МИНИСТЕРСТВО ОБРАЗОВАНИЯ И НАУКИ КЫРГЫЗСКОЙ РЕСПУБЛИКИ
ОШСКИЙ ГОСУДАРСТВЕННЫЙ УНИВЕРСИТЕТ
МЕЖДУНАРОДНЫЙ МЕДИЦИНСКИЙ ФАКУЛЬТЕТ

Кафедра патология базисной и клинической фармакологии

УТВЕРЖДЕНО на заседании кафедры патологии, базисной и клин. фармакологии . Протокол №1 от 14 сентября 2023 г.

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УТВЕРЖДЕНО
Председатель УМС факультета к.э.н Базиевой А

ФОНД ТЕСТОВЫХ ЗАДАНИЙ для итогового контрольного по дисциплине

Общая патологическая анатомия
На 2023-2024 учебный год
Направление: 560001- ЛЕЧЕБНОЕ ДЕЛО (GM)

Курс: 2 Семестр: 3

Наименование дисциплины	Всего	Кредит	Аудиторные занятия (60 ч.)		СРС
			Лекции	Практические	
Клиническая фармакология	120ч.	4 кр.	24 ч.	3бч.	60 ч.
Кол-во тестовых вопросов	352				

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ЭКСПЕРТНОЕ ЗАКЛЮЧЕНИЕ БАНКА ТЕСТОВЫХ ЗАДАНИЙ

кафедры «Патология, бучной и клинической фармакологии»

от «24» ноября 2023 г.

на

разработанные тестовые задания по дисциплине

«Общая патологическая анатомия»

наименование дисциплины

Смлов А.М(проф), Умурзакова Г.М (ст.преп), Мурашева Я(преп).

/указать должность, ученую степень, Ф.И.О. автора (авторов)!

Тестовые задания проверены членом экспертной группы тестологов

[указать должность, ученую степень, Ф.И.О./

Нап авления п оведения оценки ст кт ы и соде жания тестового задания

	Нап авление экспе тизы	Оценка экспе тов	
	Соответствие задания программам и стандартам обучения	Соответств ет	Не соответств ет
2	Включение в тесты только наиболее важных, базовых знаний	Соответств ет	Не соответств ет
3	Ясность смысла тестовой ситуации и представления ТЗ	ясно	Не ясно
4	Правильность ответа на вопрос ТЗ	Соответств ет	Не соответств ет
5	Значимость содержания тестового задания (0сомнительный, 1-допустимый, 2-важный, 3с щественный)	З— балл(ов)	
6	Соответствие необходимое число заданий по каждому разделу дисциплины исходя из его важности и числа часов, отведенных на его изучение в программе.	Соответствует	соответствуе

Членом экспертной группы выявлены следующие недостатки в тестовом задании

Отмечено недостаточно ответов

Членом экспертной группы внесены следующие исправления (корректировки) в тестовое задание

На основании представления тестовых заданий автором (авторами) и проведенной проверки сделала следующее заключение:

1) Содержание тестовых заданий соответствует (не соответствует) содержанию УМКД (нужное подчеркнуть)

2) Представленные тестовые задания в следующем объеме з ^{0}6 вопросов: соответствуют (не соответствуют) требованиям, предъявляемым к количеству, уровням сложности и формам заданий для составления тестов. (нужное поДче к ть)

Тестолог Се.а-по--К© 4. С-/ 44. 23 /

подпись дата

Малушова АА 24.11.23.

подпись дата

Ознакомлен зав. кафедрой

Exam mcq's in general pathological anatomy

l. Necrosis it is death:

a) of cells due to metabolic disorders;

b) ofparenc hymatous cells only;

c) of cells and tissues in a living organism;

d) program med, genetically determined death of cells; 2. Call morphological type of necrosis:

a) vascular;

b) allergic;

c) coagulative,

d) traumatic, 3. Dry necrosis has following colour:

a) whitish-yellowish;

b) black;

c) dark-red,

d) cyanotic; 4. Show wrong characteristic ofwet 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; 5. What colour does gangrene have?

a) yellow;

b) whitish-grayish;

c) black;

d) dark-red, 6. What morphologic al type of necrosis in the myocardium does develop?

a) wet gangrene;

b) dry gangrene;

c) wet necrosis;

d) dry necrosis; 7. What is the most often localization ofcolliquativ e necrosis?

a) spleen;

b) kidney;

c) liver;

d) brain; 8. Wet gangrene usually develops in:

a) bowel;

b) kidney;

c) liver;

d) brain; 9. Show the example of wet necrosis:

a) caseous necrosis;

b) fibrinoid necrosis;

c) ischemic infarction of the spleen;

d) ischemic infarction of the brain; 10. Piece of dead tissue without any changes is calling:

a) petrifacate;

b) bedsore;

c) infarction;

d) sequester; I l . Complication of necrosis is:

a) resorption;

b) organization;

c) encapsulation;

d) rupture of cavitary organ wall; 12. Inflammatory reaction accompanies:

a) necrosis;

b) apoptosis;

c) proliferation;

d) cytoplasm vacuolization; 13. Apoptosis it is:

a) death ofcells in a living organism; 'b) controlled process of cellular self-destruction;

c) death oftissues in a dead organism;

d) death of parenchymatous cells.

