INTRODUCTION TO PATHOLOGY AND PATHOLOGICAL ANATOMY. RESEARCH METHODS IN PATHOLOGICAL ANATOMY. INTRODUCTORY LESSON. RESEARCH METHODS IN PATHOLOGICAL ANATOMY. SHOW AUTOPSY

1 What the term pathology?

2 Define pathological anatomy

3 What is a pathological process?

4 What is called a disease?

5 Why is Rudolf Virchow considered the founder of modern pathological anatomy?

6 What are the levels of disease research in modern pathology

7 What does modern pathological anatomy study in the study of diseases?

8 What is called pathomorphosis?

9 What does the term thanatogenesis mean?

10 What does the term iatrogenia (iatrogenic pathology) mean?

11 Name the groups of subjects studied by the pathologist

12 Name the types of intravital substrates obtained from patients.

13 What is the subject of a post-mortem examination (autopsy, section)?

14 What are the levels of study of cadaveric material?

15 What additional research methods does the pathologist attract at autopsy?

16 Name the main purpose of the autopsy of a deceased patient.

17 Determine the value of the autopsy of the deceased patient.

18 For what purpose is the material taken during the patient's lifetime for microscopic examination?

19 What kind of examination is the surgical and biopsy material subjected to? What is called a biopsy?

20 Define a biopsy.

21 Why does a pathologist examine surgical material?

22 Why is a minor operation performed and biopsy material taken with it?

23 Why is an urgent histological examination performed during surgery?

24 Name the time spent on the preparation of a histological preparation for an urgent histological examination

25 Name the most commonly used material for histological examination by a gynecologist

26 Name the widely used universal histological coloration of tissue sections

27 Name sources for cytological examination

28 What does Papanicolaou reaction mean?

29 How is aspiration puncture of organs performed in oncological practice?

30 What is the Pap-NET screening system?

31 What is the immunohistochemical method of investigation?

32 What are the types of antigens that the immunohistochemical method is used to detect?

33 How is the antigen determined using the immunohistochemical method?

34 Why are monoclonal antibodies used?

35 What does the term marker indicate?

36 What are molecular biology methods used for?

37 What are the main molecular biology techniques used in the histological section?

38 What is the flow cytometry method used for?

39 How is the in situ hybridization technique applied?

40 What is the in situ hybridization technique used for?

41 What is transmission electron microscopy?

42 What is scanning electron microscopy?

43 What is electron microscopy used for?

44 What slices are used in electron microscopy?

45 How thick are the slices used in microscopic examination?

46 What is the purpose of electron microscopy?

47 What is the experimental material used for by the pathologist?

48 What are experimental studies in vivo?

49 What are experimental in vitro studies?

50 What is the basis of modern pathology?

51 Where are the genetic variations associated with diseases detected at the present stage located?

52 Finding out which factors underlying diseases is the main theme of modern medicine.

53 What mechanisms of development and progression of the disease are studied at the current level?

54 Why is pathological anatomy important for practical medicine in our time?

55 Why do clinicians need research by pathologists?

56 What is devoted to the field of pathological anatomy in medicine?

57 What is the cadaveric material taken for examination by a pathologist?

58 What is the main purpose of an autopsy

59 What does a pathologist study before an autopsy?

60 Who should be present at the autopsy other than the pathologist?

61 What is the size of the piece taken for biopsy examination?

62 What is investigated in oncology using electron microscopy?

63. Why does a future doctor need a pathological anatomy?

64. What contributes to the formation of clinical thinking of the student in the study of pathological anatomy?

65. What did I.V. Davidovsky prove in the general biological significance of pathological reactions of the body?

66. What is the basis of biological feasibility?

67 Name the basic pattern underlying the functioning of the body

67 What is the basis of biological expediency?

68 What is the main pattern underlying the functioning of the body

69 What determines the specifics of each disease?

70 What principle of teaching pathological anatomy was introduced thanks to the initiative of I.V. Davidovsky?

71. What reactions of the body provide homeostasis?

72. Name the conditions for the decline in function in the disease.

73. What is the basis of decompensation and organ death?

74 What is called clinical thinking?

75 The task of the general course of pathological anatomy

76 Why is it necessary to study the general pathological reactions of the body?

77 What is studied in the section of private pathological anatomy?

78 What underlies the functioning of the body at all levels of its organization?

79 Why is pathological anatomy a "bridge" between fundamental and practical medicine?

80 What does the term etiology mean?

81 What does the term pathogenesis mean?

CIRCULATORY DISORDERS (PLETHORA, ANEMIA, STASIS, BLEEDING). VIOLATION OF THE CONTENT OF TISSUE FLUID.

1.		What is Hyperemia?		P=1
2.		List the types of hyperemia depending on the location. a), b)	P = 2	
	3.	List the types of hyperemia by duration of development. a), b)		P = 2
4.		List the types of hyperemia by pathogenesis a), b)		P = 2
5.		What is working (functional) hyperemia?	P=1	
6.		Explain the development of postischemic hyperemia.		P=1
7.		Explain the development of vacate (decompression) hyperemia.		P=1

8.	Explain the development of collateral hyperemia.	$\mathbf{P} = 2$
9.	Name the development of angioneurotic hyperemia. a), b), c)	P=3
10.	Explain the development of hyperemia in the presence of an arteriovenous sl	
11.	What causes the development of hyperemia in the presence of an arteriovene	
	a), b), c)	P=3
12.	What is venous congestion (venous hyperemia, venous congestion)?	P=3
13.	What are the types of venous congestion by prevalence. $P = 2$	
14.	What is heart failure? P=3	
15.	What are the types of heart failure in the clinical course.	
16.	What are the types of heart failure depending on the damage to the heart. a)	\mathbf{b} \mathbf{c}
10.		, 0), 0)
17	P=3	
17.	In what circle of blood circulation does venous congestion develop in a patie	ent with left
	ventricular heart failure? P=1	
18.	In what circle of blood circulation does venous congestion develop in a patie	ent with right
	ventricular heart failure? P=1	
19.	What diseases develop acute venous congestion?	
	a), b), c), d) P=4	
	20.What diseases develop chronic venous congestion?	
	a), b), c), d), e)	P = 6
	21.What morphological changes develop in the lungs in acute venous conges	
		suon? a), 0),
	c) P=3	0 1 1
	1) What morphological changes develop in the liver in acute venous pletho	ra? a), b), c)
	, d) P = 4	
	23. What morphological changes develop in the kidneys in acute venous con-	gestion? a),
	b), c), d) $P = 4$	
	24. What is the main etiological factor of pathogenesis, leading to the develo	pment of
	changes in organs in a patient with chronic venous stasis.	P=1
	25.Name the morphological changes that develop in the organs of chronic ve	enous stasis. a)
	, b), c), d), e), e), f), g) $P = 7$	
	26. What characterizes the morphologically capillary-parenchymal block?	P-2
	27. List the morphological changes in the lungs in chronic venous congestion	
		n (stagnation).
20	a), b), c), d), e), f) $P = 6$	
28	3. What are heart failure cells (heart defect cells) called? $P = 2$	
	29. List the morphological changes in the liver in chronic venous plethora. a), b), c),
	d), e) $P = 5$	
30	D. Define ischemia. $P = 2$	
31	.List the types of ischemia by duration. $P = 2$	
32	2. List the types of ischemia by etiology. $P = 4$	
	33. In what diseases, conditions does angiospastic (neurogenic) ischemia dev	velop?
	P = 4	1
34	. What are the main mechanisms for the development of angiospastic ischemi	P = 2
	5. What are the causes of obstructive ischemia. $P = 4$	u. 1 – 2
55	36. What are the reasons for the development of compression ischemia.	P = 1
	37.What are the reasons for the development of collateral ischemia.	P = 2
	38. What factors determine the outcomes of ischemia?	$\mathbf{P} = 4$
	. List the possible outcomes of ischemia.	P = 4
). List the types of physiological arterial hyperemia.	P = 3
41	.What is reactive (post-occlusive) hyperemia?	P = 1
42	List the types of pathological arterial hyperemia.	P = 6
	B.What is bleeding?	P = 2
	0	
	44. What is hemorrhage (hemorrhage)?	$\mathbf{P} = 2$
45	44. What is hemorrhage (hemorrhage)? 5. List the mechanisms of bleeding and name them in Latin.	P = 2 $P = 6$

