1. **General nosology**
2. What studies etiology?
3. What factors are considered endogenous cause of disease?
4. What factors are considered exogenous causes of diseases?
5. What pathological processes can be caused by endogenous causes?
6. What is the role of cause in disease development?
7. What factors influence specificity of disease?
8. What factors influence the features of disease evolution?
9. In what disorders the cause plays only a trigger effect?
10. In what disorders the presence of cause is compulsory during all disease development?
11. What exogenous conditions can contribute to onset of disease?
12. What effects play favorable conditions for the body?
13. What effects play unfavorable conditions for the body?
14. What endogenous conditions can influence the action of harmful factors?
15. What exogenous conditions can influence the action of harmful factors?
16. What conditions disturb the metabolic processes and can contribute to disease development?
17. What factors enhance probability of disease development when the cause is acting?
18. What the role of conditions in disease development?
19. What studies general pathogeny?
20. What are the relations between local and general injuries in pathogeny of different diseases?
21. What represent the pathogenetic factors in pathologic process?
22. What is the chain of cause-effects in pathogeny of diseases?
23. What represents the main pathogenetic loop?
24. What is the role of main pathogenetic loop in disease development?
25. What represents etiotropic, pathogenetic and symptomatic treatment of disorders?
26. How the specific and non-specific prophylaxis of diseases is performed?
27. What are the characteristics of physiological reactions of the body?
28. What are the characteristics of pathological reactions of the body?
29. What reactions can be considered adaptative, protective, compensatory and reparative?
30. What are periods of disease development?
31. What are the characteristics of every stages of disease development?
32. What are the variants for disease resolution (ending)?
33. What represents pathological process?
34. What are primary sanogenetic mechanisms?
35. What are secondary sanogenetic mechanisms?
36. What represents vicious circle in pathogeny of diseases?
37. What are the characteristics of vicious circle in pathogeny of diseases?
38. **Cell injuries**
39. What changes at cellular level can be considered cellular injuries?
40. What cell injuries can be considered primary and secondary?
41. What primary injuries cause hypoxia, reactive oxygen species, high or low temperature, electrical current, phospholipases?
42. What cell injuries cause hyperosmolarity and hypoosmolarity of interstitial space?
43. What primary injury develops at interaction of antibodies with cell antigens and compliment activation?
44. What is the pathogenetic chain of cell death in hypoxia?
45. What is the pathogenetic chain of cell death in oxidative stress?
46. What is the pathogenetic chain of cell death at action of low temperature?
47. What is the pathogenetic chain of cell death in high temperature?
48. What is the pathogenetic chain of cell death triggered by lipolytic enzymes?
49. What is the pathogenetic chain of cell death in hyperosmolar environment?
50. What is the pathogenetic chain of cell death in hypoosmolar environment?
51. What is the pathogenetic chain of cell death triggered by continuous electrical current?
52. What is the pathogenetic chain of cell death caused by antibodies?
53. The presence in the blood of what intracellular enzymes are markers of injuries at the level of liver, pancreas, heart and epithelial cell of biliary tract?
54. How the intra-extracellular electrolyte ratio is changed at disintegration of cytoplasmic membrane?
55. What is the effect of action of electrical current on excitable cells?
56. What endogenous enzymes can trigger injuries of cytoplasmic membrane?
57. What cells can release harmful enzymes for other cells?
58. What electrolytic dyshomeostases develop during cell injuries?
59. What is the normal ratio of intra-to extracellular concentration of potassium ions?
60. What are the effects of increased level of potassium ions in the interstitium?
61. What is the normal ratio of intra-to extracellular concentration of sodium ions?
62. What is the effect of increased sodium ions concentration in the interstitial space?
63. What is the effect of reduced sodium ions concentration in the interstitial space?
64. What pathological process can reduce the electric resistance of cytoplasmic membrane?
65. What is the normal ratio of calcium ions concentration in the cell hyaloplasm and interstitial space and in the cell hyaloplasm and endoplasmic reticulum?
66. What intracellular enzymes are activated by increased level of calcium ions in the cell hyaloplasm?
67. What intracellular enzymes are activated at destruction of membrane of endoplasmic reticulum?
68. What are final effects of activation of phospholipases, ATP-ases, endonucleases and cell proteases?
69. What can be the causes of intracellular acidosis?
70. What are the consequences of decompensated cell acidosis?
71. What pathological processes can decouple the process of oxidation and phosphorylation in the mitochondria?
72. What the final effects of decoupling of mitochondrial oxidation and phosphorylation?
73. What are the consequences of energy deficiency in the cell?
74. What are the consequences of lysosomal membrane destabilization?
75. What are factors which stabilize and factors which destabilize lysosomal membrane?
76. What pathological processes lead to formation of reactive oxygen species?
77. What substances refer to free radicals?
78. What substances represent the endogenous antioxidant system?
79. What are the effects of reactive oxygen species?
80. What are the final effects of action of free radicals on cells?
81. **Cell dystrophy**
82. What are the causes of cell metabolic disorders?
83. What is the pathogenetic chain for development of fatty liver in persistent hypoglycemia?
84. What is the pathogenetic chain for development of fatty liver in persistent hyperglycemia?
85. What cellular pathological process can trigger cell dystrophy?
86. What are specific manifestations for cell dystrophy?
87. What are the causes for liver lipid metabolic disorders?
88. What is the pathogenetic chain for development of lipid dystrophy in long-lasting inanition?
89. What are possible consequences of cell dystrophy?
90. **Apoptosis**
91. Whatcells in the human bodiesare subjected to apoptosis?
92. What are manifestations of apoptosis in the initial period?
93. What factors represent positive apoptotic signals and negative apoptotic signals for initiation of apoptosis?
94. For what cells lack of growth factors, thyroid stimulation hormone, ACTH, estrogens, androgens represent a specific negative signals for initiation of apoptosis?
95. What condition is compulsory for complete evolution of apoptosis?
96. What are manifestations of apoptosis in the middle stage?
97. What are ending phenomena in apoptosis?
98. **Necrosis**
99. What represents necrosis?
100. What is the main pathogenetic loop of necrosis in injury of cytoplasmic membrane?
101. What is the main pathogenetic loop of necrosis in injury of mitochondria?
102. What is the main pathogenetic loop of necrosis in oxidative stress?
103. What are local consequences of necrosis?
104. What are general consequences of necrosis?
105. What biochemical tests can be used for diagnosis of tissue in which necrosis occurred?
106. **Tissular pathological processes**
107. What regenerative processes are possible in the cell at molecular level?
108. What regenerative processes are possible at cell organelle level?
109. What represents physiological regeneration?
110. What regeneration is considered to be homeostatic, adaptative, reparative, protective and compensatory?
111. What regeneration is quantitatively and qualitatively inadequate?
112. What represent anaplasia, metaplasia, dysplasia and sclerosis?
113. What represent hypertrophy and hyperplasia?
114. What physiological hypertrophy is considered adaptative, compensatory, functional?
115. What pathological hypertrophy is considered inflammatory, endocrine, neurotrophic and tumoral?
116. What atrophy is considered physiologic?
117. What atrophy can be considered functional, involutive, senile, posthypertrophic and endocrine?
118. What atrophy is considered pathologic?
119. What represent sclerosis?
120. What factors trigger sclerosis in the tissue?
121. What is the pathogenetic chain of sclerosis triggered by cell injury?
122. In what organs irreversible cell injuries lead to sclerosis?
123. What is the source of conjunctive tissue in pathogeny of sclerosis?
124. What is the mechanism of reducing excessive collagen fibers in the tissue?
125. What pathological processes lead to progressive sclerosis?
126. What are consequences of sclerosis?
127. What are principles of pathogenetic correction of sclerosis

