1. Absorption of what substances is affected in disorders of large intestine?
2. proteins
3. aminoacids
4. mineral salts \*
5. monosaccharides
6. water \*
7. Absorption of what substances is affected in disorders of small intestine mucosa?
8. proteins
9. aminoacids \*
10. disaccharides
11. monosaccharides \*
12. wate
13. Activation of what nervous structures can trigger temporo-mandibular pain?
	1. sensitive neurons of posterior medullar horns \*
	2. sensitive neurons of cerebral cortex \*
	3. sensitive neurons of limbic system
	4. motor neurons of posterior medullar horns
	5. motor neurons of the cerebral cortex
14. By what experimental method arterial hyperemia has been modelled on frog’s tongue?
	1. mechanical excitation of the tongue \*
	2. chemical excitation of the tongue
	3. ligation of lingual artery
	4. unilateral ligation of main veins of the tongue
	5. administration of adrenaline solution
15. By what experimental method has been modelled the development of red thrombus in mesenterial vessels in the frog?
	1. by ligation of mesenterial vessels
	2. by applying a crystal of NaCl on mesenterial vessel bifurcation
	3. by applying a crystal of AgNO3 on mesenterial vessel bifurcation
	4. by applying heparin solution on mesenterial vessels
	5. by mechanical injury of the vessel \*
16. By what experimental method ischemia has been modelled on frog’s swimming membrane?
	1. mechanical excitation of the swimming membrane
	2. chemical excitation of the swimming membrane
	3. ligation of veins on swimming membrane
	4. section of main veins on swimming membrane
	5. administration of adrenaline solution on swimming membrane \*
17. By what experimental method stasis has been modelled on frog’s tongue?
	1. mechanical excitation of the tongue
	2. chemical excitation of the tongue
	3. bilateral ligation of lingual veins \*
	4. section of main lingual vein
	5. unilateral ligation of main vein of the tongue
18. By what experimental method venous hyperemia has been modelled on frog’s tongue?
	1. mechanical excitation of the tongue
	2. chemical excitation of the tongue
	3. ligation of lingual artery
	4. unilateral ligation of main vein of the tongue \*
	5. bilateral ligation of lingual veins
19. By what experimental method was modelled the development of white thrombus in mesenterial vessels in the frog?
	1. by ligation of mesenterial vessels
	2. by applying a crystal of NaCl on mesenterial vessel bifurcation \*
	3. by applying a crystal of AgNO3 on mesenterial vessel bifurcation
	4. by applying heparin solution on mesenterial vessels
	5. by applying adrenalin solution on mesenterial vessels
20. By what method has been modelled hyperthyroidism in rats?
	1. by administration of methyluracil
	2. by administration of caffeine
	3. by administration of chloral hydrate
	4. by administration of L – thyroxin \*
	5. by administration of NaCl
21. By what method has been modelled hypothyroidism in rats?
	1. by administration of methyluracil \*
	2. by administration of caffeine
	3. by administration of chloral hydrate
	4. by administration of L – thyroxin
	5. by administration of NaCl
22. From what value of K+ ions concentration in the blood there is considered hypokalemia?
	1. less than 5,5 mEq/L
	2. less than 4,5 mEq/L
	3. less than 3,5 mEq/L \*
	4. less than 2,5 mEq/L
	5. less than 7,5 mEq/L

1. From what value of K+ ions concentration in the blood there is considered hyperkalemia?
	1. above 5,5 mEq/L \*
	2. above 4,5 mEq/L
	3. above 3,5 mEq/L
	4. above 7,5 mEq/L
	5. above 2,5 mEq/L
2. From what value of the concentration of Ca++ ions in the blood there is considered hypocalcemia?
	1. less than 5,3 mmol/L
	2. less than 0,5 mmol/L
	3. less than 2,1 mmol/L \*
	4. less than 1,0 mmol/L
	5. less than 7,0 mmol/L
3. From what value of the concentration of Ca++ ions in the blood there is considered hypercalcemia?
4. above 2,6 mmol/L \*
5. above 3,5 mmol/L
6. above 1,6 mmol/L
7. above 2,0 mmol/L
8. above 7,0 mmol/L
9. From what value of the concentration of Na+ ions in the blood there is considered hyponatremia?
	1. less than 100 mEq/L
	2. less than 152 mEq/L
	3. less than 135 mEq/L \*
	4. less than 120 mEq/L
	5. less than 300 mEq/L
10. From what value of the concentration of Na+ ions in the blood there is considered hypernatremia?
	1. above 100 mEq/L
	2. above 152 mEq/L \*
	3. above 142 mEq/L
	4. above 132 mEq/L
	5. above 300 mEq/L
11. How alteration has been modelled on frog’s tongue?
	1. by administration of adrenalin solution 0,1%
	2. by bilateral ligation of lingual veins
	3. by applying crystal of AgNO3 on the tongue \*
	4. by ligation of lingual arteries
	5. by unilateral ligation of lingual vein
12. How anaphylactic shock was triggered in the experimental rabbit?
	1. by parenteral administration of horse serum \*
	2. by parenteral administration of glucose solution
	3. by parenteral administration of isotonic solution
	4. by parietal administration of adrenaline solution
	5. by parenteral administration of bicarbonate solution
13. How are classified emboli by the direction of their circulation?
	1. Orthograde \*
	2. Anterograde
	3. Retrograde \*
	4. Paradoxical \*
	5. Turbulent
14. How blood pressure (BP) and breathing rate (BR) change in painful stimulation?
	1. BP increase, BR increase \*
	2. BP decrease, BR decreases
	3. BP unchanged, BR increase
	4. BP increase, BR unchanged
	5. BP decrease, BR unchanged
15. How can be performed the specific hyposensitisation in anaphylactic reaction?
	* 1. fractional administration high doses of specific allergen
		2. fractional administration of low doses of specific allergen \*
		3. administration of antibodies specific for allergen
		4. fractional administration of high doses of non-specific allergen
		5. transfer of sensitized lymphocytes from another sensitized person
16. How does carbohydrates metabolism change in liver failure?
	1. exaggerate postprandial hyperglycemia \*
	2. fasting hypoglycemia \*
	3. fructosemia
	4. glycogen storages diminish
	5. glycogen storage increases

1. How does lipid metabolism change in liver failure?
	1. there is intense lipolysis in the liver
	2. there is steatosis of the liver due to the accumulation of triglycerides
	3. there is intense lipolysis in adipose tissue with transport hyperlipidemia
	4. there is steatosis of the liver due to accumulation of cholesterol
	5. there is increased production of lipoproteins
2. How does protein metabolism change in liver failure?
	1. develops hyperglobulinemia \*
	2. develops hypoalbuminemia \*
	3. develops hyperaminoacidemia
	4. synthesis of gamma-globulins is disturbed
	5. there is increased concentration of urea in the blood
3. How does stomach tonus and motility change in hyperchlorhydria?
	1. hypotonus
	2. hypertonus \*
	3. accelerated evacuation
	4. stomachal chymostasis \*
	5. vomiting \*
4. How does stomach tonus and motility change in hypochlorhydria?
	1. Hypotonus \*
	2. hypertonus
	3. accelerated evacuation \*
	4. stomachal chymostasis
	5. vomiting
5. How evacuation function of the stomach is affected in hypersecretion with hyperacidity?
	1. increases
	2. decreases \*
	3. doesn’t change
	4. develops gastric chymostasis \*
	5. develops dumping syndrome
6. How has been evaluated the role of thyroid hormones in pathology?
	1. Expose of animals with hypo- and hyperthyroidism at hyperoxia
	2. Expose of animals with hypo- and hyperthyroidism at hypobaric hypoxia \*
	3. Expose of animals with hypo- and hyperthyroidism at normobaric hypoxia
	4. Expose of animals with hypo- and hyperthyroidism at hyperbaric hypoxia
	5. Expose of animals with hypo- and hyperthyroidism at hypothermia
7. How has been modelled acute adrenocortical insufficiency in rats?
	1. due to ligation of suprarenal arteries
	2. due to administration of drugs that block cholesterol metabolism
	3. due to bilateral surgical removal of adrenal glands \*
	4. due to unilateral surgical removal of adrenal glands
	5. due to administration of cytostatic

1. How has been modelled experimental fat embolism in the mesenterial vessels in the frog?
	1. by administration of oil emulsion in the heart ventricles \*
	2. by administration of oil emulsion orally
	3. by administration of oil solution in the dorsal lymphatic sack
	4. by administration of oil solution intraperitoneally
	5. by administration of oil solution transcutaneous
2. How has been modelled experimental hypervolemia?
	1. by administration of isotonic solution in the vascular bed \*
	2. by administration of adrenalin in the vascular bed
	3. by administration of noradrenaline in the vascular bed
	4. by administration of caffeine in the vascular bed
	5. by administration of hypertonic solution in the vascular bed
3. In what conditions can develop atrophy of the mouth mucosa?
	1. Bacterial infection
	2. collagen disease \*
	3. chronic inflammation \*
	4. acute inflammation
	5. viral infection
4. In what conditions can develop gaseous embolism?
	1. Decrease of dissolved gas concentration in the blood
	2. Sudden decrease in atmospheric pressure \*
	3. Decreased solubility of gases in the blood \*
	4. Increased solubility of gases in the blood
	5. Sudden increase in atmospheric pressure
5. In what disorders can be found hyperkalemia?
	1. hyperhydration with gain of body fluids
	2. hypersecretion of aldosterone
	3. enhanced catabolism of tissue proteins \*
	4. increased plasma renin concentration
	5. decreased plasma renin concentration \*
6. Lack of what digestive enzymes lead to lipid maldigestion?
	1. pancreatic amylase
	2. pancreatic carboxypeptidase
	3. salivary lipase \*
	4. pancreatic lipase \*
	5. hepatic lipase
7. Lack of what digestive enzymes lead to protein maldigestion?
	1. intestinal peptidases \*
	2. pancreatic carboxypeptidase \*
	3. pancreatic amylase
	4. salivary proteases
	5. salivary amylase
8. On what does the localization of general injuries depend?
	1. different sensibility of the body’s structures to the harmful factor \*
	2. the intensity of etiological factor that causes the disorder
	3. the affinity of pathogenic factor to the body structures \*
	4. on blood velocity in the tissue
	5. the intensity of pathogenic factor action
9. To what stressful factor were subjected laboratory animals with acute experimental hypocorticosolism?
	1. hypobaric hypoxia
	2. cold water \*
	3. starvation
	4. physical effort
	5. electrical shock
10. To what stressful factors were subjected laboratory animals in experimental hypocorticosolism?
	1. hypobaric hypoxia
	2. artificial hypervolemia
	3. starvation
	4. cold water \*
	5. electrical shock
11. Under what conditions can be found oligocythemic hypervolemia?
	1. massive infusion of saline solution \*
	2. blood transfusion
	3. body dehydration
	4. body hyperhydration \*
	5. diarrhea
12. Under what conditions can be found polycythemic hypervolemia?
	1. In case of erythremia \*
	2. In case of erythropenia
	3. blood transfusion \*
	4. In case of body dehydration
	5. plasma transfusion
13. Under what conditions can be found polycythemic hypovolemia?
	1. In case of body dehydration \*
	2. In case of burns \*
	3. In case of erythremia
	4. In case of anemia
	5. In case of body hyperhydration
14. Under what pathological conditions can be found monocytosis?
	1. in acute infection
	2. granulomatous inflammation \*
	3. in chronic infection \*
	4. bronchial asthma
	5. bacterial infection
15. Under what pathological conditions can be found neutrophilia?
	1. bacterial infection \*
	2. myocardial infarction
	3. purulent otitis \*
	4. viral infections
	5. influenza
16. Under what pathological conditions can be found primary absolute lymphocytosis?
	1. tuberculosis
	2. septicemia
	3. bronchial asthma
	4. chronic lymphoid leucosis \*
	5. Hodgkin lymphomas \*
17. What animals are more sensible to action of hypobaric hypoxia?
	1. adult animals \*
	2. new-born animals
	3. animals with excited CNS \*
	4. animals with inhibited CNS
	5. animals with intensified anaerobic metabolism
18. What are causes of pathologic hyposalivation?
	1. emotional states
	2. ingestion of fluid aliments
	3. dehydration\*
	4. parotiditis
	5. salivatory ducts obstruction \*
19. What are compensatory reactions in experimental hypervolemia?
	1. blood storage \*
	2. reduced diuresis
	3. increased diuresis \*
	4. enhanced capillary reabsorption
	5. enhanced aldosterone production
20. Under what conditions can be found oligocythemic hypovolemia?
21. first minutes after acute bleeding
22. 24 hours after acute bleeding \*
23. 30-40 minutes after acute bleeding
24. In case of erythremia
25. In case of body overheating
26. What are consequences of HCl absence in gastric juice?
	1. increased intestinal peristaltic movement \*
	2. decreased intestinal peristaltic movements
	3. maldigestion \*
	4. malabsorption
	5. constipations
27. What are digestive changes in bile secretion insufficiency?
	1. polysaccharides maldigestion
	2. intestinal atonia \*
	3. steatorrhea \*
	4. amylorrhea
	5. Creatorrhea
28. What are digestive disturbances in case of salivary amylase lack?
	1. disorders of polysaccharides digestion
	2. disorders of disaccharides digestion
	3. disorders of cellulose digestion \*
	4. disorders of proteins digestion
	5. disorders of lipid digestion
29. What are excitatory mediators?
	1. glycine
	2. Acetylcholine \*
	3. Dopamine \*
	4. gamma-oxibutyric acid
	5. serotonin
30. What are exogenous infectious pyrogenic factors?
	* 1. bacterial antigens \*
		2. bacterial endotoxin \*
		3. hyperimmune sera
		4. heterogeneous blood compounds in hemotransfusion
		5. products of cell disintegration caused by bacteria
31. What are exogenous non-infectious pyrogenic factors?
	* 1. hyperimmune sera \*
		2. heterogeneous blood and plasma \*
		3. bacterial lipopolysaccharides
		4. isotonic sodium chloride solution
		5. fungal proteins
32. What are factors involved in stomach ulcerogenesis?
	1. HCl \*
	2. Bile
	3. Helicobacter pylori \*
	4. Salmonella
	5. anaerobe flora
33. What are forms of heart excitability disorders?
	* 1. ventricular extrasystole \*
		2. paroxysmal tachycardia \*
		3. sinus tachycardia
		4. atrio-ventricular block
		5. sinus bradycardia
34. What are forms of myocardial conductibility disorders?
	1. ventricular extrasystole
	2. paroxysmal tachycardia
	3. atrial fibrillation
	4. atrio-ventricular block \*
	5. sino-atrial block \*
35. What are general manifestations of inflammation?
36. enhanced synthesis of acute-phase proteins in the liver \*
37. reduced erythrocyte sedimentation rate
38. reduced synthesis of acute-phase proteins in the liver
39. increased erythrocytes sedimentation rate \*
40. pain
41. What are inhibitory mediators?
	1. noradrenalin
	2. acetylcholine
	3. dopamine
	4. serotonin \*
	5. gamma-oxibutyric acid \*
42. What are local manifestations in allergic reaction type III?
43. arterial hyperemia \*
44. proliferative inflammation
45. ischemia
46. infiltration with neutrophils leukocytes \*
47. infiltration with T lymphocytes

