteristics. Kinetics of wound healing. Scar morphogenesis, reconstruction (remodeling) of the extracellular matrix during scarring. The role of humoral and cellular factors in the repair process.

47. Definition of the term "tumor". Epidemiology of oncological diseases. Morphological oncology as one of the leading branches of science studying tumors. The importance of biopsy in oncology. Nomenclature and principles of classification of tumors (clinical and morphological, according to the level of histological differentiation, according to the TNM system).

48. Etiology of tumors. Risk factors for the development of tumors. The role of environmental factors (carcinogenic substances, physical, infectious) and genetic factors in the development of tumors. The main causes of tumors in humans.

49. The structure of the tumor. The structure of the tumor depends on the severity of the stroma and parenchyma in it, the source of development, and the degree of its differentiation. Structural and functional features of the tumor cell and stroma. Types of atypism. Morphological atypism (tissue and cellular).

50. Biological and clinical-morphological signs of a tumor that distinguish it from normal tissues. Concepts of cataplasia. Types of tumor growth. Conditions for the development of relapses, metastases. Local and general effects of tumors on the body.

51. Features of the concepts of "benign" and "malignant" tumor. Their clinical and morphological characteristics.

52. Morphogenesis of tumors. Clinical and morphological substantiation of the stages of tumor development: background diseases, facultative and obligate precancerous tissue changes. The origin of the tumor germ, the concept of the "tumor field" and tumor progression.

53. Features of the structure of epithelial tumors. Sources of their occurrence. Benign tumors from the epithelium:

a) papilloma – the main sources of tumor development and localization. Features of the structure, clinical course. Forecast.

b) adenoma-sources of development, localization. Types of adenomas depending on the structure. Features of the clinical course of endocrine adenomas. Adenomatous polyps their localization, prognosis.

54. Precancerous processes. Definition of the concept. The main morphological changes in the epithelium during the formation of cancer. The concept of epithelial dysplasia, intraepithelial cancer. Clinical and morphological substantiation of precancerous processes: background conditions, facultative and obligate precancerous changes. Examples.

55. Malignant tumors from the epithelium (cancer). Definition. The main sources of development. Histological variants of cancer depending on the degree of differentiation of the tumor. Clinical and anatomical features of growth, metastasis, and effects on the body.

56. Mesenchymal tumors. Sources of their occurrence, classification and nomenclature depending on histogenesis and their differentiation. Features of mesenchymal tumors.

57. Benign mesenchymal tumors: fibroma, lipoma, myoma, angioma, chondroma, osteoma. The main places of their localization, features of growth, structure, and clinical manifestation.

58. Malignant mesenchymal tumors (sarcomas): structure, clinical and morphological manifestations, features of various histological variants, the role of previous benign tumors.

59. Connective tissue tumors with locally destructive growth-fibromatoses. Types, localization, and their clinical significance.

60. Tumors of melanin-forming tissue. Source of development and localization:

a) pigmented skin formations. Morphology of various types of nevi. Clinical significance. Pigmented formations with a high probability of malignancy,

b) melanoma, clinical and morphological classification and characteristics of the main variants, prognosis.

61. Tumors of the nervous system and brain membranes:

a) neuroectodermal and meningovascular tumors of the central nervous system, sources of development, clinical and morphological characteristics.

b) tumors of the autonomic and peripheral nervous system, macro-microscopic characteristics.

MICROPREPARATIONS

1. Coagulation necrosis of the muscles.

2. Curd necrosis of the lymph node in tuberculosis.

3. Anemic kidney infarction.

4. Anemic spleen infarction.

5. Hemorrhagic lung infarction.

6. Fatty myocardial dystrophy.

7. Fatty liver dystrophy.

8. Myocardial obesity.

9. Hyalinosis of the spleen vessels.

10. Liver with mechanical jaundice.

11. Pigmented nevus.

12. Brown liver atrophy

13. Brown myocardial atrophy

14. Brown induration of the lungs.

15. Acute venous fullness (edema) of the lungs.

16. Hemorrhage in the brain.

17. Brown lung induration.

19. Chronic venous fullness of the liver ("muscat liver").

20. Ischemic kidney infarction.

21. Myocardial infarction.

22. Hemorrhagic lung infarction.

23. Obturating blood clot with organization and sewerage.

24. Metastatic abscesses in the lung.

25.Metastatic ulcers in the kidneys (embolic purulent nephritis).

26. Serous pneumonia.

27. Croup pneumonia.

28 Diphtheria colitis.

29. Purulent nephritis.

30 Miliary tuberculosis of the lungs.

31 Actinomycosis of the liver.

32 Single-chamber echinococcus cyst wall.

33.Muscle trichinosis.

34. Syphilitic encephalitis.

35. Rhinoscleroma.

36. Granulation chamber

37. Myocardial hypertrophy

38. Hydronephrosis

39. Brown liver atrophy

40. Brown myocardial atrophy

41 Emphysema of the lungs

42 Glandular endometrial hyperplasia

43 Glandular-muscular hyperplasia of the prostate gland

44 Spleen, lymph nodes with antigenic irritation:

45 Accidental involution of the thymus gland.

46 Peyer's plaques in typhoid fever.

47 Rheumatic myocarditis.

48 Amyloidosis of the spleen.

49 Amyloid nephrosis.

50 Skin papilloma.

51. Papilloma of the bladder.

52. Fibroadenoma of the breast.

53 Squamous cell carcinoma with keratinization of the lower lip.

54 Squamous cell carcinoma of the cervix without keratinization.

55. Gastric adenocarcinoma.

56. Fibroma:

57. Lipoma.

58. Angioma.

59. Chondroma.

60. Osteoma.

61. Leiomyoma:

62. Malignant fibrous histiocytoma.

63. Polymorphocellular fibrosarcoma.

64. Fusiform cell sarcoma.

MACROPREPARATIONS.

1. Anemic kidney infarction.

2. Hemorrhagic lung infarction.

3. Curd necrosis of the lymph nodes in tuberculosis.

4. Gangrene of the toes (dry).

5. Fatty myocardial dystrophy ("tiger heart»)

6. Fatty liver dystrophy.

7. Myocardial obesity.

8. Hyalinosis of the spleen capsule.

9. Skin with Addison's disease

10. Birthmark (pigmented nevus).

11. Silicosis of the lungs.

12. Gallstones:

13. Kidney stones:

14. Hydronephrosis:

15. Acute fullness of the membranes of the brain in influenza.

16 Nutmeg liver.

17 Brown lung induration.

18. Hemopericardium with cardiac tamponade.

19. Brain hematoma.

20 Hemorrhage in the ventricles of the brain.

21 Myocardial infarction (white with a hemorrhagic corolla).

22 Ischemic brain infarction.

23 Ischemic spleen infarction.

24 Pulmonary embolism.

25 Croup pneumonia (stage of gray hepatica).

26 Fibrinous pericarditis

27. Diphtheria colitis:

28. Purulent nephritis.

29. Liver abscess.

30. Milliard tuberculosis of the lungs.

31. Gumma of the brain.

32. Syphilitic mesoaortitis and aortic aneurysm.

33. Single-chamber echinococcus of various organs

34. Cysticercosis of the brain.

35. Hypertrophy of the heart

36. Splenomegaly

37. Hypertrophy of the bladder wall

38. Brown heart atrophy

39. Hydronephrosis

40. Brain atrophy in hydrocephalus

41. Spleen hyperplasia.

42. Peyer's plaques and solitary follicles in typhoid fever.

43. Hyperplasia of the thymus gland.

44. Amyloidosis of the spleen (sebaceous spleen).

45. Amyloidosis of the kidneys.

56. Skin papilloma:

57. Papillomatosis of the bladder mucosa:

58. Skin cancer:

59. Adenomatous polyps of the intestine

60. Various forms of stomach cancer.

a) Polypous cancer

b) Infiltrative-ulcerative stomach cancer

61. Breast fibroadenoma

62. Cancer of the vaginal portion of the cervix

63. Skin fibroma

64. Lipoma

65. Uterine fibromyomatosis

66. Liver angioma

67. Chondroma

68. Fibrosarcoma

69. Liposarcoma

70. Malignant fibrous histiocytoma

71. Malignant mesothelioma

**Module 2. "Private pathological anatomy".**

**theme 1: Anemia. Hemoblastoses.**

Form(s) of the current control of academic performance(testing, interview, diagnosis of macropreparation and micro-preparation, solution of situational problems,abstract, report).

Assessment materials of the current control of academic performance

Test tasks

Select one or more correct answers

**1. Signs of acute lymphoblastic leukemia**

1) The peak incidence at age 60

2) develops mainly in children

3) the prevalence of lymphoblasts in the bone marrow and blood

4) struck lymph nodes

5) in the leukemic infiltrates predominate myeloblasts

**2. Clue cells in lymphoid tissues IN lymphogranulomatosis**

1) cells Anichkova

2) cells of Hodgkin

3) cells Mikulic

4) cells Reed-Sternberg

**3. Morphological changes lymph nodes in lymphogranulomatosis**

1) amyloid deposition in the stroma

2) numerous cells Reed-Sternberg

3) cells of Hodgkin

4) necrosis, sclerosis

5) hyperplasia bright centers of follicles

**4. Characteristic signs of chronic myeloid leukemia**

1) pyoid bone marrow

2) axillary resorption and osteoporosis

3) intralobular leukemic infiltration of the liver

4) infiltration of the portal stroma leukemia cells

5) aleukemic leukemia

**5. Bence-Jones protein was detected in the urine at**

1) chlamydia

2) Multiple Myeloma

3) chronic myeloid leukemia

4) chronic lymphocytic leukemia

**6. spleen weight increase greatly the in leukemia**

1) acute

2) chronic

**7. The features typical of Burkitt's lymphoma**

1) a high degree of malignancy

2) is more common in people of Europe

3) the picture "starry sky"

4) marked cellular polymorphism of tumor tissue

5) detection of tumor cells virus Epstein – Bar

**8. TYPE lymphogranulomatosis with the most unfavorable prognosis**

1) predominance of lymphoid tissue

2) mixed cell

3) nodular sclerosis

4) Lymphocyte depletion

5) lymphohistiocytic

**9. Do not look at treatment with cytostatics leukemic infiltrates STORED IN**

1) heart

2) Kidney

3) brain

4) spleen

**10. Anemia characterizes**

1) reduction in the number of red blood cells

2) an increase in hemoglobin in the blood

3) decrease in circulating blood volume

4) decrease in hemoglobin, and often in the number of erythrocytes per unit volume of blood

**11. BONE MARROW strikes PRIMARY**

1) malignant lymphoma

2) in leukemia

**12. Bone marrow involvement lymphoma**

1) secondary to metastatic

2) primary

**13. Causes of nutritional iron deficiency anemia**

1) Gastrectomy

2) Pregnancy

3) autoimmune gastritis

**14. The cause of iron deficiency anemia in pregnant and nursing mothers**

1) lack of receipt of exogenous iron

2) lack of synthesis of endogenous iron

**15. CAUSE hemolytic anemia**

1) lack factor Castle

2) incompatible blood transfusion

**16. In chronic leukemia is more characteristic**

1) The proliferation of undifferentiated (**blast**) cells

2) the proliferation of ripening (**cytic**) cells

**17. The forms of leukemia in children, with a favorable prognosis**

1) T-lymphoblastic

2) B-lymphoblastic

3) myeloblastic

4) undifferentiated

5) plazmoblastic

Questions for the oral survey

1. Anemia. Definition and classification. Acute and chronic anemia due to blood loss (posthemorrhagic): causes, clinical and morphological characteristics, diagnosis.

2. Anemia due to increased blood loss (hemolytic): hereditary, acquired, autoimmune, isoimmune, mixed genesis. Classification, pathogenesis, diagnosis, clinical and morphological characteristics, causes of death. Hypersplenism.

3.Anemia with insufficient reproduction of red blood cells (dyserythropoietic). Classification. Anemia megaloblastic (B12 - and folic-deficient), pernicious, iron-deficient, with iron metabolism disorders, hypoplastic and aplastic. Etiology, pathogenesis and morphogenesis, clinical and morphological characteristics and diagnostic methods, complications, causes of death. Diseases and conditions accompanied by anemia.

4. Tumors of hematopoietic tissues (leukemias). Classification, etiology of leukemias, chromosomal and antigenic rearrangements. General characteristics. Membrane cell antigens-markers of differentiation of tumor cells and cytogenetic variants of leukemias.

5. Acute leukemias: lymphoblastic and myeloblastic. Modern diagnostic methods, clinical and morphological characteristics, complications. Drug pathomorphosis, age characteristics, causes of death.

6. Chronic myelocytic leukemia:

a) chronic myeloid leukemia, diagnostic signs, stages, morphology.

b) true polycythemia, myelofibrosis, thrombocytemia: diagnostic methods, clinical and morphological characteristics, causes of death.

7. Chronic lymphocytic leukemias:

a) chronic lymphocytic leukemia, diagnostic methods, clinical and morphological characteristics. Modern methods of treatment: bone marrow transplantation.

b) tumors from plasma cells (paraproteinemic). General characteristics. Classification: monoclonal gammapathy of unknown nature, multiple myeloma, plasmocytoma, Waldenstrom's macroglobulinemia, Franklin's heavy chain disease. Modern diagnostic methods, etiology, pathogenesis, morphological characteristics, clinical manifestations, prognosis, causes of death.

8. Hodgkin's disease (lymphogranulomatosis): clinical stages, pathohistological types, morphological characteristics and diagnostic methods, clinical manifestations, prognosis, causes of death.

9. Non-Hodgkin's lymphomas. General characteristics, localization, forecast, typing, and classification. Immunohistochemical markers, cell types in non-Hodgkin's lymphomas. Tumors from T-and B-lymphocytes: types, morphological characteristics, immunophenotypic variants, cytogenetic and molecular genetic markers, clinical manifestations, prognosis, causes of death.

The practical part

micropreparations:

**1. The liver in chronic myeloid leukemia.** H & E stain.

The liver lobules in the course of a marked infiltration of the sinusoids leukemia cells of the myeloid series (a) and a small tumor infiltration of myelocytes portal tracts (b), in a state of hepatocytes steatosis (c), observed their lipofusinoz (g).

**2. The liver in chronic lymphocytic leukemia**. H & E stain.

In the course of the capsule and portal tracts massive clusters of tumor cells of lymphoid series (a) in hepatocytes marked protein and fatty degeneration (b) lipofuscinosis (c).

**3. Hodgkin Lymphoma lymph node (Mixed cellularity type.).** H & E stain.

In the lymph node tissue the expressed cellular polymorphism: visible large mononuclear cells Hodgkin (a) multinucleated Reed - Sternberg cells (b), the lymphocytes (B), plasma cells (g), eosinophils (d), neutrophils (e). Determined nodules undergoing necrosis and sclerosis (g).

**macropreparations:**

**1. The liver in chronic lymphocytic leukemia.**

The liver is enlarged in size, dense consistency, light brown color on the surface and cross-section are seen small gray-white nodules.

Causes hematogenous metastasis of leukemic cells from the bone marrow.

The outcome: adverse, liver failure.

**2. necrotic angina at acute leukemia**.

The tonsils are enlarged, on the surface and in the depths of visible areas of coagulation necrosis and ulceration of gray-black. Tissue around the tonsils is hydropic, hyperemic. In the mucosa of tongue and throat diapedetic it has many small and larger hemorrhages.

Reasons: leukemic infiltration of lymphoid tissue.

Complications: bleeding, infection joining.

**3. The lymph nodes in chronic lymphocytic leukemia**.

Lymph nodes were increased, merge into a huge plotnovata packages, the boundary between them is preserved in some places, but the capsule is soldered to the surrounding tissue. On a section of the fabric uniform, juicy, white and pink.

Reasons: leukemic infiltration of the lymph nodes, leading to a sharp disruption of their structure and the surrounding tissue.

Complications: compression of the adjacent organs.

Outcome: necrosis adhesions.

**4. The spleen in chronic myeloid leukemia**.

The spleen is greatly increased in size, weighing about 3 kg, dense, smooth capsule, speckled appearance. In the context of the parenchyma dark red color, with white foci of ischemic infarcts.

Reasons: diffuse leukemic infiltration, occlusion of blood vessels of the spleen tumor cells.

Complications: rupture of the capsule and parenchymal bleeding.

The outcome: poor: dysfunction of the spleen.

**5. The bone marrow in chronic myeloid leukemia**. Bone marrow epiphyseal and diaphyseal long bones replaced by lush gray-pink or greenish tissue grows into the medullary canal ("pyoid " bone marrow).

Reasons: replacement of normal bone marrow tumor tissue.

The outcome: the suppression of hematopoiesis, anemia, opportunistic infections.

**6. The spleen in the lymphogranulomatosis.**

Spleen increased in size, the cut red-brown organ parenchyma replaced by yellow-white tumor tissue, forming pockets of irregular shape, or growths with foci of necrosis and sclerosis ("porphyry" spleen).

The reasons: generalized cancer.

Outcome: dysfunction of the spleen.

**theme 2: diseases of the cardiovascular system**

The form(s) of the current control of academic performance(testing, oral survey, diagnosis of macropreparation and micro-preparations, solution of situational problems, abstract, report).

Assessment materials of the current control of academic performance

Test tasks

Test control

Select one or more correct answers.

1. When atherosclerosis affects

1) veins

2) capillaries

3) arterioles

4) large and medium arteries

2. successive stages of atherosclerosis

1) aterocalcinosis (4)

2) fibrous plaques (2)

3) complicated lesions (3)

4) fatty Streaks (1)

3. TYPES lipoprotein metabolism disorders in atherosclerosis

1) reduction in LDL - cholesterol

2) increase the level of LDL - cholesterol

3) increase in HDL - cholesterol

4) improving the abnormal lipoprotein

4. IMPROVING LDL in plasma leads to

1) the destruction of elastic fibers

2) increased permeability of the endothelium

3) damage and loss of endothelial cells

4) increasing adhesion of monocytes to Endothelial cells

5. foam cells come from

1) macrophages

2) lymphocytes

3) mast cells

4) plasma cells

5) adventitial cells

6) smooth muscle cells

6. Mönckeberg medial sclerosis strikes lining of arteries

1) Internal

2) Medium

3) outer

7. The risk factors for atherosclerosis

1) stress

2) obesity

3) hyperuricemia

4) male

5) fermentopathy

6) hypercalcemia

8. abdominal aortic aneurysm is typical for

  1) Syphilis

  2) Rheumatism

  3) atherosclerosis

4) arteriosclerosis

9. The form of clinical course of arterial hypertension

1) Secondary

2) idiopathic

3) malignant

4) benign

10. The causes and risk factors of development of secondary hypertension

1) pheochromocytoma

2) coarctation of the aorta

3) violation of the separation of sodium by the kidneys

4) genetic disorders of the renin-angiotensin system

11. Morphological changes of arterioles in hypertension

1) arteriolitis

2) caseous necrosis

3) fibrinoid necrosis

4) nodular periarteritis

5) hyaline arteriolosclerosis

6) hyperplastic arteriolosclerosis

12. arteriolosclerosis hyaline vascular lumen

1) is narrowed

2) expanded

3) is not changed

13. renal size with hypertension

1) Increase

2) conventional

3) Reduce

14. The possibility of changes in the kidney with renal artery atherosclerosis

1) infarcts

2) amyloidosis

3) embolic purulent nephritis

4) hydronephrosis transformation

15. CHANGES large vessels in essential hypertension

1) fat spots and stripes

2) hyalinosis walls

3) productive vasculitis

4) circular arteriosclerosis

5) aneurysm

16. The possibility of changes in the arterioles in essential hypertension

1) proliferation of endothelial

2) hyalinosis

3) giperelastoz

4) hypertrophy of muscle cells

5) fibrinoid necrosis

17. fatal complications in atherosclerosis mesenteric artery

1) intestinal obstruction

2) bowel gangrene

3) fibrinous colitis

4) ischemic colitis

18. SIGNS eccentric myocardial hypertrophy in hypertension

1) an increase in heart size in diameter

2) the expansion of adipose tissue

3) expansion of cavities

4) myocardial atrophy

19. Ischemic heart disease pathogenetic CONNECTION

1) with rheumatoid koronariitom

2) with mitral valve stenosis

3) with coronary atherosclerosis

4) with essential hypertension

20. forms of acute ischemic heart disease

1) cardiomyopathy

2) acute focal ischemic myocardium dystrophy

3) myocardial infarction

4) chronic cardiac aneurysm

5) sudden cardiac death

21. The most frequent localization of myocardial infarction

1) the right atrium

2) the left atrium

3) the right ventricle

4) the left ventricle

22. stage during myocardial infarction

1) necrotic

2) Mixed

3) ischemic

4) Organization

5) Compensatory

23. BASE cerebrovascular disease is

1) syphilis, cerebrovascular

2) atherosclerosis of cerebral arteries

3) essential hypertension

4) Hydrocephalus

5) rheumatoid vasculitis

24. ischemic cerebral infarction develops in

1) vessels in the brain rupture of microaneurysms

2) thrombosis of cerebral arteries

3) thrombosis of the carotid and vertebral arteries

25. Morphological manifestations of cerebrovascular Diseases

1) congenital aneurysm of the brain arteries

2) ischemic cerebral infarction

3) bleeding in the brain

4) Encephalitis

26. Types of Angina pectoris

1) labile

2) stable

3) disappear

4) spastic

5) Prinzmetala

6) unstable

27. macroscopic myocardial infarction revealed through

1) 1-2h

2) 4-6 h

3) 18-24h

4) 72 hours

28. microscopic features of myocardial infarction

1) plasma-coagulation

2) fatty degeneration

3) mucoid swelling

4) vacuolization of the cytoplasm

5) karyopyknosis, karyorrhexis

29. TYPES myocardial infarction localization

1) subendocardial

2) chordal

3) transmural

4) intramural

5) the valve

6) subepicardial

30. CHANGES cardiomyocytes of Angina pectoris

  1) atrophy

  2) kariolizis

  3) fatty degeneration

  4) the disappearance of glycogen

Questions for the oral survey.

1. Atherosclerosis and arteriosclerosis. Epidemiology, etiology, risk factors. Modern ideas about the pathogenesis of the disease.

2. Characteristics of macroscopic changes and morphogenetic stages of atherosclerosis, the structure of atherosclerotic plaque. The main clinical and morphological forms of atherosclerosis, their manifestations, complications and outcomes.Arteriosclerosis (mediacalcinosis) Menckeberg, morphological characteristics.

3. Hypertension (essential hypertension). Benign and malignant forms of arterial hypertension. The concept of a hypertensive crisis. Prevalence, etiology, and pathogenesis. Symptomatic hypertension.

4. Benign hypertension, stages, pathogenesis. Clinical and morphological forms: brain, cardiac, renal, hypertensive retinopathy. Morphological changes in blood vessels (hyaline and hyperplastic arteriolosclerosis) and in organs.

5. Malignant form of hypertension, clinical manifestations and morphological changes, complications, outcomes, causes of death.

6. Hypertensive heart disease. Myocardial hypertrophy. Chronic and acute pulmonary heart disease:causes of development, clinical and morphological characteristics.Congestive heart failure:etiology, pathogenesis, and morphogenesis. Clinical and morphological characteristics of left ventricular and right ventricular insufficiency.

7. Coronary heart disease (coronary heart disease). Concept, epidemiology, relationship with atherosclerosis and hypertension. Etiology and pathogenesis, risk factors, course, clinical and morphological forms.

8. Sudden coronary death. Causes of development. Pathogenesis, morphogenesis, and causes of death.

9. Angina pectoris: classification, clinical and morphological characteristics.

10. Myocardial infarction: definition of the disease, causes, classification, dynamics of biochemical and morphofunctional changes in the myocardium. Morphology of acute, recurrent, and recurrent myocardial infarction. Outcomes, complications, changes in thrombolytic therapy, causes of death.

11. Chronic ischemic heart disease: forms, clinical and morphological characteristics, complications, causes of death.

12. Cerebrovascular diseases. Etiology, association with atherosclerosis and hypertension. To define the concept of "stroke" and transient transient circulatory disorders of the brain. The most frequent localization of lesions of the cerebral vessels.

13. Clinical and morphological forms of CVB:

а)Brain diseases with ischemic injuries:hemorrhagic and ischemic brain infarcts,ischemic encephalopathy. Morphology, causes, and outcomes.

b) Intracranial hemorrhages. Types, causes, mechanism of development, complications and outcomes.

c) Pathological anatomy of hypertensive cerebrovascular diseases.

The practical part of the subject:

Micropreparations: In the study micropreparations pay attention to educational elements lettered in parentheses.

1. **Atherosclerotic plaque in the aorta**. H & E stain. The aortic intima visible sediments fat-protein mass and proliferation of connective tissue (a). In the center of the plaque observed foam cells, cholesterol crystals, necrotic detritus (b). The surface of the fibrous plaque has hyalinized tire (c) lined with endothelium, (d) In the interior of the plaque can be seen smooth muscle cells, macrophages, lymphocytes, (e), section atheromatosis with parietal thrombus (f). On the periphery of the newly formed vessels are marked (g).
2. **The myocardium in hypertensive disease**. H & E stain. In the myocardial hypertrophic cardiomyocytes increased with hyperchromatic nuclei (a) in the interstitial tissue is observed proliferation of connective tissue (b), the walls of the arterioles hyalinized (c).
3. **Arteriolosklerotic kidney**. H & E stain.  The walls of the arterioles of kidney significantly thickened due to accumulation of hyaline (a), the lumen of narrowed in some places obliterated (b). Most of the glomeruli sleeping, replaced by connective tissue or hyaline masses (c), the tubular epithelium atrophy, flattened (d). Surviving nephrons compensatory hypertrophied (t). Number of interstitial connective tissue is increased (f)
4. **Myocardial infarction**. H & E stain. In Myocardium observed necrosis area of muscle fibers (a), non-nuclear cardiomyocytes (b). On the periphery of the necrotic zone demarcation inflammation observed: advanced full-blooded, thin-walled vessels with a marginal standing leukocytes (c), perivascular hemorrhage (d), expressed infiltration of polymorphonuclear leukocytes (e).
5. **Postinfarction macrofocal cardiosclerosis**. H & E stain. The myocardium is observed large cicatricial field (a) represented by mature fibrous connective tissue with single fibrocytes, on the periphery of the focus of increasing muscle fiber with large hyperchromatic nuclei (b).

**Macropreparations.**

1. **Atherosclerosis of the aorta.**

In preparation of the abdominal aorta, sharply distorted by the presence of multiple saccular protrusions in the wall (aneurysm) in the cavity which are thrombotic overlay (dilatation thrombi). Uneven Intima with a lot of dense yellowish-whitish structures (plaques), protruding into the lumen. Some ulceration and deposition of calcium salts in the form of a gray-white solid mass.

The reasons: violation of combinations of fat and protein metabolism to damage the endothelium of the arteries.

Complications: thromboembolism in a large circle of blood circulation, with possible myocardial infarction and brain, kidneys and spleen, intestine and gangrene of the lower limbs; rupture of an aortic aneurysm.

Outcome: determined by the development of complications.

2**. cardiac hypertrophy in hypertensive disease**.

The heart increased in size considerably thickened wall of the left ventricle to 3.5 cm, increased trabecular volume and papillary muscle of the left ventricle. Heart weight of 800 grams. The cavity of the left ventricle expanded. In the context of the myocardium dim, clay species.

Causes: Chronic hemodynamic load on the heart.

Complications: tonogennaya dilation and concentric hypertrophy (step compensation) is replaced with the development of myogenic dilatation eccentric hypertrophy (decompensated).

The outcome: chronic heart failure.

3. **Primary-contracted kidney**.

The kidney is significantly reduced in size, pale, thick consistency, fine grain surface. In the context of a typical pattern renal erased border cortical and medullary layer is not defined in the parenchyma proliferation of connective tissue gray-white color.

Reasons: chronic circulatory failure resulting hyalinosis arterioles and circulatory sclerosis branches of the renal artery in hypertensive disease.

Complications: uremia.

The outcome: chronic renal failure.

4. **Atherosclerotic nephrosclerosis**. Kidney slightly reduced in size, its surface is large tuberous, due to the plurality of star-shaped scar retraction. The consistency is firm, drawing on the cut renal relatively preserved, visible wedge portions subcapsular parenchyma atrophy.

Reasons: chronic circulatory failure as a result of a partial obstruction of the lumen of the renal artery atherosclerotic plaque (arteriosclerosis segmental).

Complications: symptomatic renovascular hypertension.

The outcome: chronic renal failure.

5. **Myocardial infarction**. In the side wall of the left ventricle, in the apex and the anterior part of the interventricular septum of the heart, there is a pathological site irregular sink on the cut submitted drain pockets of gray-yellow in color (coagulation necrosis), around the area of hemorrhage and hyperemia (demarcation zone). In the lumen of the descending branch of the left coronary artery occlusive thrombus. Sclerotic coronary arteries with fibrous plaques. From the endocardial thrombotic visible overlay.

Causes: thrombosis, long spasm, thromboembolism, functional overstrain infarction in the presence of arteriosclerotic occlusion.

Complications in the early period- pulmonary edema, cardiogenic shock, arrhythmias and conduction, myocardial rupture (3-10 day when transmural infarction) or acute rupture of the aneurysm (4-14 days), thromboembolism

Complications of late period: chronic cardiac aneurysm, Dressler's syndrome (pericarditis, pleurisy, fever, blood eosinophilia).

Outcome: congestive heart failure, pulmonary edema, or brain macrofocal cardiosclerosis, chronic coronary artery disease.

1. **Hemopericardium with cardiac tamponade.**

In preparation of the heart with heart shirt in cross section. Pericardial accumulation of coagulated blood. On the back wall of the left ventricle there is a section of necrosis in violation of the integrity of the myocardium, measuring about 2cm.

The reasons: the gap acute or chronic heart aneurysm, rupture of the heart wall when transmural infarction (under miomalyatsii), rupture of the heart wall in obesity.

Outcome: unfavorable.

1. **Hematoma of the brain.**

The parietal-temporal region of the right hemisphere - the accumulation of coagulated blood brownish-red color. In the matter of the brain hemorrhage destroyed "red softening of the brain."

Reason: breaks microaneurysm arterioles and small arteries, or hyalinized vascular microcirculation, at least in the ulceration of an atherosclerotic plaque.

Complications: paralysis.

The outcome: poor in breaking the blood into the ventricles, rarely a cyst.

**8. Ischemic cerebral infarction**.

The left hemisphere of the brain, in the subcortical nuclei, seen hotbed of irregular shape, presented this mass of gray, the size of 1.5 cm × 3 cm, with clear boundaries - a "hotbed of gray softening." The surrounding brain tissue swelling with diapedetic hemorrhages.

Causes of thrombosis and atherosclerotic lesions of cerebral arteries precerebral rarely thromboembolism, long spasm.

Complications are determined localization nekroza- paralysis, paresis.

The outcome: a cyst.

**theme 3:** HEART DISEASE. Valvular Heart Disease. Myocardial disease. Diseases of the pericardium. Rheumatism. Congenital Heart Disease.

CONTROL QUESTIONS

1. Diseases of the valve holes of the heart and great arteries: classification, functional disorders. Congenital and acquired heart disease: clinical and morphological characteristics.
2. Endocarditis: classification, etiology, pathogenesis, morphological characteristics, complications, prognosis. Primary endocarditis (bacterial sepsis, endocarditis Leffler). Non-infectious nonbacterial thrombotic endocarditis. Endocarditis in rheumatic diseases (true rheumatism, systemic lupus erythematosus, rheumatoid arthritis). Carcinoid endocarditis.
3. Diseases of the myocardium. Classification. Myocarditis. Definition, etiology. Patho- and morphogenesis, clinical and morphological characteristics, consequences, causes of death:

a) primary myocarditis Abramov - Fidler,

b) viral, microbial and parasitic myocarditis, infectious-allergic myocarditis,

c) myocardial diseases caused by toxic, metabolic and other impacts,

d) heart disease in pregnancy and childbirth, amyloidosis, excess iron, hyper- and hypothyroidism.

4. Diseases of the pericardium. Pericarditis: classification, etiology, pathogenesis, clinical and morphological characteristics, outcomes. Hydropericardium, hemopericardium.

5. Cardiomyopathy: classification. Primary cardiomyopathy, the value of genetic factors, pathological and morphogenesis, clinical and morphological characteristics of different forms, causes of death. Secondary cardiomyopathy etiology, pathogenesis, morphological changes of heart complications.

6. Rheumatic diseases. Classification. General characteristics. Rheumatic fever: etiology, patho- and morphogenesis, the characteristic clinical and morphological forms, methods of diagnosis, clinical symptoms and syndromes forecast.

7. Congenital heart disease. Etiology. Vices "blue" and "white" types. Congenital defects of the atrial and ventricular walls, arterial trunks of the heart (transposition, stenoses and anomalies in the mouths of the great arteries, aortic coarctation, patent ductus arteriosus), combined heart defects (the triad, tetrad, pentad Fallot). Clinical and morphological characteristics.

The practical part of the subject:

Slides: In the study micropreparations pay attention to the education elements, designated by the letters in parentheses.

1. **Return warty endocarditis**. H & E stain. The valve is thickened, sclerotic and hyalinized (a), with foci of fibrinoid necrosis, necrosis of the area destroyed by the endothelium (b) the organized and fresh thrombi (c) in the thickness of the valve - the diffuse lymphoid-macrophage infiltration (g).
2. **Rheumatoid myocarditis** (granulomatous). H & E stain. The stroma of the myocardium observed tricks fibrinoid necrosis (a) around the necrosis observed focal perivascular cell infiltrates (Aschoff's body) (b), the Anitschkow cells (c) -macrophages, lymphocytes, histiocytes. The fatty degeneration of cardiomyocytes (g).
3. **Fibrinous pericarditis**. H & E stain. In epicardial tissue visible fibrin strands (a), edema and hyperemia of the vessels (B) and macrophage infiltration (c).

