Appendix to the working program

Federal State Budgetary Educational Institution of Higher Education
"Privolzhsky Research Medical University"
Ministry of Health of the Russian Federation

BANK OF ASSESSMENT TOOLS FOR DISCIPLINE/PRACTICE

PATHOLOGICAL ANATOMY, CLINICAL PATHOLOGICAL ANATOMY

Training program (specialty): _**31.05.01 GENERAL MEDICINE**
code, name

Department: ____**PATHOLOGICAL ANATOMY_**

Mode of study _____**FULL-TIME_**
(full-time/mixed attendance mode/extramural)

Nizhniy Novgorod
2022_

1. Bank of assessment tools for the current monitoring of academic performance, mid-term assessment of students in the discipline / practice

This Bank of Assessment Tools (BAT) for the discipline "Pathological anatomy, clinical pathological anatomy" is an integral appendix to the working program of the discipline "Pathological anatomy, clinical pathological anatomy". All the details of the approval submitted in the WPD for this discipline apply to this BAT.

(Banks of assessment tools allow us to evaluate the achievement of the planned results stated in the educational program.

Assessment tools are a bank of control tasks, as well as a description of forms and procedures designed to determine the quality of mastering study material by students.)

2. List of assessment tools

The following assessment tools are used to determine the quality of mastering the academic material by students in the discipline/ practice:

No.	Assessment tool	Brief description of the assessment tool	Presentation of the assessment tool in the BAT
1	Test tasks	Test tasks with a choice of one and several correct answers	Bank of test tasks
2	Situational cases	Situational cases, questions have a specified number of answers	List of situational cases

Approximate list of assessment tools (select the one you need)

№	Name of assessment tool	Brief description of the assessment tool	Presentation of assessment tool in the bank
1	Test №1	A system of standardized tasks that allows you to automate the procedure of measuring the level of knowledge and skills of a student	Bank of test tasks
	Test №2		
2	Control work	A tool of checking the ability to apply acquired knowledge for solving problems of a certain type by topic or section	Set of control tasks in variants
3	Individual survey	A control tool that allows you to assess the degree of comprehension of the material	List of questions
4	Interview	A tool of control organized as a special conversation between the teacher and the student on topics related to the discipline being studied, and designed to clarify the amount of knowledge of the student on a specific section, topic, problem, etc.	Questions on topics/sections of the discipline
5	Situational tasks	A method of control that allows you to assess the criticality of thinking and the degree of the material comprehension, the ability to apply theoretical knowledge in practice.	List of tasks

3. A list of competencies indicating the stages of their formation in the process of mastering the educational program and the types of evaluation tools

Code and formulation of competence*	Stage of competence formation	Controlled sections of the discipline	Assessment tools
UC-1 *the ability to carry out a critical analysis of problem situations based on a systematic approach, to develop a strategy of action*	Current	**Section 1** Damage to cells and tissues. **Section 2** Morphology of protein and lipid metabolism disorders. **Section 3** Morphology of pigment metabolism disorders **Section 4** Circulatory disorders **Section 5** Acute inflammation. **Section 6** Chronic inflammation. **Section 7** Adaptation and adaptive processes. **Section 8** Tumors. General provisions. Tumors of mesenchymal derived tissues. Tumors from melanin-forming tissue. **Section 9** Tumors from the epithelium. **Section 10** Cancer of individual localizations. **Section 11** Tumors of hematopoietic and lymphoid tissue. **Section 12** Diseases of the cardiovascular system **Section 13** Lung diseases. **Section 14** Diseases of the gastrointestinal tract **Section 15** Diseases of the liver and biliary tract. **Section 16** Kidney diseases. **Section 17** Intestinal infections. Sepsis. **Section 18** Tuberculosis. **Section 19** Bacterial and viral infections transmitted by airborne droplets (diphtheria, scarlet fever, measles, meningococcal infection). HIV infection. **Section 20** Organization of pathanatomic service in medical institutions (its prospects, further improvement). The importance and role of the pathology service in the health care system (tasks). **Section 21** Tasks, documentation of PAO medical institutions. The significance and tasks of pathoanatomic autopsy. Execution of the autopsy report.	*Specify the assessment tool(s)* *E.g:* *Interview 1* *(number – if there are several assessment tools of such forms)* *Credit*

		Section 22 Biopsy work.	
GPC-5	Able to assess morphofunctional, physiological conditions and pathological processes in the human body to solve professional problems	**Section 1** Damage to cells and tissues. **Section 2** Morphology of protein and lipid metabolism disorders. **Section 3** Morphology of pigment metabolism disorders **Section 4** Circulatory disorders **Section 5** Acute inflammation. **Section 6** Chronic inflammation. **Section 7** Adaptation and adaptive processes. **Section 8** Tumors. General provisions. Tumors of mesenchymal derived tissues. Tumors from melanin-forming tissue. **Section 9** Tumors from the epithelium. **Section 10** Cancer of individual localizations. **Section 11** Tumors of hematopoietic and lymphoid tissue. **Section 12** Diseases of the cardiovascular system **Section 13** Lung diseases. **Section 14** Diseases of the gastrointestinal tract **Section 15** Diseases of the liver and biliary tract. **Section 16** Kidney diseases. **Section 17** Intestinal infections. Sepsis. **Section 18** Tuberculosis. **Section 19** Bacterial and viral infections transmitted by airborne droplets (diphtheria, scarlet fever, measles, meningococcal infection). HIV infection. **Section 20** Organization of pathanatomic service in medical institutions (its prospects, further improvement). The importance and role of the pathology service in the health	…

		care system (tasks). **Section 21** Tasks, documentation of PAO medical institutions. The significance and tasks of pathoanatomic autopsy. Execution of the autopsy report. **Section 22** Biopsy work.	

* - *not provided for postgraduate programs*

4. The content of the assessment tools of entry, current control

Entry /current control is carried out by the discipline teacher when conducting classes in the form of: assessment tool 1, assessment tool 2, etc. *(list the forms, for example, control work, organization of a discussion, round table, abstract, etc.)*

Assessment tools for current control.

(the teacher specifies all types of tasks for conducting current control, if this is provided for in the WPD, in the form given below as an example. The current control is carried out in the context of the assessment of competencies provided for in the WPD, and not topics or sections of the discipline)

4.1. Tasks for the assessment of competence "UC-1, GPC-5" *(specify the competence code):*

1. Necrosis it is death:
    a) of cells due to metabolic disorders;
    b) of parenchymatous cells only;
    c) of cells and tissues in a living organism;
    d) programmed, genetically determined death of cells;
    e) of cells and tissue in dead organism.

2. Causes of necrosis are following:
    a) infectious agents;
    b) allergic factors;
    c) chemical substances;
    d) blood circulation disturbances;
    e) all the enumerated.

3. Call morphological type of necrosis:
    a) vascular;
    b) allergic;
    c) coagulative;
    d) traumatic;
    e) all the enumerated.

4. Dry necrosis has following colour:
    a) whitish-yellowish;
    b) black;
    c) dark-red;
    d) cyanotic;
    e) rusty.

5. Show wrong characteristic of wet necrosis:
    a) it has black colour;
    b) it contains a lot of fluid;
    c) it disturbs function of organ;
    d) it develops in the brain only;
    e) cyst formation – it is its often local result.

6. What colour does gangrene have?
    a) yellow;
    b) whitish-grayish;
    c) black;
    d) dark-red;
    e) cyanotic.

7. Show wrong characteristic of gangrene:
    a) it has black colour;
    b) it has contact with the environmental surrounding;
    c) it develops in bowel often;
    d) it disturbs function of organ;
    e) cyst formation – it is its local result.

8. Show wrong characteristic of dry necrosis:
    a) it has whitish-yellowish colour;
    b) it can develop in the spleen, kidney;
    c) its often outcome is organization;
    d) it can be vascular;
    e) it is direct always.

9. What morphological type of necrosis in the myocardium does develop?
    a) wet gangrene;
    b) dry gangrene;
    c) wet necrosis;
    d) dry necrosis;
    e) bedsore.

10. What is the most often localization of colliquative necrosis?
    a) spleen;
    b) kidney;
    c) liver;
    d) brain;
    e) myocardium.

11. Wet gangrene usually develops in:
    a) bowel;
    b) kidney;
    c) liver;
    d) brain;
    e) myocardium.

12. The cause of the indirect necrosis is:
    a) infectious agents;
    b) toxins;
    c) chemical substances;
    d) traumatic factors;
    e) stopping of blood flow.

13. Show the example of wet necrosis:
    a) caseous necrosis;
    b) fibrinoid necrosis;
    c) ischemic infarction of the spleen;

d) ischemic infarction of the brain;
e) waxy necrosis.

14. Bedsore it is the type of:
a) infarction;
b) gangrene;
c) dry necrosis;
d) wet necrosis;
e) ulceration.

15. In necrosis there is:
a) cytoplasm vacuolization;
b) nuclei vacuolization;
c) plasmolysis;
d) disappearance of glycogen;
e) all the enumerated.

16. In necrosis there is:
a) cytoplasm vacuolization;
b) nuclei vacuolization;
c) disappearance of glycogen;
d) kariolysis;
e) all the enumerated.

17. Caseous necrosis develops in:
a) rheumatic fever;
b) gas gangrene;
c) brain infarctions;
d) myocardial infarctions;
e) tuberculosis.

18. Show wrong characteristic of bedsore:
a) it is the type of gangrene;
b) develops in tissues due to prolonged compression;
c) has local metabolic disturbances in its development;
d) petrifaction it is its typical outcome;
e) develops in recumbent patients.

19. Fallowing change forms around the necrotic focus through few days:
a) calcium salts sedimentation;
b) osseous tissue;
c) demarcation inflammation;
d) fibrous capsule;
e) fibrous connective tissue.

20. Unpleasant outcome of necrosis is:
a) encapsulation;
b) organization;
c) petrifaction;
d) suppuration;
e) ossification.

21. Piece of dead tissue without any changes is calling:
a) petrifacate;
b) bedsore;

c) infarction;
d) sequester;
e) scar.

22. Complication of necrosis is:
a) resorption;
b) organization;
c) encapsulation;
d) rupture of cavitary organ wall;
e) petrifaction.

23. Black colour of dead tissues in gangrene is brought about:
a) melanin;
b) hemosiderin;
c) bilirubin;
d) hydrochloride acid hematin;
e) iron sulfide.

24. Gangrene is possible into:
a) kidney;
b) myocardium;
c) brain;
d) soft tissues of low extremities;
e) in all enumerated localizations.

25. Gangrene is possible into:
a) kidney;
b) myocardium;
c) lung;
d) liver;
e) brain.

26. Inflammatory reaction accompanies:
a) necrosis;
b) apoptosis;
c) proliferation;
d) cytoplasm vacuolization;
e) hyperemia.

27. Apoptosis it is:
a) death of cells in a living organism;
b) controlled process of cellular self-destruction;
c) death of tissues in a dead organism;
d) death of parenchymatous cells.

28. Usually apoptosis takes:
a) single cells;
b) foci of organ's parenchyma;
c) a part of organ;
d) whole organ.

29. At the light microscopy apoptosis bodies look like:
a) basophilic, round small bodies;
b) eosinophilic, round small bodies;

c) vacuoles;
d) crystals;
e) glomeruloid balls.

30. What does happen with the chromatin in apoptosis?
a) lyses;
b) dispersion;
c) condensation;
d) geterochromism.

31. The component of the apoptosis bodies is:
a) nuclei with nucleoli;
b) vacuoles filled with lipids inside;
c) giant mitochondria;
d) tightly packed cellular organelles;
e) dilated cisterns of endoplasmatic reticulum.

32. Apoptosis bodies are being exposed to:
a) autolysis;
b) phagocitosis;
c) organization;
d) encapsulation;
e) mucoidazation.

33. What cells can phagocitosis of apoptosis bodies?
a) macrophages;
b) lymphocytes;
c) monocytes;
d) plasmatic cells;
e) fibroblasts.

34. What is the outcome of apoptosis?
a) phagocitosis;
b) organization;
c) encapsulation;
d) tissue repair;
e) petrifaction.

35. Genetically programmed cellular death is called:
a) necrosis;
b) autolysis;
c) apoptosis;
d) mummify;
e) sequester.

36. Superficial defect of mucosa after necrotic masses disattachment is called:
a) ulcer;
b) erosion;
c) atrophy;
d) sequester;
e) apoptosis.

37. Deep defect of mucosa (or organ wall) after necrotic masses disattachment is called:
a) ulcer;

b) erosion;
c) atrophy;
d) sequester;
e) apoptosis.

38. Black colour necrosis due to iron sulfide accumulation is called:
a) infarction;
b) ulcer;
c) erosion;
d) sequester;
e) gangrene.

39. Fibrinoid necrosis develops often into:
a) nerve cells;
b) lung;
c) blood vessels' walls;
d) liver;
e) oral mucosa.

40. Caseous necrosis develops in:
a) rheumatic fever;
b) tuberculosis;
c) arterial hypertension;
d) Schigella dysentery;
e) diphtheria.

41. What is the most often morphological type of the brain necrosis?
a) gangrene;
b) wet necrosis;
c) dry necrosis;
d) cyst;
e) sequester.

42. Organization of necrosis it is a:
a) capsule formation;
b) calcium salts deposition;
c) osseous tissue formation;
d) cyst formation;
e) growth of connective tissue into necrotic focus.

43. Cyst it is a:
a) focal growing of connective tissue;
b) capsule formation on peripheral zone of pathological focus;
c) pathological cavity with walls and different containment;
d) calcium salts deposition;
e) focus of wet necrosis.

44. Petrifaction it is a:
a) formation of a bone;
b) growth of connective tissue;
c) calcium salts deposition;
d) capsule formation;
e) suppuration.

45. Deposition of calcium salts into necrotic focus it is a:
    a) organization;
    b) ossification;
    c) petrifaction;
    d) bedsore;
    e) infarction.

46. Choose the unpleasant outcome of necrosis:
    a) organization;
    b) suppuration;
    c) petrifaction;
    d) ossification;
    e) cyst formation.

47. The injury characterizes by the intra- and extracellular accumulations of abnormal quantities of a substances is calling:
    a) necrosis;
    b) apoptosis;
    c) degeneration;
    d) atrophy;
    e) hypertrophy.

48. The liver steatosis (fat degeneration) is characterized by the:
    a) sizes decreasing;
    b) dense consistency;
    c) nodular surface;
    d) accumulation of lipids in hepatocites' cytoplasm;
    e) disappearance of hepatocites' nuclei.

49. The cause of the liver fat degeneration (steatosis) is:
    a) increased blood circulation;
    b) hypoxia;
    c) hypertension;
    d) acute rheumatic fever;
    e) goiter (struma).

50. In protein starvation the fat degeneration develops into:
    a) liver;
    b) kidney;
    c) myocardium;
    d) adrenals;
    e) spleen.

51. The fat degeneration of the myocardium is characterized by the:
    a) appearance of connective tissue septas;
    b) enlargement of myocites;
    c) decreasing of myocites' sizes;
    d) accumulation of lipids in cytoplasm of several cardiomyocites only;
    e) accumulation of lipids in cytoplasm of all cardiomyocites.

52. At the microscopic investigation the myocardial fat degeneration can be define with help of:
    a) hematoxylin and eosin;
    b) sudan III;
    c) picrine acid;

d) Van Geison;
e) PAS-reaction.

53. The clinical manifestation of the myocardial fat degeneration is:
a) decreasing of systolic function;
b) increasing of systolic function;
c) hypertension;
d) rupture of the heart wall.

54. The liver steatosis is possible in:
a) alcoholism;
b) hypertension;
c) viral hepatitis A;
d) viral hepatitis B;
e) goiter (struma).

55. The fat degeneration of myocardium is possible in:
a) hypertension;
b) infectious diseases;
c) protein starvation;
d) acute rheumatic fever;
e) hemosiderosis.

56. The liver was called “gooses” in:
a) chronic venous congestion;
b) protein degeneration;
c) its capsule hyalinosis;
d) steatosis;
e) amyloidosis.

57. More typical outcome of the liver steatosis is:
a) structural restoration;
b) coming to protein degeneration;
c) coming to the massive progressive liver necrosis;
d) coming to liver cirrhosis.

58. Accumulation of lipids into arterial wall there is in:
a) inflammation;
b) aneurysm;
c) atherosclerosis;
d) cachexia;
e) obesity.

59. The heart was called “tiger” because of:
a) fat tissue grew into myocardium;
b) foci of necrosis there were into myocardium;
c) accumulation of lipids there was into some myocites;
d) accumulation of protein masses there was into some myocites;
e) uneven hyperemia there was into myocardium.

60. Severe hydropic degeneration is called:
a) ballooning degeneration;
b) hyaline-drop degeneration;
c) fat degeneration;

d) mucoid degeneration;
e) keratinization (horny).

61. Hydropic degeneration of hepatocites there is in:
a) liver steatosis;
b) viral hepatitis B;
c) liver ehynococcus;
d) diabetes mellitus;
e) obesity.

62. Hydropic degeneration of tubular epithelium in kidney there is in:
a) obesity;
b) nephrotic syndrome;
c) viral hepatitis B;
d) hypertension;
e) atherosclerosis.

63. Alcohol hyaline it is the product of following process:
a) destruction;
b) synthesis;
c) autolysis;
d) mucoidazation;
e) phagocitosis.

64. Accumulation of protein masses into tubular epithelium in kidney can be in:
a) hydropic degeneration;
b) mucoid degeneration;
c) steatosis;
d) hyaline-drop degeneration;
e) atrophy.