1. What are manifestations of chronic bleeding in the mouth?
	1. atrophy of mouth mucosa \*
	2. hyperplasia of mouth mucosa
	3. hypertrophy of mouth mucosa
	4. redness of mouth mucosa
	5. paleness of mouth mucosa \*
2. What are manifestations of hemolytic anemia in the mouth?
	1. hyperemia of the mouth mucosa
	2. ischemia of the mouth mucosa \*
	3. gingival micro-bleeding \*
	4. gingival hyperplasia
	5. hyperplasia of lingual papilla
3. What are manifestations of intestinal autointoxication?
	1. arterial hypotension \*
	2. arterial hypertension
	3. headache \*
	4. hypoglycemia \*
	5. hyperglycemia
4. What are manifestations of iron deficiency anemia in the mouth?
	1. atrophy of mouth mucosal \*
	2. hypertrophy of mouth mucosa
	3. green-grey coloration of mouth mucosa \*
	4. yellow coloration of mouth mucosa
	5. hyperplasia of mouth mucosa
5. What are manifestations of venous hyperemia on frog’s tongue?
	1. reduced filling of arterioles with blood
	2. overfilling of capillaries with blood \*
	3. Reduced volumetric blood velocity \*
	4. Increased linear blood velocity
	5. Reduced number of functional capillaries

1. What are metabolic effects of glucagon?
	1. stimulates glycogenogenesis
	2. stimulates glycogenolysis \*
	3. stimulates lipogenesis
	4. stimulates lipolysis \*
	5. stimulates glycolysis
2. What are microcirculatory changes on frog’s tongue in arterial hyperemia?
	1. dilation of arterioles, capillaries and venules
	2. increased number of functional capillaries \*
	3. decreased linear blood velocity
	4. increased hydrostatic pressure \*
	5. decreased number of functional capillaries

1. What are microcirculatory changes on frog’s tongue in prestasis?
	1. pulsatile movements \*
	2. pendulatory movements \*
	3. turbulent movements
	4. diminished inflow
	5. centripetal movements
2. What are microcirculatory changes on frog’s tongue in prestasis?
	1. accelerated movements
	2. pendulatory movements \*
	3. turbulent movements
	4. diminished arterial inflow
	5. centripetal movements
3. What are microcirculatory changes on frog’s tongue in venous hyperemia?
	1. increased volumetric blood velocity
	2. dilation of venules \*
	3. increased venous outflow
	4. narrowing of arterioles
	5. reduced linear blood velocity \*
4. What are negative signals of apoptosis initiation?
	1. absence of the growth factors \*
	2. absence of growth hormone (somatotropin)
	3. absence of estrogens for the endometrium \*
	4. absence of estrogens for the mammary gland
	5. absence of prolactin for the endometrium
5. What are positive signals of apoptosis initiation?
	1. irreparable cell injuries of any origin
	2. lack of prolactin for the mammary gland
	3. glucocorticoids for lymphocytes
	4. testosterone for the prostate
	5. lack of estrogens for the endometrium
6. What are signs of secondary absolute erythrocytosis?
	1. Low erythropoietin level
	2. High erythropoietin level \*
	3. erythrocytes count more than 5,5 ×1012/L \*
	4. erythrocyte count less than 5,5 ×1012/L
	5. Reticulocyte count less than 1,5%
7. What are the carbohydrate metabolic disorders in starvation?
	* + 1. Excess of acetyl KoA \*
			2. deficiency of oxaloacetate \*
			3. Excessive oxaloacetate
			4. Metabolic alkalosis
			5. Deficiency of acetyl KoA
8. What are the cardiac mechanisms of compensation in circulatory failure?
	1. increased force of heart contraction \*
	2. increased end-diastolic volume
	3. increased systolic volume \*
	4. reduced cardiac output
	5. increased end-systolic volume
9. What are the causes for thrombus development?
	1. Endothelial injury \*
	2. increased blood velocity
	3. increased level of pro-coagulant factors \*
	4. intensification of blood flow
	5. activation of the complement system
10. What are the causes of air embolism?
	1. Trauma of aorta
	2. Trauma of venous cranial sinus \*
	3. Trauma carotid artery
	4. Trauma of jugular veins \*
	5. Trauma of pulmonary artery
11. What are the causes of carbohydrates malabsorption?
	1. atrophy of mucosal layer in the small intestine \*
	2. atrophy of mucosal layer in the large intestine
	3. atrophy at the level of gastric mucosal layer
	4. inflammation at the level of small intestine mucosal layer \*
	5. inflammation at the level of large intestine mucosal layer
12. What are the causes of death in rats exposed to reduced atmospheric pressure?
13. decreased pressure of oxygen in inspiratory air
14. hypobaria
15. respiratory hypoxia
16. hypoxemia \*
17. circulatory hypoxia
18. What are the causes of death of the rabbit in experimental anaphylactic shock?
19. Cerebral coma
20. Cardiac arrest
21. respiratory arrest \*
22. Arterial collapse \*
23. Ischemia in the brain
24. What are the causes of exocrine insufficiency of the pancreas?
	1. chronic pancreatitis \*
	2. pancreatic tumor
	3. pancreatic duct obturation \*
	4. vagotonia
	5. sympathicotonia
25. What are the causes of eosinophilia?
26. insufficiency of adrenal glands \*
27. insufficiency of thyroid gland
28. allergic diseases \*
29. bacterial infection
30. viral infection
31. What are the causes of facial pain?
	1. inflammatory processes in the mouth \*
	2. trauma of dental-maxillary apparatus \*
	3. periosteal disorders
	4. injuries of salivary glands
	5. injuries of perivascular tissue
32. What are the causes of hypercalcemia?
	1. hypersecretion of mineralocorticoids
	2. hyposecretion of calcitonin \*
	3. excessive vitamin D \*
	4. deficient vitamin D
	5. deficiency of mineralocorticoids
33. What are the causes of hypercalcemia?
	1. hypersecretion of parathyroid hormone \*
	2. hereditary defective of calcium-dependent receptors (for parathyroid hormone)
	3. hyposecretion of calcitonin \*
	4. deficiency of vitamin D
	5. hyposecretion of parathyroid hormone
34. What are the causes of hypercoagulation?
	* + - 1. reduced level of thromboplastin
				2. surplus of thromboplastin \*
				3. increased plasminogen level in the blood
				4. decreased plasminogen level in the blood \*
				5. increased level of heparin
35. What are the causes of hyperphosphatemia?
	1. mobilization from bone matrix
	2. hyposecretion of PTH \*
	3. hypersecretion of PTH
	4. intracellular-extracellular shift \*
	5. hypersecretion of calcitonin
36. What are the causes of hypocalcemia?
	1. hypersecretion of calcitonin \*
	2. hypersecretion of parathyroid hormone
	3. hyposecretion of calcitonin
	4. Hyperphosphatemia \*
	5. Hypophosphatemia
37. What are the causes of intestinal auto-intoxication?
	1. liver failure \*
	2. protein maldigestion and malabsorption \*
	3. lipid maldigestion and malabsorption
	4. enteritis \*
	5. carbohydrates maldigestion and malabsorption
38. What are the causes of neutrophilia?
	1. Viral infection
	2. allergic diseases
	3. cocci infection \*
	4. acute infection disease \*
	5. chronic infection disease
39. What are the causes of primary endocrine disorders?
	1. disorders of endocrine hypothalamus
	2. disorders of adenohypophysis
	3. disorders of neurohypophysis
	4. disorders of peripheral endocrine glands \*
	5. disorders of peripheral hormonal reception \*
40. What are the causes of pulmonary restrictive disease?
	1. pulmonary fibrosis \*
	2. pulmonary emphysema
	3. obstruction of superior airways
	4. pulmonary atelectasis \*
	5. obstruction of inferior airways
41. What are the causes of resistance overload of the heart?
	1. stenosis of aortic valve \*
	2. insufficiency of mitral valves
	3. hypervolemia
	4. insufficiency of aortic valves
	5. arterial hypertension \*
42. What are the causes of secondary endocrine disorders?
	1. disorders of endocrine hypothalamus
	2. disorders of adenohypophysis \*
	3. disorders of neurohypophysis
	4. disorders of peripheral endocrine glands
	5. disorders of peripheral hormonal reception
43. What are the causes of sinus bradycardia?
	1. intoxication with β-adrenergic drugs
	2. increased intracerebral pressure \*
	3. heart sympathicotonia
	4. intoxication with digitalis \*
	5. intoxication with α-adrenergic drugs
44. What are the causes of tertiary endocrine disorders?
	1. disorders of endocrine hypothalamus \*
	2. disorders of adenohypophysis
	3. disorders of neurohypophysis
	4. disorders of peripheral endocrine glands
	5. disorders of peripheral hormonal reception
45. What are the causes of volume overload of the heart?
	1. stenosis of aortic valves
	2. insufficiency of mitral valves \*
	3. arterial hypertension
	4. insufficiency of aortic valves \*
	5. stenosis of aortic valves
46. What are the cellular sources of proliferation in the inflammatory focus?
	1. fibrocytes
	2. monocytes emigrated from the blood in the inflammatory focus \*
	3. neutrophils emigrated from the blood in the inflammatory focus
	4. fibroblasts \*
	5. resident parenchymal cells
47. What are the changes in exogenous hypobaric hypoxia?
	1. arterial hypoxemia, hypercapnia, alkalosis
	2. arterial hypoxemia, hypocapnia, alkalosis
	3. arterial hypoxemia, hypocapnia, acidosis \*
	4. arterial hypoxemia, hypercapnia, acidosis
	5. hypoxia, hypercapnia, alkalosis
48. What are the changes in respiratory hypoxia?
	1. arterial hypoxemia, hypocapnia, respiratory acidosis
	2. arterial hypoxemia, hypercapnia, respiratory acidosis \*
	3. arterial hypoxemia, hypercapnia, respiratory alkalosis
	4. arterial hypoxemia, normal CO2 pressure, respiratory alkalosis
	5. arterial hypoxemia, hypocapnia, respiratory alkalosis
49. What are the characteristics of inflammatory stasis?
50. is associated with intravascular aggregation of blood cells \*
51. enhances the spreading of secondary alteration in the inflammatory focus
52. limits the spreading of primary alteration in the inflammatory focus
53. limits the spreading of secondary alteration in the inflammatory focus \*
54. is associated with reduced platelets aggregation
55. What are the characteristics of type I allergic reactions?
56. reaction between free allergen in circulation and fixed antibodies on parenchymal cells
57. reaction between allergen fixed on the cells and free antibodies in circulation
58. reaction between allergen and antibodies both in circulation
59. reaction between allergen and sensitized lymphocytes
60. reaction between free allergen in circulation and antibodies fixed on mast cells \*
61. What are the characteristics of type II allergic reactions?
62. reaction between free allergen in circulation and fixed antibodies on parenchymal cells
63. reaction between allergen fixed on the cells and free antibodies in circulation \*
64. reaction between allergen and antibodies both in circulation
65. reaction between allergen and sensitized lymphocytes
66. reaction between free allergen in circulation and antibodies fixed on mast cells
67. What are the characteristics of type III allergic reaction?
68. reaction between free allergen in circulation and fixed antibodies on parenchymal cells
69. reaction between allergen fixed on the cells and free antibodies in circulation
70. reaction between allergen and antibodies both in circulation \*
71. reaction between allergen and sensitized lymphocytes
72. reaction between free allergen in circulation and antibodies fixed on mast cells