**Questions II totalisation**

**MICROCIRCULATORY DISORDERS**

1. What is the main pathogenic link of arterial hyperemia?

2. What is the correlation between influx and reflux in arterial hyperemia?

3. What is the pathogenic mechanism of neurotonic arterial hyperemia?

4. What is the pathogenic mechanism of neuroparalytic arterial hyperemia?

5. What is the pathogenic mechanism of neuromyoparalytic arterial hyperemia?

6. What is the pathogenic mechanism of functional arterial hyperemia?

7. What are the manifestations of arterial hyperemia?

8. What does venous hyperemia represent?

9. What is the cause of venous hyperemia?

10. What is the main pathogenic link of venous hyperemia?

11. What are the external manifestations of venous hyperemia?

12. What is the cause of increasing organ volume in venous hyperemia?

13. What is the cause of decreasing local temperature in venous hyperemia?

14. What are the consequences of venous hyperemia?

15. What are the local pathogenic mechanisms of ischemia?

16. What local hemodynamic changes are characteristic for the ischemia?

17. How does the cellular metabolism change in ischemia?

18. What are the external manifestations of ischemia?

19. What organ has collaterals total functional insufficient?

20. What type of embolism is considered to be as endogenous?

21. What type of embolism is considered to be as exogenous?

22. What are the causes of air embolism?

23. What are the causes of gaseous embolism?

24. What vessel is obturated in case of embolism with amniotic liquid?

25. What are the consequences of artery embolism?

26. What factor disturbs rheological properties of the blood?

27. What does the edema represent?

28. What are the pathogenic links of hydrostatic edemas?

29. What are the pathogenic links of hypooncotic edemas?

30. What are the pathogenic links of hyperosmotic edemas?

31. What are the pathogenic links of membranogenic edemas?

32. What are the pathogenic links of lymphogenic edemas?

33. What are the pathogenic links of edemas in circulatory failure?

34. What are the pathogenic links of edemas in nephritic syndrome?

35. What are the pathogenic links of edemas in nephrotic syndrome?

36. What are the pathogenic links of edemas in liver failure?

37. What are the pathogenic links of edemas in cachexia?

38. What are the pathogenic links of edemas in allergy?

**Inflammation**

1. What is the etiology of inflammation?

2. Which harmful factors can cause primary alteration?

3. What are the manifestations of primary alteration in the inflammatory site?

4. What are the factors that cause secondary alteration in the inflammatory site?

5. What carbohydrates metabolism disorders are found in the inflammatory site?

6. What pathogenic factors determine local acidosis in the inflammatory site?

7. What are the sources of accumulated histamine in the inflammatory site?

8. What is the key enzyme that causes prostaglandin synthesis?

9. What is the origin of inflammatory mediators?

10. What are the cells-sources(precursors) of inflammatory mediators? Biological effects of cell mediators

11. What are mast cells mediators with chemotactic effect?

12. What is the key enzyme for leukotrienes synthesis?

13. What are the biological effects of prostaglandins PGD2, PGE2, PGF2?

14. What mediators are derived from arachidonic acid? List their biological effects

15. What are the biological effects of IL-1?

16. What are derived mediators from neutrophil leukocytes? List their biological effects

17. What bactericidal factors are generated by neutrophil leukocytes?

18. What biologically active factors are formed due to activation of the complement?

19. What biological effects do the components of the activated complement have?

20. What are plasma-derived inflammatory mediators? List their biological effects

21. What is the consequence of vascular reaction in the inflammatory site?

22. What mediators determine the development of arterial hyperemia in the inflammatory site?

23. What are the peculiarities of arterial hyperemia in the inflammation?

24. Which pathogenetic factors determine the increase of vascular permeability in inflammation?

25. Which factors determine the occurrence of venous hyperemia in the inflammatory focus?

26. What is the biological significance of arterial and venous hyperemia and stasis in the inflammation?

27. What are the pathogenic factors of exudation in the inflammation?

28. Give the characteristic of different kinds of exudates?

29. How does the transudate differ from exudate?

30. What are the cells that have phagocytic activity?

31. Which cells are macrophages?

32. What factors contribute to the leukocyte migration in the inflammatory focus?

33. What is the biological significance of leukocyte emigration in the inflammatory focus?

34. What are the phagocytosis phases? List their succession

35. What is the succession of the phenomena involved in devitalizing of internalized microbe?

36. What are the sources of hydrolases in the inflammatory focus?

37. What is the sequence of white blood cell migration in the inflammatory focus?

38. What are the cellular sources of regeneration and proliferation in the inflammatory focus?

39. What processes include physiological regeneration in the inflammatory focus?

40. What are the disorders in the body that reflect the acute phase response to inflammation?

41. What are systemic manifestations in the organism during the inflammation?

42. What disorders of the internal environment attest the presence of an inflammatory process in the organism?

43. Which hormones have pro-inflammatory action?

44. Which hormones have anti-inflammatory action?

**Hypersensibility disorders**

1. What does represent allergy?
2. What is the difference between immune reactions and allergic reactions?
3. What is the name of the factors which trigger allergic reactions?
4. What substances can be considered complete allergen?
5. What substances can be considered haptenes?
6. In what conditions the haptenes can trigger allergic reactions?
7. What are the conditions which favorise the development of cross-linked allergic reactions?
8. What endogenous allergen are natural natives allergenes?
9. What antigenes and tissues are considered „sequestred antigenes”?
10. What endogenous allergen are infectious aquired allergens?
11. What endogenous allergen are non-infectious aquired allergens?
12. What does represent exogenous allergenes?
13. What phenomena underlie on the basis of allergic reactions?
14. What are the stages of allergic reactions? The characteristics of stages
15. In what conditions there is considered that the body is sensitized?
16. In what conditions can develop passive sensitization?
17. What immunoglobulin is involved in type I allergic reaction?

18. On what immune cell the immunoglobulin E is attached in type I hypersensibility?

19. Where does occur the interaction between immunoglobulin and antigen in type I hypersensibility?

20.What is the characteristic of immunologial phase in hypersensibility disorders?

21. What are the sources of primary anaphylaxia mediators?

22.What mediators are released in the result of mast cell degranulation?

23. What are the biological effects of histamin?

24. What are the sources of secondary anaphylaxia mediators?

25. What mediators are derived from arachydonic acid?

26.What are mediators with brnchoconstrictor and bronchodilator effects?

27. What are mediators with vasoconstrictor and bronchoconstrictor effects?

28.What phenomena are characteristic for pathophysiological stage in hypersensibility disorders?

29. What are the characteristics of immunological stage in reaginic allergic reactions?

30. What are the mediators of anaphylactic reaction?

31. What mediator triggers bronchial spam in allergic bronnchial asthma?

32. What disorders underlie on the basis of type I hypersensibility?

33.What disorders underlie on the basis of cytotoxic-cytolytic allergic reaction?

34.What immunoglobulines are involved in type II hypersensibility?

35.What are mediators involved in type II hypersensibility?

 36.What complement fractions represents anaphylatoxins?

37. What does represent anaphylatoxin?

38. What are the mechanisms of cytotoxicity in type II hypersensibility?

39.What are the characteristics of pathochemical stage in type II hypersensibility?

40. What is the common pathogenetic mechanism in type III hypersensibility?

41. In what conditions the circulating immune complexes become aggresive?

42. What are the conditions which favor the long-lasting circulation of immune complexes?

43.What are the mechanisms of tissue injury in type III hypersensibility?

44. What is the sequence of phenomena which lead to injuries in type III hypersensibility?

45. What diseases underlie on the basis of type III hypersensibility?

46. What are the characteristics of immunological stage in delayed hypersensibility?

47. What cells are involved in type IV allergic reactions?

48. In what type of hypersensibility there is production of sensitized lymphocytes?

49. What are the mediators of pathochemical stage in delayed hypersensivbility?

50.What are the mechanisms of tissue injury in type IV allergic reaction?

51. What disorders underlie on the basis of delayed hypersensibility?

52. What does represent antibody mediated cellular dysfunction? What does represent the allergen in this reaction?

53. What immunoglobulins are involved in antibody mediated cellular dysfunction?

54. Where does occur the interaction between the antigen and antibodies in Graves disease?

55. What is the difference between antibody mediated cellular dysfunction and classical type II allergic reactions with cell injury?

56.What is the main pathophysiological mechanism on which underlie the autoimmune reactions?

57. In what autoimmune disorders the auto-antibodies are organ-specific?

58. In what autoimmune disorders the auto-antibodies are not organ-specific?

59. What type of allergic reaction underlies on the basis of autoimmune thyrotoxicosis?

60. In what type of allergic reaction there is recommended specific hyposensibilization?

61.What substances are used to perform specific hyposensibilization?

62. What does represent primary and secondary immunodefficiency?

**Questions III totalisation**

Metabolic dyshomeostasis

1. What substances are produced under bacterial fermentation in the digestive tract?

2. What conditions cause bacterial fermentation of carbohydrates in the stomach?

3. What is the consequence of bacterial fermentation of the carbohydrates in the stomach?

4. What is the cause of zaharides maldigestion?

5. How does the secretion of insulin and glucagon change in excessive ingestion of carbohydrates?

6. How does the secretion of insulin and glucagon change in period of starvation?

7. How does change the glycemia in liver failure?

8. In what cases is increased bacterial fermentation of carbohydrates at the level of large intestine?

9. What are the consequences of carbohydrates fermentation in the large intestine?

10. What is the consequence of cellulose deficiency in food ration?

11. How does the carbohydrate metabolism change in liver failure?

12. What is the cause of monozaharides malabsorption at the level of small intestine?

13. What is the consequence of carbohydrates malabsorption?

14. How does the content of nutritive substances from the liver change in carbohydrates deficiency?

15. What are the compensatory reactions for maintaining normoglycemia in long lasting starvation?

16. How does the function of endocrine glands change in carbohydrate deficiency?

17. What are the endogenous sources of glucose for maintaining normal value of glycemia in long lasting starvation?

18. What is the mechanism of protein usage for gluconeogenesis during starvation?

19. What proteins undergo catabolic processes during carbohydrate deficiency?

20. What are the possible causes for increased gluconeogenesis from endogenous proteins?

21. What are the endocrine reactions to excessive consumption of carbohydrates?

22. Which hormone inhibits process of gluconeogenesis?

23. Which hormone inhibits process of glycogenolysis?

24. Which hormone activates process of hepatic glycogenogenesis?

25. What are the homeostatic reactions of hyperglycemia?

26. What is the mechanism of glucosuria in excessive carbohydrates consumption?

27. What are the causes of hypoglycemia?

28. What are the compensatory reactions of hypoglycemia?

29. What are the consequences of hypoglycemia?

30. How does the lipidemia change in hypoglycemia?

31. How does the content of lipids and glycogen in the liver change in hypoglycemia?

32. What type of liver dystrophy is possible in case of long lasting hypoglycemia?

33. What are the consequences of alimentary hyperglycemia?

34. What are the consequences of long lasting hyperglycemia?

35. What are the compensatory mechanisms in alimentary hyperglycemia?

36. What is the pathogenic chain of disturbances as result of hyperglycemia?

37. How does the circulatory volume and viscosity of the blood change in hyperglycemia caused by hypoinsulinism?

38. What are the effects of catecholamines in the period of carbohydrate deficiency?

39. How does hydric metabolism change in diabetic hyperglycemia?

40. What factors determine the pathogenesis of diabetic ketoacidosis?

41. What is the pathogenetic mechanism of diabetic ketoacidosis?

42. What are the adverse effects of gluconeogenesis intensification from aminoacids in hypoglycemia, as compensatory mechanism?

43. How does the blood osmolarity, glycemia, the volume of circulatory blood, arterial systemic pressure and diuresis change in diabetic ketoacidosis?

