1. What does etiology study? (1)
2. Causes and conditions of the disease appearance
3. Causes of the disease appearance
4. Conditions of the disease appearance
5. Mechanisms of the disease resolution
6. Mechanisms of the disease evolution
7. What factor could be considered as the endogenous cause of the disease? (5)
8. Changes of blood pH
9. Pathogenic strain of E.coli in the intestine
10. Ascaridae infestation
11. Genetic mutations appeared during the life of the patient
12. Inherited genetic mutations
13. What factors could be considered as the exogenous cause of the disease? (2,3,4)
14. Changes of blood pH
15. Pathogenic of E.coli in the intestine
16. Acaride infestation
17. Genetic mutations appeared during life course of patient
18. Genetic mutations inherited from predecessors
19. What pathological process is provoked by endogenous cause? (5)
20. Metabolic acidosis in hypoxia
21. Septicemia caused by intestinal E.coli dissemination
22. Embolism with ascaridae larvae
23. Tumor caused by genetic mutations
24. Inherited haemophilia from mother
25. What pathological process is provoked by endogenous cause? (4)
26. Metabolic acidosis in diabetes mellitus type I
27. Septicemia caused by intestinal E.coli dissemination
28. Embolism with ascaride larvae
29. Milk intolerance caused by deficiency of lactase gene
30. Tumor caused by genetic mutations

6, What exogenous conditions could lead to the disease appearance? (1,4,5)

1. Family conflicts
2. Pathogenic biologic field generated by other persons
3. Telepathic influences of by other persons
4. Changes of atmospheric pressure
5. Ultraviolet irradiation

7, What exogenous conditions could lead to the disease appearance? (1,4,5)

1. Starvation
2. Pathogen biologic field generated by other persons
3. Telepathic influences sent by other persons
4. Physical overload
5. Expose to low temperatures

8, What effects exert favorable conditions for the body? (2,3)

1. Enhance the action of the cause and favors the disease appearance
2. Enhance the body’s resistance to the action of harmful agents
3. Reduces the action of the cause and delay the disease appearance
4. Reduces the body’s reactivity to the action of harmful agents
5. Annihilates the cause of the disease

9, What effects exert favorable conditions for the body? (1,4)

1. Consolidate the physiologic reaction of the body
2. Change the superior nervous activity
3. Purifies aura and bio-field of the body
4. Change the person’s phenotype
5. Provoke the favorable mutations in the genome

10, What effects exert favorable conditions for the body? (1,3)

1. Improve the metabolism
2. Change the type of superior nervous activity
3. Contribute to the accumulation of nutritive substances in the body
4. Change the person’s phenotype
5. Change the person’s genotype

11, What effects exert unfavorable conditions for the body? (1,3,4)

1. Enhance the action of the cause and favor the disease appearance
2. Enhance the body’s resistance to the action of harmful agents
3. Reduce the body’s resistance to the action of harmful agents
4. Reduce the body’s reactivity to the action of harmful agents
5. Annihilate the cause of the disease

12, What effect exert unfavorable conditions for the body? (1)

1. Decreases physiologic reactions of the body
2. Changes the type of superior nervous activity
3. Pollutes the aura and bio-field of the body
4. Changes the person’s genotype
5. Provokes unfavorable mutations in the genome

13, What effects exert unfavorable conditions for the body? (1,3)

1. Metabolic disturbances
2. Change the activity of nervous system
3. Depletion of nutritive substances
4. Change the person’s genotype
5. Provoke unfavorable mutations in the genome

14, What endogenous conditions could influence the action of harmful agents? (1,2,3)

1. The state of immun system
2. Metabolic disturbances
3. Associated diseases
4. Personal hygiene
5. Working and resting regime

15, What exogenous conditions could influence the action of harmful agents? (1,2)

1. Ecological factors
2. Climatic factors
3. The body’s constitution
4. The state of immune system
5. Metabolic disturbances

16. What exogenous conditions could influence the action of harmful agents? (3,4)

1. The body’s constitution
2. The state of immunity system
3. Microclimate factors
4. The psychological microclimate in family and working groups
5. Personal hygiene

17. What conditions disturb the metabolism and lead to the disease appearance? (1,4)

1. Deficiency of vitamins in the diet
2. Deficiency of carbohydrates in the diet
3. Deficiency of triglycerides in the diet
4. Deficiency of polyunsaturated fatty acids in the diet
5. Deficiency of nonessential aminoacids in the diet

18. What conditions disturb the metabolism and lead to the disease appearance? (1,4)

1. Deficiency of microelements in the diet
2. Deficiency of carbohydrates in the diet
3. Deficiency of triglycerides in the diet
4. Deficiency of essential aminoacids in the diet
5. Deficiency of nonessential aminoacids in the diet

19. What conditions disturb the metabolism and lead to the disease appearance? (4)

1. Deficiency of carbohydrates in the diet
2. Deficiency of triglycerides in the diet
3. Deficiency of nonessential aminoacids in the diet
4. Deficiency of insulin
5. Deficiency of cellulose in the diet

20. What conditions disturb the metabolism and lead to the disease appearance? (4)

1. Deficiency of carbohydrates in the diet
2. Deficiency of triglycerides in the diet
3. Deficiency of nonessential aminoacids in the diet
4. Deprivation of food
5. Deficiency of cellulose in the diet

21. What conditions disturb the metabolism and lead to the disease appearance? (5)

1. Deficiency of carbohydrates in the diet
2. Deficiency of triglycerides in the diet
3. Deficiency of thyroid hormones
4. Deficiency of nonessential aminoacids in the diet
5. Excess of thyroid hormones

22. What conditions disturb the metabolism and lead to the disease appearance? (5)

1. Deficiency of carbohydrates in the diet
2. Deficiency of triglycerides in the diet
3. Deficiency of polyunsaturated fatty acids in the diet
4. Deficiency of catecholamines
5. Excess of catecholamines

23. What does study general pathology? (1)

1. General laws of appearance, evolution and resolution of pathological processes
2. General laws of body’s dying processes
3. General laws of disease resolution
4. General laws of disease origin
5. General laws of recovery processes

24. What is the role of the cause in disease appearance? (2)

1. Determines the disease duration
2. Determines the disease specificity
3. Determines the moment of disease appearance
4. Determines the disease complications
5. Determines the disease prognostic

25. What does determine the specificity of the disease? (1)

1. The cause that provoked disease
2. The age of the person
3. The gender of the person
4. The hereditary predisposition
5. The conditions of disease appearance

26. What does determine the character of the disease? (3,4,5)

1. The cause that has provoked the disease
2. The age of the person
3. The hereditary predisposition
4. The conditions of the disease appearance
5. The reactivity of the body

27. What factors increase the probability of the disease appearance to the cause action ? (3,4,5)

1. The cause that has provoked the disease
2. The hereditary predisposition
3. Unfavorable conditions
4. The state of immune system
5. The state of the central nervous system

28. What factors increase the probability of disease appearance to the action of the cause? (2,4,5)?

1. The cause that provoked disease
2. The old age of the person
3. The gender of the person
4. Unfavorable conditions
5. The state of immun system

29. What is the role of conditions in disease appearance? (4)

1. Determine the disease duration
2. Determine the specificity of the disease
3. Determine the possibility of disease appearance
4. Prevent or accelerate the disease appearance
5. Determine the specific symptoms of the disease

30. What is the role of conditions in the disease appearance? (4)

1. Determines the disease duration
2. Determines the specificity of the disease
3. Determines the possibility of disease appearance
4. Favor or worses the disease appearance
5. Determines the specific symptoms of the disease

31. What is the role of conditions in the disease appearance? (4)

1. Determine the disease duration
2. Determine the specificity of the disease
3. Determine the possibility of disease appearance
4. Favor or retain the recovery process
5. Determine the specific symptoms of the disease

32. In what disorders the cause has trigger role and further disease develops without action of it? (3,4)

1. Chronic infection disease
2. Acute intoxication
3. Hereditary disorders
4. Mechanical trauma
5. Acute infection disease

33. In what disorders the cause has trigger role and further disease develops without action of it? (3,4)

1. Chronic infection disease
2. Acute intoxication
3. Hereditary disorders
4. Thermal trauma
5. Acute infection disease

34. In what disorders the presence of the cause is necessary for all period of the disease? (2,3,5)

1. Hereditary disorders
2. Acute intoxication
3. Chronic infection disease
4. Mechanical trauma
5. Acute infection disease

35. In what disorders the presence of the cause is necessary for all period of the disease? (1,2,5)

1. Chronic infection disease
2. Acute intoxication
3. Hereditary disorders
4. Thermal trauma
5. Acute infection disease

36. What are the correlations between local and general injuries in pathogenesis of different disease? (3,4,5)

1. There are diseases with only local injuries for all periods of them
2. There are diseases with only general injuries for all period of them
3. There are diseases with balance between local and general injuries
4. There are diseases which begin with general injuries and then associate local injuries
5. There are diseases which begin with local injuries and then associate general injuries

37. What are the relations between local and general injuries in pathogenesis of different disease? (3,4,5)

1. There are diseases with only local injuries for all period of them
2. There are diseases with only general injuries for all period of them
3. There are diseases with balance between local and general injuries
4. There are disease with predominance of local injuries
5. There are disease with predominance of general injuries

38. What cellular changes could be considered as injury? (5)

1. Myocardial hypertrophy at functional cell overload
2. Decreased number of myocyte’s ribosomes in hypokinesis
3. Accumulation of lipids in adipocytes
4. Apoptosis of the tumor cells
5. Disintegration of cytoplasmatic membrane in hypoosmolar environment

39. What cellular changes could be considered as injury? (4)

1. Enhancement of anaerobic glycolisis in hypoxia
2. Synthesis of heat shock proteins in hyperthermia
3. Enhancement of protein synthesis in hyperfunctional myocytes
4. Inhibition of oxidative enzymes in hypoxia
5. Enhancement of antioxidative processes in oxidative stress

40. What cellular changes could be considered as injury? (1,4,5)

1. Diminishing mitotic processes in hypoxia
2. Diminishing oxidative processes in case of increased cellular ATP
3. Irrecoverable defects into cytoplasmatic membrane
4. Excessive accumulation of lipids into hepatocytes
5. Cessation of membrane Na/K pumps in hypoxia

41. What is pathogenetic factor in pathological processes? (2,3)

1. The field where acts disease cause
2. The totality of events which develop under the action of primary cause
3. The primary effect provoked direct by action of first cause
4. The cause that provoked disease
5. The conditions which favored action of disease cause

42. What is the pathogenetic chain in pathological processes? (5)

1. The totality of events which develop under the action of primary cause
2. The totality of injuries that are encountered during the disease
3. The totality of body reactions that are encountered during the disease
4. The totality of body injuries and reactions that are encountered during the disease
5. The totality of body injuries and reactions that are linked by cause - effect relations

43. What is the main link of pathogenesis? (1,5)

1. Pathogenetic factor which ones is removed the pathogenetic chain disappears
2. The cause that has provoked the disease
3. Injuries provoked by action of the disease cause
4. The last pathogenetic factor of the disease
5. Pathogenetic factor by which depends the disease development

44. What is the role of main link in the disease pathogenesis? (4,5)

1. Determines the whole evolution of the disease
2. Triggers all physiological reactions of the body
3. Triggers all injuries during the disease
4. Maintains pathogenetic chain of the disease
5. It is the target point for pathogenetic therapy of the disease

45. What is the aim of etiotropic therapy of the disease? (1)

1. Removal of the disease cause
2. Removal of primary injuries
3. Removal of main pathogenetic link
4. Removal of vicious cycle
5. Removal of conditions in what the disease appeares

46. What is the aim of etiotropic therapy of the disease? (2)

1. Removing of primary injuries
2. Decreasing of pathogenic action of etiological factor
3. Removing of main pathogenetic link
4. Removing of vicious cycle
5. Removing of conditions in what disease appeared

47. What is pathogenetic therapy? (1,5)

1. Breaking the vicious cycle
2. Removing of disease cause from the body
3. Removing of primary injuries
4. Decreasing of pathogenic action of etiological factor
5. Removing of main pathogenetic link

48. What is pathogenetic therapy? (2,5)

1. Removal of the disease cause from the body
2. Removal of all pathogenetic factors
3. Removal of primary injuries
4. Decreased action of pathogenic etiological factor
5. Removal of main pathogenetic link

49. What is symptomatic therapy? (5)

1. Removing of primary injuries clinically manifested
2. Decreasing of pathogenic action of etiological factor
3. Removing of main pathogenetic link
4. Removing of vicious cycle
5. Removing of life threatening disturbances

50. What is symptomatic therapy? (1)

1. Correction of life threatening states
2. Removing of primary injuries manifested clinically
3. Decreasing of pathogenic action of etiological factor
4. Correction of vicious cycle
5. Correction of main pathogenetic link

51. How is performed the specific prophylaxis of disease? (4)

1. By administration of vitamins and oligoelements
2. By tempering the body
3. By creation of favorable conditions for the person
4. By active immunization
5. By highlighting individual characters of the patient

52. How is performed the specific prophylaxis of the disease? (3)

1. By administration of vitamins and oligoelements
2. By tempering the body
3. By passive immunization
4. By creation of favorable conditions for the person
5. By highlighting individual characters of the patient

53. How is performed the nonspecific prophylaxis of the disease? (2,4)

1. By active or passive immunization
2. By administration of vitamins and oligoelements
3. By prophylactic administration of antibiotics
4. By tempering the body
5. By avoiding contact with infected persons

54. How is performed the nonspecific prophylaxis of disease? (2,4)

1. By active or passive immunization
2. By healthy activity and rest regime
3. By prophylactic administration of antibiotics
4. By follow a healthy diet
5. By avoiding contact with infected persons

55. What is the characteristic for body’s physiologic reaction? (1)

1. Corresponds to the excitant’s specificity
2. Does not correspond to excitant’s specificity
3. Leads to persistent dyshomeostasis
4. It is inferior to the excitant’s intensity
5. Exceeds the excitant’s intensity

56. What is the characteristic for body’s physiologic reaction? (2)

1. Does not correspond to excitant’s specificity
2. Corresponds quantitatively to intensity of excitant
3. Leads to persistent dyshomeostasis
4. Does not correspond quantitatively to intensity of excitant
5. Exceeds the excitant’s intensity

57. What is the characteristic for body’s physiologic reaction? (3)

1. Does not correspond to excitant’s specificity
2. Leads to persistent dyshomeostasis
3. It has homeostatic character
4. Does not correspond quantitatively to intensity of excitant
5. Exceeds the excitant’s intensity

58.. What is the characteristic for body’s pathologic reaction? (1,5)

1. It is inferior to the excitant’s intensity
2. Leads to restoration of body’s homeostasis
3. It is specific only for one excitant
4. Corresponds to intensity of excitant
5. Exceeds the excitant’s intensity
6. . What are features of body’s pathologic reaction? (2,5)
7. Leads to restoration of body’s homeostasis
8. Exceeds the excitant’s intensity
9. It is specific only for one excitant
10. Corresponds to intensity of excitant
11. It is inferior to the excitant’s intensity
12. What is the characteristic for body’s pathologic reaction? (3)
13. Leads to restoration of body’s homeostasis
14. It is specific only for one excitant
15. Leads to dyshomeostasis
16. Corresponds to the excitant’s intensity
17. Corresponds to the excitant’s specificity
18. What physiologic reaction could be considered as adaptive? (1)
19. Increased erythropoiesis in a health person at an altitude of 3000m
20. Increased erythropoiesis in a person with heart vice
21. Increased erythropoiesis in a person with tumor of erythroblast series
22. Increased leukocytopoesis in cocci infection
23. Increased leukocytopoesis in a person with tumor of myeloblast series
24. What physiologic reaction could be considered as adaptive? (3)
25. Increased erythropoiesis in a person with heart vice
26. Increased erythropoiesis in a person with tumor of erythroblast series
27. Increased erythropoiesis in a perform athlete
28. Increased leukocytopoesis in cocci infection
29. Increased leukocytopoesis in a person with tumor of myeloblast series
30. 61. What physiologic reaction could be considered as compensatory? (2)
31. Increased erythropoiesis in a health person at an altitude of 3000m
32. Increased erythropoiesis in a person with heart vice
33. Increased erythropoiesis in a person with tumor of erythroblast lineage
34. Increased leukocytopoesis in cocci infection
35. Increased leukocytopoesis in a person with tumor of myeloblast lineage
36. What physiologic reaction could be considered as compensatory? (3)
37. Increased erythropoiesis in a health person at an altitude of 3000m
38. Increased erythropoiesis in a person with tumor of erythroblast series
39. Increased erythropoiesis in a person with lung resection
40. Increased leukocytopoesis in cocci infection
41. Increased leukocytopoesis in a person with tumor of myeloblast series
42. What physiologic reaction could be considered as protective? (4)
43. Increased erythropoiesis in a health person at an altitude of 3000m
44. Increased erythropoiesis in a person with heart vice
45. Increased erythropoiesis in a person with tumor of erythroblast series
46. Increased leukocytopoesis in cocci infection
47. Increased leukocytopoesis in a person with tumor of myeloblast series
48. What physiologic reaction could be considered as reparative? (3)
49. Increased erythropoiesis in a health person at an altitude of 3000m
50. Increased erythropoiesis in a person with heart vice
51. Increased erythropoiesis in convalescence period of actinic disease
52. Increased leukocytopoesis in cocci infection
53. Increased leukocytopoesis in a person with tumor of myeloblast lineage
54. What physiologic reaction could be considered as reparative? (3)
55. Increased erythropoiesis in a health person at an altitude of 3000m
56. Increased erythropoiesis in a person with heart vice
57. Increased erythropoiesis in blood donors
58. Increased leukocytopoesis in cocci infection
59. Increased leukocytopoesis in a person with tumor of myeloblast series
60. What is adaptive reaction? (1)
61. Reaction that is orientated to change the body’s structure and function according to new life conditions
62. Reaction that is orientated to remove harmful factor from the body
63. Reaction that is orientated to maintaine the function of damaged organ by hyperfunction of other synergist one
64. Reaction that is orientated to the recovery of structural defect
65. Reaction that is orientated to change the genotype according to life conditions
66. What is compensatory reaction? (3)
67. Reaction that is orientated to change the body’s structure and function according to new life conditions
68. Reaction that is orientated to remove harmful factor from the body
69. Reaction that is orientated to maintain the function of damaged organ by hyperfunction of other synergist one
70. Reaction that is orientated to recovery the structural defect
71. Reaction that is orientated to change the genotype according to life conditions
72. What is protective reaction? (2)
73. Reaction that is orientated to change the body’s structure and function according to new life conditions
74. Reaction that is orientated to remove the harmful factor from the body
75. Reaction that is orientated to maintaine the function of damaged organ by hyperfunction of other synergist one
76. Reaction that is orientated to the recover the structural defect
77. Reaction that is orientated to change the genotype according to life conditions
78. What is reparative reaction? (4)
79. Reaction that is orientated to change the body’s structure and function according to new life conditions
80. Reaction that is orientated to remove the harmful factor from the body
81. Reaction that is orientated to maintaine the function of damaged organ by hyperfunction of other synergist one
82. Reaction that is orientated to the recovery of structural defect
83. Reaction that is orientated to change the genotype according to life conditions
84. What is the first period of disease? (4)
85. Prodromal period
86. Period of complete disease manifestation
87. Resolution period
88. Latent period
89. Exacerbation period
90. What is the second period of the disease? (1)
91. Prodromal period
92. Period of complete disease manifestations
93. Resolution period
94. Latent period
95. Exacerbation period
96. What is the third period of the disease? (2)
97. Prodromal period
98. Period of complete disease manifestations
99. Resolution period
100. Latent period
101. Exacerbation period
102. What is the fourth period of the disease? (2)
103. Latent period
104. Resolution period
105. Prodromal period
106. Period of complete disease manifestation
107. Exacerbation period
108. By what is manifested the latent period of the disease? (3)
109. Absence of specific clinical manifestations
110. Presence of nonspecific clinical manifestations
111. Absence of specific and nonspecific manifestations
112. Presence of specific and nonspecific manifestations
113. Temporary disappearance of disease manifestations
114. By what is manifested prodromal period of the disease? (3,4)
115. Absence of specific and nonspecific manifestations
116. Presence of local manifestations
117. Presence of nonspecific clinical manifestations
118. Absence of specific clinical manifestations
119. Temporary disappearance of disease manifestations
120. By what is manifested period of complete disease manifestations ? (4)
121. Absence of specific and nonspecific manifestations
122. Absence of specific clinical manifestations
123. Presence of nonspecific clinical manifestations
124. Presence of specific and nonspecific manifestations
125. Disappearance of disease manifestations
126. What clinical manifestations can be considered as resolution of the disease? (3)
127. Temporary disappearance of all disease manifestations
128. Disappearance of disease manifestations
129. Death of the body
130. Disappearance of nonspecific manifestations
131. Improving the patient’s conditions
132. What clinical manifestations can be considered as resolution of the disease? (2)
133. Temporary disappearance of all disease manifestations
134. Complete recovery
135. Disappearance of specific manifestations
136. Disappearance of nonspecific manifestations
137. Improving the patient’s condition
138. What clinical manifestations can be considered as resolution of the disease? (2)
139. Temporary disappearance of all disease manifestations
140. Disappearance of specific manifestations
141. Disappearance of nonspecific manifestations
142. Incomplete recovery
143. Improving the patient’s condition
144. Which one is the variant of disease’s resolution? (5)
145. Remission of the disease
146. Relapse of the disease
147. The occurrence of complications
148. The progression of the disease to chronic form
149. The death of the body
150. Which is one of the variants of disease’s resolution? (5)
151. Remission of the disease
152. Relapse of the disease
153. The progression of the disease to chronic form
154. The occurrence of complications
155. Complete recovery
156. Which one is the variant of the disease’s resolution? (2)
157. Remission of the disease
158. The occurrence of pathological condition
159. Relapse of the disease
160. The progression of the disease to chronic form
161. The occurrence of complications
162. Which is one of the variants of disease’s resolution? (5)
163. Remission of the disease
164. Relapse of the disease
165. The progression of the disease to chronic form
166. The occurrence of complications
167. Incomplete recovery
168. What is the pathological process? (3)
169. Primary injuries provoked by action of aetiological factor
170. The totality of primary and secondary injuries caused by action of aetiological factor
171. The totality of physiological and pathological events triggered by aetiological factor
172. The totality of local and general injuries caused by action of aetiological factor
173. The totality of physiological reactions of the body triggered by action of aetiological factor
174. Which are the primary sanogenetic mechanisms? (1)
175. Adaptive, protective and compensatory reactions
176. Protective, compensatory and terminal reactions
177. Adaptive, compensatory and terminal reactions
178. Initial and terminal mechanisms
179. Adaptive, protective, compensatory and terminal reactions
180. Which are secondary sanogenetic mechanisms? (2)
181. Adaptive, protective and compensatory reactions
182. Protective, compensatory and terminal reactions
183. Adaptive, compensatory and terminal reactions
184. Initial and terminal mechanisms
185. Adaptive, protective, compensatory and terminal reactions
186. What does the vicious cycle in pathogenesis represent? (2)
187. Any pathogenetic chain
188. Closed pathogenetic chain
189. The totality of pathological processes
190. The totality of injuries that are linked by relations of cause and effect
191. The totality of physiologic reactions
192. What is characteristic for vicious cycle in pathogenesis? (2,5)
193. It has tendency to relapse
194. It has tendency to progress
195. Provokes complications
196. It is incurable
197. It has tendency to reverberate
198. Cell dystrophies can affect one or several organs. What can be the causes of it? (a,e)
     1. persistent hyperglycemia
     2. acute general hypoxia
     3. body dehydration
     4. body overhydration
     5. chronic general hypoxia
199. What general dyshomeostasis leads to cell dystrophy? (e)
     1. general dehydration
     2. general hyperhydrationc.
     3. persistent hypernatremia
     4. calcium deficiency
     5. persistent chronic general hypoxia
200. Cell metabolic disorders can affect one or several organs. What can be the causes of multiple cell metabolic disorders? (a)
     1. persistent hyperlipidemia with chylomicrons
     2. acute general hypoxia
     3. body dehydration
     4. body overhydration
     5. persistent hyperlipidemia with high density lipoproteins
201. Cell metabolic disorders can affect one or several organs. What can be the causes of multiple cell metabolic disorders? (a)
     1. persistent hypercholesterolemia
     2. acute general hypoxia
     3. body dehydration
     4. excessive protein intake
     5. persistent hyperlipidemia with high density lipoproteins
202. Cell metabolic disorders can affect one or several organs. What can be the causes of multiple cell metabolic disorders? (a)
     1. persistent hypoglycemia
     2. acute general hypoxia
     3. body dehydration
     4. excessive protein intake
     5. persistent hyperlipidemia with high density lipoproteins
203. Cell metabolic disorders can affect one or several organs. What can be the cause of multiple cell metabolic disorders? (a)
     1. galactosemia
     2. acute general hypoxia
     3. body dehydration
     4. excessive protein intake
     5. deficiency of carbohydrates in the diet
204. Cell metabolic disorders can affect one or several organs. What can be the causes of multiple cell metabolic disorders? (a,c)
     1. chronic general hypoxia
     2. acute general hypoxia
     3. persistent hyperlipidemia with chylomicrons
     4. excessive protein intake
     5. persistent hyperlipidemia with HDL
205. Cell metabolic disorders can affect one or several organs. What can be the causes of multiple cell metabolic disorders? (a,c)
     1. hypoglycemia in long-lasting starvation
     2. acute general hypoxia
     3. persistent hyperglycemia
     4. excessive protein intake
     5. persistent hyperlipidemia with HDL
206. What is the cause of cell dystrophy? (b)
     1. acute general hypoxia
     2. vitamin deficiency
     3. general dehydration
     4. protein excess in the diet
     5. excessive protein in the diet