1. What are the clinical manifestations of hypercalcemia?
	1. neuronal inhibition \*
	2. neuronal excitation
	3. muscle hypertonia
	4. hyperreflexia
	5. muscle paralysis \*
2. What are the compensatory reactions in hyperglycemia?
	1. glucocorticoid hyposecretion \*
	2. inhibition of gluconeogenesis \*
	3. glucocorticoid hypersecretion
	4. increased lipolysis
	5. enhanced glycogenolysis
3. What are the compensatory reactions in hypoglycemia?
	1. glucagon hypersecretion \*
	2. glucagon hyposecretion
	3. increased glycogenolysis \*
	4. glucocorticoid hyposecretion
	5. increased lipogenesis
4. What are the compensatory reactions in long-lasting hypoxia?
	1. hypersecretion of erythropoietin \*
	2. mitochondrial hyperplasia and hypertrophy \*
	3. hyposecretion of erythropoietin
	4. hypersecretion of thyroid hormones
	5. hyposecretion of thyroid hormones
5. What are the conditions for formation of parietal thrombus?
6. exposure of subendothelial matrix \*
7. turbulent blood flow \*
8. enhanced blood velocity
9. increased platelet count
10. linear blood flow
11. What are the consequences of activation of the nonspecific intracellular phospholipases?
	1. Breakdown of intracellular proteins and initiation of the cell autolysis
	2. Breakdown of nucleoproteins and initiation of apoptosis
	3. Breakdown of macroergic complexes and energy depletion
	4. Breakdown of membrane phospholipids \*
	5. Initiation of anaerobic glycolysis
12. What are the consequences of annihilation of the transmembrane Ca2+ ions gradient?
	1. Myocyte relaxation
	2. Inhibition of intracellular enzymes
	3. Activation of the intracellular enzymes \*
	4. Activation of several extracellular enzymes
	5. Myocyte contraction \*
13. What are the consequences of dystrophy?
	1. Inflammation \*
	2. Sclerosis \*
	3. aplasia
	4. hyperplasia
	5. hypertrophy
14. What are the consequences of excessive carbohydrates intake?
	1. hyposecretion of glucagon \*
	2. enhanced protein synthesis
	3. enhanced lipid synthesis \*
	4. hypersecretion of glucagon
	5. enhanced lipolysis
15. What are the consequences of lipid deficiency in the diet?
	1. hypercoagulation state
	2. hypocoagulation state \*
	3. decreased blood level of saturated fatty acids
	4. decreased blood level of polyunsaturated fatty acids \*
	5. increased blood level of polyunsaturated fatty acids
16. What are the consequences of the intracellular ATP-ases activation?
	1. Breakdown of intracellular proteins and initiation of the cell autolysis
	2. Breakdown of nucleoproteins and initiation of apoptosis
	3. Breakdown of macroergic complexes and energy depletion \*
	4. Breakdown of AMP and ADP
	5. Initiation of anaerobic glycolysis
17. What are the consequences of the intracellular proteases activation?
	1. Breakdown of intracellular proteins and initiation of the cell autolysis \*
	2. Breakdown of nucleoproteins and initiation of apoptosis
	3. Breakdown of macroergic complexes and energy depletion
	4. Breakdown of AMP and ADP
	5. Initiation of anaerobic glycolysis
18. What are the disorders that could lead to teeth attrition?
	1. Disorders of thyroid gland \*
	2. Disorders of parathyroid gland \*
	3. Disorders of adrenal glands
	4. Disorders of gonads
	5. Disorders of liver
19. What are the effects of mediators involved in the allergic reaction type III?
20. bronchoconstriction
21. injury of endothelial cells \*
22. alterative inflammation \*
23. proliferative inflammation
24. granulomatous inflammation
25. What are the end-effectors in anaphylactic allergic reactions?
26. immunoglobulins E \*
27. immunoglobulin A
28. immunoglobulin G4 \*
29. sensitized B lymphocytes
30. sensitized T lymphocytes
31. What are the endogenous causes of diseases?
32. Mechanical, physical, chemical and biological factors from the environment
33. Resident microflora of the intestines and respiratory pathways
34. Intestinal parasites and blood parasites
35. Chromosome aberration resulting from the action of ionizing radiation
36. Inherited genetic defects \*
37. What are the etiological factors of lipid dystrophy?
	1. alimentary hyperglycemia \*
	2. deficiency of lipids in the diet
	3. deficiency of proteins in the diet \*
	4. excess of proteins in diet
	5. Alimentary hypolipidemia
38. What are the exogenous causes of diseases?
	1. Mechanical, physical, chemical and biological factors from the environment \*
	2. Resident microflora of the intestines and respiratory pathways
	3. Intestinal parasites and blood parasites \*
	4. Chromosome aberration resulting from the action of ionizing radiation \*
	5. Inherited genetic defects
39. What are the external changes of arterial hyperemia?
40. Diffuse general erythema
41. Local erythema \*
42. Decreased tissue turgor
43. Increased local temperature \*
44. Hyperthermia of the body
45. What are the external manifestations of ischemia?
	1. Increased turgor of the skin
	2. Decreased turgor of the skin \*
	3. Redness of ischemic tissue
	4. Paleness of ischemic tissue \*
	5. Swelling of the tissue
46. What are the external manifestations of venous hyperemia?
	1. Decreased tissular turgor
	2. Decreased local temperature \*
	3. Decreased body temperature
	4. General cyanosis
	5. Local cyanosis \*
47. What are the factors that may cause hyperlipidemia?
	1. hypersecretion of insulin
	2. hypersecretion of glucocorticoids \*
	3. hyposecretion of insulin \*
	4. hyposecretion of glucagon
	5. hyposecretion of glucocorticoids
48. What are the final effects in type II allergic reactions?
49. hemolytic anemia \*
50. neutropenia \*
51. lymphocytosis
52. thrombocytosis
53. lymphocytopenia
54. What are the general causes of energy depletion that trigger cell dystrophy?
	1. Chronic local hypoxia \*
	2. Deprivation of food \*
	3. Acute hypoxia
	4. Activation of oxidative processes by toxins
	5. Activation of anabolic processes in acute stress
55. What are the general consequences of apoptosis for the organism?
56. Cell death by apoptosis initiate fever
57. Cell death by apoptosis doesn’t provoke quantitative modifications of the cellular population
58. Cell death by apoptosis initiates the acute phase reaction
59. Cell death by apoptosis has no negative consequences for other structures of the organism \*
60. Cell death by apoptosis has negative consequences for the whole organism
61. What are the general consequences of necrosis for the organism?
	1. death of the whole organism
	2. enzymemia \*
	3. hyperkalaemia \*
	4. hypernatremia
	5. hyperchloremia
62. What are the general consequences of the cell necrosis for the whole organism?
	1. Cell death by necrosis initiate cell dystrophy in the affected tissue
	2. Cell death by necrosis has negative consequences for the affected organ only
	3. Cell death by necrosis has no negative consequences for the whole organism
	4. Cell death by necrosis initiates the acute phase reaction \*
	5. Cell death by necrosis initiates fever \*
63. What are the hallmarks of serous exudate?
64. small molecular weight proteins up to 2-3% \*
65. high molecular weight proteins up to 2-3%
66. many polymorphonuclear leukocytes
67. lack of polymorphonuclear leucocytes \*
68. presence of lysosomal enzymes
69. What are the hematologic signs of absolute secondary erythrocytosis?

a) hemoglobin content more than 160 g/L \*

b) hemoglobin content less than 160g/l

c) erythrocyte count more than 5,5 ×1012/L \*

d) total blood volume less than 7% from body weight

e) total blood volume less than 5% from body weight

1. What are the immediate extracardiac mechanisms of compensation in circulatory failure?
2. increased volume of circulating blood by mobilization of stored blood \*
3. even distribution of heart output to organs
4. distribution of cardiac output to vital organs \*
5. generalized vascular spasm to maintain arterial pressure
6. reduction of circulatory blood volume by partly blood sequestration
7. What are the late extracardiac mechanisms of compensation in circulatory failure?
	1. hydro-electrolytic retention \*
	2. pulmonary hyperventilation
	3. hyposecretion of vasopressin
	4. heart hypertrophy
	5. hypersecretion of erythropoietin \*
8. What are the local consequences of necrosis?
	1. Inflammation \*
	2. Demarcation \*
	3. encapsulation
	4. Infiltration with platelets
	5. Infiltration with erythrocytes
9. What are the local manifestations of glosalgia?

