1. **What does general pathophysiology study**?
2. General rules of the origin, onset, evolution and resolution of typical pathological processes
3. General rules of the origin, onset, evolution and resolution of pathological processes in organs and systems
4. General rules about pathogenesis of clinical syndromes and nosological entities
5. General rules about structural modifications and dysfunctions at the cellular, tissue, system and integral level in typical pathological processes
6. General rules about structural modifications and dysfunctions of organs and systems in typical pathological processes
7. **What does special pathophysiology study?**
8. General rules of the origin, onset, evolution and resolution of typical pathological processes
9. General rules of the origin, onset, evolution and resolution of pathological processes in organs and systems
10. General rules about the pathogenesis of clinical syndromes and nosological entities
11. General rules about structural modifications and dysfunctions at the cellular, tissue, system and integral level in typical pathological processes
12. General rules about structural modifications and dysfunctions of organs and systems in pathological processes
13. **What does clinical pathophysiology study?**
14. General rules of the origin, onset, evolution and resolution of typical pathological processes
15. General rules of the origin, onset, evolution and resolution of pathological
16. General rules about the pathogenesis of clinical syndromes and nosological entities
17. General rules about structural modifications and dysfunctions at the cellular, tissue, system and integral level in typical pathological processes
18. General rules about structural modifications and dysfunctions of organs and systems in pathological processes
19. **What is general etiology?**
20. The compartment of pathophysiology that studies causes and conditions of the disease’s onset
21. The compartment of pathophysiology that studies causes of the disease’s development
22. The compartment of pathophysiology that studies conditions of the disease’s development
23. The compartment of pathophysiology that studies causes and conditions of the disease’s development and evolution
24. The compartment of pathophysiology that studies the role of pathogenetic factors in disease’s evolution
25. **Which factors can cause the disease development?**

a) Action of energy on the organism

b) Action of substance on the organism

c) Action of information on the organism

d) Action of the heterogenous biological field on the organism

e) Action of hostile aura on the other person

6. **What are the exogenous causes of diseases?**

a) Mechanical, physical, chemical and biological factors from the environment

b) Resident microflora of the intestines and respiratory pathways

c) Intestinal parasites and blood parasites

d) Chromosome aberration resulting from the action of ionizing radiation

e) inherited genetic defects

7. **What are the endogenous causes of diseases?**

a) Mechanical, physical, chemical and biological factors from the environment

b) Resident microflora of the intestines and respiratory pathways

c) Intestinal parasites and blood parasites

d) Chromosome aberration resulting from the action of ionizing radiation

e) Inherited genetic defects

8. **What conditions are necessary for the disease onset?**

a) Different forms of energy

b) Material factors

c) Informational factors

d) Proper and heterogenous biological field

e) Interaction between proper aura and aura of another person

1. **What conditions are considered favorable for the organism?**
2. Conditions that promote action of the cause and disease appearance
3. Conditions that impede action of the cause and retain the disease appearance
4. Conditions that decrease body’s resistance
5. Conditions that increase the body’s resistance
6. Genetic defects
7. **What conditions are considered unfavorable for the organism?**
8. Conditions that promote action of the cause and disease appearance
9. Conditions that impede action of the cause and retain the disease appearance
10. Conditions that decrease the organism’s resistance
11. Conditions that increase the organism’s resistance
12. Genetic defects
13. **Which conditions are exogenous?**
14. Ecological factors
15. Climate factors
16. The body’s constitution
17. The body’s reactivity
18. The body’s resistance
19. **Which conditions are endogenous?**
20. Climate factors
21. Microclimate factors
22. The body’s constitution
23. The body’s reactivity
24. The body’s resistance
25. **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
26. **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
27. **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
28. **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
29. **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
30. **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
31. **What is the main link of pathogenesis?**

a) the cause that provoked disease

b) the injuries caused by the action of the primary cause

c) the injuries that provoke directly the death of the body

d) the pathogenetic factor on which depends the development of the disease and which removal can stop the disease

e) 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 pathogenetic therapy?**

a) the therapy oriented to remove the cause of disease from the organism

b) the therapy oriented to remove the primary injuries

c) the therapy oriented to attenuate the pathogenic action of the etiological factor

d) the therapy oriented to remove the main pathogenetic link

e) active or passive immunization

**25. What is the symptomatic therapy?**

a) the therapy oriented to remove the primary injuries

b) the therapy oriented to attenuate the pathogenic action of the etiological factor

c) the therapy oriented to remove the main pathogenetic link

d) the therapy oriented to remove the main clinical manifestations

e) the therapy oriented to remove the disturbances that threatens the patient’s life

1. **What is the specific prophylaxis of the disease?**

a) prophylaxis by active or passive immunization

b) prophylaxis by the consumption of vitamins and oligoelements

c) prophylaxis by „tempering” the body

d) prophylaxis that is effective only for one disease

e) prophylaxis that is effective for many diseases

1. **What is the non specific prophylaxis of the disease?**

a) prophylaxis by active or passive immunization

b) prophylaxis by the consumption of vitamins and oligoelements

c) prophylaxis by „tempering” the body

d) prophylaxis that is effective for only one disease

e) prophylaxis that is effective for many diseases

1. **What does a physiological reaction mean?**

 a) it is a reaction that is adequate to the specific excitation

 b) it is a reaction that has a dyshomeostatic character

 c) it is a reaction that has a homeostatic character

 d) it is a reaction that is inferior to the excitant’s intensity

 e) it is a reaction that exceeds the excitant’s intensity

1. **What does a pathological reaction mean?**

a) it is a reaction that doesn’t correspond to the excitant’s specificity

 b) it is a reaction that quantitatively corresponds to the excitant’s intensity

 c) it is a reaction that has a homeostatic character

 d) it is a reaction that is inferior to the excitant’s intensity

 e) it is a reaction that exceeds the excitant’s intensity

1. **What is adaptive 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 compensatory 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 protective 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 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 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
2. **What is characteristic for the prodromal period of the disease?**

a) absence of any clinical manifestations

b) absence of specific manifestations

c) presence of non-specific manifestations

d) presence of both specific and non-specific manifestations

e) disappearance of the disease’s manifestations

1. **What is characteristic for the period of complete disease manifestation?**

a) absence of any clinical manifestations

b) absence of specific manifestations

c) presence of non-specific manifestations

d) presence of both specific and non-specific manifestations

e) disappearance of the disease’s manifestations

1. **What does the pathological process include?**

a) the totality of injuries caused by the action of primary cause

b) the totality of injuries caused by the action of primary cause and the subsequent pathogenetic factors

c) the totality of injuries caused by the action of primary cause plus the subsequent pathogenetic factors plus the protective, compensatory and reparative reactions

d) the totality of local injuries

e) the totality of local and general injuries

1. **What does the disease represent?**

a) the combination of injuries and homeostatic reactions of the organism

b) the combination of local and general injuries

c) the combination of structural and functional processes

d) the combination of pathogenetic and sanogenetic processes

e) the pathological process localized in one organ

1. **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
2. **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
3. **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. Myocytes contraction
4. **What are the consequences of activation of the non specific 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
5. **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
6. **What are the consequences of the intracellular proteases activation?**
	1. Breakdown of the intracellular proteic complexes and initiation of the cell autolysis
	2. Breakdown of nucleoproteins and initiation of cell apoptosis
	3. Breakdown of macroergic complexes and energy depletion in the cell
	4. Breakdown of membrane phospholipids
	5. Breakdown of the intracellular proteic complexes and initiation of the cell apoptosis
7. **What are the consequences of the intracellular nucleoproteases activation?**
	1. Breakdown of intracellular proteins and initiation of the cell autolysis
	2. Breakdown of nucleic acids and initiation of cell apoptosis
	3. Breakdown of macroergic complexes and energy depletion
	4. Breakdown of membrane phospholipids
	5. Breakdown of nucleic acids and initiation of cell autolysis
8. **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
9. **What are the general causes of energy depletion that trigger cell dystrophy?**
10. Chronic local hypoxia
11. Deprivation of food
12. Acute hypoxia
13. Activation of oxidative processes by toxins
14. Activation of anabolic processes in acute stress
15. **What pathological processes are developed in dystrophy of desmodontal structures?**
16. Progressive tissues atrophy
17. Progressive tissues hypertophy
18. Progressive tissue hyperplasia
19. It is followed by retraction of periodontium
20. It is not followed by retraction of periodontium
21. **What are the organs that most often are prone to develop lipid dystrophy?**
22. liver
23. lungs
24. kidneys
25. brain
26. sexual glands
27. **What are the etiological factors of lipid dystroph**y?