100. Cell metabolic disorders can affect one or several organs. What can be the causes of multiple cell metabolic disorders? (a,c)

* 1. starvation
  2. acute general hypoxia
  3. persistent hyperglycemia
  4. excessive protein intake
  5. persistent hyperlipidemia with HDL

1. Cell metabolic disorders can affect one or several organs. What can be the causes of multiple cell metabolic disorders? (c,e)
   1. hypersecretion of insulin
   2. deficiency of glucocorticoids
   3. chronic intoxications
   4. excessive protein intake
   5. persistent hyperlipidemia with VLDL
2. Cell metabolic disorders can affect one or several organs. What can be the causes of multiple cell metabolic disorders? (a,c)
   1. vitamin deficiency
   2. acute general hypoxia
   3. chronic intoxications
   4. excessive protein intake
   5. insulin excess
3. Cell metabolic disorders can affect one or several organs. What can be the causes of multiple cell metabolic disorders? (a,c)
   1. inanition
   2. acute general hypoxia
   3. persistent hyperlipidemia with VLDL
   4. excessive protein intake
   5. persistent hyperlipidemia with HDL
4. What pathological cellular process can trigger cell dystrophy? (a)
   1. inhibition of Krebs cycle
   2. cell apoptosis
   3. cell necrosis
   4. cell hypertrophy
   5. enhanced Krebs cycle
5. What pathological cellular process can trigger cell dystrophy? (a)
   1. ATP depletion
   2. inhibition of anaerobic glycolysis
   3. cell necrosis
   4. sclerosis
   5. increased intracellular pH
6. What pathological cellular process can trigger cell dystrophy? (a)
   1. accumulation of calcium ions within hyaloplasm
   2. inhibition of anaerobic glycolysis
   3. deficiency of potassium in the cell
   4. accumulation of sodium ions within hyaloplasm
   5. increased intracellular pH
7. What pathological cellular processes can trigger cell dystrophy? (a,b)
   1. accumulation of free fatty acids within hyaloplasm
   2. intracellular acidosis
   3. accumulation of glucose within hyaloplasm
   4. accumulation of sodium ions within hyaloplasm
   5. intracellular alkalosis
8. What pathological cellular processes can trigger cell dystrophy? (a,b)
   1. cell enzymatic defects
   2. intracellular acidosis
   3. accumulation of glucose within hyaloplasm
   4. accumulation of sodium ions within hyaloplasm
   5. inhibition of anaerobic glycolysis
9. What pathological cellular processes can trigger cell dystrophy? (a,d)
   1. inhibition of Krebs cycle
   2. inhibition of anaerobic glycolysis
   3. activation of Krebs cycle
   4. cell enzymatic defects
   5. intracellular hyperosmolarity
10. What pathological cellular processes can trigger cell dystrophy? (a, d)
    1. inhibition of Krebs cycle
    2. inhibition of anaerobic glycolysis
    3. activation of Krebs cycle
    4. accumulation of calcium ions within cell
    5. accumulation of sodium ions within cells
11. What pathological cellular processes can trigger cell dystrophy? (a, d)
    1. accumulation of free fatty acids within cells
    2. inhibition of anaerobic glycolysis
    3. accumulation of glucose within cells
    4. accumulation of calcium ions within cell
    5. accumulation of sodium ions within cells
12. What pathological cellular processes can trigger cell dystrophy? (a, d)
    1. energy deficiency
    2. inhibition of anaerobic glycolysis
    3. accumulation of glucose within cells
    4. intracellular acidosis
    5. accumulation of sodium ions within cells
13. What represents a specific manifestation of cell dystrophy? (e)
    1. mitochondrial swelling
    2. excessive glucose storages
    3. reduction of endoplasmic reticulum
    4. injuries of cytoplasmatic membrane
    5. excessive glycogen storages
14. What represents a specific manifestation of cell dystrophy? (e)
    1. mitochondrial swelling
    2. excessive glucose storages
    3. reduction of endoplasmic reticulum
    4. injuries of cytoplasmatic membrane
    5. excessive triglyceride storages
15. What represents a specific manifestation of cell dystrophy? (e)
    1. mitochondrial swelling
    2. excessive glucose storages
    3. reduction of endoplasmic reticulum
    4. injuries of cytoplasmatic membrane
    5. excessive deposition of abnormal proteins
16. What represent specific manifestations of cell dystrophy? (d, e)
    1. mitochondrial swelling
    2. excessive deposition of glucose
    3. swelling of endoplasmic reticulum
    4. excessive deposition of triglycerides
    5. excessive deposition of abnormal proteins
17. What is the cause of parenchymatous lipid dystrophy? (a)
    1. excessive intake of carbohydrates
    2. excessive intake of proteins
    3. high level of insulin in the blood
    4. hypersecretion of thyroid hormones
    5. hypersecretion of glucocorticoids
18. What is the cause of parenchymatous lipid dystrophy? (a)
    1. high level of VLDL in the blood
    2. excessive intake of proteins
    3. high level of high density lipoproteins in the blood
    4. deficiency of thyroid hormones
    5. deficiency of glucocorticoids
19. Liver is the parenchymatous organ frequently affected by lipid dystrophy. What is one of the causes of liver steatosis? (a)
    1. inflammation in the liver
    2. excessive proteins in the diet
    3. high level of insulin in the blood
    4. fibrosis in the liver
    5. high level of glucocorticoids in the blood
20. Liver is the parenchymatous organ frequently affected by lipid dystrophy. What is one of the causes of liver steatosis? (a)
    1. chronic hypoxia in the liver tissue
    2. excessive proteins in the diet
    3. acute hypoxia in the liver
    4. liver fibrosis
    5. high level of glucocorticoids in the blood
21. Liver is the parenchymatous organ frequently affected by lipid dystrophy. What is one of the causes of liver steatosis? (a)
    1. venous hyperemia in the liver
    2. excessive proteins in the diet
    3. acute hypoxia in the liver
    4. deficiency of thyroid hormones
    5. deficiency of glucocorticoids
22. Liver is the parenchymatous organ frequently affected by lipid dystrophy. What is one of the causes of liver steatosis? (a)
    1. long-lasting starvation
    2. excessive proteins in the diet
    3. acute hypoxia in the liver
    4. deficiency of thyroid hormones
    5. high level of proteins in the blood
23. Liver is the parenchymatous organ frequently affected by lipid dystrophy. What are the causes of liver steatosis? (a,d)
    1. insulin deficiency
    2. excessive proteins in the diet
    3. acute hypoxia in the liver
    4. deficient protein intake
    5. hypersecretion of insulin
24. Liver is the parenchymatous organ frequently affected by lipid dystrophy. What are the causes of liver steatosis? (a,d)
    1. chronic hypoxia in the liver
    2. excessive proteins in the diet
    3. acute hypoxia in the liver
    4. protein malnutrition
    5. deficient carbohydrates in the diet
25. What is the cause of parenchymatous lipid dystrophy? (c)
    1. hypersecretion of insulin
    2. inability of the cell to synthetize glycogen
    3. persistent hyperlipidemia with chylomicrons
    4. inability of the cell to transform lipids into glycogen
    5. persistent hyperlipidemia with high density lipoproteins
26. What are the causes of parenchymatous lipid dystrophy? (c, e)
    1. hypersecretion of insulin
    2. inability of the cell to synthetize glycogen
    3. persistent hyperlipidemia with chylomicrons
    4. inability of the cell to transform lipids into glycogen
    5. excessive carbohydrates intake
27. What are the causes of parenchymatous lipid dystrophy? (c, e)
    1. hypersecretion of insulin
    2. inability of the cell to synthetize glycogen
    3. deficiency of lipoprotein lipase
    4. inability of the cell to transform lipids into glycogen
    5. excessive carbohydrates intake
28. What are the causes of parenchymatous lipid dystrophy? (c, e)
    1. excessive proteins in the diet
    2. deficient carbohydrates in the diet
    3. deficient proteins in the diet
    4. inability of the cell to transform lipids into glycogen
    5. excessive carbohydrates intake
29. What are the causes of parenchymatous lipid dystrophy? (c, e)
    1. excessive proteins in the diet
    2. deficient carbohydrates in the diet
    3. deficient proteins in the diet
    4. inability of the cell to transform lipids into glycogen
    5. denaturation of apoproteins
30. What are the causes of parenchymatous lipid dystrophy? (c, e)
    1. excessive proteins in the diet
    2. excessive production of apoproteins
    3. excessive lipid intake
    4. inability of the cell to transform lipids into glycogen
    5. denaturation of apoproteins
31. What are the causes of parenchymatous lipid dystrophy? (c, e)
    1. excessive proteins in the diet
    2. excessive production of apoproteins
    3. long-lasting starvation
    4. inability of the cell to transform lipids into glycogen
    5. depletion of apoproteins
32. What are the causes of parenchymatous lipid dystrophy? (c, e)
    1. deficient carbohydrate intake
    2. acute general hypoxia
    3. inability to produce lipoproteins
    4. inability of the cell to transform lipids into glycogen
    5. chronic long-lasting hypoxia
33. What are the causes of parenchymatous lipid dystrophy? (c, e)
    1. deficiency of intracellular glycolytic enzymes
    2. acute general hypoxia
    3. inability to produce lipoproteins
    4. inability of the cell to transform lipids into glycogen
    5. deficiency of intracellular lipolytic enzymes
34. What are the causes of parenchymatous lipid dystrophy? (c, e)
    1. deficiency of intracellular glycolytic enzymes
    2. acute general hypoxia
    3. inability to produce ketone bodies from lipids
    4. inability of the cell to transform lipids into glycogen
    5. deficiency of intracellular lipolytic enzymes
35. What can be the consequence of cell dystrophy? (e)
    1. cell dedifferentiation
    2. cell hypertrophy
    3. cell hyperplasia
    4. hypoplasia
    5. cell apoptosis
36. What can be the consequence of cell dystrophy? (e)
    1. cell dedifferentiation
    2. cell hypertrophy
    3. cell hyperplasia
    4. hypoplasia
    5. cell necrosis
37. What can be the consequence of cell dystrophy? (e)
    1. cell dedifferentiation
    2. cell hypertrophy
    3. cell hyperplasia
    4. hypoplasia
    5. inflammatory reaction
38. What can be the consequence of cell dystrophy? (e)
    1. cell dedifferentiation
    2. cell hypertrophy
    3. cell hyperplasia
    4. tissular hypoplasia
    5. tissular sclerosis
39. What can be the consequences of cell dystrophy? (d,e)
    1. cell dedifferentiation
    2. cell hypertrophy
    3. cell hyperplasia
    4. inflammatory reaction
    5. tissular sclerosis
40. What can be the consequences of cell dystrophy? (d,e)
    1. cell dedifferentiation
    2. cell hypertrophy
    3. cell hyperplasia
    4. cell apoptosis
    5. tissular sclerosis
41. What cell injury could be considered as primary? (1)
42. The disruption of cytoplasmic membrane under the action of mechanical factor
43. The destabilization of lysosomal membrane caused by intracellular acidosis
44. The initiation of apoptosis under the action of cytochrome C leaked from mitochondria
45. The osmotic disruption of cytoplasmic membrane at intracellular Na+ accumulation
46. The breakdown of cytoplasmic membrane under the action of lysosomal phospholipase
47. What cell injury could be considered as primary? (2,3)
48. The disruption of cytoplasmic membrane under the action of bacterial phospholipases
49. The destabilization of lysosomal membrane caused by ionizing radiation
50. The disruption of cytoplasmic membrane under the action of free radicals
51. The initiation of apoptosis under the action of cytochrome C getting out of mitochondria
52. The breakdown of cytoplasmic membrane under the action of lysosomal phospholipase
53. What cell injuries could be considered as secondary? (2,3,4)
54. The disruption of cytoplasmic membrane under the action of mechanical factor
55. The destabilization of lysosomal membrane caused by intracellular acidosis
56. The initiation of apoptosis under the action of cytochrome C getting out of mitochondria
57. The osmotic disruption of cytoplasmic membrane at intracellular Na+ accumulation
58. The electrical breakdown of cytoplasmic membrane under the action of electrical current
59. What cell injuries could be considered as secondary? (2,3,4)
60. The disruption of cytoplasmic membrane under the action of free radicals
61. The disruption of cytoplasmic membrane under the action of bacterial phospholipases
62. The destabilization of lysosomal membrane caused by ionizing radiation
63. The initiation of apoptosis under the action of cytochrome C getting out of mitochondria
64. The breakdown of cytoplasmic membrane under the action of lysosomal phospholipase
65. Under the action of hypoxia in the cell are triggered multiple injuries. What primary injury is caused by hypoxia? (2)
66. Cessation of ionic pumps
67. Decreased synthesis of ATP
68. Intracellular hyperosmolarity
69. Electrolytic intra–extracellular disbalance
70. Cytolysis
71. Under the action of free radicals in the cell are triggered multiple injuries. What primary injury is caused by free radicals? (3)
72. Electrical breakdown of cytoplasmic membrane
73. Intracellular hyperosmolarity
74. Peroxidation of membrane phospholipids
75. Electrolytic intra - extracellular disbalance
76. Cytolysis
77. Under the action of high temperature in the cell are triggered multiple injuries. What primary injury is caused by high temperature? (3)
78. Nonselective permeability of cytoplasmic membrane
79. Intracellular hyperosmolarity
80. Denaturation of membrane proteins
81. Electrolytic balance intra - extracellular
82. Cytolysis
83. Under the action of low temperature in the cell are triggered multiple injuries. What primary injury is caused by low temperature? (2)
84. Mechanical breakdown of cytoplasmic membrane
85. Crystallization of intracellular water
86. Intracellular hyperosmolarity
87. Electrolytic balance intra - extracellular
88. Cytolysis
89. Under the action of lipolytic extracellular enzymes in the cell are triggered multiple injuries. What primary injury is caused by phospholipids? (3)
90. Nonselective permeability of cytoplasmic membrane
91. Intracellular hyperosmolarity
92. Breakdown of membrane phospholipids
93. Electrolytic balance intra - extracellular
94. Cytolysis
95. Under the action of hyperosmolar environment in the cell are triggered multiple injuries. What kind of primary injury does the interstitial hyperosmolarity cause in the cells? (4)
96. Intracellular hyperosmolarity
97. Dehydration of cellular organelles
98. Mitochondrial disintegration
99. Dehydration of cellular hyaloplasm
100. Apoptosis
101. Under the action of hypoosmolar environment in the cell are triggered multiple injuries. What kind of primary injury does the interstitial hypoosmolarity cause in the cells? (4)
102. Hypoosmolarity of the cell hyaloplasm
103. Increases mechanical intracellular pressure
104. Cellular intumescences
105. Hyperhydration of the cell hyaloplasm
106. Apoptosis
107. Under the action of continue electrical current in the cell are triggered multiple injuries. What kind of primary injury does continue current cause? (3)
108. Polarization of the cell hyaloplasm
109. Intracellular electrolytic imbalance
110. Guided torrent by ions to anode and cathode
111. Disturbance of mitochondrial function
112. Apoptosis
113. Under the action of autoantibodies on the cell there can be causes death of it. What primary event occurs at interaction of autoantibodies and cellular antigens? (2)
114. Destruction of cellular antigens
115. Cell opsonization with Fc of the antibody
116. Compliment activation
117. Cell opsonization with C3b of activated compliment
118. Phagocytosis of the cell by macrophages
119. Under the action of autoantibodies on the cell is activated compliment, which in turn leads to the death of the cell. What primary phenomenon does appear under the activation of compliment in autoimmune reaction that leads to cellular death? (4)
120. Destruction of cytoplasm membrane
121. Cell opsonization with Fc of the antibody
122. Apoptosis
123. Cell opsonization with C3b of activated compliment
124. Phagocytosis of the cell by macrophages
125. Intracellular enzymes in the blood. What enzymatic changes are characteristic for hepatocyte injury? (2)
126. Increased activity of creatine phosphatase fraction MM in the blood
127. Increased activity of AST and ALT in the blood
128. Increased activity of cytochrome oxidase in the blood
129. Increased activity of acid phosphatase in the blood
130. Increased activity of alkaline phosphatase in the blood
131. The cell injury results in realizing of intracellular enzymes in the blood. What enzymatic changes are characteristic for pancreas injury? (1)
132. Increased activity of amylase in the blood
133. Increased activity of AST in the blood
134. Increased activity of cytochrome oxidase in the blood
135. Increased activity of ALT in the blood
136. Increased activity of alkaline phosphatase in the blood
137. The cell injury results in release of intracellular enzymes in the blood. What enzymatic changes are characteristic for cardiomyocytes injury? (4)
138. Increased activity of acid phosphatase in the blood
139. Increased activity of creatine kinase fraction MM in the blood
140. Increased activity of cytochrome oxidase in the blood
141. Increased activity of creatine kinase fraction MB in the blood
142. Increased activity of alkaline phosphatase in the blood
143. The cell injury results in release of intracellular enzymes in the blood. What enzymatic changes are characteristic for bile duct epithelial injury? (5)
144. Increased activity of creatine kinase fraction MB in the blood
145. Increased activity of AST in the blood
146. Increased activity of acid phosphatase in the blood
147. Increased activity of creatine kinase fraction MM in the blood
148. Increased activity of alkaline phosphatase in the blood
149. The disintegration of cytoplasmic membrane leads to electrolytic imbalance in intra- and extracellular space. How does the ratio of electrolytes in intra- and extracellular space change? (1)
150. Equilibration of intra- and extracellular ions concentration
151. Decreased the intracellular concentration of Na+
152. Increased the intracellular concentration of K+
153. Decreased the concentration of Ca++ in hyaloplasm
154. Increased the concentration of Ca++ in endoplasmic reticulum
155. The disintegration of cytoplasmic membrane leads to electrolytic imbalance in intra- and extracellular space. How does the ratio of electrolytes in intra- and extracellular space change? (2)
156. Imbalance of intra- and extracellular ions concentration
157. Increased intracellular concentration of Na+
158. Increased intracellular concentration of K+
159. Decreased concentration of Ca++ in hyaloplasm
160. Increased concentration of Ca++ in endoplasmic reticulum
161. The disintegration of cytoplasmic membrane leads to electrolytic imbalance in intra- and extracellular space. How does the ratio of electrolytes in intra- and extracellular space change? (2)
162. Imbalance of intra- and extracellular ions concentration
163. Decreased the intracellular concentration of K+
164. Increased the intracellular concentration of K+
165. Decreased the concentration of Ca++ in hyaloplasm
166. Increased the concentration of Ca++ in endoplasmic reticulum
167. The disintegration of cytoplasmic membrane leads to electrolytic imbalance in intra- and extracellular space. How does the ratio of electrolytes in intra- and extracellular space change? (5)
168. Imbalance of intra- and extracellular ions concentration
169. Decreased intracellular concentration of Na+
170. Increased intracellular concentration of K+
171. Decreased concentration of Ca++ in hyaloplasm
172. Increased concentration of Ca++ in hyaloplasm
173. What is the effect of electric current action on the excitable cells? (1)
174. Depolarization of cellular membrane
175. Hyperpolarization of cellular membrane
176. Repolarization of cellular membrane
177. Inhibition of electric impulse propagation
178. Decreased myocyte contractility
179. What is the effect of electric current action on the excitable cells? (2)
180. Opening of Cl ̅ channels
181. Opening of Na+ channels
182. Hyperpolarization of cellular membrane
183. Inhibition of electric impulse propagation
184. Decreased myocyte contractility
185. What is the effect of electric current action on the excitable cells? (1,2)
186. Depolarization of cellular membrane
187. Opening of Na+ channels
188. Repolarization of cellular membrane
189. Inhibition of electric impulse propagation
190. Decreased myocyte contractility
191. What is the effect of action of continue electrical current on the cell? (2)
192. Hyperpolarization of cellular membrane
193. Electrolysis of intracellular substances
194. Denaturation of ions
195. Polarization of cytoplasm membrane
196. Dissociation of intracellular substances
197. What is the effect of action of continue electrical current on the cell? (2)
198. Hyperpolarization of cellular membrane
199. Polarization of cytoplasm
200. Formation in excess of NaCl
201. Polarization of cytoplasm membrane
202. Dissociation of intracellular substances
203. Which endogenous enzymes could lead to cell injuries? (1,5)
204. Lysosomal enzymes
205. Superoxide dismutase
206. Cytochrome oxidase
207. Dehydrogenases
208. Pancreatic amylase
209. Which endogenous enzymes could lead to cytoplasm membrane injuries? (2,5)
210. Superoxide dismutase
211. Phagocytic cell enzymes
212. Cytochrome oxidase
213. Dehydrogenases
214. Pancreatic amylase
215. Which endogenous enzymes could lead to cytoplasm membrane injuries? (1,5)
216. Pancreatic trypsin
217. Superoxide dismutase
218. Cytochrome oxidase
219. Dehydrogenases
220. Pancreatic amylase
221. Which endogenous enzymes could lead to cytoplasm membrane injuries? (1,2,5)
222. Pancreatic trypsin
223. Lysosomal enzymes
224. Cytochrome oxidase
225. Dehydrogenases
226. Pancreatic amylase
227. Which endogenous enzymes could lead to cytoplasm membrane injuries? (1,4,5)
228. Pancreatic trypsin
229. Superoxide dismutase
230. Cytochrome oxidase
231. Phagocytic cell enzymes
232. Pancreatic amylase
233. What cells can release harmful enzymes for other cells? (1,4)
234. Cells of the pancreas
235. Cells of the salivary gland
236. Cells of the liver
237. Polymorphonuclear leukocytes
238. Lymphocytes
239. What is the effect of direct action of high temperature on the cell? (1)
240. Denaturation of the proteins with enzymatic activity
241. Denaturation of membrane phospholipids
242. Loss of electrolytes
243. Denaturation of ATP
244. Dehydration of the cell
245. What is the effect of direct action of high temperature on the cell? (1,3)
246. Denaturation of the proteins with enzymatic activity
247. Denaturation of membrane phospholipids
248. Synthesis of heat shock proteins
249. Denaturation of ATP
250. Increased synthesis of ATP
251. What is the effect of direct action of low temperature on the cell? (1)
252. Crystallization of water
253. Denaturation of the proteins with enzymatic activity
254. Denaturation of phospholipids and cellular membrane injury
255. Activation of ATP synthesis
256. Dehydration of the cell
257. What is the effect of direct action of low temperature on the cell? (1)
258. Mechanical injury of the cellular membrane
259. Denaturation of the proteins with enzymatic activity
260. Denaturation of phospholipids and cellular membrane injury
261. Activation of ATP synthesis
262. Dehydration of the cell
263. What electrolytic dyshomeostasis of internal environment leads to cell injury? (1)
264. Hyponatremia
265. Hypocalcemia
266. Hypokalemia
267. Hypomagnesemia
268. Hypermagnesemia
269. What electrolytic dyshomeostasis of internal environment leads to cell injury? (5)
270. Hypocalcemia
271. Hypokalemia
272. Hypomagnesemia
273. Hypermagnesemia
274. Hypernatremia
275. What interstitial dyshomeostasis changes the function of excitable cells? (1,2)
276. Hypercalcemia
277. Hypocalcemia
278. Hypochloremia
279. Hypomagnesemia
280. Hypermagnesemia
281. What interstitial dyshomeostasis changes the function of excitable cells? (1,2)
282. Hypercalcemia
283. Hyperkalemia
284. Hypomagnesemia
285. Hypermagnesemia
286. Hyperchloremia
287. What interstitial dyshomeostasis develops in injury of cytoplasmic membrane? (b)
     1. increased interstitial sodium concentration
     2. increased interstitial potassium concentration
     3. reduced interstitial sodium concentration
     4. reduced interstitial potassium concentration
     5. increased interstitial calcium concentration
288. What interstitial dyshomeostasis develops in injury of cytoplasmic membrane? (a)
     1. Increased interstitial hydrogen ion concentration
     2. Increased interstitial sodium ions concentration
     3. Decreased interstitial hydrogen ions concentration
     4. Reduced interstitial sodium ions concentration
     5. Increased interstitial calcium ions concentration
289. (2) What is the normal ratio of intracellular K+ to extracellular K+ ?
290. 1: 5
291. 4: 1
292. 1:20
293. 1: 10000
294. 100: 1
295. In cytoplasmatic membrane injury there is equilibration of intra-extracellular electrolytes levels. What is the effect of equilibration of intra-extracelular levels of K+? (a)
     1. inhibition of resting membrane potential
     2. hyperpolarization of cytoplasmatic membrane
     3. inhibition of active membrane potential
     4. intracellular hyperosmolarity
     5. activation of resting membrane potential
296. In cytoplasmatic membrane injury there is equilibration of intra-extracellular electrolytes levels. What is the effect of equilibration of intra-extracelular levels of K+? (a)
     1. inhibitory depolarization
     2. hyperpolarization of cytoplasmatic membrane
     3. inhibition of active membrane potential
     4. increased resting membrane potential
     5. intracellular hyperhydration
297. In cytoplasmatic membrane injury there is equilibration of intra-extracellular electrolytes levels. What is the effect of increased level of K+ in the interstitium? (c)
     1. hyperosmolarity of interstitial fluid
     2. reduced pH of interstitial fluid
     3. depolarization of neighboring cells
     4. hyperpolarization of neighboring cells
     5. inflammatory reaction
298. (c) What is the normal ratio of intracellular Na+ to extracellular Na+ ?
299. 1: 5
300. 4: 1
301. 1:20
302. 1: 10000
303. 100: 1
304. What is the efect of increased sodium ions level in the interstitium? (a)
     1. hyperosmolarity of interstitial fluid
     2. reduced pH of interstitial fluid
     3. cell swelling
     4. cell membrane depolarization
     5. inflammatory reaction
305. What is the efect of increased sodium ions level in the interstitium? (a)
     * 1. cell dehydration
       2. reduced pH of interstitial fluid
       3. cell swelling
       4. cell membrane depolarization
       5. increased pH of interstitial fluid
306. What is the efect of increased sodium ions level in the interstitium? (a)
     * 1. cell membrane hyperpolarization
       2. reduced pH of interstitial fluid
       3. cell swelling
       4. cell membrane depolarization
       5. increased pH of interstitial fluid
307. What are the effects of increased sodium level in the cell hyaloplasm? (a,c)
     1. cell membrane depolarization
     2. cell membrane hyperpolarization
     3. intracellular hyperosmolarity
     4. intracellular hypoosmolarity
     5. intracellular acidosis
308. What is the effect of increased sodium level in the cell hyaloplasm? (a)
     1. cell membrane depolarization
     2. cell membrane hyperpolarization
     3. reduced intracellular pH
     4. intracellular hypoosmolarity
     5. increased intracellular pH
309. What pathological process can reduce electrical resistance of cytoplasmatic membrane?(a)
     1. breakdown of membranary phospholipids
     2. depolarization of cytoplasmatic membrane
     3. hyperpolarization of cytoplasmatic membrane
     4. activation and opening of sodium channels
     5. inhibition of membranary pumps
310. (4) What is the normal ratio of intracellular Ca++ to extracellular Ca++ ?
311. 1: 5
312. 4: 1
313. 1:20
314. 1: 10000
315. 100: 1
316. What intracellular enzyme is activated by increased calcium in the cytoplasm? (a)
     1. endonucleases
     2. Krebs cycle enzymes
     3. glycolytic enzymes
     4. lipolytic enzymes
     5. glycogen-synthase
317. What intracellular enzyme is activated by increased calcium in the cytoplasm? (a)
     1. ATP-ases
     2. Krebs cycle enzymes
     3. glycolytic enzymes
     4. glucose-6-phosphatase
     5. glycogen-synthase
318. What intracellular enzymes are activated by increased calcium in the cytoplasm? (a,e)
     1. ATP-ases
     2. Krebs cycle enzymes
     3. glycolytic enzymes
     4. glucose-6-phosphatase
     5. proteases
319. What intracellular enzymes are activated by increased calcium in the cytoplasm? (a,e)
     1. AND-ases
     2. Krebs cycle enzymes
     3. glycolytic enzymes
     4. glucose-6-phosphatase
     5. proteolytic enzymes
320. What intracellular enzymes are activated by increased calcium in the cytoplasm? (a,e)
     1. AND-ases
     2. Krebs cycle enzymes
     3. glycolytic enzymes
     4. glucose-6-phosphatase
     5. phospholipases
321. What intracellular enzyme is activated at disintegration of endoplasmic reticulum membrane? (a)
     1. endonucleases
     2. lipolytic enzymes
     3. hydrolytic enzymes
     4. glycolytic enzymes
     5. glycogen-synthase
322. What intracellular enzyme is activated at disintegration of endoplasmic reticulum membrane? (a)
     1. phospholipases
     2. lipolytic enzymes
     3. enzymes of Krebs cycle
     4. glycolytic enzymes
     5. glycogen-synthase
323. What intracellular enzyme is activated at disintegration of endoplasmic reticulum membrane? (a)
     1. proteolytic enzymes
     2. lipolytic enzymes
     3. enzymes of Krebs cycle
     4. glycolytic enzymes
     5. glucooso-6-phosphatase
324. What intracellular enzyme is activated at disintegration of endoplasmic reticulum membrane? (a)
     1. ATP-ase