14. Usually apoptosis takes:

a) single cells;

b) foci oforgan's parenchyma;

c) a part of organ;

d) whole organ. 15. At the light microscopy apoptosis bodies look like.

a) basophilic, round small bodies,

b) eosinophilic, round small bodies;

c) vacuoles;

d) crystals; 16. What does happen with the chromatin in apoptosis?

a) lyses;

b) dispersion;
c) condensation;
d) geterochromism. 17. The component ofthe apoptosis bodies is:
a) nuclei with nucleoli;
b) vacuoles filled with lipids inside;
c) giant mitochondria;
d) tightly packed cellular organelles ;18. What is the outcome of apoptosis?
a) phagocitosis;
b) organization;
c) encapsulation;
d) tissue repair; 19. Genetically programmed cellular death is called:
a) necrosis;
b) autolysis;
c) apoptosis;
d) mummify; 20. Caseous necrosis develops in:
a) rheumatic fever;
b) tuberculosis,
c) arterial hypertension;
d) Schigella dysentery; 21. What is the most often morphological type of the brain necrosis?
a) gangrene;
b) wet necrosis;
c) dry necrosis;
d) cyst;
22. 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; 23. Deposition of calcium salts into necrotic focus it is a:
a) orga nization;
b) ossi fication;
c) petri faction;
d) beds ore; 24. The injury characterizes by the intraO and extracellular accumulations ofabnormal quantities of a substances is calling:
a) necrosis;
b) apoptosis;
c) degeneration;
d) atrophy; 25. The cause of the liver fat degeneration (steatosis) is:
a) increas ed blood circula tion; b) hypoxi a;
c) hypertension ;
d) acute rheumatic fever;

O the 26. myocardium The fat degeneration is of characterized by the:
a) appearance of connective tissue septas;
b) enlargement of myocites;
c) decreasing ofmyocites' sizes;
d) accumulation oflipids in cytoplasm of several cardiomyocite s only; 27. The clinical manifestation ofthe myocardial fat degeneration is:
a) decreasing ofsystolic ñnction;
b) increasing of systolic function;
c) hypertension;
d) rupture of the heart wall.
28. The liver steatosis is possible in:
a) alcoholism;
b) hypertension;
c) viral hepatitis A;
d) viral hepatitis B, 29. The fat degeneration of myocardium is possible in:
a) hypertension;
b) infectious diseases;
c) protein starvation;
d) acute rheumatic fever; 30. Accumulation of protein masses into tubular epithelium in kidney can be in:

a) hydropic degeneration,
b) mucoid degeneration,
c) steatosis;
d) hyaline-drop degeneration; 31. Reversible stage ofthe

connective tissue disorganization it is:
a) sclerosis;
b) fibrinoid swelling;
c) mucoid swelling;
d) granulomatousis; 32. Hyalinosis ofthe heart valves' casps there is in:
a) hereditary defective valvular heart diseases;
b) rheumatic fever,
c) arterial hypertension;
d) diabetes mellitus, 33. Amyloid it is the protein which can deposit:
a) into cells;
b) into cellular nuclei;
c) between cells;
d) into necrotic focus; 34. At the histological investigation amyloid can be recognized with help of:
a) hematoxylin and eosin;
b) congo-red;
c) sudan Ill;
d) method of Van Geison; 35. Amyloid can be the complication of:
a) bronchiectases;
b) arterial hypertension;
c) atherosclerosis;
d) acute pneumonia; 36. What is the name of etiopathogenetic variant of amyloidosis which develops as complication of another disease?
a) primary;
b) secondary;
c) elderly;
d) hereditary; 37. 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;
38. 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;
39. 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; 40. 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;
41. In obesity in heart there is:
a) appearance oflipids into myocites' cytoplasm;
b) appearance offat tissue septas in myocardium;
c) appearance of connective tissue septas in myocardium;
d) calcium salts deposition;
42. Group ofendogen 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; 43. Call hemoglobin derivate:
a) melanin;
b) hemosiderin;
c) lipofuscin,
d) lipochromine;
44. What does Perl's test show?
a) hemosiderin;
b) bilirubin,
c) porphirin,
d) melanin, 45. What pigment does accumulate in the liver in cachexia?
a) hemosiderin;
b) bilirubin;
c) melanin;
d) lipofuscin; 46. Pigments they are substances:
a) changing colour;
b) can be receptive to stains;
c) of protein nature;
d) soluble in lipids. 47. The group ofexogenic