**macropreparations:**

**1. Acute warty endocarditis**.

Heart enlarged, left ventricular wall thickened, enlarged cavity. On the edge of the mitral valve are seen small granular thrombotic imposing a "warts", size 1 cm or more, a dark brown color.

Reasons: infectious-allergic (rheumatic diseases), infectious diseases, intoxication.

Complications thromboembolic syndrome: infarctions of the spleen, kidneys, brain, bowel gangrene.

Outcomes: valvular heart disease.

2. **polypoid - ulcerative endocarditis of the aortic valve**.

Hearts increased in size. The walls of the left and right ventricle are thickened, widened chamber. The flaps of the aortic valve thickened, sclerotic, hyalinized, deformed and fused. On the edge of the flap and rounded ulceration visible defects. On the surface of the flap seen massive crumbling thrombotic imposition of polyps (vegetations). In tendon chords and parietal endocardium organized thrombotic overlap.

Reasons: bacteremia in severe infections and septicopyemia (drug addicts, complications intracardiac catheter) is often the background is a previous infectious diseases and illness, leading to severe changes in heart valves (atherosclerosis, syphilis, brucellosis, congenital heart disease, patients on hemodialysis, immunosuppressive therapy ).

Complications: thrombosis, aneurysm wings, perforation, detachment of the valve and tendon chords. Rarely glomerulonephritis.

Outcomes: heart disease.

**3.Fibrosis mitral valve.**

Mitral valve thickened, sclerotic, twisted and spliced. Chord shortened and thickened. Along the edges of the deformed valves are arranged fresh thrombotic overlay, and organized, which leads to even more wrinkling of the valve leaflets and insufficient clamping.

Reasons: rheumatism, systemic lupus erythematosus, rheumatoid arthritis.

Complications: thromboembolism.

Outcomes: chronic heart failure, decompensation blemish.

1. **Fibrinous pericarditis ("hairy heart**"). Heart increased its surface is covered with rough gray overlays as filaments resembling the scalp. The threads of fibrin are easily separated.

Reasons: tuberculosis, rheumatic fever, uremia; nonspecific bacterial infection complicating pyosepticemia (quickly becomes purulent); severe course of viral infections (influenza, of poliomyelitis, infectious mononucleosis).

Complications: a large accumulation of fluid leads to cardiac tamponade.

Outcomes: absorption of exudate; adhesions, obliteration of the pericardial cavity with the development of constrictive pericarditis; armored heart.

**5. Congenital heart disease (pentad Fallot).**

In the heart there is a ventricular septal defect, pulmonary stenosis, right ventricular hypertrophy, dekstrapozition of aorta (aorta that overrides the VSD) and atrial septal defect.

Reasons: gene mutations, chromosomal aberrations, exposure to teratogens on the embryo in the 3-11 th week of fetal development.

Eastern Promises "blue type" (**RIGHT-TO-LEFT SHUNTS**) blood flow right to left, followed by a sharp decrease in the volume of blood in the pulmonary circulation, and severe hypoxia.

The complications and causes of death: right ventricular failure, bacterial endocarditis, embolic brain abscesses, lung infections.

Outcome: unfavorable.

**6. The artificial heart valve.**

In the area of the mitral valve is a metal structure provided "flapping" disc enclosed in a rigid cage, performing the function of the valve.

Causes: congenital and acquired valve.

Complications: infective endocarditis, thromboembolism, dysfunction of the valve.

Test control

Select one or more correct answers

**1. cardiomyopathy is characterized**

1) valves lesion

2) Coronary thrombosis

3) focal granulomas in the myocardium

4) exudative interstitial inflammation

5) dystrophic cardiomyocytes

**2. PRIMARY cardiomyopathy**

1) The hypertrophic form

2) Dilated

3) constrictive

4) canalicular

5) The restrictive

**3. TITLE rheumatic granuloma**

1) focus Abrikosov

2) focus Aschoff Bullet

3) Aschoff's body

**4. CELLS IN THE COMPOSITION rheumatic granuloma**

1) Lymphocytes

2) macrophages

3) plasma cells

4) foam cells

5) fibrocytes

**5. the TRUE NAME RHEUMATISM**

1) Lyell's disease (total cutaneous epidermolysis)

2) Buerger's disease (systemic vasculitis)

3) parietal endocarditis with eosinophilia Loeffler

4) All the answers are correct

5) disease Sokolsky-Buyo

**6. pathological process of disorganization of connective tissue rheumatism**

1) Sclerosis

2) mucoid swelling

3) inflammatory reaction

4) metaplasia

5) fibrinoid changes

**7. The primary lesion ORGANS in rheumatism**

1) heart and blood vessels

2) the small joints

3) renal pelvis

4) these organs are not affected

**8. Inflammatory reactions in rheumatism**

1) purulent - exudative

2) predominantly alterative

3) intermediates

4) granulomatous

**9. warty endocarditis imposed on in rheumatism CONSTITUTE**

1) Aschoff's body

2) granulomas Berezovsky - Sternberg

3) imposition of thrombotic

**10. pathogenesis of rheumatic diseases**

1) immunodeficiency syndrome

2) violation of transplantation immunity

3) autoimmune reactions

**11. MOST PROVEN role in the development RHEUMATISM**

1) beta-hemolytic streptococcus group A

2) beta-hemolytic streptococcus group B

3) Herpes Virus

4) kampillobakter

**12. The structure of typical rheumatoid granulomas**

1) the focus of fibrinoid necrosis

2) focus caseation

3) macrophage cells Anichkova

4) focus liquefactive necrosis

5) reaction neutrophilic

**13. SIGNS dilated cardiomyopathy**

1) dilatation of the heart chambers

2) dilation of the left ventricle

3) dilation of both atria

4) hypertrophy of the left ventricular wall

5) hypertrophy of the walls of the heart chambers

6) hypertrophy of the right ventricular wall

**14. The size of the heart in dilated cardiomyopathy**

1) significantly reduced

2) slightly decreased

3) is not changed

4) slightly increased

5) increased significantly

**15. CELLS Anichkova in rheumatoid granuloma BE CONSTRUED AS A**

1) lymphoid cells

2) plump histiocytes

3) activated plasma cells

4) epithelioid cells

**16. EXODUS rheumatic endocarditis**

1) brown atrophy of the myocardium

2) the formation of heart disease

3) small-focal cardiosclerosis

4) carcinoid defeat valve

**17. RHEUMATIC pancarditis this defeat**

1) endocardium and myocardium

2) endocardial and pericardial

3) the myocardium and pericardium

4) endocardium, pericardium and myocardium

**18. METAPHORICAL NAME THE HEART AT fibrinous pericarditis**

1) "armored"

2) "muscatel"

3) "hairy"

4) "tiger"

5) "sago"

**19. endocarditis Libman - Sachs is typical for**

1) Rheumatism

2) atherosclerosis

3) Ankylosing spondylitis

4) systemic lupus erythematosus

**20. The forms of infectious endocarditis**

1) acute

2) subacute

3) chronic

4) undulating

**21. Cause of infarction of internal organs in rheumatism**

1) aneurysms of large vessels

2) thromboembolic complications

3) beta-hemolytic streptococcus

**theme 4:** lung diseases

CONTROL QUESTIONS

1. Acute inflammatory lung disease. The role of homeostasis in the development of lung pneumonia. Classification of pneumonia. Pneumonia under immunosuppression. The concept of nosocomial infections, the causes.
2. Bacterial pneumonia. Classification. Focal pneumonia (bronchopneumonia). The etiology and pathogenesis, morphological features. Complications focal pneumonia outcomes.
3. Lobar (croupous pneumonia). Etiology, pathogenesis, clinical and morphological characteristics, stage of development, pulmonary and extrapulmonary complications, outcomes.
4. Acute interstitial pneumonitis. Viral and mycoplasma pneumonia. Clinical and morphological characteristics, outcomes.
5. Lung abscess. Classification, morphogenesis and pathological, clinical and morphological characteristics, complications, outcomes. Acute and chronic abscesses.
6. Diffuse chronic lung disease. Definition and classification. Chronic obstructive pulmonary disease. General characteristics.
7. Chronic obstructive pulmonary emphysema - definition, classification, epidemiology, etiology, patho- and morphogenesis, morphological characteristics, clinical manifestations, complications, consequences, causes of death. Other types of emphysema (compensatory, senile, vicarious, interstitial): clinical and morphological characteristics.
8. Chronic obstructive bronchitis. Definition, classification, etiology, epidemiology, patho- and morphogenesis, morphological characteristics, clinical manifestations, complications, outcomes.
9. Bronchiectasis. Definition, etiology, epidemiology, patho- and morphogenesis, morphological characteristics, clinical manifestations, complications, outcomes.
10. Diffuse interstitial (infiltrative and restrictive) lung disease. Classification, clinical and morphological characteristics, pathogenesis. Alveoli. Morphological characteristics, pathogenesis.
11. Pneumoconiosis (anthracosis, silicosis, asbestosis, berylliosis). Pato, and morphogenesis, morphological characteristics, clinical manifestations, complications, causes of death.
12. The tumors of the bronchi and lungs. Epidemiology, principles of classification. Benign tumors. Malignant tumor. Lung cancer. Bronchogenic cancer. Epidemiology, etiology, the principles of the International Classification.

The practical part of the subject:

Slides. In the study micropreparations pay attention to the education elements, designated by the letters in parentheses.

1. **Bronchopneumonia**: H & E stain. The light in the mucosa of small bronchi visible desquamation of the epithelium, edema, hyperemia of the vessels, inflammatory infiltration, in the lumen of the alveoli accumulation of leukocytes (a) and peribronchial at adjacent alveoli visible exudate consisting of desquamated alveolar epithelium, neutrophils, fibrin and individual erythrocytes (b) surrounding the alveoli are expanded, filled with air (c).
2. Lobar pneumonia (stage gray hepatization) H & E stain. The visible light alveolus filled with fibrin, neutrophils, macrophages with hemosiderin (a) collapse of pulmonary capillaries (b).
3. Carnification lung tissue: H & E stain.

In the lung alveoli are seen filled with granulation or connective tissue (a), substitute fibrinous exudate. Reason- complication of lobar pneumonia as a result of the organization of exudate. Exodus - fibrosis, development of chronic cardiopulmonary failure.

1. **Bronchiectasis with symptoms of pulmonary fibrosis**. H & E stain. The area is easily visible lumen of the bronchi, which contains white blood cells, mucus, fibrin (a), bronchial epithelial sometimes listening, sometimes with signs of squamous metaplasia (b), the basement membrane of the epithelium thickened hyalinized (c) in the submucosal layer - sclerosis, diffuse lympho-macrophage infiltration with an admixture of neutrophils (d), mucous glands in the area of atrophic sclerosis (g).
2. **Obstructive emphysema**. H & E stain. In visible light gleams respiratory bronchioles and alveoli extended (a), the alveolar walls are straight, thinned (b), have the form endplates clavate thickening due to smooth muscle cell hypertrophy (c), the vessel walls are thickened, sclerotic (g).

macropreparations:

1. **Bronchopneumonia:** in the lung on the cut seen coalescing airless pockets of dense granular appearance, is in the center of the small airways in the lumen of which is determined by the contents of grayish cloudy. The walls of the larger bronchi thickened, dull grayish gaps in content. Causes: bacteria, viruses, pathogenic fungi, rarely - protozoa. Complications: carnification, abscess formation, purulent pleurisy. Outcome: recovery; unfavorable when abscess pneumonia.
2. **Lobar pneumonia** (stage gray hepatization): the proportion of light is increased in size, dense, gray, grainy appearance, with pressure from the cut surface flows muddy liquid. The pleura dull, covered with a gray-yellow coating of fibrin. Causes - pneumococci types 1, 2 and 3, at least diplobatsilla Friedlander (Klebsiella).

Complications: pulmonary - carnification lung - the organization of exudate; the formation of acute lung abscess or gangrene; empyema; extrapulmonary to relate to the possibility of infection by lymphogenous and blood ways - with lymphogenous generalization there festering mediastenit and pericarditis, with hematogenous - metastatic abscesses in the brain, purulent meningitis, severe ulcerative and polypous ulcerative endocarditis, most of the tricuspid valve, purulent arthritis, peritonitis .

1. **Lung abscess**: In the middle lobe of the lung cavity is visible rounded with conspicuous whitish-gray wall in the cavity - a greenish-gray dense content. In the surrounding lung tissue, there are pockets of coalescing greyish grainy appearance.

The reason: a complication of acute pneumonia.

Complications and outcomes: the development of a chronic abscess, empyema, extrapulmonary suppurative complications.

1. **Bronchiectasis:** In the fragment lung bronchi greatly expanded in the form of bags or cylinder walls are thickened, whitish in the gaps defined by the contents of a thick grayish - pus. The walls of the bronchial tubes protrude above the surface of the cut in the lung tissue visible thin white layer of dense tissue, forming a mesh pattern (net diffuse pulmonary fibrosis). Causes: congenital bronchiectasis, chronic obstructive bronchitis, bronchopneumonia.

Complications: pulmonary hemorrhage, lung abscess, empyema secondary AA amyloidosis, brain abscesses.

Outcomes: chronic pulmonary heart disease

1. **Emphysema:** lung to increase in size, their edges cover the anterior mediastinum, swollen, pale pink, soft, do not collapse, cut with a crunch. In section the bronchial walls do not collapse, sticking out from the surface of the cut as "chicken feathers" in the lumen of mucopurulent contents. Peribronchial and perivascular proliferation of connective tissue. Causes: genetic predisposition, chronic non-specific inflammation in the bronchi and bronchioles, bronchiectasis, pulmonary fibrosis of various origins, old age.

Complications: the development of hypertension in the pulmonary circulation and right ventricular hypertrophy - pulmonary heart.

Outcomes: pulmonary heart disease.

1. **Silicosis of the lungs**. lobe of the lung is sealed on the visible section thick blackened scars, scars in the heart of the individual cavities can be seen arising at the site of the local ischemic necrosis. Around the deformed blood vessels and bronchi and proliferation of connective tissue. Reason: aspiration of particles of quartz dust.

Complications often joins tuberculosis.

Outcomes: pulmonary fibrosis, pulmonary heart development.

1. **Peripheric lung cancer.** At the top of the unit is easily visible round shape with sharp edges, the cut gray-white, with hemorrhages and necrosis. The reasons: the influence of various oncogenic factors. Complications: often the first clinical signs caused by hematogenous metastasis. The outcome: poor
2. **Central lung cancer**. In the left lung root node is visible gray-pink in color, with no clear contours of the node in the lung tissue strands grow grayish tissue. Lymph nodes of the root easily increase in size, the cut gray-pink with a splash of black coal dust. The reasons: the influence of various oncogenic factors. Complications: atelectasis, pneumonia, tumor lysis with pulmonary hemorrhage, abscess. Lymphatic and then hematogenous metastasis. Outcome: unfavorable.

Test control

Select one or more correct answers

**1. pneumonia refers to groups**

1) dyscirculatory diseases

2) the tumor disease

3) inflammatory diseases

4) processes of disregeneration

5) All the answers are correct

**2. For lobar pneumonia is characterized by**

1) defeat of an entire lobe or more lung lobes

2) purulent inflammatory exudate

3) fibrinous pleuritis

4) the gradual onset of the disease

5) the primary lesion of the bronchi

**3. carnification lungs at lobar pneumonia**

1) the manifestation of the disease

2) complication of the disease

3) the outcome of the disease.

**4. The stage of development of lobar pneumonia**

1) gray hepatization

2) yellow dystrophy

3) mucoid swelling

4) congestion

5) Resolution

6) red hepatization

**5. AGENTS lobar pneumonia**

1) pneumococcus

2) Staphylococcus aureus

3) Streptococcus

4) virus pneumotropic

5) Klebsiella

**6. morphological characters lobar pneumonia AT THE STAGE OF GREY hepatization**

1) fibrin in alveoli

2) the leukocytes and macrophages in the alveolar exudate

3) purulent exudate meltdown

4) edematous fluid in the alveoli

5) collapse of pulmonary capillaries

**7. Pulmonary complications of lobar pneumonia**

1) Encephalitis

2) carnification

3) lung abscess

4) purulent mediastinitis

5) a pleura empyema

6) gangrene (wet)

**8. carnification - IT IS**

1) Organization of exudate in the alveoli with the formation of granulation tissue initially, and then the mature connective tissue

2) excessive activity of leukocytes in the exudate

3) suppuration of exudate

4) bleeding in the exudate

**9. CAUSE carnification**

1) enhanced fibrinolysis of exudate

2) joining pyogenic flora

3) insufficient fibrinolytic activity of leukocytes

4) the presence of extrapulmonary complications

5) All the answers are correct

6) All the answers are incorrect

**10. A characteristic morphological features of focal pneumonia**

1) involvement of the pleura

2) acute bronchitis and bronchiolitis

3) necrosis cheesy of exudate

4) fibrinous exudate in the lumen of the alveoli

5) defeat lobe

**11. AGENTS interstitial pneumonia**

1) Streptococcus

2) pneumococcus

3) Staphylococcus aureus

4) viruses

5) mycoplasma

**12. Bronchiectasis - a pathological EXPANSION**

1) the lumen of the alveoli

2) one or more of the lumen of the bronchi, comprising cartilaginous plate and glands with destruction lamina propria and muscularis

3) small bronchi

**13. MAIN TYPES of bronchiectasis**

1) varicose

2) Cylinder

3) mushroom

4) saccular

5) stellate

**14. chronic nonspecific lung diseases**

1) pneumonia

2) chronic obstructive pulmonary emphysema

3) chronic bronchitis

4) bronchiectasis

5) brown induration of lungs

**15. emphysema may be a manifestation**

1) chronic nonspecific pulmonary inflammation

2) age-related processes

3) compensatory and adaptive processes

4) All the answers are correct

5) All the answers are incorrect

**16. morphological characters DESTRUCTIVE of bronchiectasis**

1) perifocal inflammation

2) stretching acini

3) carnification

4) the gap mezhalveolyarnyh partitions.

**17. The changes in the lungs with diffuse chronic bronchitis**

1) small focal atelectasis

2) macrofocal sclerosis

3) the formation of destructive bronchiectasis

4) a mesh fibrosis

5) carnification

**18. Complications of chronic obstructive bronchitis**

1) cardiopulmonary failure

2) pneumonia

3) gangrene

4) pulmonary hemorrhage.

**19. CAUSES pneumoconiosis**

1) industrial poisons

2) the effect of physical factors

3) Infection

4) industrial dust

**20. REASON IS silicosis dust containing**

1) silicon dioxide

2) the particles of coal

3) talc

4) silicates

**21. obstructive pulmonary disease**

1) asthma

2) chronic obstructive bronchitis

3) chronic obstructive pulmonary emphysema

4) bronchiectasis

5) chronic bronchiolitis

**22. The most commonly histologic type of central lung cancer**

1) adenocarcinoma

2) the bronchioles-alveolar

3) squamous

4) small-cell

5) large cell

**23. The most frequently histologic type of peripheral lung cancer**

1) adenocarcinoma

2) the bronchioles-alveolar

3) squamous

4) small-cell

5) large cell

**24. The important factor for developing chronic bronchitis**

1) heart failure

2) Smoking

3) pulmonary hemosiderosis

4) lymphostasis

5) industrial dust

**25. ELEMENTS sarcoid granulomas**

1) hearth cheesy necrosis

2) Neutrophils

3) CD4 + T lymphocytes

4) fibroblasts

5) epithelioid cells

**26. bronchiectasis IT IS**

1) expansion of the lumen of the alveoli

2) expansion of the lumen and increase the size of the bronchial glands

3) expansion of the bronchi

4) retention cyst

**theme 5:** **gastrointestinal tract diseases**

CONTROL QUESTIONS

1. Inflammation and damage to salivary gland tumor. Sialadenitis, sialolithiasis: etiology, pathogenesis pas, morphology, outcomes. Benign and malignant tumors, tumor-like diseases. Classification, morphological characteristics, complications, prognosis.

2. Diseases of the esophagus. Diverticula of the esophagus (congenital and acquired). Ruptures of the esophagus (Mallory-Weiss syndrome). Esophagitis. Barrett's esophagus. Etiology, morphogenesis, clinical and morphological characteristics, complications, outcomes.

3. Tumors of the esophagus. Benign tumors: classification. Malignant tumor. Cancer of the esophagus, morphological characteristics, complications, outcomes, prognosis.

4. Diseases of the stomach. Gastritis. Definition. Acute gastritis. Etiology, pathogenesis, morphological forms. Clinical and morphological characteristics.

5. Chronic gastritis, the essence of the process. Etiology, pathogenesis. Principles of classification. Forms allocated on the basis of the study gastrobiopsy, morphological characteristics. Complications, outcomes, prognosis. Chronic gastritis as a precancerous condition.

6. Peptic ulcer disease. Definition. General characteristics of peptic (chronic) ulcers of different locations. Epidemiology, etiology, morphogenesis. Morphological characteristics of chronic ulcers in the period of exacerbation and remission. Complications, outcomes. Acute gastric ulcers: etiology, pathogenesis, morphological characteristic,outcomes.

7. Tumors stomach. Classification. Hyperplastic polyps. Adenoma of the stomach. Morphological characteristics. Malignant tumors of the stomach. Stomach cancer. Epidemiology, etiology, classification principles. Features metastasis. Macroscopic and histological forms.

8.Sindromy malabsorption. The role of the morphological study biopsy in the diagnosis of diseases of the colon.

9. Whipple's disease. Ulcerative colitis. Crohn's Disease. Etiology, morphogenesis, morphological ical characteristics, complications, outcomes, prognosis. The criteria for the differential diagnosis of chronic colitis.

10. Appendicitis. Classification, etiology, classification. Morphological characteristics of appendicitis. Complications. Features of the disease in children and the elderly.

The practical part of the subject:

Slides. In the study micropreparations pay attention to the education elements, designated by the letters in parentheses.

**1. Chronic gastric ulcer during the exacerbation**. H & E stain. In the area of the defect of the stomach wall has a fibrinous-purulent exudate (a) to be an extensive area of fibrinoid necrosis (b), the presence of granulation tissue (c), and the growth of coarse fiber connective tissue, penetrating to different depths of the muscular layer (d). Serous membrane of the stomach wall preserved (e).

**2. Adenocarcinoma**. H & E stain. All the layers of the stomach wall infiltrated tumor tissue with signs of cell irregularities (a). Abnormal mitoses seen in multiple hyperchromatic (b) and polymorphic tumor cells (c).

**3. Crohn's disease**. H & E stain. The wall of the colon has ulcer (1) penetrating into the muscular layer in the mesentery tissue, forming a fistulous tract (2). Lymphoplasmacytic inflammatory infiltrate (3) applies to all membrane of the intestinal wall, preserving architectonic crypts and the number of goblet cells. Thickened bowel wall due to edema, inflammatory infiltrate (4) areas of fibrosis and hypertrophy of the muscle membrane (5). Reveals a granuloma (6), composed of epithelioid and giant cell type Pirogov-Langhans (7) surrounded by a belt of lymphocytes, without clear boundaries (8). Unlike the granulomas in sarcoidosis, no fibrous ring and from tuberculous granulomas - no cheesy necrosis.

**4. Phlegmonous appendicitis**. H & E stain. All the layers of the wall of the appendix is diffusely infiltrated with polymorphonuclear leukocytes (a). There are abundant fibrinous deposits in the serous membrane (b). In the lumen of the process of accumulation of pus (c). The mesentery of the appendix congestion of vessels and inflammatory infiltration - mezenteriolit (g).

**macropreparations:**

**1. Acute catarrhal gastritis**: in preparation stomach mucosa is thickened, congested with high folds, covered with thick viscous mucus, with petechial hemorrhages. Causes: poor quality food, drinking alcohol surrogates, antineoplastic chemotherapy, burns, acids and alkalis, uremia, salmonellosis, shock, severe stress.

Complications: acute ulcer, the transition to chronic gastritis.

**2. Erosions and acute gastric ulcer**: in Preparation stomach mucosa swelling, on the surface there are multiple petechiae and conical shape defects of different sizes, their bottom edges and black. Erosions localized within the mucosa and ulcers penetrate to different depths of the mucosa, and some reach the muscle membrane. The reasons: endocrine disease (Zollinger-Ellison syndrome, hyperparathyroidism), acute and chronic circulatory disorders, poisoning, allergies, chronic infections (tuberculosis, syphilis), postoperative, steroid and stress ulcers

Complications of perforation, peritonitis.

Outcome: erosion epiteliziruyutsya, ulcerative defect is replaced by scar tissue.

**3. Chronic gastric ulcer in remission**: in preparation stomach on the lesser curvature has a pathological lesion in a recess of the mucous membrane, a rounded shape, the size of 3 cm in diameter. In the context of the inlet hole crater smaller than the inside of the ulcer. Edge facing a cardia undermining the mucous membrane over it hangs. Edge facing away gatekeeper sloping, terraced. Column ulcers presented connective tissue, gray-white, 2.5 cm. At the bottom of the ulcer sclerotic vessels, clearance of their gapes.

Causes: genetic predisposition, Helicobacter pylori, and disregeneratornye inflammatory mucosal changes, the effects of the factors leading to peptic aggression (hydrochloric acid and pepsinogen).

Complications: perigastrit, bleeding, perforation, penetration, scar deformity of the stomach with the development of stenosis input or output openings. Against the backdrop of a chronic ulcer may develop a second disease - cancer of the stomach.

**4. stomach polyps** (adenomas). in the antrum, there are two tumor formation size of a pigeon egg on thin legs, irregular oval shape with villous surface, soft consistency. In the context of pathological growths richly vascularized and localized exclusively on the mucosal surface, not germinating underlying tissues.

Complications: bleeding, obstruction of output or inlet.

Outcome: malignancy.

**5. Various forms of stomach cancer**. a) Mushroom cancer: at mucosal surfaces has tumor formation, growing into the gastric lumen, irregularly rounded shape measuring 5 cm in diameter, on a broad base in the form of a mushroom cap with the retraction of the center. In the section shows that the entire tumor invades the stomach wall.

b) diffuse gastric cancer: the body is reduced in size, the wall throughout the thickened up to 1cm thick 'woody' consistency on the cut shows a pinkish-gray tissue. The mucous membrane is uneven, it folds of varying thickness, serosa is thickened, dense, hilly. The lumen of the stomach contractions.

Causes: food (smoked, canned, pickled vegetables, pepper), biliary reflux (after operations on the stomach, especially Billroth II), Helicobacter pylori (promotes mucosal atrophy, intestinal metaplasia, dysplasia).

Metastasis: 1. orthograde nodal metastases in regional nodes in the small and large curvature, retrograde nodal metastases in the left supraclavicular lymph node - metastasis Virchow, in the ovaries - Krukenberg cancer, adrectal fiber - metastases Schnitzler.

2. Hematogenous metastases to the liver, lungs, brain, bones, kidneys, adrenal glands and less frequently in the pancreas. 3. Implantation - carcinomatosis of the pleura, pericardium, diaphragm, peritoneum, omentum.

**6. Ulcerative Colitis**. In colon preparation with sharply sanguineous mucosa. Along the mucosal erosions and ulcers of various changes in size and shape. The bottom of the ulcers clean without festering overlays, covered with a thin layer of fibrin brilliant. The surviving islets mucosa numerous pseudopolyps small size (0.2 cm - 0.5 cm), with no clear division into the leg and body with a smooth surface.

Causes: genetic predisposition, disturbance cenosis bacterial, viral or bacterial beginning, an autoimmune reaction to antigens, food allergy, changes in the immunological reactivity.

Intestinal complications: toxic dilatation of the colon, perforation, gangrene. Extraintestinal complications: skin lesions (erythema, nodular, massive leg ulcers, gangrenous pyoderma), arthritis, eye damage (episcleritis, uveitis, iridocyclitis), rarely sepsis, amyloidosis, pericholangitis with the development of fibrotic changes with the outcome of biliary cirrhosis.

The outcome: a partial or complete epithelialization of ulcers, the formation of scar tissue within the mucosa. Against the background of epithelial dysplasia may develop cancer.

**7. Crohn's disease**. The preparation portion of the transverse colon, descending colon, the mucosa on the cut even near ulcers -Pink pale color. There is an alternation of the affected areas with no changes in the mucous. Deep slit-like ulcers are oriented along and across the axis of the colon are smooth and not saped edge and preserved between the areas of edematous mucosa attached to the surface of the colon similar to the "cobblestone street". Characteristically segmental narrowing of the intestinal lumen extending from 5 cm to 10 cm - "threadlike colon." Some ulcers penetrate the muscular layer, forming a fistula connecting the different parts of the colon and small intestine. Serous membrane dull, gray, edematous mesentery, there are extensive adhesions between loops of intestine.

Causes: genetic predisposition, disturbance cenosis bacterial, viral or bacterial beginning, autoimmune reaction to hypertension, a food allergy, the change of immunological reactivity.

Complications: perforation in the free abdominal cavity fistulas with the outlet on the skin of the abdominal wall, fistulas connecting the intestine to the bladder, uterus, stomach, rectal fistula.

Outcome: Strictures fine, colon and rectum occur in ¼ of patients. Cancer on the background of Crohn's disease less often than with ulcerative colitis.

**8. Phlegmonous appendicitis.** Appendix enlarged and thickened to 1.5 cm in diameter, with serosa dull gray overlays fibrin. Mesenteric vessels congested. In the context of appendiceal lumen process with accumulation of purulent exudate impregnating the entire wall.

Reasons: circulatory disorders, obstruction of the lumen, followed by compression of the veins and the development of ischemia, infection. Complications: ulceration, perforation, paraappendicitis, mezenteriolit, gangrenous inflammation, peritonitis, liver abscesses.

Test control

Select one or more correct answers

**1. The symptoms of acute catarrhal gastritis**

1) mucosal thickening

2) atrophy of the glands

3) multiple erosions

4) mucous sclerosis

5) neutrophilic infiltration of the mucous

6) mucosal lymphoid infiltration

**2. morphological forms of acute gastritis**

1) fibrinous

2) atrophic

3) hypertrophic

4) catarrhal

5) Corrosive (necrotic)

**3. changes in the epithelium in chronic gastritis**

1) atrophy

2) intestinal metaplasia

3) hyperplasia

4) dysplasia

**4. Clinical and morphological signs of chronic atrophic gastritis in the acute stage**

1) occurs frequently in patients with alcoholism

2) the mucosa is not changed

3) diffuse lymphoid infiltration plasmocytic with considerable admixture of PMN

4) focuses pyloric and intestinal metaplasia

5) gastric hyperacidity

**5. Sclerotic deformation of stomach is the outcome**

1) catarrhal gastritis

2) diphtheritic gastritis

3) corrosivity gastritis

4) abscess gastritis

**6. Local factors in the development of gastric ulcer**

1) increase the aggressiveness of gastric juice

2) campylobacter

3) presence of chronic gastritis

4) poor circulation

5) All the answers are correct

6) All the answers are incorrect

**7. CAUSES OF ACUTE stomach ulcers**

1) corticosteroids

2) Stress

3) Aspirin

4) Smoking

5) increasing vagal tone

**8. Signs of chronic gastric ulcer during the exacerbation**

1) the presence of fibrinopurulent exudate on the surface

2) scar tissue interrupts muscle membrane at different depths

3) endangitis

4) fibrinoid changes in vascular walls and in the bottom of ulcers

5) The deepest zone is located is represented by coarse fiber ulcer scar tissue

**9. MECHANISM OF BLEEDING IN ULCER**

1) arrosive

2) diapedetic

3) due to rupture of the vessel

4) as a result of purulent fusion

**10. Complications of chronic ulcers**

1) penetration

2) perforations

3) empyema

4) hypercalcemia

5) scarry stenosis and deformation of the wall

6) bleed

**11. Benign tumors STOMACH**

1) angiosarcoma

2) adenoma

3) leiomyoma

4) adenocarcinoma

**12. ADENOMA THIS IS**

1) benign tumor of glandular epithelium

2) malignant tumor of glandular epithelium

3) epidermal cancer

4) malignant tumor of the transitional epithelium

5) benign tumor of squamous epithelium

**13. Microscopic characteristics scirrhous stomach cancer**

1) atypical cells with large nuclei are arranged in groups

2) atypical cells form cancer

3) massive proliferation of connective tissue

4) the abundance of mucus in the lumen of the glands

5) atypical cancer cells do not form

**14. COMPLICATIONS OF GASTRIC CANCER**

1) hemoptysis

2) dilation of the pylorus

3) perforation

4) depletion

5) gastric bleeding

**15. The cause of development of appendicitis**

1) blockage of coprolites

2) appendicular artery thrombosis

3) obstruction of gallstones

4) compression of the veins process

5) microbial flora

**16. The characteristic signs of ulcerative colitis**

1) the place of defeat - rectum

2) chronic inflammation affects the entire thickness of the bowel

3) the mucous membrane looks like a cobblestone street

4) is characterized by crypt abscesses

5) characterized pseudopolyps

6) often leads to the development of colon cancer

7) is often complicated by fistulas interintestinal

**17. Characteristic signs CROHN'S DISEASE**

1) the place of defeat - rectum

2) chronic inflammation affects the entire thickness of the bowel

3) the mucous membrane looks like a cobblestone street

4) is characterized by crypt abscesses

5) characterized pseudopolyps

6) often leads to the development of colon cancer

7) is often complicated by fistulas interintestinal

**18. morphological characters CROHN'S DISEASE**

1) is characterized by segmental defeat cancer, "Hose stricture"

2) crypt abscesses

3) fibrinous plaques on the mucous membranes

4) atrophy of the mucosa

5) Noncaseating granulomas

**19. morphological signs of ulcerative colitis**

1) ulcerative process within the mucosa

2) lymphoplasmacytic infiltrate all layers of the bowel wall

3) deep ulcers to the muscle layer

4) crypt abscesses

5) lymphoplasmacytic infiltration of the lamina propria with an admixture of eosinophils and leukocytes

**20. The cause of development of ischemic colitis**

1) Atherosclerosis mesenteric arteries

2) nonbacterial thrombotic endocarditis

3) the absence of ganglion cells in the submucosal layer

4) systemic vasculitis

5) Meckel's diverticulum

**21. Morphological changes in ischemic colitis**

1) hemorrhagic infarction

2) gangrene

3) megacolon

4) the proliferation of granulation tissue with subsequent fibrosis

5) polyposis mucosa

**theme 6: Diseases of liver and biliary system.**

**Hepatitis. Cirrhosis. LIVER CANCER. Cholelithiasis.**

CONTROL QUESTIONS

1. **The role of the liver in the functioning of the organism**. Characteristics of the main groups of pathological processes in the liver. Classification. Epidemiology.
2. **Hepatitis.** Definition. Classification principles: the current, etiology, origin and morphology.
3. **Viral hepatitis:**  
   a) characteristics of the etiological factors (A, B, C, D-type virus). Priority ways of infection. Pathogenesis. The morphological changes in the liver: morphological cytolysis, cell reactions, the bile production and biliary excretion,

b) morphology of acute viral hepatitis: acute cyclic form, form with massive necrosis, pericholangiolitic form. Outcomes,

c) morphological manifestations of chronic hepatitis, pathogenesis, outcomes.