     2. lipolytic enzymes
     3. enzymes of Krebs cycle
     4. glycolytic enzymes
     5. glucooso-6-phosphatase
325. What intracellular enzymes are activated at disintegration of endoplasmic reticulum membrane? (a,c)
     1. endonucleases
     2. lipolytic enzymes
     3. phospholipases
     4. glycolytic enzymes
     5. glucooso-6-phosphatase
326. What is the effect of activation of intracellular ATP-ases? (a)
     1. exhaustion of ATP storages in the cell
     2. intense consumption of ATP for intracellular energy-dependent processes
     3. stimulates ATP regeneration
     4. exhaustion of ADP storages in the cell
     5. increases oxidative phosphorylation
327. What is the effect of activation of cell endonucleases? (b)
     1. triggers cell necrosis
     2. triggers cell apoptosis
     3. triggers cell autolysis
     4. triggers cell dystrophy
     5. triggers cell sclerosis
328. What is the effect of activation of cell endonucleases? (e)
     1. cleaves aminoacids from protein molecule
     2. cleave aminoacids from nucleoprotein molecule
     3. cleave timidine from nucleoprotein molecule
     4. cleaves oligopeptides from protein molecules
     5. cleave secvences from nucleoprotein molecule
329. What is the effect of activation of cell phospholipases? (e)
     1. activates synthesis of prostaglandins
     2. cell apoptosis
     3. disintegration of nucleic acids
     4. excessive accumulation of arachidonic acid
     5. disintegration of cell membranes
330. What is the effect of activation of cell phospholipases? (e)
     1. release of cytochrome c from mitochondria
     2. activates synthesis of leukotrienes
     3. disintegration of nucleic acids
     4. excessive accumulation of arachidonic acid
     5. destabilization of lysosomal membranes
331. What is the effect of activation of cell proteases? (a)
     1. triggers cell necrosis
     2. triggers cell apoptosis
     3. triggers cell dystrophy
     4. triggers cell dysplasia
     5. triggers sclerotic process in the cell
332. What can be the cause of intracellular acidosis? (e)
     1. activation of Krebs cycle
     2. activation of oxidative processes
     3. reduced excretion of H+ with urine
     4. excessive intake of acid substances
     5. exhaustion of intracellular buffer systems
333. What can be the cause of intracellular acidosis? (c)
     1. activation of glycogenolysis
     2. activation of oxidative processes
     3. activation of glycolysis
     4. excessive intake of acid substances
     5. inhibition of glycolysis
334. What can be the cause of intracellular acidosis? (c)
     1. activation of Krebs cycle
     2. activation of oxidative processes
     3. increased inflow of H+ ions in the cell
     4. excessive intake of acid substances
     5. inhibition of glycolysis
335. What can be the cause of intracellular acidosis? (c)
     1. cell hyperoxia
     2. activation of oxidative processes
     3. cell hypoxia
     4. excessive intake of acid substances
     5. inhibition of glycolysis
336. Cell hypoxia leads to intracellular acidosis. What are the consequences of decompensated intracellular acidosis? (b)
     1. hyperpolarization of cytoplasmatic membrane
     2. inactivation of Krebs cycle enzymes
     3. reduced permiability of cytoplasmatic membrane for sodium
     4. activation of oxidative processes
     5. reduced permiability of cytoplasmatic membrane for calcium
337. Cell hypoxia leads to intracellular acidosis. What are the consequences of decompensated intracellular acidosis? (b)
     1. hyperpolarization of cytoplasmatic membrane
     2. increased permiability of cytoplasmatic membrane for sodium
     3. reduced permiability of cytoplasmatic membrane for sodium
     4. activation of oxidative processes
     5. reduced permiability of cytoplasmatic membrane for calcium
338. Cell hypoxia leads to intracellular acidosis. What are the consequences of decompensated intracellular acidosis? (b)
     1. hyperpolarization of cytoplasmatic membrane
     2. inactivation of glycolytic enzymes
     3. reduced permiability of cytoplasmatic membrane for sodium
     4. activation of oxidative processes
     5. activation of glycolytic enzymes
339. Cell hypoxia lead to intracellular acidosis. What is a consequence of decompensated intracellular acidosis? (4)
     1. activation of oxidative phosphorylation
     2. activation of glycolytic enzymes
     3. hyperpolarization of cytoplasmic membrane
340. destabilization of lysosomal membranes
341. Reduced membranary permeability for calcium ions
342. Biologic oxidation is coupled with phosphorylation of ADP. What factor can decouple the process of oxidation and phosphorylation in mitochondria?
     1. thyroid hormones
     2. reduced hemoglobin
     3. stercobilin
     4. oxidized hemoglobin
     5. glucocorticoids
343. What is the effect of decoupling of oxidation and phosphorylation? (a)
     1. reduced efficacy of biologic oxidation
     2. increased efficacy of biologic oxidation
     3. reduced oxygen requirements
     4. reduced calorigenesis
     5. reduced non-effective cell consumption of ATP
344. . What is the effect of decoupling of oxidation and phosphorylation? (a)
     1. increased non-effective oxygen consumption
     2. increased efficacy of biologic oxidation
     3. reduced oxygen requirements
     4. reduced calorigenesis
     5. reduced non-effective cell consumption of ATP
345. What is the effect of decoupling of oxidation and phosphorylation? (a)
     1. increased calorigenesis
     2. increased efficacy of biologic oxidation
     3. reduced oxygen requirements
     4. reduced calorigenesis
     5. reduced cell consumption of ATP
346. What is the effect of decoupling of oxidation and phosphorylation? (a)
     1. reduced ATP production
     2. increased efficacy of biologic oxidation
     3. reduced oxygen requirements
     4. reduced calorigenesis
     5. increased ATP production
347. What are the effects of decoupling of oxidation and phosphorylation? (a,b)
     1. increased non-effective oxygen consumption
     2. increased calorigenesis
     3. reduced non-effective oxygen consumption
     4. reduced calorigenesis
     5. reduced non-effective cell consumption of ATP
348. What are the effects of decoupling of oxidation and phosphorylation? (a,b)
     1. reduced ATP production
     2. reduced efficacy of biologic oxidation
     3. reduced oxygen requirements
     4. reduced calorigenesis
     5. enhanced ATP production
349. For every vital function the cell needs energy. What is a consequence of energy depletion for cells? (c)
     1. inactivation of intracellular enzymes
     2. compensatory activation of membranary pumps
     3. inactivation of membranary pumps
     4. activation of anabolic metabolic processes in the cell
     5. closing of membrane ionic channels
350. For every vital function the cell needs energy. What is a consequence of energy depletion for cells? (c)
     1. inactivation of intracellular enzymes
     2. compensatory opening of membranary pumps
     3. cessation of intra-extracelular ionic gradient
     4. activation of anabolic metabolic processes in the cell
     5. closing of membranary ionic channels
351. For every vital function the cell needs energy. What is a consequence of energy depletion for cells? (c)
     1. hyperpolarization of cell membrane
     2. compensatory opening of membranary pumps
     3. depolarization of cell membrane
     4. activation of anabolic metabolic processes in the cell
     5. closing of membranary ionic channels
352. For every vital function the cell needs energy. What is a consequence of energy depletion for cells? (c,d)
     1. hyperpolarization of cell membrane
     2. compensatory opening of membranary pumps
     3. depolarization of cell membrane
     4. activation of anaerobic glycolysis
     5. inhibition of anaerobic glycolysis
353. What is a biochemical compensatory reaction in intracellular hypoxia? (c)
     1. activation of oxidative metabolic reactions
     2. activation of anabolic metabolic reactions
     3. activation of anaerobic metabolic reactions
     4. activation of Krebs cycle
     5. inhibition of glycolysis
354. Maintenance of intra-extracellular ionic gradient is energy-dependent. What is the consequence of cell ATP depletion? (a)
     1. increased calcium level in the hyaloplasm
     2. increased calcium level in the endoplasmic reticulum
     3. increased calcium level in mitochondria
     4. reduced calcium level in hyaloplasm
     5. reduced calcium level in endoplasmic reticulum
355. Some intracellular metabolic reactions are energy-dependent. What is the consequence of cell ATP deficiency? (a)
     1. reduced anabolic metabolic processes
     2. reduced catabolic metabolic processes
     3. increased anabolic metabolic processes
     4. reduced anaerobic metabolic processes
     5. increased catabolic metabolic processes
356. What is the consequence of lysosomal membrane destabilization? (a)
     1. hydrolysis of proteic components in hyaloplasm
     2. release of calcium ions in hyaloplasm
     3. release of sodium ions in hyaloplasm
     4. release of potassium ions in hyaloplasm
     5. cell apoptosis
357. What is the consequence of lysosomal membrane destabilization? (a)
     1. cell necrosis
     2. release of calcium ions in hyaloplasm
     3. release of sodium ions in hyaloplasm
     4. release of potassium ions in hyaloplasm
     5. cell apoptosis
358. Cell organelles are separated from hyaloplasm by membrane. What is the consequence of lysosomal membrane destabilization? (a)
     1. cell autolysis
     2. cell dystrophy
     3. release of sodium ions in cytoplasm
     4. release of potassium ions in cytoplasm
     5. cell apoptosis
359. What is the consequence of lysosomal membrane destabilization? (a)
     1. release of lysosomal enzymes in hyaloplasm with cell autolysis
     2. release of lysosomal enzymes in hyaloplasm with cell apoptosis
     3. release of sodium ions in cytoplasm with intracellular hyperosmolarity
     4. release of potassium ions in cytoplasm with hyperpolarization of cell membrane
     5. release of calcium ions in cytoplasm with activation of intracellular enzymes
360. What are the consequences of lysosomal membrane destabilization? (a,d)
     1. release of lysosomal enzymes in hyaloplasm with cell autolysis
     2. release of lysosomal enzymes in hyaloplasm with cell apoptosis
     3. release of sodium ions in cytoplasm with intracellular hyperosmolarity
     4. release of lysosomal enzymes in cytoplasm and hydrolysis of proteic compounds
     5. release of calcium ions in cytoplasm with activation of intracellular enzymes
361. What are the consequences of lysosomal membrane destabilization? (a,b)
     1. hydrolysis of proteic compound in hyaloplasm
     2. cell autolysis
     3. release of sodium ions in hyaloplasm
     4. release of potassium ions in hyaloplasm
     5. cell apoptosis
362. Lysosomal membrane traps the lysosomal enzymes within organelle. What is the factor which can destabilize lysosomal membrane? (a)
     1. hypoxia
     2. glucocorticoids
     3. vitamin E
     4. vitamin C
     5. glutation
363. Lysosomal membrane traps the lysosomal enzymes within organelle. What is the factor which can destabilize lysosomal membrane? (a)
     1. ionizing radiation
     2. glucocorticoids
     3. vitamin D
     4. vitamin C
     5. superoxide-dismutase
364. Lysosomal membrane traps the lysosomal enzymes within organelle. What is the factor which can destabilize lysosomal membrane? (a)
     1. reactive oxygen species
     2. glucocorticoids
     3. vitamin A
     4. vitamin D
     5. superoxide-dismutase
365. Lysosomal membrane traps the lysosomal enzymes within organelle. What factors can destabilize lysosomal membrane? (a,b)
     1. reactive oxygen species
     2. hypoxia
     3. catalase
     4. thyroid hormones
     5. superoxide-dismutase
366. Lysosomal membrane traps the lysosomal enzymes within organelle. What factors can destabilize lysosomal membrane? (a,b)
     1. reactive oxygen species
     2. ionizing radiation
     3. glucocorticoids
     4. catalase
     5. superoxide-dismutase
367. Lysosomal membrane traps the lysosomal enzymes within organelles. What factor works as endogenous stabilization factor for lysosomal membrane? (c)
     1. Vitamin A
     2. lactic acid
     3. glucocorticoids
     4. thyroid hormones
     5. pyruvic acid
368. Lysosomal membrane traps the lysosomal enzymes within organelles. What factor works as endogenous stabilization factor for lysosomal membrane? (c)
     1. Vitamin A
     2. lactic acid
     3. vitamin E
     4. thyroid hormones
     5. vitamin D
369. Lysosomal membrane traps the lysosomal enzymes within organelles. What factors work as endogenous stabilization factors for lysosomal membrane? (b,c)
     1. Vitamin A
     2. glucocorticoids
     3. vitamin E
     4. thyroid hormones
     5. vitamin D
370. Oxidative processes in the cell lead to generation of free radicals. What pathological process leads to generation of reactive oxygen species. (e)
     1. cell apoptosis
     2. cell dystrophy
     3. hyperthermia
     4. hypothermia
     5. hypoxia
371. Oxidative processes in the cell lead to generation of free radicals. What pathological process leads to generation of reactive oxygen species (e)
     1. cell apoptosis
     2. cell dystrophy
     3. hyperthermia
     4. arterial hyperemia
     5. hyperoxia
372. Oxidative processes in the cell lead to generation of free radicals. What pathological process leads to generation of reactive oxygen species. (e)
     1. cell apoptosis
     2. arterial hyperemia
     3. hyperthermia
     4. hypothermia
     5. inflammatory reaction
373. Oxidative processes in the cell lead to generation of free radicals. What pathological process leads to generation of reactive oxygen species. (e)
     1. cell apoptosis
     2. arterial hyperemia
     3. cell dystrophy
     4. hypothermia
     5. ischemia
374. Oxidative processes in the cell lead to generation of free radicals. What pathological processes lead to generation of reactive oxygen species. (a,e)
     1. hyperoxia
     2. arterial hyperemia
     3. hyperthermia
     4. hypothermia
     5. ischemia
375. Oxidative processes in the cell lead to generation of free radicals. What pathological processes lead to generation of reactive oxygen species. (a,e)
     1. hypoxia
     2. arterial hyperemia
     3. hyperthermia
     4. hypothermia
     5. ionizing radiation
376. Oxidative processes in the cell lead to generation of free radicals. What pathological processes lead to generation of reactive oxygen species. (a,e)
     1. inflammatory reaction
     2. arterial hyperemia
     3. hyperthermia
     4. hypothermia
     5. hyperoxia
377. Oxidative processes in the cell lead to generation of free radicals. What pathological processes lead to generation of reactive oxygen species. (a,e)
     1. ionizing radiation
     2. arterial hyperemia
     3. hyperthermia
     4. hypothermia
     5. inflammation
378. Oxidative processes in the cell lead to generation of free radicals. What substance represents reactive oxygen species? (c)
     1. phosphate anion
     2. catalase
     3. peroxide of hydrogen
     4. hydrogen ions
     5. bicarbonate ion
379. Oxidative processes in the cell lead to generation of free radicals. What substance represents reactive oxygen species? (c)
     1. phosphate anion
     2. hydrocarbonat anion
     3. oxygen superoxide
     4. H+ ions
     5. ferritin
380. Oxidative processes in the cell lead to generation of free radicals. What substance represents reactive oxygen species? (c)
     1. phosphate anion
     2. catalase
     3. hydroxyl radical
     4. hydrogen ions
     5. Na+ and Ca++ cations
381. Oxidative processes in the cell lead to generation of free radicals. What substance represents reactive oxygen species? (b)
     1. phosphate anion
     2. peroxynitrite
     3. hydrocarbonat anion
     4. hydrogen ions
     5. Cl- anion
382. Oxidative processes in the cell lead to generation of free radicals. What substances represent reactive oxygen species? (a,b)
     1. superoxide of oxygen
     2. peroxynitrite
     3. hydrocarbonat anion
     4. hydrogen ions
     5. superoxide of hydrogen
383. Oxidative processes in the cell lead to generation of free radicals. What substances represent reactive oxygen species? (a,b)
     1. superoxide of oxygen
     2. peroxide of hydrogen
     3. hydrocarbonat anion
     4. hydrogen ions
     5. superoxide of hydrogen
384. Oxidative processes in the cell lead to generation of reactive oxygen species, which have negative effects on cell structures. What substance represents endogenous antioxidant system? (a)
     1. vitamin E
     2. cytochrome oxidase
     3. enzymes of Krebs cycle
     4. prostaglandins
     5. phospholipase A2
385. Oxidative processes in the cell lead to generation of reactive oxygen species, which have negative effects on cell structures. What substance represents endogenous antioxidant system? (a)
     1. catalase
     2. cytochrome oxidase
     3. enzymes of Krebs cycle
     4. prostaglandins
     5. phospholipase A2
386. Oxidative processes in the cell lead to generation of reactive oxygen species, which have negative effects on cell structures. What substance represents endogenous antioxidant system? (a)
     1. superoxide dismutase
     2. cytochrome oxidase
     3. enzymes of Krebs cycle
     4. prostaglandins
     5. phospholipase A2
387. Oxidative processes in the cell lead to generation of reactive oxygen species, which have negative effects on cell structures. What substances represent endogenous antioxidant system? (a,b)
     1. superoxide dismutase
     2. catalase
     3. cytochrome oxidase
     4. phospholipase
     5. prostaglandins
388. Oxidative processes in the cell lead to generation of reactive oxygen species, which have negative effects on cell structures. What substances represent endogenous antioxidant system? (a,b)
     1. superoxide dismutase
     2. vitamin E
     3. cytochrome oxidase
     4. phospholipase
     5. vitamin D
389. Many cellular pathological processes lead to generation of reactive oxygen species. What is negative efect of ROS? (a)
     1. lipid peroxidation
     2. intensification of energogenesis with excessive ATP generation
     3. increased oxygen consumption
     4. intensification of anaerobic glycolysis
     5. inhibition of energogenesis with ATP deficiency
390. Many cellular pathological processes lead to generation of reactive oxygen species. What is negative efect of ROS? (a)
     1. peroxidation of nucleic acids
     2. intensification of energogenesis with excessive ATP generation
     3. increased oxygen consumption
     4. intensification of anaerobic glycolysis
     5. inhibition of energogenesis with ATP deficiency
391. Many cellular pathological processes lead to generation of reactive oxygen species. What are negative effects of ROS? (a,c)
     1. peroxidation of nucleic acids
     2. activation of glycolysis
     3. peroxidation of membranary phospholipids
     4. peroxidation of glucose in cytoplasm of the cell
     5. inhibition of energogenesis with ATP deficiency
392. Many cellular pathological processes lead to generation of reactive oxygen species. What are negative effects of ROS? (a,c)
     1. peroxidation of nucleic acids
     2. activation of glycolysis and intracellular acidosis
     3. release of cytochrome c from mitochondria and cell apoptosis
     4. release of cytochrome c from mitochondria and cell necrosis
     5. inhibition of energogenesis with ATP deficiency
393. Many cellular pathological processes lead to generation of reactive oxygen species. What are negative effects of ROS? (b,c)
     1. activation of anaerobic glycolysis with intracellular acidosis
     2. disintegration of membranary phospholipids
     3. release of cytochrome c from mitochondria and cell apoptosis
     4. release of cytochrome c from mitochondria and cell necrosis
     5. inhibition of energogenesis with ATP deficiency
394. Many cellular pathological processes lead to generation of reactive oxygen species. What are negative effects of ROS? (b,c)
     1. activation of anaerobic glycolysis with intracellular acidosis
     2. disintegration of membranary phospholipids
     3. protein misfolding
     4. release of cytochrome c from mitochondria and cell necrosis
     5. inhibition of energogenesis with ATP deficiency
395. Many cellular pathological processes lead to generation of reactive oxygen species. What are negative effects of ROS? (b,c)
     1. activation of anaerobic glycolysis with intracellular acidosis
     2. cell autolysis
     3. protein misfolding
     4. increased calcium level in the cells
     5. inhibition of energogenesis with ATP deficiency
396. Many cellular pathological processes lead to generation of reactive oxygen species. What is a negative effect of ROS? (c)
     1. inhibition of anabolic metabolic processes
     2. increased potassium level in the cells
     3. disintegration of lysosomal membranes
     4. increased sodium level in the cell
     5. increased calcium level in the cell
397. (1) What does necrosis represent?
     1. Cell death due to pathogenic factors action
     2. Cell death after organism death
     3. Cell death due to genetic material exhaustion
     4. Cell death due to functional capacity exhaustion
     5. Cell death during the senile involution of organism
398. (4) What is the main link of necrosis pathogenesis in the cell membrane lesion?
     1. Ion channel dysfunction
     2. Mitochondria dysfunction
     3. Ion pumps dysfunction
     4. Balancing of the intra- and extracellular ion concentration
     5. Cell nucleus dysfunction
399. (3) What is the main link of necrosis pathogenesis in the mitochondrial lesion?
     1. Cytochrome c release from mitochondria
     2. Intracellular protein synthesis cessation
     3. ATP synthesis cessation
     4. Membrain channel function cessation
     5. Membrain pump function cessation
400. (1) What is the main link of necrosis pathogenesis during free radicals action?
     1. Cytoplasmic membrane disintegration
     2. Intracellular hyperosmolarity
     3. Genetic mutations
     4. Intercellular connections destruction
     5. Inflammation
401. (4) What is one of the necrosis consequences?
     1. Hypernatriemia
     2. Hypokaliemia
     3. Hypercalciemia
     4. Inflammation
     5. Apoptosis of adjacent cells
402. (3) What is one of the necrosis consequences?
     1. Hypernatriemia
     2. Hypokaliemia
     3. Enzymemia
     4. Hypercalciemia
     5. Apoptosis of adjacent cells
403. (5) What is one of the necrosis consequences?
     1. Hypernatriemia
     2. Hypokaliemia
     3. Hypercalciemia
     4. Apoptosis of adjacent cells
     5. Hyperkaliemia
404. (2,5) What are the necrosis consequences?
     1. Hypernatriemia
     2. release of proinflammatory cytokines
     3. Hypokaliemia
     4. Hypercalciemia
     5. enzymemia
405. (4) What is one of the necrosis consequences?
     1. Hypernatriemia
     2. Hypokaliemia
     3. Hypercalciemia
     4. Intracellular antigen release
     5. Apoptosis of the adjacent cells
406. (1,4) What are the necrosis consequences?
     1. Inflammation
     2. Hypokaliemia
     3. Hypercalciemia
     4. Enzymemia
     5. Apoptosis of the adjacent cells
407. (2,4) What are the necrosis consequences?
     1. Hypokaliemia
     2. Enzymemia
     3. Hypercalciemia
     4. Pro-inflammatory mediators formation
     5. Apoptosis of the adjacent cells
408. (1) What is one of the general consequences of necrosis?
     1. Fever
     2. Leukopenia
     3. Bacteremia
     4. Hypernatriemia
     5. Hypercalciemia
409. (1,2) What are the general consequences of necrosis?
     1. Acute phase reaction
     2. Fever
     3. Bacteremia
     4. Hypernatriemia
     5. Hypercalciemia
410. (3) What biochemical tests can be used to detect necrosis in organs?
     1. Dosing blood level of potassium
     2. Dosing blood level of calcium
     3. Dosing the blood level of enzymes
     4. Dosing of pyrogenic interleukins
     5. Dosing of blood count of leukocytosis
411. (1,2,3) What is sclerosis of the organ?
     1. Substitution of parenchyma with connective tissue
     2. Absolute increase of the connective tissue rate concerning the normal parenchyma
     3. Absolute increase of the connective tissue rate concerning the reduced parenchyma
     4. Cholesterol deposition in organ
     5. Organ stroma reducing
412. (1) What does the sclerosis of organ mean?
     1. Pathological regeneration
     2. Reparative physiological regeneration
     3. Compensatory physiological regeneration
     4. Protective physiological regeneration
     5. The last phase of the inflammation
413. (1) What factor induces sclerosis?
     1. Cell lessions
     2. Cell mitosis cessation
     3. Primary hypofunction of the organ
     4. Growth factors lack
     5. Apoptosis
414. (2) What factor induces sclerosis?
     1. Cell mitosis cessation
     2. Cell dystrophy
     3. Primary hypofunction of the organ
     4. Growth factors lack
     5. Apoptosis
415. (3) What factor induces sclerosis?
     1. Cell mitosis cessation
     2. Primary hypofunction of the organ
     3. Cell necrosis
     4. Growth factors lack
     5. Apoptosis
416. (4) What factor induces sclerosis?
     1. Cell mitosis cessation
     2. Primary hypofunction of the organ
     3. Growth factors lack
     4. Chronic inflammation
     5. Apoptosis
417. (1,3) What factors induce sclerosis?
     1. Chronic inflammation
     2. Cell mitosis cessation
     3. Cell lesions
     4. Growth factors lack
     5. Apoptosis
418. (2,3) What factors induce sclerosis?
     1. Cell mitosis cessation
     2. Chronic inflammation
     3. Cell dystrophy
     4. Growth factors lack
     5. Apoptosis
419. (3,4) What factors induce sclerosis?
     1. Cell mitosis cessation
     2. Cell necrosis
     3. Growth factors lack
     4. Chronic inflammation
     5. Apoptosis
420. (3) What process certainly is involved in the sclerosis evolution?
     1. cell lession,
     2. fibroblast activation
     3. necrosis
     4. inflammation
     5. macrophage activation
421. (2) In which organ the irreparable cell lesions obligatory provoke sclerosis?
     1. Liver
     2. Brain
     3. Intestinal mucose
     4. Thyroid gland
     5. Skin
422. (3) In which organ the irreparable cell lesions obligatory provoke sclerosis?
     1. Liver
     2. Intestinal mucose
     3. Myocardium
     4. Thyroid gland
     5. Skin
423. (1) What is the source of connective tissue in the sclerosis pathogenesis?
     * + 1. Neogenesis of the connective tissue due to fibroblast proliferation
         2. Dedifferentiation of the fibroblasts and their opulent proliferation
         3. Collagen abundant production by macrophages
         4. Release of the intracellular collagen fibers in interstitium
         5. Transformation of the base substance in collagen
424. (2) What is the source of connective tissue in the sclerosis pathogenesis?
     * + 1. Dedifferentiation of the fibroblasts and their opulent proliferation
         2. Opulent synthesis of collagen without fibroblast proliferation
         3. Collagen abundant production by macrophages
         4. Release of the intracellular collagen fibres in interstitium
         5. Transformation of the base substance in collagen
425. (3) What is the source of connective tissue in the sclerosis pathogenesis?
     * + 1. Dedifferentiation of the fibroblasts and their opulent proliferation
         2. Collagen abundant production by macrophages
         3. Increase of the connective tissue mass due to parenchyma reduction
         4. Release of the intracellular collagen fibres in the interstitium
         5. Transformation of the base substance in collagen
426. (3) What is the source of connective tissue in the sclerosis pathogenesis?
     * + 1. Dedifferentiation of the fibroblasts and their opulent proliferation
         2. Collagen abundant production by macrophages
         3. Opulent stimulation of fibroblasts
         4. Release of the intracellular collagen fibers in interstitium
         5. Transformation of the base substance in collagen
427. (2) A homeostatic process precluding sclerosis is collagenolysis. What is the mechanism of collagen excess reducing in the organ?
     1. Collagen excess excretion by urine
     2. Collagen fibers phagocytosis with intracellular spliting
     3. Transformation of collagen fibers in elastic fibers
     4. Transformation of fibrocytes in parenchymal cells
     5. Fibrocyte apoptosis
428. (4) A homeostatic process precluding sclerosis is collagenolysis. What is the mechanism of collagen excess reducing in the organ?
     1. Collagen excess excretion by urine
     2. Transformation of collagen fibers in elastic fibers
     3. Transformation of fibrocytes in parenchymal cells
     4. Secretion of collagenolytic enzymes by macrophages
     5. Fibrocyte apoptosis
429. (1,5) What are the mechanisms of collagen excess reducing in the organ?
     1. Intracellular degradation by collagenolytic enzymes
     2. Transformation of collagen fibers in elastic fibers
     3. Transformation of fibrocytes in parenchymal cells
     4. Extracellular degradation by collagenolytic enzymes
     5. Fibrocyte apoptosis
430. (3) Which pathological process leads to progressive sclerosis?
     1. Cell dedifferentiation
     2. Prolonged arterial hyperemia
     3. Prolonged local hypoxia
     4. Boosted apoptosis of parenchyma
     5. Hypersecretion of glucocorticoids
431. (4) Which pathological process leads to progressive sclerosis?
     1. Cell dedifferentiation
     2. Prolonged arterial hyperemia
     3. Boosted apoptosis of parenchyma
     4. Massive alteration and collapse of parenchyma
     5. Hypersecretion of glucocorticoids
432. (3) Which pathological process leads to progressive sclerosis?
     1. Cell dedifferentiation
     2. Prolonged arterial hyperemia
     3. Chronic inflammation
     4. Boosted apoptosis of parenchyma
     5. Hypersecretion of glucocorticoids
433. (5) Which pathological process leads to progressive sclerosis?