pigment includes:

a) melanin,
b) lipofuscin,
c) hemosiderin;
d) iron sulfide. 48. What pigment does accumulate in brown enduration ofthe lung?
a) hydrochloride acid hematin;
b) bilirubin,
c) hemosiderin,
d) lipofuscin, 49. 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; 50. 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; 51. Choose the type of the pathological calcification:
a) metabolic;
b) focal;
c) diffuse,
d) metastatic; 52. Metastatic calcification develops in:
á) anemia;
b) hypoxia;
c) hyperlipidemia,
d) hypercalcaemia; 53. 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; 54. Where calcium salts do deposit in metastatic calcification?
a) in connective tissue scars;
b) in connective tissue adhesions;
c) in thrombus,
d) in kidneys, lungs in hypercalcaemia. 55. What pigment can appear in zone of hemorrhage?
a) adrenochromine;
b) melanin,
c) lipofuscin;
d) hemosiderin; 56. What process is the result of melanin metabolism disturbances?
a) vitiligo;
b) leukoplakia;
c) hemochromatosis;
d) jaundice; 57. Keratinization there is in:
a) vitiligo;
b) leukoplakia,
c) widespread melanosis;
d) skin melanoma; 58. Choose the name of an arterial hyperemia type.
a) obstructive;
b) postanemic;
c) ischemic;
d) hydrostatic; 59. Vacate arterial hyperemia develops in:
a) capping glasses applying;
b) an artery forceps removing off;
c) obstruction ofmagistral artery lumen by thrombus;
d) paralysis ofvascular constrictor nerve;
60. What is for the venous congestion development necessary?
a) increasing ofblood flow; b) decreasing ofblood flow;
c) increasing of blood outflow; d) decreasing ofblood outflow. 61. Local venous congestion develops in:
a) obstruction ofan artery lumen by thrombus; b) obstruction of a vein lumen by thrombus;
c) compression of an artery by tourniquet;
d) myocardial infarction; 62. General venous congestion develops in:
a) decompensation ofheart in its hypertrophy;
b) a vein compression;
c) obstruction of a vein lumen by thrombus; d) narrowing of vein lumen by growing tumour; 63.

Acute general venous congestion develops in:
a) myocardial infarction;
b) cardiosclerosis;
c) chronic heart aneurysm;
d) defective valvulars heart diseases; 64. Chronic general venous congestion develops in:
a) myocardial infarction;
b) acute myocarditis;
c) severe myocardial degeneration;
d) cardiosclerosis; 65. What does develop in tissues in acute venous congestion? a) sclerosis;
b) atrophy;
c) petrifaction;
d) edema;

66. 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;

67. 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; 68. What does develop in lung in chronic venous congestion?
a) necrotic foci;
b) inflammation;
c) sclerosis;
d) amyloidosis; 69. What does develop in lung in acute venous congestion?
a) hemosiderosis;
b) edema;
c) sclerosis;
d) hyalinosis; 70. What is the figurative name of the liver in chronic venous congestion?
a) sebaceous;
b) sago;
c) brown;
d) nutmeg; 71. The liver was called "nutmeg" in:
a) acute venous congestion; b) chronic venous congestion;
c) anemia;
d) shock; 72. 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 ofperipheral parts of lobules;
d) beginning ofconnective tissue growth; 73. The result (outcome) of the nutmeg liver is:
a) hepatitis;
b) liver cirrhosis;
c) steatosis;
d) massive necrosis; 74. What does develop in general chronic venous congestion?
a) nutmeg liver;
b) hydrocephalus;
c) big white kidney;
d) big sebaceous kidney; 75. Accumulation of hemosiderin in lung is observed in:
a) acute venous congestion; b) chronic venous congestion;
c) acute pneumonia;
d) emphysema; 76. 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. 77. Acute venous congestion in lesser circulation can develop in:
a) decompensation ofheart hypertrophy;
b) valvular heart diseases;
c) cardiosclerosis;
d) myocardial infarction;
b) blood viscous increasing;
c) difficulties ofblood outflow;
d) stopping ofblood flow in microcirculatory bed; 99. The most severe result of long stasis is:
a) sludge phenomenon;