44. What are the changes of acid – base balance in the intra- and extracellular space in diabetic ketoacidosis?

45. What are the paraclinic signs of hydroelectrolitic imbalance in diabetic ketoacidosis?

46. What is the pathogenetic mechanism of hydroelectrolytic imbalance in diabetic ketoacidosis?

47. What is the pathogenetic mechanism of Kussmaul breathing in diabetic ketoacidosis?

48. What is the consequence of persistent hyperglycemia in insulin deficiency?

49. What is the pathogenetic mechanism of dehydration in diabetic ketoacidosis?

50. What is the pathogenetic mechanism of hypoglycemia in administration of exogenous insulin?

51. What are the functional changes of neuron in carbohydrate starvation?

52. What is the pathophysiologic mechanism of ketogenesis in carbohydrate starvation?

53. What is the pathogenetic mechanism of hypoglycemia in case of insulinom (tumor of beta cells of pancreas)?

54. What is the “threshold” value of glucose reabsorption in the primary urine at the level of renal tubes?

55. What is the role of kidney in carbohydrate starvation?

56. How does the carbohydrate metabolism change in primary hypercorticism?

57. What are the causes of galactosemia?

58. What are the consequences of galactosemia in new-borns?

59. How does the blood content of the blood change in excessive consumption of the lipids?

60. What are the metabolic consequences of excessive consumption of the lipids?

61. What are the consequences of lipids insufficiency?

62. What are the causes of lipids maldigestion?

63. What is the pathogenetic mechanism of lipids maldigestion?

64. What are the consequences of lipids malabsorption?

65. What substances are not absorbed at the level of intestinal epithelium in case of enteritis?

66. Which endocrine disorders lead to intense mobilization of lipid reserves with transport hyperlipidemia?

67. What is the pathogenetic mechanism of lipid maldigestion in inflammation of small intestine mucosa?

68. What is the pathogenetic mechanism of transport hyperlipidemia in excess of glucocorticoids?

69. Which are the digestive effects under excessive consumption of fats?

70. What are the consequences of lipid maldigestion?

71. Which lipoproteins fraction will increase in case of retention hyperlipidemia in insulin deficiency?

72. How does the lipids fraction from the blood change in lipid maldigestion?

73. In what form of lipoproteins are transported absorbed lipids from the small intestine?

74. In what form of lipoproteins are transported synthesized lipids in the liver?

75. In what form of lipoproteins are transported mobilized lipids from adipose tissue?

76. In what form is transported cholesterol to the organs?

77. What are the consequences of transport hyperlipidemia in insulin deficiency?

78. In what forms of lipoproteins is transported cholesterol from the organs to the liver?

79. What are the causes of retention hyperlipidemia?

80. What type of hyperlipidemia shows increased level of chylomicrons n the blood?

81. What are the consequences of alimentary hyperlipidemia?

82. What are the changes of lipid metabolism in liver failure?

83. What is the pathogenetic mechanism of hyperlipidemia in insulin deficiency?

84. What is the pathogenetic mechanism of hyperlipidemia in stress reaction?

85. What is the pathogenetic mechanism of transport hyperlipidemia in case of catecholamines hypersecretion?

86. How does the lipid metabolism change in excessive ingestion of sodium chloride?

87. How does the lipid metabolism change in chronic pathology of pancreas?

88. What is the pathogenetic mechanism of lipid malabsorption in affection of terminal ileum?

89. What is the pathogenetic mechanism of lipid malabsorption in liver failure?

90. What is the pathogenetic mechanism of lipid malabsorption in occlusion of intestinal lymphatic vessels?

91. What is the pathogenetic mechanism of retention hyperlipidemia in case of nephritic syndrome?

92. What is the pathogenetic mechanism of lipid dystrophy of liver in case of protein starvation?

93. What are the pathogenetic factors of fatty liver in case of excessive lipid consumption?

94. What are the etiological factors of atherosclerosis?

95. Which hormones accelerate the metabolic rate and reduce serum level of blood cholesterol?

96. What does represent hypoproteinemia?

97. What is the etiological factor of Kwashiorkor syndrome?

98. What are the consequences of excessive consumption of proteins?

99. What are the pathological factors of protein maldigestion in protein starvation?

100. What is the consequence of absorption of native alimentary proteins in the digestive tract?

101. How does the blood protein content change in liver failure?

102. What disturbances of digestive tract lead to maldigestion of proteins?

103. How does the protein metabolism change in maldigestion of proteins?

104. How does the digestive processes from large intestine change in maldigestion of protein?

105. In what pathological processes develops hypoproteinemia?

106. In what pathological processes develops maldigestion of proteins?

107. What is the mechanism of dehydration in protein starvation?

108. What are the changes of oncotic pressure and diuresis in protein starvation?

109. What is the cause of hemorrhage syndrome in liver failure?

110. What are the causes of aminoacids malabsorption at the level of intestinal mucosa?

111. How does the protein metabolism change in liver failure?

112. What are the consequences of hypoproteinemia?

113. In what pathological processes develops hyperproteinemia?

114. What are the consequences of hyperproteinemia?

115. What substances are formed in excess under intensification of nucleoproteins catabolism?

116. In what cases is found negative nitrogen balance?

117. In what cases is found positive nitrogen balance?

118. What is the mechanism of peripheral edemas in protein starvation?

119. What are the pathogenetic factors of immunodeficiency in protein starvation?

120. What is the pathogenetic factor of hyperaminoacidemia in liver failure?

121. How does the protein metabolism change in liver failure?

122. What are the consequences of reduced detoxification function of the liver in liver failure?

123. What are the consequences of protein metabolic changes in liver failure?

124. What substances are formed into the large intestine under the protein putrefaction?

125. What toxic substances provoke intestinal autointoxication?

126. What pathological processes provoke intestinal autointoxication?

127. What is the pathologic factor of hemic hypoxia in protein starvation?

128. What is the consequence of hyperamonemia in liver failure?

129. What is the pathogenetic mechanism of infections in liver failure?

130. What type of acid –base imbalance is specific for exaggerated consumption of proteins?

**Hydroelecrolytic dishomeostasis**

1. What does represent dehydration/overhydration?
2. What are the causes of hypertonic/isotonic/hypotonic hyperhydration?
3. What are the causes of hypertonic/isotonic/hypotonic dehydration?
4. What is the pathogeny of hypertonic/isotonic/hypotonic hyperhydration?
5. What is the pathogeny of hypertonic/isotonic/hypotonic dehydration?
6. In what pathological processes do develop hypertonic/isotonic/hypotonic hyperhydration?
7. In what pathological processes do develop hypertonic/isotonic/hypotonic dehydration?
8. How the plasma osmolarity and cell volume is changed in hypertonic/isotonic/hypotonic hyperhydration?
9. How the plasma osmolarity and cell volume is changed in hypertonic/isotonic/hypotonic dehydration?
10. How the capillary-interstitial and interstitium-cell water exchange are changed in hypertonic/isotonic/hypotonic dehydration?
11. How the capillary-interstitial and interstitium-cell water exchange are changed in hypertonic/isotonic/hypotonic hyperhydration?
12. What are the changes in the blood (hemic changes) in hypertonic/isotonic/hypotonic hyperhydration?
13. What are the changes in the blood (hemic changes) in hypertonic/isotonic/hypotonic dehydration?
14. What are electrolytic changes in the blood in hypertonic/hypotonic hyperhydration?
15. What cardiovascular manifestations are present in overhydration/dehydration?
16. What hemic changes are present in overhydration/dehydration?
17. What is the pathogenetic chain of arterial hypotension in dehydration?
18. How the protein blood level is changed in dehydration/overhydration?
19. What hydroelectrolytic disorders develop in vasopressin deficiency/vasopressin excess?
20. How the blood osmolarity and sodium concentration is changed in vasopressin deficiency/vasopressin excess?
21. How the blood osmolarity and cell volume is changed in vasopressin deficiency/vasopressin excess?
22. What is the pathogeny of hydroelectrolytic imbalance in vasopressin deficiency/vasopressin excess?
23. What is the pathogeny of hydroelectrolytic imbalance in water deprivation?
24. How the blood osmolarity and sodium concentration are changed in water deprivation?
25. How the blood osmolarity and cell volume is changed in water deprivation?
26. How the blood osmolarity, sodium concentration and protein level in the blood are changed in water deprivation?
27. What is the pathogeny of hypernatremia in water deprivation?
28. How the blood osmolarity, sodium concentration and cell volume are changed in excessive intake of water?
29. How the blood osmotic pressure and oncotic pressure are changed in excessive intake of water?
30. What is the pathogeny of hydroelectrolytic imbalance in enhanced sweating?
31. How the blood osmolarity, sodium concentration and protein level in the blood are changed in excessive sweating?
32. How the plasma osmolarity and cell volume are changed in excessive sweating?
33. How the plasma osmolarity and sodium concentration in the blood are changed in excessive sweating?
34. What is the pathogeny of hydroelectrolytic imbalance in pulmonary hyperventilation?
35. How the plasma osmolarity and cell volume are changed in pulmonary hyperventilation?
36. How the plasma osmolarity and sodium concentration in the blood are changed in pulmonary hyperventilation?
37. How the blood osmolarity, chlorine concentration and hydrogen ion level area changed in incoercible vomiting?
38. What hydroelectrolytic dyshomeostasis does develop in hypertonic dehydration?
39. How does occur the compensation of hydric dyshomeostasis in hypertonic dehydration?
40. What hydroelectrolytic dyshomeostasis does develop in diarrhea?
41. What electrolytic dyshomeostasis does develop in hypertonic/isotonic/hypotonic dehydration?
42. What is the pathogenetic mechanism of hyperkalemia in hypotonic dehydration?
43. What is the compensatory mechanism of osmotic dyshomeostasis in hypotonic dehydration?
44. What is the pathogeny of hydroelectrolytic imbalance in mineralocorticoid deficiency?
45. What change of circulatory blood volume is attested in hyperhydration?
46. How the oncotic pressure is changed in hyperhydration?
47. What are the pathogenetic mechanisms of edema in hyperhydration?
48. What are the compensatory reactions in isotonic hyperhydration?
49. How is define hypotonic/isotonic/hypertonic hyperhydration?
50. What are the pathogenetic mechanisms of hyperkalemia in hypotonic hyperhydration?
51. What pathological manifestations develop in hypotonic hyperhydration?
52. What is the mechanism of hemoglobinuria in hypotonic hyperhydration?
53. What is the pathogeny of hydric dyshomeostasis in aldosteron hypersecretion/aldosteron hyposecretion?
54. How the blood osmolarity, sodium level and hydrogen ion level are changed in diarrhea?
55. How the blood osmolarity, protein level in the blood, and potassium level in the blood are changed in II-III degree burns?
56. How the blood volume and concentration of blood cells are changed in intravascular dehydration?
57. What are compensatory reactions in intravascular dehydration/intravascular overhydration?
58. How the osmotic and oncotic blood pressure are changed in massive infusion of isotonic NaCl solution?
59. How the volume of interstitial and intracellular fluids is changed in massive infusion of NaCl solutions?
60. What are the immediate changes of blood oncotic and osmotic pressures after massive infusion of 5% glucose solution?
61. What are the late changes of blood oncotic and osmotic pressures after massive infusion of 5% glucose solution?
62. What are the immediate changes of interstitial and intracellular fluid volume after massive infusion of 5% glucose solution?
63. What are the late changes of interstitial and intracellular fluid volume after massive infusion of 5% glucose solution?
64. How is defined hypernatremia/hyponatremia?
65. What does represent hypernatremia/hyponatremia?
66. How the circulatory blood volume and blood viscosity are changed in absolute hypernatremia/relative hypernatremia?
67. How the circulatory blood volume and concentration of blood cells are changed in absolute hypernatremia/relative hypernatremia?
68. How the circulatory blood volume and blood viscosity are changed in absolute hyponatremia/relative hyponatremia?
69. How the circulatory blood volume and concentration of blood cells are changed in absolute hyponatremia/relative hyponatremia?
70. What hydric dyshomeostasis does represent absolute hypernatremia/relative hypernatremia?
71. What hydric dyshomeostasis does represent absolute hyponatremia/relative hyponatremia?
72. What is the pathogeny of absolute hypernatremia/relative hypernatremia?
73. What is the pathogeny of absolute hyponatremia/relative hyponatremia?
74. In what pathological processes develops absolute hypernatremia/relative hypernatremia?
75. In what pathological processes develops absolute hyponatremia/relative hyponatremia?
76. What sodium dyshomeostasis does develop in hypersecretion of vasopressin/hyposecretion of vasopressin?
77. What sodium dyshomeostasis does develop in hypersecretion of mineralocorticoids/hyposecretion of mineralocorticoids?
78. How the blood osmolarity, cell volume and intravascular volume are changed in absolute hypernatremia/relative hypernatremia?
79. How the blood osmolarity, cell volume and intravascular volume are changed in absolute hyponatremia/relative hyponatremia?
80. What is the pathogeny of cell volume changes in absolute hypernatremia/relative hypernatremia?
81. How the arterial blood pressure is changed in sodium deficiency?
82. What is the pathogeny of sodium imbalance in chronic liver disorders?
83. What sodium dyshomeostasis does develop in chronic liver disorders?
84. What are the compensatory reactions in absolute hypernatremia?
85. What is the final consequence of hypernatremia for cells
86. What are the electrolytic changes in primary hyperaldosteronism?
87. What is the pathogeny of edema in primary hyperaldosteronism?
88. In what pathological processes develop secondary hyperaldosteronism?
89. What is the normal concentration of potassium ions in the blood?
90. What value of potassium in the blood does represent hyperkalemia/hypokalemia?
91. In what pathological processes does develop hyperkalemia/hypokalemia?
92. What acidobazic dyshomeostasis does develop in hyperkalemia/hypokalemia?
93. What is the pathogeny of acidobazic dyshomeostasis in hyperkalemia/hypokalemia?
94. What are the changes on ECG in hyperkalemia/hypokalemia?
95. How does the resting membrane potential and neuromuscular excitability change in moderate hyperkalemia( 5,5 - 6,0 mEq/L)?
96. How does the resting membrane potential and neuromuscular excitability change in severe hyperkalemia(peste 6,5 mEq/L)?
97. How does the resting membrane potential and neuromuscular excitability change in hypokalemia?
98. What are the electrophysiological changes in the heart in hyperkalemia/hypokalemia?
99. What is the pathogeny of potassium dysbalance in chronic liver disorders?
100. What is the pathogeny of potassium dysbalance in treatment with insulin?
101. What is the pathogeny of potassium dysbalance in hyperglycemia?
102. What is the pathogeny of potassium dysbalance in acidosis/alkalosis?
103. What are the clinical manifestations of hyperkalemia/hypokalemia?
104. What is the mechanism of increased amplitude of T wave on ECG in hyperkalemia?
105. What is the mechanism of additional U wave on ECG in hypokalemia?
106. What is the normal Ca2+ions concentration in the blood?
107. What hormones control the calcium balance in the body?
108. What hormones have hypercalcemiant/hypocalcemiant effect?
109. What is the mechanism by which parathyroid hormone regulates the calcium balance?
110. What is the mechanism by which thyreocalcitoninregulates the calcium balance?
111. What are the biological functions of calcium ions?
112. What are the causes of hypercalcemia/hypocalcemia?
113. What are the pathogenetic mechanisms of hypercalcemia/hypocalcemia?
114. What are the pathophysiological mechanisms of calcium dyshomeostasis in liver disorders/renal disorders?
115. What is the pathogeny of calcium dysbalance in catecholamine hypersecretion?
116. What is the pathogeny of calcium dyshomeostasis in excess of parathyroid hormone/deficiency of parathyroid hormone?
117. What is the pathogeny of neuromuscular excitability disorders in hypercalcemia/hypocalcemia?
118. What are the cardiovascular manifestations in hypocalcemia?
119. What are the manifestations in hypercalcemia/hypocalcemia?
120. What is pathogeny of osteomalacia in hypocalcemia?
121. What are the compensatory reactions in hypercalcemia/hypocalcemia?
122. What severe complications of hypocalcemia can develop in children?
123. What is the mechanism of hypocalcemia in bile deficiency in the small intestine?