46. Name in Russian and Latin types of internal bleeding into serous cavities. a), b	
P = 6 47. List the types of bleeding depending on the source. a), b), c), d), e) P = 5	
48. List the types of external bleeding and name them in Latin. $1 - 5$	
a), b), c), d), e), e) $P = 6$	
49. List the vessels from which bleeding per diapedesin is possible.	
a), b), c) $P = 3$	
50. List stomach diseases complicated by bleeding per diabrosin.	
a), b), c) $P = 3$	
51. List diseases complicated by bleeding per rhexin.	
a), b), c), d) $P = 4$	
52. List the diseases in which bleeding per diapedesin may develop. a), b), c), c	l), e),
e), f), g), h), i) $P = 9$	
53. What are the types of hemorrhages?	$\mathbf{P} = 6$
54. What is a hematoma? Name the mechanism of development.	$\mathbf{P}=4$
55. What is hemorrhagic infiltration? What are the mechanisms of development	$\mathbf{P} = 4$
56. What is bruising? What are the mechanisms of development?	P = 4
57. What are petechiae? What is the mechanism of development?	P = 3
58. What is purpura? What is the mechanism of development?	$\mathbf{P}=3$
59. List the types of circulatory disorders.	D O
a), b), c), d), e), f), g), h) 60. What is stasis?	P =9 P = 4
61.List the stages of formation and resolution of stasis a), b), c)	P = 4 P = 3
62. What are the sequentially developing changes in the blood that contribute to	1 – 5
the development of stasis. a), b), c)	P = 3
63.List the most common causes of blood stasis.	1 = J
a), b), c), d), e)	P = 5
64.What is plasmorrhagia?	P = 2
65.What is edema?	P = 3
66.List the processes that contribute to the formation of edema.	
a), b), c), d)	$\mathbf{P} = 4$
67. What disorders of the lymphatic system contribute to the formation of edema?	
a), b), c), d)	P = 4
68. What is the name of the accumulation of fluid	
a) in the pleural cavity?	
b) in the abdomen?	
c) in the pericardial cavity?	P = 3
69.List the pathological changes (microscopic) with edema and swelling of the brain.	
70. What is the outcome of the increasing cerebral edema?	$\mathbf{P}=2$
71. What are the two main pathogenetic factors in the development of cardiac edema	D 0
in the decompensation of heart disease.	P=2
72.What is Anasarca?P =73.What is chylothorax?P =	
73. What is chylotholdx? $P = 74.$ What are the reasons for the development of hillous ascites. a), b). $P = 74.$	
TASKS	<i>L</i>
1 A 72-year-old patient suffered from rheumatic mitral valve disease for a lor	o time th

1. A 72-year-old patient suffered from rheumatic mitral valve disease for a long time, the immediate cause of death was decompensated chronic cardiovascular insufficiency. What are the changes that have developed in: a) lungs, b) liver, c) kidneys, d) spleen. (p=4). Name the accumulation of fluid in: e) pleural cavities, f) heart chamber, g) abdominal cavity. (p=3).P = 7

2. A 68-year-old patient suffered a myocardial infarction 4 years ago, and therefore there is a large-focal postinfarction cardiosclerosis (scar) in the posterior wall of the left ventricle. At the time of going to the doctor, he complains of increasing shortness of breath, cough with sputum of a "rusty" color. Explain the color of sputum in the patient: a) name the pigment, b) name the group of pigments to which it belongs, c) name the cells, in which it is formed, d) give the figurative name of these cells. P = 4

3. A 76-year-old patient died from decompensated chronic cardiovascular insufficiency with atherosclerotic aortic valve disease. At the autopsy, among other changes, nutmeg liver was found. List: a) stages of liver damage (p = 4), b) the main microscopic changes in different stages of the muscat liver (p = 3). P = 7

4. A 12-year-old child suffered from acute myeloid leukemia and died. At the autopsy, multiple small-focal hemorrhages were found in the brain, as well as in the region of the subcortical nuclei of the left hemisphere there is a focus with a diameter of 2.5 cm, in the form of a cavity filled with blood clots. Indicate in the answer: a) the name of multiple small foci of hemorrhages, b) their pathogenesis, c) the name of the major hemorrhage, d) its pathogenesis, e) Name the term used to refer to the tendency to bleed. P = 5

5. A 23-year-old patient was admitted to the surgical department with a diagnosis of blunt trauma to the abdomen. During the operation, the following was diagnosed: rupture of the spleen; in the abdominal cavity - 300 ml of liquid blood. Name: a) accumulation of blood in the abdominal cavity, b) the type of bleeding in this patient at the source.

 $\mathbf{P}=\mathbf{2}$

6. A 70-year-old patient died in intensive care. At the autopsy: the brain is moderately enlarged. The substance of the brain on the incision is moist, shiny. The lateral ventricles are dilated, filled with a clear liquid. On the posterior surface of the tonsils of the cerebellum, the sulcus from the wedging into the foramen magnum is determined. Based on the described macroscopic signs, name the process that has developed in the brain. P = 1

7. A 52-year-old patient was admitted to the intensive care unit with a diagnosis: "Central cancer of the upper lobe of the right lung." At the same time, the patient has massive hemoptysis. Name: a) the term for this bleeding, b) the mechanism of its development. P = 2

8. In a deceased patient who had been suffering from arterial hypertension for a long time, an autopsy found: an extensive accumulation of blood coagulations in the subcortical nuclei of the left hemisphere of the brain (4.0 cm in diameter). Name: a) the type of circulatory disorder in the brain in this case. (p = 2), b) the mechanism of development of this circulatory disorder. (p=1) P = 3

9. During the autopsy of a deceased patient, a chronic ulcer with signs of exacerbation was found in the wall of the pyloric part of the stomach. In the lumen of the stomach - 800 ml. liquid blood and its convolutions. Name: a) complication of chronic gastric ulcer (p = 2) b) the mechanism of development of this complication. (p=1) P=3

10. A 64-year-old patient was admitted to the emergency room with complaints of pain in the epigastric region; Vomiting of gastric contents of the color of "coffee grounds" was noted. On an emergency basis, the patient is sent for gastroscopy. List stomach diseases that may be complicated by the development of gastric bleeding. (p=3). What is a common mechanism for the development of gastric bleeding. (p=1) P = 4

11. In a 45-year-old patient, an X-ray of the lungs revealed a tuberculous cavity in the I-II segments of the right lung; There is a cough, sputum mixed with blood. Name in Latin the type of external bleeding and the mechanism of its development. P = 2

12. An elderly woman had uterine bleeding. Endometrial scraping has been diagnosed with cancer. Explain the cause of bleeding, name in Latin this type of external bleeding and its mechanism. P = 3

13. With the rapid release of ascitic fluid, the patient lost consciousness. Explain this phenomenon. P = 2

14. When quickly lifting from the caisson, the worker had bleeding from the nose and ears. Explain the reason. P = 2

15. A patient with a festering wound bleeding from the wound occurred 7 days after the injury. Explain its cause and name its mechanism in Latin. P = 2

THROMBOSIS, EMBOLISM

2. List the stages of blood clotting (coagulation). a), b), c), d) P = 4 3. Reveal the main mechanisms of action of the anticoagulant system of blood (fibrinolysis). a), b), c) P = 3 4. What are the three main groups of pathogenetic factors of thrombosis (Virchow's triad). a), b), c) P = 3 5. What are the main causes of damage to the walls of blood vessels and endocardium, contributing to the formation of a blood clot a), b), c), d), e) P = 5 7. What are the main causes of slowing and impaired blood flow in the vessels, contributing to the formation of a blood clot a), b), c), d), e) P = 5 7. What are the main causes of slowing and impaired blood flow in the vessels, contributing to thrombosis. a), b), c), d), e) P = 3 10. What are the types of blood clots in relation to the lumen of blood vessels. a), b) P = 2 11. List the stages of thrombosis. a), b), c), d) P = 3 10. What are the types of blood clots, depending on their composition a), b), c), d) P = 4 12. List relatively favorable outcomes of thrombosis. a), b), c), d), e) P = 5 13. List the adverse outcomes of thrombosis. a), b) P = 5 13. List the adverse outcomes of thrombosis. a), b) P = 5 14. Define thrombosindowing in the interial bed. a), b), c) P = 5 15. List the vessels, thrombosis (localization of blood clots) that contribute to the development of pulmonary embolism. (PE). a), b), c) P = 3 19. List the types of embolisms depending on the direction of movement of the embolus. a), b), c) P = 3 20. Define embolism. P = 1 21. Define retrograde embolism. P = 3 23. What are the processes by which paradoxical embolism develops. a), b), c) P = 3 24. Name the types of embolisms, depending on the nature of the embolus. a), b), c) P = 3 25. What is a microbial embolism? P = 2 26	1.	Define thrombosis.	P = 3
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19.List the types of embolisms depending on the direction of movement of the embolus.a), b), c)P = 320.Define orthograde embolism.P = 121.Define retrograde embolism.P = 222.Define paradoxal embolism.P = 323.What are the processes by which paradoxical embolism develops.a), b), c)a), b), c)P = 324. Name the types of embolisms, depending on the nature of the embolus.a), b), c)a), b), c), fl, gl, h)P = 825. What is a tissue embolism?P = 226.What is a microbial embolism?P = 327.What is thromboembolism?P = 3			
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27.What is thromboembolism? $P = 3$		25. What is a tissue embolism?	
		26.What is a microbial embolism?	$\mathbf{P}=2$
28 Name the veins, the damage of which leads to the development of air embolism		27.What is thromboembolism?	P = 3
20.1 tune the venis, the dumage of which leads to the development of an embolism.		28.Name the veins, the damage of which leads to the development of air embol	ism.
a), b), c) P = 3		a), b), c)	P = 3
20 What are the most common causes of fat ambalian?		29.What are the most common causes of fat embolism?	
2.5 what are the most common causes of tall emponsiti /		2). That are the most common causes of fat emborishit.	