b) perivascular edema;
c) plasmorrhagia ;
d) necrosis of parenchymatous cells. 100. What does develop in stasis?
a) sludge phenomenon;
b) erythrocytes diapedesis;
c) perivascular edema; d) all enumerated. 101. What does sludge phenomenon mean?
a) adhesion of blood cells to each other; b) erythrocytes agglutination;
c) increasing ofblood cells number; d) increasing ofblood viscous; 102. Edema it is:
a) increased blood filling of organ, tissue;
b) increased containment of interstitial fluid;
c) difficulties ofvenous blood outflow;
d) exudate accumulation, 103. In nephrotic syndrome edemas are:
a) hydrostatic;
b) oncotic;
c) membranogenic;
d) electrolyte; 104. In acute glomerulonephritis edemas are:
a) hydrostatic,
b) oncotic;
c) membranogenic;
d) electrolyte; 105. What is leading there in edemas development in chronic cardiac failure? a) increased hydrostatic pressure;
b) decreasing of colloidosmotic pressure;
c) increased aldosterone secretion;
d) damage of endothelium and basement membranes of capillary, 106. What is observed in lung edema?
a) increasing oflungs sizes;
b) increasing of lungs weight;
c) flabby consistency of lungs;
d) flowing down of foamy fluid at the incision; 107. What is observed in lung edema?
a) increasing oflungs sizes;
b) decreasing of lungs weight;
c) increased air filling of lungs;
d) all enumerated. 108. What does develop in lung edema?
a) extension of alveolars spaces;
b) accumulation of edematous fluid in alveolars spaces;
c) sclerosis ofinter-alveolar septas;
d) deposition of hemosiderin; 109. What does develop in lung edema?
a) hyperemia of capillars;
b) accumulation of edematous fluid in alveolars spaces;
c) narrowing ofalveolars spaces;
d) erythrocytes diapedesis; 110. 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, 111. Call the certain variant ofedematous fluid containment increasing:
a) hematoma;
b) ascitis;
c) petechia,
d) exicosis; 112. 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; 113. Choose the cause of acute ischemia:
a) obturation of vein by thrombus; b) obturation ofartery by thrombus;
c) embolism;
d) compression of artery by tumour;
e) all enumerated.
114. What is important result of acute ischemia possible?

a) sclerosis;
b) necrosis;
c) hemosiderosis;
d) atrophy; 115. What is the reversible change of cell in ischemia?
a) kariopicnosis;
b) kariorrhexis;
c) plasmolysis
d) disappearance of glycogen. 116. What is the cause of thrombus formation?
a) damage ofblood vessel wall;
b) slow blood flow;
c) turbulent blood flow;
d) all enumerated.

117. What morphological type of thrombus is nonexistent? a) red;
b) white,
c) mixed;
d) white with red rim; 118. More often white thrombi form in:
a) veins;
b) arteries;
c) aneurysm cavity; d) capillars. 119. More often red thrombi form in:
a) veins;
b) arteries;
c) capillars;
d) heart chambers;
120. One ofunfavorable thrombus formation outcomes is:
a) organization;
b) thromboembolism,
c) petrifaction;
d) vascularization; 121. Obstructive thrombus of artery can cause:
a) venous congestion;
b) arterial hyperemia;
c) infarction;
d) thromboembolism, 122. Obstructive thrombus of vein can cause:
a) venous congestion;
b) arterial hyperemia;
c) infarction;
d) petrifaction;
123. Favorable outcome of thrombus formation is:
a) septic autolysis;
b) suppuration;
c) organization;
d) thromboembolism,
124. Thrombus which is consisting of alternating red thrombus particles with white thrombus particles is called:
a) red;
b) white;
c) mixed;
d) hyaline, 125. Thrombus which contains lot oferythrocytes is called: a) red;
b) white,
c) mixed;
d) flaky; 126. Thrombus which contains lot ofleukocytes and fibrin is called:
a) red;
b) white,
c) mixed;
d) flaky;
127. What does develop in low extremity in artery femoralis obturation by thrombus?
a) dry necrosis;
b) wet necrosis;
c) gangrene;
d) infarction, 128. Call a type of embolism:
a) ischemic,
b) air;
c) angioneurotic;
d) vacate; 129. Thromboembolism of šmall brunches of pulmonary artery can cause:
a) pulmonocoronary reflex;
b) lung infarction;
c) athelectasis;
d) shock; 130. 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; 131. Thromboembolism of pulmonary trunk and its large brunches results as:
a) pulmonocoronary reflex;
b) lung infarction,
c) athelectasis,
d) shock; 132. 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 oflarge circulation;