**Acido-base dyshomeostasis**

1. What does represent acidosis/alkalosis?
2. What are the criteria of acidosis/alkalosis?
3. What does represent respiratory acidosis/respiratory alkalosis?
4. What does represent metabolic acidosis/metabolic alkalosis?
5. What does represent compensated acidosis/decompensated acidosis?
6. What does represent compensated alkalosis/decompensated alkalosis?
7. In what processes does develop metabolic acidosis/respiratory acidosis?
8. In what processes does develop metabolic alkalosis/respiratory alkalosis?
9. What are the pathogenetic factors of metabolic acidosis/respiratory acidosis?
10. What are the pathogenetic factors of metabolic alkalosis/respiratory alkalosis?
11. How do change pH and PaCO2 in metabolic acidosis/metabolic alkalosis?
12. How do change pH and PaCO2 in respiratory acidosis/respiratory alkalosis?
13. How do change pH and NaHCO3in metabolic acidosis/metabolic alkalosis?
14. How do change pH and NaHCO3 in respiratory acidosis/respiratory alkalosis?
15. How do change respiratory frequency and PaCO2in metabolic acidosis/metabolic alkalosis?
16. What acidobazic dyshomeostasis develop in aldosteron hypersecretion/aldosteron hyposecretion?
17. What is the pathogeny of acidobazic dyshomeostasis in aldosteron hypersecretion/aldosteron hyposecretion?
18. Accumulation of what endogenous substances can lead to acidosis?
19. Accumulation of what acid compounds lead to development of metabolic acidosis in insulin deficiency?
20. What is the pathogenetic factor of acidobazic dyshomeostasis in insulin deficiency?
21. How do change pH and bicarbonate in insulin deficiency?
22. What is the pathogeny of acidobazic dyshomeostasis in inanition (starvation)?
23. How do change pH and bicarbonate in inanition?
24. What is the pathogeny of acidobazic dyshomeostasis in hypoxia?
25. How do change pH and bicarbonate in hypoxia?
26. What is the pathogeny of acidobazic dyshomeostasis in diarrhea?
27. What is the pathogeny of acidobazic dyshomeostasis in liver disorders?
28. What is the pathogeny of acidobazic dyshomeostasis in pulmonary hyperventilation/pulmonary hypoventilation?
29. How do change pH and PaCO2in pulmonary hypoventilation/pulmonary hyperventilation?
30. With what electrolytic dyshomeostasis are associated metabolic acidosis/metabolic alkalosis?
31. What is the pathogeny of sodium dyshomeostasis in acidosis/alkalosis?
32. What is the pathogeny of potassium dyshomeostasis in acidosis/alkalosis?
33. What is the pathogeny of calcium dyshomeostasis in acidosis/alkalosis?
34. What is the cause of pulmonary hyperventilation in metabolic acidosis?
35. What is the cause of pulmonary hypoventilation in metabolic alkalosis?
36. What are the causes of respiratory changes in metabolic acidosis/metabolic alkalosis?
37. In what acidobazic dyshomeostasis does develop compensatory pulmonary hyperventilation?
38. In what acidobazic dyshomeostasis does develop compensatory pulmonary hypoventilation?
39. What are the compensatory reactions in metabolic acidosis/metabolic alkalosis?
40. What are clinical manifestations in acidosis/alkalosis?
41. What are the pathophysiological mechanisms of reduced neuromuscular excitability in acidosis?
42. What are the changes in the blood in metabolic acidosis/respiratory acidosis?
43. What are the changes in the blood in metabolic alkalosis/respiratory alkalosis?
44. What is pathogeny of arterial hypotension in acidosis?
45. How des involve the hemoglobin buffer system in acidosis compensation?
46. What is the pathogenetic factor of osteomalacia and osteoporosis in acidosis?
47. What are the effects of hypercapnia in respiratory acidosis?
48. How does change the oxyhemoglobin dissociation curve and oxygen affinity to hemoglobin in acidosis/alkalosis?
49. What are the compensatory reactions in acidosis/alkalosis?
50. What does hypoxia represent?
51. What does hypoxemia represent?
52. On what depends vulnerability of the different organs to hypoxia?
53. Which is the vulnerability of different organs to hypoxia (in descending order)?
54. What type of hypoxia develops in alpine disease?
55. What type of hypoxia develops in disturbance of processes of intracellular using of oxygen?
56. What is the pathogenesis of hemic hypoxia in intoxication with nitrates?
57. What is the pathogenesis of hemic hypoxia in hemoglobinopathy?
58. What is the pathogenesis of hemic hypoxia in bleeding?
59. What is the pathogenesis of hemic hypoxia in intoxication with carbon monoxide?
60. In what cases develops histotoxic hypoxia?
61. What pathological conditions characterized by deviation to the right of oxyhemoglobin dissociation curve deviates to the right?
62. What pathological conditions characterized by deviation to the right of oxyhemoglobin dissociation curve deviates to the left?
63. What are the manifestations of brain hypoxia?
64. What pathological processes develop in the brain in condition of decreased arterial partial pressure of O2 bellow 20mmHg
65. In what conditions increases the rate of dissolved O2 in the blood?
66. What does hyperoxia represent?
67. In what pathological processes develops the hyperdynamic hyperoxia?
68. What does the dysmetabolic hyperoxia represent?
69. In what pathology is contraindicated therapeutic application of oxygenum?
70. What are the harmful effects of hyperoxia?
71. What is the mechanism of CO2 accumulation in hyperoxia?
72. How does the acid-base balance change in hyperoxia?