alimentary hyperglycemia

deficiency of lipids in the diet

deficiency of proteins in the diet

excess of proteins in diet

Alimentary hypolipidemia

1. **What are the pathogenic mechanisms of lipid dystrophy?**
	1. inability of cell to catabolise lipid excess
	2. Increased synthesis of apoproteins and lipoproteins that increases export of lipids from cells
	3. Decreased apoprotein level and inability to export lipids form cells
	4. Hypoxia inhibits fatty acid oxidation and leads to acumulation of lipids
	5. Hypoxia activates fatty acid oxidation and leads to acumulation of lipids
2. **What are the consequences of dystrophy?**
3. inflamation
4. sclerosis
5. aplasia
6. hyperplasia
7. hypertrophy
8. **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
9. **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
10. **What are negative signals of apoptosis initiation?**
11. absence of the growth factors
12. absence of growth hormone (somatotropin)
13. absence of estrogens for the endometrium
14. absence of estrogens for the mammary gland
15. absence of prolactin for the endometrium
16. **What are the general consequences of necrosis for the organism?**
17. Cell death by necrosis initiate cell dystrophy in the affected tissue
18. Cell death by necrosis has negative consequences for the affected organ only
19. Cell death by necrosis has no negative consequences for the whole organism
20. Cell death by necrosis initiates the acute phase reaction
21. Cell death by necrosis initiates fever
22. **What are the general consequences of apoptosis for the organism?**
	1. Cell death by apoptosis initiates fever
	2. Cell death by apoptosis doesn’t provoke quantitative modifications of the cellular population
	3. Cell death by apoptosis initiates the acute phase reaction
	4. Cell death by apoptosis has no negative consequences for another structures of the organism
	5. Cell death by apoptosis has negative consequences for the whole organism
23. **What are the general consequences of the cell necrosis for the whole organism?**
24. death of the whole organism
25. enzymemia
26. hyperkaliemia
27. hypernatriemia
28. hypercloremia
29. **What is the pathogenetic role of free radicals in necrosis?**
	1. Peroxidation of membrane phospholipids
	2. Peroxidation of saturated fatty acids
	3. Peroxidation of carbohydrates
	4. Peroxidation of cations
	5. Peroxidation of nucleic acids
30. **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
31. **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 aerobicglycolysis that lead to intracellular alkalosis
		5. inhibition of anaerobic glycolysis that lead to intracellular acidosis
32. **What are the manifestations of cell necrosis?**
	1. leakage of lysososmal enzymes in hyaloplasm
	2. no leakage of lysosomal enzymes in hyaloplasm
	3. cell swelling
	4. cell shrinkage
	5. fragmentation of the cytoplasm and formation of apoptotic bodies
33. **What are the local consequences of necrosis?**