133. Call the localization of thrombi in large circulation arteries thromboembolism.
a) valvulars ofthe left part of heart:
b) valvulars ofthe right part of heart,
c) veins of lesser circulation; d) veins oflarge circulation; 134. 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, 135. Infarction it is necrosis:
a) with different etiology;
b) with curtain localization;
157. The basic cells in a focus of acute inflammation are:
a) monocytes;
b) macrophages;
c) histiocytes;
d) neutrophilic leukocytes;
158. What is the morphological appearance of alteration in inflammation?
a) atrophy;
b) necrosis;
c) hyperplasia;
d) apoptosis;
159. What is exudate? a) edematous fluid;
b) inflammatory fluid;
c) pathological fluid with protein containment;
d) inflammatory fluid with erythrocytes containment;
160. Call morphological type of inflammation:
a) specific;
b) proliferative;
c) immune;
d) acute; 161. Call morphological form of exudative inflammation:
a) serous;
b) granulomatous;
c) interstitial;
d) mucoid; 162. The most often outcome of serous exudate is:
a) organization;
b) petrifaction;
c) resolution;
d) coming to purulent; 163. Call the form of fibrinous inflammation:
a) purulent;
b) putrificant;
c) croupous;
d) catharal; 164. In what localization does croupous form of fibrinous inflammation develop only?
a) pleura;
b) tonsils;
c) colon;
d) urinary bladder; 165. In what localization is diphtheroid form of fibrinous inflammation possible only?
a) pleura;
b) peritoneum;
c) pericardium;
d) tonsils; 166. What morphological type of fibrinous inflammation does on oral mucosa develop?
a) phlegmonous;
b) interstitial;
c) hemorrhagic;
d) diphtheroid 167. Call the localization where both types of fibrinous inflammation can develop:
a) tonsils;
b) oral cavity;
c) pleura;
d) colon. 168. 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; 169. The most often outcome of fibrinous iñflammation is:
a) resolution;
b) coming to purulent;
c) organization;

d) mucoidazation; 170. 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; 171. Heart was named "testaceous" in following changes of pericardium.
a) fibrin sedimentation;
b) pus appearance;
c) growth oftumour;
d) organization and petrifaction of exudate; 172. What is the most often cause ofpurulent inflammation?
a) viruses;
b) protozoa;
c) chemical substances;
d) staphylococci. 173. At microscopic investigation purulent exudate is diagnosing on to lot of:
a) fibrin;
b) neutrophyls;
c) macrophages;
d) lymphocytes; 174. The basic part of purulent exudate is:
a) water;
b) neutrophyls;
c) necrotic debris;
d) fibroblasts;

175. Call the form of purulent inflammation:
a) abscess;
b) granuloma;
c) gangrene;
d) cyst; 176. What morphological form of inflammation does develop on tonsils in diphtheria?
a) diphtheroid;
b) croupous;
c) catharal;
d) purulent; 177. What morphological form of inflammation does develop in larynx and trachea in diphtheria?

196. Usual outcome of acute catharal inflammation is:
a) sclerosis and deformation;
b) organization and petrifaction;
c) resolution and tissue repair;
d) ulceration and perforation;
197. Exudate which is containing little of leukocytes and lot of fluid is calling: a) serous;
b) purulent;
c) fibrinous;
d) hemorrhagic;
198. Exudate which is containing lot of neutrophils is calling:
a) serous;
b) purulent;
c) fibrinous;
d) hemorrhagic;
199. Exudate which is containing lot of fibrin is calling:
a) serous;
b) purulent;
c) fibrinous;
d) hemorrhagic;
200. What is hematogenic cell of inflammatory infiltration?
a) endothelial;
b) tissue basophils;
c) fibroblast;
d) lymphocyte;
201. What is histiogenic cell of inflammatory infiltration?
a) monocytes;
b) lymphocytes;
c) epithelioid;
d) neutrophils;
202. What is usual outcome ofchronic inflammation?
a) suppuration;
b) sclerosis;
c) petrifaction;
d) ossification; 203. Call the morphological type of proliferative inflammation:
a) granulomatous;
b) purulent;
c) hemorrhagic;
d) anaerobic; 204. Proliferative inflammation it is inflammation with:
a) acute alteration,
b) granulomas formation,
c) predomination of proliferation;
d) growing ofconnective tissue; 205. Usual clinical course of proliferative inflammation is:
a) acute,
b) subacute;
c) chronic;