\mathbf{a} \mathbf{b} \mathbf{a}	P = 3	
a), b), c) 30.List the outcomes of fat embolism in the lungs.	$\Gamma = 3$	
a), b), c), d)	P = 4	
31. What develops in a patient as a result of embolism with tumor cells?	P = 1	
32. Give examples of the development of embolism by foreign bodies a), b)		
33. What develops in a patient as a result of microbial embolism?	P = 1	
34.Define DIC syndrome (thrombohemorrhagic syndrome).	$\mathbf{P} = \mathbf{I}$ $\mathbf{P} = 8$.	
35.List the etiological factors in the development of DIC syndrome.	$\Gamma = 0.$	
a), b), c), d), e), f)	P = 6.	
36.List the types of DIC syndrome by mechanisms of development. a), b),	1 = 0.	
c)	P = 3.	
37.List the stages of development of DIC syndrome a), b), c), d)	P = 4.	
38.Give a description of the I-stage DIC syndrome. a), b), c)	P = 4.	
39.What are the morphological compositions of blood clots formed in	г – 4.	
the vessels of microcirculation in DIC syndrome? a), b), c), d)	P = 4.	
40.Give a description of the II-stage DIC syndrome. a), b), c), d), e) .		
41. Give a description of the III stage of DIC syndrome. a), b), c), d), e) .	P = 4.	
42.Give a description of the IV-stage DIC syndrome. a), b), c), d)	P = 4.	
	r – 4.	
43.List the morphological changes in the lungs in DIC.	P = 5.	
a), b), c) d), e) 44 List the main changes in the kidneys in DIC	$\mathbf{P}=\mathbf{J}.$	
44.List the main changes in the kidneys in DIC.	P = 5.	
a), b), c), d), e)	$\mathbf{P}=\mathbf{J}.$	
45.List the main changes in the liver in DIC.	P = 4.	
a), b), c), d)	P = 4.	
46.List the main changes in the adrenal glands in DIC.	$\mathbf{D}=4$	
a), b), c), d)	$\mathbf{P}=4.$	
47. What pathological processes develop in the myocardium, brain	D 1	
with DIC syndrome? a), b), c), d)	$\mathbf{P}=4.$	
48. What pathological processes develop in the gastrointestinal tract	D 4	
with DIC syndrome? a), b), c), d)	P = 4.	
49.Name two types of blood clots in conditions of stagnation of blood: a), b) 50 Give a description of a subgriged thrombus:	P = 2	•
50. Give a description of a spherical thrombus:	D _ 2	
(a) How does this blood clot form?	P = 2. P = 1.	
b) Where is the globular thrombus localized?	$\mathbf{P} = \mathbf{I}.$	
51. What diseases, conditions cause marantic blood clots?		
Where do these blood clots form? a), b)	P = 6.	
52.How do tumor blood clots form?	P = 3.	
53. What are septic blood clots?	P = 5.	
54. In which vessels do red blood clots predominantly form?	P = 3.	
55. Where do white blood clots predominantly form?	P = 4.	
56.In which vessels are hyaline blood clots predominantly formed?	P = 1.	
57. Where do mixed blood clots form?	P = 3.	
58. What is hemostasis?	P = 5. P = 2.	
59. What is normal blood coagulation?		
60. Indicate the location of the first hematogenous metastases of osteosarco	na	D 1
of the hip.		P = 1
61 What type of embolism develops with decompression sickness?		P = 1 P = 2
62.What is metastasing?		P = 3 P = 2
63.What is metastas?		P = 3 P = 2
64.What types of embolism can metastasing exist?		P = 2
65.What is a dilatation thrombus?		P = 1
66.What are phleboliths?		P = 2

67.What does a white blood clot consist of?	P = 3
68. What does a red blood clot consist of?	P = 3
69.What does a hyaline blood clot consist of?	P = 4
70.What does a mixed blood clot consist of?	P = 4
71. What are the compositions of blood clots represented by parts of a mixed throm	bus:
a) the head? b) the body? c) the tail?	P = 3
72. What is a blood clot organization?	$\mathbf{P}=2$
73.What is aseptic autolysis?	P = 3
74.What is septic autolysis?	P =3

1. An elderly patient after gastrectomy for cancer, when trying to turn on his side, suddenly developed suffocation, cyanosis of the upper body. With the phenomena of acute heart failure, the patient died. At the autopsy in the trunk and the main branches of the pulmonary artery, thrombotic masses rolled into a ball, not soldered to the wall, were found. What pathological process are we talking about? Explain the mechanism of death of the patient.

P = 6

2 . A woman a few hours after childbirth had sudden death with acute right ventricular failure. At the autopsy, when the right ventricle was punctured under water, air bubbles were released from it, the blood in it turned out to be foamy. What pathological process are we talking about? What is its source?

$$\mathbf{P}=\mathbf{2}$$

3 . The patient, who suffered from sarcoma of the soft tissues of the left lower limb, died. At the autopsy, multiple rounded nodes of soft consistency were found in the lungs, on the section having the appearance of "fish meat". Name the process that is characterized by the development of these nodes. What process preceded their development? P = 24. A patient suffering from cancer of the duodenal sigma has died. At the autopsy, multiple liver metastases were found. Name the mechanism development of metastases. P = 1

5. The patient died after extraction of a carious tooth against the background of osteomyelitis of the upper jaw and phlegmon of the soft tissues of the face. At the autopsy, ulcers were found in many organs. What is the mechanism of development of these ulcers and how can these ulcers be called? Name the process as a whole, given its prevalence in Body? P = 3

6. A 50-year-old man suffered from prostate cancer. He died at home, suddenly. At the autopsy: in the trunk of the pulmonary artery, free-lying multiple thrombotic masses were found, completely obstructing the lumen. Name the cause of death and explain thanatogenesis (the mechanism of development of sudden death). P = 57. Surgical material was sent to the pathology department for biopsy: a section of the stomach with a tumor formation and lymph nodes of the omentum. Microscopically: gastric cancer (adenocarcinoma) diagnosed; in the lymph nodes - the growth of cancer cells. Name: a) changes in the lymph nodes, b) the general pathological process that led to these changes, c) the path of the spread of the tumor process. P = 3

8. The patient was admitted to the surgical department with a diagnosis: phlegmonous appendicitis, in connection with which an appendectomy was performed. However, after 8 days the patient died. At autopsy, multiple metastatic abscesses were found in the liver. Name: a) the general pathological process underlying the formation of purulent metastases in the liver, b) the path of the spread of the inflammatory process. (p = 2) Name the vessels (in Russian and Latin) through which there was a spread of the purulent process from the appendix to the liver. a) ..., b) ..., c) ..., d) (p = 8) P = 109. An 18-year-old patient hospitalized with fractures of both femurs and crushing of subcutaneous fat in this area developed acute pulmonary insufficiency on the second day, and then cerebral coma. The patient died. Histological examination of frozen sections in capillaries and arteriovenous shunts of the lungs, as well as in the vessels of the microcirculation of the brain, revealed discrete formations that give a positive color with Oil red and Sudan 3 dyes. Name: a) a fatal complication that has developed in a patient with fractures of long tubular bones, b) formation in the lumen of the vessels of the lungs and brain, c) two sources of these formations, d) two possible ways of their entry into the vessels of the brain, e) the causes of the development of cerebral coma. P = 7 10. In the deceased patient at the autopsy, the following were found: in the thoracic part of the aorta, a parietal thrombus measuring 4.0 cm x 1.5 cm with uneven edges in the tail area. The mouth of the superior mesenteric artery is obstructed by thromboembolism. The area of the small intestine (30 cm) is purple - black. A) Name the syndrome developed in the deceased patient. (p = 1) B) List other possible sources (localization of blood clots) for the development of this syndrome in a large circle of blood circulation: a) ..., b) ..., c) ..., d), e) ... (p=5) P = 6

11. A 27-year-old patient was admitted to the intensive care unit with a diagnosis of multiple

Combined trauma: fracture of the pelvic bones, chest, open fracture of the right femur. With increasing phenomena of multiple organ failure on the second day, the patient died. At the autopsy during histological examination, multiple hemorrhages in the lungs, liver, kidneys, adrenal glands were found; In the vessels of the microvasculature of the brain, lungs, liver - multiple fibrin blood clots. A) Name the syndrome that developed in the deceased, taking into account the development of multiple hemorrhages and blood clots (p = 1) B What are the synonyms for this syndrome? a) ..., b) (p=2) P = 3 12. A 62-year-old patient was admitted to the intensive care unit with a diagnosis: acute transmural myocardial infarction of the anterior wall of the left ventricle with a parietal thrombus. On the 2nd day, the patient was diagnosed: infarction of the spleen, left kidney.

Name the syndrome and explain the pathogenesis of lesions of the spleen and kidney.