inflammation

demarcation

incapsulation

Infiltration with platelets

Infiltration with erythrocytes

1. **What factors can cause necrosis of the oral cavity?**
2. Lead compounds
3. Ibuprofen administration
4. Ethanol administration in high concentration
5. Aspirin administration
6. Antiviral substances administration
7. **What disorders can lead to ulcerative changes of oral mucosa?**
	1. Stomach ulcer
	2. Hepatitis
	3. Thyroiditis
	4. Enterocolitis
	5. Glomerulonephritis
8. **What disorders can lead to ulcerative changes of oral mucosa?**
	1. Pancreatitis
	2. Hepatitis
	3. Thyroiditis
	4. Enterocolitis
	5. Glomerulonephritis
9. **What are the conditions for formation of parietal thrombus?**
	1. exposure of subendothelial matrix
	2. turbulent blood flow
	3. enhanced blood velocity
	4. increased platelet count
	5. linear blood flow
10. **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
11. **What are the pro-coagulant factors?**
	1. plasminogen
	2. thromboplastin
	3. prothrombin
	4. antithrombin III
	5. plasmin
12. **What pathological processes can lead to development of vascular purpura?**
	1. disorders of vascular intima trophicity
	2. vascular hyperpermeability
	3. changes of vascular tonus
	4. spasm of smooth muscles of vessels
	5. atheromatous change of blood vessels
13. **What does represent thrombocytopathy**?
	1. decreased thrombocyte number
	2. thrombocyte areactivity to thrombin
	3. thrombocyte areactivity to ADP
	4. deficit of coagulation plasmatic factors
	5. excessive release of thromboxane by thrombocytes
14. **What are the manifestations of cellular alterations in the inflammatory focus?**
	1. cell injury
	2. cell dystrophy
	3. cell apoptosis
	4. fibrosis
	5. cell hyperplasia
15. **What is the sequence of vascular reactions in the inflammatory focus?**
	1. ischemia - arterial hyperemia - venous hyperemia – stasis
	2. ischemia – venous hyperemia - arterial hyperemia – stasis
	3. stasis - ischemia – arterial hyperemia - venous hyperemia
	4. ischemia - arterial hyperemia - Stasis - venous hyperemia
	5. arterial hyperemia - venous hyperemia - stasis – ischemia
16. **What are the features of inflammatory arterial hyperemia?**
	1. persistent character
	2. myoparalitic mechanism
	3. neurotonic mechanism
	4. is combined with decreased permeability of the blood vessels
	5. is combined with increased peripheral vascular resistance
17. **What is the pathogenesis of inflammatory venous hyperemia?**
	1. effect of fraction C3a and C5a of the complement system
	2. effect of bradykinin released into inflammatory focus
	3. efect of histamine released into inflammatory focus
	4. endothelial cell contraction
	5. alteration of rheological proprieties of the blood
18. **What are the characteristics of inflammatory stasis?**
	1. is associated with intravascular aggregation of blood cells
	2. enhances the spreading of secondary alteration in the inflammatory focus
	3. limits the spreading of primary alteration in the inflammatory focus
	4. limits the spreading of secondary alteration in the inflammatory focus
	5. is associated with reduced platelets aggregation
19. **What are the hallmarks of serous exudate?**
	1. small molecular weight proteins up to 2-3%
	2. high molecular weight proteins up to 2-3%
	3. many polymorphonuclear leukocytes
	4. lack of polymorphonuclear leucocytes
	5. presence of lysosomal enzymes
20. **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
21. **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
22. **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
23. **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
24. **What are general changes in inflammation?**
	1. increased level of acute-phase proteins in the blood
	2. stress
	3. reduced level of acute-phase proteins in the blood
	4. leukocytopenia
	5. immunodeficiency
25. **What are general manifestations of inflammation?**
	1. enhanced synthesis of acute-phase proteins in the liver
	2. reduced erythrocyte sedimentation rate
	3. reduced synthesis of acute-phase proteins in the liver
	4. increased erythrocytes sedimentation rate
	5. pain
26. **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. e. ) is the result of opsonization of microbes with the complement fraction C5a and easier recognition by receptors on phagocytic cells
27. **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
28. **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
29. **What is the result of proliferation in the inflammatory focus?**
	* 1. restoration of altered parenchymal structures
		2. restoration of altered mesenchymal structures
		3. abundance increase of parenchymal structures
		4. angiogenesis de novo
		5. tissue hyperplasia
30. **What is the definition of fever?**
	1. pathologic compensatory reaction which is manifested by increased body’s temperature
	2. protective physiological reaction manifested by increased body’s temperature
	3. disturbance of thermoregulatory center manifested by increased body temperature
	4. pathologic process manifested by restructuring in activity of thermoregulatory center and increased body temperature
	5. pathologic state caused by increased body temperature in increased environmental temperature
31. **What does represents fever?**
	1. persistent increasing of body temperature at the action of high environmental temperature
	2. persistent increasing of body temperature at the action of catabolic substances on the thermoregulatory center in the brain
	3. episodic increasing of body temperature at the action of excitatory substances on the SNS
	4. episodic increasing of body temperature at the action of non-infectious agents
	5. persistent increasing and maintaining of high body temperature at the action of infectious agents
32. **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
33. **What are exogenous noninfectious pyrogenic factors?**
	1. hyperimmune sera
	2. heterogeneous blood and plasma
	3. bacterial lipopolysaccharides
	4. isotonic sodium chloride solution
	5. fungal proteins
34. **What are the primary endogenous pyrogenic factors?**
	1. products of cell necrosis
	2. products of cell disintegration
	3. interleukin IL-1
	4. bacterial lipopolysaccharides
	5. tumor necrosis factor
35. **What are the secondary endogenous pyrogenic factors?**
	1. products of cell necrosis
	2. prostaglandins
	3. acute phase proteins
	4. immunoglobulin
	5. bacterial endotoxin released in the blood
36. **What is a secondary endogenous pyrogenic factor?**
	1. products of cell necrosis
	2. products of hemolysis
	3. acute phase proteins
	4. immunoglobulin
	5. progestagenic hormones
37. **What is the pathogenesis of fever?**
	1. increased body temperature in increased environmental temperature
	2. increased thermolysis
	3. reduced thermolysis
	4. excessive catabolic processes in the body
	5. restructuration in the activity of thermoregulatory center
38. **What are the mechanisms of increased thermogenesis in fever?**
	1. inhibition of catabolic reactions
	2. simpatho-adrenal inhibition
	3. activation of catabolic reactions
	4. muscle hyperkinesia
	5. activation of anabolic reactions
39. **What are the mechanisms of reduced thermolysis in the initial period of fever**?
	1. spasm of peripheral blood vessel
	2. pulmonary hypoventilation
	3. tonic muscular contractions
	4. excessive sweating
	5. pulmonary hyperventilation
40. **What are the mechanisms of enhanced thermolysis in the final stage of fever?**
	1. peripheral vasodilation
	2. bradypnea
	3. tachypnea
	4. clonic muscular contractions
	5. reduced sweating
41. **What are the mechanisms of activation of thermogenesis in fever?**
	1. tonic muscular contractions
	2. excitation of the parasympathetic nervous system
	3. activation of catabolic reactions
	4. clonic muscular contractions
	5. hypersecretion of insulin
42. **What are the metabolic changes in the second stage of fever?**
	1. enhanced anabolic processes
	2. depletion of liver glycogen
	3. enhanced lipolysis and glycogenolysis
	4. enhanced lipogenesis and glycogenogenesis
	5. positive nitrogen balance
43. **What are the cardiovascular changes in the second period of fever?**
44. tachycardia
45. generalized spasm of blood vessels
46. centralization of blood circulation and redistribution to vital organs (brain, lungs)
47. arterial hypertension
48. arterial hypotension caused by excessive sweatining
49. **What are the cardio-vascular changes in the third period of fever?**
	1. tachycardia
	2. generalized spasm of blood vessels
	3. centralization of blood circulation and redistribution to vital organs (brain, lungs)
	4. arterial hypertension caused by peripheral vasoconstriction
	5. arterial hypotension
50. **What is the biological significance of fever?**
	1. reduces the systemic manifestations of inflammation
	2. stimulates phagocytosis
	3. inhibits allergic reactions
	4. reduces the local manifestations of inflammation
	5. enhances the bacteriostatic effect of antibiotics
51. **When the pyrotherapy is justified?**
	1. hypoergic inflammation
	2. chronic inflammation
	3. acute inflammation
	4. inflammatory reaction with immunodeficiency
	5. hyperergic inflammation
52. **When the antipyretic therapy is justified?**
	1. hyperergic inflammation
	2. autoimmune processes
	3. allergic diseases
	4. moderate fever but intolerable for patients
	5. in all cases of fever, antipyretic therapy is indicated
53. **What is the biological significance of allergic reactions?**
	1. represents a pure immune reaction which underlies on the basis of natural immunity
	2. represents an immune reaction with elements of cell injury
	3. represents an immune reaction associated with inflammation
	4. represents a pure immune reaction which underlie on the basis of adaptive immunity
	5. represents a pure inflammatory reaction
54. **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
55. **What is the feature of delayed 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
56. **What substances are complete antigens?**
	1. nucleoproteins
	2. proteins
	3. lipopolysaccharides
	4. simple organic substances
	5. inorganic substances
57. **What substances are incomplete antigens?**
	1. nucleoproteins
	2. proteins
	3. lipopolysaccharides
	4. simple organic substances
	5. inorganic substances
58. **What does represents the endogenous antigen?**
	1. products of cell injury which are released in the blood
	2. products of the bacterial or fungal origin which are released in the blood
	3. natural components of the human body which lack the immunological tolerance
	4. natural components of the human body which have immunological tolerance
	5. natural components of the human body changed by action of harmful factors
59. **What are the characteristics of type I allergic reactions?**
	1. reaction between free allergen in circulation and fixed antibodies on parenchymal cells
	2. reaction between allergen fixed on the cells and free antibodies in circulation
	3. reaction between allergen and antibodies both in circulation
	4. reaction between allergen and sensitized lymphocytes
	5. reaction between free allergen in circulation and antibodies fixed on mast cells
60. **What are the characteristics of type II allergic reactions?**
	1. reaction between free allergen in circulation and fixed antibodies on parenchymal cells
	2. reaction between allergen fixed on the cells and free antibodies in circulation
	3. reaction between allergen and antibodies both in circulation
	4. reaction between allergen and sensitized lymphocytes
	5. reaction between free allergen in circulation and antibodies fixed on mast cells
61. **What are the characteristics of type III allergic reactions?**
	1. reaction between free allergen in circulation and fixed antibodies on parenchymal cells
	2. reaction between allergen fixed on the cells and free antibodies in circulation
	3. reaction between allergen and antibodies both in circulation
	4. reaction between allergen and sensitized lymphocytes
	5. reaction between free allergen in circulation and antibodies fixed on mast cells
62. **What are the end-effectors in anaphylactic allergic reactions?**
	1. immunoglobulins E
	2. immunoglobulin A
	3. immunoglobulin G4
	4. sensitized B lymphocytes
	5. sensitized T lymphocytes
63. **What mediators are produced in the mast cells via cyclooxygenase pathway?**
	1. histamine
	2. chemotactic factors
	3. platelet activating factor
	4. leukotrienes
	5. prostaglandins
64. **What mediators are produced in the mast cells via lipoxygenase pathway?**
	1. histamine
	2. chemotactic factors
	3. platelet activating factor
	4. leukotrienes
	5. prostaglandins
65. **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
66. **What are the characteristics of allergic reactions type II?**
	1. are directed against own defective, mutant, degenerated cells
	2. are directed toward own aging cells
	3. directed against own healthy cells, which are presenting the antigen
	4. directed against own healthy cells coated with antibodies IgE
	5. directed against own healthy cells coated with antibodies IgM
67. **What are the final effects in type II allergic reactions?**
	1. hemolytic anemia
	2. neutropenia
	3. lymphocytosis
	4. thrombocytosis
	5. lymphocytopenia
68. **What are the characteristics of type III allergic reaction?**
	1. interaction between antigen and IgM and IgG in the blood
	2. interaction between antigen and IgE fixed on mast cells
	3. interaction between IgM and IgG with antigen fixed on somatic cells
	4. involvement of T helper lymphocytes and B lymphocytes
	5. involvement of T helper lymphocytes and sensitized lymphocytes
69. **What are the effects of mediators involved in the allergic reaction type III?**
	1. bronchoconstriction
	2. injury of endothelial cells
	3. alterative inflammation
	4. proliferative inflammation
	5. granulomatous inflammation
70. **What structures are frequently involved in allergic reactions type III?**
	1. lymph nodes
	2. skeletal muscles
	3. renal glomeruli
	4. gastrointestinal tract
	5. bronchial mucosal layer
71. **What are local manifestations in allergic reaction type III?**
	1. arterial hyperemia
	2. proliferative inflammation
	3. ischemia
	4. infiltration with neutrophils leukocytes
	5. infiltration with T lymphocytes
72. **What disorders underlie on the basis of II allergic reactions?**
	1. myasthenia gravis
	2. endemic goiter
	3. cretinism
	4. thyroid adenocarcinoma
	5. thyrotoxicosis
73. **What is the pathogenesis of allergic reaction type II?**
	1. antigen-antibody interaction on T cell membrane
	2. antigen-antibody interaction on mast cell membrane
	3. antigen-antibody interaction on the surface of parenchymal cells
	4. interaction of parenchymal cells with sensitized T cells
	5. antigen-antibody interaction on the surface of mesenchymal cells
74. **What can be the antigen in type II allergic reaction?**
	1. specific antigens in the bloodstream
	2. specific receptors on the cell membrane
	3. exogenous substance in the bloodstream
	4. endogenous substance in the bloodstream
	5. hyperimmune sera in the blood
75. **What antigens can trigger delayed hypersensibility?**
	1. autoantigens
	2. bacteria
	3. sequestered unchanged endogenous antigen
	4. endogenous antigens changed by pathogenetic factors
	5. endogenous antigen with immunological tolerance
76. **What does represent allergic reactions type IV?**
	1. immediate hypersensibility
	2. delayed hypersensibility
	3. allergic reactions involving sensitized T lymphocytes
	4. allergic reactions with the participation of B lymphocytes and plasma cells
	5. allergic reactions involving activation of the complement system
77. **What is the pathogenesis of physiological phase in allergic reactions type IV?**
	1. direct action of cytotoxic lymphocytes
	2. proteolysis performed by lysosomal enzymes
	3. effects of lymphotoxin
	4. direct action of IgM and IgG
	5. direct action of natural killer lymphocytes
78. **What is the final effect of allergic reactions type IV?**
	1. exudative inflammation
	2. fibrinous inflammation
	3. proliferative inflammation
	4. necrosis with scarring
	5. purulent inflammation
79. **What disorders underlie on the basis of delayed hypersensibility?**
	1. tuberculosis
	2. serum sleekness
	3. glomerulonephritis
	4. Sjogren's syndrome
	5. bronchial asthma
80. **What ions have vasoconstrictive effect?**
81. sodium
82. potassium
83. calcium
84. hydrogen
85. magnesium
86. **What ions have vasodilatory effect?**
87. zinc
88. potassium
89. hydrogen
90. carbon
91. calcium
92. **What is the correlation between the inflow and outflow of blood in arterial hyperemia?**
	1. Inflow and outflow are reduced
	2. Inflow is increased and outflow is reduced
	3. Inflow and outflow are enhanced
	4. Outflow is increased and inflow is unchanged
	5. Inflow is increased and outflow is unchanged
93. **What is characteristic for neurotonic mechanism of arterial hyperemia?**
94. Decreased parasympathetic influences of arterioles
95. Increased parasympathetic influences on arteries with their dilation
96. Increased parasympathetic influences on arterioles
97. Decreased sympathetic influences on arteries
98. Decreased vascular reactivity to catecholamines
99. **What is characteristic for neuroparalytic mechanism of arterial hyperemia?**
100. Decreased parasympathetic influences on arterioles
101. Decreased vascular reactivity to acetylcholine
102. Decreased vascular reactivity to catecholamines
103. Increased parasympathetic influences on arterioles
104. Decreased sympathetic influences on arterioles
105. **What mechanisms are specific for functional arterial hyperemia?**
	1. Metabolic
	2. Neurotonic
	3. Mechanical
	4. Neuroparalytic
	5. Humoral
106. **What are the metabolic changes in arterial hyperemia?**
107. Decreased arterial - venous oxygen difference
108. Increased arterial - venous oxygen difference
109. Increased oxygen pressure in venous blood
110. Increased oxygen pressure in arterial blood
111. Increased supply and decrease consumption of oxygen
112. **What are the external changes of arterial hyperemia?**
113. Diffuse general erythema
114. Local erythema
115. Decreased tissue turgor
116. Increased local temperature
117. Hyperthermia of the body
118. **What etiological factors are responsible for developing of venous hyperemia?**
119. Increased elasticity of the venous wall
120. Decreased arterial-venous pressure difference in heart failure
121. Decreased aspiration force of the thorax
122. Increased aspiration force of the thorax
123. Increased arterial-venous pressure difference in heart failure
124. **What are the metabolic changes in venous hyperemia?**
	1. Decreased aerobic metabolic process
	2. Increased aerobic metabolic process
	3. Increased anaerobic metabolic processes
	4. local metabolic alkalosis
	5. Decreased anaerobic metabolic processes
125. **What are the metabolic changes in venous hyperemia?**
	1. Enhanced glycolysis
	2. Increased energogenesis
	3. Reduced glycolysis
	4. Metabolic alkalosis
	5. Metabolic acidosis
126. **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
127. **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
128. **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
129. **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
130. **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
131. **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
132. **What is the pathogenetic mechanism of decreased volume of the ischemic organ?**
	1. Increased volume of interstitial fluid
	2. Decreased volume of interstitial fluid
	3. Increased volume of intracellular fluid
	4. Decreased volume of intracellular fluid
	5. Reduced lymphogenesis and lymphodynamics
133. **What types of embolisms are considered as endogenous?**
	1. Atheromatous
	2. Gaseous
	3. With amniotic fluid
	4. With air
	5. Microbial
134. **How are classified emboli by the direction of their circulation?**
	1. Orthograde
	2. Anterograde
	3. Retrograde
	4. Paradoxical
	5. Turbulent
135. **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
136. **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
137. **When can develop embolism in the systemic circulation?**
	1. Thrombophlebitis in arm vena
	2. Thrombophlebitis of leg vena
	3. Dilation varices
	4. Myocardial aneurism
	5. Paradoxical embolism
138. **What are the general consequences of embolism?**
	1. pulmonary failure
	2. Thrombophlebitis
	3. Hypertension in systemic circulation
	4. Sudden death
	5. Hypotension in pulmonary circulation
139. **What are the pathogenetic factors of capillary stasis?**
	1. Decreased blood velocity
	2. Hemodilution
	3. Decreased concentration of plasma globulins
	4. Increased concentration of plasma globulins
	5. Increased blood velocity
140. **What are the manifestations of stasis?**
	1. Decreased local temperature
	2. Decreased body temperature
	3. Reduced tissular turgor
	4. Local cyanosis
	5. General cyanosis
141. **What are the manifestations of stasis?**
142. Microhemorrahages due to increased hydrostatic pressure
143. Microhemorrhages due to decreased hydrostatic pressure
144. Microhemorrhages due to increased permiability of the vessels
145. Microhemorrhages due to decreased permiability of the vessels
146. Microhemorrhages due to increased blood velocity
147. **What is pathological regeneration?**
	1. Dysplasia
	2. Metaplasia
	3. Hyperplasia
	4. Hypertrophy
	5. Anaplasia
148. **What** **does the physiological regeneration represent?**
149. Defect regeneration with a recovery of the initial tissue volume
150. Defect regeneration with tissue excess
151. Defect regeneration with tissue deficiency
152. Regeneration of the pathogen factor induced defect with a similar tissue
153. Regeneration of the pathogen factor induced defect with an atypical tissue
154. **What does the sclerosis of organ mean?**
155. Pathological regeneration
156. Reparative physiological regeneration
157. Compensatory physiological regeneration
158. Protective physiological regeneration
159. The last phase of the inflammation
160. **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
161. **In what conditions can develop symptomatic gingival hypertrophy?**
	1. Endocrine disorders
	2. electrolytic disorders
	3. anemia
	4. Glomerulopathy
	5. Liver disorders
162. **What are the disorders that could lead to teeth attrition?**
163. Disorders of thyroid gland
164. Disorders of parathyroid gland
165. Disorders of adrenal glands
166. Disorders of gonads
167. Disorders of liver
168. **In what conditions can develop atrophy of the mouth mucosa?**
169. Bacterial infection
170. collagen disease
171. chronic inflammation
172. acute inflammation
173. viral infection
174. **What are the pathogenetic mechanisms of scleroderma in oral cavity?**
175. lingual edema
176. macrocirculatory disorders
177. tissue atrophy
178. tissue hypertrophy
179. tissue hyperplasia
180. **What are the trigger factors for development of hypertrophy?**
181. Increased workload of the organ
182. Decreased workload of the organ
183. Hormonal hypersecretion
184. Hormonal hyposecretion
185. Hypersecretion of vasoactive agents
186. **What atrophy is considered as physiological?**
187. Peripheral endocrine gland atrophy in the hypophyseal tropic hormone hyposecretion
188. Atrophy of hormone dependent organ in the deficiency of peripheral hormone
189. Organ atrophy due to organism aging
190. Organ atrophy in the hyponutrition
191. Atrophy of organ in the denervation
192. **What factor induces sclerosis?**
193. Cell lesions
194. Cell mitosis cessation
195. Primary hypofunction of the organ
196. Growth factors lack
197. Apoptosis
198. **What is one of the consequences of sclerosis?**
199. Organ malignancy
200. Fatty dystrophy of the organ
201. Organ deformation
202. Hypertrophy of the organ
203. Cell dedifferentiation
204. **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
205. **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
206. **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 140 mEq/L
	4. less than 120 mEq/L
	5. less than 300 mEq/L
207. **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
208. **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
209. **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
210. **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
211. **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
212. **What is the physiological role of potassium in the body?**