a Cell dedifferentiation

1. Prolonged arterial hyperemia
2. Boosted apoptosis of parenchyma
3. Hypersecretion of glucocorticoids
4. Persistent hemo-lymphodynamic disorderes
5. (2) Which pathological process leads to progressive sclerosis?
   1. Cell dedifferentiation
   2. Congenital defects of the collagenolytic mechanisms
   3. Prolonged arterial hyperemia
   4. Boosted apoptosis of parenchyma
   5. Hypersecretion of glucocorticoids
6. (1,3) Which pathological processes lead to progressive sclerosis?
   1. Congenital defects of the collagenolytic mechanisms
   2. Cell dedifferentiation
   3. Prolonged local hypoxia
   4. Boosted apoptosis of parenchyma
   5. Hypersecretion of glucocorticoids
7. (2,3) Which pathological processes lead to progressive sclerosis?
   1. Cell dedifferentiation
   2. Congenital defects of the collagenolytic mechanisms
   3. Massive alteration and collapse of parenchyma
   4. Boosted apoptosis of parenchyma
   5. Hypersecretion of glucocorticoids
8. (1,3) Which pathological processes lead to progressive sclerosis?
   1. Congenital defects of the collagenolytic mechanisms
   2. Cell dedifferentiation
   3. Local hemo-lymphodynamic disorderes
   4. Boosted apoptosis of parenchyma
   5. Hypersecretion of glucocorticoids
9. (4) What is one of the consequences of sclerosis?
   1. Organ malignancy
   2. Fatty dystrophy of the organ
   3. Hypertrophy of the organ
   4. Hypofunction of the organ
   5. Cell dedifferentiation
10. (3) What is one of the consequences of sclerosis?
    1. Organ malignancy
    2. Fatty dystrophy of the organ
    3. Organ deformation
    4. Hypertrophy of the organ
    5. Cell dedifferentiation
11. (2) What is one of the consequences of sclerosis?
    1. Organ malignancy
    2. Organ remodeling
    3. Fatty dystrophy of the organ
    4. Hypertrophy of the organ
    5. Cell dedifferentiation
12. (1,3) What are the consequences of sclerosis?
    1. Organ remodeling
    2. Organ malignancy
    3. Hypofunction of the organ
    4. Hypertrophy of the organ
    5. Cell dedifferentiation
13. (4) What is the principle of the pathogenetic correction of sclerosis?
    1. Local inflammation triggering
    2. Surgical removal of connective tissue excess
    3. Collagenolysis inhibition
    4. Fibrogenesis cessation
    5. Stimulation of the parenchymal cell multiplication
14. (3) What is the principle of the pathogenetic correction of sclerosis?
    1. Local inflammation triggering
    2. Surgical removal of connective tissue excess
    3. Collagenolysis stimulation
    4. Collagenolysis inhibition
    5. Stimulation of the parenchymal cell multiplication
15. (1,2) What are the principles of the pathogenetic correction of sclerosis?
    1. Collagenolysis stimulation
    2. Fibrogenesis cessation
    3. Surgical removal of connective tissue excess
    4. Collagenolysis inhibition
    5. Stimulation of the parenchymal cell multiplication
16. (2) What cell is exposed to apoptosis in a mature organism?
    1. Aged normal cells
    2. Normal cells which exceeded functional needs
    3. Dead cells
    4. Sclerosed cells
    5. Cells with reversible injury
17. (3) What cell is exposed to apoptosis in a mature organism?
    1. Aged normal cells
    2. Dead cells
    3. Cells with genetic defects
    4. Sclerosed cells
    5. Cells with reversible injury
18. (4) What cell is exposed to apoptosis in a mature organism?
    1. Aged normal cells
    2. Dead cells
    3. Sclerosed cells
    4. Cells infected by virus
    5. Cells with reversible injury
19. (5) What cell is exposed to apotosis in a matur organism?