1. **Alcoholic liver disease.** Alcoholic fatty liver. Alcoholic hepatitis. Alcoholic cirrhosis. Epidemiology, pathogenesis, morphogenesis, morphological characteristics, clinical manifestations, complications and causes of death, outcomes, prognosis.
2. **Hepatosis.** Definition. Etiology. Pathogenesis. The acute toxic degeneration of the liver. Meaning of sensitization in its development. Period of yellow and red dystrophy. Outcomes. Causes of death.
3. **Chronic steatosis.** Reasons. Clinical and morphological manifestations. Outcomes.
4. **Cirrhosis.** Definition. Etiology. Pathogenesis. Classification principles.
5. pathological anatomy of postnecrotic, portal, biliary and mixed cirrhosis.
6. the major complications of portal hypertension and hepatic (hepatocellular) failure. Hepatic encephalopathy. Icterus. Renal failure. Ascites and edema. Endocrine disorders. Circulatory disorders and infectious complications. Pathogenesis, clinical and morphological characteristics.
7. **Hepatic tumors**. Benign and malignant neoplasms. Epidemiology. Histogenesis. Macro- and microscopic picture. Regularities metastasis.
8. **Cholecystitis, cholangitis.** Ways of infection. Meaning of the stones in the development of cholecystitis. Types of acute cholecystitis (catarrhal, purulent, gangrenous). Chronic cholecystitis. Morphology. Complications. Tumors and congenital anomalies of biliary tract. Classification. Clinical and morphological characteristics.

The practical part of the subject:

Slides: In the study **micropreparations** pay attention to the education elements, designated by the letters in parentheses.

1. **Hepatitis.** H & E stain. In the hepatic parenchyma are observed hydropic degeneration (a), necrosis of hepatocytes (b) with the presence of Councilman’s body (c), hyperemia of vessels and edema of the stroma (d). In the portal tracts are observed diffuse lymphohistiocytic infiltration (e), hyperplasia of stellate reticuloendotheliocytes (Kupffer’s cells).
2. **Steatosis.** Stain Sudan III. Fatty degeneration and necrosis of hepatocytes (a), in the stroma - cellular reaction and proliferation of connective tissue (b).
3. **Toxic liver degeneration.** H & E stain. The structure of hepatic lobules changed, hepatocytes in the center of the lobules is in a state of fatty degeneration and necrosis (a), on the periphery of the lobules hepatocytes with signs of reparative regeneration (b). Newly formed bile ducts determined (c).
4. **Postnecrotic cirrhosis.** H & E stain. Hepatocytes is in a state fatty degeneration and necrosis (a). There is a violation of the trabecular structure of the liver and the formation of false lobules (nodes regenerates) (b), between which proliferation of connective tissue are observed (c). Portal triads close together and with the central veins (d). Proliferating bile ducts (e) and lympho-macrophage infiltration (f) are observed.
5. **Biliary cirrhosis (secondary).** H & E stain. In the central parts of liver lobules are observed the focal necrosis of hepatocytes (a) and periportal necrosis with the formation of "bile lakes" (b). Around nodes- regenerators (c) are observed proliferation of connective tissue that connects the central and portal zones of lobules (d). Bile capillaries expanded with signs of cholestasis (e).
6. **Muscat cirrhosis.** H & E stain. In the center of hepatic lobule stagnant hyperemia (a), fatty degeneration and necrosis of hepatocytes (b), in peripheral parts - hypertrophied hepatocytes (c). Around nodes- regenerates (d) and in the portal tracts are observed proliferation of connective tissue, the compound of the central vein with portal fields (e).

**macropreparations:**

**1. Postnecrotic cirrhosis:** the liver is greatly reduced in size, yellowish gray color, in the surface are observed **large** hillocks. Consistency liver is dense. On the cut organ have the nodular structure in a spherical form foci of various sizes, up to 3 cm in diameter. Between nodes – regenerates are observed wide layer of fibrous tissue**.** **Reasons:** acute toxic degeneration of the liver, viral hepatitis with massive necrosis, chronic hepatitis high activity, hepatotoxic poisons.  
**Complications:** hepatocellular insufficiency - hepatic encephalopathy, jaundice, hemorrhagic syndrome, hormonal disorders, hepatorenal syndrome, dyspepsia; hepatocellular carcinoma. **Outcome:** hepatocellular insufficiency.

**2. Muscat cirrhosis:** the liver is reduced in size, in the surface are observed **small** hillocks, dense texture. On the cut on the background of diffuse nodes regenerates with narrow interlayers of connective tissue, is determined mottled parenchyma in the form of reddish inclusions. **Reasons:** Chronic heart failure, chronic venous stasis in the systemic circulation. **Complications:** the syndrome of portal hypertension, ascites, splenomegaly, varicose portocaval anastomosis, bleeding, anemia. **Outcome:** portal hypertension.

**3. Biliary cirrhosis (secondary):** liver slightly enlarged, light brown color with green blotches, surface is small hilly, texture is dense, on the cut - structure of the parenchyma nodular structure, divided by gray narrow interlayers of fibrous tissue. Bile ducts are dilated, filled with bile.

**Reasons**: blockage of the large bile ducts - gallstone disease, inflammatory narrowing (stricture) of the biliary tract, primary and metastatic tumors gepatopankreoduodenalnoy zone, parasitic diseases of the liver and biliary tract (hydatid disease, ascariasis, opistorhoz), congenital biliary atresia, cyst ducts less sclerosing cholangitis. **Complications:** pneumonia, abscess formation, sepsis. **Outcome:** hepatocellular insufficiency.

**4. Multiple liver abscesses:** liver slightly enlarged, on the cut in the subcapsular parenchyma there are multiple pathological foci round shape, different sizes, containing pus. **Reasons:** purulent destructive cholangitis and cholangiolitis.

**Complications:** jaundice, hepatic failure. **Outcome:** unfavorable, pyosepticemia.

**5. Gallstones:** the gall bladder enlarged in size, on the serosa have gray-white imposing of fibrin with the organization. On the cut on the wall of gall bladder is thickened to 0,8 mm, the mucous membrane is smoothed. The lumen of the gall bladder has multiple stones, smooth, green-brown. The stones are located compactly. **Complications:** of perforation, abscess and gangrenous cholecystitis, peritonitis, jaundice. **Outcome:** determined complications.

Test control

Select one or more correct answers

1. **Morphological basis of hepatosis**  
   1) degeneration of hepatocytes  
   2) necrosis of hepatocytes  
   3) inflammation in the liver  
   4) pylephlebitis
2. **Morphological signs of toxic hepatic dystrophy**   
   1) reducing the size of the liver  
   2) imposing of fibrin on the capsule  
   3) extensive necrosis of hepatocytes  
   4) grainy surface  
   5) increase the size of the liver  
   6) the flabby consistency of liver
3. **Outcomes of** **toxic hepatic dystrophy**   
   1) Portal cirrhosis  
   2) Postnecrotic cirrhosis  
   3)Biliary cirrhosis  
   4) Muscat cirrhosis
4. **CAUSES OF steatosis**  
   1) poor quality food poisoning  
   2) Alcohol  
   3) mushroom poisoning  
   4) diabetes  
   5) viral hepatitis
5. **PRIMARY HEPATITIS**  
   1) septic hepatitis  
   2) drug-induced hepatitis  
   3) alcoholic hepatitis  
   4) Viral Hepatitis  
   5) tuberculous hepatitis
6. **Signs of chronic persistent forms of viral hepatitis "B"**  
   1) stored lobular structure  
   2) periportal fibrosis  
   3) infiltration in the portal tracts  
   4) expressed cholestasis  
   5) apoptotic Mallory’s bodies
7. **Signs of** **chronic viral hepatitis "C"**  
   1) macrovesicular steatosis of hepatocytes  
   2) the formation of lymphoid follicles in the portal tracts  
   3) confluent and bridging necrosis of hepatocytes  
   4) apoptotic Councilman’s bodies   
   5) expansion of portal tracts due to fibrosis
8. **Signs of acute alcoholic hepatitis**1) fatty degeneration of hepatocytes  
   2) leukocyte infiltration  
   3) the presence of cells Mallory  
   4) the formation of Councilman’s bodies  
   5) focal necrosis of individual hepatocytes
9. **Chronic viral hepatitis develop after**1) Hepatitis "B"  
   2) hepatitis "C"  
   3) Hepatitis "A"  
   4) the combined hepatitis "B" and «D»  
   5) Hepatitis "E"
10. **Cirrhosis can be caused by**  
    1) a fulminant hepatitis  
    2) diabetes  
    3) purulent osteomyelitis  
    4) the alimentary protein deficiency  
    5) alcoholism
11. **Morphological signs of postnecrotic cirrhosis**  
    1) the approach of portal triads with each other and the central veins  
    2) degeneration and necrosis of hepatocytes  
    3) lympho-macrophage infiltration  
    4) leukocyte infiltration  
    5) cholangitis, cholestasis
12. **Morphological signs of portal cirrhosis**  
    1) small tuberosity of liver  
    2) wide connective field  
    3) fine-meshed network of connective tissue in the lobules  
    4) early hepatic failure  
    5) early portal hypertension
13. **Histological features of primary biliary cirrhosis**  
    1) granulomatous cholangitis  
    2) decrease in the number of bile ducts  
    3) infiltration of portal tracts  
    4) expansion of portal tracts due to fibrosis
14. **SECONDARY biliary cirrhosis is characterized by**  
    1) in the surface large hillocks  
    2) liver dark green color  
    3) bile stasis  
    4) in the surface small hillocks  
    5) associated with progressive massive hepatic necrosis  
    6) associated with obstruction of extrahepatic bile ducts
15. **SIGNS hepatocellular insufficiency**  
    1) hyperalbuminemia  
    2) icterus  
    3) encephalopathy  
    4) hepatorenal syndrome  
    5) coagulopathy
16. **General factors of stone formation**1) violation of the osmotic pressure  
    2) violation of protein metabolism  
    3) violation of mineral metabolism  
    4) avitaminoses  
    5) increase in blood viscosity
17. **Histological signs of acute cholecystitis**  
    1) neutrophilic infiltration of the bladder wall  
    2) sclerosis of the bladder wall  
    3) lymphoid infiltration of the bladder  
    4) necrosis of the bladder wall  
    5) imposition of fibrin on the mucous
18. **Histological signs of chronic cholecystitis**  
    1) atrophy of the mucosa  
    2) sclerosis of the bladder wall  
    3) lymphoid infiltration of the bladder  
    4) necrosis of the bladder wall  
    5) imposition of fibrin on the mucous
19. **Histological forms of gallbladder cancer**1) scirrhus  
    2) adenocarcinoma  
    3) epidermal cancer  
    4) mucosal cancer
20. **Histological forms of liver cancer**  
    1) hepatocellular carcinoma  
    2) cholangiocellular cancer  
    3) anaplastic cancer  
    4) small cell cancer
21. **The cause of death of patients with cirrhosis**  
    1) pulmonary embolism  
    2) hepatocellular insufficiency  
    3) complications of portal hypertension  
    4) hepatocellular carcinoma  
    5) secondary bacterial infection

**theme 7: Renal disease.**

**Glomerular diseases. Glomerulonephritis. Tubulopathy. Nephrotic syndrome. Diseases of the urinary system and the male reproductive system.**

CONTROL QUESTIONS

1. **Glomerulardisease. Glomerulonephritis**. Сlassification, etiology, pathogenesis, immunomorphological characteristics.
2. **Acute glomerulonephritis.** **Post-streptococcal glomerulonephritis** and non streptococcal glomerulonephritis. Rapidly progressive glomerulonephritis. Etiology, pathogenesis, morphological characteristics and outcomes.
3. **Chronic glomerulonephritis**.Determination, macro- andmicroscopic characteristics. Uremia. Etiology, pathogenesis,clinical andmorphological characteristics.
4. **Nephrotic syndrome**.Classification. **Membranous nephropathy. Lipoid nephrosis. Focal segmental glomerulosclerosis**. Etiopathogenesis, morphological characteristics. Electron microscopic differential - diagnostic features. **Membranoproliferative glomerulonephritis. IgA-nephropathy.** **Focal proliferative and necrotic glomerulonephritis.** Etiopathogenesis, morphological characteristics.
5. **Glomerular lesions associated with systemic diseases:** Systemic lupus erythematosus. Henoch-Schonlein purpura. Bacterial endocarditis. Goodpasture's syndrome, essential cryoglobulinemia, plasma celldyscrasias. Pathogenesis,clinical andmorphological characteristics. Forecast. Renal amyloidosis. Methods of diagnosis, clinical manifestations.
6. **Hereditary nephritis**. Epidemiology, classification, pathogenesis, morphological characteristics.
7. **Kidney disease associated with damage to the tubules and interstitium.** Classification. Acute tubular necrosis(necrotic nephrosis). Etiology, pathogenesis, morphological characteristics, clinical manifestations, prognosis.
8. **Tubulointerstitial nephritis.** Classification, etiology, morphological characteristics, clinical manifestations, outcome. Tubulointerstitial nephritis induced by drug sand toxins. Analgesic nephropathy, pathogenesis, morphological characteristics.
9. **Pyelonephritis, and urinary tract infections.** Definition, classification. Etiological and contributing factors, pathways of infection in the kidney. Acute pyelonephritis. Definition, etiology, contributory diseases and pathogenesis, morphological characteristics, complications.
10. **Chronic pyelonephritis and reflux nephropathy.** Etiology, pathogenesis, morphological variations and morphological characteristics, clinical manifestations, outcomes.
11. **Nephrolithiasis.** General and local factors playing a role in stone formation. Patho- and morphogenesis, clinical and morphological characteristics, outcomes. Urate nephropathy.
12. **Tumors of the kidney.** Epidemiology, contributing factors, classification. Benign tumors: histogenesis, clinical and morphological characteristics, prognosis. Malignant tumors: renal cell carcinoma, urothelial carcinoma. Morphological characteristics, especially metastasis, clinical manifestations, prognosis.
13. **Diseases of the prostate gland**. Classification. Inflammatory disease. Prostatitis: acute bacterial, chronic. Etiology, morphogenesis, clinical and morphological characteristics, complications, outcomes.
14. **Benign nodular hyperplasia of the prostate gland.** Causes, clinical manifestations, histological variants, complications and outcomes.
15. **Tumors of the prostate gland.** Classification. Epidemiology, risk factors, causes, pathogenesis, and morphogenesis. Prostate cancer, histological variants, molecular markers, clinical manifestations, complications, outcomes.
16. **Tumors of the urinary bladder.** Classification. Morphological characteristics of benign transitional cell tumors, prognosis. Malignant epithelial tumors. Epidemiology, risk factors, etiology and morphogenesis, clinical and morphological characteristics of the different histological types of cancer, prognosis. Mesenchymal tumor. Secondary neoplastic lesions.

The practical part of the subject:

Slides: In the study **micropreparations** pay attention to the education elements, designated by the letters in parentheses.

1. **Necrotic nephrosis.** H &E stain.   
   in theconvoluted tubuleepithelialnecrosis ofthe kidneys(a) and the site of the destroyedbase membrane (b) are observed. Occlusion of lumenof thedistal tubule by cylinders (c), with the expressedvascularhyperemiaof medulla(d), interstitial edema, hemorrhage and accumulation of leukocytes in the dilated blood vessels (e)
2. **Amyloid nephrosis.** Stained Congo-Roth. In mesangial glomerular amyloid brown-red (a), as well as along thebasalmembraneof tubules(b) in the walls ofarteriolesand(c). The lumen ofthe tubularexpandedpackedcylindersin theirepithelium ofmanylipids (d). Therediffuse sclerosisof the stromamedulla(d).
3. **Intracapillary proliferative glomerulonephritis**. H &E stain. Renal glomerulus is enlarged, anemic (a), there is swelling and proliferation of endothelial cells and a slight mesangial cell proliferation (b), in a lumen of capillary loops single neutrophils (c).
4. **Chronic nephritis (secondary contracted kidney).** H &E stain. The glomeruli in a state of collapse, replaced by connective tissue or hyaline (a), tubules is atrophic, epithelium is flattened (b), the walls of the arterioles thickened and replaced by hyaline, its lumen is significantly narrowed (c). Number of interstitial connective tissue increased (nephrosclerosis) (d), stored nephrons is hypertrophied (e).
5. **Chronic pyelonephritis.** H & E stain.

Most of the tubules is expanded and filled with colloid-cylinder (a), there is a diffuse interstitial sclerosis cortex and medulla (b), lympho-histiocytic infiltration with an impurity of neutrophils (c), part of the glomeruli saved with a marked periglomerular sclerosis (d).

1. **Glandular-muscular hypertrophy of the prostate gland.** H & E stain.

Adenomere is extended with proliferation of the glandular epithelium and the formation of papillary structures, directed at glandular cavity (a), the proliferation of fibromuscular stroma (b), some acini is cystically dilated, lined by flattened epithelium (c), in the stroma are observed periglandular lymphohistiocytic infiltrates (d).

**Macropreparations:**

**1. Acute glomerulonephritis.**

The kidneys are enlarged, flabby, with a wide full-blooded cortex, which is visible in the red specks - "motley kidneys".

**Reasons:** nephritogenic strains of beta-hemolytic streptococcus group A.

**Outcome:** recovery; transition to chronic glomerulonephritis.

**2. Subacute glomerulonephritis with acute exacerbation.**

The kidneys are enlarged, pale, flabby consistency, with petechial hemorrhages on the surface. In the cut cortex dim, yellowish-gray with red specks, sharply demarcated from dark red pyramid - the "big motley kidney" or "big red kidney".

**Reasons:** systemic diseases, primary renal lesion (idiopathic and related with antibody to glomerular basement membrane, or related with immune complexes).

**Complications:** anuria, pulmonary hemorrhage (Goodpasture's syndrome), malignant hypertension.

**The outcome:** the early development of renal failure, secondary contracted kidney.

**3. Kidneys with chronic glomerulonephritis (secondary contracted kidney).**

The kidneys are symmetrically contracted, dense, gray, fine-grained surface. In the context of the layers thinned, the boundary between the cortex and medulla is not expressed. Around the renal pelvis proliferation of adipose tissue.

**Reason:** terminal stage glomerular inflammatory diseases.

**Complications:** bleeding in the brain, heart attack.

**The outcome:** chronic renal failure.

**4.** **Necrotic nephrosis.**

The kidney is enlarged, swollen and edematous, fibrous capsule is tense and easily removed. In the cut a wide cortical substance is anemic, pale gray, separated from the dark -red pyramids. In the intermediate zone and renal pelvis are observed hemorrhage.

**Reasons:** **ischemic -** a sharp decrease in blood pressure, decrease in blood volume associated with blood loss or dehydration (prolonged vomiting, profuse diarrhea, burns, prolonged use of diuretics), renal artery stenosis. **Toxic** - heavy metals, drugs, severe infection, massive hemolysis, "crash" syndrome, endogenous intoxication, snake bites and insect.

**Complication:** uremic coma.

**Outcomes:** restoration of the structure and function of the kidneys; acute renal failure; nephrosclerosis and chronic renal failure.

**5. Lipoid nephrosis.**

Kidneys greatly enlarged, flabby, the capsule can be easily removed. In a cut wide kidney bark, pale yellow or pale gray, gray-red pyramid - the "big white kidney".

**Cause:** unknown, but sometimes develops after a respiratory infection or after prophylactic immunization.

**Outcomes:** relatively favorable; nephrotic syndrome.

**6. Renal amyloidosis.**

The kidneys are increased in size, dense, waxy. In a cut a cortical substance is wide, matte, and medulla is gray-pink - "big greasy bud" or "large white kidney".

**Causes:** a) **congenital** genetic amyloidosis is fermentopathy, b) **primary** amyloidosis at myeloma: tumor plasma cells synthesize a low molecular weight abnormal protein that fills the stroma of the kidneys and leads to atrophy of the renal parenchyma, c) causes of **secondary** amyloidosis - chronic infectious disease with purulent destructive processes and with the disintegration of own tissues, that leading to a deep intoxication and violation of general protein metabolism. It is a complication of tuberculosis, chronic suppurative osteomyelitis and bronchiectasis.

**Complications:** infection (pneumonia, erysipelas, mumps), infarcts, hemorrhage, cardiac failure.

**The outcome:** acute or chronic renal failure.

1. **Purulent pyelonephritis**.

Kidney enlarged, swollen, with hyperemia, thickened capsule is easily removed. On the surface of the kidney are observed subcapsular small abscesses. In a cut the renal parenchyma motley - gray and yellow areas of necrosis and suppuration, hemorrhage. Cavities pelvis and cups expanded, in the its lumen - cloudy urine with pus. The mucous membrane of the pelvis dim, with foci of hemorrhage, necrosis, and fibrin gray coating.

**Reasons:** infection - most commonly E. coli, Proteus, Enterococcus, Streptococcus, and others.

**Complications:** carbuncle kidney (at the confluence of major abscesses or occlusion of large vessels by septic emboli), pyonephrosis (breakthrough of pus from abscesses in the pelvis), perinephritis and paranephritis (propagation of purulent process in the capsule and perirenal fat), papillonekrosis (in elderly diabetics at urinary stasis).

**Outcomes:** acute renal failure.

1. **Kidney stones (nephrolithiasis).**

The kidney is enlarged, pale. In a cut the kidney parenchyma is thinned, cups and pelvis expanded, filled with stones pale yellow, coral-shaped.

**Causes** 1**. General:** a violation of mineral metabolism, purine metabolism, diet (mineral composition of drinking water, the predominance of carbohydrates and animal protein in the diet) - endemic nephrolithiasis.

2**. Local:** - dyskinesia of the urinary tract,

- Inflammation of the urinary tract,

- Stagnation of urine.

**Complications:** hydronephrosis, pyelonephritis, pyonephrosis, urosepsis. **Outcomes:** acute and chronic renal failure.

1. **Nodular hyperplasia of the prostate gland.**

The prostate gland is enlarged, to the greatest degree medium share, who eminent in the lumen of the urethra and bladder neck. Surface of the gland is knobby, texture is elastic-dense, nodes is well-demarcated, various sizes, yellow and pink color. At the cut of gland flow down milky white prostatic fluid.

**Causes** associated with a progressive increase in the concentration of serum 17β-estradiol and estrone, formed due to the metabolic conversion from androstenedione and testosterone in men over 50 years.

**Complications:** compression and deformation of the urethra and bladder neck, cystitis, pyelitis, pyelonephritis, hydrouretra, hydronephrosis, obstruction of urinary outflow, rarely - anuria with the development of acute renal failure.

**Outcome:** favorable, extremely rare - malignancy.

Test control

Select one or more correct answers

1. Diseases leading to the development of primarily contracted kidney

1) Glomerulonephritis

2) pyelonephritis

**3) hypertension**

**4) Atherosclerosis**

2. Outcomes on amyloid nephrosis

1) recovery

2) heart failure

**3) uremia**

4) death of autoinfection

3. GLOMERULONEPHRITIS CHARACTERISTIC

**1) bilateral renal damage**

**2) the primary lesion glomeruli**

3) unilateral renal damage

4) purulent inflammation

**5) non purulent inflammation**

6) predominant involvement of the interstitial tissue

4. LOCATION IN characteristic changes at the membranous glomerulonephritis

1) proximal tubules

2) the distal tubules

**3) the basement membrane of the tubules**

4) basement membrane glomerular capillaries

5. Morphological signs of acute nephrosis

1) tubular atrophy

2) hyalinosis glomeruli

**3) necrosis of the tubular epithelium**

**4) the formation of cylinders**

6. The disease characterized by the formation of glomerular lunate

1) membranous nephropathy

2) lipoid nephrosis

**3) subacute glomerulonephritis**

4) acute post-streptococcal glomerulonephritis

7. Macroscopic picture of subacute glomerulonephritis

**1) great spotted kidney**

2) a large white kidney greasy

3) primary contracted kidney

4) kidney with necrosis papillae of the pyramids

8.POSSIBLE COMPLICATIONS of chronic glomerulonephritis

1) anemia

**2) chronic renal failure**

3) hyperglycemic coma

**4) brain hemorrhage**

**5) cardiovascular failure**

9. CHANGES IN PRODUCTIVITY extracapillary glomerulonephritis

**1) protein dystrophy tubular epithelium**

**2) necrosis of the glomerular capillary loops**

**3) fibrin in the lumen of the capsule glomerulus**

**4) proliferation nephrothelial and podocyte to form lunate**

5) nodules Kimmelstilya-Wilson

10. Changes arise in the kidney with chronic glomerulonephritis

1) thrombosis, necrosis of the glomerular loops

2) cell proliferation in renal corpuscles

**3) glomerular sclerosis and hyalinosis**

4) fibrinopurulent hemorrhagic exudate

**5) infiltration in the stroma of histiocytes, and plasma cells**

11. Macroscopic picture of amyloidosis kidney

1) Great Spotted kidney

**2) a large white kidney greasy**

3) The big red kidney

4) Kidney with the foci of purulent inflammation

12. The disease with development of secondary nephrotic syndrome

**1) renal amyloidosis**

**2) diabetic nephropathy**

3) membranous nephropathy

4) focal segmental glomerular hyalinosis

**5) lupus nephritis**

13. The outcome in necrotic nephrosis

**1) recovery**

**2) acute renal failure**

**3) chronic renal failure**

4) primary contracted kidney

14. Stages of acute renal failure

1) uremic

**2) shock**

**3) restoration of diuresis**

4) nephrotic

**5) oligoanuria**

15. The disease with development of primary nephrotic syndrome

1) renal amyloidosis

2) diabetic nephropathy

**3) membranous nephropathy**

**4) focal segmental glomerular hyalinosis**

5) lupus nephritis

16. SIGNS of the Alport syndrome

**1) hereditary disease**

2) acquired disease

**3) Glomerulopathy**

4) tubulointerstinalnoe disease

17. Contributing factors in the development of pyelonephritis

**1) obstruction of the urinary tract**

**2) vesicoureteral reflux**

3) hypertension

**4) Pregnancy**

5) atherosclerosis

**6) diabetes**

18. Signs of chronic pyelonephritis

1) symmetrically evenly contracted

**2) asymmetric uneven contracted**

**3) lymphohistiocytic infiltration, sclerosis of the stroma and periglomerular sclerosis**

**4) cystic tubular atrophy with the advent of lumens in their dense eosinophilic masses**

5) glomerular sclerosis and hyalinosis.

19. Histological forms of the PROSTATE CANCER

1) squamous

**2) solid**

**3) high-grade carcinoma**

**4) anaplastic carcinoma**

**5) transitional cell**

20. Basic morphological signs of acute pyelonephritis

**1) leukocyte infiltration interstitial**

2) degenerative changes in tubular epithelium

3) protein cylinders in the tubules

21. GROWTH IN NODES at the nodular prostatic hyperplasia starts at

1) The posterior lobe

**2) preprostatic area**

3) the prostatic urethra

4) anterior lobe

22. Macroscopic characteristics of nodes at the nodular prostatic hyperplasia

**1) Yellow-pink**

**2) soft consistency**

3) foci of hemorrhage on the cut

**4) with the cut surface prostatic fluid flow down**

23. KIDNEY SYMPTOMS PYELONEPHRITIS

1) oligouriya

2) hematuria

**3) leukocyturia**

**4) bacteriuria**

5) dysuria

6) pain

24. The relevant factor in the development of pyelonephritis

1) megauretra

**2) stricture of the urethra**

**3) purulent cystitis**

4) chronic tonsillitis

**5) sepsis**

25. The etiology of acute pyelonephritis

1) immune complexes

2) viruses

**3) gram-negative bacteria**

4) Gram-positive bacteria

5) hyperoxaluria

26. The conditions that predispose to DEVELOPMENT of nephrolithiasis

1) sickle cell nephropathy

**2) hyperparathyroidism**

**3) gout**

4) amyloid nephropathy

**5) hyperoxaluria**

27 bladder tumors

**1) papilloma**

2) condyloma

**3) adenocarcinoma**

**4) transitional cell cancer**

**5) leiomyoma**

28. Complications of chronic pyelonephritis

1) perinefrit

2) recovery

**3) bleeding in the brain**

**4) uremia**

29. Tubulointerstitial nephritis caused immune disorders and associated with antibody and glomeruli were observed at

**1) syndrome Gudspachera**

2) Albright syndrome

3) intoxication

30. Pathological processes in the kidney with acute pyelonephritis

1) serous exudation into the lumen of the glomerular capsule

**2) multiple abscesses**

**3) fibropurulent a pyelitis**

4) fibrinous exudate in the lumen of the glomerular capsu

**theme 8: DISEASEs of female genital organs. DISEASEs of uterus. Preneoplastic diseases and tumors of the cervix and uterus. DISEASEs of ovaries, fallopian tubes and mammary gland.**

CONTROL QUESTIONS

1. **Diseases of the cervix.** Acute and chronic cervicitis. Endocervical polyps. Epidemiology, etiology, risk factors, morphogenesis, morphological characteristics, clinical manifestations, outcomes.
2. **Cervical cancer.** Epidemiology, risk factors, diagnostic methods, precancerous conditions. Cervical intraepithelial neoplasia and invasive cervical cancer. Classification, morphological characteristics, clinical manifestations, prognosis.
3. **Diseases of the body uterus and endometrium**. Classification: inflammatory, dyshormonal and neoplastic. Risk factors.

**a) acute and chronic endometritis**. Morphological characteristics and clinical manifestations.

**b) Adenomyosis. Endometriosis**. Morphological characteristics, theories, clinical manifestations and clinical significance.  
**c) Glandular endometrial hyperplasia**. The classification, causes, morphological characteristics, the prognosis (risk of malignancy).

**d) tumors of the uterus body**. Classification. Benign tumors of the epithelium. Endometrial polyps. Clinical and morphological characteristics.

1. **Cancer of the endometrium**. Epidemiology, predisposing factors, classification. Macroscopic characteristics, histological forms, regularity of metastasis, prognosis.
2. **Mixed and mesenchymal tumors**. Fibromioma. Morphological characteristics, classification. Leiomyosarcoma. Malignant mixed mesodermal tumors. Endometrial stromal tumors. Clinical and morphological characteristics, prognosis.
3. **Tumors of the of ovaries**. Risk factors. Classification. Features histogenesis. Benign and malignant tumors of the superficial epitheliaum, the genital and germ cells, ovarian stroma. Ovarian cancer: morphological characteristics, prognosis. Metastatic lesions of the ovaries (Krukenberg tumor).
4. **Fibrocystic changes mammary glands**. Breast tumors. Classification. Benign tumors. Fibroadenoma. Leaf-shaped tumor. Intraductal papilloma. Morphological characteristics, clinical significance.
5. **Mammary cancer**. Epidemiology, risk factors and pathological morphogenesis, morphological types and morphological characteristics, clinical manifestations. Stage distribution by TNM. Prognosis and long-term outcomes.

The practical part of the subject:

Slides: In the study **micropreparations** pay attention to the education elements, designated by the letters in parentheses.

1. **Glandular hyperplasia endometrium.** H & E stain. In the endometrium amounts of iron is increased, they have different size and configuration (a), the epithelium of proliferative type, multirowed, sometimes epithelium formed to papillary outgrowths into the lumen of the glands  (b), in the stroma histiocytic and lymphocytic infiltrates (c).
2. **Fibroadenoma of the breast.** H & E stain. Glands ducts extended and lined by multilayer multirowed epithelium (a), are observed the glandular tumor formation (b), surrounded by connective tissue (c).
3. **Acute septic endometritis.** H & E stain. In the endometrium are observed epithelial desquamation (a), areas of necrosis (b), the deposition of fibrin (c), leukocyte infiltration (d). The myometrium is infiltrated leukocytes (e), vein is obturated by thrombi (f).
4. **Endocervicoses (cervical ectopia of the cervix).** H & E stain. In the mucosa of the exocervix are observed papillary growths of high prismatic epithelium (a), subiculum with glandular structures (b) and thin-walled blood vessels (c). Transition boundary stratified squamous epithelium in a prismatic epithelium (g) are observed.
5. **Adenocarcinoma of the endometrium.** H & E stain.  
   In the endometrium are determined atypical endometrial glandular complexes of different sizes and shapes (a), constructed of polymorphic cells endometrioid type (b), epithelium is multirowed, its polarity is broken (c), the nucleus is large, hyperchromatic, with the presence of mitosis (d).
6. **Squamous cell carcinoma of the cervix.** H & E stain.  
   In the cervical tissue are determined a solid structure, built of polymorphic polygonal cells with large hyperchromatic nuclei (a). In the tumor tissue are visible necrosis (b) and inflammatory foci (c).
7. **Scirrhous breast cancer.** H & E stain.

a- strands of tumor cells, b - the stroma

**Macropreparations:**

**1.Cancer of the endometrium.**  
In preparation of the uterus with appendages, uterus is increased to the size of a 16-week pregnancy. In the bottom are visible tumor site, exophytic growing into the cavity, irregular and rounded form, motley appearance, soft texture, 2 cm in diameter. On the cut tumor invades the uterine wall.  
**The reasons:** infringement of hormonal background, the influence of carcinogenic factors.