P = 4

13. The patient died as a result of fulminant meningococcemia. At the autopsy found: in the brain - blood clots in the capillaries, multiple small hemorrhages; in the liver - hemorrhages, necrosis of hepatocytes, fibrin thrombi in the central veins; in the lungs - fibrin and hyaline blood clots in the capillaries; in the right adrenal gland - extensive hemorrhages

(a) Name the syndrome (complication) developed in the patient. (p=1)
b) List the stages of development of this syndrome. (p=4) P = 5

14. In a 32-year-old patient suffering from deep vein thrombophlebitis of the left lower leg, Suddenly, there were pains in the lateral parts of the chest, it became difficult breathing, cough and haemoptoe. Your diagnosis: a) the name of the syndrome, b) changes in the lungs, taking into account the clinical picture P = 2

MORPHOLOGY OF IRREVERSIBLE DAMAGE TO CELLS AND TISSUES. NECROSIS. APOPTOSIS

1.	Define necrosis.	P = 3.
2.	What is apoptosis?	P = 3.
3.	Is necrosis a pathological form of cell death?	P = 1
4.	Is apoptosis a physiological process of cell self-destruction? P=1	
5.	Is apoptosis accompanied by the development of an inflammatory reaction?	P = 1.

6.	Is necrosis accompanied by the development of an inflammatory reaction?	P = 1
7.	Give a classification of necrosis depending on the cause. a), b), c), d), e).	P = 5.
8.	List the clinical and morphological forms of necrosis. a), b), c), d), e).	P = 5
9.	List the types of necrosis depending on the mechanism of action of	
	the damaging factor. a), b).	P = 2
10.	How does direct performed develop?	P = 3
10. 11.	How does direct necrosis develop? How does indirect necrosis develop?	P = 3 P = 3
11.	List the stages of development of necrosis a), b), c), d).	P = 4
12.	What do paranecrosis characterize?	P = 2
13. 14.	What is necrobiosis?	P = 2
14.	What is autolysis?	P = 2
1 <i>5</i> . 16.	What research methods are used to identify early signs	$\mathbf{I} = \mathbf{L}$
10.	of necrosis?	P = 2.
17.	List the changes in the nucleus of the cell during the development of necrosis.	
	a), b), c).	P = 3
18.	List the changes in the cytoplasm of the cell during the development of necros	
	a), b), c).	P = 3.
19.	What is karyopyknosis?	$\mathbf{P}=2.$
20.	What is karyorhexis?	P = 2.
21.	What is karyolysis?	P = 2.
22.	What is plasma coagulation?	P = 3.
23.	What is pplasmorhexis?	P = 2.
24.	What is plasmolysis?	P = 2.
25.	The destruction of the cell membrane is a sign of irreversible damage?	$\mathbf{P} = 1$
26.	List the 2 main pathogenetic factors of membrane damage cells. a), b)	$\mathbf{P}=2$
27.	What morphological changes develop in the intercellular substance after	
	cell necrosis? a), b), c).	P = 3.
28.	List the successive stages of changes in fibrous structures during necrosis:	
	a), b), c).	P = 3.
29.	List the possible causes of necrosis:	
	a), b), c), d), e), f), g), h).	$\mathbf{P} = 8$
30.	What characterizes colliquation (wet) necrosis?	$\mathbf{P}=2.$
31.	What characterizes coagulation (dry) necrosis?	$\mathbf{P}=2.$
32.	In which tissues does colliquation (wet) predominantly develop?	
	necrosis?	$\mathbf{P}=2.$
33.	In which tissues does coagulation (dry) necrosis predominantly develop?	$\mathbf{P}=2.$
34.	List the organs in which colliquation most often occurs	D 0
25	necrosis. a), b).	$\mathbf{P}=2.$
35.	List the organs in which coagulation most often occurs $(a, b, b, c) = (a, b, c)$	D 7
26	necrosis. a), b) c) d) e) f) g).	P = 7 P = 4
36. 27	In what diseases does caseous necrosis develop? a), b), c), d).	P = 4. $P = 3$
37.	In what diseases does waxy (Zenker) necrosis develop? a), b), c).	$\mathbf{F} = \mathbf{J}$
38.	In what diseases does fibrinoid necrosis develop? a), b).	$\mathbf{P} = 2$
39.	What diseases cause fat necrosis? a), b), c).	P = 3
40.	What is sequestration?	P = 4
41.	Where does sequestration develop more often? a), b).	$\mathbf{P}=2$
42.	Define gangrenous necrosis.	P = 3
43.	Why are necrotic tissues black with gangrene?	P = 1
44.	List the morphological types of gangrene. a), b), c)	P = 3

45.	How does wet gangrene develop?	P = 4.
46.	In which organs does wet gangrene develop? a), b), c), d).	P = 4.
47.	What develops on the border of dry gangrene with healthy tissues?	P = 2.
48.	What is heterolysis?	P = 3
49.	List the causes of intestinal gangrene? a), b), c).	P = 3.
50.	In which case is a favorable outcome of wet gangrene of the intestinal	1 – 5.
50.		P = 1.
51	area possible?	Γ – Ι.
51.	What diseases in the lungs can develop wet gangrene?	
	a), b), c).	P = 3.
52.	What explains the rapid spread of wet gangrene? a), b)	P = 2.
53.	In which case is a favorable outcome of wet gangrene of the lower limb possible	e? $P = 1$
54.	What is noma, where is it observed?	P = 4.
55.	How does gas gangrene develop?	P = 2.
56.	What is the most common causative agent of gas gangrene?	P = 1.
57.	What is characteristic of gas gangrene? a), b).	P = 2.
58.	What is a bed sore?	P = 3
59.	On which parts of the body do pressure sores develop more often? a), b), c), d).	P = 4
60.	What is mummification?	P = 2
61.	What is mutilation?	P = 2
62.	What are the types of ischemia according to the duration of development. a), b)	
02.	that are the types of isenenna according to the datation of development. a), b)	. 1 – 2
63.	What are the possible outcomes of acute ischemia. a), b).	P = 2
64.	Do you mean acute ischemia is a reversible pathological process or not?	P = 1
65.	Why are lung and liver tissues relatively resistant to ischemia?	$\mathbf{P}=2$
66.	Due to what can there be compensation for blood supply during obturation	1 – 2
00.	main artery?	$\mathbf{P} = 2$
67	•	1 - 2
67.	What are the organs in which collateral blood supply is relatively poorly	D 4
60	developed? a), b), c), d).	$\mathbf{P} = 4$
68.	Name the consistency of necrotic tissue a) with dry gangrene,	-
	b) with wet gangrene	$\mathbf{P}=2$
69.	Define myocardial infarction.	P = 3
70.	What is the name of necrosis, accompanied by compaction and dehydration	
	ткани.	$\mathbf{P} = 1$
71.	List the common causes of heart attack. a), b), c), d).	$\mathbf{P} = 4$
72.	Name the organs in which heart attacks most often develop. a), b), c), d).e) e)	P = 6
73.	Name the types of heart attack on the macroscopic picture. a), b), c).	P = 3
74.	List the organs whose heart attack may be hemorrhagic.	
	a), b), c), d).	P = 4
75.	Name the organs in which ischemic infarction develops. a), b), c).	P = 3
76.	In which organs does ischemic infarction develop with hemorrhagic crown?	P = 2
70.	in which organs does is chemic indiction develop which hemoninagie crown.	1 – 2
77.	What features of the blood supply to the lungs determine the development of	
	hemorrhagic infarction in them? a), b).	$\mathbf{P} = 4$
78.	What are the stages of development of a heart attack. a), b), c).	P = 3
70.		1 0
79.	Name the diseases against which patients often develop myocardial infarction	
	a), b), c).	P = 3
80.	List the types of myocardial infarction depending on the lesion of the layers	
50.	of the myocardium. a), b), c), d).	P = 4
81.	What is formed (often) in the area of a heart attack on the endocardium?	P = 1
82.		P = 1 P = 1
o∠.	What develops on the pericardium in the area of a heart attack?	Γ – Ι

83.	What does myocardial infarction look like macroscopically (from 3 days)?	P = 6
84.	What is the name of the inflammation that develops around dead tissue?	P = 1
85.	What is represented (morphologically) by the zone of demarcation	
	inflammation around necrosis? a), b), c).	P = 3
86.	On what day from the onset of myocardial infarction begins to develop	
	Granulative tissue?	P = 1
87.	List the possible outcomes of necrosis. a), b), c), d), e).	P = 5
88.	What is the favorable outcome of myocardial infarction.	P = 1
89.	What is the favorable outcome of ischemic cerebral infarction.	P = 1
90.	List the possible outcomes of a heart attack in the lungs. a), b).	P = 2
91.	Name the favorable outcome of a heart attack: a) kidneys, b) spleen.	P = 2
92.	In which organs do heart attacks have a wedge-shaped shape? a), b), c).	P = 3
93.	Describe how macroscopically a pulmonary infarction looks: a) consistency,	
	c) color, d) attitude to the pleura.	$\mathbf{P} = 4$
94.	What is the name of necrosis, accompanied by enzymatic softening	
	and melting of tissue?	P = 1
95.	What is the geometric shape of myocardial infarction?	P = 1
96.	How does the volume of necrotic tissue change in dry gangrene?	P = 1
97.	How does the volume of necrotic tissue change in wet gangrene?	P = 1