a) burn sensation \*

b) hypersalivation

c) hyposalivation

d) paraesthesia \*

e) tongue edema

1. What are the main manifestations of hyperphosphatemia?
	1. muscle cramps \*
	2. blood hyperosmolarity
	3. hypocalcemia \*
	4. hypercalcemia
	5. muscle paralysis
2. What are the main manifestations of hypophosphatemia?
	1. hemolytic anemia \*
	2. osteomalacia \*
	3. tetany
	4. muscle cramps
	5. enhanced neuronal excitability
3. What are the main mechanisms which maintain the calcium homeostasis?
	1. redistribution of Ca++ ions between intra- and extracellular compartment of the body
	2. removing of calcium salts with bile in the gastrointestinal tract
	3. incorporation and mobilization from bone matrix \*
	4. incorporation and mobilization from teeth matrix
	5. reabsorption on calcium in the proximal renal tubules \*
4. What are the main pathogenetic mechanisms of hypercalcemia?
	1. redistribution between intra- and extracellular compartment
	2. increased mobilization from teeth matrix
	3. increased mobilization from bone matrix \*
	4. reduced renal reabsorption
	5. reduced renal secretion \*
5. What are the main pathogenetic mechanisms of hypernatremia?
	1. dehydration with excessive loss of body water \*
	2. release of sodium ions from damaged cells
	3. decreased synthesis of renin in the kidneys
	4. hyperhydration with excessive gain of body water
	5. increased synthesis of renin in the kidneys \*
6. What are the main pathogenetic mechanisms of hyponatremia?
	1. excessive gain of body fluids \*
	2. excessive loss of body fluids
	3. deficiency of mineralocorticoids \*
	4. excessive release of sodium from damaged cells
	5. hypersecretion of mineralocorticoids
7. What are the main pathophysiological mechanisms of hypocalcemia?
	1. deficient mobilization from the bone in deficiency of PTH \*
	2. deficient mobilization from the bone in excessive PTH
	3. decreased incorporation in the bone in hyposecretion of calcitonin
	4. enhanced calcium secretion in the proximal renal tubes \*
	5. enhanced calcium reabsorption in the proximal renal tubes
8. What are the manifestations of agranulocytosis in the oral cavity?
	1. Ulcero- necrotic tonsillitis \*
	2. hyperemia of mouth mucosa
	3. thinning of tooth enamel
	4. thinning of dentine
	5. ischemia of mouth mucosa
9. What are the manifestations of B12 deficiency anemia in the oral cavity?
10. Presence of painful sensation of the tongue \*
11. atrophy of lingual mucosa \*
12. hyperplasia of lingual mucosa
13. hyperplasia of lingual papilla
14. absence of painful sensation of the tong
15. What are the manifestations of cell necrosis?
16. leakage of lysosomal enzymes in hyaloplasm \*
17. no leakage of lysosomal enzymes in hyaloplasm
18. cell swelling \*
19. cell shrinkage
20. fragmentation of the cytoplasm and formation of apoptotic bodies
21. What are the manifestations of cellular alterations in the inflammatory focus?
22. cell injury \*
23. cell dystrophy \*
24. cell apoptosis
25. fibrosis
26. cell hyperplasia
27. What are the manifestations of stasis?
	1. Decreased local temperature \*
	2. Decreased body temperature
	3. Reduced tissular turgor
	4. Local cyanosis \*
	5. General cyanosis
28. What are the manifestations of stasis?
	1. Microhaemorrhages due to increased hydrostatic pressure \*
	2. Microhaemorrhages due to decreased hydrostatic pressure
	3. Microhaemorrhages due to increased permeability of the vessels \*
	4. Microhaemorrhages due to decreased permeability of the vessels
	5. Microhaemorrhages due to increased blood velocity
29. What are the mechanisms of increased thermogenesis in fever?
30. inhibition of catabolic reactions
31. simpatho-adrenal inhibition
32. activation of catabolic reactions \*
33. muscle hyperkinesia \*
34. activation of anabolic reactions
35. What are the mechanisms of enhanced thermolysis in the final stage of fever?
36. peripheral vasodilation \*
37. bradypnea
38. tachypnoea \*
39. clonic muscular contractions
40. reduced sweating
41. What are the mechanisms of activation of thermogenesis in fever?
42. tonic muscular contractions
43. excitation of the parasympathetic nervous system
44. activation of catabolic reactions
45. clonic muscular contractions
46. hypersecretion of insulin
47. What are the mechanisms of phagocytosis?
	1. is the result of opsonization of microbes with the complement fraction C3a and easier recognition by receptors on phagocytic cells
	2. is the result of interaction between specific Fc receptors on phagocytic cells and microorganisms
	3. is the result of opsonization of microbes with the complement fraction C3b and easier recognition by receptors on phagocytic cells \*
	4. is the result of electrostatic interaction between the phagocyte receptors and microbe
	5. is the result of opsonization of microbes with the complement fraction C5a and easier recognition by receptors on phagocytic cells
48. What are the mechanisms of reduced thermolysis in the initial period of fever?
49. spasm of peripheral blood vessel \*
50. pulmonary hypoventilation \*
51. tonic muscular contractions
52. excessive sweating
53. pulmonary hyperventilation
54. What are the metabolic and digestive disorders in maldigestion of proteins?
	1. low osmotic pressure
	2. low oncotic pressure \*
	3. food allergy
	4. gastrointestinal auto-intoxication \*
	5. inhibition of putrefaction processes in the bowels
55. What are the metabolic changes in arterial hyperemia?
56. Decreased arterial - venous oxygen difference \*
57. Increased arterial - venous oxygen difference
58. Increased oxygen pressure in venous blood \*
59. Increased oxygen pressure in arterial blood
60. Increased supply and decrease consumption of oxygen
61. What are the metabolic changes in hypersecretion of thyroid hormones?
	1. increased intracellular synthesis of ATP
	2. increased intracellular concentration of ADP \*
	3. glycogenolysis \*
	4. increased glycogenogenesis
	5. increased lypogenesis
62. What are the metabolic changes in hyposecretion of thyroid hormones?
	1. Decreased protein anabolism \*
	2. Increased protein anabolism
	3. Decreased glycogenolysis \*
	4. Increased glycogenolysis
	5. Increased lipolysis
63. What are the metabolic changes in ischemia?
	1. local acidosis \*
	2. local alkalosis
	3. Decreased aerobic metabolic processes \*
	4. Increased aerobic metabolic processes
	5. Decreased anaerobic metabolic processes
64. What are the metabolic changes in the second stage of fever?
65. enhanced anabolic processes
66. depletion of liver glycogen \*
67. enhanced lipolysis and glycogenolysis \*
68. enhanced lipogenesis and glycogenogenesis
69. positive nitrogen balance
70. What are the metabolic changes in venous hyperemia?
71. Decreased aerobic metabolic process \*
72. Increased aerobic metabolic process
73. Increased anaerobic metabolic processes \*
74. local metabolic alkalosis
75. Decreased anaerobic metabolic processes
76. What are the metabolic changes in venous hyperemia?
	1. Enhanced aerobic glycolysis
	2. Increased energogenesis
	3. Reduced aerobic glycolysis \*
	4. Metabolic alkalosis
	5. Metabolic acidosis \*
77. What are the metabolic consequences of excessive consumption of fat?

a) high blood level of VLDL \*

b) enhanced lipogenesis \*

c) enhanced lipolysis

d) enhanced production of ketone bodies

e) infiltration of tissues with chylomicrons

1. What are the metabolic consequences of lipid maldigestion?
	1. enhanced lipolysis
	2. disturbances of steroid hormones synthesis \*
	3. high level of chylomicrons in the blood
	4. excess of liposoluble vitamins
	5. deficiency of liposoluble vitamins \*
2. What are the metabolic effects of insulin?
	1. stimulates glycogenogenesis \*
	2. stimulates glycogenolysis
	3. stimulates lipogenesis \*
	4. stimulates lipolysis
	5. stimulates gluconeogenesis
3. What are the metabolic manifestations of glucocorticoids hypersecretion?
	1. Increased gluconeogenesis \*
	2. Decreased gluconeogenesis
	3. Increased glycogenogenesis
	4. Increased proteolysis \*
	5. Increased proteosynthesis
4. What are the metabolic manifestations of somatotropin hypersecretion?
	1. intensification of carbohydrates catabolism \*
	2. intensification of carbohydrates anabolism
	3. intensification of lipid catabolism \*
	4. intensification of lipid anabolism
	5. intensification of protein catabolism
5. What are the organs that most often are prone to develop lipid dystrophy?
	1. Liver \*
	2. lungs
	3. kidneys \*
	4. brain
	5. sexual glands
6. What are the pathogenetic factors of capillary stasis?
	1. Decreased blood velocity \*
	2. Haemodilution
	3. Decreased concentration of plasma globulins
	4. Increased concentration of plasma globulins \*
	5. Increased blood velocity
7. What are the pathogenetic mechanisms of edema in venous hyperemia?
	1. Increased hydrostatic pressure in the arterioles
	2. increased hydrostatic pressure in the capillaries \*
	3. decreased lymphatic reflux from the organ \*
	4. decreased oncotic pressure in the capillaries
	5. decreased lymphogenesis and lymphodynamics
8. What are the pathogenetic mechanisms of external changes in venous hyperemia?
	1. Decreased local temperature due to reduction of arterial blood inflow \*
	2. Decreased local temperature due to tissular swelling
	3. Increased local temperature due to increased energogenesis
	4. Decreased local temperature due to decreased energogenesis \*
	5. Increased local temperature due to increased metabolic processes
9. What are the pathogenetic mechanisms of ischemia?
	1. Vasoconstriction, vasodilation, acute hemorrhage
	2. Neurogenic mechanism, obstruction, compression, redistribution of blood \*
	3. Neurogenic mechanism, obstruction, compression, storage of the blood
	4. Neurogenic, endocrine, neuroparalytic, cardiogenic mechanism
	5. Redistribution of blood, compression, renal mechanism by vasodilation
10. What are the pathogenetic mechanisms of scleroderma in oral cavity?
	1. lingual edema \*
	2. macrocirculatory disorders
	3. tissue atrophy \*
	4. tissue hypertrophy
	5. tissue hyperplasia
11. What are the pathogenetic mechanisms that contribute to development of hypokalemia?
	1. disturbances of glomerular filtration and water retention in the body
	2. excessive loss of potassium in the kidneys in hypersecretion of aldosterone \*
	3. excessive loss of potassium in the kidneys in hyposecretion of aldosterone
	4. hypersecretion of vasopressin
	5. deficiency of vasopressin
12. What are the pathogenic mechanisms of lipid dystrophy?
13. inability of cell to catabolise lipid excess
14. Increased synthesis of apoproteins and lipoproteins that increases export of lipids from cells
15. Decreased apoprotein level and inability to export lipids form cells \*
16. Hypoxia inhibits fatty acid oxidation and leads to accumulation of lipids \*
17. Hypoxia activates fatty acid oxidation and leads to accumulation of lipids
18. What are the possible causes of hypoproteinemia?
19. deficiency of pancreatic amylase
20. Hemodilution \*
21. proteinuria \*
22. renal failure \*
23. hemoconcentration
24. What are the possible consequences of hyperglycemia in healthy persons?
	1. enhanced oncotic pressure in the blood
	2. enhanced lipid synthesis \*
	3. increased osmotic pressure in the blood \*
	4. stimulation of cortisol secretion
	5. enhanced lipid breakdown
25. What are the possible consequences of hypoglycemia in healthy persons?
	1. enhanced lipid synthesis
	2. enhanced lipid breakdown \*
	3. stimulation of cortisol secretion \*
	4. stimulation of insulin secretion
	5. inhibition of glucagon secretion
26. What are the possible consequences of hypoproteinemia?
	1. energy deficiency
	2. low oncotic pressure \*
	3. high osmotic pressure
	4. low osmotic pressure
	5. edema \*
27. What are the possible variants of relationship between general and local injuries?
	1. There are diseases with exclusive locally injuries
	2. There are diseases with exclusive general injuries
	3. There are some diseases with combination of local and general injuries
	4. Any disease is an integrity of local and general injuries \*
	5. The disease starts with local or general injuries, and later makes an integration between them\*
28. What are the primary endogenous pyrogenic factors?
29. products of cell necrosis \*
30. products of cell disintegration \*
31. interleukin IL-1
32. bacterial lipopolysaccharides
33. tumor necrosis factor
34. What are the pro-coagulant factors?
35. plasminogen
36. thromboplastin \*
37. prothrombin \*
38. antithrombin III
39. plasmin
40. What are the secondary endogenous pyrogenic factors?
41. products of cell necrosis
42. prostaglandins \*
43. acute phase proteins\*
44. immunoglobulin
45. bacterial endotoxin released in the blood
46. What are the signs of intracellular hemolysis?
	1. Hemoglobinemia
	2. Hemosiderinuria
	3. Hemoglobinuria
	4. Hyperbilirubinemia with free bilirubin (indirect bilirubin) \*
	5. Hyperbilirubinemia with conjugated bilirubin (direct bilirubin)
47. What are the signs of primary absolute erythrocytosis?
	1. Low erythropoietin level \*
	2. High erythropoietin level
	3. Reticulocyte count more than 2.5% \*
	4. Thrombocytopenia
	5. Reticulocyte count less than 0,5%
48. What are the signs of relative erythrocytosis?
	1. erythrocyte count more than 5×1012/L \*
	2. erythrocyte count less than 5×1012/L
	3. reticulocyte count more than 0,5%
	4. total blood volume less than 7% from body weight \*
	5. total blood volume more than 7% from body weight
49. What are the signs of right ventricular failure?
	1. hypertension in systemic circulation
	2. hypertension in pulmonary circulation
	3. pulmonary edema
	4. ascites \*
	5. liver enlargement \*
50. What are the signs of vascular insufficiency?
	1. decreased volume of circulating blood \*
	2. decreased arterial pressure
	3. decreased central venous pressure
	4. increased central venous pressure \*
	5. increased volume of circulating blood
51. What are the somatic effects in hypersecretion of thyroid hormones?
	1. Periorbital edema
	2. Retrobulbar edema \*
	3. Weight gain
	4. hypertrophy of skeletal muscles
	5. atrophy of skeletal muscles \*
52. What are the trigger factors for development of hypertrophy?
	1. Increased workload of the organ \*
	2. Decreased workload of the organ
	3. Hormonal hypersecretion \*
	4. Hormonal hyposecretion
	5. Hypersecretion of vasoactive agents \*
53. What atrophy is considered as physiological?
	1. Peripheral endocrine gland atrophy in the hypophyseal tropic hormone hyposecretion \*
	2. Atrophy of hormone dependent organ in the deficiency of peripheral hormone \*
	3. Organ atrophy due to organism aging \*
	4. Organ atrophy in the hyponutrition
	5. Atrophy of organ in the denervation
54. What can be causes of achlorhydria?
	1. gastrin lack \*
	2. atrophic chronic gastritis \*
	3. gastric cancer
	4. hypertrophic gastritis
	5. gastric ulcer
55. What can be causes of intestinal autointoxication?
	1. intensification of putrefaction processes in the intestine
	2. excessive consumption of proteins
	3. constipations \*
	4. diarrhea
	5. liver failure \*
56. What can be causes of pathologic hypersalivation?
	1. in children during teeth eruption
	2. ingestion of dry aliments
	3. stomatitis \*
	4. mouth tumors \*
	5. Parkinson disease
57. What can be causes of steatorrhea?
	1. Acholia \*
	2. insufficiency of pancreatic lipase \*
	3. pepsin insufficiency
	4. cholemia
	5. hyperlipidemia
58. What can be consequences of disaccharides maldigestion?
	1. Diarrhea \*
	2. Dehydration \*
	3. constipations
	4. hyperhydration
	5. hypoglycemia \*
59. What can be consequences of lipid maldigestion?
	1. Hyperlipidemia
	2. Steatorrhea \*
	3. blood hypocoagulation \*
	4. diarrhea
	5. constipations
60. What can be consequences of protein maldigestion?
	1. Hypoproteinemia \*
	2. decreased oncotic pressure \*
	3. edemas \*
	4. proteinuria
	5. immunodeficiency
61. What can be consequences of vomiting?
	1. Hypochloraemia
	2. hyperkalaemia
	3. alkalosis \*
	4. acidosis
	5. activation of renin-angiotensin-aldosterone system \*
62. What can be the antigen in type II allergic reaction?
63. specific antigens in the bloodstream
64. specific receptors on the cell membrane \*
65. exogenous substance in the bloodstream
66. endogenous substance in the bloodstream
67. hyperimmune sera in the blood
68. What can be the causes of carbohydrates maldigestion?
	1. injuries at the level of small intestine mucosal layer \*
	2. insufficiency of salivary amylase \*
	3. insufficiency of gastric pepsin
	4. insufficiency of pancreatic amylase \*
	5. insufficiency of pancreatic carboxypeptidase
69. What can be the causes of hypoglycemia?
	1. enhanced gluconeogenesis
	2. reduced gluconeogenesis \*
	3. hypersecretion of glucocorticoids
	4. hyposecretion of glucocorticoids \*
	5. enhanced glycogenolysis
70. What can be the causes of hypokalemia?
71. deficiency of renin in the blood
72. hypersecretion of glucocorticoids \*
73. excessive renin in the blood \*
74. deficiency of glucocorticoids
75. overhydration of the body
76. What carbohydrates can be absorbed from the gastrointestinal tract?
	1. lactose
	2. glucose \*
	3. glycogen
	4. maltose
	5. galactose \*
77. What cells are involved in apoptosis?
	1. cells with congenital defects \*
	2. senescent old cells
	3. cells infected with viruses \*
	4. ischemic body's cells
	5. cells with reparable injuries
78. What changes of hemogram are characteristic for iron deficiency anemia?
	1. Macrocytosis
	2. hypochromic erythrocytes \*
	3. microcytosis \*
	4. hyperchromic erythrocytes
	5. drepanocytes
79. What conditions are considered favorable for the organism?
	1. Conditions that promote action of the cause and disease appearance
	2. Conditions that impede action of the cause and retain the disease appearance \*
	3. Conditions that decrease body’s resistance \*
	4. Conditions that increase the body’s resistance
	5. Genetic defects
80. What conditions are considered unfavorable for the organism?
	1. Conditions that promote action of the cause and disease appearance \*
	2. Conditions that impede action of the cause and retain the disease appearance
	3. Conditions that decrease the organism’s resistance \*
	4. Conditions that increase the organism’s resistance
	5. Genetic defects
81. What conditions are necessary for the disease onset?
82. Different forms of energy \*
83. Material factors \*
84. Informational factors \*
85. Proper and heterogenous biological field
86. Interaction between proper aura and aura of another person
87. What digestive changes can be found in exocrine insufficiency of the pancreas?
	1. develops maldigestion of polysaccharides \*
	2. develops maldigestion of proteins \*
	3. develops maldigestion of lipids \*
	4. improvement of intestinal digestion
	5. develops cellulose maldigestion
88. What disorders can lead to ulcerative changes of oral mucosa?
89. Stomach ulcer \*
90. Hepatitis \*
91. Thyroiditis
92. Enterocolitis \*
93. Glomerulonephritis
94. What disorders underlie on the basis of delayed hypersensibility?
95. Tuberculosis \*
96. serum sleekness
97. glomerulonephritis
98. Sjogren's syndrome \*
99. bronchial asthma
100. What disorders underlie on the basis of II allergic reactions?
101. myasthenia gravis \*
102. endemic goitre
103. cretinism
104. thyroid adenocarcinoma
105. thyrotoxicosis \*
106. What disturbances lead to extra-parenchymatous restriction?
	1. bronchial asthma
	2. pneumothorax \*
	3. pulmonary hypoperfusion
	4. disorders of neuro-muscular apparatus \*
	5. pulmonary fibrosis
107. What do pathogenetic factors represent?
	1. the effects of the primary cause action \*
	2. the chain of effects resulting from the action of conditions of disease
	3. the chain of effects resulting from the primary cause action \*
	4. the cause and conditions that provoked the disease
	5. the conditions that promote the action of primary cause
108. What does a pathological reaction mean?
109. it is a reaction that doesn’t correspond to the excitant’s specificity \*
110. it is a reaction that quantitatively corresponds to the excitant’s intensity
111. it is a reaction that has a homeostatic character
112. it is a reaction that is inferior to the excitant’s intensity \*
113. it is a reaction that exceeds the excitant’s intensity \*