**Complications and outcomes**: sprouting in adjacent organs, metastasis to the lymph nodes.

**2.Cervical cancer.**  
In preparation of the uterus with appendages, normal size, the cervix is deformed in a "cauliflower".  
**Reason:** infringement of hormonal background, the influence of carcinogenic factors, chronic cervicitis with metaplasia and dysplasia.  
**Complications and outcomes:** sprouting the bladder, colon, metastasis to the lymph nodes.

**3.Mammary cancer.**  
In preparation of the mammary gland, on the cut is determined by the tumor site dense gray with indistinct borders sprouting surrounding tissue.  
**Reason:** infringement of hormonal background, the influence of carcinogenic factors.  
**Complications and outcomes:** metastasis to regional lymph nodes, later hematogenous metastases.

**4.Serous ovarian cyst.**  
In preparation of the ovary, it increased in size due to the thin-walled cavity formation, spherical shape, measuring 5 cm filled with a clear liquid. In a cut the internal cyst smooth. Ovarian parenchyma atrophy.  
**Reason:** infringement of hormonal background.  
**Outcomes:** the gap with the development peritonitis, hemoperitoneum.

Test control

Select one or more correct answers

1.TERM INTERNAL "adenomyosis" means  
1) heterotopic pancreatic tissue site, located in the muscular layer of the gastrointestinal tract  
**2) tissue complexes consisting of glandular and stromal elements in the myometrium without signs of tumor growth**3) the growth of ectopic endometrial elements

2. Physiological proliferative changes in the endometrium  
1) cystic expansion of the lumen with hyperplasia of glandular epithelium  
**2) the presence of tubular glandular crypts with high dark cell epithelium**3) the presence of sawtooth glandular crypts with a flattened cuboidal epithelium  
4) rejection of menstrual endometrium  
5) basal cell hyperactivity

3. LAYERS endometrial a mature woman  
**1) Functional**  
2) intermediate  
**3) basal**  
  
4. PHASE ovarian-menstrual cycle in childbearing years  
**1) follicular  
2) progestin**  
3) secretory  
**4) luteal**  
  
5. Diffuse desquamation glandular epithelium with hemorrhagic infiltration of the stroma observed at  
1) ovulatory endometrium hyperemia  
2) acute viral endometritis  
3) Botkin's disease  
**4) rejection of menstrual endometrium**  
5) all of the above is true

6. CHARACTERISTICS OF CERVICAL leukoplakia  
1) violation of the maturation of the epithelium with a predominance of immature cell forms  
**2) an increase in the differentiation of cellular elements with a tendency to keratinization squamous epithelium cells**  
3) the appearance of glandular structures in the ectocervix  
4) papillomatous proliferation of the squamous epithelium

7.The characteristics of the true cervical erosion  
1) an increase in the differentiation of cellular elements with a tendency to keratinization squamous epithelium  
**2) destruction of the epithelium with inflammatory infiltration**  
**of the underlying tissue**

3) The proliferation of backup squamous cells  
4) retention cysts of the cervix

8. Histological features of atypical endometrial hyperplasia  
1) severe atrophy glands in combination with increased proliferative activity of the epithelium part of the gland  
**2) marked proliferation of glands with a change in their pattern ("gland in gland") and the appearance of papillary structures**3) in some epithelial cells are observed tumor polymorphism  
4) the predominance of stromal component

9. The more common ovarian tumors  
**1) Benign**2) malignant  
  
10. Epithelial ovarian tumors  
**1) serous  
2) mucinous  
3) endometrioid**4) fibroma  
**5) Brenner tumor**

11. Stomach cancer metastases in the ovary  
1) Brenner tumor  
**2) tumor Krukenberg**  
3) tumor Paget  
4) Leydig tumor

12. FREQUENTLY histological forms of cancer endometrial  
1) squamous cell carcinoma  
**2) adenocarcinoma**  
3) scirrhoma (fibrous)

13. The basic theory of endometriosis  
**1) The theory of regurgitation  
2) the theory of metaplasia  
3) The theory of hematogenous and lymphatic spread**4) the anomaly of the uterus  
  
14. Cyst at the endometriosis  
1) follicular  
2) luteinized  
**3) "chocolate"**  
4) common  
5) serous  
  
15. Benign tumors of the corpus uteri  
**1) fibroma**  
2) papilloma  
3) chondroma  
4) ganglioma

16. Endometrial cancer T4, ACCORDING TNM system

1) carcinoma in situ

2) the tumor is within the uterus

**3) sprouting to the wall of the bladder, rectum**

4) the tumor grows into the myometrium

17. The most common tubal pathology

**1) salpingitis**

2) Brush

3) tumor

4) malformations

18. Benign breast disease

**1) fibroadenoma**

**2) leaf-formed tumor**

**3) intraductal papilloma**

4) carcinoma «in situ"

19. SELECT invasive forms of breast cancer:

1) intraduct cancer

2) lobular carcinoma in situ

**3) medullary cancer**

**4) mucinous carcinoma**

**5) tubular cancer**

20. What is Paget's disease:

**1) superficial cancer of the nipple and areola of the breast**

2) lobular carcinoma in situ

3) medullary cancer

4) mucinous carcinoma

**theme 9: Pathology of pregnancy, postpartum and placenta.**

**Pre- and postnatal disorders. Congenital defects**

CONTROL QUESTIONS

**1. Pathology of pregnancy**. Spontaneous abortion. Epidemiology, causes, features of the morphological study. Ectopic pregnancy. Classification. Reasons morphological diagnosis, complications and outcomes. Causes of death.

**2. Placenta:** morpho-functional characteristics. The main types of pathological processes:  
a) infectious processes, ways of infection of the placenta and fetus. Etiology, morphological manifestations, effects on the fetus and the mother, outcomes.

b) the types of circulatory disorders: fibrinoid deposition. hematoma, infarction, thrombosis, fetal vessels. Etiology, features of morphogenesis, morphology and clinical significance.  
c) abnormalities of placental disk and placental localization. Classification, morphological characteristics, clinical significance.  
d) placenta of twins: classification, clinical significance. Placental transfusion syndrome. Pathology of the umbilical cord.

**3. Toxemia of pregnancy (gestosis).** Classification, Epidemiology. Clinical manifestations, causes, pathogenesis, morphological characteristics. Effect on the fetus. The causes of death of women.

**4. Trophoblastic disease.** Classification. Vesical drift, invasive hydatidiform mole, chorionepithelioma. Trophoblastic tumor of placental bed. Epidemiology, morphological characteristics. Clinical manifestations, prognosis.

**5. Fetal age and weight of the fetus**. Periods of development of the fetus and newborn.

**6. Perinatal pathology.** Prematurity and postmaturity. Intrauterine growth retardation of fetal growth. Causes, clinical and morphological characteristics, prognosis.

**7. Congenital malformations.** Frequency, etiology and pathogenesis. Classification. Types of teratogens, and features of their impact on the organs of the fetus. Malformations multifactorial etiology.

**8. Diseases and malformations** characteristic of individual periods: chromosomal and genetic diseases.

**9. Hemolytic disease of the newborn.** Etiology, pathogenesis. Clinical and morphological forms and manifestations. Prognosis.  
**10. Mucoviscidosis.** Etiology and pathogenesis. Pathologic characteristics of lesions of the pancreas and other organs. Complications and outcomes.

**11. Pneumopathy. The notion of respiratory distress syndrome** and its causes. Classification. Hyaline membrane disease, clinical and morphological characteristics. Other pneumopathy. Complications and outcomes.

**12. Birth trauma:** contributing factors and their causes. Birth tumor. Cephalohematoma. Hemorrhage (epidural, subdural, the adrenal glands in the brain and spinal cord).

The practical part of the subject:

Slides: In the study **micropreparations** pay attention to the education elements, designated by the letters in parentheses.

1. **Vesical drift.** H & E stain.

Placental villi significantly increased in size with a sharp swelling and formation of cavities filled with fluid (a), there is a marked proliferation of syncytiotrophoblast (b).

1. **Chorionepithelioma of uterus.** H & E stain.

In the tumor tissue there are cells of cytotrophoblast with the absence of true villi, stroma and vessels (a), are defined polymorphic atypical huge elements of syncytiotrophoblast (b) and foci of hemorrhage (c).

1. **Tubal pregnancy.** H & E stain.

In the mucosa of the fallopian tube there is decidua tissue (a), chorionic villi, which penetrate into the thickness of muscular layer (b), the extravillous trophoblast cells (c), fibrinoid (d).

1. **Abortion in scraping.** H & E stain.

Placental tissue is represented by chorionic villi (a) and decidua tissue (b).

1. **The pancreas in mucoviscidosis..** H & E stain.

Terminations departments of pancreas and small ducts are dilated (a), in the lumen of glands and ducts copious amounts of layered secret (b). Glandular parenchyma is atrophic (c), in storoma there is proliferation of fibrous tissue (d) with infiltrating lymphocytes, plasmocytes and histiocytes (e).

1. **Kernicterus hemolytic disease.** Painting on Schmorl.

The brain tissue is determined by acute swelling of neurons with conversion cells in a "shadow" (a) pronounced proliferation of oligodendroglia (b), the concentration of indirect bilirubin in neurons (c) and small vessels (d) in glial cells (e) and myelinated fibers (f).

1. **Acute venous hyperemia the lung**. H & E stain.

The capillaries and arterioles of the lung expanded and full-blooded (a), in the lumen of alveolar there is accumulation of edema fluid (b), and a few red blood cells (c).

1. **Bleeding in the brain.** H & E stain.

In the brain tissue there is vascular hyperemia (a), diapedetic perivascular hemorrhage (b) pericellular (c) and perivascular edema (d).

**Macropreparations:**

**1. Tubal pregnancy**: In preparation fallopian tube as expanded in ampullar department up to 1.5 cm, on the cut is determined by fetal egg with a massive hemorrhage.

**Causes:** chronic inflammatory diseases of the uterus, the development of adhesions and narrowing of the lumen of the fallopian tubes. Violation of peristaltic tube and narrowing of the lumen at the infantilism. Structural pathology tubes and hypoplasia of the corpus luteum of the ovary. Malformations of the uterus, and uterine hormonal contraception.

**Complications:** bleeding, tubal abortion, rupture of the fallopian tubes.

**The outcome:** abortion in term of 5-6 weeks.

**2. Ovarian pregnancy:** Ovary dramatically increased in size, there is damage to the parenchyma in the form of hemorrhage, on the cut in the thickness is determined by fetal egg. **Causes:** a structural abnormality of the fallopian tubes. **Complications:** internal bleeding, peritonitis. **Outcome:** ovarian apoplexy.

**3.** **Chorionepithelioma of uterus.** The uterus is enlarged, mucous membrane thickened, in the uterus is determined tumor on a broad basis in the form of juicy motley cancellous node, sprouting in the myometrium. **Causes:** pregnancy complicated vesical drift, after abortion, after ectopic pregnancy, after clinically normal pregnancy.

**4. Vesical drift:** The uterus is enlarged in the cavity acinar accumulations consisting of multiple cystic formations to 1 cm in size, filled with a clear liquid. The fetus is missing. **Reasons:** 1) partial vesical drift - predominance fetal karyotype paternal chromosomes, 2) complete vesical drift - chromosome set of the sperm is doubled and egg cell nucleus is inactivated or killed. **Complications:** bleeding in the I trimester, choriocarcinoma. **Outcome:** incomplete pregnancy.

**5. Hypoplasia of the kidneys.** a) For one-sided defeat: the kidney is reduced in size, its surface is lobed. **Reasons:** teratogenetic period - up to 8 weeks. It occurs as an isolated defect and match. It occurs as an isolated defect and combined. **Complications** at isolated malformation: growth retardation, renal rickets, proteinuria, hypertension. **The outcome:** chronic renal failure.  
b) In bilateral lesions: the size and weight of the kidneys is reduced by 1/3, and a decrease in the number of cups - 5 or less (normal 8-10). **Outcome** – unfavorable.

**6. Anencephaly** is absence of the brain, cranial vault bones and soft tissues. The preparation of premature infants, on the site of the brain is the connective tissue with cystic cavities and blood vessels. The bones of the skull are absence. **The reasons:** the simultaneous action of certain environmental factors, teratogenic period - up to 8 weeks of fetal development. Accompanied by the adrenal hypoplasia and aplasia neurohypophysis. **Outcome:** intrauterine fetal death or in the first days of life.

**7. Craniocerebral hernia** - hernial protrusion in the defect of the skull bones. Reasons: teratogenetichesky period up to 4 months of fetal development (infections, drugs, metabolic and embryo-fetopathy).  
**Localization:** a) between the frontal bone, b) at the root of the nose, and c) between the parietal and temporal bone, d) at the junction of the parietal bones and the occipital bone, etc.) near the inner corner of the eye.

**Forms:** 1) meningocele - hernial sac presented dura mater and leather, and the contents of the cerebrospinal fluid. 2) meningoencephalocele - in the hernial sac sticks out one or another part of the brain. Outcome: large hernia lead to brain disorders and fetal death.

Test control

Select one or more correct answers

1. The most frequent localization of ectopic pregnancy  
1) ovaries  
**2) the fallopian tubes**  
3) abdomen  
4) the cervix  
5) vagina

2. The frequency of spontaneous abortion  
1) 5-10%  
**2) 10-20%**  
3) 30-40%  
4) 40-50%  
5) 50%

3. Morphologically eclampsia is characterized   
**1) systemic fibrinoid necrosis of small vessels  
2) disseminated thrombosis of small vessels  
3) necrosis and hemorrhage in the organs**  
4) suppurative metastases  
5) vomiting

4. The reasons for late GESTOSIS  
**1) violation of trophoblast invasion into the myometrium**2) cystic degeneration of placenta  
**3) The pathology of the uterine spiral arteries**  
4) calcification of the placenta  
**5) the allocation of the ischemic placenta tromboplastic substances**

5. SIGNS OF INVASIVE vesical drift  
**1) lung metastases**  
2) tumor growth  
**3) transformation of cystic villi  
4) the proliferation of trophoblast**  
**5) swollen villi in the vessels of the myometrium**

6. SIGNS OF infarction OF THE PLACENTA  
**1) a sharp narrowing of the intervillous space**  
**2) narrowing of blood vessels in the villi**3) hemorrhagic impregnation of placenta  
**4) infiltration of polymorphonuclear leukocytes**  
5) calcification

7. CAUSES PLACENTAL hematoma  
1) thrombosis of the spiral arteries  
2) infarction  
**3) placental abruption**  
4) breaking of spiral arteries  
**5) breaking of arterioles decidua**

8. SIGNS OF THROMBOSIS of fetal ARTERY villi

**1) fibrosis and obliteration of small vessels  
2) fetal a bleeding disorder**  
3) bleeding in intervillous space  
**4) intervillous space free**  
5) the umbilical sepsis

9. Trophoblastic disease  
1) **Vesical drift**  
2) placental transfusion syndrome  
3) adenocarcinoma  
**4) invasive vesical drift**  
**5) choriocarcinoma**

10. Morphological signs of hemolytic disease  
1) arteritis and phlebitis umbilical vessels  
2) brown atrophy of the liver  
**3) hemolytic jaundice  
4) anemia and edema  
5) kernicterus**

11. Morphological characters intrauterine hypoxia  
1) thrombotic complications  
**2) diapedetic hemorrhage and edema  
3) aspiration of amniotic fluid**4) depression of the respiratory center of the brain  
**5) meconium in the amniotic fluid**

12. Morphological characters of postmaturity   
1) calcification of the placenta  
**2) absence of lubrication   
3) dry skin with maceration  
4) the appearance of ossification centers in the proximal epiphysis of the tibia and humerus**  
5) loss of the umbilical cord

13. Morphological manifestations of mucoviscidosis  
1) hemorrhagic syndrome  
**2) retention cysts of pancreas and other organs  
3) secondary fibrosis**  
4) jaundice  
**5) cirrhosis**

14. morphological signs of prematurity  
**1) the absence** **of ossification centers in the epiphysis**  
2) imperforate fontanelles  
**3) soft skull bones**  
4) the absence of vellus hair, face, shoulders, back,  
**5) underdevelopment of the nail plate**

**Topic 10.Final lesson on lecture and theoretical material on the module "Private pathological anatomy".**

**Form(s) of the current control of academic performance(testing, interview, diagnosis of macropreparation and micropreparation).**

**Evaluation materials of the current control of academic performance**

**Test tasks**

**Test tasks for the current classes.**

1. Anemia. Definition and classification. Acute and chronic anemia due to blood loss (posthemorrhagic): causes, clinical and morphological characteristics, diagnosis.

2. Anemia due to increased blood loss (hemolytic): hereditary, acquired, autoimmune, isoimmune, mixed genesis. Classification, pathogenesis, diagnosis, clinical and morphological characteristics, causes of death. Hypersplenism.

3.Anemia with insufficient reproduction of red blood cells (dyserythropoietic). Classification. Anemia megaloblastic (B12 - and folic-deficient), pernicious, iron-deficient, with iron metabolism disorders, hypoplastic and aplastic. Etiology, pathogenesis and morphogenesis, clinical and morphological characteristics and diagnostic methods, complications, causes of death. Diseases and conditions accompanied by anemia.

4. Tumors of hematopoietic tissues (leukemias). Classification, etiology of leukemias, chromosomal and antigenic rearrangements. General characteristics. Membrane cell antigens-markers of differentiation of tumor cells and cytogenetic variants of leukemias.

5. Acute leukemias: lymphoblastic and myeloblastic. Modern diagnostic methods, clinical and morphological characteristics, complications. Drug pathomorphosis, age characteristics, causes of death.

6. Chronic myelocytic leukemia:

a) chronic myeloid leukemia, diagnostic signs, stages, morphology.

b) true polycythemia, myelofibrosis, thrombocytemia: diagnostic methods, clinical and morphological characteristics, causes of death.

7. Chronic lymphocytic leukemias:

a) chronic lymphocytic leukemia, diagnostic methods, clinical and morphological characteristics. Modern methods of treatment: bone marrow transplantation.

b) tumors from plasma cells (paraproteinemic). General characteristics. Classification: monoclonal gammapathy of unknown nature, multiple myeloma, plasmocytoma, Waldenstrom's macroglobulinemia, Franklin's heavy chain disease. Modern diagnostic methods, etiology, pathogenesis, morphological characteristics, clinical manifestations, prognosis, causes of death.

8. Hodgkin's disease (lymphogranulomatosis): clinical stages, pathohistological types, morphological characteristics and diagnostic methods, clinical manifestations, prognosis, causes of death.

9. Non-Hodgkin's lymphomas. General characteristics, localization, forecast, typing, and classification. Immunohistochemical markers, cell types in non-Hodgkin's lymphomas. Tumors from T-and B-lymphocytes: types, morphological characteristics, immunophenotypic variants, cytogenetic and molecular genetic markers, clinical manifestations, prognosis, causes of death.

10. Thymus (thymus gland). The concept of thymus hyperplasia, thymitis, tumors. Changes in the thymus in immunogenesis disorders. Age-related and accidental involution( transformation), hypoplasia of the thymus. Thymomegaly as an expression of innate immune deficiency.

11. Atherosclerosis and arteriosclerosis. Epidemiology, etiology, risk factors. Modern ideas about the pathogenesis of the disease.

12. Characteristics of macroscopic changes and morphogenetic stages of atherosclerosis, the structure of atherosclerotic plaque. The main clinical and morphological forms of atherosclerosis, their manifestations, complications and outcomes. Arteriosclerosis (mediacalcinosis) Menckeberg, morphological characteristics.

13. Hypertension (essential hypertension). Benign and malignant forms of arterial hypertension. The concept of a hypertensive crisis. Prevalence, etiology, and pathogenesis. Symptomatic hypertension.

14. Benign hypertension, stages, pathogenesis. Clinical and morphological forms: brain, cardiac, renal, hypertensive retinopathy. Morphological changes in blood vessels (hyaline and hyperplastic arteriolosclerosis) and in organs.

15. Malignant form of hypertension, clinical manifestations and morphological changes, complications, outcomes, causes of death.

16. Hypertensive heart disease. Myocardial hypertrophy. Chronic and acute pulmonary heart: causes of development, clinical and morphological characteristics. Congestive heart failure: etiology, pathogenesis and morphogenesis. Clinical and morphological characteristics of left ventricular and right ventricular failure.

17. Coronary heart disease (coronary heart disease). Concept, epidemiology, relationship with atherosclerosis and hypertension. Etiology and pathogenesis, risk factors, course, clinical and morphological forms.

18. Sudden coronary death. Causes of development. Pathogenesis, morphogenesis, and causes of death.

19. Angina pectoris: classification, clinical and morphological characteristics.

20. Myocardial infarction: definition of the disease, causes, classification, dynamics of biochemical and morphofunctional changes in the myocardium. Morphology of acute, recurrent, and recurrent myocardial infarction. Outcomes, complications, changes in thrombolytic therapy, causes of death.

21. Chronic ischemic heart disease: forms, clinical and morphological characteristics, complications, causes of death.

22. Cerebrovascular diseases. Etiology, association with atherosclerosis and hypertension. To define the concept of "stroke" and transient transient circulatory disorders of the brain. The most frequent localization of damage to the cerebral vessels.

23. Clinical and morphological forms of Cerebrovascular diseases:

a) Brain diseases with ischemic injuries: hemorrhagic and ischemic brain infarcts, ischemic encephalopathy. Morphology, causes, and outcomes.

b) Intracranial hemorrhages. Types, causes, mechanism of development, complications and outcomes.

c) Pathological anatomy of hypertensive cerebrovascular diseases.

1. 24.Diseases of the valve holes of the heart and great arteries: classification, functional disorders. Congenital and acquired heart disease: clinical and morphological characteristics.
2. 25.Endocarditis: classification, etiology, pathogenesis, morphological characteristics, complications, prognosis. Primary endocarditis (bacterial sepsis, endocarditis Leffler). Non-infectious nonbacterial thrombotic endocarditis. Endocarditis in rheumatic diseases (true rheumatism, systemic lupus erythematosus, rheumatoid arthritis). Carcinoid endocarditis.
3. 26. Diseases of the myocardium. Classification. Myocarditis. Definition, etiology. Patho- and morphogenesis, clinical and morphological characteristics, consequences, causes of death:

a) primary myocarditis Abramov - Fidler,

b) viral, microbial and parasitic myocarditis, infectious-allergic myocarditis,

c) myocardial diseases caused by toxic, metabolic and other impacts,

d) heart disease in pregnancy and childbirth, amyloidosis, excess iron, hyper- and hypothyroidism.

4. 27. Diseases of the pericardium. Pericarditis: classification, etiology, pathogenesis, clinical and morphological characteristics, outcomes. Hydropericardium, hemopericardium.

5. 28.Cardiomyopathy: classification. Primary cardiomyopathy, the value of genetic factors, pathological and morphogenesis, clinical and morphological characteristics of different forms, causes of death. Secondary cardiomyopathy etiology, pathogenesis, morphological changes of heart complications.

6. 29.Rheumatic diseases. Classification. General characteristics. Rheumatic fever: etiology, patho- and morphogenesis, the characteristic clinical and morphological forms, methods of diagnosis, clinical symptoms and syndromes forecast.

7. 30.Congenital heart disease. Etiology. Vices "blue" and "white" types. Congenital defects of the atrial and ventricular walls, arterial trunks of the heart (transposition, stenoses and anomalies in the mouths of the great arteries, aortic coarctation, patent ductus arteriosus), combined heart defects (the triad, tetrad, pentad Fallot). Clinical and morphological characteristics.

31. Acute inflammatory lung diseases. The role of lung homeostasis disorders in the development of pneumonia. Classification of pneumonia. Pneumonia in conditions of suppressed immunity. The concept of nasocomial infection, the causes of its occurrence.

32. Bacterial pneumonia. Classification. Focal pneumonia (bronchopneumonia). Etiology and pathogenesis, morphological features. Complications of focal pneumonia, outcomes.

33. Lobar (croup pneumonia). Etiology, pathogenesis, clinical and morphological features, stages of development, pulmonary and extrapulmonary complications, outcomes.

34. Acute interstitial pneumonitis (alveolitis). Viral and mycoplasma pneumonia. Clinical and morphological characteristics, outcomes.

35. Lung abscess. Classification, pathogenesis and morphogenesis, clinical and morphological characteristics, complications, outcomes. Acute and chronic abscesses.

36. Diffuse chronic lung lesions. Definition of the concept and classification. Chronic obstructive pulmonary diseases. General characteristics.

37. Chronic obstructive pulmonary emphysema-definition, classification, epidemiology, etiology, pathogenesis and morphogenesis, morphological characteristics, clinical manifestations, complications, outcomes, causes of death. Other types of emphysema (compensatory, senile, vicar, inter-daily): clinical and morphological characteristics.

38. Chronic obstructive bronchitis. Definition, classification, etiology, epidemiology, pathogenesis and morphogenesis, morphological characteristics, clinical manifestations, complications, outcomes.

39. Bronchiectatic disease. Definition, etiology, epidemiology, pathogenesis and morphogenesis, morphological characteristics, clinical manifestations, complications, outcomes.

40. Diffuse interstitial (infiltrative and restrictive) lung diseases. Classification, clinical and morphological characteristics, pathogenesis. Alveolitis. Morphological characteristics, pathogenesis. Idiopathic pulmonary fibrosis. Classification, etiology, pathogenesis and morphogenesis, stages and variants, clinical and morphological characteristics, prognosis.

41. Pneumoconiosis (anthracosis, silicosis, asbestos, berylliosis). Pathogenesis and morphogenesis, morphological characteristics, clinical manifestations, complications, causes of death.

42. Tumors of the bronchi and lungs. Epidemiology, principles of classification. Benign tumors. Malignant tumors. Lung cancer. Bronchogenic cancer. Epidemiology, etiology, principles of international classification. Biomolecular markers of lung cancer. Precancerous changes in the bronchi and lung. The concept of "cancer in the rumen". Clinical manifestations.

43. Gastritis. Definition. Acute gastritis. Etiology, pathogenesis, Clinical and morphological characteristics of various forms (catarrhal, erosive, phlegmonous, fibrinous, necrotic). Complications. Outcomes.

44. Chronic gastritis, the essence of the process. Etiology, pathogenesis. Classification principles. Forms identified on the basis of the study of gastrobiopsias, morphological characteristics. Complications, outcomes, prognosis. Chronic gastritis as a precancerous condition.

45. Peptic ulcer of the stomach and duodenum 12:

a) Definition. General characteristics of peptic ulcers of the stomach and duodenum 12. Epidemiology, etiology, pathogenesis, and morphogenesis, especially in pyloroduodenal and medio-gastric ulcers,

b) Morphological characteristics of chronic ulcers in the period of exacerbation and remission. Complications, outcomes.

46. Acute gastric ulcers: etiology, pathogenesis, morphological characteristics, complications, outcomes.

47. Diseases of the stomach of various etiologies. Gastropathy. Classification, morphological variants, clinical and morphological features. Outcomes. Varicose veins of the stomach. Causes, complications.

48. Stomach tumors. Epidemiology, etiology, principles of classification.

a) Hyperplastic (hyperplasiogenic) polyps. Gastric adenoma. Morphological characteristics.

b) Malignant tumors of the stomach. Stomach cancer. Precancerous processes. Macroscopic and histological forms. Features of lymphogenic metastasis.

49. Enterocolitis. Diarrhea syndrome: definition, main types, causes. Infectious enterocolitis. Necrotizing enterocolitis.

50. Pseudomembranous colitis. Etiology, pathogenesis, clinical and morphological characteristics, prognosis. Ischemic colitis. Causes, clinical and morphological manifestations. Complications, outcome.

51. Idiopathic inflammatory bowel diseases.

Non-specific ulcerative colitis. Crohn's disease. Epidemiology, etiology, pathogenesis and morphogenesis, morphological characteristics, clinical manifestations, complications, outcomes, prognosis. Criteria for the differential diagnosis of chronic colitis.

52. Diseases of the appendix of the caecum. Anatomical and histological features. Appendicitis. Classification, epidemiology, etiology, pathogenesis. Morphological characteristics and clinical manifestations of acute and chronic appendicitis. Complications. Features of the disease in children and the elderly. Tumors of the appendix. Classification, clinical and morphological characteristics, prognosis.

53. The role of the liver in the vital activity of the body. Characteristics of the main groups of pathological processes in the liver. Classification, epidemiology.

54. Hepatitis. Definition. Principles of classification: by current, etiology, origin, and morphology.

55. Viral hepatitis:

a) characteristics of etiological factors (A, B, C, D-types of viruses). Preferred routes of infection. Pathogenesis. Morphological changes in the liver: morphological cytolysis, cellular reactions, violation of bile formation and bile excretion,

b) morphology of acute forms of viral hepatitis: acute cyclic form, forms with massive necrosis, pericholangiolytic form, outcomes,

c) morphological manifestations of chronic forms of hepatitis, pathogenesis, outcomes.

56. Alcoholic liver damage. Alcoholic liver obesity. Alcoholic hepatitis. Alcoholic cirrhosis of the liver. Epidemiology, pathogenesis and morphogenesis, morphological characteristics, clinical manifestations, complications and causes of death, outcomes, prognosis.

57. Hepatoses. Definition. Etiology. Pathogenesis. Acute toxic liver dystrophy. The importance of sensitization in its development. The period of yellow and red dystrophy. Outcomes. Causes of death.

58. Chronic fatty hepatosis. Reasons. Clinical and morphological manifestations. Outcomes.

59. Cirrhosis of the liver. Definition. Etiology. Pathogenesis. Classification principles.

a) pathological anatomy of postnecrotic, portal, biliary and mixed cirrhosis of the liver.

b) the most important complications of portal hypertension and hepatic (hepatocellular) insufficiency. Hepatic encephalopathy. Jaundice. Kidney failure. Ascites and edema. Endocrine disorders. Circulatory disorders and infectious complications. Pathogenesis, clinical and morphological characteristics.

60. Liver tumors. Benign and malignant neoplasms. Epidemiology. Histogenesis. Macro-and microscopic picture. Patterns of metastasis.

61. Cholecystitis and cholangitis. Ways of infection penetration. The importance of stones in the development of cholecystitis. Types of acute cholecystitis (catarrhal, purulent, gangrenous). Chronic cholecystitis. Morphology. Complications. Tumors and congenital anomalies of the biliary tract. Classification. Clinical and morphological characteristics.

62. Diseases of the pancreas. Diseases of the exocrine part of the pancreas. Acute pancreatitis (pancreonecrosis) and chronic. Epidemiology, etiology, pathogenesis, morphological characteristics. Tumors of the exocrine part of the pancreas. Pancreatic cancer, classification, morphological characteristics, prognosis.

63. Glomerular kidney diseases. Glomerulonephritis. Modern classification, etiology, pathogenesis, immunomorphological characteristics. Acute glomerulonephritis. Post-streptococcal and non-streptococcal glomerulonephritis. Rapidly progressive glomerulonephritis. Etiology, pathogenesis, morphological characteristics, outcomes.

64. Chronic glomerulonephritis. Definition, macro-and microscopic characteristics. Uremia. Etiology, pathogenesis, clinical and morphological characteristics.

65. Nephrotic syndrome. Classification. Membranous nephropathy. Lipoid nephrosis. Focal segmental glomerulosclerosis. Etiopathogenesis, morphological characteristics. Electron microscopic differential diagnostic signs. Membranoproliferative glomerulonephritis. IgA-nephropathy. Focal proliferative and necrotizing glomerulonephritis. Etiopathogenesis, morphological characteristics.

66. Damage to the renal glomeruli associated with systemic diseases: Systemic lupus erythematosus. The Schenlein-Genoch purple. Bacterial endocarditis. Goodpasture's syndrome, essential cryoglobulinemia, plasma cell dyscrasia. Pathogenesis, clinical and morphological characteristics. Forecast. Amyloidosis of the kidneys. Diagnostic methods, clinical manifestations.

67. Hereditary nephritis. Epidemiology, classification, pathogenesis, morphological characteristics.

68. Kidney diseases associated with the defeat of the tubules and interstitium. Classification. Acute tubular necrosis (necrotic nephrosis). Etiology, pathogenesis, morphological characteristics, clinical manifestations, prognosis.

69. Tubulointerstitial nephritis. Classification, etiology, morphological characteristics, clinical manifestations, outcome. Tubulointerstitial nephritis caused by drugs and toxins. Analgesic nephropathy, pathogenesis, morphological characteristics.Development of practical skills (Study of macropreparations in the department's museums. Microscopic examination and drawing of histological preparations ).

70. Pyelonephritis and urinary tract infections. Definition, classification. Etiological and predisposing factors, ways of spreading infection in the kidneys. Acute pyelonephritis. Definition, etiology, predisposing diseases and pathogenesis, morphological characteristics, complications.

71. Chronic pyelonephritis and reflux nephropathy. Etiology, pathogenesis, morphological variants and morphological characteristics, clinical manifestations, outcomes.

72. Nephrolithiasis. General and local factors that play a role in stone formation. Pathogenesis and morphogenesis, clinical and morphological characteristics, outcomes. Urate nephropathy.