65. Reversible stage of the connective tissue disorganization it is:
a) sclerosis;
b) fibrinoid swelling;
c) mucoid swelling;
d) granulomatousis;
e) hyalinosis.

66. Hyalinosis of the heart valves' casps there is in:
a) hereditary defective valvular heart diseases;
b) rheumatic fever;
c) arterial hypertension;
d) diabetes mellitus;
e) alcoholism.

67. Widespread (systemic, general) hyalinosis of arterioles there is in:
a) atherosclerosis;
b) alcoholism;
c) arterial hypertension;
d) tuberculosis;
e) syphilis.

68. Development of hyalinosis is possible inside:
a) petrifacates;

b) amyloid masses;
c) connective tissue;
d) osseous tissue;
e) cartilages.

69. Amyloid it is the protein which can deposit:
a) into cells;
b) into cellular nuclei;
c) between cells;
d) into necrotic focus;
e) into focus of petrifaction.

70. At the histological investigation amyloid can be recognized with help of:
a) hematoxylin and eosin;
b) congo-red;
c) sudan III;
d) method of Van Geison;
e) toluoidine-blue.

71. Amyloid can be the complication of:
a) bronchiectases;
b) arterial hypertension;
c) atherosclerosis;
d) acute pneumonia;
e) acute Schigella dysentery.

72. Amyloid can be the complication of:
a) arterial hypertension;
b) atherosclerosis;
c) liver cirrhosis;
d) chronic lung abscess;
e) rabies.

73. Amyloid can be the complication of:
a) tuberculosis;
b) diabetes mellitus;
c) arterial hypertension;
d) atherosclerosis;
e) hepatitis.

74. What is the name of etiopathogenetic variant of amyloidosis which develops as complication of another disease?
a) primary;
b) secondary;
c) elderly;
d) hereditary;
e) family.

75. At visual inspection the kidney in amyloidosis is:
a) big motley;
b) big white;
c) primary shrunken;
d) small-nodular;
e) big-lobular.

76. What is typical there in appearance of organ in amyloidosis at visual inspection?
    a) flabby consistency;
    b) dense consistency;
    c) granular picture at incision;
    d) nodular surface;
    e) scars.

77. What is typical there in appearance of organ in amyloidosis at visual inspection?
    a) flabby consistency;
    b) granular picture at incision;
    c) sebaceous view at incision;
    d) small-nodular surface;
    e) big-nodular surface.

78. Where does amyloid deposit in the kidney?
    a) into glomeruli;
    b) into epithelium of proximal tubules;
    c) into epithelium of distal tubules;
    d) into fibrous capsule;
    e) into all enumerated objects.

79. The most often cause of death in secondary amyloidosis it is:
    a) chronic cardiac failure;
    b) acute cardiac insufficiency;
    c) chronic renal failure;
    d) acute renal insufficiency;
    e) acute adrenocortical insufficiency.

80. Obesity it is a predisposing factor of:
    a) brown atrophy of heart development;
    b) myocarditis development;
    c) ischemic heart disease development;
    d) acute pancreatitis development;
    e) goiter development.

81. In obesity in heart there is:
    a) appearance of lipids into myocites' cytoplasm;
    b) appearance of fat tissue septas in myocardium;
    c) appearance of connective tissue septas in myocardium;
    d) calcium salts deposition;
    e) foci of cardiomyocites necrosis.

82. What is it right for hyperplasic variant of obesity?
    a) has bad prognosis;
    b) number of adipocites (lipocites) is increased;
    c) adipocites contain a lot of triglycerides:
    d) it is associated with metabolic disorders;
    e) all enumerated is true.

83. What is it right for hypertrophic variant of obesity?
    a) has bad prognosis;
    b) number of adipocites (lipocites) is increased;
    c) function of adipocites is not disturbed;

d) there are not metabolic disorders;
e) all enumerated is true.

84. Group of endogen pigments includes:
a) lipids in hepatocites' cytoplasm;
b) proteins in tubular epithelium in kidney;
c) bilirubin in hepatocites' cytoplasm;
d) calcium salts in connective tissue;
e) all enumerated.

85. Call hemoglobin derivate:
a) melanin;
b) hemosiderin;
c) lipofuscin;
d) lipochromine;
e) adrenochromine.

86. What does Perl's test show?
a) hemosiderin;
b) bilirubin;
c) porphirin;
d) melanin;
e) lipofuscin.

87. What pigment does accumulate in the liver in cachexia?
a) hemosiderin;
b) bilirubin;
c) melanin;
d) lipofuscin;
e) ferritin.

88. Pigments they are substances:
a) changing colour;
b) can be receptive to stains;
c) of protein nature;
d) soluble in lipids.

89. The group of exogenic pigment includes:
a) melanin;
b) lipofuscin;
c) hemosiderin;
d) bilirubin;
e) iron sulfide.

90. What is it true about melanin?
a) it is exogenic pigment;
b) it is hemoglobin's derivate;
c) contains iron;
d) yellow;
e) is synthesizing in melanocites.

91. What is it true about hemosiderin?
a) it is exogenic pigment;
b) it is hemoglobin's derivate;

c) black;
d) there is not in health;
e) does not contain iron.

92. What is it true about bilirubin?
a) it is bile pigment;
b) is not defining in blood in health;
c) contains iron;
d) it is melanin derivate;
e) it is lipidogenic pigment.

93. What is it true about lipofuscin?
a) it is exogenic pigment;
b) it is hemoglobin's derivate;
c) contains iron;
d) accumulates in hepatocites cytoplasm;
e) makes function of hepatocite worse.

94. What pigment does accumulate in brown enduration of the lung?
a) hydrochloride acid hematin;
b) bilirubin;
c) hemosiderin;
d) lipofuscin;
e) coal dust.

95. What is the one of morphological features of brown enduration of the lung?
a) lungs are dark-red;
b) lungs are dense;
c) bronchi lumens are extended;
d) alveolar spaces are extended;
e) it is the example of general hemosiderosis.

96. What is the one of morphological features of brown enduration of the lung?
a) accumulations of hemosiderin;
b) thinning of interalveolar septas;
c) alveolar spaces are extended;
d) develops in acute venous congestion;
e) it is the example of general hemosiderosis.

97. The example of degenerative calcification is:
a) calcium salts into healthy gastric mucosa;
b) calcium salts metastases in kidneys;
c) petrifaction of necrosis;
d) calcium salts into healthy lungs;
e) calcium salts into myocardium in hypercalcaemia.

98. Choose the type of the pathological calcification:
a) metabolic;
b) focal;
c) diffuse;
d) metastatic;
e) idiopathic.

99. Choose the type of the pathological calcification:

a) metabolic;
b) focal;
c) diffuse;
d) degenerative;
e) idiopathic.

100. Metastatic calcification develops in:
a) anemia;
b) hypoxia;
c) hyperlipidemia;
d) hypercalcaemia;
e) hypocalcaemia.

101. What is wrong there in the characteristic of degenerative calcification?
a) it is local process;
b) there is not hypercalcaemia;
c) calcium salts deposit in organs with pathological changes;
d) there is not functional disorder;
e) can be in surplus filling of vitamin D.

102. What is not right about metastatic calcification?
a) there is hypercalcaemia in organism;
b) several organs are damaged;
c) function of organs is not disturbed;
d) calcium salts deposit in organs with pathological changes;
e) can be in surplus filling of vitamin D.

103. Call example of degenerative calcification:
a) calcium salts deposition in gastric mucosa in hypercalcaemia;
b) calcium salts deposition in heart valvulars in rheumatic fever;
c) calcium salts deposition in myocardium in hypercalcaemia;
d) calcium salts deposition in healthy kidneys;
e) calcium salts deposition in healthy lungs.

104. Where calcium salts do deposit in metastatic calcification?
a) in connective tissue scars;
b) in connective tissue adhesions;
c) in thrombus;
d) in heart valvulars sclerosis;
e) in kidneys, lungs in hypercalcaemia.

105. As result of caseous necrosis petrifacates occur in:
a) rheumatic fever;
b) tuberculosis;
c) atherosclerosis;
d) arterial hypertension;
e) Schigella dysentery.

106. Gout it is disturbed metabolism of:
a) lipids;
b) nucleoproteins;
c) aminoacids;
d) pigments;
e) calcium.

107. Uric acid infarction it is the result of what metabolism disturbances?
a) calcium;
b) potassium;
c) lipidogenic pigments;
d) hemoglobinogenic pigments;
e) nucleoproteins.

108. What pigment can appear in zone of hemorrhage?
a) adrenochromine;
b) melanin;
c) lipofuscin;
d) hemosiderin;
e) lipochromine.

109. What process is the result of melanin metabolism disturbances?
a) vitiligo;
b) leukoplakia;
c) hemochromatosis;
d) jaundice;
e) Gilbert syndrome.

110. Keratinization there is in:
a) vitiligo;
b) leukoplakia;
c) widespread melanosis;
d) skin melanoma;
e) in all enumerated.

111. Choose the name of an arterial hyperemia type:
a) obstructive;
b) postanemic;
c) ischemic;
d) hydrostatic;
e) mechanical.

112. Vacate arterial hyperemia develops in:
a) capping glasses applying;
b) an artery forceps removing off;
c) obstruction of magistral artery lumen by thrombus;
d) paralysis of vascular constrictor nerve;
e) in all enumerated.

113. What is for the venous congestion development necessary?
a) increasing of blood flow;
b) decreasing of blood flow;
c) increasing of blood outflow;
d) decreasing of blood outflow.

114. Local venous congestion develops in:
a) obstruction of an artery lumen by thrombus;
b) obstruction of a vein lumen by thrombus;
c) compression of an artery by tourniquet;
d) myocardial infarction;

e) decompensation of heart in its hypertrophy.

115. General venous congestion develops in:
a) decompensation of heart in its hypertrophy;
b) a vein compression;
c) obstruction of a vein lumen by thrombus;
d) narrowing of vein lumen by growing tumour;
e) varicous dilation of veins.

116. Acute general venous congestion develops in:
a) myocardial infarction;
b) cardiosclerosis;
c) chronic heart aneurysm;
d) defective valvulars heart diseases;
e) pneumosclerosis.

117. Chronic general venous congestion develops in:
a) myocardial infarction;
b) acute myocarditis;
c) severe myocardial degeneration;
d) cardiosclerosis;
e) acute heart aneurysm.

118. What does develop in tissues in acute venous congestion?
a) sclerosis;
b) atrophy;
c) petrifaction;
d) edema;
e) hyalinosis.

119. What does develop in organs and tissues in acute venous congestion?
a) sclerosis;
b) atrophy of parenchymatous cells;
c) hypertrophy of parenchymatous cells;
d) diapedesis of erythrocytes;
e) all enumerated.

120. What does develop in organs and tissues in chronic venous congestion?
a) atrophy of parenchymatous cells;
b) calcium salts deposition;
c) amyloid accumulation;
d) inflammation;
e) all enumerated.

121. What does develop in lung in chronic venous congestion?
a) necrotic foci;
b) inflammation;
c) sclerosis;
d) amyloidosis;
e) all enumerated.

122. What does develop in lung in acute venous congestion?
a) hemosiderosis;
b) edema;

c) sclerosis;
d) hyalinosis;
e) all enumerated.

123. What is the figurative name of the liver in chronic venous congestion?
a) sebaceous;
b) sago;
c) brown;
d) nutmeg;
e) glazed.

124. The liver was called “nutmeg” in:
a) acute venous congestion;
b) chronic venous congestion;
c) anemia;
d) shock;
e) DIC-syndrome.

125. In nutmeg liver all enumerated develops except:
a) hyperemia of central veins;
b) hyperemia of portal vein brunches;
c) atrophy of hepatocites;
d) fat degeneration of hepatocites;
e) hemorrhages into centers of lobules.

126. The liver has nutmeg appearance due to:
a) hemorrhages into centers of lobules:
b) atrophy of hepatocites in centers of lobules;
c) hypertrophy of hepatocites of peripheral parts of lobules;
d) beginning of connective tissue growth;
e) structural reorganization of lobules.

127. The result (outcome) of the nutmeg liver is:
a) hepatitis;
b) liver cirrhosis;
c) steatosis;
d) massive necrosis;
e) obstructive jaundice.

128. What does develop in general chronic venous congestion?
a) nutmeg liver;
b) hydrocephalus;
c) big white kidney;
d) big sebaceous kidney;
e) all enumerated.

129. What does develop in general chronic venous congestion?
a) nutmeg liver;
b) brown enduration of lung;
c) cyanotic enduration of kidney;
d) ascitis;
e) all enumerated.

130. What does develop in the liver in chronic venous congestion?

a) amyloid deposition;
b) calcium salts deposition;
c) atrophy of hepatocites;
d) cholestasis;
e) all enumerated.

131. Accumulation of hemosiderin in lung is observed in:
a) acute venous congestion;
b) chronic venous congestion;
c) acute pneumonia;
d) emphysema;
e) shock.

132. What can develop in myocardial infarction of left heart ventricle?
a) acute venous congestion in large circulation;
b) acute venous congestion in lesser circulation;
c) chronic venous congestion in large circulation;
d) chronic venous congestion in lesser circulation.

133. Acute venous congestion in lesser circulation can develop in:
a) decompensation of heart hypertrophy;
b) valvular heart diseases;
c) cardiosclerosis;
d) myocardial infarction;
e) in all enumerated.

134. What does develop in lung in decompensation of mitral stenosis?
a) pneumonia;
b) brown enduration;
c) hematoma;
d) amyloid deposition;
e) calcium salts deposition;

135. What does develop in lung in left ventricle myocardial infarction?
a) brown enduration;
b) pneumosclerosis;
c) edema;
d) hemosiderosis;
e) inflammation.

136. What does develop in lung in decompensation of mitral stenosis?
a) tumour;
b) necrosis;
c) atrophy;
d) inflammation;
e) sclerosis.

137. In nutmeg liver there is:
a) decreasing of the organ sizes;
b) particoloured view at incision;
c) flabby consistency;
d) nodular surface;
e) all enumerated.

138. In nutmeg liver there is:
a) enlargement of the organ;
b) dense consistency
c) particoloured view at incision;
d) rounded low margin;
e) all enumerated.

139. In nutmeg liver there is:
a) ischemia of central parts in lobules;
b) hyperemia of central parts in lobules;
c) hemosiderosis;
d) hypertrophy of hepatocites of central parts in lobules;
e) all enumerated.

140. What cannot develop in organs and tissues in acute venous congestion?
a) edema;
b) plasmorrhagia;
c) sclerosis;
d) erythrocytes diapedesis;
e) parenchymatous cells degeneration.

141. What is observed in central parts of lobules in nutmeg liver?
a) hemorrhage;
b) hyperemia;
c) hepatocites atrophy;
d) the connective tissue growth beginning;
e) all enumerated.

142. Hemorrhage it is:
a) blood accumulation in cavities;
b) blood accumulation in tissues;
c) blood running out from blood vessel;
d) running out of blood in environmental surrounding;
e) rupture of blood vessel wall.

143. Accumulation of blood inside anatomical cavity is called:
a) hydrothorax;
b) hydroperitoneum;
c) hematoma;
d) hemopericardium;
e) hemorrhage.

144. Call a possible mechanism of bleeding:
a) stasis;
b) plasmorrhagia;
c) hemorrhage;
d) diapedesis;
e) angiospasm.

145. Call morphological variant of hemorrhage:
a) hematoma;
b) hemorrhagia;
c) ascitis;
d) edema;

e) all enumerated.

146. Rapid massive bleeding can result as:
a) venous congestion;
b) edemas;
c) stasis;
d) acute ischemia;
e) chronic ischemia.

147. What pigment in zone of a hemorrhage can appear?
a) melanin;
b) lipofuscin;
c) hemosiderin;
d) lipochromine;
e) hemoglobin.

148. "Rusty" cyst in brain develops on the place of:
a) necrosis;
b) hematoma;
c) ischemic infarction;
d) tumour;
e) ehynococcus.

149. What does form on hematoma place in the brain usually?
a) cyst;
b) scar;
c) tumour;
d) calcium salts deposition;
e) capsule.

150. Unfavorable outcome of hemorrhage is:
a) cyst;
b) suppuration;
c) scar;
d) petrifaction;
e) resolution.

151. What is hematoma?
a) accumulation of blood inside serous cavities;
b) accumulation of blood in tissues without their destruction;
c) accumulation of blood in tissues with their destruction;
d) bruise;
e) petechia.

152. When does bleeding develop due to blood vessel wall erosion?
a) in purulent inflammation;
b) in chronic venous congestion;
c) in acute venous congestion;
d) in hypertensive crisis;
e) in traumas.

153. When does bleeding develop due to blood vessel rupture?
a) in purulent inflammation;
b) in chronic venous congestion;

c) in acute venous congestion;
d) in hypertensive crisis;
e) in tumours.

154. When does bleeding develop due to diapedesis?
a) in traumas;
b) in chronic venous congestion;
c) in tumours;
d) in tubal pregnancy;
e) in necrosis.

155. Hemorrhage which is associated with tissue necrosis is called:
a) hemorrhagia;
b) hematoma;
c) hemorrhagic saturation;
d) petechia;
e) bruise.

156. What can be outcome of hemorrhage?
a) hematoma;
b) organization;
c) necrosis;
d) petechia;
e) functional disturbances.

157. Brain hematoma in arterial hypertension develops as result of:
a) blood vessel rupture;
b) blood vessel wall erosion;
c) increased blood vessel wall permeability.