maintains the plasmatic osmotic pressure

maintains the plasmatic oncotic pressure

ensures the active membrane potential in the excitable cells

ensures the resting membrane potential in the excitable cells

maintains the threshold potential in the excitable cells

1. **In what disorders can be found hyperkalemia?**
	1. hyperhydration with gain of body fluids
	2. hypersecretion of aldosteron
	3. enhanced catabolism of tissue proteins
	4. increased plasma renin concentration
	5. decreased plasma renin concentration
2. **What can be the causes of hypokalemia?**

deficiency of renin in the blood

hypersecretion of glucocorticoids

excessive renin in the blood

deficiency of glucocorticoids

overhydration of the body

1. **What are the pathogenetic mechanisms that contribute to development of hypokalemia?**

disturbances of glomerular filtration and water retention in the body

excessive loss of potassium in the kidneys in hypersecretion of aldosteron

excessive loss of potassium in the kidneys in hyposecretion of aldosteron

hypersecretion of vasopressin

deficiency of vasopressin

1. **What is the normal concentration of Ca++  ions in the blood?**

1,5 – 2,5 mmol/L

2,1 – 2,6 mmol/L

4,5 – 5,5 mmol/L

less than 1,0 mmol/L

more than 3,5 mmol/L

1. **From what value of the concentration of Ca++ ions in the blood there is considered hypercalcemia?**
2. above 2,6 mmol/L
3. above 3,5 mmol/L
4. above 1,6 mmol/L
5. above 2,0 mmol/L
6. above 7,0 mmol/L
7. **From what value of the concentration of Ca++ ions in the blood there is considered hypocalcemia?**
8. less than 5,3 mmol/L
9. less than 0,5 mmol/L
10. less than 2,1 mmol/L
11. less than 1,0 mmol/L
12. less than 7,0 mmol/L
13. **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
14. **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
15. **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
16. **What are the causes of hypercalcemia?**

hypersecretion of mineralocorticoids

hyposecretion of calcitonin

excessive vitamin D

deficient vitamin D

deficiency of mineralocorticoids

1. **What are the main pathogenetic mechanisms of hypercalcemia?**

redistribution between intra- and extracellular compartment

increased mobilization from teeth matrix

increased mobilization from bone matrix

reduced renal reabsorption

reduced renal secretion

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 causes of hypocalcemia?**

hypersecretion of calcitonin

hypersecretion of parathyroid hormone

hyposecretion of calcitonin

Hyperphosphatemia

hypophosphatemia

1. **What are the main pathophysiological mechanisms of hypocalcemia?**

deficient mobilization from the bone in deficiency of PTH

deficient mobilization from the bone in excessive PTH

decreased incorporation in the bone in hyposecretion of calcitonin

enhanced calcium secretion in the proximal renal tubes

enhanced calcium reabsorption in the proximal renal tubes

1. **What are the causes of hyperphosphatemia?**

mobilization from bone matrix

hyposecretion of PTH

hypersecretion of PTH

intracellular-extracellular shift

hypersecretion of calcitonin

1. **What are the main manifestations of hyperphosphatemia?**

muscle cramps

blood hyperosmolarity

hypocalcemia

hypercalcemia

muscle paralysis

1. **What is the cause of hypophosphatemia?**

hypersecretion of PTH

hyposecretion of PTH

hypersecretion of calcitonin

hyposecretion of calcitonin

metabolic alkalosis

1. **What are the main manifestations of hypophosphatemia?**

hemolytic anemia

osteomalacia

tetany

muscle cramps

enhanced neuronal excitability

1. **What is normal blood glucose level?**

120 -140 mg/dl

60 -80 mg/dl

5,5 -6,0 mmol/L

4,5 – 5,5 mmol/L

80 – 120 mg/dl

1. **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 pepsi
		4. insufficiency of pancreatic amylase
		5. insufficiency of pancreatic carboxypeptidase
2. **What are the causes of carbohydrates malabsorption?**

atrophy of mucosal layer in the small intestine

atrophy of mucosal layer in the large intestine

atrophy at the level of gastric mucosal layer

inflammation at the level of small intestine mucosal layer

inflammation at the level of large intestine mucosal layer

1. **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
2. **What are the consequences of excessive carbohydrates intake?**