73. Kidney tumors. Epidemiology, predisposing factors, classification. Benign tumors: histogenesis, clinical and morphological characteristics, prognosis. Malignant tumors: renal cell carcinoma, urothelial carcinoma. Morphological characteristics, features of metastasis, clinical manifestations, prognosis.

74. Diseases of the bladder. Inflammatory and hyperplastic pathological processes and diseases, clinical and morphological characteristics. Tumors of the bladder. Classification. Morphological characteristics of benign transitional cell tumors, prognosis. Malignant epithelial tumors. Epidemiology, risk factors, etiology and morphogenesis, clinical and morphological characteristics of various histological types of cancer, prognosis. Mesenchymal tumors. Secondary tumor lesions.

75. Diseases of the male genitals. Diseases of the testicles and their appendages. Non-specific and specific inflammatory diseases of the testicles and their appendages. Tumors.

76. Diseases of the prostate gland. Classification. Inflammatory diseases. Prostatitis: acute bacterial, chronic. Etiology, morphogenesis, clinical and morphological characteristics, complications, outcomes.

77. Benign nodular hyperplasia of the prostate gland. Causes, clinical manifestations, histological variants, complications and outcomes.

78. Prostate tumors. Classification. Epidemiology, risk factors, causes, pathogenesis and morphogenesis. Prostate cancer, histological variants, molecular markers, clinical manifestations, complications, outcomes.

79. Diseases of the cervix. Acute and chronic cervicitis. Endocervical polyps. Epidemiology, etiology, risk factors, morphogenesis, morphological characteristics, clinical manifestations, outcomes. Cervical cancer. Epidemiology, risk factors, diagnostic methods, precancerous conditions. Cervical intraepithelial neoplasia and invasive cervical cancer. Classification, morphological characteristics, clinical manifestations, prognosis.

80. Diseases of the body of the uterus and endometrium. Classification: inflammatory, dyshormonal and tumor. Risk factors.

a) Acute and chronic endometritis. Morphological characteristics and clinical manifestations.

b) Adenomyosis. Endometriosis. Morphological characteristics, theories of occurrence, clinical manifestations and clinical significance.

c) Glandular endometrial hyperplasia. Classification, causes, morphological characteristics, prognosis (risk of malignancy).

d) Tumors of the uterine body. Classification. Benign tumors from the epithelium. Endometrial polyp. Clinical and morphological characteristics. Endometrial cancer. Epidemiology, predisposing factors, classification. Macroscopic characteristics, histological forms, patterns of metastasis, prognosis. Mixed and mesenchymal tumors. Fibromyoma. Morphological characteristics, classification. Leiomyosarcoma. Malignant mixed mesodermal tumors. Endometrial stromal tumors. Clinical and morphological characteristics, prognosis.

81. Ovarian tumors. Risk factors. Classification. Features of histogenesis. Benign and malignant tumors from the surface epithelium, from germ and germ cells (germinogenic), from the ovarian stroma (stroma of the genital cord). Ovarian cancer: morphological characteristics, prognosis. Metastatic ovarian lesions (Krukenberg tumor).

82. Fibrocystic changes and breast cancer. Breast tumors. Classification. Benign neoplasms. Fibroadenoma. Phelloid (leaf-shaped) tumor. Intra-flow papilloma. Morphological characteristics, clinical significance.

83. Breast cancer. Epidemiology, risk factors, pathogenesis and morphogenesis, morphological types and morphological characteristics, clinical manifestations. Stages of distribution by TNM. Prognosis and long-term results of treatment.

6. Pathology of pregnancy. Spontaneous abortions. Epidemiology, causes, features of morphological research. Ectopic pregnancy. Classification. Causes, morphological diagnosis, complications, and outcomes. Causes of death.

84. Placenta: morphofunctional characteristics. The main types of pathological processes:

a) infectious processes, ways of infection of the afterbirth and fetus. Etiology, morphological manifestations, effects on the fetus and the mother's body, outcomes.

b) types of circulatory disorders: fibrinoid deposition. hematoma, heart attack, fetal vascular thrombosis. Etiology, features of morphogenesis, morphology and clinical significance.

c) anomalies of the placental disc, localization and attachment of the placenta. Classification, morphological features, clinical significance.

d) twin placentas: classification, clinical significance. Placental transfusion syndrome. Pathology of the umbilical cord.

85. Toxicosis of pregnant women (gestosis). Classification, epidemiology. Clinical manifestations, causes, pathogenesis, morphological characteristics. Effect on the fetus. Causes of death of a woman.

86. Trophoblastic disease. Classification. Cystic drift, invasive cystic drift, chorioepithelioma. Trophoblastic tumor of the placental bed. Epidemiology, morphological characteristics. Clinical manifestations, prognosis.

87. Prenatal age and fetal weight. Periods of fetal and newborn development.

88. Perinatal pathology. Prematurity and portability. Delay in intrauterine development of fetal growth. Causes, clinical and morphological characteristics, prognosis.

89. Congenital malformations. Frequency, etiology, and pathogenesis. Classification. Types of teratogens and features of their impact on the organs of the fetus. Malformations of multifactorial etiology.

90. Diseases and malformations characteristic of individual periods of cymatogenesis: chromosomal and gene diseases.

91. Hemolytic disease of newborns. Etiopathogenesis. Clinical and morphological forms and their manifestations. Forecast.

92. Cystic fibrosis. Etiology and pathogenesis. Pathoanatomic characteristics of lesions of the pancreas and other organs. Complications and outcomes.

93. Pneumopathies. The concept of the syndrome of respiratory disorders and the causes of its occurrence. Classification. Hyaline membrane disease, clinical and morphological characteristics. Other types of pneumopathies. Complications and outcomes.

94.Birth trauma: predisposing factors and causes of their occurrence. A birth tumor. Cephalohematoma. Hemorrhages (epidural, subdural, in the adrenal glands, in the spinal cord and brain).

MICROPREPARATIONS

1. The liver in chronic myeloid leukemia.

2. The liver in chronic lymphocytic leukemia.

3. Lymphogranulomatosis of the lymph node

4. Hemosiderosis of the spleen.

5. Atherosclerotic plaque in the aorta.

6. Myocardium in hypertension.

7. Arteriolosclerotic kidney.

8. Diffuse cardiosclerosis.

9. Myocardial infarction.

10. Postinfarction large-focal cardiosclerosis.

11. Brain hemorrhage

12.Recurrent warty endocarditis.

13. Rheumatic myocarditis (granulomatous).

14. Rheumatic endocarditis.

15. Fibrinous pericarditis.

16. Chronic venous fullness of the liver.

17. Bronchopneumonia.

18. Croup pneumonia (stage of gray hepatica).

19. Lung abscess.

20. Bronchiectasis with the phenomena of pneumosclerosis.

21. Obstructive emphysema of the lungs.

22. Lung with silicosis.

23. Lung cancer (Squamous cell lung cancer with keratinization).

24. Chronic stomach ulcer in the period of exacerbation.

25. Chronic atrophic gastritis.

26. Gastric adenocarcinoma.

27. Gastric mucosal cancer.

28. Stomach skirr.

29. Ulcerative colitis.

30. Crohn's disease.

31. Phlegmonous appendicitis.

32. Chronic appendicitis.

33. Hepatitis.

34. Fatty hepatosis.

35. Toxic liver dystrophy.

36. Postnecrotic cirrhosis of the liver.

37. Biliary cirrhosis of the liver (secondary).

38. Muscat fibrosis of the liver.

39. Necrotic nephrosis.

40. Amyloid nephrosis.

41. Intracapillary proliferative glomerulonephritis.

42. Chronic nephritis (secondary-shrunken kidney).

43. Chronic pyelonephritis.

44. Glandular-muscular hypertrophy of the prostate gland.

45. Glandular hyperplasia of the uterine mucosa.

46. Fibroadenoma of the breast.

47. Acute endometritis.

48. Endocervicosis (cervical ectopia of the cervix).

49. Endometrial adenocarcinoma.

50. Squamous cell non-cancerous cervical cancer.

51. Bubble drift

52. Chorioepithelioma of the uterus

53. Tubal pregnancy

54. Abortion in

a scraper 55. Pancreas in cystic fibrosis

56. Nuclear jaundice in hemolytic disease

57. Acute venous fullness of the lung

58. Brain hemorrhage.

MACROPREPARATIONS.

1. The liver in chronic lymphocytic leukemia.

2. Necrotic angina in acute leukemia.

3. Lymph nodes in chronic lymphocytic leukemia.

4. Spleen in chronic myeloid leukemia.

5. Bone marrow hyperplasia in chronic myeloid leukemia.

6. Lymphatic formations of the intestine in lymphocytic leukemia.

7. Spleen in lymphogranulomatosis.

8. Aortic atherosclerosis.

9. Hypertrophy of the heart in hypertension.

10. Primary-shriveled kidney.

11. Atherosclerotic nephrosclerosis.

12. Gangrene of the toes.

13. Myocardial infarction.

14. Hemopericardium with cardiac tamponade.

15. Brain hematoma.

16. Ischemic brain infarction.

17. Acute warty endocarditis.

18. Polypous-ulcerative endocarditis of the aortic valves.

19. Fibrosis of the mitral valve flaps.

20. Fibrinous pericarditis ("hairy heart»)

21. Acquired heart disease.

22. Congenital heart disease (Fallot's pentad).

23. Nutmeg liver.

24. Heart with artificial valve.

25. Bronchiectasis.

26. Emphysema of the lungs.

27. Silicosis of the lungs.

28. Peripheral lung cancer.

29. Central lung cancer.

30. Acute catarrhal gastritis.

31. Erosions and acute stomach ulcers.

32. Chronic gastric ulcer in remission.

33. Stomach polyps (adenomas).

34. Various forms of stomach cancer:

a) Fungal cancer.

b) Diffuse stomach cancer.

c) Saucer-shaped stomach cancer.

35. Ulcerative colitis.

36. Crohn's disease.

37. Phlegmonous appendicitis.

38. Chronic appendicitis.

39. Gangrenous appendicitis

40.Post-necrotic cirrhosis of the liver:

41. Biliary cirrhosis of the liver (secondary).

42.Muscatine fibrosis of the liver.

43.Multiple liver abscesses:

44. Gallbladder stones

45.Acute glomerulonephritis.

46. Subacute glomerulonephritis with exacerbation.

47. Kidneys in chronic glomerulonephritis (secondary-shrunken kidneys).

48. Necrotic nephrosis.

49. Lipoid nephrosis.

50. Amyloidosis of the kidneys.

51. Purulent pyelonephritis.

52. Kidney stones.

53. Prostate adenoma.

54. Endometrial cancer.

55. Cervical cancer.

56. Breast cancer.

57. Serous ovarian cysts.

58. Tubal pregnancy

59. Ovarian pregnancy

60. Chorionepithelioma of the uterus

61. Bubble drift

62. Thoracopagi

63. Agenesis (unilateral absence ) kidneys.

64. Hypoplasia of the kidney.

65. Horseshoe kidney

66. Microcephaly

67. Anencephaly

68. Exencephaly

69. Craniocerebral hernias

70. Congenital heart defects.

a) Atrial defect

b) Ventricular septal defect

c) Tricameral heart

**Module 3. Pathological anatomy of infectious diseases.**

**Theme 1**. Tuberculosis. Sepsis.

CONTROL QUESTIONS

1. **Sepsis as a special form of infection**. Differences from other infections. Etiology, pathogenesis, relationship between macro- and micro-organism.

2. **The concept of the septic focus, the entrance gate (classification, morphology)**. Classification sepsis. Clinical and anatomical form of sepsis: septicemia, pyosepticemia, sepsis (infection), endocarditis. Pathological changes. Outcomes of inflammatory changes in infections.

    3. **Bacterial shock.** Etiology. Pathogenesis. Pathological changes. Outcomes and complications.

4. **Tuberculosis.** Epidemiology, etiology, patho- and morphogenesis. Classification of Tuberculosis: primary, hematogenous secondary.

5. **Primary tuberculosis**: morphology and ways of progression of primary tuberculosis complex. The complications and causes of death of patients.

6. **Hematogenous tuberculosis**: Pathogenesis, morphology different forms, complication and cause of death.

7. **Secondary tuberculosis**: path formation, morphology of various forms, complication and cause of death. The incidence of tuberculosis in modern conditions. Pathomorphosis tuberculosis.

**The practical part of the subject**

Slides: In the study **micropreparations** pay attention to the education elements, designated by the letters in parentheses.

1. **Metastatic abscesses in the kidneys.** H & E stain. In the cortex and medulla of the kidneys can be seen numerous foci of purulent inflammation presented an accumulation of polymorphonuclear leukocytes (a), in the center of foci kidney tissue is melted (b) visible microbial emboli (c) around the foci of inflammation vessels are dilated, congested (d).
2. **Metastatic abscesses in the lungs.** H & E stain. In lung tissue shows numerous of purulent inflammation lesions presented accumulation of polymorphonuclear leukocytes (a), melting the lung tissue in the heart chambers (b) and foci of inflammation around blood vessels, dilated, congested (c).
3. **Diffuse purulent meningitis.** H & E stain. Pia infiltrated a large number of polymorphonuclear leukocytes with an admixture of macrophages (a) identifies fibrin strands (b). Vessels dramatically congested (c), there diapedetic hemorrhage (d).
4. **Miliary tuberculosis the lung.** H & E stain. In lung tissue, determined by multiple foci (tubercular tubercles) caseous necrosis (a), around which the circular layer of activated macrophages are located - epithelioid cells (b), multinucleated giant cells Pirogov-Langhans resulting fusion of epithelioid cells (c). The outer layers of the cellular infiltrate represented by T-lymphocytes (d).

**Macropreparations.**

**1.** **Metastatic abscesses in the lungs**: The easy preparation of a newborn baby. In lung tissue visible yellowish multiple small foci of purulent inflammation, and subpleural foci of hemorrhage of dark red color.

Causes: bacterial thromboembolism, often with umbilical sepsis.

**2. Metastatic abscesses in the kidneys:**

In preparation - the kidney increased in size, loose consistency. The cortex and medulla in the section seen numerous rounded small foci grayish-yellow size of a pinhead, often surrounded by hemorrhagic rim.

Causes: the result of a bacterial thromboembolism is an expression of an inadequate response to the infectious agent.

**3. Septic spleen:** The spleen is enlarged in size, with hard capsule on a cut crimson, it is often found infarcts, scraping the pulp a abundant.

Causes: a manifestation of the general changes to the hyperplasia of lymphoid tissue in the intoxication can be associated with various pathogens.

**4. Polypous ulcerous endocarditis:** The aortic valve can be seen extensive foci of necrosis and ulceration, frequently with a margin of the valves and the formation of holes (fenestrae), thrombotic massive overlay of polyps in the areas of ulceration.

Causes: The most frequent pathogens - white and Staphylococcus aureus, streptococcus and other zelenyaschy.

Complications with the development of thromboembolic syndrome, heart attacks and gangrene (in the spleen, kidney, brain).

Outcomes: - the formation of valvular defects, thromboembolism with of heart attacks and gangrene (in the spleen, kidney, brain).

**5. Diffuse purulent meningitis:** In preparation of the brain. Shell thickened, dull, yellow-green color, purulent exudate diffusely infiltrates the meninges. The vessels dilated, congested. Brain tissue swelling with diapedetic hemorrhages.

Causes: pathogens often are Staphylococcus and Pseudomonas aeruginosa, less other pathogens.

**6.** **Primary pulmonary tuberculous complex.** In macropreparations right lung. Subpleural in the III segment is the pathological focus, irregular shape with sharp edges in the form of crumbling, curdled mass of gray-yellow in color (hearth caseous necrosis - primary affect). From the necrotic zone to hilar lymph nodes is determined by the "track" of the multiple and small, whitish-yellow hillocks (tuberculous lymphangitis). Regional lymph nodes are enlarged in size, the cut gray-yellow in color with confluent areas of necrosis caseous (caseous tuberculous lymphadenitis).

Causes: Mycobacterium tuberculosis primary infection (airborne path).

Complications: the transition to the primary progressive form of tuberculosis or hematogenous tuberculosis until the development of tuberculous sepsis.

Outcome: fibrosis and fibrosis may lead to the development of pulmonary-cardiac insufficiency. Tuberculous meningitis, caseous pneumonia, tubercular sepsis, bleeding from acute cavities can lead to death.

**7. Primary pulmonary cavity.** In macropreparations lung, deep in the tissue which has a cavity of irregular shape, communicating with the lumen of the segmental bronchi. Content cavity dirty-gray masses, the inner layer of the cavity is formed caseous masses. Cavity walls are thin, without a clear demarcation from surrounding edematous lung parenchyma.

Causes: Mycobacterium tuberculosis primary infection (airborne route), with the progression of infection and subsequent focal caseous destruction of lung tissue.

Complications: bleeding, pneumothorax and tubercular pleurisy, tubercular sepsis.

Outcomes: drainage cavity with subsequent scarring, less chronic process.

**8. TB lymphadenitis.** In macropreparations right lung. Lymph nodes are the root of the lung has increased several times, soldered together in a package on the cut lymphoid tissue is substituted by white-yellow crumbling necrotic masses.

Causes: lymphogenous form of progression of primary tuberculosis.

Complications depend on the localization of primary tuberculosis complex (pulmonary or gastrointestinal) and the involvement of relevant regional (or not only regional) lymph nodes: the development skrofulёza (loss shёynyh lymph nodes), atelectasis (caseous-altered enlarged lymph nodes compress the bronchi or caseous mass occlusive bronchial lumen), the formation of sinus tracts, the generalization of infection and the development of tuberculous sepsis.

Outcomes: depend on the intensity of necrotic processes of the lymphoid tissue; caseous pneumonia; ulceration of the intestinal wall.

**9. Miliary tuberculosis.** Macropreparations presented with lung, which slightly increased in size, increased lightness in their thicker palpated small nodules. In the context of all lobes of both lungs scattered foci thick grayish-white color size of a millet seed.

Causes: hematogenous progression of primary tuberculosis complex; hematogenous generalization of infection foci of screenings after suffering a primary tuberculosis.

Complications: caseous pneumonia, tuberculous meningitis, tuberculous sepsis.

Outcomes: unfavorable.

**10. Fibro-cavernous pulmonary tuberculosis (chronic pulmonary consumption).** Macropreparations presented lungs, which are sealed and are slightly smaller in size. At the top of the right lung cavity (cavity) of irregular round shape with a diameter of 2-5 cm. Thick wall cavity, the inner surface is uneven, the cavity cross sclerotic vessels and bronchi, is seen draining bronchus. Outside the cavity among the layers of connective tissue visible areas of atelectasis and perifocal emphysema. In the middle and lower parts of the lung focal and diffuse sclerosis, petrifikaty, foci of caseous pneumonia.

Characteristically, the oldest changes are observed in the upper parts of the lungs, and the most recent - in the bottom.

Causes: secondary progressive chronic tuberculosis.

Complications: bleeding, hemorrhagic anemia, tuberculous pleural effusion and pneumothorax, secondary renal amyloidosis.

Outcomes: pulmonary fibrosis and emphysema razvitieim with chronic pulmonary heart. Chronic renal failure.

**Test control**

**Select one or more correct answers**

**1. Process name and nature of changes the valve for pyosepticemia**

1) acute warty endocarditis

2) acute polypous ulcerous endocarditis

3) the focus of purulent inflammation in the flap valve

4) focus fibrinous necrosis, surrounded by lymphoid macrophage infiltration

5) often affects the tricuspid valve

**2. Distinctive signs of sepsis FROM OTHER INFECTIOUS DISEASES**

1) strong immunity

2) infectivity

3) acyclicity

4) the specificity of the pathogen

5) poly etiology

**3. general changes in sepsis**

1) parenchymal degeneration of internal organs

2) interstitial inflammation

3) flebotromboz

4) hemorrhagic syndrome

5) hyperplasia of the lymph nodes and spleen

**4. FORMS OF SEPSIS characterized by METASTASIS**

1) chroniosepsis

2) pyosepticemia

3) septicemia

4) protracted septic endocarditis

**5. MACROSCOPIC SIGNS spleen sepsis**

1) reduction of body

2) abundant scraping pulp

3) dense consistency

4) loose consistency

5) hyperplasia pulp

**6. FORM sepsis predominance of general changes over local**

1) urosepsis

2) pyosepticemia

3) septicemia

4) the umbilical sepsis

**7. FEATURE AT septic focus cryptogenic sepsis**

1) the septic focus from the entrance gate of infection

2) septic focus away from the entrance gate of infection

3) septic focus is not detected

4) septic focus - carious teeth

**8. SPECIFIC clinical-morphological characters pyosepticemia**

1) suppurative lymphadenitis and lymphangitis

2) bacterial embolism to form metastatic abscesses in various organs

3) hemorrhagic syndrome

4) suppurative thrombophlebitis

**9. SPECIFIC clinical - morphological signs of septicemia**

1) the absence of purulent metastases

2) fast flowing

3) purulent lymphangitis and lymphadenitis

4) hemorrhagic syndrome

5) hemolytic jaundice

**10. Features characteristic of septic (bacterial) endocarditis**

1) often caused by gram-negative organisms

2) occurs more often in the perverse valves

3) characterized by gross defects valve leaflets

4) often affects the tricuspid valve

5) often affects the aortic valve

**11. POSSIBLE COMPLICATIONS bacterial endocarditis**

1) ischemic cerebral infarction

2) embolic purulent nephritis

3) diffuse glomerulonephritis

4) separation valve

5) renal infarction

**12. Causes of death in septic endocarditis**

1) amyloidosis

2) brain hemorrhage

3) acute heart failure

4) myocardial ischemic brain

5) myocardial infarction

**13. Background diseases or condition for DEVELOPMENT bacterial endocarditis**

1) hypertensive disease

2) alcoholic cardiomyopathy

3) ball prosthetic aortic valves

4) congenital heart disease

5) rheumatic heart disease

**14. TITLE septic endocarditis, develops on unchanged VALVES**

1) primary bacterial endocarditis

2) Sokolsky - Buyo disease

3) Chernogubov disease

4) ankylosing spondylitis

5) endocarditis Libman - Sachs

**15. Microscopic DIFFERENCE OF CHANGES IN HEART VALVES in a bacterial endocarditis of the rheumatic returnable-warty endocarditis**

1) the presence of thrombotic overlays

2) the nature of the cellular infiltrate

3) valve sclerotic changes

4) the presence of bacteria

5) the formation of foci of calcification.

**16. Characteristic manifestations of septicemia**

1) hemolytic jaundice, hemorrhagic syndrome

2) is characterized by peripheral characters: stains Lukin-Liebmann, Osler nodes

3) thrombotic overlay on the valve

4) septic focus away from the entrance gate

5) multiple abscesses in organs

6) the absence of purulent metastases

**17. PROCESS AND NATURE OF CHANGES IN HEART VALVES for pyosepticemia**

1) acute polypous ulcerous endocarditis

2) acute warty endocarditis

3) the focus of purulent inflammation in the flap valve

4) the focus of fibrinoid necrosis, surrounded by lymphoid macrophage infiltration

5) often affects the tricuspid valve

**18. Characteristic signs chroniosepsis**

1) long-term for long-term

2) severe hyperergy

3) reduce the reactivity of the organism

4) septic focus is not detected

5) splenomegaly

6) the availability of long-term healing septic focus

**19. FORMS OF SEPSIS depending on the input GATE**

1) surgery

2) wound

3) septicemia

4) bacterial endocarditis

5) the umbilical sepsis

6) iatrogenic sepsis

**20. Local changes in sepsis**

1) lymphadenopathy

2) septic spleen

3) the focus of purulent inflammation

4) lymphangitis

5) thrombophlebitis

6) interstitial hepatitis

**21. Characteristic manifestations pyosepticemia**

1) hemolytic jaundice, hemorrhagic syndrome

2) is characterized by peripheral characters: stains Lukin-Liebmann, Osler nodes

3) overlay on the thrombotic leaflets

4) septic focus away from the entrance gate

5) multiple abscesses in organs

6) the absence of purulent metastases

**22. Characteristic manifestation of bacterial endocarditis**

1) hemolytic jaundice, hemorrhagic syndrome

2) is characterized by peripheral characters: stains Lukin-Liebmann, Osler nodes

3) overlay on the thrombotic leaflets

4) septic focus away from the entrance gate

5) multiple abscesses in organs

6) the absence of purulent metastases

**23. tubercle bacilli that can cause tuberculosis in MAN**

1) bovine type

2) the type of bird

3) human-type

4) cold-blooded animals

**24. Possible ways ingestion of tubercle bacilli**

1) the alimentary

2) airborne

3) transmissible

4) parenteral

5) transplacental

**25. complications, characteristic of tuberculosis**

1) pulmonary hemorrhage

2) tuberculous pleurisy

3) amyloidosis bodies

4) suppurative metastases in parenchymal organs

**26. The correct definition of TB FOCI**

1) focus Gon - healed primary focus of tuberculosis

2) The focus of Abrikosov - center caseous pneumonia - initial manifestations of secondary tuberculosis

3) focus Assman-Redeker - healed center Abrikosov

4) focus Aschoff Bullet - focus infiltrative tuberculosis in secondary tuberculosis

**27. Wrong definition of TB FOCI**

1) focus Gon - healed hotbed of secondary tuberculosis

2) The focus of Abrikosov - center caseous pneumonia - initial manifestations of secondary tuberculosis

3) focus Aschoff Bullet - healed center Abrikosov

4) focus Assman-Redeker - focus infiltrative tuberculosis in the hematogenous tuberculosis

**28. TYPICAL FOR tuberculous inflammation exudate**

1) purulent

2) fibrinous

3) serous desquamative

**29. SIGNS primary tuberculosis**

1) subpleural localization of the lower lobe of the lung

2) caseous lymphadenitis

3) intrakanalikulyarny route of infection

4) lymphogenous generalization

**30. The form of tuberculosis with the most part of the development of tuberculous meningitis**

1) hematogenous form of progression of primary tuberculosis complex

2) generalized hematogenous tuberculosis

3) Tuberculosis of the genitourinary system

4) secondary tuberculosis

**31. Forms secondary tuberculosis**

1) infiltrative

2) cirrhotic

3) productive

4) interstitial

5) cavernous

**32. FORMS OF TUBERCULOSIS to enter a tubercle**

1) alopecia

2) infiltrative

3) primary affect

**33. Phase reflects the "characteristics" of tuberculosis process**

1) alterative (necrotic)

2) exudative (inflammatory)

3) proliferative (granulomatous)

**34. tissue changes during exacerbation of tuberculosis**

1) the formation of granulomas

2) exudation

3) caseous necrosis

**35. The lymph nodes are affected in form of tuberculosis**

1) hematogenous

2) the secondary

3) primary

4) fibronodular

5) fibrocavernous

**36. VARIETY hematogenous tuberculosis**

1) caseous pneumonia

2) acute cavernous

3) cirrhotic

4) miliary

5) infiltrative

6) tuberculoma

**37. PATHWAYS infection in secondary tuberculosis**

1) intrakanalikulyarny

2) lymphogenous

3) contact

**38. The forms of TB, not included in secondary tuberculosis**

1) acute focal

2) acute cavernous

3) infiltrative

4) miliary

5) cirrhotic

**39. The elements of primary tuberculosis complex**

1) The primary affect and lymphangitis

2) lymphadenitis and lymphangitis

3) lymphangitis, lymphadenitis and primary affect

**40. PRIMARY tuberculosis lymph node CHARACTERIZED**

1) total caseous necrosis

2) "specific" granulomas

3) hyperplasia of lymphoid tissue

4) medullary swelling of lymphoid tissue

**41. hematogenous-disseminated tuberculosis, usually in the dissemination**

1) in one lung

2) both lungs

3) of the upper lobe of the left lung

**42. The changes in the lungs in acute tuberculous sepsis**

1) small foci of necrosis

2) miliary tubercles

3) large pockets caseous necrosis

4) subpleural cavity

**43. The signs of a general acute miliary tuberculosis**

1) small necrotic bumps in all organs

2) nodosa pockets in all organs

3) lymphocytic bumps in all organs

4) multiple large foci of necrosis

**44. ORGAN characteristic localization of secondary tuberculosis**

1) brain

2) the pancreas

3) lung

4) bone

5) the joints

**45. SIGNS infiltrative pulmonary tuberculosis**

1) the prevalence of pronounced perifocal inflammation of caseous necrosis

2) the form of the secondary tuberculosis

3) a small portion of caseous necrosis

4) changes in the prevalence of caseous over the perifocal inflammation

5) fibrous cavity wall

6) of the cavity containing the mass of caseous, not circumscribed by fibrous tissue from the surrounding lung tissue

**46. tuberkuloma lung presented**

1) multi-specific granulation

2) a single major focus of caseous necrosis

3) with a cavity wall of the fibrous

4) cystic cavity with hemorrhagic content

**47. ACUTE cavernous pulmonary tuberculosis SUBMITTED**

1) cavity wall with fibrous

2) cystiform cavity

3) cavity containing a caseous mass, not circumscribed by fibrous tissue from the surrounding lung tissue

4) all of the above is true

**48. CONTENT cavity with fibro-cavernous pulmonary tuberculosis**

1) the caseous mass, specific granulation and fibrous tissue

2) necrotic masses

3) lined by a multilayer flat epithelium neorogovevayuschy

4) lined with prismatic epithelium

**49. SIGNS cirrhotic pulmonary tuberculosis**

1) fibrosis, bronchiectasis, cavity cystiform

2) emphysema

3) specific inflammation

4) suppurative inflammation

**50. expressed aggravation of tuberculosis process is an inflammatory tissue reaction**

1) Productive

2) exudative

3) productive-infiltrative

4) productive necrotic

**51. OUTCOMES tuberculous granulomas**

1) abscess

2) hemorrhagic infiltration

3) putrefaction

4) scarring

4) atrophy

6) petrification

**52. CELLS tuberculous granulomas**

1) epithelioid

2) fat

3) Pirogov-Langans giant cells

4) Anichkov cells

**53. The characteristic manifestations of primary tuberculosis**

1) caseous lymphadenitis

2) pathway intrakanalikulyarny

3) the affected area - apex

4) the most frequent localization process - III segment of the right lung

5) subpleural focus localization

**54. The characteristic manifestations of secondary tuberculosis**

1) caseous lymphadenitis

2) pathway intrakanalikulyarny

3) the affected area - apex

4) the most frequent localization process - III segment of the right lung

5) subpleural focus localization

**55. FORM OF PROGRESSION OF PRIMARY TUBERCULOSIS With the development of "galloping consumption"**

1) hematogenous

2) lymphogenous

3) the growth of primary affect

4) Mixed

**56. COMPLICATIONS OF PRIMARY TUBERCULOSIS**

1) fibrinous pleurisy

2) tuberculous meningitis

3) amyloidosis

4) fistulas

5) bleeding in the formation of cavities

6) sequestrations, bone deformities

**57. COMPLICATIONS OF SECONDARY TUBERCULOSIS**

1) fibrinous pleurisy

2) tuberculous meningitis

3) amyloidosis

4) fistulas

5) bleeding in the formation of cavities

6) sequestrations, bone deformations

**58. Complications hematogenically TUBERCULOSIS**

1) fibrinous pleurisy

2) tuberculous meningitis

3) amyloidosis

4) fistulas

5) bleeding in the formation of cavities

6) sequestrations, bone deformities

**Theme 2**. **intestinal infections**

CONTROL QUESTIONS

**1. Typhoid fever.**  
a) etiology, mechanism for dissemination of, modern doctrine of the pathogenesis of typhoid fever,  
b) major general and local morphological changes in typhoid fever

c) intestinal and extra-intestinal complications of typhoid fever.

**2. Salmonellosis.** Basic forms: intestinal, septic and typhoid. Etiology, ways of infection, pathogenesis, morphology, complications, outcomes.

**3. Bacillary dysentery (Shigellosis).**  
a) etiology. Ways of infection, the modern theory of the pathogenesis of dysentery,  
c) forms of dysentery and their morphological characteristics,  
d) outcomes and complications of dysentery.

**3. Cholera.** Epidemiology, etiology, patho- and morphogenesis, morphological characteristics, clinical manifestations, complications, outcomes, causes of death.

**4. Amoebiasis.** Epidemiology, etiology, patho- and morphogenesis, morphological characteristics, clinical manifestations, complications, outcomes, causes of death.

**5. Viral enteritis and diarrhea.** Etiology, morphological characteristics, clinical manifestations, complications, outcomes.

**6. Campylobacter’s enteritis, Yersinia enteritis.** Epidemiology, etiology, patho- and morphogenesis, morphological characteristics, clinical manifestations, complications, outcomes, causes of death.

The practical part of the subject:

Slides: In the study **micropreparations** pay attention to the education elements, designated by the letters in parentheses.

**1.Medullary swelling of Peyer's patches in typhoid fever.** H & E stain.

In the mucosa of the small bowel there is edema and hyperemia (a), in the lamina propria there is "typhoid granuloma" (b), consisting of typhoid cells - macrophages, histiocytes, reticular cells and monocytes (c).

**2. Necrosis of the Peyer's patch.** H & E stain.

In the Peyer's patches of the jejunum there are typhoid granulomas (a) with necrosis (b), penetrating to different depths, with the formation of the ulcer (c). The mucous membrane is swelling and hyperemia (d).

**3. Mesenteric lymph node at a typhoid fever.** H & E stain.

In the lymph node there is "typhoid granuloma" (a), consisting of an accumulation of macrophages, histiocytes, reticular cells and monocytes (b). There are areas of necrosis of the lymphoid tissue (c), a significantly reduction of lymphoid cells (d).

**4. Diphtheritic colitis.** H & E stain.

On the surface of the colon mucosa is fibrinous membrane (a), edema, hemorrhage (b), and infiltration of polymorphonuclear leukocytes (c) in the submucosal layer. There is necrosis of the mucosa with formation of the ulcer (d), the bottom of which is the muscle layer (e).