158. Multiple petechias in skin in infectious diseases develop due to:
a) blood vessel rupture;
b) blood vessel wall erosion;
c) increased blood vessel wall permeability.

159. Choose the definition of the stasis:
a) decreased arterial blood flow;
b) blood viscous increasing;
c) difficulties of blood outflow;
d) stopping of blood flow in microcirculatory bed;
e) stopping of arterial blood flow.

160. The most severe result of long stasis is:
a) sludge phenomenon;
b) perivascular edema;
c) plasmorrhagia;
d) erythrocytes diapedesis;
e) necrosis of parenchymatous cells.

161. What does develop in stasis?
a) sludge phenomenon;
b) erythrocytes diapedesis;
c) perivascular edema;
d) necrosis of tissue elements;

e) all enumerated.

162. What does sludge phenomenon mean?
a) adhesion of blood cells to each other;
b) erythrocytes agglutination;
c) increasing of blood cells number;
d) increasing of blood viscous;
e) stopping of blood flow in microcirculatory bed.

163. Edema it is:
a) increased blood filling of organ, tissue;
b) increased containment of interstitial fluid;
c) difficulties of venous blood outflow;
d) exudate accumulation;
e) plasmatic infiltration.

164. In nephrotic syndrome edemas are:
a) hydrostatic;
b) oncotic;
c) membranogenic;
d) electrolyte;
e) due to lymphostasis.

165. In acute glomerulonephritis edemas are:
a) hydrostatic;
b) oncotic;
c) membranogenic;
d) electrolyte;
e) due to lymphostasis.

166. What is leading there in edemas development in chronic cardiac failure?
a) increased hydrostatic pressure;
b) decreasing of colloid-osmotic pressure;
c) increased aldosterone secretion;
d) damage of endothelium and basement membranes of capillars;
e) increased permeability of capillars membranes.

167. What is observed in lung edema?
a) increasing of lungs sizes;
b) increasing of lungs weight;
c) flabby consistency of lungs;
d) flowing down of foamy fluid at the incision;
e) all enumerated.

168. What is observed in lung edema?
a) increasing of lungs sizes;
b) decreasing of lungs weight;
c) increased air filling of lungs;
d) dense consistency of lungs;
e) all enumerated.

169. What does develop in lung edema?
a) extension of alveolars spaces;
b) accumulation of edematous fluid in alveolars spaces;

c) sclerosis of interalveolar septas;
d) deposition of hemosiderin;
e) all enumerated.

170. What does develop in lung edema?
a) hyperemia of capillars;
b) accumulation of edematous fluid in alveolars spaces;
c) narrowing of alveolars spaces;
d) erythrocytes diapedesis;
e) all enumerated.

171. A transudate is characterized by the fallowing feature:
a) muddy;
b) bad smelling;
c) contains proteins less than 2%;
d) there is lot of cells;
e) all enumerated.

172. Call the certain variant of edematous fluid containment increasing:
a) hematoma;
b) ascitis;
c) petechia;
d) exicosis;
e) hemothorax.

173. Brain edema is characterized by the:
a) volume decreasing in association with convolutions flattening;
b) volume increasing in association with cerebellum wedge in major occipital hole;
c) extension of brain ventricles by transparent fluid;
d) extension of brain ventricles by muddy fluid;
e) picture of brain tissue is blurred at the incision.

174. Choose the cause of acute ischemia:
a) obturation of vein by thrombus;
b) obturation of artery by thrombus;
c) embolism;
d) compression of artery by tumour;
e) all enumerated.

175. Choose the cause of acute ischemia:
a) spasm of artery;
b) obturation of artery by thrombus;
c) obturation of artery by thromboembolus;
d) compression of artery by forceps;
e) all enumerated;

176. What is important result of acute ischemia possible?
a) sclerosis;
b) necrosis;
c) hemosiderosis;
d) atrophy;
e) degeneration.

177. What is important result of chronic ischemia possible?

a) degeneration and necrosis;
b) atrophy and sclerosis;
c) edema and plasmorrhagia;
d) hyperemia and diapedesis.

178. What is the reversible change of cell in ischemia?
a) kariopicnosis;
b) kariorrhexis;
c) plasmolysis;
d) rupture of membranes;
e) disappearance of glycogen.

179. What is the cause of thrombus formation?
a) damage of blood vessel wall;
b) slow blood flow;
c) turbulent blood flow;
d) increasing of blood viscous;
e) all enumerated.

180. What is the cause of thrombus formation?
a) damage of blood vessel wall;
b) number of erythrocytes decreasing;
c) number of thrombocytes decreasing;
d) diapedesis of erythrocytes;
e) plasmorrhagia.

181. Call stages of thrombus formation:
a) agglutination of thrombocytes;
b) fibrinogen coagulation;
c) agglutination of erythrocytes;
d) plasma proteins precipitation;
e) all enumerated.

182. What morphological type of thrombus is non-existent?
a) red;
b) white;
c) mixed;
d) white with red rim;
e) hyaline.

183. More often white thrombi form in:
a) veins;
b) arteries;
c) aneurysm cavity;
d) capillars.

184. More often red thrombi form in:
a) veins;
b) arteries;
c) capillars;
d) heart chambers;
e) aorta.

185. More often hyaline thrombi form in:

a) veins;
b) arteries;
c) capillars;
d) heart chambers;
e) aorta.

186. One of unfavorable thrombus formation outcomes is:
a) organization;
b) thromboembolism;
c) petrifaction;
d) vascularization;
e) recanalization.

187. Obstructive thrombus of artery can cause:
a) venous congestion;
b) arterial hyperemia;
c) infarction;
d) thromboembolism;
e) atrophy.

188. Obstructive thrombus of vein can cause:
a) venous congestion;
b) arterial hyperemia;
c) infarction;
d) petrifaction;
e) thromboembolism.

189. Favorable outcome of thrombus formation is:
a) septic autolysis;
b) suppuration;
c) organization;
d) thromboembolism;
e) obstruction of blood vessel lumen.

190. Thrombus which is consisting of alternating red thrombus particles with white thrombus particles is called:
a) red;
b) white;
c) mixed;
d) hyaline;
e) mural glomerular.

191. Thrombus which contains lot of erythrocytes is called:
a) red;
b) white;
c) mixed;
d) flaky;
e) hyaline.

192. Thrombus which contains lot of leukocytes and fibrin is called:
a) red;
b) white;
c) mixed;
d) flaky;

e) hyaline.

193. What does develop in low extremity in artery femoralis obturation by thrombus?
a) dry necrosis;
b) wet necrosis;
c) gangrene;
d) infarction;
e) hyperemia.

194. Call a type of embolism:
a) ischemic;
b) air;
c) angioneurotic;
d) vacate;
e) inflammatory.

195. Thromboembolism of small brunches of pulmonary artery can cause:
a) pulmonocoronary reflex;
b) lung infarction;
c) athelectasis;
d) shock;
e) DIC-syndrome.

196. Gross characteristics of a thrombus include:
a) rough surface;
b) smooth surface;
c) contains lot of fluid;
d) it is not attached to blood vessel wall;
e) all enumerated is right.

197. Gross characteristics of a thrombus include:
a) rough surface;
b) crimped surface;
c) dull surface;
d) it is attached to blood vessel wall;
e) all enumerated is right.

198. Thromboembolism of pulmonary trunk and its large brunches results as:
a) pulmonocoronary reflex;
b) lung infarction;
c) athelectasis;
d) shock;
e) DIC-syndrome.

199. Call the localization of thrombi in pulmonary thromboembolism:
a) valvulars of the left part of heart;
b) aorta;
c) arteries of large circulation;
d) veins of large circulation;
e) veins of lesser circulation.

200. Call the localization of thrombi in large circulation arteries thromboembolism:
a) valvulars of the left part of heart:
b) valvulars of the right part of heart;

c) veins of lesser circulation;
d) veins of large circulation;
e) arteries of lesser circulation.

201. Fat embolism of what organs capillars is most dangerous?
a) kidney;
b) liver;
c) lungs;
d) intestine;
e) spleen.

202. Call the outcome of large circulation arteries thromboembolism:
a) hyperemia of inner organs;
b) infarctions in organs;
c) edema;
d) exicosis;
e) cachexia.

203. Pulmonocoronary reflex develops in:
a) fat embolism of lung blood vessels;
b) amniotic fluid embolism;
c) microbe embolism of lung blood vessels;
d) pulmonary trunk thromboembolism;
e) thromboembolism of pulmonary artery small brunches.

204. Fat embolism is possible in:
a) ulceration and disattachment of atherosclerotic plaque particles;
b) massive traumas of subcutaneous fat tissue;
c) mistaken intramuscular injections of oil-based drugs;
d) amniotic fluid embolism;
e) all enumerated.

205. Infarction it is necrosis:
a) with different etiology;
b) with curtain localization;
c) with vascular genesis (due to blood circulation disturbances);
d) due to microcirculation disturbances;
e) in organ due to stopping of arterial blood flow.

206. What is not a morphological type of infarction?
a) white;
b) red;
c) mixed;
d) white with red rim.

207. Call the most often cause of infarction development:
a) venous congestion;
b) arterial thrombosis;
c) thrombosis of large veins;
d) microcirculatory bed embolism;
e) microcirculatory bed thrombosis.

208. The most main condition of hemorrhagic infarction development is:
a) massive blood loss;

b) arterial thrombosis;
c) venous congestion;
d) anemia;
e) anastamoses insufficiency.

209. Red infarction is usual for:
a) myocardium;
b) lung;
c) spleen;
d) kidney;
e) liver.

210. White infarction with red rim is usual for:
a) intestine;
b) skin;
c) brain;
d) myocardium;
e) liver.

211. White infarction is usual for:
a) spleen;
b) intestine;
c) lung;
d) liver;
e) skin.

212. What is wrong about lung infarction?
a) it has pyramidal form;
b) dark-red colour;
c) develops in venous congestion:
d) cyst – is its result;
e) its cause is thrombosis (or thromboembolism).

213. What is wrong about myocardial infarction?
a) it has pyramidal form;
b) it has whitish-yellowish colour;
c) it has red rim;
d) its consistency is dense;
e) thrombus there is near on endocardium always;

214. What is wrong about kidney infarction?
a) it has pyramidal form;
b) it has whitish-yellowish colour;
c) it has red rim;
d) it has soggy mass consistency;
e) its cause is thrombosis (or thromboembolism).

215. What is wrong about spleen infarction?
a) it has pyramidal form;
b) its colour is red;
c) its consistency is dense;
d) its cause is thrombosis (or thromboembolism);
e) its outcome is connective tissue scar.

216. What is wrong about brain infarction?
a) it has pyramidal form;
b) it has whitish-grayish colour;
c) its consistency is soft;
d) localizes into subcortical nuclei;
e) often develops in atherosclerosis.

217. Infarction of what organ has the most severe results?
a) spleen;
b) kidney;
c) brain;
d) lung;
e) bones.

218. Unfavorable outcome of infarction is:
a) organization;
b) petrifaction;
c) cyst formation;
d) suppuration;
e) encapsulation.

219. What does develop as myocardial infarction outcome usually?
a) cyst;
b) abscess;
c) scar;
d) hemosiderosis;
e) petrifaction.

220. What does develop as brain infarction outcome usually?
a) cyst;
b) abscess;
c) scar;
d) hemosiderosis;
e) petrifaction.

221. What does develop as kidney (spleen) infarction outcome usually?
a) cyst;
b) abscess;
c) hemosiderosis;
d) scar;
e) petrifaction.

222. Call the type of shock:
a) acute;
b) hypovolumic;
c) reversible;
d) irreversible;
e) all is true.

223. What does develop in kidney in shock?
a) acute tubular necrosis;
b) inflammation;
c) hemosiderosis;
d) petrifaction;

e) urates accumulation.

224. What is morphological change in kidney in shock observed?
a) tubular atrophy;
b) tubular necrosis;
c) stromal sclerosis;
d) inflammation;
e) all is true.

225. What does develop in lung in shock?
a) necrosis;
b) fat degeneration;
c) disappearance of glycogen;
d) edema;
e) inflammation.

226. What is morphological change in lung in shock observed?
a) degeneration;
b) necrotic foci;
c) edema;
d) inflammation;
e) all is true.

227. What does develop in lung in shock?
a) hyperemia;
b) hemorrhage;
c) edema;
d) athelectasis;
e) all enumerated.

228. What does develop in liver in shock?
a) ischemia;
b) necrotic foci;
c) hemosiderosis;
d) sclerosis;
e) inflammatory infiltration.

229. What does develop in myocardium in shock?
a) petrifaction;
b) hemosiderosis;
c) necrosis of cardiomyocites;
d) sclerosis;
e) inflammatory infiltration.

230. In what organ ulcers and erosions develop in shock more often?
a) stomach;
b) esophagus;
c) oral cavity;
d) rectum;
e) bronchi.

231. What is “shock organ”?
a) an organ, pathology of that causes death;
b) an organ, changes of that causes of shock development;

c) an organ with morphological changes due to shock.

232. What is main in DIC-syndrome development?
a) thrombocitopenia;
b) anemia;
c) insufficiency of fibrinogen synthesis;
d) increased intravascular blood coagulation;
e) decreased volume of circulating blood.

233. What is the starting moment in the DIC-syndrome development?
a) coagulation of fibrinogen with the formation of fibrin;
b) appearance lot of thromboplastin in blood;
c) hypofibrinogenemia;
d) increased formation of thrombin from plasma prothrombin;
e) formation of thrombi in microcirculatory bed.

234. What is shock accompanied with often?
a) nephrotic syndrome;
b) DIC-syndrome;
c) hepatico-renal syndrome;
d) hepatico-lienal syndrome;
e) chronic renal failure.

235. What is the phase of inflammation?
a) hyperemia;
b) degeneration;
c) exudation;
d) reparation;
e) regeneration.

236. Show unfavorable result of inflammation:
a) killing of microbes;
b) neutralization of toxins;
c) restitution;
d) massive sclerosis of organ;
e) phagocitosis of necrotized cells.

237. What does happen in exudation?
a) arterial and venous hyperemia;
b) increasing of blood vessel wall permeability;
c) migration of blood cells;
d) phagocitosis;
e) all enumerated.

238. The basic cells in a focus of acute inflammation are:
a) monocytes;
b) macrophages;
c) histiocytes;
d) neutrophilic leukocytes;
e) fibroblasts.

239. What is the morphological appearance of alteration in inflammation?
a) atrophy;
b) necrosis;

c) hyperplasia;
d) apoptosis;
e) all enumerated.

240. What is exudate?
a) edematous fluid;
b) inflammatory fluid;
c) pathological fluid with protein containment;
d) inflammatory fluid with erythrocytes containment;
e) every pathological fluid.

241. Call morphological type of inflammation:
a) specific;
b) proliferative;
c) immune;
d) acute;
e) chronic.

242. Call morphological form of exudative inflammation:
a) serous;
b) granulomatous;
c) interstitial;
d) mucoid;
e) chronic.

243. What is wrong about serous exudate?
a) protein containment is less than 2%;
b) at visual inspection it is transparent fluid;
c) at visual inspection it looks like transudate;
d) contains low number of leukocytes;
e) easy resolves.

244. The most often outcome of serous exudate is:
a) organization;
b) petrifaction;
c) resolution;
d) coming to purulent;
e) sclerosis.

245. What is wrong about fibrinous exudate?
a) contains lot of proteins;
b) whitish-grayish membranes form on mucosa surface;
c) resolve perfectly;
d) its most often localizations are mucosa membranes;
e) contains little of fluid.

246. What is wrong about fibrinous pericarditis?
a) develops in rheumatic fever;
b) its figurative name is “bread and butter heart”;
c) inflammation is diphtheroid;
d) connective tissue adhesions form as outcome;
e) its clinical manifestation is pericardial rub.

247. Choose the wrong name of exudative inflammation form:

a) serous;
b) purulent;
c) fibrinous;
d) fibrous;
e) anaerobic.

248. Call the form of fibrinous inflammation:
a) purulent;
b) putrificant;
c) croupous;
d) catharal;
e) hemorrhagic.

249. In what localization does croupous form of fibrinous inflammation develop only?
a) pleura;
b) tonsils;
c) colon;
d) urinary bladder;
e) uterine body.

250. In what localization is diphtheroid form of fibrinous inflammation possible only?
a) pleura;
b) peritoneum;
c) pericardium;
d) tonsils;
e) in all enumerated localizations.

251. What morphological type of fibrinous inflammation does on oral mucosa develop?
a) phlegmonous;
b) interstitial;
c) hemorrhagic;
d) anaerobic;
e) diphtheroid.

252. Call the localization where both types of fibrinous inflammation can develop:
a) tonsils;
b) oral cavity;
c) pleura;
d) pericardium;
e) colon.

253. What is wrong about diphtheroid inflammation?
a) it develops on tonsils;
b) membrane of exudate is strongly attached to mucosa;
c) ulcers form at the exudate membrane disattachment;
d) it is the variant of catharal inflammation;
e) in diphtheria develops.

254. What is wrong about croupous inflammation?
a) it develops on trachea mucosa;
b) it is the variant of fibrinous inflammation;
c) membrane of exudate is not strongly attached to mucosa;
d) deep ulcers form at the exudate membrane disattachment;
e) in diphtheria develops.

255. Development of croupous or diphtheroid form of fibrinous inflammation in colon is defined by:
a) type of infectious agent;
b) form of clinical course;
c) strength of blood circulation disturbances;
d) depth of necrosis;
e) leukocytes activity.

256. The most often outcome of fibrinous inflammation is:
a) resolution;
b) coming to purulent;
c) organization;
d) mucoidazation;
e) functional disorders.