1. What does a physiological reaction mean?
	1. it is a reaction that is adequate to the specific excitation \*
	2. it is a reaction that has a dyshomeostatic character
	3. it is a reaction that has a homeostatic character \*
	4. it is a reaction that is inferior to the excitant’s intensity
	5. it is a reaction that exceeds the excitant’s intensity
2. What does agranulocytosis represent?
	1. severe increased of lymphocytes in peripheral blood
	2. severe decrease or absence of agranulocytes in peripheral blood
	3. severe increased count of agranulocytes in peripheral blood
	4. severe decreased or absence of granulocytes in peripheral blood \*
	5. increased number of hyper-segmented neutrophils in peripheral blood

1. What does clinical pathophysiology study?
	1. General rules of the origin, onset, evolution and resolution of typical pathological processes
	2. General rules of the origin, onset, evolution and resolution of pathological
	3. General rules about the pathogenesis of clinical syndromes and nosological entities \*
	4. General rules about structural modifications and dysfunctions at the cellular, tissue, system and integral level in typical pathological processes
	5. General rules about structural modifications and dysfunctions of organs and systems in pathological processes
2. What does general pathophysiology study?
3. General rules of the origin, onset, evolution and resolution of typical pathological processes\*
4. General rules of the origin, onset, evolution and resolution of pathological processes in organs and systems
5. General rules about pathogenesis of clinical syndromes and nosological entities
6. General rules about structural modifications and dysfunctions at the cellular, tissue, system and integral level in typical pathological processes
7. General rules about structural modifications and dysfunctions of organs and systems in typical pathological processes
8. What does injury represent?
	1. functional disturbances at any level of the organism’s organization
	2. structural changes at any level of the organism’s organization
	3. the combination of structural changes and functional disturbances at any level of the organism’s organization
	4. structural, biochemical and functional dyshomeostasis at any level of the organism’s organization \*
	5. persistent and irreparable disturbances of the structural, biochemical and functional homeostasis \*
9. What does represent allergic reactions type IV?
10. immediate hypersensibility
11. delayed hypersensibility \*
12. allergic reactions involving sensitized T lymphocytes \*
13. allergic reactions with the participation of B lymphocytes and plasma cells
14. allergic reactions involving activation of the complement system
15. What does represent dyspnea?
	1. changes of respiratory frequency and amplitude \*
	2. changes of gas diffusion through the alveolar-capillary membrane
	3. changes of pulmonary ventilation with hypercapnia
	4. changes of gaseous composition of the blood
	5. subjective sensation of air insufficiency
16. What does represent expiratory dyspnea?
	1. prolonged duration of inspiration and expiration
	2. prolonged duration of expiration \*
	3. increased inspiratory effort with passive expiration
	4. forced expiration \*
	5. reduced inspiratory effort with passive expiration
17. What does represent hypercapnia?