- A 67-year-old woman suffered from atherosclerosis of the aorta and its main branches, was admitted to the surgical department with a symptom of an acute abdomen. In case of emergency laparotomy, loops of the small intestine of purple-black color were found, the serous membrane was dull, with fibrin overlays. (a) What is bowel damage called? b) What is the most likely cause of intestinal damage? P=3
- 2. At the autopsy, the patient was found: a blood clot in the abdominal aorta 6.0 cm long; in the lumen of the superior mesenteric artery thromboembolism. The wall of the small intestine for 45 cm is purple black. a) Name the process that has developed in the intestine; b) What was (what syndrome) caused the development of pathology in the intestine? P=3
- 3. The patient was admitted to the intensive care unit with a diagnosis: acute transmural myocardial infarction of the anterior wall of the left ventricle. On the 5th day he died. At the autopsy: in the area of the infarction, a slit-like rupture of the heart wall, and in the heart shirt 300 ml. liquid blood. a) What led to the rupture of the heart wall in the area of the infarction? b) Name the process in the heart shirt. P = 2
- 4. A patient with tuberculous spondylitis died of kidney failure. At the autopsy on the cut of the spinal column in the vertebral bodies there are structureless, dry, crumbling foci of yellow-white color; Some of them are in a state of melting. Name the process in the vertebrae, taking into account its clinical and anatomical shape and its variety. P = 2
- 5. The patient died suddenly. At the autopsy: the lumen of the left middle cerebral artery is closed by a thrombus; In the parietal-temporal region of the left hemisphere of the brain there is a focus of destruction of a mushy consistency of whitish-gray color, measuring $5.0 \ge 6.0 \text{ cm}$. Name the process. P = 2
- 6. In a patient with a fracture of the spine and paralysis of both lower limbs on the skin in the sacrum, a pink, and later purple-black area appeared. Name the changes.

7. A 65-year-old patient suffering from atherosclerosis had pain in the right lower limb. The soft tissues of the toes became swollen, black, with an unpleasant odor and exfoliated epidermis. Name the process according to its variety. P = 2

 $\mathbf{P} = 1$

- 8. A patient with coronary heart disease died of heart failure. At the autopsy in the anterior wall of the left ventricle of the heart, large foci of scar tissue were found, capturing the entire thickness of the myocardium. What process preceded the development of the described changes? Name it based on prevalence in the myocardium. P = 2
- 9. A lymph node measuring 2.5 cm x 1.5 cm x 2.0 cm, of a dense consistency, yellowishgray in the section, cheesy in appearance, was delivered to the pathology department for biopsy. Name the process, taking into account its clinical and morphological form.

$$\mathbf{P}=2.$$

- 10. At the autopsy of an 87-year-old patient suffering from cerebral atherosclerosis, a cavity was found in the left temporal lobe, measuring 3.5 cm x 3.0 cm x 4.5 cm, the walls of the cavity are even, smooth, white, and the lumen of the cavity contains a clear yellowish liquid. a) name this formation in the brain, b) the outcome of which acute process, apparently, is the described formation of the brain? P = 2.
- 11. At the autopsy of the patient in the tissue of the right lung there are areas of triangular shape, located sub pleural, dense consistency, dark red color. Name this process in the lungs, taking into account the macroscopic appearance. P = 2.
- 12. A vermiform appendix measuring 12.0x3.0 cm, purple-black in color, was delivered for biopsy. Name the type of appendicitis, taking into account the type of necrosis that has developed in the appendix. P = 1.
- 13. At the autopsy, the patient in both kidneys has triangular areas, the base facing the capsule, bloodless, grayish-brownish in color, surrounded by a hemorrhagic corolla. Name the pathological process in the kidneys, taking into account its macroscopic appearance. P = 2.
- 14. An amputated right lower limb at the level of the lower third of the thigh was delivered for biopsy. The tissues of the limb are reduced in volume, dry, black. Name the disease. P = 2.

15. At the autopsy of an 84-year-old patient in the lateral and posterior walls of the left ventricle in all layers of the myocardium there is an irregularly shaped area, measuring 7.0 x 6.0 x 1.5 cm, variegated, with foci of pale gray, yellowish, dark red, delimited from a healthy myocardium by a dark red corolla. Name this disease, taking into account the defeat of all layers of the myocardium. P = 2.

MORPHOLOGY OF CELLULAR AND TISSUE METABOLISM DISORDERS. PARENCHYMAL DYSTROPHIES.

- What is meant by tissue trophism? (from the Greek trophe eating) P = 4
 What mechanisms provide trophism? a) ... b) ... P = 2
 What are the main factors that cause a disorder of cell autoregulation?

 (a)... b) ... c)... d) ... P = 4

 What does the violation of the autoregulation of the cell lead to? a) ... b) ... P = 2
- 5. What are the main pathogenetic links, in violation of the cellular

	mechanisms of trophism? a) b)	P = 2
6.	What is the leading cause in the pathogenesis of discirculatory dystrophi	es? P=1
7.	Define infiltration as one of the mechanisms of dystrophies.	P = 3
8.	Define decomposition (phanerosis) as one of the mechanisms	
	dystrophies.	P = 2
9.	Define transformation as one of the mechanisms of dystrophies.	P = 3
10.	Define perverted synthesis as one of the mechanisms of dystrophies.	P = 3
11.	What are the leading morphogenetic mechanisms of dystrophies: a) b)	$\mathbf{P}=2$
12.	Specify the principles of classification of dystrophies: a) b) c) d)	P = 4
13.	List dystrophies depending on the predominance of morphological	
	changes in the parenchyma or stroma and in the vascular wall: a) b) c)	P = 3
14.		
	type of exchange. a) b) c) d)	$\mathbf{P} = 4$
15	List dustrophies depending on the influence of constitutions a) h	D – 2
15.	List dystrophies depending on the influence of genetic factors. a) b)	P = 2
16.	List dystrophies according to prevalence. a) b)	P = 2
17.	Bring the morphogenetic chain of parenchymal disproteinosis with	
	the outcome in coagulation total necrosis.	P = 3
18.	Bring the morphogenetic chain of parenchymal disproteinosis with	
	the outcome in colliquation total necrosis of the cell.	P = 3
19.		P = 4
20.		P = 6
21.		P = 3
22.	1	
	a) b) c) d)	P = 4
	23. The most common causes of hydropic dystrophy are.	
	a) b) c)	$\mathbf{P}=3$
	24 List the main causes of parenchymal disproteinosis.	D 5
25	a) b) c) d) e) In the colle of which organs hydronic dystrophy is often cheeryod	P = 5
25.		P = 3
	a) b) c)26. Name the successive stages of cell damage with continued exposure to	$\Gamma = 3$
	the pathogenetic factor. a) b) c) d)	P = 4
	27. What substances accumulate intracellularly in dystrophies.	P = 5
	28. In cells, lipids can accumulate in the form of. a) b) c)	P = 3
	29. In which organs can steatosis develop: a) b)	P = 2
	30. In what diseases is hepatic steatosis observed?	1 - 2
	a) b) c) d)	$\mathbf{P} = 4$
31.	In what composition lipids accumulate in hepatocytes and what color can	
	be used to identify these compounds. a) b)	P = 2
32.		
	c) size, d) view on the section, e) figurative name.	P = 5
	33. Causes of myocardial fatty degeneration: a) b)	P = 2
	34. What are the histological features of myocardial fatty degeneration.	
	(a) b)	P = 2
	35. Describe the appearance of the heart with fatty degeneration:	
	a) the size of the chambers, b) consistency, c) figurative name.	P = 3
	36. Clinical manifestation of myocardial fatty degeneration.	$\mathbf{P} = 1$
	37. What clinical symptom develops with protein dystrophy of the kidney	P = 1

 38. What is the typical adverse outcome of balloon cell dystrophy. 39. What is the name of hereditary storage diseases. 40. List the outcomes of fatty degeneration. a) b) 41. List the main systemic lipidosis, taking into account the type of 	P = 1 $P = 1$ $P = 2$
 lipids accumulating in the cells. a) b) c) 42. List what determines the morphological manifestations of the cell's response to the effects of a pathogenic factor. a) b) 43. What are some examples of cells that have high a) medium; b, c, d) 	P = 6 $P = 4$
and low; (d, e, f) sensitivity to ischemia (hypoxia).	$\mathbf{P}=7$
44. What are the manifestations of violations of the glycogen content in the tissues.45. In what diagona are the most pronounced violations of the glycogen	P = 2
45. In what diseases are the most pronounced violations of the glycogen content in the tissues?46. Why does fatty liver develop in diabetes mellitus?	P = 2 $P = 3$
47. What changes in the nuclei of hepatocytes are observed in fatty degeneration the liver?	P = 2
48. What microscopic changes in the tubules of the kidneys in	1 – 2
diabetes mellitus are associated with glucosuria.	P = 3
49. Indicate the morphological features of damage to cardiomyocytes in moderate and deep hypoxia: a) b)?50. What are the names of hereditary carbohydrate dystrophies associated	P = 2
with impaired glycogen metabolism. Give synonyms. 51. Indicate how long it takes for irreversible ischemic damage to	P = 2
cardiomyocytes (a) to occur, and when can they be diagnosis using conventional light microscopy (b)?	P = 2
52. What is mucous dystrophy?	$\mathbf{P} = 4$
53. What microscopic changes are observed in mucous dystrophy.	P = 3
54. What is the common cause of mucous dystrophy.	P = 1
55. What are the main morphological signs of cell damage by viruses: a) b) c) d)	P = 4
56. List the cells in which hydropic dystrophy is often observed a) b). c) d) e). f)	P = 6
57. Name and reveal the essence of the two main mechanisms of the appearance of fatty inclusions in cardiomyocytes during hypoxia.	P = 6
58. Give examples of cell damage caused by free radicals. $P = 5$ 59. What substances accumulate in the intima of the aorta in atherosclerosis	
	– .
60. What are the clinical symptoms and what type of nephrocyte dystrophy protein reabsorption in the kidney is impaired?	develops when $P = 2$
61. What is alteration?	P = 3
62. What are general pathological processes alterative? a) b)63. What are the main pathogenetic mechanisms of irreversible cell change	P = 2
during hypoxia?	P = 4
TASKS	