257. Heart was named “hairy” (“bread and butter”) in following changes of pericardium:
a) organization of exudate;
b) fibrin sedimentation;
c) pus appearance;
d) development of connective tissue adhesions;
e) exudate petrifaction.

258. Heart was named “testaceous” in following changes of pericardium:
a) fibrin sedimentation;
b) pus appearance;
c) growth of tumour;
d) organization and petrifaction of exudate;
e) development of connective tissue adhesions.

259. What is the most often cause of purulent inflammation?
a) viruses;
b) protozoa;
c) chemical substances;
d) toxins;
e) staphylococci.

260. At microscopic investigation purulent exudate is diagnosing on to lot of:
a) fibrin;
b) neutrophyls;
c) macrophages;
d) lymphocytes;
e) erythrocytes.

261. The basic part of purulent exudate is:
a) water;
b) neutrophyls;
c) necrotic debris;
d) fibroblasts;
e) microbes.

262. Usual localization of purulent inflammation is:
a) serous membranes;
b) mucous membranes;

c) soft tissues;
d) any organ;
e) all enumerated.

263. Call the form of purulent inflammation:
a) abscess;
b) granuloma;
c) gangrene;
d) cyst;
e) hematoma.

264. What morphological form of inflammation does develop on tonsils in diphtheria?
a) diphtheroid;
b) croupous;
c) catharal;
d) purulent;
e) putrificant.

265. What morphological form of inflammation does develop in larynx and trachea in diphtheria?
a) diphtheroid;
b) croupous;
c) catharal;
d) purulent;
e) putrificant.

266. Call non-individual form of exudative inflammation which was emerged on the base of topography:
a) purulent;
b) putrificant;
c) hemorrhagic;
d) serous;
e) catharal.

267. Phlegmone it is the form of:
a) catharal inflammation;
b) croupous inflammation;
c) diphtheroid inflammation;
d) purulent inflammation;
e) putrificant inflammation.

268. Choose the complication of purulent inflammation:
a) hyperemia;
b) atrophy;
c) erosive bleeding;
d) edema;
e) cellular proliferation.

269. At visual inspection purulent exudate looks like:
a) transparent fluid;
b) muddy fluid;
c) creamy, greenish-yellowish fluid;
d) coloured by blood fluid;
e) mucus.

270. Localized (focal) purulent inflammation with the tissues necrosis and cavity formation is called:
a) abscess;
b) phlegmone;
c) empyema;
d) cyst;
e) granuloma.

271. The most often outcome of acute abscess is:
a) coming to chronic;
b) pus resorption and walls constriction;
c) pus condensation and its petrifaction;
d) pus condensation and its organization;
e) pus drain and cyst formation.

272. What does develop in abscess wall in its chronization?
a) necrotic debris;
b) tissue of organ infiltrated by leukocytes;
c) epithelial tissue;
d) fibrous tissue;
e) necrotic debris infiltrated by leukocytes.

273. Show the complication of chronic purulent inflammation:
a) hyperemia;
b) edema;
c) cellular proliferation;
d) secondary amyloidosis;
e) systemic hyalinosis.

274. Show the complication of acute purulent inflammation:
a) hyperemia;
b) edema;
c) severe intoxication;
d) secondary amyloidosis;
e) systemic hyalinosis.

275. Multiply small abscesses were found in organs of the dead patient with a purulent wound of the femur and regional thrombophlebitis. How had being named the developed complication?
a) abscess;
b) phlegmone;
c) gangrene;
d) septicemia;
e) septicopiemia.

276. What is the most often outcome of purulent inflammation?
a) organization;
b) petrifaction;
c) ossification;
d) vascularization;
e) amyloidosis.

277. What layer does form into chronic abscess wall?

a) necrotized tissue with leukocytes;
b) purulent exudate;
c) fibrous tissue;
d) epithelial tissue;
e) osseous tissue.

278. What is wrong about catharal inflammation?
a) develops on mucous membranes only;
b) it is the form of exudative inflammation;
c) it has acute clinical course only;
d) the most often its cause is infection;
e) changing of exudates is usual.

279. Show forms of acute catharal inflammation:
a) serous;
b) mucous;
c) purulent;
d) anaerobic;
e) all enumerated.

280. The duration of acute rinitis is about:
a) 24 hours;
b) 2-3 days;
c) 7 days;
d) 2-3 weeks;
e) 1 month.

281. Catharal inflammation it is:
a) exudative inflammation of mucous membranes with discharge of exudate;
b) exudative inflammation of mucous membranes with acute hyperemia;
c) inflammation with changing of exudates.

282. Call the change of a mucosa which is specific for chronic catharal inflammation:
a) edema;
b) hyperemia;
c) sclerosis;
d) desquamation of epithelium;
e) ulcers formation;

283. Call the change of a mucosa which is specific for chronic catharal inflammation:
a) edema;
b) hyperemia;
c) atrophy;
d) desquamation of epithelium;
e) ulcers formation;

284. Chronic catharal inflammation is dangerous because:
a) narrowing of lumen can develop;
b) malignant tumour development is possible;
c) ulcers with their following perforation can develop;
d) massive bleeding is possible;
e) it is associated with severe intoxication.

285. Catharal inflammation is characterized by:

a) discharge and flowing of exudate;
b) formation of membrane;
c) formation of ulcers and erosions;
d) deformation of lumen.

286. What is wrong about acute catharal inflammation?
a) localizes on mucous membranes;
b) exudate flows from surface;
c) there is mucus in exudate;
d) there is fibrin in exudate;
e) restoration of tissue it is its outcome.

287. What is wrong about chronic catharal inflammation?
a) can develop on bronchi, gastric mucosa, etc.;
b) it can be the cause of severe intoxication;
c) displasia of epithelium can develop;
d) carcinoma can develop;
e) it has long time duration (years).

288. Precanceromatous change of epithelium in chronic catharal inflammation is:
a) atrophy;
b) degeneration;
c) desquamation;
d) displasia;
e) all is true.

289. Usual outcome of acute catharal inflammation is:
a) sclerosis and deformation;
b) organization and petrifaction;
c) resolution and tissue repair;
d) ulceration and perforation;
e) development of carcinoma.

290. Exudate which is containing little of leukocytes and lot of fluid is calling:
a) serous;
b) purulent;
c) fibrinous;
d) hemorrhagic;
e) putrificant.

291. Exudate which is containing lot of neutrophyls is calling:
a) serous;
b) purulent;
c) fibrinous;
d) hemorrhagic;
e) putrificant.

292. Exudate which is containing lot of fibrin is calling:
a) serous;
b) purulent;
c) fibrinous;
d) hemorrhagic;
e) putrificant.

293. What is hematogenic cell of inflammatory infiltration?
a) endothelial;
b) tissue basophils;
c) fibroblast;
d) lymphocyte;
e) epithelioid.

294. What is histiogenic cell of inflammatory infiltration?
a) monocytes;
b) lymphocytes;
c) epithelioid;
d) neutrophils;
e) eosinophils.

295. What is usual outcome of chronic inflammation?
a) suppuration;
b) sclerosis;
c) petrifaction;
d) ossification;
e) tissue autolysis.

296. Call the morphological type of proliferative inflammation:
a) granulomatous;
b) purulent;
c) hemorrhagic;
d) anaerobic;
e) serous.

297. Proliferative inflammation it is inflammation with:
a) acute alteration;
b) granulomas formation;
c) predomination of proliferation;
d) growing of connective tissue;
e) acute exudation.

298. Usual clinical course of proliferative inflammation is:
a) acute;
b) subacute;
c) chronic;
d) rapid progressive.

299. What cells can multiply in focus of proliferative inflammation?
a) macrophages;
b) reticulocytes;
c) erythrocytes;
d) neutrophils;
e) basophils.

300. What is specific for the proliferative interstitial myocarditis?
a) foci of dry necrosis;
b) abscess formation;
c) acute clinical course;
d) round-cell infiltration there is in the stroma;
e) formation of giant-cell granuloma.

301. Usual outcome of proliferative interstitial inflammation is:
a) edema;
b) sclerosis;
c) suppuration;
d) petrifaction;
e) ossification.

302. Granuloma it is focus of:
a) purulent inflammation;
b) accumulation of lymphoid cells;
c) accumulation of cells can do phagocitosis;
d) caseous necrosis;
e) fibrous tissue.

303. What type of granuloma does not present?
a) epithelioid;
b) giant-cellular;
c) immune;
d) specific;
e) neutrophilic.

304. Show the type of granuloma according to the cellular composition:
a) specific;
b) giant-cellular;
c) immune;
d) lipogranuloma;
e) acute.

305. Choose non-infectious granuloma:
a) tuberculous;
b) oleogranuloma;
c) syphilitic;
d) in scleroma;
e) in leprosy.

306. Choose infectious granuloma:
a) oleogranuloma;
b) lipogranuloma;
c) syphilitic;
d) around of foreign body;
e) in asbestosis.

307. Non-immune granuloma develops in:
a) in alveococcosis;
b) in tuberculosis;
c) in syphilis;
d) in scleroma;
e) in leprosy.

308. Immune granuloma develops in:
a) in alveococcosis;
b) in asbestosis;
c) around of foreign body;

d) in tuberculosis;
e) in silicosis.

309. In what acute infectious disease granulomas are usual?
a) Schigella dysentery;
b) diphtheria;
c) scarlet fever;
d) salmonellosis;
e) yersiniosis.

310. What is wrong about tuberculous granuloma?
a) miliary;
b) there is wet necrosis in the center;
c) epithelioid;
d) immune;
e) specific.

311. What is wrong about tuberculous granuloma?
a) miliary;
b) there is dry necrosis in the center;
c) lot of epithelioid cell;
d) healing with small scar formation;
e) its suppuration is possible.

312. What cells are there in tuberculous granuloma?
a) Aschoff cells;
b) Hodgkin cells;
c) Reed- Schternberg cells;
d) Virchov cells
e) Pirogov-Langhance cells.

313. What is wrong about syphilitic granuloma?
a) solitary;
b) immune;
c) specific;
d) non-infectious;
e) healing with large scar formation.

314. Fallowing elements are parts of gumma, except:
a) necrotic debris;
b) Reed- Schternberg cells;
c) lymphoid cells;
d) plasmatic cells;
e) epithelioid cells.

315. What granuloma's type does develop around of suture material?
a) immune;
b) specific;
c) giant-cellular;
d) injections;
e) with intensive metabolism.

316. What is the outcome of granuloma?
a) sclerosis;

b) suppuration;
c) mucoidazation;
d) resolution;
e) cyst formation.

317. What is favorable outcome of tuberculous granuloma?
a) suppuration;
b) hemorrhagic infiltration;
c) putrefaction;
d) scaring;
e) necrosis.

318. What is the complication of syphilitic mesaortitis?
a) aneurysm of abdominal aorta;
b) aneurysm of thoracic aorta;
c) myocardial infarction;
d) cardiosclerosis;
e) aortic valve defect.

319. Granuloma with unknown etiology develops in:
a) rheumatic fever;
b) tuberculosis;
c) syphilis;
d) sarcoidosis (Besnier-Boeck-Schaumann's disease);
e) scleroma.

320. Usual localization of inflammatory polyps is:
a) serous membranes;
b) meningeas;
c) anal-genital area mucosa;
d) nasal mucosa;
e) everywhere.

321. Usual localization of pointed condilomas is:
a) serous membranes;
b) meningeas;
c) anal-genital area mucosa;
d) bronchial mucosa;
e) nasal mucosa.

322. What is wrong about syphilitic granuloma?
a) its synonym is gumma;
b) at visual inspection it is one large focus;
c) big scar develops as its result;
d) develops in tertiary syphilis;
e) localizes in liver only.

323. Choose non-immune granuloma:
a) in tuberculosis;
b) in syphilis;
c) in scleroma;
d) in leprosy;
e) around of foreign body.

324. What granuloma does in tuberculosis develop?
a) macrophagal;
b) epithelioid;
c) giant-cellular;
d) necrotic;
e) regenerative.

325. Specific granuloma develops in:
a) rheumatic fever;
b) tuberculosis;
c) yersiniosis;
d) echinococcus;
e) around of suture material.

326. Intensive metabolism is observed in:
a) granuloma around of foreign body;
b) lipogranuloma;
c) tuberculous granuloma;
d) granuloma around of suture material;
e) echinococcal granuloma.

327. What is the one of characteristics of granulomatous diseases?
a) acute clinical course;
b) total recovery is often;
c) accompanied with immunity disorders;
d) exudation develops always;
e) caseous necrosis occurs always.

328. What is it proliferation?
a) cellular death;
b) cellular injury;
c) result of inflammation;
d) multiplication of cells;
e) synonym of regeneration.

329. Increasing of functional elements volume is accompanied with increasing of function is called:
a) degeneration;
b) displasia;
c) hypertrophy;
d) atrophy;
e) metaplasia.

330. Hypertrophy it is:
a) restoration of tissues after injury;
b) increasing of cellular, tissue or organ's volume;
c) decreasing of cellular, tissue or organ's volume;
d) changing type of tissue;
e) substitution by connective tissue.

331. Increased number of cellular elements is called:
a) degeneration;
b) displasia;
c) hypertrophy;

d) hyperplasia;
e) metaplasia.

332. Choose the type of hypertrophy:
a) working (compensatory);
b) neurotic;
c) compressive;
d) cerebral;
e) dysfunctional.

333. Glandular hyperplasia of endometrium it is:
a) working hypertrophy;
b) vicar hypertrophy;
c) correlative hypertrophy;
d) neurohumoral hypertrophy.

334. What type of myocardial hypertrophy does in defective valvular heart disease develop?
a) working hypertrophy;
b) vicar hypertrophy;
c) correlative hypertrophy;
d) neurohumoral hypertrophy.

335. What type of myocardial hypertrophy does in arterial hypertension develop?
a) working hypertrophy;
b) vicar hypertrophy;
c) correlative hypertrophy;
d) neurohumoral hypertrophy.

336. Myocardial hypertrophy develops as result of:
a) cardiomyocites sizes increasing;
b) cardiomyocites number increasing;
c) stromal edema;
d) cardiomyocites intracellular accumulations.

337. The cause of physiological myocardial hypertrophy is:
a) defective valvular heart disease;
b) cardiosclerosis;
c) physical training;
d) arterial hypertension;
e) toxic myocarditis.

338. Myocardial hypertrophy in phase of its compensation is characterizing by fallowing sign only :
a) decreasing of heart sizes;
b) thickening of ventriculars walls;
c) dilation of chambers;
d) flabby consistency of myocardium;
e) fat degeneration of cardiomyocites.

339. What does develop in hypertrophic myocardium in phase of decompensation?
a) atrophy of cardiomyocites;
b) hyperplasia of cardiomyocites;
c) degeneration of cardiomyocites;

d) tissue repair;
e) hypertrophy of cardiomyocites.

340. What does develop in heart in decompensation?
a) increasing of cardiomyocites number;
b) increasing of cardiomyocites sizes;
c) atrophy of cardiomyocites;
d) degeneration of cardiomyocites.

341. What is the manifestation of hypertrophic heart decompensation?
a) myogenic dilation of chambers;
b) brown atrophy of myocardium;
c) rheumatic myocarditis;
d) fibrinous pericarditis;
e) acute verrucous endocarditis.

342. In what organ does vicar hypertrophy develop?
a) heart;
b) stomach;
c) kidney;
d) uterus;
e) uterine bladder.

343. The phase of hypertrophic heart decompensation is characterizing by the fallowing sign:
a) flabby consistency of myocardium;
b) paleness of myocardium;
c) thickening of ventricles walls;
d) increasing of heart mass;
e) increasing of cardiomyocites sizes.

344. Neurohumoral hypertrophy develops in:
a) heart in arterial hypertension;
b) breasts in pregnancy;
c) urinary bladder in prostatic hyperplasia;
d) kidney in removing of second one;
e) stomach wall in pilorostenosis.

345. Decreasing of structural elements volume in living organism is called:
a) hypertrophy;
b) hyperplasia;
c) atrophy;
d) hypoplasia;
e) displasia.

346. What is the example of local atrophy?
a) dysfunctional;
b) cancеromatous cachexia;
c) hypophysial cachexia;
d) cerebral cachexia;
e) alimentary emaciation;

347. What is the example of general atrophy?
a) alimentary emaciation;

b) neurotic atrophy;
c) atrophy due to long time compression;
d) dysfunctional;
e) all enumerated.

348. What is the example of local atrophy?
a) vicar;
b) carcinomatous;
c) ischemic;
d) cerebral;
e) hypophysial.

349. The example of atrophy due to long time compression is:
a) atrophy of bone marrow in radial illness;
b) atrophy of kidney in urolithiasis;
c) atrophy of muscles in bone fracture;
d) atrophy of myocardium in coronary atherosclerosis.

350. The example of atrophy due to influence of physics factors is:
a) atrophy of bone marrow in radial illness;
b) atrophy of kidney in urolithiasis;
c) atrophy of muscles in bone fracture;
d) atrophy of adrenals cortex in corticosteroid hormones using.

351. The example of atrophy due to chronic ischemia is:
a) focal atrophy of myocardium in coronary atherosclerosis;
b) atrophy of adrenals cortex in corticosteroid hormones using;
c) atrophy of muscles in bone fracture;
d) atrophy of optic nerve after eyeball removing.

352. Brown atrophy can develop in:
a) stomach;
b) lung;
c) prostate;
d) kidney;
e) liver.

353. What does in brain develop as result of liquor outflow difficulties?
a) edema and swelling;
b) hydrocephalus;
c) tumour;
d) meningitis;
e) encephalitis.