hyposecretion of glucagon

enhanced protein synthesis

enhanced lipid synthesis

hypersecretion of glucagon

enhanced lipolysis

1. **What factors can cause hyperglycemia?**

reduced glycogenolysis

enhanced glycogenolysis

enhanced glycolysis

reduced gluconeogenesis

enhanced gluconeogenesis

1. **What can be the causes of hypoglycemia?**

enhanced gluconeogenesis

reduced gluconeogenesis

hypersecretion of glucocorticoids

hyposecretion of glucocorticoids

enhanced glycogenolysis

1. **What are the compensatory reactions in hyperglycemia?**

glucocorticoid hyposecretion

inhibition of gluconeogenesis

glucocorticoid hypersecretion

increased lipolysis

enhanced glycogenolysis

1. **What are the compensatory reactions in hypoglycemia?**

glucagon hypersecretion

glucagon hyposecretion

increased glycogenolysis

glucocorticoid hyposecretion

increased lipogenesis

1. **What endocrine factors can contribute to development of hyperglycemia?**

excessive insulin

excessive glucagon

deficiency of glucagon

deficiency of thyroid hormones

excessive glucocorticoids

1. **What endocrine factors can contribute to development of hypoglycemia?**

deficiency of insulin

excessive glucagon

deficiency of glucagon

excessive thyroid hormones

deficiency of glucocorticoids

1. **What carbohydrates can be absorbed from the gastrointestinal tract?**

lactose

glucose

glycogen

maltose

galactose

1. **What are the possible consequences of hyperglycemia in healthy persons?**

enhanced oncotic pressure in the blood

enhanced lipid synthesis

increased osmotic pressure in the blood

stimulation of cortisol secretion

enhanced lipid breakdown

1. **What are the possible consequences of hypoglycemia in healthy persons?**

enhanced lipid synthesis

enhanced lipid breakdown

stimulation of cortisol secretion

stimulation of insulin secretion

inhibition of glucagon secretion

1. **What are the factors that may cause hyperlipidemia?**

hypersecretion of insulin

hypersecretion of glucocorticoids

hyposecretion of insulin

hyposecretion of glucagon

hyposecretion of glucocorticoids

1. **Lack of what digestive enzyme leads to lipid maldigestion?**

pancreatic amylase

pancreatic carboxypeptidase

salivary lipase

pancreatic lipase

hepatic lipase

1. **What lipid substances are synthesized in the body?**
	1. triglyceride
	2. urea
	3. polyunsaturated fatty acids
	4. Phospholipids
	5. liposoluble vitamins
2. **What are the metabolic consequences of excessive consumption of fat?**
	1. high blood level of VLDL
	2. enhanced lipogenesis
	3. enhanced lipolysis
	4. enhanced production of ketone bodies
	5. infiltration of tissues with chylomicrons
3. **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
4. **What are the metabolic consequences of lipid maldigestion?**

enhanced lipolysis

disturbances of steroid hormones synthesis

high level of chylomicrons in the blood

excess of liposoluble vitamins

deficiency of liposoluble vitamins

1. **What are the possible causes of hypoproteinemia?**

deficiency of pancreatic amylase

* 1. Hemodilution
	2. proteinuria
	3. renal failure
	4. hemoconcentration
1. **What are the possible consequences of hypoproteinemia?**
	1. energy deficiency
	2. low oncotic pressure
	3. high osmotic pressure
	4. low osmotic pressure
	5. edema
2. **Lack of what digestive enzymes lead to protein maldigestion?**

intestinal peptidases

pancreatic carboxypeptidase

pancreatic amylase

salivary proteases

salivary amylase

1. **What is the possible consequence of direct absorption of protein from the digestive tract?**
	1. hyperproteinemia
	2. food allergy
	3. hypoproteinemia
	4. anaphylactic shock
	5. infiltration of the liver with protein
2. **What are the metabolic and digestive disorders in maldigestion of proteins?**

low osmotic pressure

low oncotic pressure

food allergy

gastrointestinal auto-intoxication

inhibition of putrefaction processes in the bowels

1. **What pathological states are associated with hypoproteinemia?**

diarrhea

Hemodilution

hemoconcentration

polyuria

combustions with plasmorrhagia

1. **What pathological states are associated with hyperproteinemia?**

Hemodilution

hemorrhage

renal insufficiency

diarrhea

polyuria

1. **What are the causes of intestinal auto-intoxications?**

liver failure

protein maldigestion and malabsorbtion

lipid maldigestion and malabsorbtion

enteritis

carbohydrates maldigestion and malabsorbtion

1. **What are excitatory mediators?**

glycine

Acetylcholine

Dopamine

gamma-oxibutyric acid

serotonin

1. **What are inhibitory mediators?**
	1. noradrenalin
	2. acetylcholine
	3. dopamine
	4. serotonin
	5. gamma-oxibutyric acid
2. **Activation of what nervous structures can trigger temporo-mandibular pain?**

sensitive neurons of posterior medullar horns

sensitive neurons of cerebral cortex

sensitive neurons of limbic system

motor neurons of posterior medullar horns

motor neurons of the cerebral cortex

1. **What are the local manifestations of glosalgia?**

burn sensation

hypersalivation

hyposalivation

paresthesia

tongue edema

1. **What are the causes of facial pain?**

inflammatory processes in the mouth

trauma of dental-maxillary apparatus

periosteal disorders

injuries of salivary glands

injuries of perivascular tissue

1. **When can develop dental hyperesthesia?**

thin enamel layer

naked dentin

injuries at the level of sensorial neurons in the spinal chord

increased excitability threshold

injuries at the level of sensorial neurons in the cerebral cortex

1. **When can develop pulp pain?**

disorders of microcirculation

excessive osteoblast proliferation

excessive osteoclast proliferation

edema in tooth alveoli

paradontosis

1. **What are the causes of primary endocrine disorders?**

disorders of endocrine hypothalamus

disorders of adenohypophysis

disorders of neurohypophysis

disorders of peripheral endocrine glands

disorders of peripheral hormonal reception

1. **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
2. **What are the causes of tertiary endocrine disorders?**

disorders of endocrine hypothalamus

disorders of adenohypophysis

disorders of neurohypophysis

disorders of peripheral endocrine glands

disorders of peripheral hormonal reception

1. **What type of hormone is increased in gigantism?**
	1. triiodothyronine
	2. somatotropin
	3. tetraiodthyronine
	4. cortisol
	5. somatoliberine
2. **What type of hormones are increased in Graves-Bazedov disease?**
	1. Triiodothyronine
	2. somatotropin
	3. somatoliberine
	4. tetraiodthyronine
	5. cortisol
3. **What hormonal disturbance is characteristic for myxedema?**
	1. deficiency of triiodothyronine
	2. excess of triiodthyronine
	3. deficiency of thyroxin
	4. excess of thyroxin
	5. deficiency of cortisol
4. **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
5. **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
6. **What are the metabolic changes in hypersecretion of thyroid hormones?**

increased intracellular synthesis of ATP

increased intracellular concentration of ADP

glycogenolysis

increased glycogenogenesis

increased lypogenesis

1. **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
2. **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
3. **What hormonal disturbances induce hyperglycemia?**

Hypersecretion of insulin

Hypersecretion of glucagon

Hypersecretion of glucocorticoids

Hyposecretion of glucagon

Hyposecretion glucocorticoids

1. **What hormonal disturbances induce hypoglycemia?**

hypersecretion of insulin

hyposecretion of insulin

hypersecretion of thyroid hormones

hyposecretion of thyroid hormones

hyposecretion of glucocorticoids

1. **What hormonal disturbance induces glycogenogenesis?**

Hypersecretion of insulin

hypersecretion of glucagon

Hyposecretion of insulin

hypersecretion of thyroid hormones

hypersecretion of glucocorticoids

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. **Which hormones have catabolic effect?**
	1. insulin
	2. glucagon
	3. glucocorticoids
	4. thyroid hormones
	5. parathyroid hormone
3. **Which hormones have anabolic effect?**
	1. Insulin
	2. glucagon
	3. glucocorticoids
	4. thyroid hormones
	5. parathyroid hormone
4. **What are the metabolic manifestations of glucocorticoids hypersecretion?**
	1. Increased gluconeogenesis
	2. Decreased gluconeogenesis
	3. Increased glycogenogenesis
	4. Increased proteolysis
	5. Increased proteosynthesis
5. **What are the somatic manifestations of glucocorticoids hypersecretion?**
	1. Increased bones demineralization
	2. Decreased bones demineralization
	3. proliferation of lymphoid tissue
	4. atrophy of lymphoid tissue
	5. hypertrophy of muscular tissue
6. **What hormonal disturbances induce hyperlipidemia?**

Hypersecretion of insulin

Hyposecretion of insulin

Hypersecretion of glucagon

Hyposecretion of glucagon

Hyposecretion of glucocorticoids

1. **What hormonal disturbances induce proteolysis?**

Excess of insulin

Excess of glucocorticoids

Deficiency of glucocorticoids

Excess of thyroid hormones

Deficiency of thyroid hormones

1. **What are the metabolic effects of insulin?**

stimulates glycogenogenesis

stimulates glycogenolysis

stimulates lipogenesis

stimulates lipolysis

stimulates gluconeogenesis

1. **What are metabolic effects of glucagon?**

stimulates glycogenogenesis

stimulates glycogenolysis

stimulates lypogenesis

stimulates lipolysis

stimulates glycolysis

1. **What is the pathogenesis of polyuria in insulin deficiency?**

insulin deficiency – ADH hypersecretion- inhibition of water canalicular reabsorbtion – polyuria

insulin deficiency – hyperglycemia – increased glomerular filtration – polyuria

insulin deficiency – hyperglycemia – incomplete glucose reabsorbtion – glucosuria – polyuria

insulin deficiency – hyperglycemia – inhibition of aldosterone secretion – hypernatremia – polyuria

insulin deficiency – hyperglycemia – glucosuria – blockade of aquaporine – polyuria