1. In a child of one of the countries of Central Africa as a result of protein starvation. There was a lag in development, loss of hair pigment, frequent gastrointestinal and pulmonary infections, an increase in the size of the abdomen due to the accumulation of fluid, an enlarged liver, signs of multiple organ failure, which served as the immediate cause of death. Name the disease (p = 1) Name the changes in the liver found at the autopsy (p = 2) Explain the pathogenesis of these changes and the outcome (p = 3)

$$P = 6$$

2.Microscopy of the organs of the deceased from anemia after staining with Sudan-3 revealed small-drop obesity of cardiomyocytes, significant small- and large-droplet obesity of hepatocytes in the center of the hepatic lobules. Describe the violation in these organs and give them a macroscopic figurative name. P = 4

3. The patient suffered from the underlying disease - chronic myeloid leukemia with severe anemia. Recently, there have been signs of progressive heart failure, against which pneumonia developed, which was the direct cause of death. A postmortem examination revealed changes in the heart characteristic of the terminal period of this disease. a) give the figurative name of the heart in this pathology, (p = 1)

b) describe the macroscopic changes in the heart, on the basis of which it was given Such a name, (p = 1) c) describe the microscopic changes in the myocardium in these areas, (p = 1) d) name the two mechanisms of dystrophy leading to such changes, (p = 2) e) name the additional histological staining for verification (p = 1) f) name the main stages of the pathogenesis of the development of changes in certain parts of the myocardium in chronic myeloid leukemia (p = 5) P = 11

4. The man died of chronic alcohol intoxication. At the autopsy, an enlarged liver of a testy consistency was found, yellow on the incision. When coloring paraffin sections hematoxylin and eosin, in the cytoplasm of hepatocytes revealed optic empty vacuoles of various sizes. What general pathological process is observed in hepatocytes? P=2

5. A man suffering from cirrhosis of the liver in the stage of decompensation, died of acute posthemorrhagic anemia. At the autopsy in the heart from the endocardium, striation is visible, due to the alternation of lighter and darker muscle fibers. Histochemical examination revealed lipids in the cytoplasm of cardiomyocytes. What is the figurative name of the heart in this case? Name the general pathological process for changes in cardiomyocytes. What mechanisms were involved in this process? What dye should be used to detect lipids? P = 6

- 6. A woman has a whitish area in the mucous membrane of her mouth. In the study of biopsy material, it was found that there is pathological keratinization in these areas of the mucous membrane. What general pathological process occurs in the mucous membrane of the mouth? What is the name of pathological keratinization of the mucous membranes? What is the possible unfavorable outcome? P = 3
- 7. From the medical history it became known that the man suffered from chronic alcoholism. Microscopic examination revealed hyaline-like bodies in hepatocytes. What general pathological process are we talking about? What are these bodies called by the author? What mechanism led to these changes? P=3
- 8. The man showed signs of renal failure after poisoning with antifreeze. Microscopic examination of the kidney biopsy specimen revealed a large number of vacuoles in the epithelial cells of the proximal tubules. What process are we talking about? What color of the micro preparation should be produced in order to exclude the presence of other substances in the vacuoles? P = 3

- 9. The following changes were found in the deceased from anemia at the autopsy: the heart is flabby, enlarged, the cavities are expanded, and a yellow-white striation is visible from the endocardium. What do you call these heart changes? With what coloring of histological specimens can this be identified? Give the figurative name of this process in the heart according to the macroscopic picture. What is the main pathogenetic factor for the development of this process? P = 4
- 10. At the autopsy of the deceased from thiophos poisoning (organophosphorus compound), the following changes in the liver were found: it is enlarged, flabby, clayey-yellow. Name these changes. Give a figurative name to the liver in this pathology. P = 2
- 11. After hypothermia, a woman suddenly had chills, herpetic eruptions on her lips. Microscopically, large vacuoles are found in most epidermal cells with the nucleus dropping to the periphery. What general pathological process in the epidermis are we talking about? What is the possible outcome? P = 3

MORPHOLOGY OF PROTEIN, LIPID AND CARBOHYDRATE DISORDERS EXCHANGE. STROMAL-VASCULAR DYSTROPHIES

 a) b) c) P = 3 2. List stromal-vascular disproteinosis: a) b) c) d) P = 4 3. Give the morphological chain of stromal-vascular disproteinosis P = 3 4. What is mucoid swelling? P = 5 5. What histological methods are used for staining: a) collagen fibers b) elastic fibers c) reticular fibers b) elastic fibers c) reticular fibers c) metachromasia? P = 2 7. An infiltrate of which cells can accompany changes in the main substance and collagen fibers? P = 3 8. In which organs is mucoid swelling most common?: a) b) c) d) P = 4 9. What are the reasons for the development of mucoid swelling: a) b) c) P = 3
 Give the morphological chain of stromal-vascular disproteinosis P = 3 What is mucoid swelling? P = 5 What histological methods are used for staining: a) collagen fibers b) elastic fibers c) reticular fibers P = 3 What is metachromasia? P = 2 An infiltrate of which cells can accompany changes in the main substance and collagen fibers? P = 3 In which organs is mucoid swelling most common?: a) b) c) d) What are the reasons for the development of mucoid swelling:
 4. What is mucoid swelling? P = 5 5. What histological methods are used for staining: a) collagen fibers b) elastic fibers c) reticular fibers P = 3 6. What is metachromasia? P = 2 7. An infiltrate of which cells can accompany changes in the main substance and collagen fibers? P = 3 8. In which organs is mucoid swelling most common?: a) b) c) d) P = 4 9. What are the reasons for the development of mucoid swelling:
 5. What histological methods are used for staining: a) collagen fibers b) elastic fibers c) reticular fibers P = 3 6. What is metachromasia? P = 2 7. An infiltrate of which cells can accompany changes in the main substance and collagen fibers? P = 3 8. In which organs is mucoid swelling most common?: a) b) c) d) P = 4 9. What are the reasons for the development of mucoid swelling:
 b) elastic fibers c) reticular fibers b) elastic fibers c) reticular fibers b) elastic fibers c) reticular fibers c) an infiltrate of which cells can accompany changes in the main substance and collagen fibers? b) elastic fibers c) P = 3 c) and collagen fibers? c) and collagen fibers (and collagen fibers) b) and collagen fibers (b) and collagen fibers) c) and collagen fibers (c) and collagen fibers) c) and collagen fibers (c) and collagen fibers) c) and collagen fibers (c) and collagen fibers) b) and collagen fibers (c) and collagen fibers) c) and collagen fibers (c) and collagen fibers (c) and collagen fibers) c) and collagen fibers (c) and collagen fibers (c) and collagen fibers) d) and collagen fibers (c) and collagen fibers (c) and collagen fibers) d) and collagen fibers (c) and collagen fibers (c)
 An infiltrate of which cells can accompany changes in the main substance and collagen fibers? P = 3 In which organs is mucoid swelling most common?: a) b) c) d) P = 4 What are the reasons for the development of mucoid swelling:
substance and collagen fibers? $P = 3$ 8.In which organs is mucoid swelling most common?: a) b) c) d) $P = 4$ 9.What are the reasons for the development of mucoid swelling:
 8. In which organs is mucoid swelling most common?: a) b) c) d) P = 4 9. What are the reasons for the development of mucoid swelling:
 c) d) P = 4 9. What are the reasons for the development of mucoid swelling:
9. What are the reasons for the development of mucoid swelling:
· · ·
a) b) c) $P = 3$
10. List the outcomes of mucoid swelling: a) b) $P = 2$
11. List the changes in tinctorial properties in the foci of
mucoid swelling with stains: a) hematoxylin and eosin,
b) picrofuxin, c) toluidine blue $P = 3$
12. What dye is used to detect mucoid swelling? $P = 1$
13. What connective tissue structures change with mucoid
swelling? $P = 1$
14. List the main processes in the development of fibrinoid swelling:
a) b) c) $P = 3$
15. What is fibrinoid swelling? $P = 5$
16.Indicate the most common localization of fibrinoid swelling:
a) b) c)
17.List the causes of fibrinoid swelling: a) b) c) d) $P = 4$
18. List the outcomes of fibrinoid changes: a) b) c) $P = 3$
19.What is hyalinosis? $P = 3$
20.In which tissue structures hyalinosis most often develops: a) b) c) $P = 3$
21.List the possible outcomes of hyalinosis: a) b) c) d) $P = 4$
22.At the end of what processes hyalinosis can develop: a), b) c) d) e) $P = 5$