354. Changing of tissue type on related one is called:
a) displasia;
b) anaplasia;
c) hyperplasia;
d) metaplasia;
e) malignancy.

355. Metaplasia of connective tissue is possible in:
a) osseous;
b) muscular;

c) nerve;
d) epithelial;
e) blood reconstruction tissue.

356. What epithelium does develop in metaplasia of bronchial mucosa?
a) columnar;
b) prismatic;
c) squamous-cell;
d) atrophic;
e) mesotelium.

357. Metaplasia of bronchial epithelium develops as result of:
a) lymphostasis;
b) hyperemia;
c) necrosis;
d) acute inflammation;
e) chronic inflammation.

358. Metaplasia of bronchial epithelium can come to:
a) degeneration;
b) atrophy;
c) necrosis;
d) carcinoma;
e) inflammation.

359. What is the synonym of general atrophy?
a) hypoplasia (aplasia);
b) emaciation (cachexia);
c) hypertrophy (hyperplasia);
d) dwarfism.

360. Growth of connective tissue into pathological focus is called:
a) metaplasia;
b) encapsulation;
c) organization;
d) petrifaction;
e) displasia.

361. Growth of connective tissue around of pathological focus is called:
a) metaplasia;
b) encapsulation;
c) organization;
d) petrifaction;
e) displasia.

362. Focal sclerosis in place of pathological focus is called:
a) cyst;
b) cardiosclerosis;
c) scar;
d) cirrhosis;
e) capsule.

363. Massive sclerosis of organ with its reorganization and deformation is called:
a) scar;

b) diffuse sclerosis;
c) cirrhosis;
d) diffuse fibrosis;
e) focal fibrosis.

364. Disturbance of cellular proliferation and differentiation with appearance of cellular atypia in some cells is called:
a) hyperplasia;
b) metaplasia;
c) anaplasia;
d) displasia;
e) organization.

365. Choose the type of wound healing:
a) organization;
b) encapsulation;
c) metaplasia;
d) primary intension;
e) all enumerated.

366. Restoration of structural tissue elements instead dead is called:
a) organization;
b) tissue repair;
c) metaplasia;
d) displasia;
e) anaplasia.

367. What is it granulation tissue?
a) fibrillary connective tissue;
b) young connective tissue;
c) mature connective tissue;
d) good-vascularized connective tissue;
e) connective tissue with lot of cells.

368. What cannot be the structural element of granulation tissue?
a) multiplying fibroblasts;
b) multiplying endothelial cells;
c) reticular fibers;
d) plenty of collagenic fibers;
e) thin blood vessels.

369. Scar tissue is characterized by the:
a) plenty of multiplying fibroblasts;
b) plenty of reticular fibers;
c) lot of collagenic fibers;
d) lot of blood vessels;
e) intensive leukocytes infiltration.

370. Compensatory heart hypertrophy develops in:
a) DIC-syndrome;
b) shock;
c) acute myocarditis;
d) arterial hypertension;
e) toxic myocardial degeneration.

371. What is classified as atrophy?
a) agenesia (absence) of organ;
b) aplasia (staing of organ as embrional rudiment) of organ;
c) hypoplasia of organ (underdeveloped organ);
d) decreasing of organ's sizes in living organism;
e) all enumerated.

372. What is classified as physiological atrophy?
a) atrophy of sexual glands in elderly age;
b) atrophy due to long time compression;
c) dysfunctional atrophy;
d) ischemic atrophy;
e) all enumerated.

373. What pigment accumulates in organs in alimentary emaciation?
a) melanin;
b) hemosiderin;
c) lipofuscin;
d) bilirubin;
e) hydrochloric acid hematin.

374. Tumour it is pathological process characterized by:
a) no adequate multiplying of immature cells;
b) proliferation and hyperplasia of cells;
c) hyperplasia and metaplasia of cells;
d) multiplying and differentiation of cells.

375. The one of the tissue atypia appearances in tumour is:

a) different forms of cells;
b) different sizes of cellular nuclei;
c) different forms of cellular nuclei;
d) disturbed alignment of fibers and cells;
e) disturbances of cellular structure.

376. Choose the type of tumour growth into tissues:
a) unicentric;
b) infiltrative;
c) exophytic
d) endophytic
e) implantation.

377. Choose the type of tumour growth in cavitary organ:
a) unicentric;
b) multicentric;
c) appositional;
d) exophytic;
e) infiltrative.

378. Call the pathway of metastatic spreading:
a) implantation;
b) infiltrative;
c) expansive;

d) appositional;
e) local destructive.

379. Call the pathway of metastatic spreading:
a) unicentric;
b) multicentric;
c) lymphogenic
d) infiltrative;
e) appositional.

380. Choose the type of tumour growth in cavitary organ:
a) unicentric;
b) multicentric;
c) appositional
d) endophytic;
e) infiltrarive.

381. Morphological atypia of tumour can be:
a) antigenic and histochemical;
b) exophytic and histochemical;
c) expansive and infiltrative;
d) tissue and cellular.

382. Organoid tumour has:
a) good developed parenchyma;
b) good developed stroma;
c) two components: parenchyma and stroma:
d) two same volume components: stroma and parenchyma.

383. Histioid tumour has:
a) good developed parenchyma;
b) good developed stroma;
c) two components: stroma and parenchyma;
d) two same volume components: stroma and parenchyma.

384. What is not there in metastatic spreading process:
a) disattachment of tumour cells from basic tumour node;
b) carrying of tumour cells;
c) development of secondary tumour nodes;
d) development of necroses and hemorrhages into tumour nodes.

385. Choose the definition of sarcoma:
a) immature tumour from fibrous tissue;
b) immature tumour from mesenchymal tissues;
c) mature tumour from mesenchymal tissues;
d) mature tumour from fibrous tissue.

386. On what principle the international tumours classification is based?
a) anatomical;
b) topographic;
c) histogenetic;
d) histochemical;
e) antigenic.

387. Choose the type of tumour growth into tissues:
a) exophytic;
b) endophytic;
c) expansive;
d) unicentric;
e) multicentric.

388. Choose the type of tumour growth in cavitary organ:
a) expansive;
b) infiltrative;
c) unicentric;
d) multicentric;
e) exophytic.

389. What is the basic structural element of a tumour?
a) stroma;
b) parenchyma;
c) blood vessels;
d) necrosis;
e) hemorrhages.

390. What is wrong about benign tumour?
a) tumour cells are differentiated;
b) it has expansive growth;
c) never spreads;
d) never relapse;
e) makes general influence on organism.

391. What is wrong about malignant tumour?
a) tumour cells are poorly-differentiated;
b) it has infiltrative growth;
c) never relapse;
d) can spread;
e) makes general influence on organism.

392. What is the tumour with local destructive growth?
a) malignant tumour with infiltrative growth;
b) tumour with only one feature of malignancy – infiltrative growth;
c) tumour which is never spreading;
d) tumour with appositional growth.

393. Choose the tumour with local destructive growth:
a) venous hemangioma;
b) cavernous hemangioma;
c) capillary hemangioma;
d) chondroma;
e) fibroma of skin.

394. Choose the tumour with local destructive growth:
a) chondroma;
b) lipoma;
c) fibroma of skin;
d) nasopharingeal angiofibroma;
e) angiosarcoma.

395. Call mesenchymal tumour:
a) adenoma;
b) angiosarcoma;
c) papilloma;
d) hepatoma;
e) osseous calculus.

396. Call benign mesenchymal tumour:
a) nasopharingeal angiofibroma;
b) fibroma of skin;
c) leiomyosarcoma;
d) desmoid;
e) chondrosarcoma.

397. What can be classified as benign mesenchymal tumour?
a) fibromyoma;
b) leiomyosarcoma;
c) osteosarcoma;
d) desmoid;
e) liposarcoma.

398. What can be classified as malignant mesenchymal tumour?
a) fibromyoma;
b) leiomyosarcoma;
c) nasopharingeal angiofibroma;
d) desmoid;
e) chondroma.

399. Cavernous hemangioma of liver is characterized by the following feature only:
a) tissue and cellular atypia;
b) immature cells;
c) malignant;
d) consists of venous type blood vessels;
e) has hematogenic spreading.

400. Choose the malignant mesenchymal tumour:
a) liposarcoma;
b) desmoid;
c) capillary hemangioma;
d) osteoma;
e) fibroma.

401. The benign tumour from muscular tissue is:
a) fibroma;
b) fibrosarcoma;
c) hemangioma;
d) leiomyoma;
e) leiomyosarcoma.

402. What is the favorite pathway of spreading in sarcomas?
a) lymphogenic;
b) hematogenic;
c) perineural;

d) contact.

403. What is wrong about capillary hemangioma?
a) it is mature tumour;
b) it has local destructive growth;
c) it has metastatic spreading;
d) it develops from blood vessels;
e) skin it is its most often localization.

404. What is the most often localization of leiomyoma?
a) skin;
b) heart;
c) uterus;
d) soft tissues;
e) stomach.

405. Choose the histological type of fibrosarcoma:
a) soft;
b) dense;
c) undifferentiated;
d) youthful;
e) desmoid.

406. What type of hemangioma is non-existent?
a) capillary;
b) venous;
c) arterial;
d) cavernous;
e) glomus-angioma.

407. What is the first metastases localization in fibrosarcoma of low extremity soft tissues?
a) bones;
b) regional lymphatic nodes;
c) kidneys;
d) liver;
e) lungs.

408. What is the first metastases localization in sarcoma of small intestinal mesentery?
a) bones;
b) regional lymphatic nodes;
c) kidneys;
d) liver;
e) lungs.

409. What is wrong about cavernous hemangioma of liver?
a) it has tissue atypia;
b) its cells are mature;
c) there are no symptoms usually;
d) it consists of venous type blood vessels;
e) it has hematogenic spreading.

410. What is the precancer disease in melanoma development?
a) nevus;
b) displasia of melanocytes;

c) pigment spot;
d) vitiligo;
e) leukodermia.

411. What is the most often localization of melanoma?
a) skin;
b) eyeball;
c) rectum;
d) oral cavity;
e) lungs.

412. What is wrong about melanoma?
a) it is malignant;
b) it has metastatic spreading;
c) it has expansive growth;
d) it can be without pigment;
e) it can relapse.

413. What is wrong about nevus?
a) it is tumour-like formation:
b) it is dangerous if displasia there is in it;
c) it always malignises;
d) it can be without pigment;
e) its usual localization is skin.

414. Choose the most malignant tumour:
a) hemangioma;
b) liposarcoma;
c) well-differentiated fibrosarcoma;
d) angiosarcoma;
e) desmoid.

415. Choose the most malignant tumour:
a) well-differentiated fibrosarcoma;
b) osteoblastoclastoma;
c) melanoma;
d) liposarcoma;
e) desmoid.

416. Sarcoma it is immature tumour from:
a) epithelium;
b) blood restoration tissue;
c) fibrous tissue;
d) mesenchymal tissues;
e) lymphoid tissue.

417. Immature tumour from blood vessels is:
a) hemangioma;
b) angiosarcoma;
c) histiocytoma;
d) lymphangioma;
e) lymphangiosarcoma.

418. Immature tumour from mesenchymal tissues is:

a) adenoma;
b) papilloma;
c) carcinoma;
d) cancer;
e) sarcoma.

419. Mature, benign tumour from fibrous (connective) tissue is:
a) angioma;
b) fibroma;
c) papilloma;
d) adenoma;
e) carcinoma.

420. Mature tumour from blood vessels is:
a) lymphagioma;
b) hemangioma;
c) hemangiosarcoma;
d) carcinosarcoma;
e) masenchymoma.

421. Disturbed cellular differentiation is called:
a) atypia;
b) anaplasia;
c) atrophy;
d) metaplasia;
e) hypoplasia.

422. Set of features these distinguish any tumour from healthy tissue is called:
a) atypia;
b) anaplasia;
c) atrophy;
d) malignancy;
e) kataplasia.

423. What is the characteristic of malignant tumour?
a) consists of poorly-differentiated cells;
b) never metastasizes;
c) never relapses;
d) it has expansive growth;
e) makes only local influence.

424. Tumour relapse it is:
a) development of new tumour;
b) renoval growth of tumour on the place where it was removed;
c) acceleration of tumour growth;
d) form of metastatic spreading;
e) development of new tumour foci.

425. One of the modern methods of tumour histogenesis defining is:
a) histochemical;
b) histological;
c) cytological;
d) immunomorphological;
e) ultrasonic investigation.

426. Realization of local influence of tumour on organism is:
a) increasing of ESR;
b) anemia;
c) cachexia;
d) destruction of surrounded tissues;
e) hormonal disorders.

427. Realization of general influence of tumour on organism is:
a) atrophy of surrounded tissues;
b) destruction of surrounded tissues;
c) cachexia;
d) compression of blood vessels;
e) deformation of organs and tissues.

428. Mature tumour it is:
a) tumour which consists of the same cells that cells of organ;
b) tumour which consists of differentiated cells;
c) tumour which consists of poorly-differentiated cells;
d) tumour without invasive growth;
e) never metastizing tumour.

429. Mature tumour it is:
a) tumour which consists of poorly-differentiated cells;
b) tumour which consists of differentiated cells;
c) never metastizing tumour;
d) does not make general influence on organism;
e) any small tumour.

430. Immature tumour it is:
a) tumour which consists of poorly-differentiated cells;
b) tumour which can metastasize;
c) tumour which can relapse;
d) tumour which can destroy surrounded tissues;
e) all enumerated.

431. What type of structure do epithelial tumours have?
a) organoid;
b) histioid.

432. Choose the name of mature epithelial tumour:
a) lipoma;
b) fibroma;
c) adenoma;
d) carcinoma;
e) cancer.

433. Choose the mature tumour which develops from squamous-cell epithelium:
a) adenoma;
b) papilloma;
c) carcinoma;
d) cystic adenoma;
e) lymphangioma.

434. Choose the morphological type of adenoma:
a) papilloma;
b) hemangioma;
c) cystadenoma;
d) adenocarcinoma;
e) angiofibroma.

435. Call the morphological type of adenoma:
a) angiosarcoma;
b) fibrosarcoma;
c) fibroadenoma;
d) adenocarcinoma;
e) angiofibroma.

436. What is right there in the characteristic of papilloma?
a) immature tumour;
b) exophitic growth is typical;
c) it can malignize;
d) rapid growth;
e) makes general influence on organism.

437. What is wrong about adenoma?
a) it is mature tumour;
b) it has only tissue atypia;
c) it has slow growth;
d) makes general influence on organism always;
e) can malignize.

438. What is wrong about benign epithelial tumour?
a) tissue atypia;
b) histioid type of structure;
c) expansive growth;
d) never metastatises;
e) never relapse.

439. What is the most often papilloma's localization?
a) stomach;
b) esophagus;
c) skin;
d) urinary tract;
e) pleura.

440. What is the most often cystadenoma's localization?
a) stomach;
b) rectum;
c) breast;
d) pancreas;
e) ovary.

441. What is the most often fibroadenoma's localization?
a) stomach;
b) rectum;
c) breast;
d) pancreas;

e) ovary.

442. What does develops in adenoma malignization?
a) adenocarcinoma;
b) ring-cell carcinoma;
c) mucoid carcinoma;
d) solid carcinoma;
e) scirrhous carcinoma.

443. What change of epithelium can be obligate precancer?
a) hyperplasia;
b) metaplasia;
c) proliferation;
d) displasia of I-II degree;
e) displasia of III degree.

444. Carcinoma it is:
a) every tumour from epithelium;
b) mature tumour from epithelium;
c) immature tumour from epithelium;
d) immature tumour from glandular epithelium;
e) every tumour from glandular epithelium.

445. What is wrong about "carcinoma in situ"?
a) does not grow right through basement membrane;
b) background is severe displasia:
c) never metastasizes;
d) never relapses;
e) has bad prognosis.

446. Histological feature of "carcinoma in situ" is:
a) invasive growth;
b) metastatic spreading;
c) intraepithelial malignant growth;
d) hemorrhages into tumour;
e) necrosis of tumour.

447. What is wrong about displasia?
a) it is cellular proliferation with disturbed differentiation;
b) part of cells with atypia;
c) it is reversible pathology;
d) its III degree malignize usually;
e) it has the beginning of invasive growth.

448. Squamous-cell carcinoma is especially typical for:
a) thyroid gland;
b) pancreas;
c) uterine cervix;
d) uterine body;
e) stomach.

449. The morphological sign of well-differentiated squamous-cell carcinoma is:
a) keratinization;
b) mucus formation;

c) solid structures;
d) pathological mitoses;
e) inflammatory infiltration.

450. Usually primary malignant tumour of esophagus it is:
a) adenocarcinoma;
b) squamous-cell carcinoma;
c) undifferentiated carcinoma;
d) melanoma;
e) leiomyosarcoma.

451. Adenogenic carcinoma more often develops in:
a) stomach;
b) esophagus;
c) bronchi;
d) uterine cervix;
e) urine bladder.

452. Morphological sign of carcinoma as malignant tumour is:
a) formation of glandular-like structures;
b) mucus formation;
c) cellular atypia;
d) keratinization;
e) less of stroma.

453. What does not define as poorly-differentiated carcinoma?
a) adenocarcinoma;
b) solid carcinoma;
c) colloid carcinoma;
d) medullar carcinoma;
e) fibrous carcinoma.