a Aged normal cells

b Dead cells

c Sclerosed cells

d Cells with reversible lessions

e Cancerogenous cells

1. (5) What cell is exposed to apotosis in a matur organism?

a normal cells

b Cells with reversible lessions

c Dead cells

d Sclerosed cells

e Cells with irreversible lessions

1. (4,5) What cells are exposed to apoptosis in a mature organism?

a Aged normal cells

b Cells with reversible injury

c Dead cells

d Normal cells which exceeded functional needs

e Cells with irreversible injury

1. (2,3) What cells are exposed to apotosis in a matur organism?

a Aged normal cells

b Cells with irreversible lesions

c Cells with genetic defects

d Sclerosed cells

e Cells with reversible lesions

1. (2,4) What cells are exposed to apoptosis in a mature organism?

a Aged normal cells

b Cells with irreversible lessions

c Sclerosed cells

d Cells infected by virus

e Cells with reversible lessions

1. (1,3) What cells are exposed to apoptosis in a mature organism?

a Normal cells which exceeded functional needs

b Aged normal cells

c Cancerous cells

d Sclerosed cells

e Cells with reversible injury

1. (1) How apoptosis is manifested in the initial period?

a Disorganization of the intercellular communication structures

b Cell membrane disintegration

c Mitochondria disintegration

d Karyorrhexis

e Karyolysis

1. (5) What factor can serve as positive signal for apoptosis triggering?

a Recoverable injuries

b Cell necrosis

c Cell hyperfunction

d Mitotic hyperactivity

e Non-viable mutations

1. (2) What trigger factor can serve as positive signaling for apoptosis?

a Recoverable injuries

b Viral infected cells

c Cell necrosis

d Cell hyperfunction

e Mitotic hyperactivity

1. (3) What factor can serve as positive signal for apoptosis triggering?

a Recoverable injuries

b Cell necrosis

c Non-recoverable injuries

d Cell hyperfunction

e Mitotic hyperactivity

1. (4) What factor can serve as positive signal for apoptosis triggering?

a Recoverable injuries

b Cell necrosis

c Cell hyperfunction

d Excess of cells in the organ

e Mitotic hyperactivity

1. (5) What hormone can serve as positive signal for apoptosis triggering?

a Androgens

b Estrogens

c ACTH

d Catecholamines

e Glucocorticoids

1. (3) What factor can serve as negative signal for apoptosis triggering?

a Thyroxine lack

b Glucocorticoids lack

c Growth hormone lack

d Insulin lack

e Catecholamines lack

1. (2) What factor can serve as negative signal for apoptosis triggering?

a Thyroxine lack

b Thyrotropic hotmone lack

c Glucocorticoids lack

d Insulin lack

e Catecholamines lack

1. (1) What factor can serve as negative signal for apoptosis triggering?

a Androgens lack

b Thyroxine lack

c Glucocorticoids lack

d Insulin lack

e Catecholamines lack

1. (5) What factor can serve as negative signal for apoptosis triggering?

a Thyroxine lack

b Glucocorticoids lack

c Insulin lack

d Catecholamines lack

e Estrogens lack

1. (2) What factor can serve as negative signal for apoptosis triggering?

a Thyroxine lack

b ACTH lack

c Glucocorticoids lack

d Insulin lack

e Catecholamines lack

1. (2,3,4) For which cells the somatotropin lack is a negative signal for apoptosis?

a Neurons

b Osteoblasts

c Chondroblasts

d Striated myocytes

e Lymphocytes

1. (2) For which cells the TSH lack is a negative signal for apoptosis?

a Neurons

b Thyroid gland cells

c Parathyroid cells gland

d Neurosecretory hypothalamus cells

e Adipocytes

1. (3) For which cells the ACTH lack is a negative signal for apoptosis?

a Neurons

b Suprarenal medular cells

c Suprarenal cortex cells

d Neurosecretory hypothalamus cells

e Lymphocytes

1. (1,2) For which cells the estrogens lack is a negative signal for apoptosis?

a Vaginal epithelium

b Endometrial cells

c Hair follicular cells

d Neurosecretory hypothalamus cells

e Ovarium cells

1. (4) For which cells the androgens lack is a negative signal for apoptosis?

a Neurosecretory hypothalamus cells

b Sertoli cells

c Leidig cells

d Hair follicular cells

e Spermatozoides

1. (3) For which cells the androgens lack is a negative signal for apoptosis?

a Neurosecretory hypothalamus cells

b Sertoli cells

b Prostate cells

d Leidig cells

e Spermatozoides

1. (4) For which cells the androgens lack is a negative signal for apoptosis?

a Neurosecretory hypothalamus cells

b Sertoli cells

c Leidig cells

d Laryngeal cartilage chondroblasts

d Spermatozoides

1. (2) For which cells the FSH lack is negative signal for apoptosis in men?

a Neurosecretory hypothalamus cells

b Sertoli cells

c Leidig cells

d Laryngeal cartilage chondroblasts

e Spermatozoids

1. (1) For which cells the FSH lack is negative signal for apoptosis in women?

a Ovarian follicule cells

b Endometrial cells

c Vaginal epithelium

d Laryngeal cartilage chondroblasts

e Mammary gland epithelium

1. (3) For which cells the LSH lack is negative signal for apoptosis in men?

a Neurosecretory hypothalamus cells

b Sertoli cells

c Leidig cells

d Laryngeal cartilage chondroblasts

e Spermatozoides

1. (5) For which cells the LSH lack is negative signal for apoptosis in women?

a Neurosecretory hypothalamus cells

b Sertoli cells

c Leidig cells

d Laryngeal cartilage chondroblasts

e Yellow body cells

1. (1) How the apoptosis is manifested in the initial period?

a Cytoplasm condensation

b Cell membrane disintegration

c Mitochondria disintegration

d Karyorrhexis

e Karyolysis

1. (3) How the initial period of apoptosis is manifested?

a Cell membrane disintegration

b Mitochondria disintegration

c Nucleus condensation

d Karyorrhexis

e Karyolysis

1. (1,5) How the apoptosis is manifested in the initial period?

a Nucleus condensation

b Cell membrane disintegration

c Mitochondria disintegration

d Karyorrhexis

e Intercellular communication structures disorganization

1. (2,5) How the apoptosis is manifested in the initial period?

a Cell membrane disintegration

b Nucleus condensation

c Mitochondria disintegration

d Karyorrhexis

e Cytoplasm condensation

1. (1) What condition is necessary for complete apoptosis evolution?

a Maintenance of the cell membrane integrity

b Maintenance of the nucleus integrity

c Maintenance of the cytoskeleton integrity

d Maintenance of the chromosome integrity

e Maintenance of the intercellular connections

1. (2) What condition is necessary for complete apoptosis evolution?

a Maintenance of the nucleus integrity

b maintenance of mitochondrial function

c maintenance of endoplasmic reticulum function

d maintenance of Golgi apparatus function

e Maintenance of the intercellular connections

1. (4) How the apoptosis is manifested in the middle-period?

a Apoptotic bodies disintegration

b Cell membrane disintegration

c Mitochondria disintegration

d Apoptotic bodies formation

e Karyorrhexis

1. (3) What is the terminal phenomenon of apoptosis?

a Phagocytosis of the cell exposed to apoptosis

b Extracellular disintegration of the apoptotic bodies with biochemical compound elimination

c Phagocytosis of the apoptotic bodies by tissular macrophages

d Biochemical degradation of the apoptotic bodies in the macrophages of the liver