23. Give a classification of hyalinosis: a) b)	$\mathbf{P}=2$
24.In which layers of the vascular wall hyaline is found and what does	it
lead to: a) b)	P = 3
25. In which organs hyalinosis of small arteries and arterioles is most p	ronounced:
a) b) c) d) e)	P = 5
26. What diseases lead to the development of hyalinosis: a) b) c)	P = 3
27. Name the varieties of vascular hyaline: a) b) c)	P = 3
28. As a result of what changes does hyalinosis of the connective tissue	
itself develop: (a) b) c)	P = 3
29. Give examples of hyalinosis as an outcome of local sclerosis:	
(a) b) c) d) e) f)	P = 6
30. What macroscopic changes in organs are observed in:	
a) hyalinosis of small arteries and arterioles	
b) hyalinosis of one's own connective tissue	$\mathbf{P}=2$
31. What is amyloidosis?	$\mathbf{P} = 4$
32 List the components of amyloid: a) b)	$\mathbf{P}=2$
33 What dyes can be used to detect amyloid in tissues: a) b)	P = 2
34 Specify the principles of classification of amyloidosis: a) b) c)	
d)	$\mathbf{P} = 4$
35 Give a classification of amyloidosis depending on the causes:	D (
a) b) c) d)	$\mathbf{P}=4$
36. List the diseases, the complication of which may be amyloidosis:	
a) b) c) d) e)	P = 5
37. What organs are often affected by senile amyloidosis: a) b) c)	d) $.P = 4$
38.List the organs in which amyloid is most often deposited in	D 5
acquired (secondary) amyloidosis.	P=5
39. What types of amyloidosis are isolated, depending on the specifics of the amyloid fibril protoint a , b , b , d , d	P = 4
the amyloid fibril protein: a) b) c) d) 40.When AL-amyloidosis can be observed and in what diseases: a) b)	
40. when AL-amyloidosis can be observed and in what diseases. a) b) 41. In what cases can AA amyloidosis be observed: a) b)	P = 2
42.What is affected by AF amyloidosis?	P = 1
43.What is affected by ASC amyloidosis?	P = 2
44. Given the prevalence of amyloidosis, its varieties are distinguished:	$\mathbf{I} = \mathbf{Z}$
a) b)	$\mathbf{P} = 2$
45. What types of amyloidosis are distinguished, taking into account clin	
manifestations: (a) b) c) d) e) f) g)	P=7
46. Which cells produce amyloid fibril protein in generalized forms of a	
a) b) c)	P = 3
47. Which cells produce amyloid fibril protein in local forms of amyloid	osis:
a) b) c) d) e) f)	P = 6
48. What are the main provisions of the theory of disproteinosis, explaining the	pathogenesis
of amyloidosis?	P = 4
49. What is the content of the immunological theory explaining the pathogenesis	1 •
of amyloidosis?	P = 3
50. What is the theory of "cellular local synthesis"?	P = 3
51. What is the mutation theory of amyloidosis?	$\mathbf{P} = 3$
52.What is called obesity?	P = 3
53.List the possible causes of death of patients with secondary amyloidosis:	
a) b) c)	$\mathbf{P}=3$
54.In which structures of the kidney amyloid is deposited: a) b) c) d)	$\mathbf{P}=4$
55.In which structures of the liver amyloid is deposited: a) b) c) d) e)	. P = 5

56. What are the two main variants of figurative terms for amyloidosis of the spleen:	
a) b)	P = 2
57.In what structures of the heart amyloid is deposited: a) b) c) d)	$\mathbf{P} = 4$
58.In which structures of the intestine amyloid is deposited: a) b) c)	P = 3
59.In what structures of the brain amyloid is deposited: a) b) c)	P = 3
60.In which structures of the pancreas amyloid is deposited: a) b)	P = 2
61.Specify the functional significance of amyloidosis depending on the degree of	
development: a) b) c)	P = 3
62. What changes can occur in the heart with obesity?	P = 3
63.Where fat is deposited with obesity: a) b) c) d) e) (e) g)	P = 7
64.Name the forms of obesity according to the etiological principle.	$\mathbf{P} = 2$
65. What types of obesity are distinguished by external manifestations: a) b) c) c	1)P = 4
66.Indicate the degree of obesity, depending on the excess body weight of the patient:	
a) b) c) d)	P =4
67. What variants of general obesity are distinguished depending on the morphological	
changes in adipose tissue: a) b)	P = 2
68. What is the antipode of general obesity?	P = 2
69.What is lipomatosis?	P = 2
70. Abnormality of predominantly what kind of metabolism is observed with	
Atherosclerosis?	P = 2
71.Name stromal-vascular dystrophy associated with impaired glycoprotein metabolism	n P = 1

- 1. A patient with fibro-cavernous pulmonary tuberculosis died of uremia. Dissemination of the tuberculosis process in the kidneys at autopsy was not detected. Explain the possibility of developing uremia in a patient? P = 4
- 2. The patient suffered from rheumatic mitral valve disease. Death was due to chronic cardiovascular failure. At the autopsy: the mitral valve leaflets are thickened, fused, dense, cartilaginous, milky white. (a) As a result of what dystrophy do the valve leaflets look like this?
 b) as a result of what two successive changes (stages of the process) in the valve apparatus this dystrophy developed? c) At what stage was the process reversible? P = 4
- 3. The patient died of cancer. On the section, it was found: the thickness of the subcutaneous tissue is reduced, the adipose tissue is flabby, gelatinous, and ochre-yellow in color, microscopically the fat cells of the fat depots are collapsed, wrinkled. What kind of lipid metabolism disorder are we talking about? P = 2
- 4. At autopsy, a systemic disease was diagnosed. In the liver, lungs, spleen and adrenal glands, deposits of an eosinophilic extracellular substance that was stained with Congo red have been revealed. Name the general pathological process. Specify macroscopic changes in organs in size, consistency, appearance on the incision. Give the figurative names of the kidneys, liver, spleen in this process. P = 8
- 5. Microscopic examination of the atherosclerotic plaque of the aorta showed that its tissue under conditions of hypoxia has increased basophile, and when stained with toluidine blue, it acquires a lilac-red color. How can the described phenomenon be named, defined and explained? P = 5
- 6. Microscopic examination of the mitral valve leaflets of a patient who died because of exacerbation of rheumatism showed that they contain areas of homogenization and destruction

of collagen fibers, which have increased eosinophilia and give a positive reaction to fibrin. Metachromasia in toluidine blue staining is not verify. Name pathological process P=2

- 7. The patient suffered peritonitis. At the autopsy, there was found the capsule of the spleen and liver is sharply thickened and has a whitish translucent appearance. What pathological process are we talking about? What are the figurative names of the spleen and liver? P = 3
- 8. After a severe burn of the flexion surface of the elbow joint, the patient developed a scar of cartilaginous consistency, sharply restricting movement. Excision of the scar was performed. On the incision, the scar tissue has a whitish translucent appearance. Microscopically the fibers of the scar are sharply thickened, homogeneous in appearance, the number of fibrocytes is insignificant. What kind of process are we talking about? What is the name of the type of skin scar? P = 2
- 9. A 50-year-old man suffered from bronchiectatic disease for many years. Recently, edema, proteinuria, hypoproteinemia, hyperlipidemia have appeared. What process in the kidneys complicated the course of bronchiectasis? Name the four structures of the kidneys that are affected by this process. What coloration of kidney tissues is necessary for process verification? P=7
- 10. The patient suffered from rheumatoid arthritis for many years. Recently, there have been increasing proteinuria, edema. A kidney biopsy is prescribed. What general pathological process in the kidneys that complicated the course of rheumatoid arthritis was complicated? What are the four structures of the kidneys that damaged by this process? What are three additional research methods needed to verify the process? P = 8
- 11. A 50-year-old man (height 180 cm, body weight 110 kg) complains of shortness of breath, a feeling of heaviness in the right hypo chondrium. Determine the degree of obesity and indicate possible changes in the organs of the patient. P = 3
- 12. Microscopic examination of the cerebral vessels of a patient who died from hemorrhage in the subcortical nodes (clinically diagnosed hypertension) found that their wall is sharply thickened, homogeneous, and eosinophilic. The number of cellular elements in the vessel wall is extremely small. How can the development of the described phenomenon be called and explained? P = 3
- 13. Patient 65 y. o. with III degree of Obesity suffered from diabetes mellitus and died of ischemic cerebral infarction. At the autopsy, the liver is flabby, enlarged in size, on the incision yellow. In the pancreas, microscopically in the islets of Langerhans, homogeneous pink masses. What general pathological processes occurred in the liver and pancreas? What are the verification methods must be applied? P=4
- 14. At the autopsy of a patient suffering from rheumatism, there were found: the heart was enlarged (heart weight 380 g), the mitral valve leaflets were evenly thickened, translucent, whitish in color, spliced. The left atrioventricular foramen is narrowed. What is the general pathological process that corresponds to these changes in the heart? What is your diagnosis? P = 5

MORPHOLOGY OF CHROMOPROTEIN AND NUCLEOPROTEIN METABOLISM DISORDERS. VIOLATION OF MINERAL METABOLISM. PATHOLOGICAL CALCIFICATION. FORMATION OF STONES.