**Questions of IV totalisation**

1. What is the measure of neuronal excitability?
2. What does represent the neuronal excitability threshold?
3. How the neuronal excitability is changed in reduced resting membrane potential?
4. How the neuronal excitability is changed in increased resting membrane potential?
5. What proces does increase the neuronal excitability?
6. How the resting memrane potential is changed when the activity of membranary Na+/K+ pumps is stopped?
7. How the intraneuronal elecrolytes concentration is changed when the activity of membranary Na+/K+ pumps is stopped?
8. How the cessation of membranary Ca, Mg pumps affects the intraneuronal Ca++ concentration ?
9. What is the pathogeny of neuronal excitability disorders?
10. What is the mechanism of neuronal excitability disorders in condition of enhanced lipid peroxidation?
11. What is the mechanism of action of excitatory neurotransmitters?
12. What is the mechanism of action of inhibitory neurotransmitters?
13. What pathological processes can lead to neuronal degeneration?
14. What is the main pathogenetic loop in neuronal degeneration?
15. What are the specific mechanisms of neuronal degeneration?
16. What are the non-specific mechanisms of neuronal degeneration?
17. What effects are induced by neuronal deafferentation?
18. What is the mechanism of enhanced neuronal excitability in hypoxia?
19. What factors stabilize the neuronal membrane?
20. What is the pathogeny of excitability disturbance in hypercalcemia?
21. How the GABA (gama-aminobutyric acid) is changing the neuronal excitability?
22. What is the pathogeny of neuronal excitability disorders in ischemia?
23. What is the pathogenetic chain of neuronal degeneration in cerebral hypoperfusion?
24. What is the role of lipid peroxide in initiation of neuronal disorders?
25. What is the mechanism by which coffein is changing the neuronal excitability?
26. What is the mechanism by which alcohol is changing the neuronal excitability?
27. What factors can trigger neuronal hyperexcitability (tetanic syndrome) ?
28. What factors can trigger neuronal hypoexcitability?
29. What pathogenetic factors disturb the neurotransmitter synthesis?
30. What is the role of Ca++ in neuronal functionality?
31. What is the mechanism of acethylcholin action on inervated structures?
32. What substances block the neuromuscular transmission?
33. What stages of neuromuscular transmission are affected by botulinus toxin?
34. What substances disturb the axonal transportation of neurotransmitters? What is the pathogenetic loop?
35. What pathogenetic factors can trigger neuronal apoptosis?
36. What toxins can reach the spinal neurons by retrograde axonal transportation?
37. What mechanisms ensure the neuronal surviving in transversal axonal section? What can be the etiological factors for these?
38. What are the pathogenetic mechanism in neuronal demielinization?
39. What neurological disturbances can develop when there is demielinization at the level of posterior medullary horns?
40. What are the effects of blockage of post-synaptic receptors?
41. What is the effect of neurotransmitter exaustion at the level of nervous ending?
42. What is the effect of noradrenalin exhaustion at the level of synaptic cleft?
43. What is the effect of reduced dopaminergic innervation?
44. What factors can affect the neurotransmitter release in the synaptic cleft?
45. What factors can enhance/decrease the neurotransmitter effect on postsynaptic neurones?
46. What factors trigger the activation of sympathetic nervous system?
47. What are the effects of enhanced tonus of sympathetic nervous system?
48. What are the etiological factors of segmentary vegetative disturbances?
49. What is the pathogeny of suprasegmentary vegetative disturbances?
50. What vegetative disturbances can be induced by glucocorticoid hormones?
51. What vegetative disturbances can be induced by gondotropines?
52. What vegetative disturbances develop in hyperexcitability of sympathetic vegetative centres in the hypothalamus?
53. What is the effect of enhanced sympathetic tonus on carbohydrate metabolic processes?
54. What is the effect of enhanced sympathetic tonus on lipid metabolic processes?
55. What is the effect of enhanced sympathetic tonus on endocrine glands?
56. What is the effect of enhanced sympathetic tonus on cardiovascular system?
57. What is the effect of enhanced sympathetic tonus on digestive tract ?
58. What is the effect of enhanced sympathetic tonus on airways?
59. What is the effect of enhanced sympathetic tonus on coronary blood vessels?
60. What is the effect of enhanced sympathetic tonus on abdominal blood vesels?
61. What are the effects of enhanced sympathetic tonus on circulatory system?
62. What is the effect of enhanced sympathetic tonus on external sexual organs?
63. What are the effect of enhanced sympathetic tonus on eyes?
64. What are the effects of enhanced sympathetic tonus on skin structures?
65. What are the effects of enhanced parasympathetic tonus?
66. What are the effects of enhanced parasympathetic tonus on digestive tract?
67. What are the effects of enhanced parasympathetic tonus on airways?
68. What are the effects of enhanced parasympathetic tonus on external sexual glands?
69. What are the effects of enhanced parasympathetic tonus on eyes?
70. What disturbances develop in hyperexcitability of hypothalamic parasympathetic neurones?
71. What is the pathogeny of generalized seisures in the frog after administartion of stryknine?
72. What are the effects of striknine?
73. What disturbances are induced by aminasin administration in the rat?
74. What is the pathogeny of adynamia in the rat aftr aminasin admnistration?
75. What does represent hypocortisolism?
76. What is the main pathogenetic loop in tertiary hypocortisolism?
77. What is the main pathogenetic loop in secondary hypocortisolism?
78. What is the main pathogenetic loop in primary hypocortisolism?
79. What is a possible causing factor of primary hypocortisolism/secondary hypocortisolism?
80. What are the factors that contribute to development of clinical manifestations in prymary/secondary and tertiary hypocortisolism?
81. What are the principles of pathogenetic therapy in primary/secondary and tertiary hypocortisolism?
82. What are the principles of direct (descendent) regulation of hypothalamus-hypophysis-adrenal axis?
83. What are the principles of retrograde (ascendent) regulation of hypothalmus-hypophysis-adrenal axis?
84. What is the pathogeny of adrenal gland atrophy in exogenous administration of glucocorticoids?
85. How the vascular tonus is changed in glucocrticoid hyposecretion?
86. What are the mechanism of circulatory disturbances in lack of glucocorticoids?
87. How the vascular tonus can be maintained in patients with hypocortisolism?
88. How the cardiovascular function is changed in hypocortisolism?
89. What is the mechanism of cardiovascular insufficiency in lack of glucocorticoids?
90. What hormonal investigations are necessary for differentiation of primary, secondary and tertiary hypocortisolism?
91. What are the hormonal changes in primary/secondary and tertiary hypocortisolism?
92. What is the clinical manifestation which develops only in primary hypocortisolism?
93. What is the clinical manifestation which develops only in secondary hypocortisolism?
94. What is the pathogeny of skin hyperpigmentation in primary hypocortisolism?
95. What is the pathogeny of skin depigmentation in secondary hypocortisolism?
96. How the resistence to stress in changed in patients with hypocortisolism?
97. What are the risk of stress in patients with hypocortisolism?
98. What is the mechanism of hypoglycemia in patients with lack of glucocorticoids?
99. What is the mechanism of arterial collapse in glucocorticoid lack?
100. What is the mechanism of cardiac failure in glucocorticoid lack?
101. How the cardiovasdcular complications in stress can be prevented in the ptients with hypocorticosolism?
102. How the adaptation abilities to stressful conditons is changed in patients with hypocortisolism?
103. What are the most severe consequences in condition of stress in patients with hypocortisolism?
104. How does carbohydrate metabolism disturb in patients with hypocortisolism?
105. What is the vital importance of glucocorticosteroids in extreme conditions?
106. How do insulin and glucagon secretion disturb in the hypocortisolism?
107. How does the inflammatory reaction occur in people with hypocortisolism?
108. What is the mechanism of hyperergic inflammatory reaction in people with hypocortisolism?
109. What is the mechanism of exaggerated arterial hyperemia into inflammatory focus due to hypocortisolism?
110. What is the mechanism of exaggerated exudation into inflammatory focus due to hypocortisolism?
111. How does allergic reactivity disturb in patients with hypocortisolism?
112. What kind of hormones are deficient in the experimental animal after the surgical removal of adrenal glands?
113. Lack of which one hormone compensates exogenous sodium chloride in the laboratory animal without adrenal glands?
114. What adaptive reactions require physical effort with adrenal gland involvement?
115. What created dishomeostasis due to lack of glucocorticosteroids diminishes the animal's resistance to physical effort?
116. What are mechanisms of body weight loss due to hypocortisolism?
117. What is pathogenesis of adipose tissue atrophy due to hypocortisolism?
118. What is pathogenesis of atrophy of skeletal muscles due to hypocortisolism?
119. What is pathogenesis of muscle weakness in total hypocortisolism?
120. How is the decrease in diastolic blood pressure explained due to hypocortisolism ?
121. How is the decrease in systolic blood pressure explained due to hypocortisolism ?
122. How is tachycardia explained in hypocortisolism?
123. What is the pathogenesis of hyperpigmentation of the skin in hypocortisolism?
124. What is the direct cause of loss of consciousness in the orthostatic test in the patient with hypocortisolism?
125. How does BCC disturb due to hypocortisolism?
126. What is the direct cause of hypoglycemia due to hypocortisolism?
127. What is pathogenesis of hyponatremia and hyperkalaemia due to total hypocortisolism?
128. In what form of hypocortisolism the cortisol concentration is low; ACTH concentration increase; aldosterone concentration - decreases?
129. What is the possible etiology of primary hypocortisolism?
130. What are the principles of the pathogenetic therapy of total primary hypocortisolism?
131. What premedication is required for patients with hypocortisolism undergoing surgery?
132. What is hypercortisolism?
133. What is the main pathogenetic link of tertiary hyperacortisolism?
134. What is the primary pathogenetic link of secondary hyperacortisolism?
135. What is the primary pathogenetic link of primary hyperacortisolism?
136. What is the possible cause of tertiary hyperacortisolism?
137. What is the possible cause of secondary hyperacortisolism?
138. What is the possible cause of primary hyperacortisolism?
139. How are the clinical manifestations of tertiary hypercortisolism explained?
140. How are the clinical manifestations of secondary hypercortisolism explained?
141. How are the clinical manifestations of primary hypercortisolism explained?
142. What are pathogenetic principles of treatment of tertiary hyperacortisolism?
143. What are pathogenetic principles of treatment of secondary hypercortisolism?
144. What are pathogenetic principles of treatment of primary hypercortisolism?
145. How does electrolyte balance disturb in hypersecretion of glucocorticosteroids?
146. What is mechanism of disorders in electrolyte balance due to glucocorticosteroids hypersecretion?
147. How do calcium metabolism and bone status disturb due to glucocorticosteroids hypersecretion?
148. How does systemic hemocirculation disturb due to glucocorticosteroids hypersecretion?
149. How does heart function disturb due to glucocorticosteroids hypersecretion?
150. What are mechanisms of cardiovascular functions disorders due to glucocorticosteroids hypersecretion?
151. How does the immune system function disturb due to glucocorticosteroids hypersecretion?
152. How do digestive functions disturb due to glucocorticosteroids hypersecretion?
153. How does specific body immunity disturb due to glucocorticosteroids hypersecretion?
154. How does carbohydrate metabolism disturb due to glucocorticosteroids hypersecretion?
155. What is pathogenesis of hyperglycaemia due to hypersecretion of glucocorticosteroids?
156. How does lipid metabolism disturb due to glucocorticosteroids hypersecretion?
157. What is pathogenesis of fatty tissue hypertrophy due to hypercortisolism?
158. How does protein metabolism disturb due to glucocorticosteroids hypersecretion?
159. What are the biochemical disorders of the blood due to hypercortisolism?
160. How does hypersecretion of glucocorticosteroids influence the lax connective tissue?
161. How does wound healing occur due to glucocorticosteroids hypersecretion?
162. What is the pathogenesis of recurrent gastric ulcer in the patient treated with glucocorticosteroids?
163. How do lymphoid tissue and thymus gland disturb due to glucocorticosteroids hypersecretion?
164. How does specific immune response disturb due to glucocorticosteroids hypersecretion?
165. How does the inflammatory reaction occur in excess of glucocorticosteroids?
166. How do vascular reactions into inflammatory focus occur in excess of glucocorticosteroids?
167. How does the exudates disturb due to excess of glucocorticosteroids?
168. How does leukocyte emigration disturb into inflammatory focus in excess of glucocorticosteroids?
169. What are mechanisms of body weight gain due to hypercortisolism?
170. What is the cause of pathological fractures due to hypercortisolism?
171. What is mechanism of muscle asthenia due to hypercortisolism?