e Apoptotic bodies excretion by kidneys

1. What are the effect of kinins in inflammation?
2. vasoconstriction
3. vasodilation \*
4. systemic hypertension
5. sensation of pain \*
6. bactericide effect
7. What are the effects of activated Hageman factor contact?
8. chemotactic effect
9. anti-inflammatory
10. activation of kininogenic system \*
11. d. direct destruction of the vascular wall
12. activation of anticlotting system \*
13. What are the effects of activated Hageman factor contact?
14. chemotactic effect
15. anti-inflammatory
16. activation of kininogenic system \*
17. direct destruction of the vascular wall
18. activation of the fibrinolytic system \*
19. What are the effects of C3a and C5a in inflammatory focus?
20. vasoconstriction action
21. stimulates prostaglandin synthesis
22. chemotactic action \*
23. degranulation of mast cells \*
24. pro-clotting activity
25. What are the effects of C3a and C5a in inflammatory focus?
26. vasoconstriction action
27. stimulates prostaglandin synthesis
28. chemotactic action \*
29. vasodilatory action \*
30. pro-clotting activity
31. What are the effects of interleukin in the inflammatory focus?
32. T lymphocytes activation \*
33. anti-inflammatory action
34. pyrogenic action \*
35. chemotactic action
36. erythropoietic action
37. What are the pathogenic factors of cardiac edemas?
38. rennin hypersecretion \*
39. increases hydrostatic pressure in capillaries \*
40. hyperonchia of blood plasma
41. arterial hypotension
42. hypoxemia
43. What are the pathogenic factors of cardiac edemas?
44. venous stasis \*
45. hypernatremia \*
46. hypovolemia
47. arterial hypotension
48. hypoxemia
49. What biological active factor forms at the complement activation?
    1. C3
    2. C5
    3. C5-C9 \*
    4. C9
    5. C1a
50. What can be the consequences of protein maldigestion?
51. disturbances in regenerative processes \*
52. proteinuria
53. reduced creatinine level in the blood
54. reduced urea level in the blood
55. reduced aminoacids concentration in the blood
56. What positive effects has the fever?
57. stimulates the phagocytosis \*
58. direct bactericide action
59. enhances the bacteriostatic action of the antibiotic \*
60. accelerates regenerative processes
61. stimulates the anabolic metabolic processes
62. Which is an external manifestation of ischemia?
    1. diffuse erythema
    2. tissue swelling
    3. decrease of skin turgor \*
    4. skin turgescence growth
    5. cyanosis
63. What process includes physiological regeneration in inflammatory focus?
64. excessive formation of specific parenchymal structures
65. restoration nonspecific mesenchymal structures \*
66. excessive formation of nonspecific mesenchymal structures
67. angiogenesis de novo
68. excessive formation of collagen fibrils
69. What cells realize inflammatory mediators?
70. platelets \*
71. parenchymatous cells
72. erythrocytes
73. skeletal myocytes
74. neurons
75. What cells realize inflammatory mediators?
76. platelets \*
77. parenchymatous cells
78. erythrocytes
79. skeletal myocytes
80. granulocytes \*
81. What cells realize inflammatory mediators?
82. granulocyte leukocytes \*
83. parenchymatous cells
84. erythrocytes
85. skeletal myocytes
86. neurons
87. What cells realize inflammatory mediators?
88. Platelets \*
89. parenchymatous cells
90. erythrocytes
91. skeletal myocytes
92. fibroblasts \*
93. What cells realize inflammatory mediators?
94. fibroblasts \*
95. parenchymatous cells
96. erythrocytes
97. skeletal myocytes
98. neurons
99. What cells realize inflammatory mediators?
100. mast cells \*
101. parenchymatous cells
102. erythrocytes
103. skeletal myocytes
104. neurons
105. Which is a manifestation of venous hyperemia?
     1. diffuse redness (erythema), swelling and decrease of local temperature
     2. diffuse redness (erythema), swelling and increase of local temperature
     3. cyanosis, swelling and increase of local temperature
     4. cyanosis, swelling and decrease of local temperature \*
     5. paleness, swelling and decrease of local temperature
106. What chemotactic factor release mast cells?
107. chemotactic factor of neutrophils \*
108. chemotactic factor of T lymphocytes
109. chemotactic factor of B lymphocyte
110. chemotactic factor of basophils
111. chemotactic factor of mast cell
112. What chemotactic factor release mast cells?
113. chemotactic factor of eosinophils \*
114. chemotactic factor of T lymphocytes
115. chemotactic factor of B lymphocyte
116. chemotactic factor of basophils
117. chemotactic factor of mast cell
118. What chemotactic factors release mast cells?
     1. chemotactic factor of T lymphocytes
     2. chemotactic factor of B lymphocyte
     3. chemotactic factor of basophils
     4. chemotactic factor of neutrophils \*
     5. chemotactic factor of eosinophils \*
119. What structure is affected in the type III allergic reactions?
120. endothelial basement membrane\*
121. myocardium
122. liver
123. smooth muscles
124. brain
125. What factor causes venous hyperemia in inflammatory focus?
126. accumulation of transudate
127. marginalization of leucocytes to microvessels \*
128. thrombosis in the arterioles
129. venule dilation
130. arteriolar dilation
131. What factor causes venous hyperemia in inflammatory focus?
132. thrombosis in the arterioles
133. narrowing of arterioles
134. accumulation of exudate \*
135. arteriolar dilation
136. dilation of venules
137. What factor causes venous hyperemia in inflammatory focus?
138. thrombosis in the venules \*
139. accumulation of transudate
140. narrowing of arterioles
141. arteriolar dilation
142. dilation of venules
143. What factors cause arterial hyperemia in the inflammatory focus?
144. vessel breakdown
145. release of mediators with vasoconstrictor effect
146. release of mediators with vasodilator effect \*
147. acidosis in inflammatory focus \*
148. thrombogenesis in inflammatory focus
149. What factors cause venous hyperemia in inflammatory focus?
150. thrombosis in the venules \*
151. narrowing of arterioles
152. accumulation of exudate \*
153. arteriolar dilation
154. dilation of venules
155. What factors cause venous hyperemia in inflammatory focus on frog’s tongue?
156. thrombosis in the venules \*
157. leucocytes margination at the level of microvessels \*
158. accumulation of transudate
159. arteriolar dilation
160. dilation of venules
161. What factors provoke pulmonary edema?
162. hypoproteinemia \*
163. pulmonary hypoperfusion
164. right-left vascular shunt
165. bronchial asthma
166. larynx stenosis
167. What factors provoke pulmonary edema?
168. left ventricle insufficiency \*
169. pulmonary hypoperfusion
170. right-left vascular shunt
171. bronchial asthma
172. larynx stenosis
173. What factors provoke pulmonary edema?
174. pulmonary circuit capillary stasis \*
175. pulmonary circuit capillary hyperpermeability \*
176. pulmonary hypoperfusion
177. bronchial asthma
178. larynx stenosis
179. What factors provoke pulmonary edema?
180. lung lymphatic stasis \*
181. protein accumulation in the pulmonary interstitium \*
182. pulmonary hypoperfusion
183. bronchial asthma
184. larynx stenosis
185. What general effect exercise interleukin 1 (IL-1)?
186. pyrogenic action \*
187. anti-inflammatory action
188. anabolic action
189. chemiotactic action\*
190. erythropoietic action
191. What hormone has direct anti-inflammatory action?
192. ACTH
193. Cortisol \*
194. aldosterone
195. thyroxine
196. testosterone
197. What inflammatory mediator comes from eosinophilic leukocytes?
198. cationic proteins \*
199. histamine
200. antiparasitic antibodies
201. lysozyme
202. hyaluronidase
203. What inflammatory mediator comes from eosinophilic leukocytes?
204. histamine
205. perforine \*
206. antiparasitic antibodies
207. lysozyme
208. hyaluronidase
209. What inflammatory mediator comes from eosinophilic?
210. cationic proteins \*
211. perforine \*
212. antiparasitic antibodies
213. lysozyme
214. hyaluronidase
215. What inflammatory mediator comes from lymphocytes?
216. mitogenic factor for lymphocytes \*
217. complement activating factor
218. lysosomal enzymes
219. integrins
220. immunoglobulins
221. What inflammatory mediator comes from lymphocytes?
222. complement activating factor
223. lymphocytotoxin \*
224. lysosomal enzymes
225. integrins
226. immunoglobulins
227. What inflammatory mediator comes from lymphocytes?
228. complement activating factor
229. chemotactic factor of lymphocytes \*
230. lysosomal enzymes
231. integrins
232. immunoglobulins
233. What inflammatory mediator comes from platelets?
234. serotonin \*
235. lysosomal enzymes
236. antiparasitic antibodies
237. lysozyme
238. hyaluronidase
239. What inflammatory mediator derives from neutrophil leukocytes?
240. histamine
241. tryptase
242. prostaglandins
243. cationic proteins \*
244. antimicrobial antibodies
245. What inflammatory mediator derives from neutrophil leukocytes?
246. reactive oxygen species \*
247. tryptase
248. prostaglandins
249. cationic proteins \*
250. antimicrobial antibodies
251. What inflammatory mediator derives from neutrophil leukocytes?
252. lysosomal enzymes \*
253. histamine
254. tryptase
255. histaminase
256. antimicrobial antibodies
257. What inflammatory mediator derives from neutrophil leukocytes?
258. halogenated compounds \*
259. tryptase
260. prostaglandins
261. cationic proteins \*
262. antimicrobial antibodies
263. What inflammatory mediator derives from neutrophil leukocytes?
264. histamine
265. tryptase
266. histaminase
267. reactive oxygen species \*
268. lysosomal enzymes \*
269. What inflammatory mediator derives from neutrophil leukocytes?
270. lysosomal enzymes \*
271. tryptase
272. prostaglandins
273. cationic proteins \*
274. antimicrobial antibodies
275. What is biological importance of eosinophils emigration into inflammatory focus?
276. release of cationic proteins and perforine \*
277. release of free radicals and halogens
278. specific local immunity (antibody synthesis)
279. realize histaminase and breakdown of histamine \*
280. phagocytosis of microorganisms
281. What is biological importance of eosinophils emigration into inflammatory focus?
282. release of free radicals and halogens
283. specific local immunity (antibody synthesis)
284. phagocytosis of dead cells and cellular compounds
285. phagocytosis of microorganisms
286. realize histaminase and breakdown of histamine \*
287. What is biological importance of lymphocytes emigration into inflammatory focus?
288. generation of bactericides (cationic proteins, perforine)
289. generation of bactericides oxygen dependent (free radicals, halogens)
290. granuloma formation \*
291. phagocytosis of microorganisms
292. phagocytosis of dead cells and cellular compounds
293. What is biological importance of lymphocytes emigration into inflammatory focus?
294. generation of bactericides (cationic proteins, perforine)
295. generation of bactericides oxygen dependent (free radicals, halogens)
296. granuloma formation \*
297. phagocytosis of microorganisms
298. specific local immunity (antibody synthesis) \*
299. What is biological importance of lymphocytes emigration into inflammatory focus?
300. generation of bactericides (cationic proteins, perforine)
301. generation of bactericides oxygen dependent (free radicals, halogens)
302. specific local immunity (antibody synthesis, cellular immune reactions) \*
303. phagocytosis of microorganisms
304. phagocytosis of dead cells and cellular compounds
305. What is biological importance of monocytes emigration into inflammatory focus?
306. generation of bactericides (cationic proteins, perforine)
307. collagen synthesis
308. specific local immunity (antibody synthesis)
309. phagocytosis of microorganisms
310. phagocytosis of dead cells and cellular compounds \*
311. What is biological importance of neutrophil emigration into inflammatory focus?
312. release of cationic proteins and perforine
313. phagocytosis of microorganisms \*
314. specific local immunity (antibody synthesis)
315. release of free radicals and halogens \*
316. intake and degradation of histamine
317. How is manifested clinic anaphylactic?
318. Bronchodilation
319. Bronchospasm \*
320. Hypersecretion of bronchus mucous \*
321. Hyposecretion of bronchus mucous
322. Arterial hypertension
323. How is manifested clinic anaphylactic?
324. Bronchodilation
325. Arterial hypertension
326. Hypersecretion of bronchus mucous \*
327. Hyposecretion of bronchus mucous
328. Arterial collapse \*
329. What is mechanism of bronchial spasm in anaphylactic shock?
330. Direct action of immune complex
331. Action of histamine \*
332. Action of adrenaline
333. Action of gamma aminobutyric
334. Action of glucocorticoids
335. What is mechanism of bronchial spasm in anaphylactic shock?
336. Direct action of immune complex
337. Action of leukotrienes \*
338. Action of adrenaline
339. Action of gamma aminobutyric
340. Action of glucocorticoids
341. What is microcirculatory change in arterial hyperemia?
342. dilation of arterioles, capillaries and venules \*
343. decreased number of functional capillaries
344. decreased blood velocity
345. decreased hydrostatic pressure
346. dilation of arterioles, narrowing of capillaries and venules
347. What is microcirculatory change in arterial hyperemia?
348. dilation of arterioles, narrowing of capillaries and venules
349. decreased number of functional capillaries
350. decreased blood velocity
351. increased blood velocity \*
352. decreased function of lymphatic vessels
353. What is microcirculatory change in prestasis?
354. accelerated movement
355. pulsatile movement \*
356. turbulent movement
357. diminished inflow
358. centripetal movement
359. What is microcirculatory change in prestasis?
360. accelerated movement
361. pendulatory movement \*
362. turbulent movement
363. diminished inflow
364. centripetal movement
365. What is microcirculatory change in venous hyperemia?
366. increased blood velocity
367. dilation of venules \*
368. increased venous outflow
369. narrowing of arterioles
370. narrowing of venules
371. What is microcirculatory change in venous hyperemia?
372. increased blood velocity
373. narrowing of venules
374. diminished venous outflow \*
375. narrowing of arterioles
376. narrowing of venules
377. What is one of the general manifestations of inflammatory reaction of the body?
378. hypothermia
379. leukocytopenia
380. decreasing of erythrocyte sedimentation rate
381. acceleration of erythrocyte sedimentation rate \*
382. increasing anabolism
383. What is one of the pathogenic factors of cardiac edemas?
384. excessive overfilling of capillaries with blood \*
385. arterial hypotension
386. hypoxia
387. hypersecretion of renin \*
388. hypersecretion of atrial natriuretic peptide
389. What is one of the pathogenic factors of cardiac edemas?
390. hypernatremia \*
391. hypokalemia
392. hyponatremia
393. hyposecretion of vasopressin
394. hypersecretion of atrial natriuretic peptide
395. What is one of the pathogenic factors of cardiac edemas?
396. increases hydrostatic pressure in the capillaries \*
397. arterial hypotension
398. hyponatremia
399. hyposecretion of vasopressin
400. hypersecretion of atrial natriuretic peptide
401. What is one of the pathogenic factors of cardiac edemas?
402. hypoonchia of blood plasma \*
403. arterial hypotension
404. hyposecretion of renin
405. hypersecretion of renin \*
406. hypersecretion of atrial natriuretic peptide
407. What is pathogenesis of inflammatory venous hyperemia?
408. vessels sclerosis in inflammatory focus
409. spherization of endothelial cells \*
410. platelets aggregation under the action of prostacyclin
411. arteriolar thrombosis in inflammatory focus
412. platelets aggregation under the action of thromboxane \*
413. What is pathogenesis of inflammatory venous hyperemia?
414. vessels sclerosis in inflammatory focus
415. adhesion of leukocytes to the capillary wall \*
416. platelets aggregation under the action of prostacyclin
417. arteriolar thrombosis in inflammatory focus
418. hemoconcentration in microvessels of inflammatory focus \*
419. What is pathogenesis of inflammatory venous hyperemia?
420. sclerosis of vessels in inflammatory focus
421. edema and increased interstitial pressure in the inflammatory focus \*
422. platelets aggregation under the action of prostacyclin
423. arteriolar thrombosis in inflammatory focus
424. adhesion of leukocytes to the capillary wall \*
425. What is pathogenesis of inflammatory venous hyperemia?
426. sclerosis of vessels in inflammatory focus
427. hemoconcentration in microvessels of inflammatory focus \*
428. platelets aggregation under the action of prostacyclin
429. thrombosis of arteriolar in inflammatory focus
430. edema and increased interstitial pressure in the inflammatory focus \*
431. What is pathogenesis of vascular hyperpermeability in inflammation?
432. thromboxanes action
433. prostacyclin action
434. glucocorticoid hormones action
435. action of active factors of C3a and C5a complement \*
436. histamine action \*
437. What is pathogenesis of vascular hyperpermeability in inflammation?
438. thromboxanes action
439. prostacyclin action
440. glucocorticoid hormones action
441. action of active factors of C3a and C5a complement \*
442. bradykinin action \*
443. What is sequence of vascular reactions in inflammatory focus?
444. arterial hyperemia→ venous hyperemia →ischemia → stasis
445. hyperemia → arterial hyperemia → ischemia → stasis
446. venous stasis → venous hyperemia → arterial hyperemia → ischemia
447. ischemia → arterial hyperemia → venous hyperemia → stasis \*
448. ischemia → venous hyperemia → arterial hyperemia → stasis
449. What is specific for inflammatory arterial hyperemia?
450. persistent character \*
451. transient character
452. neuroparalytic pathogenesis
453. neurotonic pathogenesis
454. is associated with reduced vascular permeability
455. What is specific for inflammatory arterial hyperemia?
456. transient character
457. neuroparalytic pathogenesis
458. neurotonic pathogenesis
459. is associated with increased vascular permeability \*
460. is associated with reduced vascular permeability
461. What is the biological effect of leukotrienes in the inflammatory focus?
462. vasodilatory action \*
463. clotting effect
464. stimulation of the platelet aggregation
465. inhibits the platelet aggregation
466. bronchoconstriction effect \*
467. What is the biological effect of leukotrienes in the inflammatory focus?
468. vasodilatory action \*
469. clotting effect
470. stimulation of the platelet aggregation
471. inhibits the platelet aggregation
472. vasoconstrictor action
473. What is the biological effect of prostacyclin in the inflammatory focus?
474. stimulation of the platelet aggregation
475. inhibits the platelet aggregation \*
476. vasoconstrictor action
477. bronchoconstriction action
478. uterotonic action
479. What is the biological effect of prostaglandins in the inflammatory focus?
480. vasodilation \*
481. vasoconstriction
482. bronchoconstriction
483. uterotonic action \*
484. arterial hypertension
485. What is the biological effect of thromboxanes in the inflammatory focus?
486. stimulates of the platelet aggregation \*
487. inhibits the platelet aggregation
488. vasodilation action
489. bronchoconstriction action
490. uterotonic action
491. What is the biological importance of venous hyperemia and inflammatory stasis?
492. leads to chronic inflammatory process
493. contributes to the localization of the inflammatory process \*
494. contributes to the parenchymal proliferation and regeneration in inflammatory focus
495. increases blood perfusion of the inflammatory tissue
496. contributes to the exudation \*
497. What is the biological importance of venous hyperemia and inflammatory stasis?
498. contributes to leukocytes migration \*
499. leads to chronic inflammatory process
500. contributes to the parenchymal proliferation and regeneration in inflammatory focus
501. increases blood perfusion of the inflammatory tissue
502. contributes to the spreading of pathogenic factor
503. What is the biological importance of venous hyperemia and inflammatory stasis?
504. leads to chronic inflammatory process
505. contributes to the exudation \*
506. contributes to the parenchymal proliferation and regeneration in inflammatory focus
507. increases blood perfusion of the inflammatory tissue
508. contributes to the spreading of pathogenic factor
509. 7What is the biological importance of venous hyperemia and inflammatory stasis?
510. leads to chronic inflammatory process
511. contributes to the localization of the inflammatory process \*
512. contributes to the parenchymal proliferation and regeneration in inflammatory focus
513. increases blood perfusion of the inflammatory tissue
514. contributes to the spreading of pathogenic factor
515. What is the biological importance of venous hyperemia and inflammatory stasis?
516. leads to chronic inflammatory process
517. contributes to the localization of the inflammatory process \*
518. contributes to the parenchymal proliferation and regeneration in inflammatory focus
519. increases blood perfusion of the inflammatory tissue
520. contributes to leukocytes migration \*
521. What is the cellular source of proliferation in the inflammatory focus?
522. parenchymal cells
523. cells formed as a result of metaplasia
524. mutant cells
525. monocytes migrated from blood \*
526. cells formed as a result dedifferentiation
527. What is the cellular source of proliferation into inflammatory focus?
528. parenchymal cells
529. cells formed as a result of metaplasia
530. mutant cells
531. resident mesenchymal cells \*
532. cells formed as a result dedifferentiation
533. What is the effect of activated Hageman factor contact?
534. chemotactic effect
535. anti-inflammatory
536. activation of kininogenic system\*
537. direct injury of the vascular wall
538. inactivation of thrombin
539. What is the effect of activated Hageman factor contact?
540. activation of anticlotting system \*
541. chemotactic effect
542. anti-inflammatory effect
543. direct injury of the vascular wall
544. inactivation of thrombin
545. What are the effects of activated Hageman factor contact?
546. chemotactic effect
547. anti-inflammatory
548. activation of kininogenic system \*
549. direct destruction of the vascular wall
550. activation of anticlotting system \*
551. What are the effects of activated Hageman factor contact?
552. chemotactic effect
553. anti-inflammatory
554. activation of kininogenic system \*
555. direct destruction of the vascular wall
556. activation of the fibrinolytic system \*
557. What are the effect of kinins in inflammation?
558. vasoconstriction
559. vasodilation \*
560. systemic hypertension
561. sensation of pain \*
562. bactericide effect
563. What are the effects of C3a and C5a in inflammatory focus?
564. vasoconstriction action
565. stimulates prostaglandin synthesis
566. chemotactic action \*
567. degranulation of mast cells \*
568. pro-clotting activity
569. What are the effects of C3a and C5a in inflammatory focus?
570. vasoconstriction action
571. stimulates prostaglandin synthesis
572. chemotactic action \*
573. vasodilatory action \*
574. pro-clotting activity
575. What is the effect of C3a and C5a in inflammatory focus?
576. permeability of blood vessels \*
577. vasoconstriction action
578. stimulates prostaglandin synthesis
579. anticlotting action
580. pro-clotting activity
581. What is the effect of C3a and C5a in inflammatory focus?
582. vasoconstriction action
583. vasodilator action \*
584. stimulates prostaglandin synthesis
585. anticoagulated action
586. pro-clotting activity
587. What is the effect of C3a and C5a in inflammatory focus?
588. vasoconstriction action
589. vasodilator action \*
590. stimulates prostaglandin synthesis
591. degranulation of mast cells \*
592. anticlotting action
593. What is the effect of kinins in inflammation?
594. vasoconstriction
595. contraction of smooth muscles of internal organs \*
596. systemic hypertension
597. sensation of pain \*
598. bactericide effect
599. What is the effect of kinins in inflammation?
600. vasodilation A\*.
601. vasoconstriction
602. uterus muscle relaxation
603. systemic hypertension
604. bactericidal effect
605. What is the effect of kinins in inflammation?
606. vasoconstriction
607. systemic hypotension \*
608. systemic hypertension
609. sensation of pain \*
610. bactericide effect
611. What is the effect of kinins in inflammation?
612. vasoconstriction
613. contraction of smooth muscles of internal organs \*
614. uterine muscle relaxation
615. systemic hypertension
616. bactericide effect
617. What is the effect of mastocyte tryptase in inflammation?
618. complement activation by the classical way
619. contributes to the formation of membrane attack complex \*
620. tryptamine splitting
621. vasodilative effect
622. vasoconstrictive effect
623. What is the effect of mastocyte tryptase in inflammation?
624. complement activation by the alternative way \*
625. complement activation by the classical way
626. tryptamine splitting
627. vasodilative effect
628. vasoconstrictive effect
629. What is the effect of mastocyte tryptase in inflammation?
630. contributes to the formation of fragments of complement C3a and C3b \*
631. activation of fragment of complement C1
632. inhibits the fragment of the complement C1q
633. tryptamine splitting
634. contributes to the formation of membrane attack complex \*
635. What is the effect of mastocyte tryptase in inflammation?
636. complement activation by the classical way
637. contributes to the formation of membrane attack complex \*
638. tryptamine splitting
639. vasodilative effect
640. complement activation by the alternative way \*
641. What is exudate pathogenesis in inflammatory focus?
642. increase of osmotic interstitial pressure \*
643. increase of oncotic pressure in interstitial space \*
644. decrease of oncotic interstitial pressure
645. increase of intracapillary oncotic pressure
646. decrease of interstitial osmotic pressure
647. What is exudate pathogenesis in inflammatory focus?
648. increase of osmotic interstitial pressure \*
649. vascular wall hyperpermeability \*
650. decrease of oncotic interstitial pressure
651. increase of intracapillary oncotic pressure
652. decrease of interstitial osmotic pressure
653. What is the exudate pathogenesis in inflammatory focus?
654. increase of osmotic interstitial pressure \*
655. increases of hydrostatic pressure in interstitial space
656. decrease of oncotic interstitial pressure
657. decrease of intracapillary oncotic pressure
658. decrease of interstitial osmotic pressure
659. What is the exudate pathogenesis in inflammatory focus?
660. hydrostatic pressure increases in capillaries \*
661. hydrostatic pressure increases in interstitial space
662. oncotic interstitial pressure decreases
663. intracapillary oncotic pressure decrease
664. interstitial osmotic pressure decreases
665. What is the exudate pathogenesis in inflammatory focus?
666. oncotic interstitial pressure increases \*
667. hydrostatic pressure increases in interstitial space
668. oncotic interstitial pressure decreases
669. intracapillary oncotic pressure decrease
670. interstitial osmotic pressure decreases
671. What is the exudate pathogenesis in inflammatory focus?
672. vascular wall hyperpermeability \*
673. hydrostatic pressure increases in interstitial space
674. oncotic interstitial pressure decreases
675. intracapillary oncotic pressure decrease
676. interstitial osmotic pressure decreases
677. What is the mechanism of leukocyte migration into the inflammatory focus?
678. action of chemotactic factors in inflammatory focus \*
679. passive migration of leukocytes through the vessel wall
680. laminar blood flow
681. vessels hyperpermeability \*
682. leukocyte adhesion to the vascular wall \*
683. What is the mechanism of leukocyte migration into the inflammatory focus?
684. hydrolytic enzymes action on the basement membrane vascular wall\*
685. passive migration of leukocytes through the vessel wall
686. laminar blood flow
687. vessels hyperpermeability \*
688. leukocyte adhesion to the vascular wall \*
689. What is the mechanism of leukocyte migration into the inflammatory focus?
690. hydrolytic enzymes action on the basement membrane vascular wall\*
691. passive migration of leukocytes through the vessel wall
692. laminar blood flow
693. expression of selectins and integrins on endotelial cells and leukocytes \*
694. leukocyte adhesion to the vascular wall \*
695. What is the required enzyme for leukotrienes synthesis?
696. phospholipase A2 and cyclooxygenase
697. cyclooxygenase and lipoxygenase
698. phospholipase A2 and lipoxygenase \*
699. lecithinase and cyclooxygenase
700. tryptase and cyclooxygenase
701. What is the required enzyme for prostaglandin synthesis?
702. phospholipase A2 and cyclooxygenase \*
703. cyclooxygenase and lipoxygenase
704. phospholipase A2 and lipoxygenase