454. Early pathway of carcinoma metastatic spreading is:
a) hematogenic;
b) lymphogenic;
c) contact;
d) perineural.

455. Early carcinoma metastases localize into:
a) regional lymphatic nodes;
b) distant lymphatic nodes;
c) lungs;
d) liver.

456. What is wrong about adenocarcinoma?
a) develops from columnar epithelium;
b) forms glandular-like structures;
c) has cellular atypia;
d) has high malignancy;
e) has invasive growth.

457. The basic criteria of invasive growth beginning is:
a) intensive mitotic activity;
b) sharp cellular atypia;

c) destruction of basement membrane;
d) deep akantosis;
e) severe displasia as background.

458. Carcinoma which develops from protective epithelium is called:
a) adenocarcinoma;
b) solid;
c) fibrous;
d) squamous-cell carcinoma;
e) colloid.

459. Unpleasant prognostic sign of carcinoma is:
a) mild cellular atypia;
b) non-intensive mitotic activity;
c) superficial invasion;
d) small size of tumour;
e) cancer embolism of blood vessels.

460. Colloid carcinoma is characterized by the fallowing feature only:
a) cellular atypia in association with mucus hyperproduction;
b) expansive growth;
c) absence of relapse;
d) late metastatic spreading;
e) good prognosis.

461. Fibrous (scirrhous) carcinoma is characterized by the fallowing feature only:
a) mild malignancy;
b) late metastatic spreading;
c) lot of stroma with complexes of atypical cells;
d) absence of relapse;
e) good prognosis.

462. Choose the carcinoma with high malignancy:
a) endometrial adenocarcinoma;
b) undifferentiated lung carcinoma;
c) squamous-cell carcinoma of uterine cervix;
d) squamous-cell carcinoma of lower lip.

463. Choose the carcinoma with high malignancy:
a) endometrial adenocarcinoma;
b) gastric adenocarcinoma;
c) breast scirrhous carcinoma;
d) squamous-cell lung carcinoma;
e) squamous-cell carcinoma of lower lip.

464. The basic distinguishing morphological feature of adenocarcinoma is:
a) lot of mitoses;
b) sharp cellular atypia;
c) formation of glandular-like structures;
d) mucus formation;
e) keratinization.

465. Call immature tumour from epithelium:
a) adenoma;

b) papilloma;
c) sarcoma;
d) carcinoma;
e) displasia.

466. Call mature tumour from columnar epithelium:
a) adenoma;
b) papilloma;
c) sarcoma;
d) carcinoma;
e) cancer.

467. The most pleasant prognosis there is in:
a) adenocarcinoma;
b) “carcinoma in situ”;
c) squamous-cell carcinoma;
d) fibrous carcinoma;
e) ring-cell carcinoma.

468. What is wrong about “carcinoma in situ”?
a) never spreads;
b) tumour cell grow into lymphatic vessels;
c) it is so called “zero” clinical stage of carcinoma;
d) it has good prognosis after treatment;
e) it comes in invasive carcinoma.

469. Carcinoma it is immature tumour from:
a) epithelium;
b) connective tissue;
c) blood restoration tissue;
d) serous membranes;
e) mesenchyma.

470. According to the tumour progression theory every tumour cell develops in way of:
a) increasing of maturing;
b) increasing of differentiation;
c) increasing of malignancy;
d) decreasing of malignancy.

471. Manifestation of tissue atypia in epithelial tumour is:
a) different forms and number of glandular structures;
b) different forms and number of epithelial cells;
c) increased sizes of nuclei in tumour cells;
d) intensive mitotic activity;
e) cellular pleomorphism.

472. Manifestation of cellular atypia in epithelial tumour is:
a) different forms and number of glandular structures;
b) different forms and number of epithelial cells;
c) disordered containment stroma and parenchyma;
d) predomination of stroma;
e) necrosis and hemorrhages.

473. Call the form of carcinoma:

a) adenoma;
b) fibroadenoma;
c) scirrhous;
d) cystadenoma;
e) papilloma.

474. What is right for fibrous (scirrhous) carcinoma?
a) it is poorly-differentiated carcinoma;
b) average cellular atypia;
c) builds glandular-like structures;
d) contains less of stroma;
e) has soft consistency.

475. What is right for adenocarcinoma?
a) it is poorly-differentiated carcinoma;
b) builds glandular-like structures from atypical cells;
c) has expansive growth;
d) never relapse;
e) metastatic spreading is rare.

476. What is right for squamous-cell carcinoma?
a) it can develop on base of epithelial displasia;
b) it has high malignancy;
c) never relapses;
d) makes only local influence;
e) has expansive growth.

477. What is right for pleomorphic-cell carcinoma?
a) lot of stroma;
b) slow growth;
c) consists of cells with very sharp cellular atypia;
d) has late metastatic spreading;
e) good prognosis.

478. Call usual localization of the first metastasis of carcinoma:
a) surrounded tissues;
b) regional lymphatic nodes;
c) liver;
d) lungs;
e) mesentery.

479. Retrograde metastatic spreading of tumour it is:
a) spreading of tumour cell along lymph supply;
b) spreading of tumour cell against lymph supply;
c) coming of tumour cells into lymphatic nodes;
d) coming of tumour cells in thoracic lymphatic duct.

480. Carcinoma of what organ can have early (rapid) wide-spread metastases?
a) stomach;
b) skin;
c) uterine cervix;
d) uterine body;
e) breast.

481. Carcinoma of what organ can have early (rapid) wide-spread metastases?

a) stomach;
b) skin;
c) uterine cervix;
d) uterine body;
e) lung.

RIGHT ANSWERS

1. c
2. e
3. c
4. a
5. a
6. c
7. e
8. e
9. d
10. d
11. a
12. e
13. d
14. b
15. e
16. e
17. e
18. d
19. c
20. d
21. b
22. d
23. e
24. d
25. c
26. a
27. b
28. a
29. b
30. a
31. d
32. b
33. a
34. a
35. c
36. b
37. a
38. e
39. c
40. b

41. b
42. e
43. c
44. c
45. c
46. b
47. c
48. d
49. b
50. a
51. d
52. b
53. a
54. a
55. b
56. d
57. d
58. c
59. c
60. a
61. b
62. b
63. b
64. d
65. c
66. b
67. c
68. c
69. c
70. b
71. a
72. d
73. a
74. b
75. b
76. b
77. c
78. e
79. c
80. c
81. b
82. b
83. a
84. c
85. b
86. a
87. d
88. a
89. e
90. e
91. b
92. a
93. d
94. c
95. b
96. a
97. c
98. d
99. d
100. d
101. e
102. d
103. b
104. e
105. b

106.b
107.e
108.d
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113.d
114.b
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116.a
117.d
118.d
119.d
120.a
121.c
122.b
123.d
124.b
125.b
126.a
127.b
128.a
129.e
130.c
131.b
132.b
133.d
134.b
135.c
136.e
137.b
138.e
139.b
140.c
141.e
142.b
143.e
144.d
145.a
146.d
147.c
148.b
149.a
150.b
151.c
152.a
153.d
154.b
155.b
156.b
157.a
158.c
159.d
160.e
161.e
162.a
163.b
164.b
165.d
166.a
167.e
168.a
169.b
170.e

171.c
172.b
173.b
174.b
175.e
176.b
177.b
178.e
179.e
180.a
181.e
182.d
183.c
184.a
185.c
186.b
187.c
188.a
189.c
190.c
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227.e
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234.b
235.c

236.d
237.e
238.d
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244.c
245.c
246.c
247.d
248.c
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251.e
252.e
253.d
254.d
255.d
256.c
257.b
258.d
259.e
260.b
261.b
262.e
263.a
264.a
265.b
266.e
267.d
268.c
269.c
270.a
271.d
272.d
273.d
274.c
275.e
276.a
277.c
278.c
279.e
280.c
281.a
282.c
283.c
284.b
285.a
286.d
287.b
288.d
289.c
290.a
291.b
292.c
293.d
294.c
295.b
296.a
297.c
298.c
299.a
300.d

301.b
302.c
303.e
304.b
305.b
306.c
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308.d
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363.c
364.d
365.d

366.b
367.b
368.d
369.c
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377.d
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384.d
385.b
386.c
387.c
388.e
389.b
390.e
391.c
392.b
393.c
394.d
395.b
396.b
397.a
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401.d
402.b
403.c
404.c
405.c
406.c
407.e
408.d
409.e
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411.a
412.c
413.c
414.d
415.c
416.d
417.b
418.e
419.b
420.b
421.b
422.a
423.a
424.b
425.d
426.d
427.c
428.b
429.b
430.e

431.a
432.c
433.b
434.c
435.c
436.b
437.d
438.b
439.c
440.e
441.c
442.a
443.e
444.c
445.e
446.c
447.e
448.c
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456.d
457.c
458.d
459.e
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463.c
464.c
465.d
466.a
467.b
468.b
469.a
470.c
471.a
472.b
473.c
474.a
475.b
476.a
477.c
478.b
479.b
480.e
481.e

4.3. Questions for colloquiums, interviews *(specify the competence code):*

4.4. Tasks (assessment tools) for the exam/credit

https://docs.yandex.ru/docs/view?url=ya-browser%3A%2F%2F4DT1uXEPRrJRXlUFoewruPzhnM8AyLNqGTui6m1EV0M6RPkVu1uFlYsY73XRaA74QhdbxaxlgMJAtr85ycsSxmwMvYVz2RrFcY291lNk_rFsIwWdLXnRdglfss-niO0BM329oenAgSlRPQMIcvo5Gg%3D%3D%3Fsign%3D3S3BHr-avlwOWcwGXyTEcjMBm3jhZYT3tYP2cNLkM3k%3D&name=Pathological%20anatomy%202%20term.docx&nosw=1

The full package of examination tasks/tasks is given *(specify the competence code):*

CARCINOMAS OF INDIVIDUAL ORGANS

1. The tumor of inferior left lobe bronchial tube was found at the autopsy in the 45 years old man's corpse of the lungs, which mainly had an endobronchial growth in bronchial wall with a growing into the surrounding pulmonary tissue. The tumor was pale-grey on the section. In different organs: in the brain, in the parietal pleura, in the liver, in peritoneum, in bones there were plural tumourous nodes mainly of the round form and different sizes. Under the microscopic investigation of all tumourous nodes some small undifferentiated cells of glandular epitelium were found.

1. Define name of the tumor of the dead man.
2. What ways of spreeding of this tumour do you know and where are localised by early and late metastases?
3. Can the tumor render a general influence on an organism?

2. Radial course of treatment was carried out for the patient with the uterine neck cancer before the operation. Then the extirpation of uterus with appendages and the removal of lymphatic nodes of pelvic fatty tissue were performed (the Vergerman's operation).

1. Explain is it necessary to remove lymphatic nodes and fatty tissue of the minor pelvis together with uterus?
2. Enumerate the frequentest ways of metastatic spreading of the uterine neck cancer.
3. Enumerate lymphatic nodes in which are the frequentestly located metastases of this cancer.
4. Enumerate the frequentest histological type of the uterine neck cancer which are met on the uterine neck.
5. What do you know about the progress in the treatment in case of uterine neck cancer.

3. To the pathologic laboratory an uterus with appendages was delivered from an operative room. On the mucous membrane of the uterine corpus there was the tumourous formation on the wide base, with a hilly surface which was considerably infiltrated into the lumen. Under the histological investigation of endometrium the growth of atypical cells of glandular epitelium was found which was compounded into glandular-like structures divided by layers of connective tissue. There was no an invasive growth in the myometrium.

1. What type of tumor growth as regard to a lumen of an organ?
2. What histological type of cancer was found at histological investigation?
3. What prognosis may be for the patient? What information can you get from the description of this case history?
4. What pretumourous processes of endometrium do usually precedeof development of endometrial cancer?
5. What age is the most probable for the development of cancer?

4. The mammary gland with a fatty tissue from an axillary area was delivered to the histological laboratory. On carring out parallel sections in the brest the thick formation, of 3cm in diameter, light-grey color with rough contours was found. Under the histological investigation an infiltrative ductal carcimoma was found.

1. What morphological form of the mammary gland tumor was found during the section of the operative material?
2. Why was the axillary fatty cellular tissue removed during the operation?
3. Point out the locations of the mammary gland tumour lymphogenic metastases.
4. Where are the most frequent locations of hematogenetic mammary gland tumor metastasis?
5. Name the histological forms of mammary gland tumor.
6. How can you estimate the prognosis of mammary gland cancer and what are the successes of cancer treatment nowadays?

5. The malignant tumor of the lung was detected in the patient which is suffering from chronic

bronchitis for a long time. For the diagnostic verification bronchoscopy was made. The exophytic formation was observed in the right lobar bronchial lumen. Histologically the squamous cell carcinoma was detected.

1. What anatomical (macroscopic) and histological variants of pulmonary carcinoma do you know?
2. What precancer process did the patient have?
3. What predisposing factors of pulmonary carcinoma development can you call?
4. What pathways of pulmonary carcinoma metastatic spreading do you know?
5. Where are localized early and late metastases?

6. The removed stomach has been brought to the pathologic laboratoty due to it's tumor suggestion. The gastric wall of pyloric and fundal portions is thick, pligas are big and deformed. Dense, whitish tissue there is at incision. The diagnosis of cancer has been made.

1. What is the name of this carcinoma macroscopic form?
2. What histological picture does characterize this type of gastric carcinoma usually?
3. What are localizations of gastric carcinoma metasteses?
4. What precancer diseases and processes of stomach do you know?
5. What histological variants of gastric carcinoma do you know?

LEUKAEMIAS. LYMPHOMAS

1. The patient of 16 years old has been admitted to the hematological clinic with the diagnosis of acute myeloid leukaemia.

1. What macro- and microscopic changes are there in the organism in acute leukamias?
2. What clinic-morphological differences are there between acute and chronic leukaemias?

2. The patient of 19 years old came to the clinic with the complaints of acute weakness, fever and skin hemorrhages. A high amount of the blastic cellular forms was found in the blood analysis and there was myelogramm. The patient died soon.

1. What type of leukaemia was there according to the cellular differentiation in this case?
2. What clinic-morphological changes were there in the organism in leukaemias?
3. What complications and causes of death are possible in leukaemias?

3. The corpse of the patient who died from chronic myeloid leukaemia with blastic crisis has been brought for the dissection.

1. What clinic-morphological signs of chronic myeloid leukaemias do you know?
2. What does blastic crisis mean?
3. What morphological changes were there in the patient's organism in blastic crisis?

4. The patient of 52 years old has come to the clinic for the treatment. There are 135000 intermediate lymphoid cells in his blood analysis. The main clinical symptoms are weakness and fever.

1. What form of leukaemia does the patient have?
2. What morphological changes are there in this leukaemia type?
3. What is the prognosis in this case?

4. What is the most often cause of death in such patients?

5. The patient of 20 years old has come to the clinic with the complaints of lymphatic nodes of neck increasing, weakness, fever and emaciation. There are no changes in peripheral blood and bone marrow. The diagnosis of Hodgkin's disease has been made at the biopsy of the jugular lymphatic node.

1. What is the definition of Hodgkin's disease?
2. What histological variants of the disease do you know?
3. Call please favourable and unfavourable variants.
4. What morphological changes are there in lymphatic nodes in these variants?

6. The corpse of the young man who died from generalized form of Hodgkin's disease has been brought for the dissection.

1. What changes are there in lymphatic nodes in Hodgkin's disease?
2. What groups of lymphatic nodes are usually affected?
3. What changes are there in the spleen?
4. Call please all histological variants of Hodgkin's disease.
5. What of them have unfavourable prognosis?

ATHEROSCLEROSIS, PRIMARY ESSENTIAL HYPERTENSION, ISHEMIC DISEASE OF THE HEART

1. The corpse of the patient, for a long time suffered from arterial hypertension, has been brought for the dissection. The hyalinosis of arterioles of kidneys, brain and other organs was found at histological investigation.

1. How does a blood vessel with hyalinosis look like?
2. What kind of blood vessels is hyalinosis developed in?
3. What types of the vascular hyalinosis do you know?
4. Please, describe their structure and call the diseases when every type of hyalinosis develops.

2. The corpse of the patient with the diagnosis of the acute ischemic heart disease and acute heart failure has been brought for the autopsy. The diagnosis has been confirmed at the dissection.

1. What morphological changes of the heart (macro- and microscopic) can confirm the diagnosis of the acute ischemic heart disease?

3. The patient with the chronic ischemic heart disease came to the hospital with the severe heart insufficiency. He died despite of the treatment. The pathologist found the chronic heart aneurysm at the dissection.

1. What is a chronic heart aneurysm?
2. What does it,s wall form?
3. What mortal complications do chronic heart aneurysm accompany ?
4. What other morphological changes (except aneurysm) are there in the heart in chronic ischemic heart disease?
5. What morphological signs of chronic heart insufficiency do you know?

4. The man with the obesity, arterial hypertension and smoking about 2 boxes of cigarettes a day during 10 years has come to the hospital with the attacks of severe retrosternal pain. The large focal myocardial infarction of the left ventricle was diagnosed. The death occured on the third day.

1. Please, describe gross changes that had been observed in the heart at the dissection.
2. Please, describe microscopic changes in the heart in this case.
3. What changes could have been found in the coronary arteries?
4. What risk factors did the patient have for development of myocardial infarction?

5. The patient who is suffering from arterial hypertension for a long time has come to the neurological clinic with the complaints of loss memory, disturbances of muscular coordination and gait disorders.

1. What diagnosis has been made according to the clinical symptoms?
2. What morphological changes are possble there in the brain in case of arterial hypertension during a long period of time?

6. The diagnosis of the mesenterial arteries atherosclerosis with the complication and peritonitis development was made at the dissection.