172. What is the possible pathogenesis of diminishing libido due to hypercortisolism?
173. What is the possible pathogenesis of male sterility due to hypercortisolism?
174. What is the possible pathogenesis of skin atrophy and bleeding due to primary hypercortisolism?
175. What is the possible pathogenesis of skin depigmentation due to primary hypercortisolism?
176. What is the mechanism of selective obesity due to hypercortisolism?
177. What is the mechanism of edema due to hypercortisolism?
178. What is the mechanism of hypertension due to hypercortisolism?
179. What hematological disorders are specific due to hypercortisolism?
180. What is pathogenesis of hyperglycemia due to hypercortisolism?
181. What ispathogenesis of hypernatraemia and hypokalaemia due to hypercortisolism?
182. What is mechanism of hypercalcemia due to hypercortisolism?
183. In what form of hypercortisolism the concentration of aldosterone is normal cortisol increases , ACTH - decreases?
184. What is the possible etiology of primary hypercortisolism?
185. What is the pathogenesis of atrophy of healthy adrenal glands contralateral to tumor of the adrenal glands?
186. What are principles of primary hypercortisolism therapy?
187. What postoperative complication is possible after unilateral adenoma removal of adrenal glands?
188. What is the rehabilitation method after removal of the hormone-producing tumor of adrenal glands?
189. What is hyperaldosteronism?
190. What is the cause of primary hyperaldosteronism?
191. What are the causes of secondary hyperaldosteronism?
192. What is pathogenesis of hyperaldosteronism due to liver failure?
193. What is pathogenesis of hyperaldosteronism due to renin-secreting tumor?
194. What is the pathogenesis of hyperaldosteronism in hypovolemia?
195. What is pathogenesis of secondary hyperaldosteronism in kidney ischemia?
196. What is the pathogenetic link of hyperaldosteronism in essential hypertension?
197. What are clinical manifestations of hyperaldosteronism?
198. How does the concentration of electrolytes change in case of hyperaldosteronism?
199. How does the hydroelectrolytic balance change in hyperaldosteronism?
200. How does the systemic hemocirculation change in case of hyperaldosteronism?
201. What are the hyperglycemic factors that are included in case of hypoglycemia?
202. What are the hypoglycemic factors that are included in case of hypoglycemia?
203. What is the glucostatic mechanism of glucagon?
204. What is the glucostatic mechanism of catecholamines?
205. What is the glucostatic mechanism of glucocorticoids?
206. What is the glucostatic mechanism of insulin?
207. How do the functions of endocrine glands change in lack of insulin?
208. What hormones lead to hyperglycemia in lack of insulin secretion?
209. What are the metabolic effects of glucagon?
210. What are the metabolic effects of catecholamines?
211. What are the metabolic effects of glucocorticoids?
212. What are the consequences of long standing hyperglycemia?
213. What are the consequences of glycosylation of apoproteins from VLDL, LDL and their receptors?
214. What are the consequences of glycosylation of vascular basal membrane collagen?
215. What are the effects and consequences of exaggerated hyperglycemia in diabetes mellitus type I?
216. By what is manifested transport hyperlipidemia in diabetes mellitus type I?
217. What are the consequences of transport hyperlipidemia for the liver in case of diabetes mellitus type I?
218. What are the consequences of increased non-esterified fatty acids in the liver in case of diabetes mellitus type I?
219. What are the effects of accumulation in excess of ketone bodies in case of diabetes mellitus type I?
220. What acid-base imbalance is characteristic for diabetes mellitus type I?
221. How do the ponderal mass index and adipose tissue mass change in case of diabetes mellitus type I?
222. What is the pathogenesis of polyphagia in diabetes mellitus type I?
223. What is the pathogenesis of weight loss in diabetes mellitus type I?
224. How do the metabolic processes change in lack of insulin?
225. What anabolic processes are disturbed in diabetes mellitus type I?
226. What hormones intensify the catabolic processes of carbohydrates in diabetes mellitus type I?
227. What hormones intensify the catabolic processes of proteins in diabetes mellitus type I?
228. What hormones intensify the catabolic processes of lipids in diabetes mellitus type I?
229. What is the pathogenesis of muscular weakness and atrophy of muscles in diabetes mellitus type I?
230. How does the biochemical composition of hepatocytes change in case of diabetes mellitus type I?
231. How does the biochemical composition of the blood change in case if diabetes mellitus type I?
232. What are the consequences of hyperlipidemia with VLDL and LDL in diabetes mellitus type I?
233. What cells are reach in GLUT-4 insulin-dependent receptors?
234. What are the consequences of inactivation of GLUT-4 receptors in diabetes mellitus type I?
235. What is the cause of hepatocyte inability to uptake the glucose in diabetes mellitus type I?
236. What metabolic processes are disturbed in hepatocytes in diabetes mellitus type I?
237. What conditions assure the glucose uptake by the neurons in lack of insulin?
238. What is the cause that adipocytes cannot metabolize glucose in lack of insulin?
239. What metabolic consequences are characteristic for leukocytes in lack of insulin?
240. What is the pathogenesis of hyperglycemia in diabetes mellitus type I?
241. What is the pathogenesis of glycogenolysis in diabetes mellitus type I?
242. What is the cause of inhibition the glucose uptake by the peripheral cells in diabetes mellitus type I?
243. What is the pathogenesis of decreasing the glucose tolerance in diabetes mellitus type I?
244. What is the pathogenesis of erectile dysfunction in diabetes mellitus type I?
245. What is the pathogenesis of coronary insufficiency in diabetes mellitus type I?
246. What is the pathogenesis of visual disturbances in diabetes mellitus type I?
247. What is the cause of predisposition to develop pyogenic infection of patients with diabetes mellitus type I?
248. What is the cause of decreasing microbicidial activity of phagocytes in diabetes mellitus type I?
249. How does the lipidogram change in patients with diabetes mellitus type I?
250. What is the pathogenesis of metabolic acidosis in diabetes mellitus type I?
251. What is the mechanism of glycosuria in lack of insulin?
252. At what value of glycemia the glycosuria is present in patients with diabetes mellitus type I?
253. What is the cause of polyuria in lack of insulin?
254. What is cause of urine hypersthenuria in lack of insulin?
255. What is the mechanism of albuminuria in patients with diabetes mellitus type I?
256. What is the cause of polydipsia in diabetes mellitus type I?
257. What is the pathogenesis of erythrocytosis and hemoconcentration in diabetes mellitus type I?
258. What is the significance of glycosylated hemoglobin concentration in diabetes mellitus type I?
259. What is the pathogenesis of hyperglycemia and glucose intolerance in diabetes mellitus type I?
260. What is the pathogenesis of hyperlipidemia with non-esterified fatty acids in diabetes mellitus type I?
261. What is the pathogenesis of hyperlipidemia with VLDL and LDL in diabetes mellitus type I?
262. What is the pathogenesis of hypercholesterolemia in diabetes mellitus type I?
263. What is the pathogenesis of exhaustion the alkaline reserves in the blood in diabetes mellitus type I?
264. What investigations are informative to confirm the diagnosis “Diabetes Mellitus type I”?
265. What are the distinctive signs for diabetes mellitus type I?
266. What are the distinctive signs for insulin resistance?
267. What are the consequences of thyroliberin absence?
268. What are the consequences of thyrotropin absence?
269. What are the consequences of radioactive iodine administration for thyroid gland?
270. What is the main pathogenetic link for tertiary hypothyroidism?
271. What is the main pathogenetic link for secondary hypothyroidism?
272. What is the main pathogenetic link for primary hypothyroidism?
273. What are the clinical manifestations of tertiary hypothyroidism?
274. What are the clinical manifestations of secondary hypothyroidism?
275. What are the clinical manifestations of primary hypothyroidism?
276. What are the pathogenetic principles of therapy for tertiary hypothyroidism?
277. What are the pathogenetic principles of therapy for secondary hypothyroidism?
278. What are the pathogenetic principles of therapy for primary hypothyroidism?
279. What is the mechanism of iodine incorporation into the thyroid gland from the blood?
280. What processes in thyroid gland are decreased in case of thyrotropin absence?
281. What are the causes of tertiary hypothyroidism?
282. What are the causes of secondary hypothyroidism?
283. What are the causes of primary hypothyroidism?
284. What is the pathogenesis of primary hypothyroidism caused by iodine deficiency?
285. What is the pathogenesis of hypothyroidism caused by action of antibodies against thyroglobulin?
286. What is the pathogenesis of hypothyroidism caused by action of antibodies against thyropexidase I?
287. What hormonal changes are characteristic for primary hypothyroidism?
288. What hormonal changes are characteristic for secondary hypothyroidism?
289. What hormonal changes are characteristic for tertiary hypothyroidism?
290. What pathogenetic factors are characteristic for myxedema coma?
291. What is the pathogenesis of hypothermia in hypothyroidism?
292. What is pathogenesis of bradycardia in hypothyroidism?
293. What is the pathogenesis of arterial hypotension in hypothyroidism?
294. What is the pathogenesis of persistent hypoglycemia in hypothyroidism?
295. What is the pathogenesis of persistent hyperlipidemia in hypothyroidism?
296. What are the therapeutic principles for autoimmune primary hypothyroidism?
297. What are the consequences of thyrotropin excess?
298. What are the effects of thyrotropin excess?
299. What is the main pathogenetic link of tertiary hyperthyroidism?
300. What is the main pathogenetic link of secondary hyperthyroidism?
301. What is the main pathogenetic link of primary hyperthyroidism?
302. What clinical manifestations are characteristic for tertiary hyperthyroidism?
303. What clinical manifestations are characteristic for secondary hyperthyroidism?
304. What clinical manifestations are characteristic for primary hyperthyroidism?
305. What are the principles of pathogenetic therapy for tertiary hyperthyroidism?
306. What are the principles of pathogenetic therapy for secondary hyperthyroidism?
307. What are the principles of pathogenetic therapy for primary hyperthyroidism?
308. What is the significance value of radioactive iodine uptake by the thyroid gland?
309. What factors influence the velocity of iodine uptake by the thyroid gland?
310. What is the significance of intense iodine uptake “in foci” by the thyroid gland?
311. What is the mechanism of intense iodine uptake by the thyroid tumor in lack of thyrotropin?
312. What is the significance of iodine deficiency in health parenchyma adjacent to the thyroid tumor?
313. What mechanism is responsible for iodine uptake by the malignized thyrocytes (thyroid cancer)?
314. Why the adjacent parenchyma to the tumor doesn’t uptake iodine?
315. What type of allergic reaction is characteristic for Graves’s disease?
316. What disorder is characterized by the abnormal uptake of iodine only in one delimitated zone?
317. What is the pathogenesis of autoimmune hypothyroidism Hashimoto?
318. What is the pathogenesis of Grave’s disease?
319. What somatic changes are characteristic for animal treated previously with L-thyroxine?
320. What metabolic changes are characteristic for the animal treated previously with L-thyroxin?
321. How does the basal metabolic rate change in animal with hyperthyroidism?
322. What compensatory metabolic reactions serve for the adaptation of animal to exogenous hypoxia?
323. What metabolic disturbances decreased the resistance of animal with hyperthyroidism in hypoxia?
324. What is the pathogenesis of weight loss in hyperthyroidism?
325. What is the pathogenesis of increased appetite in hyperthyroidism?
326. What is the pathogenesis of muscular asthenia in hyperthyroidism?
327. What are the consequences of hyperlipidemia in hyperthyroidism?
328. What is the pathogenesis of hyperthermia in hyperthyroidism?
329. What is the pathogenesis of tachycardia in hyperthyroidism?
330. What is the pathogenesis of decreased arterial diastolic blood pressure in hyperthyroidism?
331. What is the pathogenesis of excessive oxigenum consumption in hyperthyroidism?
332. What investigations differentiate tertiary, secondary and primary hyperthyroidism?
333. What are the causes of functional disturbances of adenohypophysis?
334. What are the triggers of antidiuretic hormone hypersecretion?
335. What is the mechanism of antidiuretic hormone hyposecretion in trauma of pituitary stock?
336. What are the manifestations of ADH hypersecretion?
337. What are the causes of prolactin hypersecretion?
338. What manifestations are characteristic for prolactin hypersecretion in women?
339. What manifestations are characteristic for prolactin hypersecretion in men?
340. What are the causes of growth hormone hypersecretion?
341. What are the causes of growth hormone hyposecretion?
342. How does the carbohydrate metabolism change in hypersecretion of growth hormone?
343. How does the protein metabolism change in hypersecretion of growth hormone?
344. What are the somatic manifestations in hypersecretion of growth hormone in children?
345. What are the somatic manifestations in hypersecretion of growth hormone in adults?
346. What are the somatic manifestations in hyposecretion of growth hormone in children?
347. What are the somatic manifestations in hyposecretion of growth hormone in adults?