705. lecitinase and cyclooxygenase
706. tryptase and cyclooxygenase
707. What is the specific sign of serous exudate?
708. contains until 2-3% of proteins \*
709. contains fibrinogen
710. contains fibrine
711. contains collagen fibers
712. contains elastic fibers
713. What is the specific sign of purulent exudate?
714. contains until 2-3% of proteins
715. contains fibrinogen
716. contains many polymorphonuclear leukocytes \*
717. contains many erythrocytes
718. contains many platelets
719. What is the specific sign of fibrinous exudate?
720. contains until 2-3% of proteins
721. contains fibrinogen
722. contains fibrine \*
723. contains collagen fibers
724. contains elastic fibers
725. What is the specific sign of hemorrhagic exudate?
726. contains collagen fibers
727. contains fibrine
728. contains elastic fibers
729. contains many erythrocytes \*
730. contains many platelets
731. What is the exogenous pyrogen infectious factor?
732. microbial, viral, fungal proteins \*
733. allogenic transplantation
734. hyperimmune serums
735. blood and heterogeneous blood plasma
736. heterogeneous proteins administered parenteral
737. What is the exogenous pyrogen infectious factor?
738. microbial lipopolysaccharide \*
739. allogenic transplantation
740. hyperimmune serums
741. blood and heterogeneous blood plasma
742. heterogeneous proteins administered parenteral
743. What is the exogenous pyrogen infectious factor?
744. bacterial antigens \*
745. allogenic transplantation
746. hyperimmune serums
747. blood and heterogeneous blood plasma
748. heterogeneous proteins parenteral administered
749. What is the relation between thermogenesis and thermolysis in the first period of the fever?
750. stimulation of thermogenesis and thermolysis in the same time
751. stimulation of thermogenesis and inhibition of thermolysis \*
752. inhibition of thermogenesis and activation of thermolysis
753. inhibition of thermogenesis and thermolysis in the same time
754. discordance between the activity of thermogenesis and thermolysis center
755. What mediator causes arterial inflammatory hyperemia?
756. catecholamines
757. interleukins
758. leukotrienes
759. C3a and C5a complement factors \*
760. C5-C9complement factors
761. What mediator causes arterial inflammatory hyperemia?
762. catecholamines
763. interleukins
764. leukotrienes
765. C3a and C5a complement factors \*
766. Bradykinin \*
767. What mediator causes arterial inflammatory hyperemia?
768. Histamine \*
769. catecholamines
770. interleukins
771. leukotrienes
772. C5-C9 complement factors
773. What mediator causes arterial inflammatory hyperemia?
774. catecholamines
775. interleukins
776. leukotrienes
777. C5-C9 complement factors
778. prostaglandins PGE2 \*
779. What mediator is pre-synthetized by mast cells?
780. Histamine \*
781. interleukins
782. leukotrienes
783. interferons
784. prostaglandins
785. What mediators are synthesized by mast cells via lipoxygenase pathway?
786. histamine
787. chemotactic factors
788. platelet-activating factor
789. leukotrienes \*
790. prostaglandins
791. What mediators are synthesized by mast cells via cyclooxygenase way?
792. histamine
793. chemotactic factors
794. platelet-activating factor
795. leukotrienes
796. prostaglandins \*
797. What mediators cause arterial inflammatory hyperemia?
798. C3a and C5a complement factors\*
799. interleukins
800. leukotrienes
801. C3b complement factors
802. Bradykinin \*
803. What mediators cause arterial inflammatory hyperemia?
804. prostaglandins PGE2 \*
805. interleukins
806. leukotrienes
807. C3b complement factors
808. Bradykinin \*
809. What mediators cause arterial inflammatory hyperemia?
810. Histamine \*
811. interleukins
812. leukotrienes
813. C3b complement factors
814. Bradykinin \*
815. What metabolic disorders can be attested in the mouse exposed to normobaric hypoxia without hypercapnia?
816. metabolic acidosis \*
817. metabolic alkalosis
818. respiratory acidosis
819. hypercapnia
820. hypoxemia \*
821. What metabolic disorders can be attested in the mouse exposed to normobaric hypoxia without hypercapnia?
822. metabolic acidosis \*
823. metabolic alkalosis
824. respiratory acidosis
825. hypercapnia
826. respiratory alkalosis \*
827. What metabolic disorders can be attested in the mouse exposed to normobaric hypoxia without hypercapnia?
828. respiratory alkalosis \*
829. metabolic alkalosis
830. respiratory acidosis
831. hypercapnia
832. hypoxemia \*
833. What microcirculatory disorders lead to stasis into inflammatory focus?
834. arterial hyperemia
835. venous hyperemia \*
836. ischemia
837. embolism
838. thrombosis \*
839. What oxygen-dependent bacteria factor is generated by neutrophil leukocytes?
840. superoxide anion O-2 \*
841. ozone O3
842. Br-
843. Ag
844. HCl
845. What oxygen -dependent bacteria factor is generated by neutrophil leukocytes?
846. hydrogen peroxide H2O2 \*
847. ozone O3
848. Br-
849. Ag
850. HCl
851. What oxygen-dependent bacteria factor is generated by neutrophil leukocytes?
852. hydroxyl radical OH- \*
853. ozone O3
854. Br-
855. Ag
856. HCl
857. What oxygen-dependent bacteria factor is generated by neutrophil leukocytes?
858. halogens OCl- \*
859. ozone O3
860. Br-
861. Ag
862. HCl
863. What oxygen-dependent bacteria factors are generated by neutrophil leukocytes?
864. halogens OCl- \*
865. ozone O3
866. Br-
867. Ag
868. superoxide anion O-2\*
869. What oxygen-dependent bacteria factors are generated by neutrophil leukocytes?
870. halogens OCl-\*
871. ozone O3
872. Br-
873. Ag
874. hydrogen peroxide H2O2 \*
875. What oxygen-dependent bacteria factors are generated by neutrophil leukocytes?
876. halogens OCl-\*
877. ozone O3
878. Br-
879. Ag
880. super-oxide anion\*
881. What is the physiological importance of sodium ions?
882. participation in the clotting of blood
883. the maintenance of extracellular fluid osmolarity \*
884. participation in the formation of resting potential of excitable cells
885. the maintenance of intracellular fluid osmolarity
886. the transmembrane transport of organic substances
887. What does represent hypernatremia?
888. blood plasma sodium concentration above 180 mecv / l
889. blood plasma sodium concentration less than 135 mecv / l
890. blood plasma sodium concentration greater than 150 mecv / l\*
891. blood plasma sodium concentration more than 120 mecv / l
892. extracellular fluid sodium concentration above 150 mecv / l
893. What are the causes of hypernatremia?
894. dehydration of the body \*
895. chronic adrenal insufficiency
896. primary aldosteronism \*
897. gaseous alkalosis
898. metabolic alkalosis
899. What is the basic pathogenetic mechanisms of hypernatremia?
900. excessive intake of water
901. redistribution of sodium ions between intra-and extracellular areas
902. excessive loss of potassium
903. excessive loose of body water\*
904. increased synthesis of renin in the kidney \*
905. What are the consequences of absolute hypernatremia?
906. increase extracellular fluid volume \*
907. intracellular fluid volume growth
908. reduction of intracellular fluid volume \*
909. reduction increasing the intravascular fluid volume
910. reduction of extracellular fluid volume
911. What is the normal concentration of Na+ ions in the blood?
912. less than 100 - mEq/L
913. 100 -125 mEq/L
914. 135 - 145 mEq/L \*
915. 140 -160 mEq/L
916. above 300 mEq/L
917. From what value of the concentration of Na+ ions in the blood there is considered hypernatremia?
918. above 100 mEq/L
919. above 152 mEq/L \*
920. above 140 mEq/L
921. above 132 mEq/L
922. above 300 mEq/L
923. From what value of the concentration of Na+ ions in the blood there is considered hyponatremia?
924. less than 100 mEq/L
925. less than 152 mEq/L
926. less than 140 mEq/L
927. less than 130 mEq/L \*
928. less than 300 mEq/L
929. **What are the main pathogenetic mechanisms of hypernatremia?**
930. dehydration with excessive loss of body water \*
931. release of sodium ions from damaged cells
932. decreased synthesis of renin in the kidneys
933. hyperhydration with excessive gain of body water
934. increased synthesis of renin in the kidneys \*
935. **What are the main pathogenetic mechanisms of hyponatremia?**
936. excessive gain of body fluids \*
937. excessive loss of body fluids
938. deficiency of mineralocorticoids \*
939. excessive release of sodium from damaged cells
940. hypersecretion of mineralocorticoids
941. **What is the normal concentration of K+ ions in the blood?**
942. 5,5 - 6,5 mEq/L
943. 3,5 – 5,5 mEq/L \*
944. 2,5 – 3,5 mEq/L
945. less than 2,5 mEq/L
946. less than 1,5 mEq/L
947. **From what value of K+ ions concentration in the blood there is considered hyperkalemia?**
948. above 5,5 mEq/L \*
949. above 4,5 mEq/L
950. above 3,5 mEq/L
951. above 7,5 mEq/L
952. above 2,5 mEq/L
953. **From what value of K+ ions concentration in the blood there is considered hypokalemia?**
954. less than 5,5 mEq/L
955. less than 4,5 mEq/L
956. less than 3,5 mEq/L \*
957. less than 2,5 mEq/L
958. less than 7,5 mEq/L
959. **What is the physiological role of potassium in the body?**
960. maintains the plasmatic osmotic pressure
961. maintains the plasmatic oncotic pressure
962. ensures the active membrane potential in the excitable cells
963. ensures the resting membrane potential in the excitable cells \*
964. maintains the threshold potential in the excitable cells
965. **In what disorders can be found hyperkalemia?**
966. hyperhydration with gain of body fluids
967. hypersecretion of aldosterone
968. enhanced catabolism of tissue proteins \*
969. increased plasma renin concentration
970. decreased plasma renin concentration \*
971. **What can be the causes of hypokalemia?**
972. deficiency of renin in the blood
973. hypersecretion of glucocorticoids \*
974. excessive renin in the blood \*
975. deficiency of glucocorticoids
976. overhydration of the body
977. **What are the pathogenetic mechanisms that contribute to development of hypokalemia?**
978. disturbances of glomerular filtration and water retention in the body
979. excessive loss of potassium in the kidneys in hypersecretion of aldosterone \*
980. excessive loss of potassium in the kidneys in hyposecretion of aldosterone
981. deficiency of glucocorticoids
982. deficiency of vasopressin
983. **What is the normal concentration of Ca++ ions in the blood?**
984. 1,5 – 2,5 mmol/L
985. 2,1 – 2,6 mmol/L \*
986. 4,5 – 5,5 mmol/L
987. less than 1,0 mmol/L
988. more than 3,5 mmol/L
989. **From what value of the concentration of Ca++ ions in the blood there is considered hypercalcemia?**
990. above 2,6 mmol/L \*
991. above 3,5 mmol/L
992. above 1,6 mmol/L
993. above 2,0 mmol/L
994. above 7,0 mmol/L
995. **From what value of the concentration of Ca++ ions in the blood there is considered hypocalcemia?**
996. less than 5,3 mmol/L
997. less than 0,5 mmol/L
998. less than 2,1 mmol/L \*
999. less than 1,0 mmol/L
1000. less than 7,0 mmol/L
1001. **What is the physiological role of Ca++ ions in the body?**
1002. acts as intracellular second messenger \*
1003. maintains plasma osmotic pressure
1004. provides electrical charge to plasma proteins
1005. participates in protein synthesis
1006. maintains resting membrane potential of excitable cells
1007. **What are the main mechanisms which maintain the calcium homeostasis?**
1008. redistribution of Ca++ ions between intra- and extracellular compartment of the body
1009. removing of calcium salts with bile in the gastrointestinal tract
1010. incorporation and mobilization from bone matrix \*
1011. incorporation and mobilization from teeth matrix
1012. reabsorption of calcium in the proximal renal tubules \*
1013. **What are the causes of hypercalcemia?**
1014. hypersecretion of parathyroid hormone \*
1015. hereditary defective of calcium-dependent receptors (for parathyroid hormone)
1016. hyposecretion of calcitonin \*
1017. deficiency of vitamin D
1018. hyposecretion of parathyroid hormone
1019. **What are the causes of hypercalcemia?**
1020. hypersecretion of mineralocorticoids
1021. hyposecretion of calcitonin
1022. excessive vitamin D \*
1023. deficient vitamin D
1024. deficiency of mineralocorticoids
1025. **What are the main pathogenetic mechanisms of hypercalcemia?**
1026. redistribution between intra- and extracellular compartment
1027. increased mobilization from teeth matrix
1028. increased mobilization from bone matrix \*
1029. reduced renal reabsorption
1030. reduced renal secretion \*
1031. **What are the clinical manifestations of hypercalcemia?**
1032. neuronal inhibition \*
1033. neuronal excitation
1034. muscle hypertonia
1035. hyperreflexia
1036. muscle paralysis \*
1037. **What are the causes of hypocalcemia?**
1038. hypersecretion of calcitonin \*
1039. hypersecretion of parathyroid hormone
1040. hyposecretion of calcitonin
1041. Hyperphosphatemia \*
1042. hypophosphatemia
1043. **What are the main pathophysiological mechanisms of hypocalcemia?**
1044. deficient mobilization from the bone in deficiency of PTH \*
1045. deficient mobilization from the bone in excessive PTH
1046. decreased incorporation in the bone in hyposecretion of calcitonin
1047. enhanced calcium secretion in the proximal renal tubes \*
1048. enhanced calcium reabsorption in the proximal renal tubes
1049. What are the clinical manifestations of severe hypernatremia?
1050. Thirsty \*
1051. bradycardia
1052. haemoconcentration
1053. increased blood coagulability
1054. tachycardia \*
1055. What does represent hyponatremia?
1056. decrease of blood plasma sodium concentration below 135 mecv / l \*
1057. reduction of extracellular fluid sodium concentration below 135 mecv / l,
1058. reduce of sodium concentration of blood plasma below 120 mecv / l,
1059. increase in blood sodium concentration more than 120 mecv / l
1060. decrease in blood plasma sodium concentration below 100 mecv / l
1061. What are the causes absolute hyponatremia?
1062. increased excretion of АDH
1063. profuse diarrhea \*
1064. excessive intake of liquids
1065. chronic adrenal insufficiency \*
1066. synthesis of biologically active renin
1067. What is the basic pathogenetic mechanisms of hyponatremia?
1068. hyper-hydration of the body \*
1069. excessive loss of potassium from the body
1070. excessive loss of sodium in the body \*
1071. cellular dehydration
1072. increase the intracellular fluid osmolality
1073. What is the physiological importance of potassium?
1074. Formation of action potential of excitable cells
1075. the maintenance of intracellular fluid osmolarity \*
1076. the maintenance of extracellular fluid osmolarity
1077. participate in protein synthesis \*
1078. maintains sympathetic nervous system activity
1079. What is the physiological importance of potassium?
1080. formation of resting potential of excitable cells \*
1081. the maintenance of intracellular fluid osmolarity \*
1082. the maintenance of extracellular fluid osmolarity
1083. participate in lipid synthesis
1084. maintains sympathetic nervous system activity
1085. What is the physiological importance of potassium?
1086. participate in lipid synthesis
1087. the maintenance of intracellular fluid osmolarity \*
1088. the maintenance of extracellular fluid osmolarity
1089. participate in protein synthesis \*
1090. maintains sympathetic nervous system activity
1091. What does represent hyperkalemia?
1092. increased concentration of potassium in blood plasma over 5.5 mecv / l \*
1093. increased concentration of potassium in blood plasma for more than 7 mecv/l
1094. increased concentration of potassium extracellular fluid more than 5 mecv / l
1095. increased potassium ion concentration in serum of more than 7 mecv / l
1096. increased concentration of potassium ions more than 3,5 mecv/l
1097. What are the causes of absolute hyperkalemia?
1098. Increase in plasma renin concentration
1099. primary hyperaldosteronism
1100. metabolic alkalosis
1101. treatment with insulin
1102. decrease in plasma renin concentration \*
1103. What are the causes of absolute hyperkalemia?
1104. Increase in plasma renin concentration
1105. metabolic acidosis \*
1106. metabolic alkalosis
1107. treatment with insulin
1108. decrease in plasma renin concentration \*
1109. What does represent hypokalaemia?
1110. reduced blood plasma level of potassium ions below 5 mecv / l,
1111. reduced blood plasma level of potassium ions below 3.5 mecv / l \*
1112. reduced blood plasma level of potassium ions below 5 mecv / l
1113. reduced blood plasma level of potassium ions below 6.0 mecv / l
1114. reduced blood plasma level of potassium ions below 2 mecv / l
1115. What are the causes of hypokalemia?
1116. treatment with glucocorticoids \*
1117. treatment of insulin \*
1118. metabolic acidosis
1119. respiratory acidosis
1120. renin hyposecretion
1121. What are the pathogenetic mechanisms that contribute to installation of hypokalemia?
1122. disturbances of glomerular filtration and water retention in the body
1123. metabolic alkalosis \*
1124. increased secretion of aldosterone \*
1125. reduced secretion of aldosterone
1126. metabolic acidosis
1127. What is the cause of hypokalemia in chronic liver affection?
1128. disruption of protein synthesis
1129. deregulation of glycolysis
1130. aldosterone metabolism disorder \*
1131. impaired glycogenolysis
1132. disturbances in the metabolism of glucocorticoids
1133. What are the physiological importance of calcium ions?
1134. participate in blood coagulation processes \*
1135. maintain body fluid osmolality
1136. represent secondary messengers on the cells \*
1137. represent primary messenger of the cells
1138. maintain cellular fluid osmolality
1139. What is the role of calcium in regulation of excitability in excitable cells?
1140. activate the sodium pump
1141. inactivate the sodium pump \*
1142. reduce the gradient of concentration of extra-and intracellular sodium \*
1143. increase the gradient of concentration of extra-and intracellular sodium
1144. activate the potasium pump
1145. Which intracellular enzymes are activated by calcium?
1146. endonucleases \*
1147. cytochrome oxidase
1148. glutation
1149. AТP-ase \*
1150. catalase
1151. What are the most important mechanisms that regulate homeostasis of calcium ions?
1152. parathyroid hormone increases bone resorption \*
1153. parathyroid hormone reduces bone resorption
1154. thyrocalcitonin increase calcium absorption from bowels
1155. thyrocalcitonin increase calcium fixation in bones \*
1156. phosphates increase calcium fixation in bones
1157. What are the causes of secondary hypercalcemia?
1158. an excessive calcium intake \*
1159. metabolic alkalosis
1160. hypervitaminosis with vitamin D \*
1161. adrenal insufficiency
1162. respiratory alkalosis
1163. What are the causes of secondary hypercalcemia?
1164. metabolic acidosis \*
1165. metabolic alkalosis
1166. hypervitaminosis with vitamin D \*
1167. adrenal insufficiency
1168. respiratory alkalosis
1169. What are the clinical manifestations of hypercalcemia?
1170. constipation \*
1171. diarrhea
1172. increased muscle tone
1173. muscle paresis \*
1174. hyperreflexia
1175. What are the causes of hypocalcemia?
1176. parathyroid gland hypofunction \*
1177. parathyroid glands hyperfunction
1178. hyperfunction of thyrocalcitonin \*
1179. renal failure \*
1180. increased sensitivity of bone tissue to parathyroid hormone
1181. What are the causes of hypocalcemia?
1182. decreased sensitivity of bone tissue to parathyroid hormone \*
1183. parathyroid glands hyperfunction
1184. hyperfunction of thyrocalcitonin \*
1185. renal failure \*
1186. increased sensitivity of bone tissue to parathyroid hormone
1187. What severe complication causes hypocalcemia in children?
1188. acute renal failure
1189. hypoosmotic coma
1190. spasmophylia
1191. tonic convulsions and asphyxia \*
1192. myasthenia
1193. What is the total water volume in healthy human body?
1194. 17 l
1195. 40% of body weight
1196. 65% of body weight \*
1197. 80% of body weight
1198. 45 l \*
1199. What are the peculiarities of body dehydration caused by water deficiency?
1200. Hypo-osmolarity, hypo-proteinemia, hyponatremia
1201. Hyper-osmolarity, hyperproteinaemia, hypernatremia \*
1202. Iso-osmolarity, hyperproteinaemia, hypernatremia
1203. Hyperosmolarity, hyperproteinaemia, hyponatremia
1204. Hypo-osmolarity, hyperproteinaemia, hyponatremia
1205. What are the peculiarities of body dehydration caused by excessive sweating?
1206. Hypo-osmolarity, hypoproteinaemia, hyponatremia
1207. Hyperosmolarity, hyperproteinaemia, hypernatremia \*
1208. Iso-osmolarity, hyperproteinaemia, hypernatremia
1209. Hyperosmolarity, hyperproteinaemia, hyponatremia
1210. Hypoosmolality, hyperproteinaemia, hyponatremia
1211. What are the peculiarities of body dehydration caused by incoercible vomiting?
1212. Hyperosmolarity, hypochloraemia acidosis.
1213. Hypoosmolarity, hypochloraemia acidosis
1214. Hyperosmolarity, hypochloraemia, alkalosis \*
1215. Hypoosmolarity, hypochloraemia, alkalosis.
1216. Iso-osmolarity, hyperchloremia, alkalosis
1217. What are the peculiarities of body dehydration caused by diarrhea?
1218. Hypoosmolarity, hyponatremia, acidosis
1219. Iso-osmolarity, hyponatremia, acidosis \*
1220. Hypoosmolarity, hyponatremia, alkalosis
1221. Hyperosmolarity, hypernatremia, alkalosis
1222. Iso-osmolarity, hypernatremia, alkalosis
1223. What are the peculiarities of body dehydration in the II-III stages of burning?
1224. Iso-osmolarity, hyponatremia, hypoproteinemia, hypopotassemia
1225. Hypoosmolarity, hyponatremia, hypoproteinemia, hyperpotassiemia
1226. Iso-osmolarity, hyponatremia, hypoproteinemia, hyperpotassiemia
1227. Hyperosmolarity, hyponatremia, hypoproteinemia, hyperpotassiemia \*
1228. Hyperosmolarity, hypernatremia, hypoproteinemia, hyperpotassiemia
1229. What are the peculiarities of body dehydration in acute hemorrhage within 2 hours?
1230. Hypoosmolarity, hyponatremia, hypovolemia
1231. Hypoosmolarity, hypernatremia, hypovolemia
1232. Hyperosmolarity, hypernatremia, hypovolemia \*
1233. Iso-osmolarity, normal values of proteins, hypovolemia
1234. Iso-osmolarity, normal values of proteins, normal values of body water
1235. What changes are attested in blood circulation during intravascular dehydration?
1236. increased peripheral resistance \*
1237. reduced peripheral resistance
1238. worsened rheological blood capacity \*
1239. improving rheological blood capacity
1240. heart hypofunction
1241. What changes are attested in blood circulation during intravascular dehydration?
1242. increased peripheral resistance \*
1243. reduced peripheral resistance
1244. improving rheological blood capacity
1245. decreased of blood flow to the organs \*
1246. heart hypofunction
1247. What are the compensatory reactions of intravascular dehydration?
1248. hypersecretion of aldosterone \*
1249. hypersecretion of vasopressin \*
1250. natriuretic hormone of hypersecretion
1251. hyposecretion of aldosterone
1252. hypersecretion of vasopressin
1253. What are the effects of aldosterone in intravascular dehydration?
1254. increased renal sodium reabsorption \*
1255. increased renal reabsorption of potassium
1256. decreased voluntary water reabsorption in the kidney
1257. hyperosmolarity of blood plasma \*
1258. polyuria
1259. What are the effects of vasopressin in intravascular dehydration?
1260. increase voluntary water reabsorption in the kidney \*
1261. increase mandatory water reabsorption in kidney
1262. polyuria
1263. oliguria \*
1264. hypoproteinemia
1265. What are the consequences of intravascular dehydration?
1266. decrease blood flow in organs \*
1267. aggregation of blood cells \*
1268. increasing of blood coagulation \*
1269. inhibition of vasopressin secretion
1270. inhibition of aldosterone secretion
1271. What are the consequences of intravascular dehydration?
1272. aggregation of blood cells \*
1273. increasing of blood coagulation \*
1274. inhibition of vasopressin secretion
1275. inhibition of aldosterone secretion
1276. decreasing of blood coagulation
1277. What are the events through drinking water, food, liquid and bowel irrigation?
1278. hypervolemia \*
1279. hyponatremia \*
1280. hypersecretion of aldosterone
1281. hypovolemia
1282. hypernatremia
1283. What are the events by infusion of isotonic NaCl solution?
1284. hypervolemia \*
1285. increased oncotic pressure
1286. izoosmolarity \*
1287. hypovolemia
1288. edema \*
1289. What are the compensatory reactions in the intravascular hyper-hydration?
1290. aldosterone hyposecretion \*
1291. vasopressin hyposecretion \*
1292. decreased glomerular filtration
1293. activation of renin-angiotensin-aldosterone system
1294. oliguria
1295. What are the compensatory reactions in the intravascular hyper-hydration?
1296. aldosterone hyposecretion \*
1297. vasopressin hypersecretion
1298. increased glomerular filtration \*
1299. activation of renin-angiotensin-aldosterone system
1300. oliguria
1301. Which factors increase the capillary hydrostatic pressure?
1302. reduce blood outflow with keeping the inflow \*
1303. arterial hyperaemia \*
1304. blood stasis
1305. capillary stasis
1306. venous hyperaemia \*
1307. Which factors that increase the oncotic pressure of blood?
1308. Hypoproteinemia \*
1309. hyperproteinaemia
1310. hyponatremia
1311. hypernatremia
1312. dehydration
1313. What conditions are associated with decrease of blood oncotic pressure?
1314. hypoalbuminemia \*
1315. dehydration
1316. proteinuria \*
1317. polycythaemia
1318. hyperalbuminemia
1319. Which factors that increase the oncotic pressure of interstitial fluid?
1320. increase of capillary permeability \*
1321. abundant filtration of plasma proteins \*
1322. hyperhydration of the interstitial space
1323. proteolysis with formation of amino acids
1324. decreased of capillary permeability
1325. What is edema?
1326. accumulation of fluid in the intracellular space \*
1327. accumulation of fluid in the interstitial space
1328. accumulation of transudate in serous cavities
1329. accumulation of effusion in serous cavities
1330. accumulation of fluid in the cerebral ventricles
1331. What is the cellular sweeling?
1332. accumulation of fluid in the intracellular space \*
1333. accumulation of fluid in the interstitial space
1334. accumulation of transudate in serous cavities
1335. accumulation of effusion in serous cavities
1336. accumulation of fluid in the cerebral ventricles
1337. Which factors that increase the permeability of vessels?
1338. Hypoxia \*
1339. acidosis \*
1340. hyperoxia
1341. alkalosis
1342. hyaluronidase \*
1343. What are the main pathogenetic link of cardiac edema?
1344. hyperpermeability of biological membranes
1345. hyperproteinaemia
1346. venous hyperaemia \*
1347. hyponatremia
1348. hypoproteinaemia \*
1349. What are the main pathogenetic link of nephritic edema?
1350. hypoproteinemia
1351. activation of the renin-angiotensin system \*
1352. venous hyperaemia
1353. hyponatremia
1354. hyperazotemia
1355. What are the main pathogenetic link of nephrotic edema?
1356. activation of the renin-angiotensin system
1357. hypoalbuminemia \*
1358. hyponatremia
1359. hyperazotemia
1360. venous hyperaemia
1361. What are the main pathogenetic link of cachectic edema?
1362. activation of the renin-angiotensin system
1363. hypernatremia
1364. hypoproteinemia \*
1365. starvation
1366. albuminuria
1367. What are the main pathogenetic link of allergic edema?
1368. increased biologically active substances
1369. increased permeability of blood vessels \*
1370. activation of the renin-angiotensin system
1371. lack of glucocorticoids
1372. hypoproteinemia
1373. What are the principles of pathogenetic therapy of cardiac edema?
1374. improvement of cardiac activity \*
1375. liquidation of venous hyperaemia \*
1376. injections of protein solutions
1377. liquidation of arterial hyperaemia
1378. administration of antihistamine preparations
1379. What are the principles of pathogenetic therapy nephritic edema?
1380. correction of blood circulation
1381. stimulation of diuresis
1382. restoration of capillary permeability \*
1383. infusions of protein solutions
1384. restoration of renal function \*
1385. What are the principles of pathogenetic therapy of nephrotic edema?
1386. correction of blood circulation
1387. stimulation of diuresis
1388. injections of protein solutions \*
1389. administration of antihistamine medication
1390. restoration of capillary permeability
1391. What are the principles of pathogenetic therapy of allergic edema?
1392. correction of blood circulation
1393. administration of antihistamine medication \*
1394. stimulation of diuresis
1395. administration of glucocorticoid hormones \*
1396. restoration of blood circulation
1397. What are the principles of pathogenetic therapy of inflammatory edema?
1398. administration of antihistamine medication \*
1399. correction of local blood circulation
1400. administration of antibacterial medication \*
1401. administration of glucocorticoid hormones\*
1402. injections of protein solutions