d) rapid progressive. 206. What cells can multiply in focus of proliferative inflammation?
a) macrophages;
b) reticulocytes;
c) erythrocytes;
d) neutrophils; 207. 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; 208. Usual outcome of proliferative interstitial inflammation is:
a) edema;
b) sclerosis,
c) suppuration;
d) petrifaction; 209. Granuloma it is focus of:
a) purulent inflammation;
b) accumulation of lymphoid cells;
c) accumulation of cells can do phagocitosis;
d) caseous necrosis; 210. What type of granuloma does not present?
a) epithelioid;
b) giant-cellular;
c) immune;
d) neutrophilic. 211. Show the type of granuloma according to the cellular composition:
a) specific;
b) giant-cellular
c) immune,
d) lipogranuloma; 212. Choose noninfectious granuloma:
a) tuberculous;
b) oleogranuloma;
c) syphilitic;
d) in scleroma; 213. Choose infectious granuloma:
a) oleogranuloma;
b) lipogranuloma,
c) syphilitic;
d) around of foreign body; 214. Non-immune granuloma develops in:
a) in alveococcosis;
b) in tuberculosis;
c) in syphilis;
d) in scleroma; 215. Immune granuloma develops in.
a) in alveococcosis;
b) in asbestosis;
c) around of foreign body;
d) in tuberculosis, 216. In what acute infectious disease granulomas are usual?
a) Schigella dysentery;
b) diphtheria;
c) scarlet fever;
d) yersiniosis. 217. What cells are there in tuberculous granuloma?
a) Aschoffcells;
b) Hodgkin cells;
c) Reed- Schternberg cells;
d) Pirogov-Langhance cells. 218. What granuloma's develop around of suture material? a) immune; b) specific; c) giant-cellular; d) injections; 226. What granuloma does develop? a) macrophagal;
a) sclerosis;
b) suppuration;
c) mucoidazation;
d) resolution; 227. Specific granuloma develops in: a) rheumatic fever; b) tuberculosis;
a) suppuration;
b) hemorrhagic infiltration;
c) putrefaction;
d) scaring; 228. Intensive metabolism is observed in: 221. is the complication of syphilitic aneurysm ofabdominal aorta;
c) anal-genital area
d) bronchial mucosa; 225. Choose non-immune type does the outcome in tuberculosis? a) in tuberculosis; b) in syphilis; c) in scleroma; d) around of foreign body.
219. What is the outcome of granuloma? a) sclerosis; b) epithelioid; c) giant-cellular; d) necrotic;
220. What is favorable outcome oftuberculous granuloma?
a) suppuration;
b) hemorrhagic infiltration;
c) putrefaction;
d) scaring;
221. is the complication of syphilitic aneurysm ofabdominal aorta;
a) granuloma around of foreign body;
b) tuberculous granuloma;
c) myocardial infarction;
d) aortic valve defect.

granulomatous diseases? 222. Granuloma with a) acute clinical course; unknown etiology b) total recovery is often; develops in: c) accompanied with

a) rheumatic fever; immunity disorders;
b) tuberculosis; d) exudation develops
c) syphilis; always;
d) sarcoidosis (Besnier- 230. What is it Boeck-Schaumann's proliferation? disease); a)' cellular death;

223. Usual localization of b) cellular injury; inflammatory polyps is: c) result of inflammation;

a) serous membranes; d) multiplication of cells;
b) meningeas; 231. Increasing of
c) anal-genital area functional elements mucosa; volume is accompanied
d) nasal mucosa; with increasing of function

224. Usual localization of is pointed condilomas is: called:

a) serous membranes; a) degeneration;
b) meningeas; b) displasia;
c) hypertrophy;
d) atrophy;

232. Hypertrophy it is:

a) restoration oftissues 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; 233. Increased number of cellular elements is called:

a) degeneration;
b) displasia;
c) hypertrophy;
d) hyperplasia; 234. Choose the type of hypertrophy:

a) working (compensatory);
a) cardiomyocites sizes increasing; b) cardiomyocites number increasing;
c) stromal edema;
d) cardiomyocites intracellular accumulations. 239. The cause of physiological myocardial hypertrophy is:
a) defective valvular heart disease;
b) cardiosclerosis;
c) physical training;
d) arterial hypertension; 240. Myocardial hypertrophy in phase of its