1. **Under what conditions can be found oligocythemic hypovolemia?**

first minutes after acute bleeding

24 hours after acute bleeding

30-40 minutes after acute bleeding

In case of erythrem

In case of body overheating

1. **Under what conditions can be found polycythemic hypovolemia?**

In case of body dehydration

In case of burns

In case of erythremia

In case of anemia

In case of body hyperhydration

1. **Under what conditions can be found oligocythemic hypervolemia?**

massive infusion of saline solution

blood transfusion

body dehydration

body hyperhydration

diarrhea

1. **Under what conditions can be found polycythemic hypervolemia?**

In case of erythremia

In case of erythropenia

blood transfusion

In case of body dehydration

plasma transfusion

1. **What are the signs of intracellular hemolysis?**

Hemoglobinemia

Hemosiderinuria

Hemoglobinuria

Hyperbilirubinemia with free bilirubin (indirect bilirubin)

Hyperbilirubinemia with conjugated bilirubin (direct bilirubin)

1. **What changes of hemogram are characteristic for iron deficiency anemia?**

Macrocytosis

hypochromic erythrocytes

microcytosis

hyperchromic erythrocytes

drepanocytosis

1. **Under what pathological conditions can be found neutrophilia?**

bacterial infection

myocardial infarction

purulent otitis

viral infections

influenza

1. **What does “left” nuclear shift represent?**

increased number of agranulocytes in peripheral blood

increased number of granulocytes in peripheral blood

increased number of immature neutrophils in peripheral blood

increased number of mature neutrophils in peripheral blood

increased number of hyper-segmented neutrophils in peripheral blood

1. **Under what pathological conditions can be found primary absolute lymphocytosis?**

tuberculosis

septicemia

bronchial asthma

chronic lymphoid leucosis

Hodgkin lymphomas

1. **Under what pathological conditions can be found secondary absolute lymphocytosis?**

tuberculosis

infectious mononucleosis

bronchial asthma

chronic lymphoid leucosis

acute lymphoid leucosis

1. **Under what pathological conditions can be found monocytosis?**

in acute infection

granulomatous inflammation

in chronic infection

bronchial asthma

bacterial infection

1. **What does agranulocytosis represent?**

severe increased of lymphocytes in peripheral blood

severe decrease or absence of agranulocytes in peripheral blood

severe increased count of agranulocytes in peripheral blood

severe decreased or absence of granulocytes in peripheral blood

increased number of hyper-segmented neutrophils in peripheral blood

1. **What are the hematologic signs of absolute secondary erythrocytosis?**

hemoglobin content more than 160 g/L

hemoglobin content less than 160g/l

erythrocyte count more than 5,5 ×1012/L

total blood volume less than 7% from body weight

total blood volume less than 5% from body weight

1. **What are the signs of relative erythrocytosis?**

erythrocyte count more than 5×1012/L

erythrocyte count less than 5×1012/L

reticulocyte count more than 0,5%

total blood volume less than 7% from body weight

total blood volume more than 7% from body weight

1. **What are the signs of primary absolute erythrocytosis?**

Low erythropoietin level

High erythropoietin level

Reticulocyte count more than 2.5%

Thrombocytopenia

Reticulocyte count less than 0,5%

1. **What are signs of secondary absolute erythrocytosis?**

Low erythropoietin level

High erythropoietin level

erythrocytes count more than 5,5 ×1012/L

erythrocyte count less than 5,5 ×1012/L

Reticulocyte count less than 1,5%

1. **What processes are disturbed in hypoplastic anemia?**

proliferation of erythroblastic series

differentiation of erythroblastic series

hemoglobin synthesis

erythrodieresis

erythrocytes maturation

1. **What processes are disturbed in hemolytic anemias?**
	1. proliferation of erythroblast series
	2. differentiation of erythroblast series
	3. hemoglobin synthesis
	4. erythrodieresis
	5. erythrocyte maturation
2. **What processes are disturbed in iron deficiency anemia?**
	1. proliferation of erythroblast series
	2. differentiation of erythroblast series
	3. hemoglobin synthesis
	4. erythrodieresis
	5. erythrocyte maturation
3. **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
4. **What is the sign of absolute leukocytosis?**
	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
5. **What are the causes of neutrophilia?**
	1. Viral infection
	2. allergic diseases
	3. cocci infection
	4. acute infection disease
	5. chronic infection disease
6. **What are the causes of eosinophilia?**
	1. insufficiency of adrenal glands
	2. insufficiency of thyroid gland
	3. allergic diseases
	4. bacterial infection
	5. viral infection
7. **What is the etiologic factor of lymphocytosis?**
	1. bacterial infection
	2. viral infection
	3. acute infection disease
	4. chronic infection disease
	5. allergic disease
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?**
	1. Presence of painful sensation of the tongue
	2. atrophy of lingual mucosa
	3. hyperplasia of lingual mucosa
	4. hyperplasia of lingual papilla
	5. absence of painful sensation of the tong
10. **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
11. **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
12. **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
13. **How has been modeled spinal reflex in the frog?**
	1. By total anesthesia
	2. By decapitation on the anterobulbar line of the brain
	3. By destroying of spinal cord
	4. By decapitation on the retrobulbar line of the brain
	5. By the section of sciatic nerve
14. **How has been modeled acute adrenocortical insufficiency in rats?**

due to ligation of suprarenal arteries

due to administration of drugs that block cholesterol metabolism

due to bilateral surgical removal of adrenal glands

due to unilateral surgical removal of adrenal glands

due to administration of cytostatics

1. **To what stressful factors were subjected laboratory animals in experimental hypocorticosolism?**

hypobaric hypoxia

cold water

starvation

physical effort

electrical shock

1. **To what stressful factor were subjected laboratory animals with acute experimental hypocorticosolism?**

hypobaric hypoxia

artificial hypervolemia

starvation

cold water

electrical shock

1. **What is the stress hormone?**

Cortisol

Insulin

Epiandrosteron

Somatotropin

Thyroxin

1. **What mechanisms determine the resistance to the action of stressful factors?**

Hyperglycemia

Hypoglycemia

Heart hyperfunction

Heart hypofunction

Increased the body’s needs of O2

1. **What mechanisms determine the resistance to the action of stressful factors?**

Hyperglycemia

Hypoglycemia

Increased the muscle tonus

Decreased the muscle tonus

Increased the body’s needs of O2

1. **What mechanism determines the resistance to the action of stressful factors?**
2. Hyperproteinemia
3. Hypoglycemia
4. Hyperlipidemia
5. Hypolipidemia
6. Increased the body’s needs of O2
7. **What does the primary hyperthyroidism mean?**