**Questions V totalisation**

Pathophysiology of the blood

1. Hypovolemia. Definition, classification, compensatory reactions.

2. Normocythemic hypovolemia. Causes, pathogeny, manifestations.

3. Policythemic hypovolemia. Causes, pathogeny, manifestations.

4. Oligocythemic hypovolemia. Causes, pathogeny, manifestations.

5. Policythemic hypervolemia. Causes, pathogeny, manifestations.

6. Oligocythemic hypervolemia. Causes, pathogeny, manifestations.

7. Normocythemic hypervolemia. Causes, pathogeny, manifestations.

8. What are the parameters of normocythemic normovolemia?

9. What are the parameters of oligocythemic hypervolemia?

10. What are the parameters of policythemic hypervolemia?

11. What are the parameters of normocythemic hypovolemia?

12. What are the parameters of oligocythemic hypovolemia?

13. What are the parameters of policythemic hypovolemia?

14. Policythemia. Definition, causes, classification.

15. Absolute primary erythrocytosis. Etiology, pathogeny, manifestations?

16. Absolute secondary erythrocytosis. Etiology, pathogeny, manifestations?

17. Relative erythrocytosis. Etiology, pathogeny, manifestations?

18. Characteristics of peripheral blood picture in absolute primary erytrocytosis/absolute secondary erythrocytosis/ relative erythrocytosis.

21. Anemia. Definition. Pathogenetic classification. consequences?

22. What are the signs of local tissular hypoxia triggered by anemia?

23. What are the pathogenetic mechanisms which explain the simptoms in anemia?

24. What laboratory parameters describe the type of anemia?

25. What laboratory parameters describe the severity of anemia?

26. What laboratory parameters are changed in anemia and what are their refrence ranges?

27. What does represent reticulocytes?

28. What are the normal rahnegs of reticulocytes in the peripheral blood?

29. What is the clinical role of evaluation of reticulocyte count in the peripheral blood?

30. What is the iron homeostasis in the human body?

31. What are the sources which ensure the iron homeostasis in the human body?

32. What is the iron metabolism in human body?

33. What are the causes of iron deficiency anemia?

34. What are the causes of absolute iron deficiency in lack of dietary supply?

35. What are the causes of relative dietary iron deficiency?

36. What are the mechanisms of iron absorbtion?

37. What are the enzymes which are involved in iron transport through the body?

38. What are the causes of iron absorbtion disturbances?

39. What are the causes of iron transport disturbances?

40. What are the mechanisms of iron storage in the human body?

41. What are the iron reserves in the body?

42. What iron reserves can compensate its depletion in the human body?

43. What biochemical parameters are the markers of iron deficiency in the body?

44. What laboratory parameters confirm the diagnosis of iron deficiency anemia?

45. What are the clinical manifestations in iron deficiency anemia?

46. What chronic disorders are associated with anemic syndrome?

47. What is the pathogeny of anemic syndrome associated with chronic diseases?

48. what is the picture of peripheral blood in iron deficiency anemia?

49. What are the compensatory reactions in acute bleeding?

50. What are the manifestations of acute bleeding?

51. What are the hematological signs of acute post-bleeding anemia?

52. What are the general characteristics of hemolytic anemias?

53. What are the intrinse/extrinsic causes of hemolytic anemias?

55. What are the causes of intravascular/extravascular hemolytic anemias?

57. What is the pathogeny of intravascular/extravascular hemolysis?

58. What are the characteristic manifestations in intravascular/extravascular hemolysis?

60. What is the pathogeny of immune hemolytic anemias?

61. What are the hematological signs of autoimmune hemolytic anemias?

62. What are the hematological signs of intravascular hemolysis?

63. What are the pathogenetic types of anemia developed in the result of diminished erytrocytopoiesis?

64. What are the causes of anemic syndrome triggered by reduced erythrocytopoiesis?

66. What are the biological roles of B12 vitamin?

67. What are the morphogenic/metabolic effects of B12 vitamin?

69. What changes are triggered by B12 vitamin deficiency?

70. What are the metabolic changes caused by B12 vitamin deficiency?

71. What are the causes of B12 vitamin deficiency?

72. What are the causes of megaloblastic anemia?

73. What is the pathogeny of pernicious anemia?

74. What types of antibodies are involved in pathogenesis of pernicious anemia?

75. What are the manifestations in pernicious anemia?

76. What are the manifestations in the peripheral blood picture in megaloblastic anemia?

77. What are the characteristics of megaloblastic anemia?

78. What is the pathogeny of anemic syndrome/neurologic syndrome/ gastro-intestinal syndrome in pernicious anemia?

81. What are the manifestations of gastro-intestinal syndrome in pernicious anemia?

82. What are the changes in the peripheral blood picture in B12 vitamin deficiency anemia?

83. In what anemia there can be attested megaloblastic erythrocytopoiesis?

84. What are the signs of disturbances in proliferation and maturation of erythroblast series?

85. What are the changes in the peripheral blood in hyperpropliferation of red bone marrow?

86. What are the signs of erythrocyte hypochromia?

87. What are the signs of erythrocyte hyperchromia?

88. What are the signs of erythrocyte macrocytosis?

89. What are the general features of aplastic anemia?

90. What does represent Fanconi anemia?

91. What are the common causes of secondary aplastic anemia?

92. What are the pathogenetic mechanisms of secondary aplastic anemia?

93. What is the picture of peripheral blood in aplastic anemia?

94. What stages of leucocytopoiesis are disturbed in the bone marrow in case of primary disorders of hematopoietic organs?

95. What stages of leucocytopoiesis are disturbed in the bone marrow in case of secondary disorders of hematopoietic organs?