O compensation is characterizing by

b) neurotic;
c) compressive;
d) cerebral; 235. Glandular hyperplasia of endometrium it is: a) working hypertrophy;
b) vicar hypertrophy;
c) correlative hypertrophy;
d) neurohumoral hypertrophy. 236. What type of myocardial hypertrophy does in defective valvular heart disease develop?
a) working hypertrophy;
b) vicar hypertrophy;
c) correlative hypertrophy;
d) neurohumoral hypertrophy. 237. What type of myocardial hypertrophy does in arterial hypertension develop?
a) working hypertrophy;
b) vicar hypertrophy;
c) correlative hypertrophy;
d) neurohumoral hypertrophy. 238. Myocardial hypertrophy develops as result of:

fallowing sign only:

a) decreasing ofheart sizes;
b) thickening of ventriculars walls;
c) dilation of chambers;
d) flabby consisten

cy of myocardium; 241. 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; 242. 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. 243. What is the manifestation of hypertrophic heart decompensation?

a) myogenic dilation of chambers;
b) brown atrophy of myocardium;
c) rheumatic myocarditis;
d) fibrinous pericarditis; 244. In what organ does vicar hypertrophy develop?
a) heart;
b) stomach;
c) kidney;
d) uterus; 245. The phase of hypertrophic heart decompensation is characterizing by the fallowing sign:
a) flabby consistency of myocardium;
b) paleness of myocardium;
c) thickening ofventricles walls; d) increasing of heart mass; 246. 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; 247. Decreasing of structural elements volume in living organism is called:
a) hypertrophy;
b) hyperplasia;
c) atrophy;
d) hypoplasia; 248. What is the example of local atrophy?
a) dysfunctional;
b) cancerомatous cachexia;
c) hypophysial cachexia;
d) cerebral cachexia;
249. What is the example of general atrophy? a) alimentary emaciation;
b) neurotic atrophy;
c) atrophy due to long time compression;
d) dysfunctional; 250. What is the example of local atrophy?
a) vicar;
b) carcinomatous;
c) ischemic;
d) cerebral; 251. 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. 252. 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. 253. 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. 254. Brown atrophy can develop in:
a) stomach;
b) lung;
c) prostate;
d) liver.
255. What does in brain develop as result of liquor outflow difficulties?
a) edema and swelling;
b) hydrocephalus;
c) tumour;
d) meningitis;
256. Changing oftissue type on related one is called:
a) displasia;
b) anaplasia;
c) hyperplasia;
d) metaplasia;
257. Metaplasia of

O connective tissue is possible in:

a) osseous;
b) muscular;
c) nerve;
d) epithelial; 258. What epithelium does develop in metaplasia of bronchial mucosa?
a) columnar;
b) prismatic;
c) squamous-cell;
d) atrophic; 259. Metaplasia of bronchial epithelium develops as result of:
a) lymphostasis;
b) hyperemia;
c) necrosis;
d) chronic inflammation. 260. Metaplasia of bronchial epithelium can come to:
a) degeneration;
b) atrophy;
c) necrosis;
d) carcinoma; 261. What is the synonym of general atrophy?
a) hypoplasia (aplasia);
b) emaciation (cachexia);
c) hypertrophy (hyperplasia);
d) dwarfism.

262. Growth of connective tissue into pathological focus is called.
a) metaplasia;
b) encapsulation;
c) organization;
d) petrifaction; 263. Growth of connective tissue around of pathological focus is called:
a) metaplasia;
b) encapsulation;
c) organization;
d) petrifaction; 264. Massive sclerosis of organ with its reorganization and deformation is called:
a) scar;
b) diffuse sclerosis;
c) cirrhosis;
d) diffuse fibrosis; 265 Restoration of structural tissue elements instead dead is called:
a) organization;
b) tissue repair;
c) metaplasia;
d) displasia; 266. Compensatory heart hypertrophy develops in:
a) DIC-syndrome;
b) shock;
c) acute myocarditis;
d) arterial hypertension; 267. 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; 268. 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; 269. 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. 270. 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, 271. Choose the type of tumour growth into tissues:
a) unicentric;
b) infiltrative;
c) exophytic
d) endophytic 272. Choose the type of tumour growth in cavitary organ:
a) unicentric;
b) multicentric;
c) appositional;
d) exophytic; 273. Call the pathway of metastatic spreading:
a) implantation;
b) infiltrative;
c) expansive;
d) appositional; 274. Morphological atypia of tumour can be:
a) antigenic and histochemical;
b) exophytic and histochemical;
c) expansive and infiltrative;
d) tissue and cellular. 275. Organoid tumour has:
a) good developed parenchyma;
b) good developed stroma;
c) two compone

nts: parenchyma and stroma: d) two same volume components: stroma and parenchyma. 276. Histioid tumour has.

