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. Following 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) heterochromism.

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) Shigella 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 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 hepatocytes’ cytoplasm;
e) disappearance of hepatocytes’ 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 myocytes;
c) decreasing of myocytes’ sizes;
d) accumulation of lipids in cytoplasm of several cardiomyocytes only;
e) accumulation of lipids in cytoplasm of all cardiomyocytes.

52. At the microscopic investigation the myocardial fat degeneration can be defined 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 hepatocytes there is in:
   a) liver steatosis;
   b) viral hepatitis B;
   c) liver echynococcus;
   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 cardiomyocytes 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?
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;
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 branches;
   c) atrophy of hepatocytes;
   d) fat degeneration of hepatocytes;
   e) hemorrhages into centers of lobules.

126. The liver has nutmeg appearance due to:
   a) hemorrhages into centers of lobules:
   b) atrophy of hepatocytes in centers of lobules;
   c) hypertrophy of hepatocytes 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 hepatocytes;
   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 a 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 alveolar spaces;
b) accumulation of edematous fluid in alveolar 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 alveolar spaces;
c) narrowing of alveolar spaces;
d) erythrocytes diapedesis;
e) all enumerated.

171. A transudate is characterized by the following 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;
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 branches of pulmonary artery can cause:
   a) pulmonocoronary reflex;
b) lung infarction;
c) atelectasis;
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 branches results as:
a) pulmonocoronary reflex;
b) lung infarction;
c) atelectasis;
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 branches.

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) atelectasis;
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 cardiomyocytes;
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;
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 neutrophils 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 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) cardiomyocytes sizes increasing;
   b) cardiomyocytes number increasing;
   c) stromal edema;
   d) cardiomyocytes 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 falling only:
   a) decreasing of heart sizes;
   b) thickening of ventriculars walls;
   c) dilation of chambers;
   d) flabby consistency of myocardium;
   e) fat degeneration of cardiomyocytes.

339. What does develop in hypertrophic myocardium in phase of decompensation?
   a) atrophy of cardiomyocytes;
   b) hyperplasia of cardiomyocytes;
   c) degeneration of cardiomyocytes;
   d) tissue repair;
   e) hypertrophy of cardiomyocytes.

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

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 following sign:  
a) flabby consistency of myocardium;  
b) paleness of myocardium;  
c) thickening of ventricles walls;  
d) increasing of heart mass;  
e) increasing of cardiomyocytes 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) canceromatous 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 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) agenesis (absence) of organ;
   b) aplasia (staining 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;
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) infiltrative.

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. Histoid 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) nasopharyngeal angiofibroma;
   e) angiosarcoma.

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

396. Call benign mesenchymal tumour:
   a) nasopharyngeal 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;
398. What can be classified as malignant mesenchymal tumour?
   a) fibromyoma;
   b) leiomyosarcoma;
   c) nasopharyngeal 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;
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) lymphangioma;
   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) renewal 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 following 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 following 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