Primary increasing of thyroid gland function

Disturbance of ratio T3/T4

Increasing of thyroid gland function due to high secretion of thyrotropin

Increasing of thyroid gland function due to high secretion of thyroliberin

Excess of thyrotropin hormone and thyroid hormones

1. **What does the primary hypothyroidism mean?**

Disturbance of ratio T3/T4

Primary decreasing of thyroid gland function

Decreasing of thyroid gland function due to deficiency of thyrotropin

Decreasing of thyroid gland function due to deficiency of thyroliberin

Deficit of thyrotropin hormone and thyroid hormones

1. **By what method has been modeled hyperthyroidism in rats?**

by administration of methyluracil

by administration of caffeine

by administration of chloral hydrate

by administration of L – thyroxin

by administration of NaCl

1. **By what method has been modeled hypothyroidism in rats?**
2. by administration of methyluracil
3. by administration of caffeine
4. by administration of chloral hydrate
5. by administration of L – thyroxin
6. by administration of NaCl
7. **What is mechanism of hypothyroidism at methyluracil administration?**
8. Intensification of iodine absorption from the blood
9. Intensification of iodine uptake by the thyrocytes
10. Inhibition of iodine uptake by the thyrocytes
11. Destruction of the thyroglobulin in thyrocytes
12. Inhibition of thyroid hormones exocytose
13. **How has been evaluated the role of thyroid hormones in pathology?**
14. Expose of animals with hypo- and hyperthyroidism at hyperoxia
15. Expose of animals with hypo- and hyperthyroidism at hypobaric hypoxia
16. Expose of animals with hypo- and hyperthyroidism at normobaric hypoxia
17. Expose of animals with hypo- and hyperthyroidism at hyperbaric hypoxia
18. Expose of animals with hypo- and hyperthyroidism at hypothermia
19. **Which of the experimental animal with changed thyroid function was more sensitive to the action of hypoxia?**
20. The animal that had received methyl uracil
21. The animal that had received mercazolil
22. The animal that had received novocaine
23. The animal that had received L-thyroxine
24. The animal that had received NaCl
25. **What mechanisms reduce the resistance of the rat with experimental hyperthyroidism to hypoxia?**
26. Decreased oxido- reduction reactions
27. increased O2 consumption
28. decreased basal metabolic rate
29. exhaustion of neuronal metabolic substrate
30. decreased O2 consumption
31. **What value of arterial pressure does represent pulmonary hypertension?**
	1. systolic pressure 20-25 mmHg
	2. systolic pressure more than 30 mmHg
	3. systolic pressure more than 60 mmHg
	4. average pressure 10-17 mmHg
	5. average pressure more than 20 mmHg
32. **What value of arterial pressure does represent systemic hypertension?**
	1. systolic pressure more than120 mmHg
	2. systolic pressure more than 140 mmHg
	3. systolic pressure more than 150 mmHg
	4. diastolic pressure more than 80 mmHg
	5. diastolic pressure more than 70 mmHg
33. **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
34. **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
35. **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
36. **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
37. **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
38. **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
39. **What pathogenic factors trigger heterometric heart hyperfunction?**
	1. stenosis of aortic valves
	2. insufficiency of mitral valves
	3. enhanced circulatory blood volume
	4. stenosis of mitral valves
	5. increased peripheral vascular resistance
40. **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
41. **What is the main pathogenic factor which triggers myocardial hypertrophy?**
	1. increased total work load performed by the heart
	2. increased work load for every mass unit of the heart
	3. increased arterial pressure in systemic circulation
	4. increased arterial pressure in pulmonary circulation
	5. increased circulatory blood volume
42. **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
43. **What are the immediate extracardiac mechanisms of compensation in circulatory failure?**
	1. reduction of circulatory blood volume by partly blood sequestration
	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
44. **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
45. **What are the signs of left ventricular failure?**
	1. hypotension in systemic circulation
	2. hypertension in systemic circulation
	3. pulmonary edema
	4. ascites
	5. hypotension in pulmonary circulation
46. **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
47. **What is the cause of sinus tachycardia?**
	1. increased intracerebral pressure
	2. intoxication with digitalics
	3. hypothermia
	4. hyperthermia
	5. vagotonia
48. **What are the causes of sinus bradycardia?**
	1. intoxication with β-adrenergic drugs
	2. increased intracerebral pressure
	3. heart sympathicotonia
	4. intoxication with digitalics
	5. intoxication with α-adrenergic drugs
49. **What are forms of heart excitability disorders?**
	1. ventricular extrasystole
	2. paroxysmal tachycardia
	3. sinus tachycardia
	4. atrio-ventricular block
	5. sinus bradycardia
50. **What are forms of myocardial conductibility disorders?**
	1. ventricular extrasystole
	2. paroxysmal tachycardia
	3. atrial fibrillation
	4. atrio-ventricular block
	5. sino-atrial block
51. **What does represent hypercapnia?**
	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 more than 46 mmHg
	4. partial pressure of CO2 in the cells more than 46 mmHg
	5. partial pressure of CO2 in arterial blood less than 40 mmHg
52. **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
53. **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
54. **What disturbances lead to extra-parenchymatous restriction?**
	1. bronchial asthma
	2. pneumothorax
	3. pulmonary hypoperfusion
	4. disorders of neuro-muscular apparatus
	5. pulmonary fibrosis
55. **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
56. **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
57. **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
58. **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
59. **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
60. **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
61. **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
62. **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
63. **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
64. **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
65. **What structure is the most sensitive to hypoxia?**
	1. bones
	2. nervous tissue
	3. connective tissue
	4. cartilage
	5. myocardium
66. **What is the cause of respiratory hypoxia?**
	1. decreased pressure of oxygen in inspired air with hypoxemia
	2. disorders of external respiration with hypoxemia
	3. heart failure and lung hypoperfusion with hypoxemia
	4. disorders of internal respiration with hypoxemia
	5. pulmonary hyperventilation with hypoxemia
67. **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
68. **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
69. **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
70. **What are the consequences of hypoxia?**
	1. alveolar hyperventilation
	2. alveolar hypoventilation
	3. respiratory acidosis
	4. respiratory alkalosis
	5. brain hemodynamic disorders
71. **What are the changes in respiratory hypoxia?**
72. arterial hypoxemia, hypocapnia, respiratory acidosis
73. arterial hypoxemia, hypercapnia, respiratory acidosis
74. arterial hypoxemia, hypercapnia, respiratory alkalosis
75. arterial hypoxemia, normal CO2 pressure, respiratory alkalosis
76. arterial hypoxemia, hypocapnia, respiratory alkalosis
77. **What represents hypersalivation?**
78. saliva secretion more than 2L/24 h
79. saliva secretion more than 1L/24 h
80. saliva secretion more than 1,5L/24h
81. saliva secretion more than 0,5 L/24h
82. saliva secretion more than 0,3L/24 h
83. **What can be causes of pathologic hypersalivation?**
84. in children during teeth eruption
85. ingestion of dry aliments
86. stomatitis
87. mouth tumors
88. Parkinson disease
89. **What are causes of pathologic hyposalivation?**
90. emotional states
91. ingestion of fluid aliments
92. dehydration
93. parotiditis
94. salivatory ducts obstruction
95. **Stomachal hypersecretion can be induced by:**
96. caffeine
97. ethanol
98. gastrin excess
99. pepsin excess
100. vagotony
101. **How evacuation function of the stomach is affected in hypersecretion with hyperacidity?**
102. increases
103. decreases
104. doesn’t change
105. develops gastric chymostasis
106. develops dumping syndrome
107. **How intestinal transit is affected in case of stomachal hypersecretion with hyperacidity?**
108. increases
109. decreases
110. doesn’t change
111. frequent constipations
112. diarrhea
113. **What represent achlorhydria?**
114. lack of Cl ions in the blood
115. absence of HCl in gastric juice
116. lack of enzymes in gastric juice
117. increased blood pH
118. decreased blood pH
119. **What can be causes of achlorhydria?**
120. gastrin lack
121. atrophic chronic gastritis
122. gastric cancer
123. hypertrophic gastritis
124. gastric ulcer
125. **What are consequences of HCl absence in gastric juice?**
126. increased intestinal peristaltic movement
127. decreased intestinal peristaltic movements
128. maldigestion
129. malabsorbtion
130. constipations
131. **What can be consequences of vomiting?**
132. Hypochloremia
133. hyperkaliemia
134. alkalosis
135. acidosis
136. activation of renin-angiotensin-aldosterone system
137. **What are the causes of exocrine insufficiency of the pancreas?**
138. chronic pancreatitis
139. pancreatic tumor
140. pancreatic duct obturation
141. vagotony
142. sympathicotonia
143. **Which are consequences of insufficient pancreatic secretion?**
144. Maldigestion
145. Malabsorbtion
146. Malnutrition
147. ulcerogenesis
148. constipations
149. **What represents steatorrhea?**
150. presence of lipids in the blood
151. excessive elimination of lipids with stool
152. excessive accumulation of lipids in hepatic parenchyma
153. lipid elimination with urine
154. lack of lipids in feces
155. **What can be causes of steatorrhea?**
156. Acholia
157. insufficiency of pancreatic lipase
158. pepsin insufficiency
159. cholemia
160. hyperlipidemia
161. **What represents acholia?**
162. lack of bile in the blood
163. lack of bile in intestine
164. presence of bile in the blood
165. decoloration of feces
166. lack of bilirubin in bile
167. **What can be consequences of disaccharides maldigestion?**
168. Diarrhea
169. Dehydration
170. constipations
171. hyperhydration
172. hypoglycemia
173. **What can be consequences of protein maldigestion?**
174. Hypoproteinemia
175. decreased oncotic pressure
176. edemas
177. proteinuria
178. immunodeficiency
179. **What can be consequences of lipid maldigestion?**
180. hyperlipidemia
181. steatorrhea
182. blood hypocoagulation
183. diarrhea
184. constipations
185. **What can be causes of intestinal autointoxication?**
186. intensification of putrefaction processes in the intestine
187. excessive consumption of proteins
188. constipations
189. diarrhea
190. liver failure
191. **What are manifestations of intestinal autointoxication?**
192. arterial hypotension
193. arterial hypertension
194. headache
195. hypoglycemia
196. hyperglycemia
197. **What pathologic processes disturb digestion in the mouth?**
198. Hypersalivation
199. Hyposalivation
200. lack of salivary amylase
201. lack of lysosim
202. alkaline reaction of the saliva
203. **What are digestive disturbances in case of salivary amylase lack?**
204. disorders of polysaccharides digestion
205. disorders of disaccharides digestion
206. disorders of cellulose digestion
207. disorders of proteins digestion
208. disorders of lipid digestion
209. **How does stomach tonus and motility change in hypochlorhydria?**
210. Hypotonus
211. hypertonus
212. accelerated evacuation
213. stomachal chymostasis
214. vomiting
215. **How does stomach tonus and motility change in hyperchlorhydria?**
216. hypotonus
217. hypertonus
218. accelerated evacuation
219. stomachal chymostasis
220. vomiting
221. **What are factors involved in stomach ulcerogenesis?**
222. HCl
223. Bile
224. Helicobacter pylori
225. Salmonella
226. anaerobe flora
227. **What stomach digestive changes can be found in hypochlorhydria?**
228. develops maldigestion of polysaccharides
229. develops maldigestion of proteins
230. develops maldigestion of lipids
231. improvement of gastric digestion
232. develops maldigestion of cellulose
233. **What stomach digestive changes can be found in hyperchlorhydria?**
234. develops maldigestion of polysaccharides
235. develops maldigestion of proteins
236. develops maldigestion of lipids
237. improvement of gastric digestion
238. develops maldigestion of cellulose
239. **What digestive changes are found in exocrine insufficiency of the pancreas?**
240. develops maldigestion of polysaccharides
241. develops maldigestion of proteins
242. develops maldigestion of lipids
243. improvement of intestinal digestion
244. develops cellulose maldigestion
245. **What are digestive changes in bile secretion insufficiency?**
246. polysaccharides maldigestion
247. intestinal atonia
248. steatorrhea
249. amylorrhea
250. Creatorrhea
251. **Absorbtion of what substances is affected in disorders of small intestine mucosa?**
252. proteins
253. aminoacids
254. disaccharides
255. monosaccharides
256. water
257. **Absorbtion of what substances is affected in disorders of large intestine?**
258. proteins
259. aminoacids
260. mineral salts
261. monosaccharides
262. water
263. **How does carbohydrates metabolism change in liver failure?**
264. exaggerate postprandial hyperglycemia
265. fasting hypoglycemia
266. fructosemia
267. glycogen storages diminishes
268. glycogen storage increases
269. **How does protein metabolism change in liver failure?**
270. develops hyperglobulinemia
271. develops hypoalbuminemia
272. develops hyperaminoacidemia
273. synthesis of gamma-globulins is disturbed
274. there is increased concentration of urea in the blood