a) partial pressure of CO2 in venous blood more than 46 mmHg

b) partial pressure of O2 in arterial blood less than 60 mmHg

c) partial pressure of CO2 in arterial blood more than 46 mmHg \*

d) partial pressure of CO2 in the cells more than 46 mmHg

e) partial pressure of CO2 in arterial blood less than 40 mmHg

1. What does represent hypoxemia?
	1. partial pressure of CO2 in venous blood more than 46 mmHg
	2. partial pressure of O2 in arterial blood less than 60 mmHg \*
	3. partial pressure of CO2 in arterial blood less than 46 mmHg
	4. partial pressure of O2 in venous blood less than 60 mmHg
	5. reduced O2 pressure in the cells
2. What does represent inspiratory dyspnea?
	1. prolonged duration of inspiration and expiration
	2. short inspiration with long expiration
	3. increased inspiratory effort with passive expiration \*
	4. increased inspiratory effort with forced expiration
	5. reduced inspiratory effort with passive expiration
3. What does represent intra-parenchymatous pulmonary restriction?
	1. reduced total compliance of respiratory system by reduced compliance of the lungs \*
	2. reduction of total compliance of respiratory system
	3. reduced total compliance of respiratory system by reduced compliance of thoracic cavity
	4. reduced total compliance and elasticity of thoracic cavity
	5. reduced total compliance and elasticity of pleura
4. What does represent pulmonary obstruction?
	1. disorders of gas diffusion thought the alveolar-capillary membrane
	2. reduced lung compliance with hypoventilation
	3. increased resistance of airways with hypoventilation \*
	4. increased resistance of airways with hyperventilation
	5. decreased resistance in airways with hypoventilation
5. What does represent pulmonary restriction?
	1. reduced compliance of lung alveoli \*
	2. reduced total compliance of thoracic cage or lungs
	3. reduced elasticity of lung alveoli \*
	4. reduced patency of superior airways
	5. reduced patency of inferior airways
6. What does represent regeneration in the inflammatory focus?
	1. restoration of specific parenchymal structures \*
	2. restoration of nonspecific mesenchymal structures \*
	3. restoration of non-specific parenchymal structures
	4. restoration of specific mesenchymal structures
	5. angiogenesis de novo
7. What does represent thrombocytopathy?
8. decreased thrombocyte number
9. thrombocyte areactivity to thrombin
10. thrombocyte areactivity to ADP \*
11. deficit of coagulation plasmatic factors
12. excessive release of thromboxane by thrombocytes
13. What does represent fever?
14. persistent increasing of body temperature at the action of high environmental temperature
15. persistent increasing of body temperature at the action of catabolic substances on the thermoregulatory center in the brain
16. episodic increasing of body temperature at the action of excitatory substances on the SNS
17. episodic increasing of body temperature at the action of non-infectious agents
18. persistent increasing and maintaining of high body temperature at the action of infectious agents \*
19. What does represent the endogenous antigen?
20. products of cell injury which are released in the blood
21. products of the bacterial or fungal origin which are released in the blood
22. natural components of the human body which lack the immunological tolerance \*
23. natural components of the human body which have immunological tolerance
24. natural components of the human body changed by action of harmful factors
25. What does the disease represent?
26. the combination of injuries and homeostatic reactions of the organism \*
27. the combination of local and general injuries \*
28. the combination of structural and functional processes
29. the combination of pathogenetic and sanogenetic processes \*
30. the pathological process localized in one organ
31. What does the pathological process include?
32. the totality of injuries caused by the action of primary cause
33. the totality of injuries caused by the action of primary cause and the subsequent pathogenetic factors
34. the totality of injuries caused by the action of primary cause plus the subsequent pathogenetic factors plus the protective, compensatory and reparative reactions \*
35. the totality of local injuries
36. the totality of local and general injuries
37. What does the physiological regeneration represent?
	1. Defect regeneration with a recovery of the initial tissue volume
	2. Defect regeneration with tissue excess
	3. Defect regeneration with tissue deficiency
	4. Regeneration of the pathogen factor induced defect with a similar tissue \*
	5. Regeneration of the pathogen factor induced defect with an atypical tissue \*
38. What does the primary hyperthyroidism mean?
	1. Primary increasing of thyroid gland function \*
	2. Disturbance of ratio T3/T4
	3. Increasing of thyroid gland function due to high secretion of thyrotropin
	4. Increasing of thyroid gland function due to high secretion of thyroliberin
	5. Excess of thyrotropin hormone and thyroid hormones
39. What does the primary hypothyroidism mean?
40. Disturbance of ratio T3/T4
41. Primary decreasing of thyroid gland function \*
42. Decreasing of thyroid gland function due to deficiency of thyrotropin
43. Decreasing of thyroid gland function due to deficiency of thyroliberin
44. Deficit of thyrotropin hormone and thyroid hormones
45. What does the sclerosis of organ mean?
	1. Pathological regeneration \*
	2. Reparative physiological regeneration
	3. Compensatory physiological regeneration
	4. Protective physiological regeneration
	5. The last phase of the inflammation
46. What does “left” nuclear shift represent?
	1. increased number of agranulocytes in peripheral blood
	2. increased number of granulocytes in peripheral blood
	3. increased number of immature neutrophils in peripheral blood \*
	4. increased number of mature neutrophils in peripheral blood
	5. increased number of hyper-segmented neutrophils in peripheral blood
47. What endocrine factors can contribute to development of hyperglycemia?
	1. excessive insulin
	2. excessive glucagon \*
	3. deficiency of glucagon
	4. deficiency of thyroid hormones
	5. excessive glucocorticoids \*
48. What endocrine factors can contribute to development of hypoglycemia?
	1. deficiency of insulin
	2. excessive glucagon
	3. deficiency of glucagon \*
	4. excessive thyroid hormones
	5. deficiency of glucocorticoids \*
49. What endogenous factors lead to different effects of hypoxia and hypobaria on rats?
	1. Metabolism intensity \*
	2. functional state of SNC \*
	3. glucose level in the blood
	4. hydric metabolism in the rat
	5. functional state of the immune system
50. What etiological factors are responsible for developing of venous hyperemia?
	1. Increased elasticity of the venous wall
	2. Decreased arterial-venous pressure difference in heart failure \*
	3. Decreased aspiration force of the thorax \*
	4. Increased aspiration force of the thorax
	5. Increased arterial-venous pressure difference in heart failure
51. What factor induces sclerosis?
	1. Cell lesions \*
	2. Cell mitosis cessation
	3. Primary hypofunction of the organ
	4. Growth factors lack
	5. Apoptosis
52. What factors can cause hyperglycemia?
	1. reduced glycogenolysis
	2. enhanced glycogenolysis \*
	3. enhanced glycolysis
	4. reduced gluconeogenesis
	5. enhanced gluconeogenesis \*
53. What factors can cause necrosis of the oral cavity?
	1. Lead compounds \*
	2. Ibuprofen administration
	3. Ethanol administration in high concentration \*
	4. Aspirin administration
	5. Antiviral substances administration
54. What factors can lead to inferior airways obstruction?
	1. hypersecretion of bronchial mucus \*
	2. spasm of terminal bronchioles \*
	3. spasm of tracheal muscles
	4. stenosis of the larynx
	5. spasm of laryngeal muscles
55. What factors can lead to upper airways obstruction?
	1. spasm of terminal bronchioles
	2. spasm of tracheal muscles
	3. tracheal obstruction \*
	4. stenosis of the larynx \*
	5. spasm of laryngeal muscles
56. What factors cause venous hyperemia in inflammatory focus on frog’s tongue?
	1. thrombosis in the venules \*
	2. leucocytes margination at the level of microvessels
	3. accumulation of exudate \*
	4. arteriolar dilation
	5. dilation of venules
57. What hormonal disturbance induces glycogenogenesis?
	1. Hypersecretion of insulin \*
	2. hypersecretion of glucagon
	3. Hyposecretion of insulin
	4. hypersecretion of thyroid hormones
	5. hypersecretion of glucocorticoids
58. What hormonal disturbance is characteristic for diabetes insipidus?
	1. deficiency of vasopressin \*
	2. excess of vasopressin
	3. excess of thyroxin
	4. deficiency of thyroxin
	5. deficiency of cortisol
59. What hormonal disturbance is characteristic for myxedema?
	1. deficiency of triiodothyronine \*
	2. excess of triiodothyronine
	3. deficiency of thyroxin \*
	4. excess of thyroxin
	5. deficiency of cortisol

1. What hormonal disturbances induce glycogenolysis?
	1. Hypersecretion of insulin
	2. hypersecretion of glucagon \*
	3. hyposecretion of glucagon
	4. hyposecretion of thyroid hormones
	5. hypersecretion of thyroid hormones \*
2. What hormonal disturbances induce hyperglycemia?
	1. Hypersecretion of insulin
	2. Hypersecretion of glucagon \*
	3. Hypersecretion of glucocorticoids \*
	4. Hyposecretion of glucagon
	5. Hyposecretion glucocorticoids
3. What hormonal disturbances induce hyperlipidemia?
	1. Hypersecretion of insulin
	2. Hyposecretion of insulin \*
	3. Hypersecretion of glucagon \*
	4. Hyposecretion of glucagon
	5. Hyposecretion of glucocorticoids
4. What hormonal disturbances induce hypoglycemia?
	1. hypersecretion of insulin \*
	2. hyposecretion of insulin
	3. hypersecretion of thyroid hormones
	4. hyposecretion of thyroid hormones \*
	5. hyposecretion of glucocorticoids \*
5. What hormonal disturbances induce proteolysis?
	1. Excess of insulin
	2. Excess of glucocorticoids \*
	3. Deficiency of glucocorticoids
	4. Excess of thyroid hormones \*
	5. Deficiency of thyroid hormones
6. What intracellular dyshomeostasis results from cessation of membrane ionic pumps function?
	1. Increased concentration of Ca2+ ions in hyaloplasm \*
	2. Increased concentration of K+ ions in hyaloplasm
	3. Increased concentration of Na+ ions in hyaloplasm \*
	4. Increased concentration of Ca2+ ions in the endoplasmic reticulum
	5. Increased concentration of Ca2+ ions in interstitial space
7. What ions have vasoconstrictive effect?
	1. sodium
	2. potassium
	3. calcium \*
	4. hydrogen
	5. magnesium
8. What ions have vasodilatory effect?
	1. zinc
	2. potassium \*
	3. hydrogen \*
	4. carbon
	5. calcium
9. What is a secondary endogenous pyrogenic factor?
10. products of cell necrosis
11. prostaglandins
12. acute phase proteins \*
13. immunoglobulin
14. bacterial endotoxin released in the blood
15. What is adaptive reaction?
16. it is a reaction that is oriented to the organism survival in the new life conditions \*
17. it is a reaction that is oriented to prevent, attenuate and eliminate the action of pathogenic factor
18. it is a reaction that is oriented to ensure the functional homeostasis in damaged structures by the hyperfunction of other synergistic structures
19. it is a reaction that is oriented to recover the structural defect and restoration of structural homeostasis
20. it is a reaction that is oriented to change the genotype according to life conditions
21. What is characteristic for neuroparalytic mechanism of arterial hyperemia?
	1. Decreased parasympathetic influences on arterioles
	2. Decreased vascular reactivity to acetylcholine
	3. Decreased vascular reactivity to catecholamines \*
	4. Increased parasympathetic influences on arterioles
	5. Decreased sympathetic influences on arterioles \*
22. What is characteristic for neurotonic mechanism of arterial hyperemia?
	1. Decreased parasympathetic influences of arterioles
	2. Increased parasympathetic influences on arteries with their dilation
	3. Increased parasympathetic influences on arterioles \*
	4. Decreased sympathetic influences on arteries
	5. Decreased vascular reactivity to catecholamines
23. What is characteristic for the latent period of the disease?
	1. absence of any clinical manifestations \*
	2. absence of specific manifestations
	3. Presence of non-specific manifestations
	4. Presence of both specific and non-specific manifestations
	5. absence of non-specific manifestation
24. What is characteristic for the period of complete disease manifestation?
25. absence of any clinical manifestations
26. absence of specific manifestations
27. presence of non-specific manifestations
28. presence of both specific and non-specific manifestations \*
29. disappearance of the disease’s manifestations
30. What is characteristic for the prodromal period of the disease?
	1. absence of any clinical manifestations
	2. absence of specific manifestations \*
	3. presence of non-specific manifestations \*
	4. presence of both specific and non-specific manifestations
	5. disappearance of the disease’s manifestations
31. What is compensatory reaction?
32. it is a reaction that is oriented to the organism survival in the new life conditions
33. it is a reaction that is oriented to prevent, attenuate and eliminate the action of pathogenic factor
34. it is a reaction that is oriented to ensure the functional homeostasis in damaged structures by the hyperfunction of other synergistic structures \*
35. it is a reaction that is oriented to recover the structural defect and restoration of structural homeostasis
36. it is a reaction that is oriented to change the genotype according to life conditions
37. What is general etiology?
	1. The compartment of pathophysiology that studies causes and conditions of the disease’s onset \*
	2. The compartment of pathophysiology that studies causes of the disease’s development
	3. The compartment of pathophysiology that studies conditions of the disease’s development
	4. The compartment of pathophysiology that studies causes and conditions of the disease’s development and evolution
	5. The compartment of pathophysiology that studies the role of pathogenetic factors in disease’s evolution
38. What is mechanism of hypothyroidism at methyluracil administration?
	1. Intensification of iodine absorption from the blood
	2. Intensification of iodine uptake by the thyrocytes
	3. Inhibition of iodine uptake by the thyrocytes
	4. Destruction of the thyroglobulin in thyrocytes
	5. Inhibition of thyroid hormones exocytosis \*
39. What is normal blood glucose level?
	1. 120 -140 mg/dl
	2. 60 -80 mg/dl
	3. 5,5 -6,0 mmol/L \*
	4. 4,5 – 5,5 mmol/L
	5. 80 – 120 mg/dl \*
40. What is one of the consequences of sclerosis?
	1. Organ malignancy
	2. Fatty dystrophy of the organ
	3. Organ deformation \*
	4. Hypertrophy of the organ
	5. Cell dedifferentiation
41. What is pathogenetic therapy?
42. the therapy oriented to remove the cause of disease from the organism
43. the therapy oriented to remove the primary injuries
44. the therapy oriented to attenuate the pathogenic action of the etiological factor
45. the therapy oriented to remove the main pathogenetic link \*
46. active or passive immunization
47. What is pathological regeneration?
	1. Dysplasia \*
	2. Metaplasia \*
	3. Hyperplasia
	4. Hypertrophy
	5. Anaplasia

1. What is protective reaction?
2. it is a reaction that is oriented to the organism survival in the new life conditions
3. it is a reaction that is oriented to prevent, attenuate and eliminate the action of pathogenic factor \*
4. it is a reaction that is oriented to ensure the functional homeostasis in damaged structures by the hyperfunction of other synergistic structures
5. it is a reaction that is oriented to recover the structural defect and restoration of structural homeostasis
6. it is a reaction that is oriented to change the genotype according to life conditions
7. What is reparative reaction?

a) it is a reaction that is oriented to the organism survival in the new life conditions

b) it is a reaction that is oriented to prevent, attenuate and eliminate the action of pathogenic factor

c) it is a reaction that is oriented to ensure the functional homeostasis in damaged structures by the hyperfunction of other synergistic structures \*

d) it is a reaction that is oriented to recover the structural defect and restoration of structural homeostasis

e) it is a reaction that is oriented to change the genotype according to life conditions