**Macropreparations:**

**1. Medullary swelling of Peyer's patches at a typhoid fever.** Preparation of the ileum. The follicles increased in size, protrude above the surface of the mucous membrane, with a rough surface, in the form of sulci and gyri, on the cut follicles is juicy, pink and gray.

Reasons: typhoid fever.

Complications: intestinal - necrosis, bleeding. Extraintestinal - purulent perichondrium laryngeal abscess, osteomyelitis, arthritis, cystitis, prostatitis, intramuscular abscesses, pneumonia, sepsis, rare.

Outcome: scarring with epithelialization.

**2. Spleen at a typhoid fever.** The spleen is greatly increased in size 18-15-10 cm, capsule is tense, easily removed, on the cut the parenchyma protrudes, dark cherry color, gives scraping pulp.

Complications: rupture of the capsule, bleeding into the abdominal cavity, peritonitis.

**3. Diphtheritic colitis.** The preparation of portion of the colon, in the cut mucosa is hyperemic, thickened by the expressed edema, on the surface of the mucosa has a dirty brown membrane, tightly soldered to the underlying tissues. After the rejection fibrinous membrane remains defects - erosion and ulceration.

Causes: Dysentery.

Complications: intestinal - bleeding, perforation, peritonitis, abscess, phlegmon. Extraintestinal complications - pneumonia, pyelonephritis, arthritis, liver abscesses, at the chronic course - amyloidosis, exhaustion.

Outcome: cicatrical deformation of the bowel wall, chronic colitis dysentery.

**4. Follicular-ulcerative colitis at a dysentery.** The preparation of portion of the colon. Group and solitary follicles are increased, due to hyperplasia, in the center of follicles there is necrosis and purulent fusion with the formation of ulcers. Complications: bleeding, perforation, peritonitis. Outcome: scarring of the bowel wall.

**5. Acute enteritis at a salmonellosis.** Preparation of the small intestine with a sharp vascular hyperemia, on the cut mucosa is edematous with hemorrhages. On the surface of the mucosa there is erosion and ulceration that penetrate to different depths.

Reason: Salmonella.

Complications: toxico - infective shock, dysbacteriosis, acute renal failure.

Outcome: epithelialization, scarring.

Test control

Select one or more correct answers

1.Characteristic signs of typhoid fever  
**1) enlargement of the spleen**  
2) suppurative osteomyelitis  
3) fibrinous-necrotic angina  
4) exantema  
5) protein and fatty degeneration of hepatocytes

2.Pathological process for uncomplicated typhoid  
1) mucoid swelling  
2) fibrinous inflammation of the intestinal mucosa  
3) the emergence of dyscirculatory ulcers in the bowel  
4) hyperplasia in the mesenteric lymph nodes intestine  
**5) medullary swelling of Peyer's patch**

3. The characteristic signs of the first stage of typhoid fever  
1) degenerative changes in the nerve fibers of intestine  
2) microperforation of plaque  
3) lymphocyte proliferation Peyer's patch  
**4) hyperplasia of Peyer's patch and catarrhal enteritis**  
5) mucoid swelling structures Peyer's patch

4. Bowel perforation at a typhoid fever occurs most frequently

1) In connection with pathological regeneration in the fifth stage of the disease

2) in the second stage in connection with hyperergic necrosis of Peyer's patch

3) in the first stage in connection with germination Peyer's patch by reticulum cells

**4) in the stage of formation clean ulcers**

5. Appearance in intestinal bleeding at a typhoid fever

1) In connection with pathological regeneration in the fifth stage of the disease

**2) in the second stage in connection with hyperergic necrosis of Peyer's patch**

3) in the first stage in connection with germination Peyer's patch by reticulum cells

4) in the stage of formation clean ulcers

6. Characteristic pathological process for the typhoid

1) spastic colitis

2) ulcerative lesion the ascending portion of the colon

3) follicular ulcers of the colon

4) occurrence of ulcers in the colon on the 10th day of illness

**5) perichondritis of the larynx**

7. FOR The characteristic signs FOR septic forms of the salmonellosis

1) Hyperemia mucosa of the gastrointestinal tract

**2) abscess subdiaphragmatic space**

3) peritonitis due to perforation of the bowel wall

4) Hyperplasia of lymphoid apparatus of the intestinal mucosa

8. The characteristic signs FOR intestinal forms of the salmonellosis

1) medullary swelling of Peyer's patches

**2) rapid dehydration, exsicosis**

3) hematogenous generalization of pathogen

4) hyperplasia of the spleen necrosis

5) metastatic abscesses in the lung

9. Causative agents of dysentery

**1) Shigella**

2) Salmonella

3) Yersinia

4) Escherichia

5) vibrio

10. Stage of the development of COLITIS at the dysentery

**1) catarrhal**

**2) fibrinous**

**3) ulcer**

4) purulent haemorrhagic

11. Intestine, most commonly affected with dysentery

1) sigmoid colon

**3) rectum and sigmoid**

4) the small intestine

5) the rectum

12. Dissemination of the pathogen from the primary focus HAPPENS

1) perineural

2) lymphogenous

3) hematogenically

**4) lymphogenous and hematogenically**

13. The most typical characteristics for catarrhal dysentery

**1) sigmoiditis**

2) encephalitis

3) involvement of the mesentery nodes of the transverse colon

4) paralytic extension of rectum

5) enteritis

14. Complications of dysentery

**1) bleeding with perforation**

2) osteomyelitis

**3) distant metastases lime**

4) necrotizing nephrosis

**5) scar stenosis of the colon**

15. FEATURE of ulcers at the dysentery ulcerative colitis

1) formed on the site of lymphoid follicles

**2) develop on melting fibrinous membrane**

**3) usually does not extend beyond the mucosa**

4) have a callous edge

5) the bottom of ulcer is black (hydrochloric acid hematin)

16. Forms of dysentery with possible perforation of the intestinal wall

**1) diphtheritic**

2) follicular-ulcer

3) chronic

4) toxic

17. Characteristic lesions at a dysentery

1) gastritis

2) ileitis

3) ileokolitis

4) typhlitis

**5) proctitis**

18. Intestine, in which the originating primary lesion at a typhoid fever

1) duodenum

2) direct

3) sigmoid

**4) ileum**

5) colon

19. Characteristic signs of the first stage of CHOLERA

1) splenomegaly

2) dehydration (exsicosis)

3) necrosis of epithelial cells of intestine

4) spleen is decreased, small, dense

**5) serous edema of the villi of the small intestine, swelling of enterocytes**

20. Complications of cholera

1) gasteroenterit

**2) sub-acute intracapillary glomerulonephritis**

3) profuse diarrhea

4) serous hemorrhagic enteritis

**Theme 3**. **respiratory viral infections. CHILDREN AND INTRAUTERINE INFECTION.**

CONTROL QUESTIONS

**1. Infection.** Concept. Infectious agents (endoparasites, ectoparasites) classification. General characteristics of the infectious process: the entrance gate of infection, primary infection complex, distribution and dissemination, ways of transmission of infectious agents. Possible local and general reactions in infections.

**2. Infection primarily affecting the respiratory system.** **Influenza.** Epidemiology, etiology, patho- and morphogenesis, clinical and morphological characteristics, complications, consequences, causes of death.

**3. Parainfluenza.** Epidemiology, etiology, patho- and morphogenesis, clinical and morphological characteristics, complications, consequences, causes of death.

**4. Respiratory syncytial infection (RS) infection.** Epidemiology, etiology, patho- and morphogenesis, clinical and morphological characteristics, complications, consequences, causes of death.

**5. Adenovirus infection.** Epidemiology, etiology, patho- and morphogenesis, clinical and morphological characteristics, complications, consequences, causes of death.

**6. Measles.** Etiology, epidemiology, pathogenesis, pathological anatomy. Complications cause of death.

**7. Diphtheria.** Etiology, epidemiology, pathogenesis, pathological anatomy, complications, causes of death.

**8. Scarlet fever.** Etiology, epidemiology, pathogenesis, pathological anatomy, complications, causes of death.

**9. Meningococcal meningitis.** Etiology and pathogenesis. Morphology. Outcomes.

**10.** **Intrauterine infection:** cytomegaly, mycoplasmosis, listeriosis, toxoplasmosis. Etiology pathogenesis, clinical and morphological characteristics, diagnosis.

**The practical part of the subject**

Slides: In the study **micropreparations** pay attention to the education elements, designated by the letters in parentheses.

**1. The purulent meningitis. H & E stain.** Pia edematous, with hyperemia vessels (a) and diffuse infiltration of polymorphonuclear leukocytes (b). The brain tissue edema peritselyullyarny (c), diapedetic perivascular hemorrhage (d).

**2. Diphtheritic angina.** H & E stain. There desquamation of stratified squamous epithelium in the tonsil (a), massive fibrinous deposits (b), in the surrounding tissue swelling and congestion of vessels (c).

**3. Myocarditis in diphtheria.** H & E stain. In cardiomyocytes, fatty degeneration and necrosis (a), in the interstitial tissue sharp hyperemia and edema of the capillary bed (b), lymphohistiocytic interstitial infiltrate with admixture of neutrophils and eosinophils (c).

**Macropreparations.**

**1. Fibrinous-hemorrhagic laryngotracheitis at the severe influenza.** In preparation section of larynx and trachea. The mucosa is hyperemic, edematous, with extensive areas of necrosis and ulceration. On the surface of mucosa is determined by gray-red membrane, which easily removed.

Causes: the influenza virus.

Complications and outcomes: false croup, descending croup, aspiration pneumonia.

**2. Bronchopneumonia at complications of influenza (great mottle lung).** Lungs are increase in volume, full-blooded, swollen, mottled appearance. In a cut are defined light gray peribronchial pneumonic lesions, with pressure from the surface of the lung flow down serous-hemorrhagic fluid. In the lumen of the bronchi there are mucous-purulent contents, streaked with blood. In the parenchyma there are multiple hemorrhages, small foci of purulent inflammation, foci of atelectasis and emphysema.

*Causes:* heavy during of influenza with the accession of secondary infection (viral and bacterial form) and with development of pulmonary complications.

*Complications:* bronchiectasis (more children), chronic obliterans bronchitis, chronic obstructive pulmonary emphysema, pulmonary fibrosis, sinusitis, encephalitis, arachnoiditis, neuritis.

*Outcomes:* poor; cardiopulmonary failure.

**3. Purulent meningitis**. In preparation of the brain, meninges cerebral hemispheres bloodshot, bleary in the subarachnoid space has a cluster of fibrinopurulent yellow exudate. Purulent accumulations exist in basal cisterns and the Sylvian fissure. With the progression of the disease the inflammatory process extends to the ventricles ependymomas.

*Causes:* meningococcal infection.

*Complications:* meningoencephalitis, brain abscesses. Rarely process can take over a sustained, growing organization of fibrinous exudate holes with obliteration of the ventricles, hydrocephalus.

**4. Necrotizing tonsillitis and sharp hyperemia throat.** In preparation organocomplexes - determined by a sharp hyperemia throat, enlarged tonsils, swollen on the surface dull greyish lesions in the form of a film, that tightly welded to the underlying tissues and after rejection in their place are deep defects - ulcers, some with purulent fusion. Submandibular and cervical lymph nodes are enlarged and juicy.

*Causes*: hemolytic streptococcus group A.

*Complications*: purulent otitis media, mastoiditis, sinusitis, brain abscess, meningitis, cellulitis maxillofacial area and neck, pyosepticemia.

**5. Croup larynx and trachea in diphtheria**. In preparation section of larynx and trachea, the cut mucosa hyperemic, edematous. On the surface it is determined by the white-yellow film loosely soldered to the underlying tissue and easily torn away, obturiruya lumen.

*Causes*: stick Leffler.

*Complications:* true croup, descending croup, aspiration pneumonia.

**6. Acute hemorrhagic glomerulonephritis.** Grossly "mottled kidney." The kidneys are enlarged, swollen parenchyma, the capsule is removed easily. In the context of the bark is pale, wide with red specks in the medulla stagnant congestion.

*Causes*: viral and mycoplasma infection.

*Outcome*: acute failure.

**Test control**

**Select one or more correct answers**

**1. Form of meningococcal infection**

1) purulent meningitis

2) toxic

3) nasopharyngitis

4) meningococcemia

5) laryngotracheitis

**2. Typical inflammation of meningococcal meningitis**

1) hemorrhagic

2) catarrhal

3) productive

4) suppurative

5) granulomatous

**3. Complications MENINGITIS**

1) Brush the brain

2) brain tumor

3) hemorrhagic cerebral infarction

4) Hydrocephalus

5) glial scar

**4. CHARACTERISTICS SPECIFIC meningococcal nasopharyngitis**

1) catarrh of the mucous membrane

2) hyperemia and edema of the posterior pharyngeal wall

3) hyperplasia of lymph follicles

4) is more common in adults

5) nasopharyngitis develops in 10-30% of cases of infection of meningococcus in the nasal mucosa

**5. SIGNS SPECIFIC meningococcal meningitis**

 1) serous exudate

 2) purulent exudate

 3) bleeding in the brain

 4) suppurative-fibrinous effusion

 5) full-fledged brain tissue, swelling

**6. SIGNS meningococcemia**

1) skin purpura

2) Endocarditis

3) septic arthritis and pericarditis

4) generalized defeat ICR

5) necrosis in the kidneys and adrenals

6) purulent iridocyclitis

**7. features are characteristic for the outcome of meningococcal meningitis**

1) absorption of exudate

2) hydrocephalus and atrophy of brain substance

3) loss of choroid

4) the development of meningococcal encephalitis

5) holes obliteration of the 4th ventricle and obstruction of cerebrospinal fluid circulation

**8. Scarlet fever AGENT**

1) diplococcus

2) E. coli

3) Streptococcus group A

4) Streptococcus viridans

5) Staphylococcus aureus

**9. typical localization lesions in scarlet fever**

1) oral mucosa

2) shed

3) Leather

4) conjunctiva

**10. Changes to regional lymph nodes in scarlet fever**

1) necrosis

2) Anemia

3) hypoplasia

4) multiple sclerosis

5) atrophy

**11. pathomorphological manifestations of scarlet fever**

1) formation of the primary complex, the primary passion, combined with regional lymphadenitis

2) "blazing shed"

3) punctulate rash is bright red, covers the entire body except for nasolabial triangle

4) toxic myocarditis

5) acute diffuse glomerulonephritis in the 2nd stage of the disease

**12. SIGNS OF SPECIFIC scarlet fever**

1) plate-shaped peeling of the epidermis

2) retropharyngeal abscess

3) fibrinous-necrotic angina

4) hyperplasia breeding centers follicles plazmatizatsiey

5) parenchymal neuritis with the disintegration of the myelin

6) myeloid metaplasia in the spleen, lymph nodes and Peyer's patches

**13. SPECIFIC complications of scarlet fever**

1) chronic otitis

2) chronic kidney disease

3) erosion of major blood vessels of the neck

4) suppurative osteomyelitis of the temporal bone

5) all of the above is true

**14. CHARACTER inflammation at the entrance gate in diphtheria**

1) productive

2) fibrinous

3) purulent

4) hemorrhagic

5) putrid

**15. RARE, entrance gate in diphtheria BE**

1) the larynx

2) palatine tonsils

3) shed

4) wound surface

**16. The change of heart in diphtheria**

1) fibrinous pericarditis

2) suppurative myocarditis

3) toxic myocarditis

4) heart defect

5) back and warty endocarditis

**17. pathomorphological manifestations Diphtheria**

1) true croup

2) parenchymatous neuritis with the disintegration of the myelin

3) alterative myocarditis

4) circulatory disorders in the ganglia

5) all but 3

6) all of the above is true

**18. One feature is not characteristic for Diphtheria**

1) necrotizing nephrosis

2) hyperplasia of the splenic follicles

3) small foci of necrosis in the cortical layer of the adrenal glands

4) a bilateral interstitial mumps

5) interstitial myocarditis

**19. Characteristic signs FOR DIPHTHERIA**

1) metaplasia of the epithelium of the upper airways in the stratified squamous

2) giant cell pneumonia

3) hyperemia oral mucosa around stennova duct

4) fibrinous-necrotic angina

5) the true croup

**20. SPECIFIC COMPLICATIONS Diphtheria**

1) the formation of bedsores traheostomaticheskoy tube

2) purulent perichondrium

3) chronic otitis

4) suppurative mediastenit

5) peribronchial pneumonia

**21. POSSIBLE WAYS fetal infection**

1) transcervically

2) transplatsentarno

3) perineural

4) downward by

**22. Common symptoms of intrauterine infection**

1) large fruit

2) hemorrhagic syndrome

3) congenital malformations

4) accelerated maturation of tissues

5) extramedullary hematopoiesis

**23. SIGNS congenital pneumonia**

1) infection by transplacental

2) the predominance of necrotic changes

3) by aspiration of infected amniotic fluid

4) E. coli infection

5) development of the disease in the first 72 hours of life

**24. The signs of fetal Sepsis**

1) jaundice and anemia

2) false croup

3) osteomyelitis

4) hyaline membrane disease

5) DIC

**25. Signs of acute forms of congenital cytomegalovirus**

1) loss of the salivary glands and hepatosplenomegaly

2) hemolytic anemia and jaundice

3) cells in the form of "owl's eye"

4) desquamative papular rash

5) Deafness

**26. The manifestations of chronic congenital cytomegalovirus**

1) the type of congenital embriopaty

2) necrotizing enterocolitis

3) fibrosis of interstitial inflammation

4) catarrhal angina

5) cytomegalovirus metamorphosis cells

**27. SIGNS congenital toxoplasmosis**

1) microcephaly

2) granulomatous inflammation

3) eye disease

4) cramps

**28. Morphological changes fetus in toxoplasmosis in between 9 and 29 weeks of pregnancy**

1) true hypoplasia of the eyes

2) a delay in the formation and differentiation of the brain

3) bleeding diathesis

4) Hydrocephalus

5) pulmonary edema

**29. Morphological changes fetus in toxoplasmosis from 29 weeks of pregnancy before birth**

1) nephrotic syndrome

2) foci of calcification in the brain

3) alterative-productive meningoencephalitis

4) pulmonary agenesis

5) retinal lesions and vascular tract

**30. Generalized forms TOXOPLASMOSIS CHARACTERISTIC**

1) interstitial pneumonia

2) acute adrenal insufficiency

3) occurs shortly before birth or during labor

4) kernicterus

5) hepatosplenomegaly and jaundice

**31. intrauterine infection MIKOPLAZ-MAMI CHARACTERIZES**

1) transplacental route of infection

2) interstitial pneumonia

3) chorioretinitis

4) foci of calcification in the brain

5) congenital malformations

**32. mycoplasma infection predominantly affects**

1) musculoskeletal system

2) genitourinary tract

3) CNS

4) the respiratory system

5) bodies of

**33. ACUTE urogenital mycoplasma infection results**

1) generic sepsis

2) septic abortion

3) mieloeritroblastozu

4) cryptorchidism

5) premature birth

**34. Congenital listeriosis CHARACTERIZED**

1) pyosepticemia

2) heart defects

3) granulomatous sepsis

4) triad Hutchinson

5) preterm birth

**35. Listeriosis CHARACTERIZED**

1) liver

2) DIC

3) lymph nodes and spleen

4) fibrosis

5) CNS damage

**36. The clinical and morphological forms of listeriosis are characteristic of the fetus and newborn**

1) anginal-septic

2) nervous

3) septic granulomatous

4) eye-glandular

5) heart

**37. Histological structure LISTERIOMY**

1) giant cells

2) the zone of necrosis

3) the granulation tissue in the center of the granuloma

4) lymphohistiocytic infiltration

5) the abundance of leukocytes

**38. SIGNS septic granulomatous FORMS listeriosis**

1) granulomatous inflammation of the liver

2) conjunctivitis

3) purulent meningitis

4) extramedullary hematopoiesis

5) purulent hemorrhagic pneumonia

**39. COMPONENTS true croup in diphtheria**

1) fibrinous exudate in the alveoli

2) diphtheritic inflammation of the larynx and trachea

3) reflex spasm

4) rapid swelling of the larynx

5) croupous inflammation of the larynx and trachea

**40. pathomorphological manifestations in the second period of scarlet fever**

1) acute diffuse glomerulonephritis

2) quinsy

3) myocarditis

4) interstitial hepatitis

5) synovitis, arthritis

**Standards of answers to test tasks ON THEME:**

**«RESPIRATORY VIRAL INFECTIONS. CHILDREN AND INTRAUTERINE INFECTION».**

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| **№** | **Ответ** | **№** | **Ответ** | **№** | **Ответ** | **№** | **Ответ** |
|  | 1,3,4 |  | 1,2,3,5 |  | 1,2,4 |  | 1,2,5 |
|  | 4 |  | 1,2,3,4,6 |  | 2,3,5 |  | 2,3,4 |
|  | 4 |  | 5 |  | 1,3,5 |  | 1,2,5 |
|  | 1,2,3,5 |  | 2 |  | 1,3,5 |  | 1,3,5 |
|  | 1,2,4,5 |  | 4 |  | 1,2,3 |  | 1,3,5 |
|  | 1,3,4,5,6 |  | 3 |  | 1,3,5 |  | 3 |
|  | 1,2,4,5 |  | 6 |  | 2,3,4 |  | 2,4,5 |
|  | 3 |  | 4 |  | 1,2,4 |  | 1,3,5 |
|  | 2,3 |  | 5 |  | 2,3,5 |  | 3,4,5 |
|  | 1 |  | 1,2,4 |  | 1,3,5 |  | 1,3,5 |

**Theme 4**. **Final lesson on lecture and theoretical material on the module "Pathological anatomy of infectious diseases".**

Form(s) of the current control of academic performance (testing, interview, diagnosis of macropreparation and micropreparation).

Evaluation materials of the current control of academic performance

Test tasks

Test tasks for the current classes.

Consolidation of theoretical material (Oral analysis of the topic and a survey of students )

1. Features of the course of the infectious process in different age groups. The concept of the reactivity of the body and the general morphology of the infectious process. Interaction of a macroorganism and infectious agents. Diseases caused by bacteria. Local and general reactions in infections. Bacteremia. General morphological characteristics. The peculiarity of the infection in connection with the characteristics of the pathogen and the method of its transmission.

2. Epidemiology of tuberculosis, etiology. Features of tubercle bacilli that are important in the development of this disease. Ways of penetration. The incidence of tuberculosis in different age groups. The importance of TB vaccination, depending on the social conditions, the reactivity of the body. The nature of inflammation in tuberculosis depends on the reactivity of the body. Types of tubercular tubercles, their morphology and stages of formation, outcomes.

3. Primary tuberculosis. Primary tuberculosis complex: its localization. Morphology of components. Outcomes of the primary complex:

a) the spread of the primary affect through the contact. Forms of lung damage,

b) lymphocytic form of progression (bronchoadenitis, mesoadenitis). Complications, c

) hematogenic generalization (miliary and large-focal),

d) Chronic forms of primary tuberculosis.

4. Hematogenic forms of tuberculosis. Forms of generalization. Organ tuberculosis (tuberculosis of the bone and joint, genitourinary system, skin and other organs). Complications, causes of death.

5. Secondary tuberculosis. Ways of infection. Localization and structure of secondary affect. The spread of the process in secondary tuberculosis. Forms of secondary tuberculosis. Complications, causes of death.

6. Sepsis as a special form of infection. Differences from other infections.

7. The etiology of sepsis, the relationship of macro-and micro-organism, pathogenesis, local and general manifestations.

8. The concept of septic foci, entrance gates (classification, morphology).

9. Classification of sepsis. Clinical and anatomical forms of sepsis: septicemia, septicopiemia, septic (infectious) endocarditis. Pathomorphological changes.

10. Bacterial shock. Etiology. Pathogenesis. Pathomorphological changes. Outcomes and complications.

11. Meningococcal infection:

a) etiology, pathways of infection and mechanism of spread,

b) modern studies on the pathogenesis of meningococcal infection,

c) the main morphological features of meningococcal infection, taking into account the various forms of the disease,

d) meningococcal nasopharyngitis, the frequency of its development, taking into account the invasion of meningococcus into the nasopharyngeal mucosa,

e) purulent meningitis, the relationship of its development with the immaturity of the blood-brain barrier,

f) meningococcemia, the dependence of its development on the state of immune reactivity of the body,

g) outcomes and complications of meningococcal infection, taking into account the forms of the disease, the cause of death.

12. Scarlet fever:

a) characteristics of the disease, etiology and pathogenesis, frequency of the disease in different age groups,

b) pathological anatomy of the first period of scarlet fever: reveal the morphology of scarlet fever, the spread of infection and general changes in the tissues,

c) describe the toxic form of the disease,

d) morphological picture of septic and toxic-septic forms of the disease,

e) pathological anatomy of the second period of scarlet fever, features of its occurrence and course,

f) complications and causes of death in severe scarlet fever.

13. Diphtheria:

a) definition of the disease, etiology and pathogenesis, clinical and anatomical forms of the disease,

b) local and regional changes in the tissues in diphtheria of the pharynx, larynx, trachea and bronchi, the concept of "true croup" and its contrast to "false croup",

c) General changes in the tissues in diphtheria, describe the processes in the nervous, cardiovascular systems, adrenal glands and associate them with the clinical manifestations of the disease.

14. Measles:

a) definition of the disease, etiology and pathogenesis, ways of infection penetration into the body

b) clinical and morphological characteristics, complications and outcomes.

15. Intrauterine infections: Cytomegaly:

a) definition of the disease, etiology and pathogenesis, ways of infection penetration into the fetus,

b) clinical and morphological characteristics of acute and chronic forms of congenital cytomegaly,

c) cytomegalovirus metamorphosis of cells of parenchymal organs, anatomical units where cytomegalic cells are mainly localized.

16. Toxoplasmosis:

a) definition of the disease, etiology and pathogenesis, diagnosis of congenital toxoplasmosis,

b) features of intrauterine infection depending on the duration of pregnancy, outcomes,

c) damage to the brain and other organs in various forms of congenital toxoplasmosis,

d) complications of toxoplasmosis.

17. Mycoplasmosis:

a) morphological and clinical features of Mycoplasma infection pathways in the body,

b) clinical and morphological characteristics of respiratory mycoplasmosis, diagnostics,

c) clinical and morphological characteristics nerespectarea lesions of the organs: lungs, liver, kidneys, Central nervous system, blood vessels,

d) urogenital mycoplasmas, organ for acute and chronic forms,

e) the role of genital mycoplasmas in the genesis of congenital CNS defects, features of congenital mycoplasmosis of the fetus and newborn.

18. Listeriosis:

a) definition of the disease, etiology and pathogenesis, diagnosis of congenital listeriosis,

b) clinical and morphological characteristics of the form of listeriosis:

- anginous-septic

- ocular-glandular

- septic-granulomatous,

c) features of the course of the septic-granulomatous form of listeriosis in the fetus and newborn, complications, outcome.

19. Viral infections. The flu. Etiology, epidemiology, pathogenesis. Clinical morphology of mild, moderate and severe forms of influenza. Complications.

20. Parainfluenza. Etiology. Clinical and morphological characteristics. Complications. Outcomes.

21. Adenovirus infection. Etiology. Clinical and morphological characteristics. Complications. Outcomes.

22. Infections that mainly affect the gastrointestinal tract.

a) Viral enteritis and diarrhea. Features. Morphology. Issue.

b) Campyloid enteritis. Epidemiology, etiology, pathogenesis and morphogenesis, morphological characteristics, clinical manifestations, complications, outcomes, causes of death.

c) Yersiniosis enteritis. Epidemiology, etiopathogenesis, clinical and morphological characteristics, complications, outcomes, causes of death.

23. Typhoid fever.

a) etiology, mechanism of distribution, modern teachings on the pathogenesis of typhoid fever,

b) the main general and local morphological changes in typhoid fever, taking into account the stage of the disease and pathogenesis,

c) clinical and morphological features of the course of the disease in children,

d) intestinal and extra-intestinal complications of typhoid fever.

24. Salmonellosis. The main forms are intestinal, septic, and typhoid. Etiology, pathways of infection, pathogenesis, morphology, complications, outcomes.

25. Cholera. Epidemiology, etiology, pathogenesis and morphogenesis, morphological characteristics, clinical manifestations, complications, outcomes, causes of death.

26. Amoebiasis. Epidemiology, etiology, pathogenesis and morphogenesis, morphological characteristics, clinical manifestations, complications, outcomes, causes of death.

27. Bacterial dysentery.

a) etiology. Ways of infection, modern teaching about the pathogenesis of dysentery, c

) forms of dysentery and their morphological characteristics,

d) outcomes and complications of dysentery,

e) features of the clinical and morphological course in childhood.

28. Quarantine infections. Anthrax. Epidemiology, etiology, pathogenesis and morphogenesis, morphological characteristics, clinical manifestations, complications, outcomes, causes of death.

29. The plague. Epidemiology, etiology, pathogenesis and morphogenesis, morphological characteristics, clinical manifestations, complications, outcomes, causes of death.

MICROPREPARATIONS

1. Healed primary tuberculous pulmonary affect.

2. Miliary tuberculosis of the lung.

3. Tuberculous leptomeningitis.

4. Purulent-necrotic omphalitis.

5. Metastatic ulcers of the kidneys.

6. Metastatic ulcers in the lungs.

7. Diffuse purulent meningitis.

8. Purulent meningitis.

9. Diphtheria angina.

10. Croup of the trachea.

11. Myocarditis in diphtheria.

12. Toxoplasmosis with brain damage.

13. Lung with cytomegaly.

14. Medullary swelling of Peyer's plaques

15. Necrosis of Peyer's plaque:

16. Mesenteric lymph node in typhoid fever

17. Diphtheria colitis:

18. Enteritis in salmonellosis

19. Fibrinous-hemorrhagic laryngotracheitis in severe influenza.

20. Bronchopneumonia in complicated influenza (large variegated lung).

MACROPREPARATIONS.

1. Primary pulmonary tuberculosis complex.

2. Primary pulmonary cavity.

3. Tuberculous lymphadenitis.

4. Miliary tuberculosis of the lungs.

5. Fibrocavernous tuberculosis of the lungs

6. Metastatic ulcers of the kidneys.

7. Metastatic ulcers in the lungs.

8. Septic spleen

9. Polypous-ulcerative endocarditis

10. Diffuse purulent meningitis

11. Purulent meningitis:

12. Necrotic tonsillitis and sharp pharyngeal hyperemia:

13. Croup of the larynx and trachea in diphtheria

14. Acute hemorrhagic glomerulonephritis.

15. Medullary swelling of Peyer's plaques

16. The spleen in typhoid fever.

17. Diphtheria colitis.

18. Follicular ulcerative colitis in dysentery.

19. Acute enteritis in salmonellosis.

20. Influenza tracheitis

21. Bronchopneumonia in complicated influenza.

**Module 4 Clinical Pathological Anatomy**

Theme 1. Organization of the pathological service in the Russian Federation. The procedure for the appointment and conduct of pathological autopsies. Pathological autopsy technique. Lifetime research methods. Goals, objectives and types of morphological research of biological material. Biopsy.

Form (s) of current monitoring of progress (testing, oral questioning, solving situational problems with the registration of a detailed pathological anatomical diagnosis and epicrisis, abstract, report).

Evaluation materials for monitoring progress

CONTROL QUESTIONS

1. Goals, objectives and structure of pathoanatomical service in the system of practical health care in Russia.

2. Organization, methods of work, basic documentation of the pathology Department. Ethical aspects of pathology practice.

3. Statement of biological death. The procedure for reporting a patient's death. Regulations on transportation, preservation and delivery of the body of the deceased.

4. Regulations on pathoanatomical autopsy of corpses. The conditions of appointment of carrying and lifting of postmortem autopsies. Responsibility of medical workers.

5. Structure and rules of registration of the Protocol of pathoanatomical opening of corpses.

6. The principles of design of pathology of the medical report and conclusion about the cause of death.

**Theme 2.** THE ROLE OF IATROGENIC DISEASE IN THE COURSE AND OUTCOME OF THE DISEASE. THE PATHOMORPHOSIS OF THE DISEASE. MEDICAL-CONTROL COMMISSION (MCC) HOSPITAL AND CLINICAL-ANATOMICAL CONFERENCE (CAC). A STUDY OF BIOPSY, SURGICAL MATERIAL AND PLACENTA.

CONTROL QUESTIONS

1. Definition of “iatrogenic pathology” and principles of its classification.

2. Assessment of the role of iatrogenic pathology in the course and outcome of the disease.

3. Definition of "pathomorphosis", causes, types and meaning.

4. The tasks of the MCC and CAC.

5. Organization of work of commissions and conferences, rules of their conduct.

6. Biopsy method of research. Definition. Types of biopsies.

7. Requirements for the processing of biopsy material.

**Theme 3.** MAKING THE DIAGNOSIS IN CLINICAL MEDICINE AND PATHOLOGY PRACTICE. THE PROCEDURE FOR FILLING AND ISSUE OF MEDICAL DEATH CERTIFICATE. ICD-10 IN BUILDING A DIAGNOSIS AND ISSUE A MEDICAL DEATH CERTIFICATE.

CONTROL QUESTIONS

1. The structure of the pathoanatomical diagnosis, the value in clinical practice.

2. Determination of the main, concomitant, competing, background diseases and their complications.

3. Basic principles of pathoanatomical diagnosis in pediatric practice

4. Comparison of clinical and pathological diagnoses.

5. Analysis of medical errors in the wrong diagnosis.

6. Evaluation of the value of incorrect diagnosis in the outcome of the disease.

7. The procedure for filling out and issuing a medical death certificate.

8. The procedure for filling out and issuing a medical certificate of perinatal death.

**Topic 4. Pathology of diagnostic and treatment procedures. Medical and Control Commission. Clinical and anatomical conference. Credit in the section "Clinical pathological anatomy".**

**Form (s) of current monitoring of progress (testing, interview, solving a situational problem with the registration of a detailed pathological anatomical diagnosis and epicrisis, registration of a medical death certificate).**

**Evaluation materials for monitoring progress**

**Test tasks**

U2 Clinical pathological anatomy

U3 Organization of pathoanatomical service in Russia. Autopsy

# THE BASIC TASKS OF THE PATHOLOGICAL ANATOMIC SERVICE INCLUDE

Post-mortem and intravital diagnosis of diseases

Monitoring the quality of clinical diagnosis and the course of the treatment process

Clarification of the structure of causes of death.