1. **How does lipid metabolism change in liver failure?**
2. there is intense lipolysis in the liver
3. there is steatosis of the liver
4. in the blood increase concentration of very low density lipoproteins
5. in the blood increase concentration of very high density lipoproteins
6. in the blood there increase concentration of non-esterified fatty acids
7. **Which are biochemical manifestations of cholemia?**
8. hyperbilirubinemia with free bilirubin
9. hyperbilirubinemia with conjugated bilirubin
10. hypercholesterolemia
11. cholalemia
12. hypoprothrombinemia
13. **Which are consequences of choledoc obstruction?**
14. hyperbilirubinemia with free bilirubin
15. cholestasis
16. acholia
17. hyperbilirubinemia with conjugated bilirubin
18. lipid maldigestion
19. **Which are manifestations of infectious hepatitis in organs of the mouth?**
20. edema of the mouth mucosa
21. jaundice of the mouth mucosa
22. teleagiectasia
23. Fourdis granules
24. paleness of mouth mucosa
25. **How is modelated 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
26. **What are compensatory reactions in experimental hypervolemia?**
27. blood storage
28. dilation of resistive blood vessels
29. increased diuresis
30. reduced erythrocytopoiesis
31. activation of renin-angiotensin-aldosteron system
32. **What is the method of measurement of blood pressure in the rabbit during experimental hypervolemia?**
33. indirect method with Riva-Roci device placed on posterior limbs
34. indirect method with Riva-Roci device placed on anterior limbs
35. direct intra-arterial method with hydrargyrum manometer
36. indirect method with Riva-Roci device placed on thorax
37. direct intra-arteriolar method with hydrargyrum manometer
38. **How blood pressure (BP) and breathing rate (BR) change in painful stimulation?**
39. BP increase, BR increase
40. BP decrease, BR decreases
41. BP unchanged, BR increase
42. BP increase, BR unchanged
43. BP decrease, BR unchanged
44. **What are the mechanisms of increased blood pressure in painful excitation?**
45. high secretion of catecholamines
46. increased number of adrenoreceptors
47. increased peripheral vascular resistence
48. activation of MAO
49. activation of kallikrein-kinin system
50. **What is the mechanism of restoration of blood pressure following painful stimulation?**
51. Activation of MAO
52. Hypersecretion of acetylcholine
53. activation of renin-angiotensin-aldosteron system
54. blood storage in deposits
55. centralization of hemocirculation
56. **What is the mechanism of restoration of blood pressure in hypercatecholaminemia?**
57. activation of monoamine-oxidase
58. increased level of acethylcholin
59. activation of renin-angiotensin-aldosteron system
60. blood storage
61. inhibition of monoamine-oxidase
62. **What are the causes of death in rats exposed to reduced atmospheric pressure?**
63. decreased pressure of oxygen in inspiratory air
64. hypobaria
65. respiratory hypoxia
66. hypoxemia
67. circulatory hypoxia
68. **What manifestations develop in the mouse in condition of normobaric hypoxia?**
69. hyperemia of sclera
70. cyanosis
71. tachycardia
72. paleness
73. meteorism
74. **What endogenous factors lead to different effects of hypoxia and hypobaria on rats?**
75. Metabolism intensity
76. functional state of SNC
77. glucose level in the blood
78. hydric metabolism in the rat
79. functional state of the immune system
80. **What animals are more sensible to action of hypobaric hypoxia?**
81. adult animals
82. new-born animals
83. animals with excited CNS
84. animals with inhibited CNS
85. animals with intensified anaerobic metabolism
86. **What are microcirculatory changes on frog’s tongue in arterial hyperemia?**
87. dilation of arterioles, capillaries and venules
88. increased number of functional capillaries
89. decreased linear blood velocity
90. decreased volumetric blood velocity
91. decreased number of functional capillaries
92. **What are microcirculatory changes on frog’s tongue in venous hyperemia?**
93. increased volumetric blood velocity
94. dilation of venules
95. increased venous outflow
96. narrowing of arterioles
97. reduced linear blood velocity
98. **By what experimental method arterial hyperemia was modelated on frog’s tongue?**
99. mechanical excitation of the tongue
100. chemical excitation of the tongue
101. ligation of lingual artery
102. unilateral ligation of main veins of the tongue
103. administration of adrenaline solution
104. **By what experimental method venous hyperemia was modelated on frog’s tongue?**
105. mechanical excitation of the tongue
106. chemical excitation of the tongue
107. ligation of lingual artery
108. unilateral ligation of main vein of the tongue
109. bilateral ligation of lingual veins
110. **By what experimental method stasis was modelated on frog’s tongue?**
111. mechanical excitation of the tongue
112. chemical excitation of the tongue
113. bilateral ligation of lingual veins
114. section of main lingual vein
115. unilateral ligation of main vein of the tongue
116. **By what experimental method ischemia was modelated on frog’s swimming membrane?**
117. mechanical excitation of the swimming membrane
118. chemical excitation of the swimming membrane
119. ligation of veins on swimming membrane
120. section of main veins on swimming membrane
121. administration of adrenaline solution on swimming membrane
122. **What are microcirculatory changes on frog’s tongue in prestasis?**
123. pulsatile movements
124. pendulatory movements
125. turbulent movements
126. diminished inflow
127. centripetal movements
128. **What are microcirculatory changes on frog’s tongue in prestasis?**
129. accelerated movements
130. pendulatory movements
131. turbulent movements
132. diminished arterial inflow
133. centripetal movements
134. **By what experimental method was modelated the development of white thrombus in mesenterial vessels in the frog?**
135. by ligation of mesenterial vessels
136. by applying a crystal of NaCl on mesenterial vessel bifurcation
137. by applying a crystal of AgNO3 on mesenterial vessel bifurcation
138. by applying heparin solution on mesenterial vessels
139. by applying adrenalin solution on mesenterial vessels
140. **By what experimental method was modelated the development of red thrombus in mesenterial vessels in the frog?**
141. by ligation of mesenterial vessels
142. by applying a crystal of NaCl on mesenterial vessel bifurcation
143. by applying a crystal of AgNO3 on mesenterial vessel bifurcation
144. by applying heparin solution on mesenterial vessels
145. by mechanical injury of the vessel
146. **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
147. **How was modelated experimental fat embolism in the mesenterial vessels in the frog?**
148. by administration of oil emulsion in the heart ventricles
149. by administration of oil emulsion orally
150. by administration of oil solution in the dorsal lymphatic sack
151. by administration of oil solution intraperitoneally
152. by administration of oil solution transcutaneously
153. **How alteration was modelated on frog’s tongue?**
154. by administration of adrenalin solution 0,1%
155. by bilateral ligation of lingual veins
156. by applying crystal of AgNO3 on the tongue
157. by ligation of lingual arteries
158. by unilateral ligation of lingual vein
159. **What is the mechanism of primary alteration on frog’s tongue after administration of AgNO3 crystals?**
160. chemical cell injury
161. development of venous hyperemia
162. development of blood stasis
163. development of arteriolar spasm
164. thrombogenesis
165. **What is the sequence of vascular reactions in inflammatory focus on frog’s tongue?**
166. arterial hyperemia→ venous hyperemia →ischemia → stasis
167. venous hyperemia → arterial hyperemia → ischemia → stasis
168. venous stasis → venous hyperemia → arterial hyperemia → ischemia
169. ischemia → arterial hyperemia → venous hyperemia → stasis
170. ischemia → venous hyperemia → arterial hyperemia → stasis
171. **What factors cause venous hyperemia in inflammatory focus on frog’s tongue?**
172. thrombosis in the venules
173. leucocytes margination at the level of microvessels
174. accumulation of exudate
175. arteriolar dilation
176. dilation of venules
177. **What are manifestations of venous hyperemia on frog’s tongue?**
178. reduced filling of arterioles with blood
179. reduced linear blood velocity
180. Reduced volumetric blood velocity
181. Increased linear blood velocity
182. Reduced number of functional capillaries
183. **What microcirculatory disorders lead to development of stasis on frog’s tongue?**
184. ischemia
185. venous hyperemia
186. arterial hyperemia
187. embolism
188. e thrombosis
189. **How anaphylactic shock was triggered in the experimental rabbit?**
190. by parenteral administration of horse serum
191. by parenteral administration of glucose solution
192. by parenteral administration of isotonic solution
193. by parietal administration of adrenaline solution
194. by parenteral administration of bicarbonate solution
195. **What are the causes of death of the rabbit in experimental anaphylactic shock?**
196. Cerebral coma
197. Cardiac arrest
198. respiratory arrest
199. Arterial collapse
200. Ischemia in the brain