1. What is the biological significance of allergic reactions?
2. represents a pure immune reaction which underlies on the basis of natural immunity
3. represents an immune reaction with elements of cell injury \*
4. represents an immune reaction associated with inflammation \*
5. represents a pure immune reaction which underlie on the basis of adaptive immunity
6. represents a pure inflammatory reaction
7. What is the biological significance of fever?
8. reduces the systemic manifestations of inflammation
9. stimulates phagocytosis
10. inhibits allergic reactions
11. reduces the local manifestations of inflammation
12. enhances the bacteriostatic effect of antibiotics
13. What is the cause of hypophosphatemia?
	1. hypersecretion of PTH \*
	2. hyposecretion of PTH
	3. hypersecretion of calcitonin
	4. hyposecretion of calcitonin
	5. metabolic alkalosis \*
14. What is the cause of sinus tachycardia?
	1. increased intracerebral pressure
	2. intoxication with digitalis
	3. hypothermia
	4. hyperthermia \*
	5. vagotonia
15. What is the correlation between the inflow and outflow of blood in arterial hyperemia?
16. Inflow and outflow are reduced
17. Inflow is increased and outflow is reduced
18. Inflow and outflow are enhanced \*
19. Outflow is increased and inflow is unchanged
20. Inflow is increased and outflow is unchanged
21. What is the definition of fever?
22. pathologic compensatory reaction which is manifested by increased body’s temperature
23. protective physiological reaction manifested by increased body’s temperature
24. disturbance of thermoregulatory center manifested by increased body temperature
25. pathologic process manifested by restructuring in activity of thermoregulatory center and increased body temperature \*
26. pathologic state caused by increased body temperature in increased environmental temperature
27. What is the etiologic factor of lymphocytosis?
	1. bacterial infection
	2. viral infection \*
	3. acute infection disease
	4. chronic infection disease \*
	5. allergic disease
28. What is the feature of delayed hypersensibility?
29. underlies on the basis of humoral immune reactions
30. underlies on the basis of cellular immune reactions \*
31. underlies on the basis of acute inflammatory reaction
32. underlies on the basis of chronic inflammatory reaction
33. underlies on the basis of mixed immune reactions - humoral and cellular
34. What is the feature of immediate hypersensibility?
	1. underlies on the basis of humoral immune reactions \*
	2. underlies on the basis of cellular immune reactions
	3. underlies on the basis of acute inflammatory reaction
	4. underlies on the basis of chronic inflammatory reaction
	5. underlies on the basis of mixed immune reactions - humoral and cellular
35. What is the final effect of allergic reactions type IV?
36. exudative inflammation
37. fibrinous inflammation
38. proliferative inflammation \*
39. necrosis with scarring
40. purulent inflammation
41. What is the hallmark of purulent exudate?
	1. small molecular weight proteins up to 2-3%
	2. high molecular weight proteins up to 2-3%
	3. many polymorphonuclear leukocytes \*
	4. hig level of fibrinogen and fibrin
	5. presence of lysosomal enzymes
42. What is the main link of pathogenesis?
43. the cause that provoked disease
44. the injuries caused by the action of the primary cause
45. the injuries that provoke directly the death of the body
46. the pathogenetic factor on which depends the development of the disease and which removal can stop the disease \*
47. the pathogenetic factor caused by the primary cause action, on which depends the development of the disease and which removal can stop the disease

1. What is the mechanism of increased blood pressure in painful excitation?
	1. high secretion of catecholamines \*
	2. increased number of adrenoreceptors \*
	3. increased peripheral vascular resistance
	4. activation of MAO
	5. activation of kallikrein-kinin system
2. What is the mechanism of leukocyte emigration in the inflammatory focus?
	1. the action of chemotactic factors \*
	2. increased permeability of the vessels \*
	3. active filtration of leucocytes trough vascular wall
	4. passive filtration of leukocytes through vascular wall
	5. increased hydrostatic pressure in the capillaries
3. What is the mechanism of primary alteration on frog’s tongue after administration of AgNO3 crystals?
	1. chemical cell injury \*
	2. development of venous hyperemia
	3. development of blood stasis
	4. development of arteriolar spasm
	5. thrombogenesis
4. What is the mechanism of restoration of blood pressure following painful stimulation?
	1. Activation of MAO \*
	2. Hypersecretion of acetylcholine
	3. activation of renin-angiotensin-aldosterone system
	4. blood storage in deposits
	5. centralization of hemocirculation
5. What is the mechanism of restoration of blood pressure in hypercatecholemia?
	1. activation of monoamine-oxidase \*
	2. increased level of acetylcholine
	3. activation of renin-angiotensin-aldosterone system
	4. blood storage
	5. inhibition of monoamine-oxidase

1. What is the method of measurement of blood pressure in the rabbit during experimental hypervolemia?
	1. indirect method with Riva-Roci device placed on posterior limbs
	2. indirect method with Riva-Roci device placed on anterior limbs
	3. direct intra-arterial method with hydrargyrum manometer \*
	4. indirect method with Riva-Roci device placed on thorax
	5. direct intra-arteriolar method with hydrargyrum manometer
2. What is the non-specific prophylaxis of the disease?
3. prophylaxis by active or passive immunization
4. prophylaxis by the consumption of vitamins and oligoelements \*
5. prophylaxis by „tempering” the body \*
6. prophylaxis that is effective for only one disease
7. prophylaxis that is effective for many diseases \*
8. What is the normal concentration of Ca++ ions in the blood?
	1. 1,5 – 2,5 mmol/L
	2. 2,1 – 2,6 mmol/L \*
	3. 4,5 – 5,5 mmol/L
	4. less than 1,0 mmol/L
	5. more than 3,5 mmol/L
9. What is the normal concentration of K+ ions in the blood?
	1. 5,5 - 6,5 mEq/L
	2. 3,5 – 5,5 mEq/L \*
	3. 2,5 – 3,5 mEq/L
	4. less than 2,5 mEq/L
	5. less than 1,5 mEq/L
10. What is the normal concentration of Na+ ions in the blood?
	1. less than 100 - mEq/L
	2. 100 -125 mEq/L
	3. 135 - 145 mEq/L \*
	4. 140 -160 mEq/L
	5. above 300 mEq/L
11. What is the pathogenesis of allergic reaction type II?
12. antigen-antibody interaction on T cell membrane
13. antigen-antibody interaction on mast cell membrane
14. antigen-antibody interaction on the surface of parenchymal cells \*
15. interaction of parenchymal cells with sensitized T cells
16. antigen-antibody interaction on the surface of mesenchymal cells
17. What is the pathogenesis of fever?
18. increased body temperature in increased environmental temperature
19. increased thermolysis
20. reduced thermolysis
21. excessive catabolic processes in the body
22. restructuration in the activity of thermoregulatory center
23. What is the pathogenesis of inflammatory venous hyperemia?
24. effect of fraction C3a and C5a of the complement system
25. effect of bradykinin released into inflammatory focus
26. effect of histamine released into inflammatory focus
27. endothelial cell contraction \*
28. alteration of rheological proprieties of the blood \*
29. What is the pathogenesis of physiological phase in allergic reactions type IV?
30. direct action of cytotoxic lymphocytes
31. proteolysis performed by lysosomal enzymes
32. effects of lymphotoxin \*
33. direct action of IgM and IgG \*
34. direct action of natural killer lymphocytes
35. What is the pathogenesis of polyuria in insulin deficiency?
	1. insulin deficiency – ADH hypersecretion- inhibition of water canalicular reabsorbtion – polyuria
	2. insulin deficiency – hyperglycemia – increased glomerular filtration – polyuria
	3. insulin deficiency – hyperglycemia – incomplete glucose reabsorbtion – glucosuria – polyuria \*
	4. insulin deficiency – hyperglycemia – inhibition of aldosterone secretion – hypernatremia – polyuria
	5. insulin deficiency – hyperglycemia – glucosuria – blockade of aquaporine – polyuria
36. What is the pathogenesis of proliferation in the inflammatory focus?
	1. is the effect of substances with proliferative effect released in the inflammatory focus by microbes
	2. is the effect of substances with proliferative effect released in the inflammatory focus from mesenchymal structures \*
	3. is the effect of substances with proliferative effect released in the inflammatory focus from thrombocytes
	4. is the effect of substances with proliferative effect released in the inflammatory focus from parenchymal structures
	5. deficiency of substances with inhibitory effects on proliferative processes in the inflammatory focus
37. What is the pathogenetic mechanism of decreased volume of the ischemic organ?
38. Increased volume of interstitial fluid
39. Decreased volume of interstitial fluid \*
40. Increased volume of intracellular fluid
41. Decreased volume of intracellular fluid \*
42. Reduced lymphogenesis and lymphodynamics
43. What is the pathogenetic role of ATP depletion in necrosis?
	1. Increases of AMP concentration \*
	2. Decreases of AMP concentration
	3. Activation of anaerobic glycolysis that lead to intracellular acidosis \*
	4. Activation of aerobic glycolysis that lead to intracellular alkalosis
	5. inhibition of anaerobic glycolysis that lead to intracellular acidosis
44. What is the pathogenetic role of free radicals in necrosis?
45. Peroxidation of membrane phospholipids \*
46. Peroxidation of saturated fatty acids
47. Peroxidation of carbohydrates
48. Peroxidation of cations
49. Peroxidation of nucleic acids \*
50. What is the pathogenetic role of hypoxia in necrosis?
	1. Increased resting potential
	2. Inhibitory depolarization \*
	3. Dysfunction of the membrane ionic pumps \*
	4. Dysfunction of the membrane ionic channels
	5. Inhibitory hyperpolarization
51. What is the physiological role of Ca++ ions in the body?
	1. acts as intracellular second messenger \*
	2. maintains plasma osmotic pressure
	3. provides electrical charge to plasma proteins
	4. participates in protein synthesis
	5. maintains resting membrane potential of excitable cells
52. What is the physiological role of potassium in the body?
	1. maintains the plasmatic osmotic pressure
	2. maintains the plasmatic oncotic pressure
	3. ensures the active membrane potential in the excitable cells
	4. ensures the resting membrane potential in the excitable cells \*
	5. maintains the threshold potential in the excitable cells
53. What is the possible consequence of direct absorption of protein from the digestive tract?
	1. hyperproteinaemia
	2. food allergy \*
	3. hypoproteinemia
	4. anaphylactic shock
	5. infiltration of the liver with protein
54. What is the result of proliferation in the inflammatory focus?
	1. restoration of altered parenchymal structures
	2. restoration of altered mesenchymal structures \*
	3. abundance increases of parenchymal structures
	4. angiogenesis de novo
	5. tissue hyperplasia
55. What is the role of conditions in the disease appearance?
	1. determines the possibility of the disease appearance
	2. determines the specificity of the disease
	3. determines the moment of the disease appearance
	4. impedes the appearance of the disease \*
	5. accelerates the appearance of the disease \*
56. What is the role of the cause in the disease appearance?
	1. determines the possibility of the disease appearance \*
	2. determines the specificity of the disease \*
	3. determines the moment of the disease appearance
	4. impedes the appearance of the disease
	5. accelerates the appearance of the disease
57. What is the sequence of leukocyte emigration into the inflammatory site?
	1. granulocytes - monocytes – lymphocytes \*
	2. polymorphonuclear - monocytes – lymphocytes
	3. lymphocytes - granulocytes – monocytes
	4. granulocytes - lymphocytes – monocytes
	5. monocytes - granulocytes – lymphocytes
58. What is the sequence of vascular reactions in inflammatory focus on frog’s tongue?
59. arterial hyperemia→ venous hyperemia →ischemia → stasis
60. venous hyperemia → arterial hyperemia → ischemia → stasis
61. venous stasis → venous hyperemia → arterial hyperemia → ischemia
62. ischemia → arterial hyperemia → venous hyperemia → stasis \*
63. ischemia → venous hyperemia → arterial hyperemia → stasis
64. What is the sign of absolute leucocytosis?
	1. increased number of young and mature leucocytes in the blood
	2. increased number of young leucocytes in the blood
	3. total blood count of leucocytes is 6-7×109/L
	4. decreased production of leucocytes in the bone marrow
	5. increased number of mature leukocytes in the blood \*
65. What is the significance of the increased intracellular enzymes activity in the blood?
	1. Activation of the intracellular metabolic processes
	2. Activation of metabolic processes
	3. Activation of catalytic activity of enzymes that lead to cell apoptosis
	4. Activation of catalytic activity of enzymes that lead to cell injuries \*
	5. Activation of catalytic activity of enzymes that lead to cell dystrophy
66. What is the specific prophylaxis of the disease?
67. prophylaxis by active or passive immunization \*
68. prophylaxis by the consumption of vitamins and oligoelements
69. prophylaxis by „tempering” the body
70. prophylaxis that is effective only for one disease \*
71. prophylaxis that is effective for many diseases
72. What is the stress hormone?
	1. Cortisol \*
	2. Insulin
	3. Epiandrosteron
	4. Somatotropin
	5. Thyroxin
73. What is the symptomatic therapy?
74. the therapy oriented to remove the primary injuries
75. the therapy oriented to attenuate the pathogenic action of the etiological factor
76. the therapy oriented to remove the main pathogenetic link
77. the therapy oriented to remove the main clinical manifestations
78. the therapy oriented to remove the disturbances that threatens the patient’s life \*
79. What lipid substances are synthesized in the body?
	1. triglyceride \*
	2. urea
	3. polyunsaturated fatty acids
	4. Phospholipids \*
	5. liposoluble vitamins
80. What manifestations develop in the mouse in condition of normobaric hypoxia?
	1. hyperemia of sclera
	2. cyanosis \*
	3. tachycardia \*
	4. paleness
	5. meteorism
81. What mechanism determines the resistance to the action of stressful factors?
	1. Hyperglycemia \*
	2. Hypoglycemia
	3. Heart hyperfunction \*
	4. Heart hypofunction
	5. Increased the body’s needs of O2 \*
82. What mechanisms are specific for functional arterial hyperemia?
83. Metabolic \*
84. Neurotonic
85. Mechanical
86. Neuroparalytic
87. Humoral
88. What mechanisms reduce the resistance of the rat with experimental hyperthyroidism to hypoxia?
	1. Decreased oxido- reduction reactions
	2. increased O2 consumption \*
	3. decreased basal metabolic rate
	4. exhaustion of neuronal metabolic substrate \*
	5. decreased O2 consumption
89. What mediators are produced in the mast cells via cyclooxygenase pathway?
90. histamine
91. chemotactic factors
92. platelet activating factor
93. leukotrienes
94. prostaglandins \*
95. What mediators are produced in the mast cells via lipoxygenase pathway?
96. histamine
97. chemotactic factors
98. platelet activating factor
99. leukotrienes \*
100. prostaglandins
101. What microcirculatory disorders lead to development of stasis on frog’s tongue?
	1. ischemia
	2. venous hyperemia \*
	3. arterial hyperemia
	4. embolism
	5. thrombosis \*
102. What pathogenic factors increase heart afterload?
	1. stenosis of aortic valves \*
	2. insufficiency of mitral valves
	3. enhanced circulatory blood volume
	4. insufficiency of aortic valves
	5. increased peripheral vascular resistance \*
103. What pathogenic factors increase heart preload?
	1. stenosis of aortic valves
	2. insufficiency of mitral valves \*
	3. stenosis of mitral valves
	4. insufficiency of aortic valves \*
	5. increased peripheral vascular resistance
104. What pathogenic factors induce homeometric heart hyperfunction?
	1. stenosis of aortic valves \*
	2. insufficiency mitral valves
	3. enhanced circulatory blood volume
	4. insufficiency of aortic valves
	5. increased peripheral vascular resistance \*
105. What pathogenic factors trigger heterometric heart hyperfunction?
	1. stenosis of aortic valves
	2. insufficiency mitral valves \*
	3. enhanced circulatory blood volume
	4. insufficiency of aortic valves \*
	5. increased peripheral vascular resistance
106. What pathologic processes disturb digestion in the mouth?
	1. Hypersalivation
	2. Hyposalivation \*
	3. lack of salivary amylase \*
	4. lack of lysozyme
	5. alkaline reaction of the saliva
107. What pathological process is associated with hemic hypoxia?
	1. hemolysis \*
	2. formation of carbhemoglobin
	3. reduced circulatory blood volume
	4. cardiac insufficiency
	5. vascular insufficiency
108. What pathological processes are activated during hypoxia?
	1. hypersecretion of glucocorticoids \*
	2. activation of respiratory chain enzyme
	3. decreased activity of antioxidant system
	4. hyposecretion of glucocorticoids
	5. reduced activity of the lysosomal enzymes
109. What pathological processes are developed in dystrophy of desmodontal structures?
	1. Progressive tissues atrophy
	2. Progressive tissues hypertrophy
	3. Progressive tissue hyperplasia
	4. It is followed by retraction of periodontium
	5. It is not followed by retraction of periodontium
110. What pathological processes can lead to development of vascular purpura?
111. disorders of vascular intima trophicity \*
112. vascular hyperpermeability \*
113. changes of vascular tonus
114. spasm of smooth muscles of vessels
115. atheromatous change of blood vessels
116. What pathological states are associated with hyperproteinaemia?
	1. Hemodilution
	2. Hemorrhage \*
	3. renal insufficiency
	4. diarrhea \*
	5. polyuria
117. What pathological states are associated with hypoproteinemia?
	1. diarrhea
	2. Hemodilution \*
	3. hemoconcentration
	4. polyuria
	5. combustions with plasmorrhagia \*
118. What process is disturbed in hypoplastic anemia?
	1. proliferation of erythroblastic series \*
	2. differentiation of erythroblastic series
	3. hemoglobin synthesis
	4. erythrodieresis
	5. erythrocytes maturation
119. What process is disturbed in iron deficiency anemia?
	1. proliferation of erythroblast series
	2. differentiation of erythroblast series
	3. hemoglobin synthesis \*
	4. erythrodieresis
	5. erythrocyte maturation
120. What processes are disturbed in B12 deficiency anemia?
	1. proliferation of erythroblast series \*
	2. differentiation of erythroblast series
	3. hemoglobin synthesis
	4. erythrodieresis
	5. erythrocyte maturation \*
121. What processes are disturbed in hemolytic anemia?
	1. proliferation of erythroblast series
	2. differentiation of erythroblast series
	3. hemoglobin synthesis
	4. erythrodieresis \*
	5. erythrocyte maturation
122. What represent achlorhydria?
	1. lack of Cl ions in the blood
	2. absence of HCl in gastric juice \*
	3. lack of enzymes in gastric juice
	4. increased blood pH
	5. decreased blood pH
123. What represents acholia?
	1. lack of bile in the blood
	2. lack of bile in intestine \*
	3. presence of bile in the blood
	4. decoloration of feces
	5. lack of bilirubin in bile
124. What represents hypersalivation?
	1. saliva secretion more than 2L/24 h
	2. saliva secretion more than 1L/24 h
	3. saliva secretion more than 1,5L/24h \*
	4. saliva secretion more than 0,5 L/24h
	5. saliva secretion more than 0,3L/24 h
125. What represents steatorrhea?
	1. presence of lipids in the blood
	2. excessive elimination of lipids with stool \*
	3. excessive accumulation of lipids in hepatic parenchyma
	4. lipid elimination with urine
	5. lack of lipids in feces
126. What stomach digestive changes can be found in hypochlorhydria?
	1. develops maldigestion of polysaccharides
	2. develops maldigestion of proteins \*
	3. develops maldigestion of lipids \*
	4. improvement of gastric digestion
	5. develops maldigestion of cellulose