1. How did mesenterial arteries look like at visual inspection?
2. What possible complications are there in the mesenterial arteries caused by atherosclerosis?
3. What complications can be developed by peritonitis?
4. What clinic-morphological forms of atherosclerosis do you know?

7. The patient of 70 years old with the severe atherosclerosis of the cerebral arteries lost his consciousness suddenly and died in a day. The pathologist deturmined the cerebral infarction at the dissection.

1. What morphological type of infarction is developed most often in the brain in case of atherosclerosis?
2. What are possible outcomes of this infarction?
3. What can you observe in the patient's cerebral blood vessels in this case?
4. What cerebral blood vessels can be damaged in case of the cerebral infarction?

8. The corpse of the patient with the symptoms of chronic heart failure due to chronic ischemic heart disease has been brought for the dissection.

1. What changes in the heart can confirm the diagnosis of chronic ischemic heart disease?
2. What extra heart changes can confirm the chronic heart failure?

9. The man of 45 years old has come to the reception ward of the hospital with the complaints of acute retrosternal pain which has radiated to the left shoulder-blade and left arm. At ECG investigation the myocardial infarction of the anterior wall of the left ventricle has been diagnosed. The patient died after 6 hours from the beginning of the pain attack. The red thrombus in a branch of a coronary artery has been found by pathologist at the dissection.

1. What stage of the myocardial infarction did the patient die in?
2. What morphological changes were there in the myocardium in this stage?
3. What additional methods of microscopy can you use for the myocardial

infarction confirming?
4. What were the probable causes of death of the patient?

10. The corpse of the patient with the diagnosis of the cardiac form of arterial hypertension has been brought for the autopsy. It's known that the patient suffered from chronic coronary insufficiency.

1. What morphological changes could have been observed in the patient's organism?

11. The woman of 75 years old who is suffering from atherosclerosis for a long time, has a progressive loosening of memory, doesn't recognize relatives, her behaviour becomes non-adequate and the communication with her is difficult.

1. What pathological process does take place in the patient's brain and cerebral blood vessels?
2. What changes of brain tissue do develop due to this disturbance of cerebral blood vessels?
3. What is the name of this clinical condition?

RHEUMATIC FEVER. CEREBROVASCULAR DISEASES

1. The child has been hospitalized at the pediatric hospital with the diagnosis of rheumatic fever. He complained of pain in small joints, which were swollen, the painful nodes appeared in surrounding tissues.

1. What changes were there in joints and surrounding tissues in rheumatic fever?
2. Describe please the dynamic of morphological changes.

2. The combined mitral valvular disease were formed in the patient after rheumatic fever attack. The chronic heart insufficiency appeared in some years. Despite of the persistent treatment the patient died.

1. What changes of chronic heart insufficiency were confirmed at the dissection?
2. Describe please all possible morphological changes of the heart and other organs.

3. The woman of 50 years old had a long time history of rheumatic fever with heart failure. She died from chronic heart insufficiency. The combined mitral valvular disease was found at the dissection.

1. Describe, please, morphological changes of the valve's casps which were observed by pathologist at visual inspection and histological investigation.
2. What morphological changes must have been found in the lesser circulation?
3. What morphological changes were found in the organs and tissues of the greater circulation at the dissection?
4. What does it mean "combined mitral valvular disease"?

4. The patient with the rheumatic combined mitral valvular disease which is complicated by the circulatory decompensation is in a ward of the therapeutic department.

1. What changes can be developed in the lungs of the patient and why?
2. Describe please possible structural changes in the lungs and liver in this patient.

PULMONARY DISEASES: ACUTE PNEUMONIAS. GRIPPE

1. The patient with a heavy form of the grippe (influenza) was admitted to the infectious hospital. In some time unpleasant feelings appeared in heart, the heart sounds were muffled. The diagnosis of myocardiodistrophy was made.

1. What morphological changes were in the heart in picture of myocardiodystrophy?
2. What was their cause?
3. What was the morphogenetic mechanism of this dystrophy development?

2. The corpse of patient who died from lobar pneumonia was brought to the pathoanatomy department. The diagnosis of lobar pneumonia was confirmed by autopsy: there was fibrinous inflammation in the lungs and pleura.

1. How does the lung and pleura look like at visual inspection and microscopic investigation?
2. What pulmonary complications of lobar pneumonia do you know?

3. The patient had suffered from arterial hypertension. During hypertonic crisis the brain hemorrhage had been developed. The patient lived for 7 days without consciousness. At the autopsy besides the brain damage the foci of pneumonia were observed in the lower parts of both lungs.

1. What clinico-morphological type of pneumonia was there in this case?
2. Describe the lung at visual inspection?
3. What predisposing factors of pneumonia development were there in this case?
4. If the patient did not die, what processes would be in the lung in recovery?

4. Patient G. of 37 years old had fallen ill with a heavy form of pneumococcal pneumonia and died from heart insufficiency. At dissection the enlarged, dense, whitish-grey, unaired left lower lobe of the lung was observed. There were also pleura changes.

1. What clinico-morphological form of acute pneumonia was there in this case?
2. What stages of development does this pneumonia have and how long does every stage take for its development?
3. Describe, please, the lung is gross appearance at visual inspection and the histological features in every stage.
4. What pathological process was developed in the pleura? Call, please, its macro- and microscopical characteristics.
5. What are possible causes of death in this form of pneumonia?

5. The child died in the children's department from confluent bacterial bronchopneumonia. The etiology defined two suggestions: either staphylococcal or streptococcal pneumonia.

1. What are the distinguishing structural criteria of these two types of pneumonia?
2. What can you say about streptococcal etiology in this case and what is about staphylococcal one?
3. What possible complications may be in these pneumonias?

6. The corpse of the 34 years old patient has been brought for the dissection with the diagnosis of lobar pneumonia which has been complicated by heart insufficiency. At autopsy some morphological changes confirming the diagnosis were found. There was the total damage of the left lung in stage of grey hepatization.

1. Describe, please, macro- and microscopic picture of grey hepatization stage.
2. What other stages of lobar pneumonia do you know?
3. What extra pulmonary changes can be developed in case of lobar pneumonia?
4. What is the etiology of lobar pneumonia?

7. The patient of 60 years old was operated on for gastric carcinoma. After the operation he stayed in heavy condition and pneumonia was developed. The patient died from cardiac-pulmonary failure.

1. What clinico-morphological form of acute pneumonia was there in this case?
2. What are the main morphological features of this pneumonia?

3. What predisposing factors of the pneumonia development were there in this case?
4. How can you avoid the pneumonia development in postoperative period?
5. Is pneumonia the basic disease or complication in this case?

8. The symptoms of cardiac insufficiency have appeared in the patient with bilateral bronchopneumonia: heart sounds are muffled, there are arrhythmia and changes of ECG.

1. What is bronchopneumonia?
2. Give, please, its morphological characteristics (macro- and microscopical).
3. What morphological changes in the heart did cause its functional disorders?
4. Why were they developed?

9. The patient was admitted to the hospital during the epidemic period of the grippe with the complaints of chills, aching joints, coughing, headache, pronounced dyspnea, and temperature risening up to 40°C. At auscultation the multiply moist rales were listened to. Despite of the treatment the patient died in 12 days due to severe pneumonia.

1. What clinico-morphological form of the grippe was there which caused death?
2. What morphological changes were there in the bronchi and lungs?
3. How did the lungs look like at visual inspection?
4. What is the figurative name of this lung?

10. During "American Legion" congress in 1976 the 48 years old man was complaining of rising temperature up to 38,7°C and pain in the chest in coughing. There was no lung pathology. The temperature retuned to normal after antibiotics treatment. The rate of immunofluorescent Legionellas antibodies was examined in 6 months. It was positive.

1. What clinical form of legionellosis was there in the patient?
2. Call, please, the cause of the disease. Is the disease pass from person to person, what are the ways of spreading?
3. Why was the congress of the "American Legion" mentioned in this case?
4. Why does it take place in the history of this disease researching?
5. What changes may occur in lungs if the process becomes severe?

PULMONARY DISEASES: CHRONIC DISEASES OF LUNGS

1. In the patient who was suffering from chronic bronchitis, tumor of lung was found. To confirm the diagnosis the bronchoscopy was made. At this examination the formation was observed which was attached to bronchial wall. At histological examination the patient was found to have carcinoma from bronchial epithelium.

1. What macro- and microscopical variants of lung carcinoma do you know?
2. Where does lung carcinoma spread?
3. What peculiarities of metastatic spreading of this tumor do you know?

2. The 47 years old patient has come to the surgery department with a high temperature and moist cough. On X-ray examination the cavity with the fluid was observed in the right lung. The operation of lobectomy was performed. At investigation of the operative material the pathoanatomist has found the cavity of 7 sm. in diameter which has contained a yellowish-green, bad smelled fluid. The wall of the cavity is thick, whitish-grey and dense.

1. What pathological process was there in the lung?
2. What could has been preceded of this pathological formation in the lung?
3. What morphological changes determined the chronic course of this process?
4. What do you think, why was the operation indicated for the patient?

5. What complications of this disease have been avoided by operation?

DISEASES OF STOMACH AND INTESTINE

1. The resectied stomach with a sticking out carcinoma in the lumen of the small curvature has been brought to the pathoanatomical laboratory. Gastric adenocarcinoma has been diagnosed at the histological investigation.

1. What is the name of the tumor growth in the lumen of the organ?
2. Give please examples of well- and poorly-differentiated adenogenic carcinomas.
3. What is adenocarcinoma?
4. What are the localizations of gastric carcinoma's metastases and what are the ways of its spreading?

2. The removed stomach has been brought to the pathoanatomical laboratory due to its tumor suggestion. The stomach wall of pyloric and fundal portions is thick, plicas are big and deformed. There is dense, whitish tissue at incision. The diagnosis of cancer has been made.

1. What is the name of this macroscopic form of carcinoma?
2. What histological picture does this type of gastric carcinoma usually characterize?
3. What are the sides of localizations of gastric carcinoma,s metastases?

3. A severe form of epithelial displasia was found in gastric mucosa at gastroscopy in the patient with a long time history of stomach functional disorders.

1. What morphological changes did appropriate to this diagnosis?
2. Why is displasia dangerous?
3. What morphological changes may be there in different forms of displasia (mild and average, except severe) and what must the doctor do in every certain case?

4. The 5 years old child has eaten much different irritated food, at the New Year,s table. In some hours he began to complain of abdominal pain, nausea and vomiting. The temperature was normal. After some time the self-beeling became better, except anorexia only. The diagnosis of gastritis was made.

1. What form of gastritis was there in this case according to the clinical course and morphology?
2. What morphological changes of gastric mucosa (at visual inspection and histological investigation) are there in this form of gastritis?
3. What is the usual outcome of this gastritis?
4. What other morphological forms of acute gastritis do you know? Describe please their morphological appearances.

5. The patient with the acute abdominal pain has been urgently transferred to the surgical clinic. The pain has appeared suddenly ("knife-like pain"). The patient is known to have had a long time history of peptic ulcer.

1. Describe please peptic ulcer`s morphology (macro- and microscopic changes) in all phases of the disease.
2. What complication could have been suspected in this case?
3. What was necessary to do and why?
4. What groups of peptic ulcer complications do you know?

6. The gastrobiopsy is often taken from the chronic gastritis patients for the diagnosis confirming.

1. What classifications of chronic gastritis do you know?
2. What structural changes could have been observed by biopsy in gastric mucosa material with different morphological types of chronic gastritis?

7. The pain in epigastrium and vomiting appeared suddenly in the woman. She came to the doctor two days after the onset. The temperature was 38°C. The woman was admitted to the hospital and was operated on. During the operation thick appendix was found, its serous membrane was hyperemic covered by fibrinouse and purulent stains.

1. What form of acute appendicitis was found in the patient?
2. What histological changes of the appendix did corresponded to this form?
3. What complications could be combined with this form of acute appendicitis?

8. The patient of 50 years old, has a long time history of chronic gastritis with the typical symptoms characterizing the disease. He came to the doctor complaning of increased pain in the abdomen (stomach). The gastroscopy with biopsy was performed. The histological diagnosis was chronic atrophic gastritis.

1. On the base of what histological changes was this diagnosis made?
2.Why was gastroscopy indicated with biopsy for the patient?
3. Why is chronic gastritis dangerous?
4. What morphological forms of chronic gastritis do you know?
5. What etiological factors take place in the chronic gastritis development?

LIVER DISEASES

1. The patient with the symptoms of hepatic failure is in the hospital. His skin is yellow.

1. What is the name of pathological condition when the skin becomes yellow?
2. What other tissue changes may have the patient?
3. Due to accumulation of what pigment patient's tissues become yellow?
4. What is the nature of this pigment?
5. Give please examples of clinical pictures and pathological conditions when this pigment can be accumulated in blood?

2. The acute hepatic insufficiency has been developed in the patient after mushrooms poisoning. The patient has had hepatic coma clinically. A sharp decrease of liver sizes has occurred.

1. What disease has been developed in the patient?
2. What morphological stages does it have?
3. What morphological changes of the liver are there in every stage of the disease (macro- and microscopic)?
4. What possible outcomes may this disease have?

3. The corpse of 54 years old woman has been brought for the dissection. There was cholelithiasis in her anamnesis. At autopsy the jaundice, decreased liver with dense consistency, small nodular surface and green color have been marked.

1. What disease was detected at dissection?
2. What morphogenetic and morphological forms of the disease were there in this case?
3. What morphological changes must be found in hepatocytes, stroma, bile capillaries and topography of blood vessels in the liver?
4. What was the cause of death in this case?

4. The patient has a long time history of chronic alcoholism (a hepatic form of disease). At the last hospitalization the liver was found with small nodular surface at laparoscopy in this patient. There were distended veins of the anterior abdominal wall ("caput Medusae").

1. What pathological process was developed in the liver?
2. Name please morphogenetic and morphological types of the process in this case.
3. What possible microscopic changes were there in the liver?
4. Why were the veins of the anterior abdominal wall distended in the patient?
5. What are the possible causes of death in this disease?

5. The 43 year old patient who was the surgeon felt ill with a viral hepatitis after the injury of the hand during the operation. He was treated for several years, but hepatitis became chronic. The severe morphological picture of chronic hepatitis was observed at puncture biopsy.

1. Why does acute hepatitis become in chronic in some cases?
2. What clinic-morphological forms of chronic hepatitis do you know?
3. Compare please these forms according to the morphological and clinical parameters?
4. What form of chronic hepatitis was there in the patient?

6. The corpse of the patient who died from liver cirrhosis has been brought for dissection. Portal liver cirrhosis has been observed.

1. What other morphogenetic types of liver cirrhosis do you know?
2. Make please comparison of all these liver cirrhosis types according to the etiology, taking into acount clinical symptoms and macro- , microscopic changes.

7. Patient M. has a severe viral hepatitis in his anamnesis. The diagnosis is liver cirrhosis at present time. At laparoscopy the liver is decreased, its surface is greatly nodular (the diameter of the nodes is up to 5 sm.) and thick connective tissue septus are observed.

1. What type of liver cirrhosis is there according to morphogenesis and morphology?
2. What morphological peculiarities has this liver cirrhosis type?
3. What do you think, in outcome of what form of viral hepatitis in this liver may be cirrhosis developed?
4. How is the decompensation of this liver cirrhosis clinically realized?

8. At dissection of the dead patient of 50 years old, the decreased, dense, small nodular liver was found and there were varicose dilation of esophageal veins and veins of the cardiac portion of the stomach. In the stomach cavity there were 900 ml. of liquid blood and blood clots, in peritoneal cavity – 700 ml. of edematous fluid.

1. What disease of the liver was there in the patient?
2. Describe, please, the morphological changes of the liver.
3. What was the cause of death in this patient?
4. Explain please why was blood found in the stomach and was there edematous fluid in the peritoneal cavity?
5. Give, please, examples of diseases which may cause liver cirrhosis.

9. The young woman has been urgently brought to the hospital with poisoning symptoms. Despite of the treatment the patient's condition was worse and the death was due to hepatic failure. An acute decrease of the liver, with flabby consistency and yellow colour was found at autopsy.

1. What disease had the patient?
2. What stages of this disease do you know?
3. In what stage did the death occur in this case?
4. Describe please the microscopic picture of the liver in this stage.
5. What other outcome of the disease is possible?
6. What possible changes were there in the patient's kidneys?

10. What different clinical forms of the viral hepatitis are known including the form with jaundice and without jaundice.

1. What other clinico-morphological forms of acute hepatitis do you know?
2. What morphological changes are there in liver in hepatitis with jaundice and without jaundice?
3. Why is jaundice developed and what is its type?

11. The patient has been admitted to the infectious hospital with the suggestion of epidemic hepatitis. The diagnosis has been confirmed by laboratory analysis.

1. What is the etiology of this hepatitis?
2. What morphological changes are there in the liver in this hepatitis?
3. What is the most frequent outcome of the disease?
4. Name please all possible outcomes of viral hepatitis.
5. What is the most severe form of viral hepatitis and why is it so?

KIDNEY'S DISEASES

1. The stomatological patient has a long time history of chronic osteomyelitis of the upper jaw. Despite of treatment, the facial edemas, protein in urine and other symptoms of kidney functional disorders appeared in some time.

1. What complication were developed in the patient?
2. Why are kidney changes possible in this patient?

2. The corpse of the patient who was suffering from secondary amyloidosis for a long time and died from combined insufficiency of kidneys, liver and adrenals glands, has been brought for dissection. The pathoanatomist made the diagnosis of secondary amyloidosis, of parenchymatous variant.