 96. What disorders develop in primary affection of the bone marrow?

97. From what cells does develop hematoblastosis?

98. What is the definition and characteristics of hematoblastosis?

99. What are the characteristics of leukemia?

100. What is the characteristic of lymphomas?

101. What are the types of acute/chronic leukemias?

102. What is the etiology of hematoblastosis?

104. What are the general concepts in development of hematoblastosis?

105. What are the pathogenetic principles in hematoblastosis?

106. What are the general features in acute leukemias/chronic leukemias?

107. What is the general pathogenesis on acute leukemia/chronic leukemia?

110. What are the features of chronic granulocyte leukemia?

111. What is the general characteristic of chronic lymphocyte leukemia?

112. What is the pathogeny of chronic lymphocyte leukemia?

113. What are the signs of absolute leukocytosis/relative leukocytosis?

115. What leukocytosis are considered physiological?

116. What are the causes of neutrophilia/eosinophilia/lymphocytosis/monocytosis?

117. What does represent ‘left shift’ neutrophilia?

118. What are the causes of hyperregenerative neutrofilia?

122. What does represent agranulocytosis?

123. In what pathological condition can be found agranulocytosis?

124. What are the hematological signs in leukemic myeloid leucosis?

125. What are the hematological signs in subleukemic myeloid leucosis?

126. What are the hematological signs in leukocytopenic myeloid leucosis?

127. What are the hematological signs in aleukemic myeloid leucosis?

Pathophysiology of cardiovascular system

1. How does the systolic volume and cardiac output change in heart failure?
2. How does the arterial and venous pressure change in heart failure?
3. How does the systolic volume and end-systolic volume change in heart failure?
4. In what disorders can develop dysmetabolic heart failure?
5. In what pathologic conditions can be attested overload of the heart with resistence/volume?
6. What is a possible cause of right heart failure/left heart failure?
7. What are characteristic manifestations for left heart failure/right heart failure?
8. What are immediate/late cardiac compensatory reactions in heart failure?
9. What are immediate/late extracardiac compensatory reactions in heart failure?
10. How is performed homeometric/heterometric hyperfunction of the myocardium?
11. For what cardiac defect there is characteristic homeometric hyperfunction/heterometric hyperfunction?
12. How does the structure of hypertrophic myocardium change?
13. What are the mechanism of functional exhaustion and cardiosclerosis of hypertrophied myocardium?
14. What are the causes of relative hypoxia in hypertrophied myocardium?
15. What are the causes of hypervolemia in chronic heart failure?
16. What are the consequences of venous stasis in circulatory insufficiency?
17. What are the consequences of venous stasis in the liver?
18. What is one of the consequences of portal hypertension in heart failure?
19. What are the pathogenic factors of cardiac edemas?
20. What is the role of kidney hypoperfusion in pathogenesis of edemas?
21. What is the pathogenesis of secondary hyperaldosteronism in circulatory failure?
22. How does the cardiac metabolism change in initial phase of hypertrophy?
23. By what is manifested disturbance of intracardiac conductibility?
24. For what diseases is characteristic sinus bradycardia?
25. What are the causes of bradycardia?
26. For what type of extrasystoles is characteristic complete compensatory pause?
27. By what is manifested disturbance of cardiac excitability?
28. By what is manifested disturbance of cardiac automatism?
29. What are the pathogenic factors of renal arterial hypertension?
30. What heart compartments undergo hyperfunction in hypertensive disease?
31. In what pathologic conditions is developed secondary arterial hypertension?

Pathophysiology of respiratory system

 1. What process characterizes external respiration?

2. What are the causes of respiratory center disturbance?

3. Which factors influence the nervous afferent structures and change activity of the respiratory center?

4. Which factors influence the nervous efferent structures and change the pulmonary ventilation?

5. What are the causes of diaphragm paralysis?

6. What pathological processes disturb neuromuscular juctions of the diaphragm?

7. What are the manifestations of diaphragm disorders and intercostal muscules disorders?

8. What is characteristic for unilateral and bilateral paralysis of the diaphragm?

9. What does respiratory reflex Kratschmer represent, what are the trigger factors, what is the mechanism?

10. What does the reflex Hering-Breuer represent, what are the trigger factors, what is the mechanism?

11. What does the hypercapnia represent?

12. What does the hypoxemia represent?

13. What does hyperpnea represent?

14. What does polypnea represent?

15. What does bradypnea represent?

16. What does hyperventelation mean?

17. What ventilation parameters are changed under condition of hyperventilation?

18 What changes of alveolar air are characteristic for hyperventilation?

19. What changes of gaseouse content in the blood are characteristic for hyperventilation?

20. What acid-base imbalance is charcteristic for hyperventilation?

21. What does hypoventilation mean?

22. What ventilation parameters are changed under condition of hypoventilation?

23. What changes of alveolar air are characteristic for hypoventilation?

24. What changes of gaseouse content in the blood are characteristic for hypoventilation?

25. What acid-base imbalance is charcteristic for hypoventilation?

26. What does pulmonary restriction mean?

27. What are the causes of extraparenchymal pulmonary restriction?

28. What are the causes of intraparenchymal pulmonary restriction?

29. What processes or disorders lead to shallow and accelearted breathing?

30 What are the mechanisms of shallow and accelerated breathing?

31 What is the pathogenetic mechanism of hypoxemia in case of restrictive lung disorders?.

32. What changes of pneumogramm are characteristic for reastrictive disorders?

33. How the intratoracic pressure and venous return to the heart are changed in shallow breathing?

34. What does pulmonary emphysema represent?

35. What is the pathogenesis of pulmonary emphysema?

36. What characteristic for pulmonary emphysema?

37. What is the main pathogenetic link of pulmonary emphysema?

38. What changes in the pulmonary parenchyma are characteristic for the emphysema?

39. What types of pulmonary emphysema exist?

40 What is the incidence of different types of pulmonary emphysema?

41. What is the mechanism of abnormal expiration in emphysema?

42. What is the mechanism of shortening expiration in emphysema?

43. What is the mechanism of increasing the volume of thoracic cavity in emphysema?

44. What does the pleural effusion mean?

45. What are the causeses of transudate in pleural cavity?

46. What are the causes of exudate in pleural cavity?

47. What does the pneumotorax mean?

48. What is the main pathogenetic link of pneumotorax?

49. What are the types of pneumotorax?

50. What does the pneumosclerosis mean?

51. Whata are the causes of pneumosclerosis?

52. What are the pathogenetic links of pneumosclerosis?

53. What are the manifestations of pneumosclerosis?

54. What does athelectasis mean?

55. What are the types of pulmonary athelectasis?

56. What is the pathogenesis of athelectasis?

57. What are the causes of nonobstructive athelectasis?

58. What are the pathogenetic links of nonobstructive athelectasis?

59. What are the consequences of athelectasis?

60. What does the pulmonary obstruction mean?

61. Which factors prorovoke obstruction of superior airways?

62. Which factors provoke obstruction of inferior airways?

63. What are the causes of stenotic breathing?

64. What are the causes of expiratory dyspneea?

65. What are the key elements of asthma?

66. What are the pathogenetic factors of asthma?

67. What are the pathophysiological mechanisms of bronchial obstruction?

68. Whata are the main manifestations of asthma?

69. Which indicices are used for determining the bronchial obstruction degree?

70. What are the diagnostic criteria of bronchila asthma (parameters of pneumogram)?

71. What biologic substances have bronchocontrictor effect?

72. What biologic substances have bronchodilator effect?

73. What is the pathogenesis of hypoxemia and hypercapnia in pulmonary congestion?

74. What is the pathogenesis of hypoxemia and hypercapnia in excessive inhalation of anorganic dusts?

75. What is the pathogenesis of hypoxemia and hypercapnia in pneumosclerosis?

76. What is the consequence of pulmonary congestion?

77. To what pathology can lead pulmonary congestion?

78. What is the pathogenesis of cardiac failure caused by pulmonary congestion?

79. What does the pulmonary edema represent?

80. What factors provoke pulmonary edema?

81. What is the pathogenesis of pulmonary edema?

82. What are the local compensatory antiedema mechanisms of the lungs?

83. What biologic active substances increase pulmonary circulation?

84. What biologic active substances reduce pulmonary circulation?

85. What does the acute respiratory distress syndrome mean?

86. What are the main characteristic for the acute respiratory distress syndrome?

87. What factors provoke acute respiratory distress syndrome in adults?

88. What are the pathogenetical mechanisms of ARDS in adults?

89. Which are the stages of ARDS in adults?

90. What are characteristic for the ARDS in adults according to each satge in part?

91. How can be calculated volume of gas deffusuion though the alveolar – capillary membrane? (Fick law)?

92. On what depend the diffusion coificient of the gas in liquid enviroinment of the body (blood)?

93. What physical parameters of alveolar air decrease the gas diffusion through the alveolar – cappilar barrier?

94. What processes decrease the gas diffusion through the alveolar – capillary barrier?

95. What does the oxygenic capacity represent?

96 What factors reduce the oxygenic capacityof the blood?

97. What haemoglobin compounds reduce oxygenic capacity of the blood?

98. What physico-chemichal parameters impede oxygen association to haemoglobin in pulmonary cisculation?

99. What physico-chemichal parameters impede oxygen dissociation of oxyhaemoglobin in systemic cisculation?

100. What physico-chemichal parameters increase oxygen disssociation of oxyhaemoglobin in systemic cisculation?

101. What does the asphyxia mean?

102. What are the stages of asphyxia?

103. How does the pneumogram change in different stages of experimental asphyxia?

**VI Totalisation**

1. Excess of salivation. Causes. Pathogenesis. Manifestations. Consequences.
2. Insufficient salivation. Causes. Pathogenesis. Manifestations. Consequences.
3. Hypersecretion of gastric glands. Causes. Features of gastric and intestinal digestion in gastric hypersecretion and hyperacidity.
4. Genesis of gastric and duodenal ulcer. Causes. Pathogenesis. Aggressive and protective gastric factors.
5. Hypo-secretion of gastric glands. Causes. Pathogenesis. Features of digestion.
6. Impaired intestinal digestion in insufficient secretion of pancreatic juice. Causes. Pathogenesis. Manifestations. Consequences.
7. Intestinal digestion in impaired bile secretion. Acholia. Causes. Pathogenesis. Manifestations. Consequences.
8. Intestinal absorption disorders. Etiology. Pathogenesis. Manifestations. Consequences.
9. Mechanical jaundice. Cholestasis. Cholemia. Etiology. Pathogenesis. Manifestations. Changes in bile pigment metabolism.
10. Parenchymatous jaundice. Etiology. Pathogenesis. Manifestations. Changes in bile pigment metabolism.
11. Hepatic insufficiency. Classification. Pathogenesis. Manifestations. Hepatic coma.
12. Glomerular filtration abnormalities. Causes. Pathogenesis. Manifestations. Consequences.
13. Disorders of renal reabsorption of organic matter (proteins, glucose, amino acids). Causes. Pathogenesis. Consequences.
14. Disorders of renal reabsorption of water and electrolytes (Na). Causes. Pathogenesis. Consequences.
15. Disorders of renal secretion (H +, K +). Causes. Pathogenesis. Manifestations. Consequences.
16. Renal failure. Causes. Pathogenesis. Manifestations. Quantitative pathological disorders of diuresis. The disorders of urine concentration function in kidney. Hypo-and isosthenuria.
17. Pathological changes in the composition of urine (proteinuria, glucosuria, and hematuria). Causes. Pathogenesis.