1. What structure is the most sensitive to hypoxia?
	1. bones
	2. nervous tissue \*
	3. connective tissue
	4. cartilage
	5. myocardium
2. What structures are frequently involved in allergic reactions type III?
3. lymph nodes \*
4. skeletal muscles
5. renal glomeruli \*
6. gastrointestinal tract
7. bronchial mucosal layer
8. What structures of cytoplasmic membrane are damaged and lead to disintegration of the cell?
	1. Membrane glycoproteins
	2. Membrane channels \*
	3. Membrane receptors
	4. Membrane pumps \*
	5. Hormonal cytoplasmic receptors
9. What substances are complete antigens?
10. nucleoproteins
11. proteins \*
12. lipopolysaccharides \*
13. simple organic substances
14. inorganic substances
15. What substances are incomplete antigens?
16. nucleoproteins
17. proteins
18. lipopolysaccharides
19. simple organic substances \*
20. inorganic substances \*
21. What type of hormone is increased in gigantism?
	1. triiodothyronine
	2. somatotropin \*
	3. tetraiodthyronine
	4. cortisol
	5. somatoliberine
22. What type of hormones are increased in Graves-Bazedov disease?
	1. Triiodothyronine \*
	2. somatotropin
	3. tetraiodthyronine \*
	4. cortisol
	5. somatoliberine
23. What types of embolisms are considered as endogenous?
24. Atheromatous \*
25. Gaseous
26. With amniotic fluid \*
27. With air
28. Microbial
29. When can develop dental hyperesthesia?
	1. thin enamel layer \*
	2. naked dentin \*
	3. injuries at the level of sensorial neurons in the spinal chord
	4. increased excitability threshold
	5. injuries at the level of sensorial neurons in the cerebral cortex
30. When can develop pulp pain?
	1. disorders of microcirculation \*
	2. excessive osteoblast proliferation
	3. excessive osteoclast proliferation
	4. edema in tooth alveoli \*
	5. parodontosis
31. When the antipyretic therapy is justified?
32. hyperergic inflammation \*
33. autoimmune processes
34. allergic diseases
35. moderate fever but intolerable for patients \*
36. in all cases of fever, antipyretic therapy is indicated
37. When the pyrotherapy is justified?
38. hypoergic inflammation \*
39. chronic inflammation \*
40. acute inflammation
41. inflammatory reaction with immunodeficiency
42. hyperergic inflammation
43. Which are consequences of insufficient pancreatic secretion?
	1. Maldigestion \*
	2. Malabsorbtion \*
	3. Malnutrition
	4. ulcerogenesis
	5. constipations
44. Which are manifestations of infectious hepatitis in organs of the mouth?
	1. edema of the mouth mucosa
	2. jaundice of the mouth mucosa \*
	3. teleagiectasia \*
	4. Fourdis granules
	5. paleness of mouth mucosa
45. Which are the biochemical manifestations of sever cholemia?
	1. hyperbilirubinemia with free bilirubin
	2. hyperbilirubinemia with conjugated bilirubin \*
	3. hypercholesterolemia \*
	4. cholalemia
	5. hypoprothrombinemia
46. Which are the consequences of choledocus obstruction?
	1. hyperbilirubinemia with free bilirubin
	2. cholestasis
	3. acholia \*
	4. hyperbilirubinemia with conjugated bilirubin \*
	5. lipid maldigestion \*
47. Which are the signs of cardiac insufficiency?
	1. systolic volume less than 60 ml
	2. systolic volume less than 50 ml
	3. cardiac output less than 5 L/min
	4. cardiac output less than 4L/min
	5. blood circulation time 20-23 sec
48. Which conditions are endogenous?
	1. Climate factors
	2. Microclimate factors
	3. The body’s constitution \*
	4. The body’s reactivity \*
	5. The body’s resistance \*
49. Which conditions are exogenous?
	1. Ecological factors \*
	2. Climate factors \*
	3. The body’s constitution
	4. The body’s reactivity
	5. The body’s resistance
50. Which factors can cause the disease development?
51. Action of energy on the organism \*
52. Action of substance on the organism \*
53. Action of information on the organism \*
54. Action of the heterogenous biological field on the organism
55. Action of hostile aura on the other person
56. Which hormone has anabolic effect?
57. Insulin \*
58. glucagon
59. glucocorticoids
60. thyroid hormones
61. parathyroid hormone
62. Which hormones have catabolic effect?
	1. insulin
	2. glucagon \*
	3. glucocorticoids \*
	4. thyroid hormones \*
	5. parathyroid hormone
63. Which structures from oral cavity have high regenerative potential?
	1. Oral cavity epithelium \*
	2. Chewing muscles
	3. Granular periodontal tissue \*
	4. Tooth enamel
	5. Odontoblasts \*
64. Which type of hypoxia does develop in alpine disease?
	1. exogenous normobaric hypoxia
	2. exogenous hyperbaric hypoxia
	3. exogenous hypobaric hypoxia \*
	4. respiratory hypoxia
	5. hystotoxic hypoxia