1. What are the basic clinical signs that characterise agony?
2. inspiratory dyspnoea
3. rare breathing with decreasing amplitude \*
4. bradycardia
5. breathing is shallow and fast
6. tachycardia
7. What are the basic clinical signs of death?
8. loss of consciousness
9. stopping heart contractions \*
10. general areflexia
11. remains the pupil reflex to light
12. respiratory arrest \*
13. By what is determined the duration of clinical death?
14. resistance of brain cortex to hypoxia \*
15. pathological antecedents
16. proprieties of tanatogenic factors
17. reactivity of the body’s
18. depth of processes excitation-inhibition in cortex
19. What time duration of first functional disorders of cortical neurons in the case of anoxia are?
20. 2-3 minutes
21. a few seconds \*
22. 15-30 minutes
23. 5-6 minutes
24. 45 minutes
25. What time duration of neuronal necrosis in the case of anoxia are?
26. 2 to 3 minutes
27. 15-30 minutes
28. 5-6 minutes
29. a few seconds \*
30. 45 minutes
31. What are the causes of carbohydrates maldigestion?
32. lack of hydrochloric acid
33. lack of pancreatic carboxypeptidase
34. lack of pepsin
35. insufficiency of pancreatic amylase \*
36. lack of intestinal disaccharidases \*
37. What are the causes of carbohydrates maldigestion?
38. lack of hydrochloric acid
39. lack of salivary amylase \*
40. lack of pepsin
41. insufficiency of pancreatic amylase \*
42. insufficiency of pancreatic lipase
43. What are the consequences of cellulose intake deficiency?
44. inhibited growth of microflora
45. intestinal blocking \*
46. increased peristalsis
47. constipation \*
48. diarrhea
49. What are the causes of carbohydrates malabsorption?
50. hyposecretion of intestine glands \*
51. hyposecretion of bile
52. gastric mucosal lesion
53. small bowel mucosal lesion \*
54. large intestine mucosal lesion
55. What are the disorders of carbohydrate metabolism in starvation?
56. excess acetyl Ko A \*
57. oxaloacetate acid deficiency \*
58. NADPH excess
59. metabolic alkalosis
60. deficiency acetyl Ko A
61. What are the disorders of carbohydrate metabolism in starvation?
62. oxaloacetate acid deficiency \*
63. deficiency acetyl Ko A
64. NADPH deficiency \*
65. metabolic alkalosis
66. NADPH excess
67. What are the disorders of carbohydrate metabolism in starvation?
68. excess acetyl Ko A \*
69. excess of oxaloacetate acid
70. excess of NADPH
71. metabolic alkalosis
72. metabolic acidosis \*
73. What are changes of endocrine glands functions in carbohydrate starvation?
74. glucagon hypersecretion \*
75. glucagon hyposecretion
76. glucocorticoids hypersecretion \*
77. glucocorticoids hyposecretion
78. adrenal hyposecretion
79. What are changes of endocrine glands functions in carbohydrate starvation?
80. glucagon hypersecretion \*
81. glucagon hyposecretion
82. glucocorticoids hyposecretion
83. adrenal hyposecretion
84. adrenal hypersecretion \*
85. What are the consequences of excessive consumption of carbohydrates?
86. insulin hyposecretion
87. protein mobilization from adipose tissue
88. increased lipogenesis \*
89. hyperglycemia \*
90. lipid mobilization from adipose tissue
91. What are the consequences of excessive consumption of carbohydrates?
92. insulin hypersecretion \*
93. protein mobilization from adipose tissue
94. increased lipogenesis \*
95. hyperglycemia \*
96. lipid mobilization from adipose tissue
97. What is the average value ​​of normal blood glycaemia?
98. 50 mg%
99. 200 mg%
100. 100 mg% \*
101. 2.0 mmol / l
102. 5.5 mmol / l \*
103. What are the causes of hypoglycaemia?
104. intense gluconeogenesis
105. intense glycogenolysis
106. hypersecretion of glucocorticoids
107. insulin hypersecretion \*
108. glucosuria \*
109. What are the causes of hypoglycaemia?
110. carbohydrate starvation \*
111. intense glycogenolysis
112. hypersecretion of glucocorticoids
113. insulin hypersecretion \*
114. hypersecretion of glucagon
115. What are the compensatory reactions in hyperglycemia?
116. insulin hypersecretion \*
117. intense glycogenogenesis \*
118. glucocorticoid hypersecretion
119. increased lipolysis
120. enhance of gluconeogenesis
121. What are the compensatory reactions in hyperglycemia?
122. insulin hyposecretion
123. intense glycogenogenesis \*
124. glucocorticoid hypersecretion
125. increased lipogenesis \*
126. enhance of gluconeogenesis
127. What are the compensatory reactions in hypoglycaemia?
128. glucagon hypersecretion \*
129. insulin hypersecretion
130. increased glycogenolysis \*
131. glucocorticoid hyposecretion
132. decreased lipolysis
133. What are the compensatory reactions in hypoglycaemia?
134. glucagon hyposecretion
135. insulin hypersecretion
136. increased glycogenogenesis
137. glucocorticoid hypersecretion \*
138. increased lipolysis \*
139. What are the potential consequences of hypoglycaemia?
140. hypoglycemic coma \*
141. metabolic alkalosis
142. lipogenesis
143. liver lipodystrophy \*
144. respiratory alkalosis
145. What are the potential consequences of hyperglycemia?
146. Obesity \*
147. hyperglycaemic coma
148. lipodystrophy and lipidic infiltration of organs \*
149. hypercetonemia
150. glucosuria \*
151. What are the potential consequences of diabetic hyperglycemia?
152. blood hyperosmolarity \*
153. cetodiabetic coma \*
154. obesity
155. metabolic alkalosis
156. blood hypoosmolarity
157. What are the causes of galactosemia?
158. intensive transformation of glucose into galactose
159. inability to use galactose bodies
160. inability of the liver to convert galactose into glucose \*
161. inability of the kidneys to secrete galactose
162. excessive consumption of galactose
163. What are the consequences of galactosemia in new-borns?
164. coma caused by hypergalactosemia
165. infiltration of organs with galactose \*
166. diabetes mellitus
167. opacification of lens \*
168. disturbed morphogenesis of central neurosis system \*
169. What lipids are synthesized in the body?
170. neutral fats \*
171. saturated fatty acids \*
172. polyunsaturated fatty acids
173. phospholipids \*
174. fat-soluble vitamins
175. What is the role of bile acids in the digestive system?
176. activation of inactive lipase \*
177. absorption of fatty acids \*
178. inhibits intestinal peristalsis
179. alkalinization of gastric contents
180. fats emulsifying \*
181. What are the consequences of lipid starvation?
182. energy deficit \*
183. fat-soluble vitamin deficiency \*
184. lack of saturated fatty acids
185. lack of polyunsaturated fatty acids \*
186. increasing of blood coagulability
187. What are the causes of lipids maldigestion?
188. salivary hyposecretion
189. bile hyposecretion \*
190. stomachal hypoacidity
191. pancreatic hyposecretion \*
192. large bowel mucosal lesions
193. What are the causes of lipids maldigestion?
194. salivary hyposecretion
195. bile hypersecretion
196. stomachal hypoacidity
197. pancreatic hyposecretion \*
198. small bowel mucosal lesions \*
199. What are the metabolic consequences of lipids maldigestion?
200. Water-soluble vitamin deficiency
201. disturbance of steroid hormone synthesis \*
202. change in cell membrane properties \*
203. hypercoagulation of blood
204. hypocoagulation of blood \*
205. What is the sequence of lipids absorption?
206. mesenterial veins, lymphatic vessels, thoracic duct
207. mesenterial veins, portal vein, liver
208. lymphatic vessels, thoracic duct, superior vena cava \*
209. mesenterial veins, superior vena cava, liver
210. lymphatic vessels, thoracic duct, liver
211. In what form are transported absorbed lipids from the small intestine?
212. free triglycerides
213. chylomicrons \*
214. free fatty acids
215. phospholipids
216. free bile acids
217. In what form are transported lipids synthesized in the liver?
218. low-density lipoproteins (beta-lipoprotein)
219. very low-density lipoproteins (pre-beta-lipoprotein) \*
220. high density lipoproteins (alpha-lipoprotein)
221. free triglycerides
222. free fatty acids
223. In what form are transported lipids mobilized from adipose tissue?
224. chylomicrons
225. very low-density lipoproteins (pre-beta-lipoprotein)
226. low-density lipoprotein (beta-lipoprotein)
227. free fatty acids conjugated albumin \*
228. high density lipoproteins (alpha-lipoprotein)
229. In components of what lipoproteins, cholesterol is mainly transported to the organs?
230. chylomicrons
231. very low-density lipoproteins (pre-beta-lipoprotein)
232. high density lipoproteins (alpha-lipoprotein)
233. low density lipoproteins (beta-lipoprotein) \*
234. free cholesterol
235. In components of what lipoproteins, cholesterol is transported from the organs to the liver?
236. chylomicrons
237. very low density lipoproteins (pre-beta-lipoprotein)
238. high density lipoproteins (alpha-lipoprotein) \*
239. low density lipoproteins (beta-lipoprotein)
240. free cholesterol
241. What are the possible consequences of direct absorption of protein from digestive tract?
242. hyperproteinaemia
243. food allergy \*
244. no pathological consequences
245. anaphylactic shock
246. infiltration of the liver protein
247. What is the protein imbalance occurring in liver failure?
248. failure to increase the synthesis of serum globulin ratio albumin/globulin
249. lack of serum albumin synthesis by lowering the albumin/globulin ratio \*
250. lack of paraproteinemia
251. insufficiency of blood coagulation factor synthesis \*
252. lack of immune deficiency immunoglobulin synthesis
253. What are the causes of protein maldigestion?
254. bile hyposecretion
255. pancreatic hyposecretion \*
256. hyposalivation
257. stomach hyperacidity
258. hyposecretion of intestinal peptidase \*
259. What are the causes of protein maldigestion?
260. bile hyposecretion
261. pancreatic hyposecretion \*
262. hyposalivation
263. stomach hypoacidity \*
264. hypersecretion of intestinal peptidase
265. What are the consequences of amino acids malabsorption in the digestive tract?
266. essential amino acid deficiency \*
267. hypoproteinemia \*
268. mobilization of storage proteins
269. immunodeficiency \*
270. mobilization of fat deposits
271. What is the normal average value of blood proteins?
272. 50 g / l
273. 120 g / l
274. 80 g / l \*
275. 200 g / l
276. 150 g / l
277. Which states are accompanied by hypoproteinemia?
278. total starvation \*
279. haemodilution \*
280. haemoconcentration
281. nephritic syndrome
282. nephrotic syndrome \*
283. Which states are accompanied by hypoproteinemia?
284. Hypoonchia \*
285. Edema \*
286. decrease of glomerular filtration
287. polyuria \*
288. oliguria
289. What are the consequences of hyperproteinaemia?
290. interstitial dehydration \*
291. edema
292. increased renal filtration
293. polyuria
294. reduction of renal filtration \*
295. What is the protein imbalance in liver failure?
296. reducing the concentration of lipoproteins in the blood \*
297. reducing the protein content of the coagulant system \*
298. increasing the ratio albumin/globulin in plasma
299. hypoalbuminemia \*
300. increasing the protein content of the coagulant system
301. Which states are accompanied by negative nitrogen balance?
302. in healthy children
303. fever \*
304. administration of insulin
305. administration of glucocorticoids \*
306. pregnancy
307. Which states are accompanied by positive nitrogen balance?
308. administration of anabolic hormones \*
309. gestation \*
310. diabetes mellitus
311. fever
312. administration of glucocorticoids.
313. What is the average value of blood pH the healthy man?
314. 6.0
315. 7.4 \*
316. 7.1
317. 7.6
318. 8.0
319. What are the basic criteria of acidosis?
320. pH decrease \*
321. increasing concentrations of H + ions \*
322. increasing the pH
323. decreasing concentrations of H + ions
324. alkaline reserve accumulation
325. What are the basic criteria of alkalosis?
326. increasing the pH \*
327. pH decrease
328. increasing of H + ion concentration
329. reduction of alkaline reserve
330. alkaline reserve accumulation \*
331. What does represent compensated acidosis?
332. decrease of alkaline reserves with decreasing pH
333. reduce of the alkaline reserves with constant pH \*
334. decrease of alkaline reserves with increasing pH
335. increasing of alkaline reserves with constant pH
336. constant alkaline reserves with constant pH
337. What does represent decompensated acidosis?
338. decrease of alkaline reserves with increasing pH
339. decrease of alkaline reserves with decreasing pH \*
340. decrease of alkaline reserves with constant pH
341. increase alkaline reserves with constant pH
342. constant alkaline reserves with constant pH
343. What does represent compensated alkalosis?
344. decrease of alkaline reserves with decreasing pH
345. decrease of alkaline reserves with increasing pH
346. increase the alkaline reserves with constant pH \*
347. reduction of alkaline reserve with constant pH
348. constant alkaline reserves with constant pH
349. What does represent decompensated alkalosis?
350. decrease alkaline reserves with decreasing pH
351. decrease alkaline reserves with increasing pH
352. decrease alkaline reserves with constant pH
353. increase alkaline reserves with increased pH \*
354. constant alkaline reserves with constant pH
355. What are the causes of excretory acidosis?
356. renal insufficiency \*
357. diabetic coma \*
358. diarrhea \*
359. asphyxia
360. incoercible vomiting
361. What are the causes of respiratory acidosis?
362. excessive formation of CO2
363. loss of bicarbonates with physiological secretions
364. disorder of CO2 diffusion from the blood into alveoli \*
365. loss of hydrochloric acid in gastric juice
366. excessive loss of CO2 in alveolar hyperventilation
367. What are the causes of alkalosis?
368. excessive formation of ketones
369. loss of hydrochloric acid in gastric juice\*
370. loss of bicarbonates by physiological secretions
371. disorder of urea synthesis from ammonia \*
372. excessive loss of CO2 in alveolar hyperventilation \*
373. What is consequence of decompensated cellular acidosis?
374. activation of oxidative processes
375. activation of glycolytic enzymes
376. hyper polarization of cytoplasmic membrane
377. lysosomal enzymes exit and activation in hyaloplasm \*
378. decrease of cellular membrane permeability for Na ions
379. What is consequence of decompensated cellular acidosis?
380. inactivation of Krebs cycle enzymes \*
381. activation of glycolytic enzymes
382. hyper polarization of cytoplasmic membrane
383. lysosomal enzymes exit and activation in hyaloplasm \*
384. decrease of cellular membrane permeability for Na ions
385. What is consequence of decompensated cellular acidosis?
386. increase of cellular membrane permeability for Na ions \*
387. activation of glycolytic enzymes
388. hyper polarization of cytoplasmic membrane
389. lysosomal enzymes exit and activation in hyaloplasm \*
390. decrease of cellular membrane permeability for Na ions
391. What is consequence of decompensated cellular acidosis?
392. inactivation of glycolytic enzymes \*
393. activation of glycolytic enzymes
394. hyper polarization of cytoplasmic membrane
395. lysosomal enzymes exit and activation in hyaloplasm \*
396. decrease of cellular membrane permeability for Na ions
397. What is consequence of decompensated cellular acidosis?
398. activation of oxidative processes
399. activation of glycolytic enzymes
400. inactivation of glycolytic enzyme \*
401. hyper polarization of cytoplasmic membrane
402. decrease of cellular membrane permeability for Na ions
403. What is consequence of decompensated cellular acidosis?
404. cytoplasmic membrane hyperpolarization
405. Krebs cycle enzymes inactivation \*
406. decrease of cellular membrane permeability for Na ions
407. activation of oxidative processes
408. glycolytic enzymes activation
409. What is consequence of decompensated cellular acidosis?
410. cytoplasmic membrane hyper polarization
411. increase of cellular membrane permeability for Na ions \*
412. decrease of cellular membrane permeability for Na ions
413. activation of oxidative processes
414. activation of glycolytic enzymes
415. What are the consequences of disaccharide maldigestion?
416. hyperglycemia
417. pancreatic insufficiency \*
418. constipation
419. hyperhydration
420. hypoglycemia \*
421. What are the consequences of lipid maldigestion?
422. Steatorrhea \*
423. blood hypocoagulation \*
424. deficiency of vitamin K \*
425. blood hypocoagulation
426. constipation
427. What are the parameters of normocythaemic normovolemia?
428. erythrocytes 5.1012 / L; total volume of blood 7% of body weight; hematocrit 42%; \*
429. erythrocytes 7.1012 / L; total volume of blood 7% of body weight; hematocrit 56%
430. erythrocytes 3.1012 / L; total blood volume of 5% of body weight; 32% hematocrit;
431. erythrocytes 3.1012 / L; total volume of blood 7% of body weight; 32% hematocrit;
432. erythrocytes 7.1012 / L; 9% total blood volume of body weight; hematocrit 56%;
433. What are the parameters of oligocythemic hypovolemia?
434. hematocrit 42%; total blood volume 5% of body weight; erythrocytes 5.1012 / L;
435. hematocrit;56%; total volume of blood 7% of body weight; erythrocytes 7.1012 / L;
436. hematocrit 32%; total blood volume 5% of body weight; erythrocytes 3.1012 / L; \*
437. hematocrit 32%; total blood volume 7% of body weight; erythrocytes 3.1012 / L;
438. hematocrit 56%; total blood volume 5% of body weight; erythrocytes 7.1012 / L;
439. What are the parameters of oligocythemic hypervolemia?
440. erythrocytes 5.1012 / L; total blood volume 7% of body weight; hematocrit 42%;
441. erythrocytes 7.1012 / L; total blood volume 7% of body weight; hematocrit 56%;
442. erythrocytes 3.1012 / L; total blood volume 5% of body weight; hematocrit 32%;
443. erythrocytes 3.1012 / L; total blood volume 7% of body weight; hematocrit 32%;
444. erythrocytes 3.1012 / L; 9% total blood volume of body weight; hematocrit 32%; \*
445. What are the parameters of polycythemic hypovolemia?
446. erythrocytes 5.1012 / L; total blood volume 5% of body weight hematocrit 42%;
447. erythrocytes 7.1012 / L; The total volume of blood 7% of body weight; hematocrit 56%;
448. erythrocytes 7.1012 / L; total blood volume 5% of body weight; hematocrit 56%; \*
449. erythrocytes 3.1012 / L; total blood volume 7% of body weight; hematocrit 32%;
450. erythrocytes 7.1012 / L; 9% total blood volume of body weight; hematocrit 56%;
451. What changes of acid-base equilibrium refer to hyperventilation?
452. alkaline reserves decrease
453. alkaline reserves increase \*
454. pH increasing \*
455. pH decreasing
456. hydrogen proton concentration increases
457. What changes of acid-base equilibrium refer to hypoventilation?
458. alkaline reserves decrease \*
459. alkaline reserves increase
460. pH increasing
461. pH decreasing \*
462. hydrogen proton concentration decreases