1. What is the secondary amyloidosis?
2. What types of amyloidosis according to the etiology do you know?
3. Where does amyloid deposit in parenchymatous type?
4. Why were there kidney, liver and adrenals insufficiencies in this case?

3. Some changes suggesting the kidney amyloidosis have been found at dissection.

1. How do kidneys look like at visual inspection in amyloidosis?
2. What test can you make for amyloid recognizing at dissection?
3. What histological stains can you use for the presence of amyloid confirmation?

4. The patient with the diagnosis of acute glomerulonephritis was in the nephrological clinic. Despite of the treatment the patient died from renal failure sooner. The autopsy confirmed the diagnosis. There were severe changes in glomeruli and ducts.

1. What changes were there in glomeruli in acute glomerulonephritis?
2. What structural changes were there in tubules and in blood vessels?
3. What certain morphological changes do the most severe clinical manifestations have?

5. The patient with the renal and extra renal symptoms of glomerulonephritis has been admitted to the hospital.

1. Name please all renal and extrarenal symptoms of glomerulonephritis which you know and their pathogenesis.

6. The young man has come to the polyclinic with the complaints of facial oedemas. At laboratory examination the protein and hyaline casts were found in urine. The patient was hospitalized with the diagnosis of glomerulonephritis with nephrotic syndrome.

1. What is the nephrotic syndrome?
2. Why is it developed (its causes and pathogenesis)?
3. What changes were there in the tubular epithelium and what was the mechanism of their development?

7. Patient P. with the wide-spreading burns has been brought to the traumatological clinic in shock. In several hours he died. The changes of the kidneys were found at dissection. The kidneys were increased, flabby, capsule was taken off without any difficulties, the cortex was pale, and pyramids were dark red at incision.

1. What pathological process was there in the kidney?
2. What was its stage?
3. Name please other stages of this pathology.
4. What probable histological changes of the kidneys were there in this case?
5. Why kidney's pathology was developed in all types of shock?

8. The patient with the manifestations of chronic renal insufficiency due to chronic glomerulonephritis had been hospitalized many times. The severe hypertension was occurred at the last time. The death was sudden due to acute cerebral circulation impairment. The changes confirming the chronic glomerulonephritis were found at autopsy and there was also brain hemorrhage in brain lateral ventricles.

1. What is the name of the kidney in chronic glomerulonephritis accompanied by chronic renal insufficiency?
2. How does this kidney look like at visual inspection?
3. Why was cerebral effusion developed?
4. What changes were there in the blood vessels?

INTESTINAL INFECTIONS. SEPSIS

1. The temperature has risen up to 41°C, the acute edema of the oral mucosa and neck tissues, cloudy consciousness, multiplied skin petechial hemorrhages have appeared in the patient of 21 years old in 3 days after teeth extraction. The death has come on the 2-d day after the symptoms appearance.

1. What clinic-morphological form of sepsis was there in this case?
2. What was type of the sepsis according to the gates of infection?

3. What local morphological changes were there in the patient?
4. What morphological changes were there in the stroma and parenchyma of the parenchymatous organs, in the hemopoetic organs and in the lymphoid tissue?

2. The woman of 44 years old, has come to the hospital with the odontogenic phlegmone of the mouth cavity and submandibullar region. Despite of the active surgical and antibiotic treatment the sepsis has been developed and the death occured due to this. The multiple abscesses of the lungs, liver, kidneys and the septic spleen were found at the dissection.

1. What clinic-morphological form of the sepsis was there in this case?
2. What primary septic focus was there in this case?
3. What morphological changes could the the pathoanatomist observed in the the septic spleen?
4. What are the principles of sepsis classifications?

3. The patient with the symptoms of the heart damage (pain, arrhythmias, weakness of the heart sounds and others) has come to the reception ward of the hospital. The diagnosis of the myocardial infarction was not confirmed by ECG and the new diagnosis of the septic endocarditis was made.

1. What possible morphological changes are there in the organism in septic endocarditis?
2. What are the modern views of etiology and pathogenesis of this disease?

4. In the 46 year old patient of the suffering from rheumatic heart valvular disease for a long time the fever, jaundice, edemas, petechial skin hemorrhages and shortness of breath at rest appeared. On the conjunctiva of both eyeballs there was petechial rash. The patient died. The diagnosis of the septic endocarditis has been confirmed at the dissection.

1. What clinic-morphological form of sepsis was developed there in the patient?
2. By what objects have been valvular casps damaged in the sepsis?
3. Compare, please, morphological changes of the valvular casps which developed in sepsis and rheumatic fever.
4. What complications were possible there in the patient if he would survive?

5. The symptoms of “acute” abdomen (acute abdominal condition) have appeared in the patient with typhoid fever on the third week of the disease and peritonitis has been diagnosed. Despite of the performed treatment the patient died.

1. Describe please the genesis and dynamic of morphological changes of the intestine with typhoid fever.
2. Why has peritonitis been developed?
3. Name please other possible local complications in typhoid fever.

6. The young woman has been hospitalized at the Emergency Department with the complaints of headache, high temperature, weakness and apathy. At the external examination the following changes have been found: the skin was clear, the abdomen was slightly painful and in the lungs the vesicular respiration was heard. The diagnosis was uncertain. Approximately on the 10-th day the roseolous popular rash on the abdomen,s skin and the disorders of the intestine appeared. The patient was directed to the infectious department with the diagnosis of typhoid fever.

1. What stages of typhoid fever do you know?
2. What changes were developed in the intestine according to the stages?
3. Why does rash appear?
4. What possible complications are there out of intestine in typhoid fever?

7. The fibrinous colitis has been found in the patient with schigella dysentery at the rectoscopy. The diagnosis of the infection has been confirmed by bacteriologic tests.

1. Describe please macro- and microscopic appearances of fibrinous colitis.
2. What stages of schigella dysentery are there in its development?
3. What changes are there at the onset at the beginning and at the end of schigella dysentery in the sigmoid intestine and rectum?

8. The rectoscopy was made in the patient with a severe form of classic schigella dysentery on the 12-th day. Some morphological changes were found.

1. What was the stage of the disease in this case?
2. What following changes were there in the intestine?
3. What regime must has been prescribed for the patient and why?

9. The woman of 32 years old was admitted to the oncology dispensary with the complaints of weakness, emaciation and liquid stool. Multiple, dense conglomerates of nodes were found in the abdomen at palpation. The diagnosis of malignant tumor of the abdominal cavity was made. Than the bilateral confluent bronchopneumonia developed and the patient died. At the dissection there was no tumor, but the generalized form of yersiniosis was found.

1. What is yersiniosis? What is the way of the infection?
Who is the origine of the infection?
2. What clinico-morphological forms of yersiniosis do you know?
3. What morphological specialties are there in the organism in yersiniosis?

10. The young military man, came to the hospital with the severe form of yersiniosis, with the damage of the intestine, mesenterial lymphatic nodes and with the symptoms of peritonitis. He died in several days due to generalization of the infection.

1. What morphological changes were there in the intestine and lymphatic nodes in case of yersiniosis?
2. What was the form of yersiniosis on the beginning of the disease and at its end in this case?

11. The patient was admitted to the hospital with the symptoms of a severe gastroenteritis and with the complaints of profuse diarrhea and vomiting. At the examination manifestations of dehydratation were observed. The cholera vibrio was found at the bacteriologic tests. Despite of the treatment the patient died.

1. What was the stage of cholera in this case?
2. What morphological changes were there in the stomach, intestine and soft tissues?
3. What are possible causes of death in cholera?

12. The man of 30 years old arrived from India. The profuse diarrhea and vomiting appeared suddenly. The temperature was high. Cholera was confirmed by bacteriological tests. The clinical

manifestations of the disease changed in some time: the skin became dry and wrinkled, the temperature decreased and the patient died due to hypovolumic coma.

1. What the stage of cholera was there in the patient?
2. Describe please morphological changes of his inner organs.
3. Name please specific and nonspecific complications of cholera.

TUBERCULOSIS

Clinical cases

1. The primary tuberculous complex (Ghon complex) has been developed in the infant of 4 years old. The diagnosis of the primary tuberculosis has been made and the treatment was administered. However, the lymphogenic spreading of the infection has appeared and tuberculous bronchoadenitis has been developed.

1. Describe please the morphology of the primary tuberculous complex.
2. What morphological changes does the tuberculous bronchoadenitis characterize?
3. What pathways of primary tuberculous spreading do you know?

2. The corpse of the boy of 6 years old who died from hematogenic spreading of the primary tuberculosis was brought for the dissection. The autopsy confirmed the presence of primary tuberculous complex and signs of primary tuberculosis spreading, specifically miliary tuberculosis of the lungs.

1. What morphological changes did confirm the presence of primary tuberculous complex and miliary tuberculosis of the lungs?

3. It is important to know the main groups of secondary tuberculosis complications fo avoiding its development.

1. What groups of secondary tuberculosis complications do you know?
2. When and why can hemorrhages be developed? What is the mechanism of their development?
3. Name please other certain examples of secondary tuberculosis complications of every group.

4. The patient of 62 years old has the cavitary fibrocaseous form of tuberculosis with the cavern in the upper lobe of the right lung. The patient has been treated for many years. Not long ago he was admitted to the hospital with the decompensation of chronic heart insufficiency.

1. What is the way of the cavern development in tuberculosis?
2. What is the morphology of cavitary fibrocaseous tuberculosis?
3. Why is heart insufficiency possble in case of secondary tuberculosis?
4. Describe, please, its pathogenesis.

5. The rounded shape of shadow was found in the upper lobe of lung in the woman of 45 years old at the photoroentgenography during dispensary observation. On following examination the lung tuberculoma was found. The woman was operated on. The histological investigation confirmed the diagnosis.

1. What is tuberculoma? What is its morphology? In what clinico-morphological form of tuberculosis is tuberculoma developed?
2. How can tuberculoma be developed (describe the mechanism of its formation)?

6. At the dissection of two men died from phthisiatric dispensary the acute focal tuberculosis was found in one and there was the fibrocaseous tuberculosis in the second. Compare, please, two tuberculosis forms according to the morphology.

7. The man of 40 years old has come to the polyclinic with the complaints of weakness, fever, night sweating. The acute focal tuberculosis has been found on examination.

1. What macro- and microscopic characteristics does acute focal tuberculosis have?
2. What is the Abricosov focus and what is its structure?

8. The tuberculous spondylitis was developed in the child of 12 years old. To avoid the deformation of vertebra column the plaster bandage was applied and bed regime was prescribed.

1. In what clinico-morphological form of tuberculosis does tuberculous spondylitis occur?
2. What is the dynamic of morphological changes in tuberculous spondylitis course?
3. Why is plaster bandage necessary?
4. What changes are possible in tuberculous foci in recovery?

9. A severe form of tuberculosis of the kidney was found in the young man. The patient was operated on and the kidney was removed.

1. What macro- and microscopic changes of the kidney were there in the patient?
2. Why do clinical manifestations of the kidney tuberculosis appear later than the morphological changes?
3. What structures can be damaged after in intracanalicular spreading of tuberculous infection in the kidney?

INFECTIONS OF CHILDHOOD. AIDS

1. The man of 57 years old, homosexual, has been hospitalized in a grave condition with the complaints of fever and increasing of all lymphatic nodes. Also the splenomegaly, infectious injuries of gastro-intestinal tract and skin tumor were found on examination. The diagnosis was AIDS.

1. What are the stages in the AIDS clinical course?
2. What stage was there in this case?
3. What changes were there in the lymphatic nodes?
4. What tumors are usually developed in AIDS?

2. A young man, the father of two children, became ill with AIDS after the blood transfusion. The disease had a rapidly-progressive clinical course; fever, diarrhea and emaciation were developed. It was the gastrointestinal form of AIDS.

1. Why were infectious injuries in AIDS called opportunistic?
2. What are clinico-morphological features of infectious diseases in AIDS?
3. What other organ systems can be affected in AIDS (excepting gastro-intestinal tract)?

3. The child has been admitted to the hospital in moderately grave condition and with average intoxication. There are signs of fibrinous inflammation on the mucous membranes of larynx and trachea. The diagnosis of diphtheria has been made after additional investigations.

1. What form of exudative inflammation is possble there on larynx and trachea mucosa in case of diphtheria?
2. What clinico-morphological forms of diphtheria do you know?

4. The acute pain on swallowing and heavy oedema of neck soft tissues had appeared in 5 years old child. The temperature had risen up to 39°C, the condition has become worth. The whitish-yellow membranes, difficulty fixing, were on the tonsils and soft palate. Despite of treatment the child has died.

1. What disease was there in this case?
2. What pathological process was developed on tonsils and soft palate?
3. What is the name of this form of disease?
4. What possible changes are there in the organism in this form?

5. The child with the confirmed diagnosis of diphtheria has been brought to the infectious hospital.

1. What forms of fauces diphtheria do you know?
2. Describe, please, their morphological appearances.
3. What complications are possible in fauces diphtheria?

6. The mother of 2 years old child called in a doctor due to the temperature rising, a sore throat and rash on the body,s skin except nasolabial triangle of the face. The doctor made the diagnosis of scarlet fever.

1. What changes are there in tonsils, oral cavity and lymphatic nodes in scarlet fever?
2. What was the period of the scarlet fever in this case?
3. Why did rash appear and what is its outcome?

7. The patient of 40 years old had progressive deafness after scarlet fever in childhood which was complicated by otitis.

1. What clinical forms of scarlet fever can be complicated by otitis?
2. Why and in what period of disease does it happen?
3. What complications can be developed in the first and second periods of scarlet fever?

8. The 1,5 years old baby died from meningococcal meningitis with the oedema and swelling of the brain. The death occured on the third day after the onset of the disease.

1. Describe, please, gross and microscopic changes of the soft brain membrane in this time?
2. What following changes of the soft brain membrane are possible?

9. The 3 months old baby was in the epidemic focus of meningococcal infection. He had an acute onset of the disease, high temperature and rash. The acute adrenals failure (Waterhouse-Friderichsen syndrome) was developed soon and the patient died from bacterial shock.

1. What is the name of this form of meningococcal infection?
2. What morphological changes could have been observed at the dissection there in the patient who died from this form of disease?
3. What is the Waterhouse-Friderichsen syndrome?

10. The child had meningococcal infection but he did not recover from it. In this period there was hydrocephalus in the patient.

1. What is hydrocephalus and why was it developed in this case?
2. Describe, please, morphological changes of the brain membranes and brain in this case?

And then the tasks are specified for all competencies provided for this discipline.

5. The content of the assessment tools of mid-term assessment

Mid-term assessment is carried out in the form of a credit / differentiated credit / exam (leave the necessary).

The content of the assessment tool (questions, topics of abstracts, round tables, etc.)

If the bank of assessment tools for conducting current control and mid-term assessment of students in this discipline is presented on the Educational Portal of the PRMU, specify a link to this electronic resource.

5.1 The list of control tasks and other materials necessary for the assessment of knowledge, skills and work experience *(the teacher indicates only those tasks and other materials that are used within the framework of this discipline)*

5.1.1. The questions for the exam pathological anatomy, clinical pathological anatomy are the situational tasks specified above (competence codes UC-1, GPC-5).

6. Criterias for evaluating learning outcomes

For the exam

Learning outcomes	**Assessment of competence developed**			
	unsatisfactory	**satisfactory**	**good**	**excellent**
Completeness of knowledge	The level of knowledge is below the minimum requirements. There were bad mistakes	The minimum acceptable level of knowledge. A lot of light mistakes were made	The level of knowledge in the volume corresponding to the training program. A few light mistakes were made	The level of knowledge in the volume corresponding to the training program, without errors
Availability of skills	Basic skills are not demonstrated when solving standard tasks. There were bad mistakes	Basic skills are demonstrated. Typical problems with light mistakes have been solved. All tasks have been completed, but not in full.	All basic skills are demonstrated. All the main tasks have been solved with light mistakes. All tasks have been completed, in full, but some of them with shortcomings	All the basic skills were demonstrated, all the main tasks were solved with some minor shortcomings, all the tasks were completed in full

Learning outcomes	Assessment of competence developed			
	unsatisfactory	**satisfactory**	**good**	**excellent**
Availability of skills (possession of experience)	Basic skills are not demonstrated when solving standard tasks. There were bad mistakes	There is a minimal set of skills for solving standard tasks with some shortcomings	Basic skills in solving standard tasks with some shortcomings are demonstrated	Skills in solving non-standard tasks without mistakes and shortcomings are demonstrated
Characteristics of competence formation*	The competence is not fully formed. The available knowledge and skills are not enough to solve professional tasks. Repeated training is required	The formation of competence meets the minimum requirements. The available knowledge and abilities are generally sufficient to solve professional tasks, but additional practice is required for most practical tasks	The formation of competence generally meets the requirements, but there are shortcomings. The available knowledge, skills and motivation are generally sufficient to solve professional tasks, but additional practice is required for some professional tasks	The formation of competence fully meets the requirements. The available knowledge, skills and motivation are fully sufficient to solve complex professional tasks
The level of competence formation*	Low	Below average	Intermediate	High

For testing:

Mark "5" (Excellent) - points (100-90%)
Mark"4" (Good) - points (89-80%)
Mark "3" (Satisfactory) - points (79-70%)

Less than 70% – Unsatisfactory – Mark "2"

Developer(s): Yunusova Ekaterina Eduardovna, Candidate of Medical Sciences, Associate Professor
Full name, position, academic degree, academic title

Date "_19_" __may____ 2023__