+All of the above

True 1 and 3

# ETHICAL STANDARDS FOR A DOCTOR (CLINICIST, PATHOLOGIST) ARE DETERMINED

Skills

Laws and orders

+The moral responsibility of the doctor to society

Ethnic features of the region

True 1 and 2

# THE SYSTEM OF PATHOLOGICAL ANATOMY IS INCLUDED

Pathological departments (prosectorial) as part of medical facilities

Centralized Pathology Departments and Pathological Bureau

Pathomorphological and pathohistological laboratories of research and educational institutes

True 1 and 2

+All of the above

# THE BASIC FUNCTIONAL TASKS OF DOCTORS OF THE PATHOLOGY ANATOMY INCLUDE

Determining the nature of the pathological process on sectional, surgical and biopsy material.

Establishment of the cause and mechanism of death of the patient with the identification of the nature and origin of the disease,

Analysis of the quality of diagnostic and therapeutic work together with the attending physicians, by comparing the clinical and pathological data and diagnoses.

+All of the above

True 1 and 2

# THE LIST OF PROFESSIONAL TASKS OF THE DOCTOR-PATHOLOGIST INCLUDES

Assessment of preliminary information and preparation for morphological research

Autopsy

Study of histological preparations and, if necessary, the involvement of consultants

True 2 and 3

+All of the above

# A SET OF PATHOLOGICAL ANATOMIC DEPARTMENT SPACES INCLUDES

Sectional and storage of corpses

Farewell room with waiting room

Doctors' offices, laboratories with utility rooms. Only 1 and 3

+All of the above

# A SET OF PREMISES FOR A HISTOLOGICAL LABORATORY INCLUDES

Room for receiving and cutting biopsy material. Histological laboratory

Fixation and washing rooms, a room for storing the histological archive

+All of the above

True 2 and 3

# THE CABIN OF THE EQUIPMENT OF THE PATHOLOGICAL ANATOMIC OFFICE INCLUDE

Devices and devices

Car for transporting corpses

Reagents, paints, chemicals, dishes

All of the above

+True 1 and 3

# WHAT STRUCTURAL DIVISIONS ARE INCLUDED IN PATHOANATOMIC EDUCATIONAL, SCIENTIFIC AND PRACTICAL ASSOCIATIONS

Departments of pathological anatomy of medical academies (institutes)

PUB-pathology offices

CPAO-Central pathology departments

Morphological laboratories of the research Institute

+everything is Correct

# THERE ARE THE FOLLOWING TYPES OF DEATH

Biological

Violent

Death caused by illness

Intrauterine

+Everything is correct

# BIOLOGICAL DEATH IS

+Complete and irreversible termination of the vital functions of the body and, above all, the systems that regulate these functions

Death resulting from any violent actions from outside (accidents, trauma, murder, suicide, poisoning)

Complete but reversible termination of the vital functions of the body and, above all, the systems that regulate these functions

Death in the midst of visible health, unexpected for others, if the deceased has no obvious manifestations of a fatal disease. Everything is correct

# SUDDEN DEATH IS

Complete and irreversible termination of the vital functions of the body and, above all, the systems that regulate these functions

Death resulting from any violent actions from outside (accidents, trauma, murder, suicide, poisoning)

Complete but reversible termination of the vital functions of the body and, above all, the systems that regulate these functions

+Death among visible health, unexpected for others, in the absence of the deceased's obvious manifestations of a fatal disease That's right

# SUDDEN DEATH IS A SPECIAL CASE

Natural death

Violent death

+Death from disease

Intrauterine death

Everything is correct

# VIOLENT DEATH IS

Complete and irreversible termination of the vital functions of the body and, above all, the systems that regulate these functions

+Death resulting from any violent actions from outside (accidents, trauma, murder, suicide, poisoning)

Complete but reversible termination of the vital functions of the body and, above all, the systems that regulate these functions

Death among visible health, unexpected for others, in the absence of the deceased's obvious manifestations of a fatal disease

Everything is correct

# CLINICAL DEATH IS

Complete and irreversible termination of the vital functions of the body and, above all, the systems that regulate these functions

Death resulting from any violent actions from outside (accidents, trauma, murder, suicide, poisoning)

+Complete but reversible termination of the vital functions of the body and, above all, the systems that regulate these functions

Death among visible health, unexpected for others, in the absence of the deceased's obvious manifestations of a fatal disease

Everything is correct

# "EARLY AUTOPSIES" INCLUDE

+Autopsies performed within the first hour after the biological death has been confirmed

Autopsies performed within the first 30 minutes after the biological death is confirmed

Autopsies performed within the first day after the biological death is confirmed

Autopsies performed within the first 3 hours after the biological death has been confirmed

# HOW LONG AFTER DEATH IS USUALLY DELIVERED THE CORPSE FROM THE MEDICAL INSTITUTION TO THE PAO

+Not earlier than 2 hours after the biological death was confirmed

1 hour after the biological death was confirmed

Immediately after the biological death is confirmed

1 day after the biological death was confirmed

# LIST THE OBJECTIVE SIGNS OF DEATH

Cadaverous spots.

the cooling of the corpse

drying of the skin and mucous membranes

shells

corneal Opacity and pupil dilation

+Everything is correct

# WHAT SHOULD THE PATHOLOGIST DO IF DURING THE AUTOPSY SUSPICION AROSE OR SIGNS OF VIOLENT DEATH WERE FOUND

the Autopsy shall be suspended

the chief medical officer or his Deputy shall be Immediately notified of this

Take measures to preserve the corpse, its organs and tissues for further forensic research

A protocol is issued for the part of the autopsy performed (in the generally accepted form)

+Everything is correct

# DOCUMENTATION OF THE SECTION OF THE WORK OF THE PATHOLOGICAL ANATOMIC DEPARTMENT INCLUDES

Books of registration of postmortem autopsy and extradition of corpses

Protocol (map) of the pathological examination

Journal of accounting biopsy and surgical material

+True 1 and 2

All of the above

# THE CHOICE OF THE METHOD AND PROCEDURE FOR CONDUCTING A PATHOANATOMIC AUTOPSY IS NOT AFFECTED BY THE REQUIREMENTS:

Effective and safe work of employees

pathology Department

Elimination of environmental pollution

Complete examination of the organs and systems of the deceased

+Requests from relatives of the deceased

Exclusion of actions leading to disfigurement of the corpse

# Have the right to attend an autopsy

Relatives of the deceased.

Doctors of the department where the patient died.

The attending physicians.

All of the above.

+Only 2 and 3

# sowing FOR BACTERIOLOGICAL RESEARCH ARE NOT PRODUCED

from organs not removed from the corpse, using a sterile tool after cauterizing the incision surface, injection

From the surface of the organ, until it comes into contact with non-sterile objects

Using blood from the right atrium, ulnar vein (before opening the skull)

+Using blood from an open aorta or inferior Vena cava

Everything is correct

# CANCELLATION OF autopsy IS NOT PERMITTED IN CASES

Staying a patient in a medical institution for less than a day

Suspected violent death and the presence of infectious diseases

An unclear lifetime diagnosis (regardless of the length of stay in the hospital), and after carrying out diagnostic and therapeutic measures that cause death

True 2 and 3

+All of the above

# IN PEDIATRIC PRACTICE autopsy IS SUBJECT

All, without exception, abortions, stillbirths and newborns who died in a medical institution

Newborns with a gestation period of more than 22 weeks

All stillborn with a body weight of more than 500 g

All of the above

+True 2 and 3

# PROTECTIVE SUIT CONSISTS OF

Overalls with a hood (kerchief), gauze mask

Anti-plague and medical gowns, rubber boots, socks, towels, rubberized aprons, arm ruffles

Two pairs of rubber gloves and goggles

True 1 and 2

+All of the above

# DURING THE PATHOLOGICAL ANALYSIS OF THE DEATH OF SPECIALLY DANGEROUS INFECTIONS, GUIDED

Features of the autopsy of the dead from infectious diseases

The established mode of operation of quarantine institutions

The requirements of instructional materials on the mode of working with material infected or suspected to be infected by pathogens of plague, cholera, smallpox, anthrax, tularemia and brucellosis

Right 2 and 3

+All of the above

# THE FOLLOWING DOCUMENTS ARE NOT REQUIRED FOR AUTOPSY

Protocol of pathoanatomic research

Medical certificate of death

Conclusion on the cause of death

+Sick leave

Everything is correct

# EXTRACTION OF ORGANS BY THREE COMPLEXES IS CHARACTERISTIC OF THE METHOD

+Abrikosov method

Virchow method

Shore method

Everything is correct

# EXTRACTION OF THE ENTIRE ORGAN COMPLEX DURING OPENING IS CHARACTERISTIC OF THE METHOD

Abrikosov method

Virchow method

+Shore method

Everything is correct

# EXTRACTION OF INDIVIDUAL ORGANS DURING AUTOPSY IS CHARACTERISTIC OF THE METHOD

Abrikosov method

+Virchow method

Shore method

Everything is correct

# DURING AUTOPSY, EXTRACTION METHODS CAN BE USED

Individual bodies

bodies on systems

Total organ complex

+Everything is correct

# THE SPECIFICS OF CONDUCTING AUTOPSIES OF CORPSES IN INFECTIOUS DISEASES DO NOT APPLY

the Presence of 15-20 liters of disinfectants and a Mat richly moistened with a disinfectant solution in front of the door leading to the sectional area

using an anti-plague suit

+possibility of accumulation and disinfection of liquids washed off from the sectional table

Presence of the attending doctor at the autopsy

Everything is correct

# When taking the material for bacteriological and virological research under the conditions of the pathology department, it is necessary to use

Sterile loops, spatulas, syringes and Pasteur pipettes

A set of culture media (broth, agar)

Petri dishes

All of the above

+True 1 and 3

# THE PROTOCOL OF THE PATHOLOGICAL ANALYSIS INCLUDES THE FOLLOWING SECTIONS

Passport part, clinical diagnoses, protocol part

Pathological diagnosis

A brief extract from the medical death certificate, brief clinical data and clinical anatomical epicrisis

+All of the above

True 1 and 2

# NEW FORMS OF THE ORGANIZATION OF PATHOLOGICAL ANATOMIC SERVICE RECEIVING A WIDE DISTRIBUTION IN THE LAST DECADES

Central Pathological Department (CPD).

Pathological Bureau (PB).

Pathology department (PD).

All of the above

+True 1 and 2

# What are the basic structural divisions of the pathoanatomical service in practical health care?

Pathology department.

Pathological Bureau: city, regional, republican

Department of academies, institutes for the improvement of doctors.

+True 1 and 2

All of the above

# INDEPENDENT ("INDEPENDENT") INSTITUTIONS OF THE PATHOLOGICAL ANATOMIC SERVICE

Pathological departments (including centralized) of medical institutions

Regional institutes of pathology

Pathological departments (departments, laboratories, groups in departments) of research institutes

Republican, regional, city, municipal pathological anatomical bureaus

+True 2 and 4

 # ANNUAL LOAD OF SECTIONAL WORK of the first category of complexity FOR ONE STAFF POSITION OF THE DOCTOR OF THE GENERAL SOMATIC TREATMENT AND PREVENTIVE INSTITUTION MAKES

100 autopsies

125 autopsy

150 autopsies

+200 autopsies

175 autopsies

 # ANNUAL LOAD OF SECTIONAL WORK of the second category of complexity FOR ONE STAFF POSITION OF THE DOCTOR OF THE GENERAL SOMATIC TREATMENT AND PREVENTIVE INSTITUTION MAKES

100 autopsies

125 autopsy

150 autopsies

200 autopsies

+175 autopsies

 # ANNUAL LOAD OF SECTIONAL WORK of the third category of complexity FOR ONE STAFF POSITION OF THE DOCTOR OF THE GENERAL SOMATIC TREATMENT AND PREVENTIVE INSTITUTION MAKES

100 autopsies

125 autopsy

+150 autopsies

200 autopsies

175 autopsies

 # ANNUAL LOAD OF SECTIONAL WORK of the fourth category of complexity FOR ONE STAFF POSITION OF THE DOCTOR OF THE GENERAL SOMATIC TREATMENT AND PREVENTIVE INSTITUTION MAKES

100 autopsies

+125 autopsy

150 autopsies

200 autopsies

175 autopsies

 # ANNUAL LOAD OF SECTIONAL WORK of the fifth category of complexity FOR ONE STAFF POSITION OF THE DOCTOR OF THE GENERAL SOMATIC TREATMENT AND PREVENTIVE INSTITUTION MAKES

+100 autopsies

125 autopsy

150 autopsies

200 autopsies

175 autopsies

# ANNUAL LOAD OF SECTIONAL WORK OF THE DOCTOR-PATHOLOGIST OF CHILDREN'S PATHOLOGY AND ANATOMY COMPOSITIONS

100 autopsies

125 autopsies.

+175 autopsies

200 autopsies

150 autopsies

# pathoanatomical autopsy (macroscopic examination) without histological examination is

+Pathoanatomical autopsy of the category I of complexity

Pathoanatomical autopsy of the category II of complexity

Pathoanatomical autopsy of the category III of complexity

Pathoanatomical autopsy of the category IV of complexity

Pathoanatomical autopsy of the category V of complexity

# pathoanatomical autopsy of a fetus, stillborn or deceased newborn is

Pathoanatomical autopsy of the category I of complexity

+Pathoanatomical autopsy of the category II of complexity

Pathoanatomical autopsy of the category III of complexity

Pathoanatomical autopsy of the category IV of complexity

Pathoanatomical autopsy of the category V of complexity

# pathoanatomical autopsy with an established clinical diagnosis, including complications of the underlying disease, in the absence of uncertainty in the interpretation of the mechanisms and cause of DEATH IS

Pathoanatomical autopsy of the category I of complexity

+Pathoanatomical autopsy of the category II of complexity

Pathoanatomical autopsy of the category III of complexity

Pathoanatomical autopsy of the category IV of complexity

Pathoanatomical autopsy of the category V of complexity

# pathoanatomical autopsy in cases of death after surgery, when difficulties arise in the interpretation of the essence pathoanatomical process, mechanisms and causes of death Is

Pathoanatomical autopsy of the category I of complexity

Pathoanatomical autopsy of the category II of complexity

+Pathoanatomical autopsy of the category III of complexity

Pathoanatomical autopsy of the category IV of complexity

Pathoanatomical autopsy of the category V of complexity

# pathoanatomical autopsy in case of combined underlying disease or polypathy, in the presence of defects in diagnosis and treatment, which caused difficulties in interpreting the nature of the pathoanatomical process, mechanisms and causes of death Is

Pathoanatomical autopsy of the category I of complexity

Pathoanatomical autopsy of the category II of complexity

Pathoanatomical autopsy of the category III of complexity

+Pathoanatomical autopsy of the category IV of complexity

Pathoanatomical autopsy of the category V of complexity

# pathoanatomical autopsy with an unidentified clinical diagnosis of the underlying disease, when there are difficulties in interpreting the nature of the pathoanatomical process and the cause of death, or it is necessary to use additional immunohistochemical, molecular biological, electron microscopic research methods Is

Pathoanatomical autopsy of the category I of complexity

Pathoanatomical autopsy of the category II of complexity

Pathoanatomical autopsy of the category III of complexity

Pathoanatomical autopsy of the category IV of complexity

+Pathoanatomical autopsy of the category V of complexity

# ANNUAL LOAD OF A DOCTOR-PATHOLOGIST EXECUTING ONLY A BIOPSY STUDY, MAKES UP

1000 biopsies

2000 biopsies

3000 biopsies

+4000 biopsies

5000 biopsies

U3 Building a diagnosis and issue a medical death certificate

# TAKING INTO ACCOUNT THE NUMBER OF DISEASES DETECTED, PATHOLOGICAL ANATOMIC DIAGNOSIS MAY BE

Monocausal

Bicausal

Polypathic

All of the above

+True 1 and 2

# DIAGNOSIS START

+From the nosological unit ("keyword", statistical units) - the underlying disease - the original cause of death

From the immediate cause of death

From the pathological process that launched the pathogenetic chain

From all of the above

# PATHOLOGICAL DIAGNOSIS STRUCTURE SHOULD RESPOND TO THE FOLLOWING PRINCIPLES

Nosological (taking into account the requirements of the ICD)

Intranosological

Pathogenetic

+All of the above

Only 2 and 3

# BASIC DISEASE - NOSOLOGICAL UNIT WHICH AT THIS TIME AND IN THESE CONDITIONS TO THE MOST DEGREE

Threatens the life, health, working capacity of the patient

Requires initial treatment and preventive measures

Itself or through complications was the cause of death.

+Correctly 1 and 3

All of the above

# The immediate cause of death is

Nosological unit (syndrome, trauma), followed by biological death

The nosological unit that caused the thanatogenetic process

The mechanism of death.

Right 2 and 3

+All of the above

# STRUCTURE OF PATHOLOGICAL ANATOMIC DIAGNOSIS INCLUDES

Underlying disease

Complications

Concomitant diseases

+All of the above

True 1 and 2

# STRUCTURE OF PATHOLOGICAL ANATOMIC DIAGNOSIS IN THE PRESENCE OF A COMBINED BASIC DISEASE INCLUDES

Two competing diseases

The main and background disease

Two concomitant diseases

+All of the above

True 2 and 3

# Combined primary disease includes

Two competing diseases

Two conjunction diseases

Main and background diseases

+Everything is correct

# nosological units, each of which, by itself or through its complications, could be fatal

+Competing diseases

Conjunction diseases

Background diseases

Everything is correct

# diseases that etiologically unrelated to the main one, but included in the general pathogenesis with the main disease, was one of the reasons for its development, later aggravated the course and contributed to the development of fatal complications that were fatal

Competing diseases

Conjunction diseases

+Background diseases

Everything is correct

# nosological units that accidentally matched in time and mutually aggravating each other, but separately, they could not be fatal

Competing diseases

+Conjunction diseases

Background diseases

Everything is correct

# CORRECT REGISTRATION OF A MEDICAL DEATH CERTIFICATE REQUIRES THE FOLLOWING CONDITIONS

The main disease (the initial cause of death) is recorded in the last of three lines (a, b, c) taking into account the number of previously filled lines

The main disease is recorded only in the third line (in)

The immediate cause of death is written only on the top line (a)

All of the above

+True 1 and 3

# COMPLICATIONS OF INTENSIVE THERAPY AND RESUSCITATION REFLECT IN PATHOLOGICAL DIAGNOSIS IN A LINE

Complications of the underlying disease

Among concomitant diseases

+In a separate line after concomitant diseases

# CLINICAL AND PATHOLOGICAL EPICRISIS INCLUDES THE FOLLOWING BASIC DATA

Anamnestic and clinical

Laboratory and radiological

Pathological and histological.

+All of the above

True 1 and 3

# CLINICAL AND ANATOMIC EPICRYSIS REFLECT

Clinical and anatomical justification for the diagnosis of the underlying disease (initial cause of death) and fatal complications (immediate cause of death)

Conclusion on the cause of death of the patient

Comparison of clinical and pathological diagnoses with the characteristic of discrepancies

+All of the above

True 1 and 2

# WHEN CLINICAL AND PATHOLOGICAL DIAGNOSES ARE COMPARED, THE FOLLOWING OF CATEGORIES OF DIFFERENCE ARE INSTALLED

According to the diagnosis of the underlying disease

For the most important complications that significantly changed the course of the underlying disease or caused death

For the second disease in the combined main (in the presence of two competing, combined, the main with the background)

True 1 and 2

+All of the above are correct.

# THE REASONS FOR DIFFERENCES OF CLINICAL AND PATHOLOGICAL DIAGNOSIS ARE

Inadequate examination of the patient and objective difficulties of the study

Underestimation of clinical and laboratory data

Reassessment of clinical and laboratory data

+All of the above

True 1 and 2

# THE REASONS FOR DIFFERENCES OF CLINICAL AND PATHOLOGICAL DIAGNOSIS ARE

Underestimation and revaluation of radiological and other functional data

Incorrect registration and construction of diagnoses

Other reasons

+All of the above

True 1 and 2

# To which category the case of DIFFERENCE of clinical and pathologic diagnosis, if the disease was not recognized in this health institutions, but not timely diagnosis would have been a positive influence on outcome, but a correct diagnosis could be determined

category 1

+category 2

category 3

All of the above

# WHAT CATEGORY DOES A CASE OF DIFFERENCE BETWEEN CLINICAL AND PATHOANATOMIC DIAGNOSES BELONG TO, IF THE DISEASE WAS NOT RECOGNIZED AT PREVIOUS STAGES, AND IN THIS MEDICAL AND PREVENTIVE INSTITUTION IT WAS IMPOSSIBLE TO ESTABLISH A CORRECT DIAGNOSIS DUE TO OBJECTIVE REASONS

+category 1

category 2

category 3

All of the above

# TO WHICH CATEGORY DOES THE CASE OF DIFFERENCE OF CLINICAL AND PATHOLOGICAL DIAGNOSIS RELATED IF THE WRONG DIAGNOSIS LED TO THE WRONG MEDICAL TACTICS THAT PLAYED A CRUCIAL ROLE IN THE ONSET OF DEATH?

category 1

category 2

+category 3

All of the above

# HOW MUCH TIME OF THE ROOT OF MEDICAL CERTIFICATES IS TO BE STORED AT THE PLACE OF THEIR ISSUANCE

+within 1 year

within 1 month

for 5 years

for 10 years

# SUBJECTIVE REASONS FOR DIFFERENCES OF DIAGNOSIS INCLUDE EVERYTHING EXCEPT

inadequate examination of the patient

underestimation of anamnestic data

underestimation of clinical data

+the short stay of the patient in a medical institution

# SELECT THE OBJECTIVE REASONS FOR MEDICAL ERRORS

+the Short duration of the patient's stay in the hospital

Defects in laboratory, hardware, instrumental, and other studies

Complexity and lack of knowledge of the disease

Underestimating or overestimating the role of consultants

Incorrect wording of the final diagnosis

# OBJECTIVE REASONS FOR DIFFERENCES OF DIAGNOSIS INCLUDE

+the severity of the patient's condition

inadequate examination of the patient

underestimation of anamnestic data

underestimation of clinical data

U3 Medical-control commission hospital and clinical-anatomical conference. Iatrogenia. Biopsy

# AT THE CLINICAL AND ANATOMICAL CONFERENCE DISCUSS

Cases of discrepancy between clinical and pathoanatomic diagnosis

Rare observations, unusual diseases, cases of drug pathology

Cases of death of patients after surgical, diagnostic and therapeutic interventions

+All of the above

True 1 and 2

# CLINICAL AND ANATOMICAL CONFERENCES DISCUSS

Cases of acute infectious diseases

Cases of delayed diagnosis and deaths that remain unclear

Report of the head of the pathology Department

All of the above

+True 2 and 3

# IATROGENIA IN THE FINAL CLINICAL AND PATHOANATOMIC DIAGNOSES MAY NOT BE PART OF THE

the Main disease

Concomitant disease

+Complications of the underlying disease

a Competing disease

Combined disease

# FOR A COMPLETE MORPHOLOGICAL DIAGNOSIS OF THE DISEASETHE ATTENDING PHYSICIAN IS NOT REQUIRED TO PROVIDE

Marking of research objects

Fixing the objects of research

Specifying the exact number of objects

filling in two copies of the referral for histological examination

+Visa of the chief doctor (or his Deputy for the medical part) for research

# UNIVERSAL WIDELY USED FIXING LIQUID

Distilled water

+10 % solution of neutral buffered formalin

96-100% ethyl alcohol

Liquid Karnua

# OPTIMAL FOR THE PREVENTION OF AUTOLYSIS IN THE OBJECTS OF RESEARCH (BIOPSIES, PIECES OF TISSUE) VOLUME OF THE FIXING FLUID

+10-50 times the volume of the object

2 times the volume of the object

Equal to the volume of the object

the Liquid covers the surface of the object

# IN THE DIRECTION OF HISTOLOGICAL EXAMINATION OF DIAGNOSTIC ENDOMETRIAL SCRAPING, THE GYNECOLOGIST DOES NOT INDICATE

Detailed clinical diagnosis

Results and coordinates of previous histological studies

the Date of the beginning and end of the last menstruation or bleeding

the Nature of menstrual function disorders

+Nationality of the woman

# INTRAOPERATIVE (URGENT) HISTOLOGICAL EXAMINATION SHOULD BE PERFORMED WITHIN

+Up to 20-25 min

Up to 1 hour

within 5 days

Up to 10 days

Up to 20-30 days

# DIAGNOSTIC (PLANNED) THE HISTOLOGICAL EXAMINATION SHOULD BE PERFORMED WITHIN

Up to 20-25 min

Up to 1 hour

+within 5 days

Up to 10 days

Up to 20-30 days

# DIAGNOSTIC HISTOLOGICAL EXAMINATION OF BONE TISSUE SHOULD BE CARRIED OUT WITHIN

Up to 20-25 min

Up to 1 hour

within 5 days

+Up to 10 days

Up to 20-30 days

# THE RANGE OF PERSONS TO WHOM INFORMATION ABOUT THE RESULTS OF MORPHOLOGICAL RESEARCH IS TRANSMITTED WITHOUT THE PRIOR CONSENT OF THE PATIENT OR HIS LEGAL REPRESENTATIVE INCLUDES

+the Attending physician and the head of the Department where the patient is located

Other officials - in the interests of examination and treatment of the patient

Employees of medical institutions-for research and publication in scientific literature

Officials for use in the educational process

# WITHOUT THE CONSENT OF THE PATIENT OR HIS LEGAL REPRESENTATIVE, INFORMATION CONSTITUTING A MEDICAL SECRET IS TRANSFERRED TO OFFICIALS IN THE FOLLOWING SITUATIONS, EXCEPT

for the purpose of examination and treatment of an incapacitated citizen

at the risk of spreading infectious diseases, mass lesions and poisoning

in cases of assistance to a minor under the age of 15

+for publication in scientific literature, use in the educational process

at the request of the bodies of inquiry and investigation, the Prosecutor and the court

# IS IT ALLOWED TO DIVIDE OBJECTS FOR SENDING TO DIFFERENT LABORATORIES

Allowed

+Not allowed

# THE IMPORTANCE OF BIOPSY IN PRACTICAL HEALTH CARE

Confirms and clarifies the clinical diagnosis

is the basis for the formulation of the final clinical diagnosis

Determination of histogenesis and initial stages of diseases

Study of the dynamics of the development of the pathological process

+All of the above is true

# Who is responsible for delivering the biopsy material to the pathology Department

Attending physician

the Doctor who prescribed the study (consultant)

Chief medical officer

True 1 and 3

+True 1 and 2

# CATEGORY I IATROGENIES ARE

pathological processes, reactions and complications caused by medical exposure, carried out according to reasonable indications and performed correctly

pathological processes, unusual fatal reactions, including those caused by inadequate, erroneous or incorrect medical influences, which were the cause of death

+pathological processes, reactions that are not pathogenetically related to the main disease or its complication and do not play a significant role in the overall thanatogenetic assessment of the case

# CATEGORY II IATROGENIES ARE

+pathological processes, reactions and complications caused by medical exposure, carried out according to reasonable indications and performed correctly

pathological processes, unusual fatal reactions, including those caused by inadequate, erroneous or incorrect medical influences, which were the cause of death

pathological processes, reactions that are not pathogenetically related to the main disease or its complication and do not play a significant role in the overall thanatogenetic assessment of the case

# CATEGORY III IATROGENIES ARE

pathological processes, reactions and complications caused by medical exposure, carried out according to reasonable indications and performed correctly

+pathological processes, unusual fatal reactions, including those caused by inadequate, erroneous or incorrect medical influences, which were the cause of death

pathological processes, reactions that are not pathogenetically related to the main disease or its complication and do not play a significant role in the overall thanatogenetic assessment of the case

# VARIETIES OF IATROGENY INCLUDE

pharmacological iatrogenic

radiation iatrogenies

instrumental diagnostic iatrogenies

surgical iatrogenies

+All of the above is true

# IN THE PATHOANATOMIC DIAGNOSIS OF CATEGORY I IATROGENIA OCCUPY A PLACE IN THE rubric

the main disease

+concomitant disease

background of the disease

competing diseases

complications of the underlying disease

# IN THE PATHOANATOMIC DIAGNOSIS OF CATEGORY II IATROGENIA OCCUPY A PLACE IN THE RUBRIC

complications of the underlying disease

combined disease

background of the disease

competing diseases

+All of the above is true

# IN THE PATHOANATOMIC DIAGNOSIS OF CATEGORY III IATROGENIA OCCUPY A PLACE IN THE RUBRIC

+the main disease

concomitant disease

background of the disease

competing diseases

complications of the underlying disease

# ACCORDING to I. V. TIMOFEEV, a NEW PATHOLOGICAL PROCESS that DEVELOPED as a RESULT of the SHOWN and CORRECTLY performed INTERVENTION, which AFFECTED the OUTCOME of the DISEASE, SHOULD be CONSIDERED AS

+complication

iatrogenic

medical care defect

# ACCORDING to I.V. TIMOFEEV, a NEW PATHOLOGICAL PROCESS that DEVELOPED as a RESULT of the SHOWN and CORRECTLY performed INTERVENTION, which did NOT AFFECT the OUTCOME of the DISEASE, SHOULD be CONSIDERED AS

+complication

iatrogenic

medical care defect

# ACCORDING to I.V. TIMOFEEV, a NEW PATHOLOGICAL PROCESS that DEVELOPED as a RESULT of the INDICATED, but INCORRECTLY PERFORMED INTERVENTION, which AFFECTED the OUTCOME of the DISEASE, SHOULD be CONSIDERED

complication

+iatrogenic

the defect of medical care

# ACCORDING to I.V. TIMOFEEV NEW PATHOLOGICAL PROCESS that DEVELOPED as a result is SHOWN, BUT MADE a WRONG INTERVENTION, NOT affect the OUTCOME of the DISEASE SHOULD be CONSIDERED

complication

iatrogenic

+the defect of medical care

# TRAUMATIC COMPLICATIONS OF CARDIAC RESUSCITATION INCLUDE

+broken ribs and sternum

cardiopulmonary syndrome

postanaoxic encephalopathy

# DISEASES OF THE ANIMATED ORGANISM INCLUDE

Malory-Weiss traumatic syndrome

broken ribs and sternum

+cardiopulmonary syndrome

# NON-INFECTIOUS COMPLICATIONS OF TRACHEOSTOMY INCLUDE

+pressure ulcers of the mucous membranes

purulent thyroiditis

laryngitis

# COMPLICATIONS OF VENTILATOR INCLUDE

postanoxic encephalopathy

posttoxic gastroenteropathy

+inflating the stomach with air

# IN CASES OF DIAGNOSIS OF MALIGNANT TUMORS, INFECTIOUS DISEASES, DISEASES REQUIRING HORMONAL, RADIATION, CYTOSTATIC THERAPY AND SURGICAL INTERVENTIONS, THE PATHOHISTOLOGICAL CONCLUSION SHALL BE SIGNED

pathologist

Head of the pathology Department

Chief medical officer

the Attending oncologist

+Pathologist and head of the pathology Department

# MANDATORY FORMS OF MEDICAL DOCUMENTATION IN THE PATHOANATOMIC DEPARTMENT FOR THE STUDY OF BIOPSIES, OPERATING MATERIAL, AND AFTERBIRTH

alphabetical journal of research registration

Forms of form 014 / u "Referral to histological research" with the results of morphological research, reset in the book

Journal of registration of results of lifetime morphological studies

Log of registration of the issue of pathoanatomic conclusions

+Everything is correct

# THE COMPONENT OF THE MORPHOLOGIST'S RESPONSE IN THE STUDY OF OPERATIONAL MATERIAL IS NOT

Macroscopic description

+Description of the fixing method

Histological description

Conclusion (diagnosis)

Everything is correct

# ORGANS AND TISSUES ARE SUBJECT TO MANDATORY MORPHOLOGICAL EXAMINATION

only in unclear cases

to clarify the dynamics of the disease

to clarify the nature and severity of the injury

removed during surgery

+everything is True

**Criteria used for assessing students at midterm attestation**

*(The disciplinary rating is calculated as follows:*

*if the form of midterm attestation in the discipline – final test: RD = Rc + Rb + Rt,*

***Rb –*** *bonus rating;*

***Rd –*** *disciplinary rating;*

***Rt-*** *test rating;*

***Rc –*** *current rating (Rating score for practical training (for completing must-have skills);*

**Assessment criteria for practice test**

|  |  |
| --- | --- |
| **Control form** | **Assessment criteria** |
| Pass | 1. Keeping a diary |
| 1. Making a practice report |
| 1. Practical skill demonstration |
| 1. Characteristics given by the head of a medical organization |

**11-15 points.** If there is no failure of meeting the deadlines for handing over reporting documents, all documentation is drawn up in accordance with the requirements; there is a positive characteristic from the practice place. A student demonstrated high activity during the practice, good practical skills during the test. The answers to the questions posed are presented logically, consistently and do not require additional explanations. The causal relationships between phenomena and events are fully disclosed. Reasonable conclusions are made. A student demonstrates in-depth knowledge of the basic regulatory legal acts. The norms of literary speech are observed.