a) good developed parenchyma;
b) good developed stroma;
c) two components: stroma and parenchyma;
d) two same volume components: stroma and O parenchyma. 277. What is not there in metastatic spreading process:
a) disattachment oftumour cells from basic tumour node;
b) carrying of tumour cells;
c) development of secondary tumour nodes;
d) development ofnecroses and hemorrhages into tumour nodes. 278. Choose the definition of sarcoma:
a) immature tumour from fibrous tissue;
b) immature tumour from mesenchymal tissues;
c) mature tumourfrom mesenchymal tissues;
d) mature tumour from fibrous tissue. 279. Choose the type of tumour growth into tissues:
a) exophytic
b) endophytic;
c) expansive;
d) unicentric;
2S() [1],Vhat is the basic structural element of a
a) stroma;
b) parenchyma;
c) blood vessels;
d) necrosis; 281. 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. 282. Choose the tumour with local destructive growth.
a) venous hemangioma;
b) cavernous hemangioma;
c) capillary hemangioma;
d) chondroma; 283. Choose the tumour with local destructive growth.
a) chondroma;
b) lipoma;
c) fibroma of skin,
d) nasopharingeal angiofibroma; 284. Call mesenchymal tumour:
a) adenoma;
b) angiosarcoma;
c) papilloma,
d) hepatoma; 285. Call benign mesenchymal tumour:
a) nasopharingeal angiofibroma;
b) fibroma of skin;
c) leiomyosarcoma,
d) desmoid, 286. What can be classified as benign mesenchymal tumour?
a) fibromyoma;
b) leiomyosarcoma;
c) osteosarcoma;
d) desmoid,
287. What can be classified as malignant mesenchymal tumour?
a) fibromyoma;
b) leiomyosarcoma;
c) nasopharingeal angiofibroma;
d) desmoid; 288. Cavernous hemangioma of liver is characterized by the following feature only:
a) tissue and cellular atypia;
b) immature cells;
c) malignant;
d) consists ofvenous type blood vessels; 289. The benign tumour from muscular tissue is:
a) fibroma;
b) fibrosarcoma;
c) hemangioma;
d) leiomyoma; 290. What is the favorite pathway of spreading in sarcomas?
a) lymphogenic;
b) hematogenic;
c) perineural;
d) contact. 291. 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; 292. What is the most often localization of leiomyoma?
a) skin;
b) heart;
c) uterus;

d) soft tissues; 293. Choose the histological type of fibrosarcoma:
a) soft;
b) dense,
c) undifferentiated;
d) youthful, 294. What is the precancer disease in melanoma development?
a) nevus;
b) displasia of melanocytes;
c) pigment spot;
d) vitiligo; 295. Choose the mature tumour which develops from squamous-cell epithelium:
a) adenoma;
b) papilloma,
c) carcinoma;

O d) 296. cystic Choose adenoma;the morphological type of adenoma:
a) papilloma;
b) hemangioma,
c) cystadenoma;
d) adenocarcinoma; 297. What is right there in the characteristic of papilloma?
a) immature tumour;
b) exophitic growth is typical;
c) it can malignize;
d) rapid growth, 298. What is the most often papilloma's localization?
a) stomach;
b) esophagus;
c) skin,
d) urinary tract; 299. Squamous-cell carcinoma is especially typical for:
a) thyroid gland,
b) pancreas,
c) uterine cervix;
d) uterine body; 300. The morphological sign ofwell-differentiated squamous-cell carcinoma is:
a) keratinization;
b) mucus formation,
c) solid structures;
d) pathological mitoses; 301. Usually primary malignant tumour of esophagus it is:
a) adenocarcinom a;
b) squamous-cell carcinoma; c) undifferentiate d carcinoma;
d) melanoma;
302. Adenogenic carcinoma more often develops in:
a) stomach;
b) esophagus;
c) bronchi;
d) uterine cervix; 303. What does not define as poorly-differentiated carcinoma?
a) adenocarcinoma;
b) solid carcinoma;
c) colloid carcinoma;
d) medullar carcinoma;
304.Colloid carcinoma is characterized by the fallowing feature only: cancer
a) cellular atypia in association with mucus hyperproduction,
b) expansive growth;
c) absence of relapse,
d) late metastatic spreading;
e) good prognosis.
305. Fibrous (cirrhous) carcinoma is characterized by the fallowing feature only:
a) mild malignancy;
b) late metastatic spreading;
c) lot of stroma with complexes ofatypical cells;
d) absence of relapse;
e) good prognosis.
306. Dysplasia is:

a. transition of one type of fabric to another, related to it

b. replacement of the site of necrosis or thrombus with connective tissue

c. intravital reduction in cell volume, accompanied by a decrease or termination of their function

d. violation of cell proliferation and differentiation with the appearance of cell atypia and violation of histoarchitectonics stomach