1. Endogenous pigments are usually divided into a) ... b) ... c) ... P = 3

2. What pigments are formed because of the physiological breakdown of red blood cells?	2
(a) b) c)	P = 3
3. What pigments, in addition to those formed normally, are formed both intra	
and extravascular in pathological conditions a) b} c)	P = 3
4. List iron-containing hemoglobinogenic pigments.	P = 3
5. What is the definition of hemosiderosis?	P = 3
6. What is the definition of hemochromatosis?	P = 3
7. List the diseases in which there is a general hemosiderosis. (a) b) c) d) e)	P = 5
8. Which cells in conditions of severe hemolysis of erythrocytes can become sideroblasts	
a) b) c) d) e)	P = 5
9. Which organs with general hemosiderosis acquire a rusty-brown color?	
a) b) c) d)	$\mathbf{P} = 4$
10. What is the primary hemochromatosis?	$\mathbf{P} = 4$
11. In which organs hemosiderosis predominantly develops in hemochromatosis	
a) b) c) d) e)f) g) h)	P = 8
12. Specify the causes of secondary hemochromatosis a) b) c) d) e)	P = 5
13. What hemoglobinogenic pigments are formed in the old hematoma? (a) b)	P = 2
14. List hemoglobinogenic pigments a) b) in $\}$ d) e) (e) g) h)	P = 8
15. List the characteristic properties of hemosiderin a) physical condition b) color	
c) site of formation d) the presence of iron e) the timing of formation	P = 5
16. List the types of jaundice according to the mechanism of development. (a) b) c)	P = 3
17. List the diseases in which mechanical (sub hepatic) jaundice may develop)	
a) b) c) d)	P = 4
18. List the diseases in which hepatic (parenchymatous) jaundice can develop a) b)	
c) d)	P = 4
19. List the diseases in which supra hepatic (hemolytic) jaundice can develop a) b)	_
c) d)	$\mathbf{P}=4$
20. Name protein genic (tyrosinogenic) pigments a) b) c)	P = 3
21. In which organs are melanoblasts localized. (a) b) c) d) e)	P = 5
22. What are the cells that synthesize melanin called and what is their origin a) b)	$\mathbf{P}=2$
23. What hormones and biologically active substances stimulate the synthesis of melanin	_
a) b) c)	$\mathbf{P}=3$
24. What explains renal failure in sub hepatic (mechanical) jaundice?	$\mathbf{P}=2$
25. What explains the hyperpigmentation of the skin in Addison's disease?	P = 9
26. Indicate the possible causes of Addison's disease.	$\mathbf{P} = 5$
27. In what diseases does local acquired melanosis occur? (a) b)	P = 5
28. In what cases is there an increased focal formation of melanin? (a) b)	$\mathbf{P}=2$
29. What is albinism and what is the essence of this melanin metabolism disorder?	$\mathbf{P}=2$
30. What are the main symptoms of the disease associated with dysregulation of melanin	
formation: a), b), c), d)	$\mathbf{P} = 4$
31.List lipid pigments. a) b) c) d)	P = 4
	D (
32. In what cases does secondary lipofuscinosis develop? a) b) c) d)	P = 4
33. In which tissues are uric acid salts deposited in gout? a) b) c) d) e)	P = 5
34. What is the microscopic structure of a gouty lump?	P = 4
35. List what is affected by Ca $++$ ions in the body? a) b) c) d) e)	P = 5
36. List the forms of calcification a) b) c)	$\mathbf{P}=3$
37. Name the most frequent localization of calcifications by type of	D 7
calcareous metastases a) b) c) d) e)	P = 5
38. Give examples of dystrophic calcification a) \dots b) \dots c) \dots d) \dots e) \dots (e) \dots g)	P = 7
39. Specify what can be the chemical composition of gallstones. a) b) c)	P = 5
40. Specify what can be the composition of urinary stones a) b) c) d) e)	P = 5

41. List the diseases and conditions leading to hypercalcemia a) b) c) d)	
e) f)	P = 6
42. Give examples of petrification of the arterial wall related to local dystrophic	
calcification a) b)	P = 2
43. Indicate which stones are formed during the acidic reaction of urine a) b)	P = 2
44. Name the variants of hemosiderosis by prevalence a) b)	P = 2
45. In which organs is hemosiderin deposited during intravascular hemolysis of	
erythrocytes? a) b) c) d)	$\mathbf{P} = 4$
46. What is jaundice?	P = 4
47. Name the group of diseases in which parenchymal jaundice can develop.	P = 3
48. What extrahepatic changes develop with obstructive jaundice. a) b) c) c	d). P = 4
49. List the manifestations of the violation of the exchange of nucleoprotein a)	
c)	P = 3
50. What changes develop with gout a) b) c) d)	$\mathbf{P} = 4$
51. What local factors are important in stone formation?	P = 3
52. Give the clinical significance of stone formation a) b) c)	P = 3
53. Give examples of melanin metabolism disorders a) b) c) d)	$\mathbf{P} = 4$
54. Name the types of hyper bilirubinemia in jaundice a) b)	P = 2
55. In what types of jaundice is non-conjugated hyperbilirubinemia observed?	
a) b)	$\mathbf{P} = 2$
56. What does jaundice conjugated hyperbilirubinemia includes?	P = 1
57. When does local hemosiderosis occur?	P = 1
58. When does general hemosiderosis occur?	P = 1
59. What type of calcification develops with hypercalcemia?	P = 1
60. What is the mechanism underlying metastatic calcification?	P = 1
61. Which organs are ferritin in normal found in the greatest amount?	P = 4

1. After falling from the scooter, the child has a bruise on his right thigh, purple-bluish color. Over time, the color of the bruise changed to yellowish-brown, yellowish-greenish. After some time, the skin at the site of the injury acquired a normal color.

What is hemoglobinogenic pigments? Indicate their timing of formation. Name the color of the skin at the site of injury. P = 4

2.At the autopsy, a hemorrhage was found in the substance of the brain with a forming cyst filled with yellow-brown contents. What explains the color of the contents of the cyst in the focus of hemorrhage? Justify approximately the duration of the hemorrhage. P = 4

3. At the autopsy of a patient who died from heart valve disease with symptoms of severe heart failure. The lungs were compacted, with a brown color on cutting. Specify the pigment, observed in the lungs. What is the name of such a lung state? P = 34. The patient died from poisoning with berthollet salt. When examining the corpse, there were the skin and visible mucous membranes yellow colored. At the autopsy, the liver has a rusty tint. Name the pigments that explain the described changes, indicate the reason for their appearance. P = 4

5. A patient with jaundice caused by pancreatic cancer, with obstruction of the common bile duct and signs of impaired renal function. A patient died from cancer cachexia. At the autopsy, cancer metastases were found in the liver. The kidneys were with an erased pattern of structure, yellowish-green in color. Describe the pathogenetic features of jaundice in this patient. P = 4

6. At the autopsy of a deceased patient with cachexia, a decrease in the size of the liver and heart, their tissue on the section was brown. Name the general pathological process and explain the brown color of the liver and the myocardium. P = 3

7. A patient with tuberculosis had hyperpigmentation of the skin, weight loss, hypotension, and insufficiency of adrenal function. What syndrome should be suspected? What causes hyperpigmentation of the skin? P = 2

8. A woman who has had syphilis has pigment-free spots on the skin of her neck. What is the term for these changes? What kind of abnormal metabolism do they testify to? P = 2

9. The patient died from typhoid fever. At autopsy, calcified foci of waxy necrosis were found in the rectus abdominis muscles. What is the type of calcification? P = 1

10. A pregnant woman periodically has cramps of the muscles of the legs. Name and explain the cause of seizures. P = 2

11. At the autopsy of the corpse of a child who underwent tuberculous broncho adenitis, in the lymph nodes of the tracheal bifurcation, whitish, crumbling masses were found. Microscopic picture revealed positive the Koss reaction. What is outcome of lymphadenitis? Name the type of calcification. P = 3

12. A 12-month-old child had growth retardation, non-fusion of fontanelles, chest deformity, thickening of the ribs at the border of the bone and cartilage parts. Specify the type of violation of mineral metabolism, its cause. What disease should be included in the diagnosis? P = 4

13. In the pelvis of the kidney, a whitish stone, resembling a mulberry, was found. Name this formation taking into account its composition. P = 2

14. At the opening of the gallbladder, dense faceted formations were revealed, consisting of yellow crystals radially diverging from the center, surrounded by a layer of dark green condensed bile. Give the full name of these formations. P = 4

15. A 12-year-old boy complains of fatigue, icteric staining of the skin, hyper kinesis, and periodic epileptiform seizures. An enlargement of the spleen, the Kaiser-Flesher ring (a greenish ring on the periphery of the cornea) were revealed. What is metabolism disturbed? What is the reason for this? P = 5