**6-10 баллов.** If there is no failure of meeting the deadlines for handing over reporting documents, the reporting documentation contains minor errors and shortcomings, indicating a certain decrease in the level of professionalism in performing tasks. A student was given a positive characteristic from the place of practice. A student demonstrates practical skill with minor errors, but without gross violations of the algorithm. The answers to the questions posed are presented in a systematic and consistent manner. The material is presented with confidence. The causal relationships between phenomena and events are revealed. The ability to analyze the material is demonstrated, but not all conclusions are reasoned and evidence-based. The norms of literary speech are observed. (Test: the number of correct answers> 70%).

**3-5 баллов.** Minor failure of meeting the deadlines for handing over reporting documents without a valid reason, there are errors and shortcomings in the reporting documentation, indicating a decrease in the level of professionalism in performing tasks. A student demonstrated practical skill with one / two gross mistakes. In the answer there are violations in the sequence of presentation. The causal relationships between phenomena and events are not fully disclosed. Superficial knowledge of the issue is demonstrated, specific tasks are difficult to solve. There are difficulties with conclusions. Violations of the norms of literary speech are allowed. (Test: the number of correct answers> 50%).

**0-2 балла.** The documentation is performed with serious comments. There is no positive characteristic from the place of work. The material is presented inconsistently, does not represent a specific system of knowledge in the discipline. The cause-and-effect relationships between phenomena and events are not disclosed. Analysis is not performed. There are no conclusions. There are no answers to additional questions. There are noticeable violations of the norms of literary speech. (Test: the number of correct answers <50%).

**Examination ticket pattern**

FEDERAL STATE BUDGETARY EDUCATIONAL INSTITUTION

OF HIGHER EDUCATION

«Orenburg State Medical University»

of the Health Ministry of Russia

the department of pathological anatomy

the direction of training (speciality): 31.05.01 General medicine

discipline: pathological anatomy, clinical pathological anatomy

**Examination ticket/card № 1**

**I. THEORETICAL QUESTIONS**

**1. Pathological anatomy.** The content, objectives, objects, methods and levels of study. Pathoanatomical service and its importance in the health care system.

**2. Atherosclerosis and arteriosclerosis.** Modern views on the etiology and pathogenesis of atherosclerosis. Morphological characteristics and stages of atherosclerosis, the structure of an atherosclerotic plaque.

**II. PRACTICAL PART**

**Stomach cancer (adenocarcinoma)** (you need to describe macro- and microscopic changes in an organ and / or tissue).

Head of the Pathological Anatomy department

MD, PhD, Professor V.S. Polyakova

Dean of the Foreign students faculty

MD, PhD A.O. Mironchev

**A list of didactic materials for students at midterm attestation.**

**Examination questions on pathological anatomy**

1. Pathological anatomy. The content, objectives, objects, methods and levels of study/

2. Methods of research in pathological anatomy. Autopsy. The value of the study of cadaveric material substrates derived from patients with life, the experimental material. Histological examination. Cytology, immunohistochemistry, electron microscopy. Biopsy - species importance in clinic.  
3. Necrosis. Reasons, mechanisms of development, morphological characteristics. Clinical and morphological forms of necrosis, pathological morphogenesis

4. Apoptosis programmed cell death. Determining the mechanisms of development, morphological characteristics and methods of diagnosis. Stages of apoptosis. Meaning of apoptosis in physiological and pathological processes.  
5. Dystrophy as a kind of tissue damage. Functional and morphological essence dystrophy. Etiological factors, the main pathogenetic links dystrophy, morphogenesis. Principles of classification dystrophies.  
6. Intracellular accumulation: definition, mechanisms of development. The accumulation of lipids (lipidoses): causes, pathological and morphogenesis, clinical and morphological characteristics, diagnostic methods, and outcomes. Steatosis. Fatty changes in the myocardium, liver, kidney.  
7. The accumulation of proteins (disproteinozy): causes, pathological and morphogenesis, morphological characteristics and methods of diagnosis, clinical symptoms and syndromes, consequences.  
8. The accumulation of glycogen: causes, pathological and morphogenesis, morphological characteristics and methods of diagnosis, clinical manifestations, consequences. Acquired and congenital glycogen storage.  
9. Stromal vascular degeneration. Definition, basic terms and mechanisms of classification. Protein stromal-vascular dystrophy: mucoid swelling, fibrinoid swelling, determination, morphological manifestations, outcomes.  
10. Fat vascular stromal dystrophies, definition, principles of classification. Causes of obesity, morphological changes in the organs, the clinical significance, outcomes of local and general obesity.

11. Hyaline changes. Intracellular and extracellular hyaline: morphogenesis, morphological characteristics. Hyaline changes in various pathological conditions.  
12. Metabolic pigments (chromoproteids). Exogenous pigments. Endogenous pigments: species formation mechanism, morphological characteristics and methods of diagnosis.  
13. Metabolic gemoglobinogennye pigments. Main causes and morphological changes in the body in violation of hemoglobin metabolism. Hemosiderosis (local, systemic), hemochromatosis.  
14. Disorders of bilirubin metabolism, morphological characteristics. Jaundice. Classification of the causes and mechanisms of development of jaundice.  
15. Disorders of the exchange of lipofuscin: clinical and morphological characteristics.  
16. Disorders of the exchange of melanin: clinical and morphological characteristics.  
17. Pathological calcification (calcifications). Types of calcifications: dystrophic, metastatic. Reasons pathological and morphogenesis, morphological characteristics, diagnosis, clinical manifestations, outcomes.  
18. Venous congestion: general and local, acute and chronic. Local venous congestion causes morphological manifestations, outcomes.  
19. Venous congestion in the pulmonary circulation: morphogenesis and pathogenesis, clinical and morphological characteristics, outcomes.  
20. Venous stasis in the systemic circulation: patho- and morphogenesis, clinical and morphological characteristics, outcomes. Venous congestion in the portal vein (portal hypertension): pathogenesis and clinical and morphological manifestations.

21. Shock. Definition, types, mechanisms of development, stage, morphological characteristics of internal organs in shock, clinical manifestations, consequences.  
22. Bleeding: external and internal hemorrhage. The reasons, types, clinical and morphological characteristics. Hemorrhagic diathesis.  
23. The syndrome of DIC (disseminated intravascular coagulation): definition, causes, mechanisms of development. Characteristic stages. Pathological changes  
24. Thrombosis. The definition of local and general factors of thrombosis. A blood clot, its types, morphological characteristics. Venous thrombosis. Thrombosis of the arteries. Thrombosis in the cavities of the heart.   
25. Embolism: definition, types, causes, morphological characteristic. Thromboembolism: causes of development, clinical significance. Pulmonary embolism, acute pulmonary heart. Thromboembolic syndrome: clinical and morphological characteristics.  
26. Ischemia. Determining the causes, mechanisms of development, morphological characteristics and methods of diagnosis, clinical significance. Acute and chronic ischemia. Infarction: definition, causes, classification, morphological characteristics of different types of infarction, complications, outcomes.  
27. Inflammation: definition, nature and biological significance. Morphological characteristics of the phases of inflammation. Principles of classification of inflammation.   
28. Acute inflammation. Etiology and pathogenesis. The reaction of the blood vessels in acute inflammation. Transudate, exudate, edema, stasis. The emigration of leukocytes, pus formation. Outcomes of acute inflammation.  
29. Exudative inflammation: serous, fibrinous, purulent, catarrhal, hemorrhagic mixed. Morphological characteristics, clinical significance.  
30. Chronic inflammation. Causes, pathogenesis, morphological characteristics, and outcomes.

31. Granulomatous inflammation. Etiology, pathogenesis, clinical and morphological characteristics, diagnostic methods, and outcomes. Types of granulomas. Granulomatous disease.  
32. Regeneration: definition, nature and biological significance, the relationship with inflammation, outcomes. The components of the healing process.  
33. Granulation tissue, angiogenesis: the stage, morphological characteristics. The kinetics of wound healing. Morphogenesis of the scar, the restructuring of the extracellular matrix with the scarring. The role of humoral and cellular factors in the process of repair. Pathological aspects of inflammation and regeneration.  
34. The immune system: structure and function. Pathological conditions of the immune system. Hypersensitivity reactions Mechanisms of development, morphological characteristics, clinical significance. Morphogenesis, morphological characteristics, clinical significance.  
35. Autoimmunity and autoimmune disease. Determining mechanisms of development, the clinical significance (role in the development of rheumatic fever, systemic lupus erythematosus, rheumatoid arthritis). Infectious agents in autoimmunity.  
36. Immune Deficiency Syndromes. Immune deficiency: definition, etiology, classification. Primary immunodeficiencies: definition, classification, diagnostic methods. Clinical and morphological characteristics of primary immunodeficiencies. Causes of death. Secondary (acquired) immunodeficiencies: definition, etiology, classification.  
37. Amyloidosis: the structure, physico-chemical properties, methods of diagnosis of amyloidosis, the theory of the etiology and pathogenesis, classification principles. Macro- and microscopic characterization of organs in amyloidosis.  
38. The concept of adaptation and compensation. Stage compensatory process. Compensatory myocardial hypertrophy: definition, causes, mechanisms, morphological characteristics, and outcomes.  
39. Hypertrophy and hyperplasia: definition, causes, mechanisms, types, clinical and morphological characteristics.  
40. Atrophy: definition, causes, mechanisms, types, clinical and morphological characteristics. Brown atrophy of the liver, myocardium, skeletal muscles.

41. Metaplasia, and dysplasia: definition, types. Metaplasia of epithelial and mesenchymal tissue: morphological characteristics, clinical significance, role in carcinogenesis.  
42. Tumors. Definition, etiology and pathogenesis. The concept of proto-oncogenes and anti-oncogenes. Molecular bases of carcinogenesis. Precancerous processes. Principles of classification of tumors. Meaning biopsy in oncology.  
43. Benign and malignant tumors: variety, comparative characteristics. The main properties of the tumor. Types of tumor growth.  
44. Epithelial tumors: benign and malignant. Cancer, its types.  
45. Mesenchymal tumors: benign and malignant. Sarcoma, its kinds. Special types of mesenchymal tumors.  
46. ​​The most important clinical and pathological manifestations of tumor growth. Characteristics of the tumor process. Local tumor effect. Violation of homeostasis. Secondary changes in the tumor. Metastases and systemic non-metastatic effects. Cancer cachexia, paraneoplastic syndromes.  
47. Biology of tumor growth. The morphogenesis of tumors. The progression and heterogeneity of tumors. Features in the tumor cell population focus. Mechanisms of invasive growth. Metastasis: forms, patterns, mechanisms. The metastatic cascade.  
48. Signs of death and postmortem changes. Death. Definition. The sudden death. The notion of fetal, clinical, biological death. Signs of biological death.  
49. Atherosclerosis and arteriosclerosis. Modern views on the etiology and pathogenesis of atherosclerosis. Morphological characteristics and stages of atherosclerosis, the structure of an atherosclerotic plaque. Organ damage in atherosclerosis. Arteriosclerosis (Menkeberg’s disease), morphological characteristics.  
50. Hypertension and arteriolosclerosis. Essential hypertension (hypertension) and secondary (symptomatic) hypertension. Benign and malignant hypertension within. Hypertensive heart disease: risk factors, causes of development, pathogenesis, morphological changes in the blood vessels and heart. Hyaline and hyperplastic arteriolosclerosis (morphological characteristics, changes in organs).

51. Ischemic heart disease (coronary heart disease). The concept, epidemiology, communication with atherosclerosis and hypertension. The etiology and pathogenesis, risk factors. The forms of CHD. Angina pectoris: classification, clinical and morphological characteristics.  
52. Myocardial infarction: causes, classification, the dynamics of biochemical and morphological and functional changes in the myocardium. Morphology of acute, recurrent, recurrent myocardial infarction. Sudden coronary (ischemic) death.  
53. Chronic ischemic heart disease: classification, clinical and morphological characteristics, complications, causes of death.  
54. Rheumatism: etiology, classification, pathological and morphogenesis, morphological characteristics and methods of diagnosis, clinical symptoms and syndromes forecast. Endocarditis, myocarditis, pericarditis, and pancarditis: classification, clinical and morphological characteristics, complications.  
55. Cerebrovascular disease. Etiology, relationship with atherosclerosis and hypertension. Clinical and morphological forms. Pathological anatomy of hemorrhagic and ischemic stroke, diffuse atrophic lesions.  
56. Infective endocarditis: classification, etiology, pathogenesis, morphological characteristics, complications, prognosis. Prosthetic heart valves: the complications arising from the presence of artificial heart valves.  
57. Cardiomyopathy: Classification, morphological characteristics of the value of genetic factors, pathological and morphogenesis, clinical and morphological characteristics, causes of death.  
58. Congenital heart defects. Etiology. Vices "blue" and "white" types. Congenital defects of the atrial and ventricular walls, arterial trunks of the heart (transposition, stenosis and anomalies in the mouths of the great arteries, aortic coarctation, patent ductus arteriosus), combined heart defects (the Fallot’s triad, tetrad, pentad). Clinical and morphological characteristics. Heart transplantation. Complications.  
59. Anemia. Definition and classification. Acute and chronic blood loss due anemia (posthemorrhagic): causes, clinical and morphological characteristics, diagnosis. Anemia due to increased destruction of blood (haemolytic). Classification, pathogenesis, diagnosis, clinical and morphological characteristics, causes of death. Hypersplenism.  
60. Anemia in low red blood cell reproduction (diseritropoetic). The classification of the causes . etiology, patho- and morphogenesis, clinical and morphological characteristics and methods of diagnosis, complications, causes of death.

61. Tumors of the hematopoietic tissue (leukemia). Classification, general clinical and morphological characteristics. Acute leukemia’s: modern methods of diagnosis, current stage, clinical and morphological characteristics, complications, age characteristics, causes of death.  
62. Chronic leukemia’s: classification, methods of diagnosis, current stage, clinical and morphological characteristics, causes of death. The etiology of leukemia, chromosome and antigenic restructuring. Modern methods of treatment: a bone marrow transplant.  
63. Tumors of plasma cells. General characteristics, diagnostic methods. Classification. Modern methods of diagnosis, etiology, pathogenesis, morphological characteristics, clinical symptoms, prognosis, cause of death.  
64. Hodgkin's disease (Hodgkin's disease): clinical stage, histopathological types, morphological characteristics and methods of diagnosis, clinical manifestations, prognosis, cause of death.  
66. Non-Hodgkin's lymphoma. General characteristics, localization, prognosis, classification and typing. Immunohistochemical markers, cell types in non-Hodgkin's lymphomas. Tumors of the T- and B-lymphocytes: types, morphological characteristics, cytogenetic and molecular genetic markers, clinical manifestations, prognosis, cause of death.  
68. Chronic obstructive bronchitis. Definition, classification, etiology, epidemiology, patho- and morphogenesis, morphological characteristics, clinical manifestations, complications, outcomes.  
69. Chronic obstructive pulmonary emphysema - definition, classification, epidemiology, etiology, patho- and morphogenesis, morphological characteristics, clinical manifestations, complications, consequences, causes of death. Other types of emphysema (clinical and morphological characteristics).  
70. Bronchial asthma. Definition, classification. Atopic asthma. Aggravating factors and pathological morphogenesis, clinical and morphological characteristics, consequences, causes of death. Other forms of bronchial asthma. Patho- and morphogenesis, clinical and morphological characteristics.

71. Bronchiectasis and bronchiectasis. The concept, classification, etiology, patho- and morphogenesis, morphological characteristics, clinical manifestations, complications, consequences, causes of death.  
72. Acute inflammatory lung disease. Bacterial pneumonia. Focal pneumonia (bronchopneumonia). Classification. Morphology and pathogenesis. Pneumonia under immunosuppression. Etiology, morphological characteristics, complications of focal pneumonia.  
73. Lobar pneumonia. Etiology, pathogenesis, clinical and morphological characteristics, stage of development, complications, outcomes.  
74. Interstitial pneumonia: etiology, pathogenesis, morphological characteristics, and outcomes.  
79. Diseases of the esophagus. Varicose veins of the esophagus. Esophagitis. Barrett's esophagus. Etiology, patho- and morphogenesis, clinical and morphological characteristics, complications, outcomes.  
81. Gastritis. Definition. Acute gastritis. Etiology, pathogenesis, morphological forms. Clinical and morphological characteristics.  
82. Chronic gastritis, the essence of the process. Etiology, pathogenesis. Principles of classification. Morphology. Complications, outcomes, prognosis.  
83. Peptic ulcer disease. Definition. General characteristics of peptic (chronic) ulcers of different locations. Epidemiology, etiology, pathogenesis, and morphogenesis, especially when it piloroduodenal and med0iogastral ulcers. Morphological characteristics of chronic ulcers in the period of exacerbation and remission. Complications, outcomes.  
86. Idiopathic inflammatory bowel disease. Nonspecific ulcerative colitis. Crohn's Disease. Epidemiology, etiology, patho- and morphogenesis, morphological characteristics, clinical manifestations, complications, outcomes, prognosis.   
87. Appendicitis. Classification, epidemiology, etiology, pathogenesis. Morphological characteristics and clinical manifestations of acute and chronic appendicitis. Complications. Features of the disease in children and the elderly.  
90. Hepatitis: definition, classification. Acute viral hepatitis. Epidemiology, etiology, ways of transmission, patho- and morphogenesis, clinical and morphological forms, morphological characteristics, viral markers outcomes.

91. Chronic hepatitis. Etiology, morphological characteristics and classification, signs of activity, outcome, prognosis. Viral hepatitis and liver cirrhosis.  
92. Alcoholic liver disease. Alcoholic fatty liver. Alcoholic hepatitis. Alcoholic cirrhosis. Epidemiology, pathogenesis, and morphogenesis, morphological characteristics, clinical manifestations, complications and causes of death, consequences, prognosis.  
93. Cirrhosis of the liver. Pathological characteristics and morphological classification of cirrhosis. The etiological classification of cirrhosis. Clinical and morphological characteristics of the major types of cirrhosis. Alcoholic cirrhosis. Cirrhosis after hepatitis. Biliary cirrhosis (primary, secondary). Changes to the liver in hemochromatosis, Wilson -Konovalova's disease, failure of alpha-1-antitrypsin. Pathogenesis, clinical and morphological characteristics.  
94. Cholelithiasis. Etiology, pathogenesis, types of stones. Cholecystitis. Definition. Acute and chronic cholecystitis. Etiology, pathogenesis, clinical and morphological characteristics, complications, causes of death.  
100. Glomerular kidney disease. Modern classification. Acute glomerulonephritis. Etiology, pathogenesis, morphological characteristics, and outcomes.

101. Nephrotic syndrome. Classification, pathogenesis, symptoms, clinical and morphological variants. Membranous nephropathy. Lipoid nephrosis. Focal segmental glomerulosclerosis. The pathogenesis, morphological characteristics.  
102. Chronic glomerulonephritis. Determination of macro- and microscopic characteristics. The concept of primary and secondary wrinkled kidney. Uremia. Etiology, pathogenesis, morphological characteristics.  
103. Renal amyloidosis. Morphological characteristics. Methods of diagnosis, clinical manifestations.  
106. Pyelonephritis. Determining Classification. Acute pyelonephritis, etiology, predisposing factors of the disease, pathogenesis, morphological characteristics, clinical manifestations, complications, outcomes.  
107. Chronic pyelonephritis and reflux nephropathy. Etiology, predisposing factors and disease pathogenesis, morphological characteristics, clinical manifestations, complications, outcomes.  
109. Nephrolithiasis. Patho- and morphogenesis, morphological characteristics, clinical manifestations. Types of stones, stone formation mechanisms, complications.  
112. Diseases of the prostate: classification. Inflammatory disease. Prostatitis: acute bacterial, chronic. Etiology, morphogenesis, morphological characteristics, complications, outcomes.

114. Diseases of the cervix. Acute and chronic cervicitis. Cervical ectopia. Endocervical polyps. Epidemiology, etiology, risk factors, morphogenesis, morphological characteristics, clinical manifestations, consequences.  
115. Diseases of the body of the uterus and endometrium. Classification Risk factors. Endometritis acute and chronic. Adenomyosis. Endometriosis. Morphological characteristics, theories, clinical manifestations, clinical significance.  
116. The glandular endometrial hyperplasia. The classification, causes, morphological characteristics, prognosis.  
124. gestosis. Classification, Epidemiology. Clinical manifestations, causes, pathogenesis, morphological characteristics. Causes of death of the woman, the effect on the fetus.  
125. Pathology of the placenta: classification. Infection in the placenta. Ways of infection of the placenta and fetus. Etiology, morphological manifestations, effects on the fetus and the woman's body, outcomes. Anomalies placental disk, localization and attachment of the placenta. Classification, morphological characteristics, clinical significance. Circulatory disorders. Etiology, especially morphogenesis, clinical significance. Pathology of the umbilical cord

126. Trophoblastic disease. Classification. Hydatidiform mole, invasive hydatidiform mole, choriocarcinoma. Trophoblastic tumor of placental bed. Epidemiology, morphological characteristics, clinical manifestations, prognosis.

**Infectious pathology**

132. Infectious and parasitic diseases. The interaction of microorganisms and infectious agents. General characteristics of the infectious process. Possible local and general reactions in infections.  
133. Bacteremia and sepsis. Sepsis as a special form of infection. Differences from other infections. Etiology, pathogenesis, relationship between macro- and micro-organism. The concept of the septic focus, the entrance gate (classification, morphology). Classification sepsis. Clinical and anatomical form of sepsis. Outcomes.  
134. Acquired Immune Deficiency Syndrome (AIDS). Epidemiology, transmission path, etiology. Biology of human immunodeficiency virus. Patho- and morphogenesis. Clinical and morphological characteristics. AIDS-associated diseases: opportunistic infections, tumors. Complications. Causes of death.  
135. Infection primarily affecting the respiratory system. Viral infections. Influenza. Etiology, epidemiology, pathogenesis. Morphological characteristics. Complications.  
136. Tuberculosis. Epidemiology, etiology, patho- and morphogenesis. Classification (primary, hematogenous, secondary). Morphological characteristics, clinical manifestations, complications, consequences, causes of death.  
137. Hematogenous tuberculosis. Classification, clinical and morphological characteristics, complications, consequences, causes of death.  
138. Secondary tuberculosis, ways of development. Classification, clinical and morphological characteristics, complications, consequences, causes of death. Pathomorphosis tuberculosis.  
139. Shigellosis. Epidemiology, etiology, patho- and morphogenesis, morphological characteristics, clinical manifestations, complications, consequences, causes of death/

140. Campylobacter and Yersinia enteritis. Epidemiology, etiology, patho- and morphogenesis, morphological characteristics, clinical manifestations, complications, consequences, causes of death.  
141. Typhoid fever and salmonellosis. Epidemiology, etiology, patho- and morphogenesis, morphological characteristics, clinical manifestations, complications, consequences, causes of death.  
142. Cholera. Epidemiology, etiology, patho- and morphogenesis, morphological characteristics, clinical manifestations, complications, consequences, causes of death.  
143. Diseases caused by protozoa and helminthes: malaria, amebiasis, hydatid disease. Epidemiology, etiology, patho- and morphogenesis, clinical and morphological characteristics, complications, consequences, causes of death.  
144. Scarlet fever. Epidemiology, etiology, pathogenesis (virulence factors), morphogenesis, morphological characteristics, clinical manifestations, complications, consequences, causes of death.  
146. Measles. Etiology, epidemiology, patho- and morphogenesis, morphological characteristics, clinical manifestations, complications, consequences, causes of death.  
147. Meningococcal disease. Etiology, epidemiology, patho- and morphogenesis, morphological characteristics, clinical manifestations, complications, consequences, causes of death.  
148. Diphtheria. Etiology, epidemiology, patho- and morphogenesis, morphological characteristics, clinical manifestations, complications, consequences, causes of death.  
  
  
  
  
  
  
Head of the Department of Pathological Anatomy  
MD, Professor Polyakova VS

**List of examination preparations**  
1. Gastric adenocarcinoma  
2. Actinomycosis liver  
3. Alcoholic monolobulyarny (portal) cirrhosis (painting pikrofuksinom)  
4. Amyloidosis Kidney (red coloration of the Congo)  
5. Amyloidosis of the spleen (Congo red stain)  
6. Arteriolosklerotichesky nephrosclerosis  
7. Atherosclerosis of the aorta  
8. Bronchopneumonia  
9. Bronchiectasis and pulmonary fibrosis  
10. Brown myocardial atrophy  
11. Brown atrophy of the liver  
12. Brown induration of lungs (Perls reaction)  
13. Intradermal nevi pigmentosa  
14. Chorionic villi and decidua tissue in spontaneous abortion  
15. Hemorrhagic pulmonary infarction  
16. Myocardial hypertrophy  
17. Hypertrophy of the prostate  
18. The purulent meningitis  
19. Granulation tissue  
20. Diphtheritic colitis dysentery  
21. Diphtheritic sore throat  
22. The glandular endometrial hyperplasia  
23. Fatty infarction (color of Sudan III)  
24. Fatty liver (coloring Sudan III)  
25. intracapillary proliferative (acute) glomerular nephritis (needle biopsy)  
26. Myocardial infarction  
27. Ischemic heart attack kidney  
28. Cavernous hemangioma  
29. carnification with lobar pneumonia  
30. Bleeding in the brain  
31. Macrofocal cardiosclerosis  
32. Lobar pneumonia  
33. Leiomyoma  
34. Lymphogranulomatosis lymph node  
35. Metastatic abscesses in the lung  
36. Myocarditis in diphtheria  
37. Medullary swelling group of follicles of the small intestine in typhoid fever  
38. The tubular epithelium kidney necrosis (necrotizing nephrosis)  
39. Obesity infarction  
40. Skin papilloma  
41. Liver myeloid leukemia  
42. Liver chronic lymphocytic leukemia  
43. Squamous cell carcinoma with keratinization  
44. Polymorphocellular sarcoma  
45. Breast cancer  
46. ​​Rheumatic back and warty endocarditis  
47. Rheumatoid nodular productive (granulomatous) myocarditis  
48. Toxic liver degeneration  
49. Tubal pregnancy  
50. TB granulomas in the lungs (miliary tuberculosis)  
51. Breast Fibroadenoma  
52. Fibroma  
53. Flegmonous appendicitis  
54. Chronic obstructive pulmonary emphysema   
55. Chronic gastric ulcer  
56. Chronic hepatic congestion - Muscat liver

**Correspondence table of training outcomes by discipline and -evaluation materials used at midterm attestation.**

|  |  |  |  |
| --- | --- | --- | --- |
| No. | controlled expertise | descriptor | Control and assessment tool (question / practical task number) |
| 1 | CCE-1 the ability for abstract thinking, analysis, synthesis. | Can the main stages in the development of pathological anatomy, its significance for practical and theoretical medicine; the main patterns of development and life of the body based on the structural organization of cells, tissues and organs; structural and functional foundations of general pathological processes; functional systems of the human body, their regulation and self-regulation when exposed to the external environment in health and disease; methods of morphological research; Latin, Greek and eponymous terminology; classification, nomenclature of anatomical names; safety rules and work in the morphological laboratory with reagents, devices and animals; | Practical tasks №1-148 |
| Have use educational, scientific, popular science literature, the Internet for professional activities; describe the morphological changes of the studied micro- and macroscopic preparations in organs and tissues; analyze information obtained using the methods of light-optical and electron microscopy; solve situational tasks | Diagnostics of the macro-preparation, micro-preparation No. 1-56. |
| Practical activities experience note taking skills; medical and anatomical conceptual apparatus; the skills of describing general pathological morphological changes in the studied macro-preparations, micro-preparations and electron diffraction patterns; skills in assessing the nature of the tumor process and its clinical manifestations based on macro- and microscopic changes in organs and tissues; the simplest medical instruments. | Practical training diary analysis  Diagnostics of the macro-preparation, micro-preparation No. 1-56 |
| 2 | Common professional expertise-9 the ability to assess morphofunctional, physiological conditions and pathological processes in the human body for solving professional problems. | Can anatomical and physiological, age-sex and individual characteristics of the structure and development of a sick organism; concepts of etiology, pathogenesis, morphogenesis and pathomorphosis of diseases, nosology, principles of classification of diseases; structural and functional bases of diseases and pathological processes; the value of the knowledge gained on human pathological anatomy for the subsequent study of clinical disciplines and in the professional activity of a doctor; | Practical tasks №1-148 |
| Have explain the nature of deviations in the course of development, which can lead to the formation of variants of anomalies and defects; use anatomical instruments and equipment correctly; substantiate the nature of the pathological process and its clinical manifestations; solving situational tasks. | Diagnostics of the macro-preparation, micro-preparation No. 1-56. |
| Practical activities experience the skills of describing morphological changes in various diseases; skills in assessing the nature of the pathological process and its clinical manifestations based on macro- and microscopic changes in organs and tissues; the simplest medical instruments. | Practical training diary analysis Diagnostics of the macro-preparation, micro-preparation No. 1-56. |
| 3… | Professional expertise – 5 the readiness to collect and analyze the patient's complaints, data from his anamnesis, examination results, laboratory, instrumental, pathological and other studies in order to recognize a condition or establish the presence or absence of a disease. | Can age-sex and individual characteristics of the structure and development of the sick organism; concepts of etiology, pathogenesis, morphogenesis and pathomorphosis of diseases, nosology, principles of classification of diseases; structural and functional bases of diseases and pathological processes; the value of the knowledge gained on human pathological anatomy for the subsequent study of clinical disciplines and in the professional activity of a doctor; | Practical tasks №1-148 |
| Have use educational, scientific, popular science literature, the Internet for professional activities; work with magnifying equipment (microscopes, optical and simple loupes); make calculations based on the results of the experiment, carry out elementary statistical processing of experimental data; describe the morphological changes of the studied micro- and macroscopic preparations in organs and tissues; explain the nature of deviations in the course of development, which can lead to the formation of variants of anomalies and defects; use anatomical instruments and equipment correctly; substantiate the nature of the pathological process and its clinical manifestations; solving situational tasks. | Diagnostics of the macro-preparation, micro-preparation No. 1-56. |
| Practical activities experience medical and anatomical conceptual apparatus; skills in assessing the nature of the pathological process and its clinical manifestations based on macro- and microscopic changes in organs and tissues; the simplest medical instruments | Practical training diary analysis Diagnostics of the macro-preparation, micro-preparation No. 1-56. |

1. **Methodical recommendations for using point-rating system.**

Within the implementation of point-rating system for assessing the educational achievements of students on «PATHOLOGICAL ANATOMY, CLINICAL PATHOLOGICAL ANATOMY» in accordance with the provision "About the point-rating system for assessing educational achievements of students", the rules for the formation of the following ratings are defined:

* Student`s current actual rating;
* Student`s bonus actual rating.

**Rules for making the student`s current actual rating for practice**

The current actual rating for the student's practice is formed as a result of current control during practice by analyzing the implementation of must-have practical skills.

The bonus actual rating for the student's practice is the result of assessing the performance of optional skills during practice.

The current actual rating for practice and the bonus actual rating for the student's practice are expressed in points and are calculated in accordance with the approaches determined by the methodological recommendations for the use of the point-rating system, located in section 3 of the Federal educational standard practice (Appendix 2).

The approaches to the current actual rating in practice and the bonus actual rating for the student's practice are the same for all types of practices and are carried out on a scale from 1 to 70 and on a scale from 1 to 15, respectively. Thus, the values ​​of the current actual rating and the bonus actual rating do not need to be brought to standardized values, since they are standardized values.

The calculation of the current actual rating for practice and the bonus actual rating for the student's practice is carried out automatically upon completion of the student's work on the report in the Information System of the University and is available for the teacher during midterm attestation.

The current actual rating for practice is formed on the basis of the "Total coefficient of mastering must-have skills" (hereinafter referred to as the total coefficient), which is calculated according to formula 3.

actual value / target value = Cumulative coefficient (3),

where

the actual value is the total number of mandatory or practical actions performed by the student during the practice, provided for by the practice report;

the planned value is the total number of planned mandatory or practical actions during the practice, provided for by the practice report;

the total coefficient is the ratio of actually performed by the student and planned for the performed actions or practical actions within the framework of the practice program. Thr actual practice rating is equal to

* 70 points, if the total coefficient is more than 0.9 and less than or equal to 1;
* 65 points if the total coefficient is more than 0.8 and less than or equal to 0.9;
* 60 points, if the total coefficient is more than 0.7 and less than or equal to 0.8;
* 0 points if the total coefficient is less than or equal to 0.7.

The actual bonus rating in practice is formed on the basis of the bonus coefficient, which is calculated according to formula 4.

(total coefficient + number of elective skills) / planned value (4),

where

the number of optional skills is the number of optional or practical actions performed during the practice within the framework of the practice program;

The actual bonus practice rating is equal to

• 15 points if the received bonus coefficient is more than 2;

• 10 points, if the received bonus coefficient is greater than or equal to 1.5 and less than or equal to 1.9;

• 5 points if the received bonus coefficient is greater than or equal to 1.1 and less than or equal to 1.4;

• 0 points if the received bonus coefficient is less than 1.1.

If the practice is carried out modularly, then

• the current actual rating is calculated as the arithmetic mean of the current actual ratings for each practice module, which, in turn, are determined in accordance with clauses 9.6, 9.7 of this regulation;

• bonus actual rating is calculated as the arithmetic average of bonus actual ratings for each practice module, which, in turn, are determined in accordance with clauses 9.6, 9.7 of these regulations.

When calculating the disciplinary rating for practice, the value of the current actual rating is used as the current standardized rating, and the value of the bonus actual rating is used as the bonus standardized rating.

Rules for transfering the disciplinary ranking in practice into a five-point system.

|  |  |  |
| --- | --- | --- |
| **PRS disciplinary rating** | **Mark for practice** | |
| Grading test | Final test |
| 91– 100 points | 5 (excellent) | passed |
| 71 – 89 points | 4 (good) | passed |
| 65–70 points | 3 (satisfactory) | passed |
| 64 and less points | 2 (unsatisfactory) | not passed |