1. What is reparative reaction?
2. In what conditions can develop symptomatic gingival hypertrophy?
3. What are the mechanisms of increased thermogenesis in fever?
4. What is the pathogenesis of allergic reaction type II?
5. What is the pathogenesis of increased permeability of the blood vessel walls in the inflammatory focus?
6. When can develop embolism in the systemic circulation?
7. What structures are frequently involved in allergic reactions type III?
8. What are the cells involved in anaphylactic allergic reactions?
9. What are the mechanisms of reduced thermolysis in the initial period of fever?
10. What can be the antigen in type II allergic reaction?
11. What does a pathological reaction mean?
12. What does represent thrombocytopathy?
13. What does the sclerosis of organ mean?
14. What is characteristic of pathological regeneration?
15. How are classified emboli by the direction of their circulation?
16. How can be diminished the pathochemical processes in anaphylactic reactions?
17. In what condition can develop insufficiency of anti-coagulant system?
18. In what conditions can develop atrophy of the mouth mucosa?
19. In what conditions can develop gaseous embolism?
20. On what does the localization of general injuries depend?
21. What antigens can trigger autoimmune reaction?
22. What antigens can trigger delayed hypersensibility?
23. What are eventual causes of the primary cell injuries?
24. What are eventual causes of the secondary cell injuries?
25. What are exogenous infectious pyrogenic factors?
26. What are exogenous non-infectious pyrogenic factors?
27. What are general changes in inflammation?
28. What are general manifestations of inflammation?
29. What are local manifestations in allergic reaction type III?
30. What are the biological effects of active complement fraction?
31. What are the biological effects of IL-1?
32. What are the biological effects of leukotrienes?
33. What are the biological effects of prostacyclin?
34. What are the biological effects of prostaglandins PGD2, PGE2, PGF2a alpha?
35. What are the biological effects of thromboxane TxA2, TxB2?
36. What are the cardio-vascular changes in the third period of fever?
37. What are the cardiovascular changes in the second period of fever?
38. What are the causes for thrombus development?
39. What are the causes of air embolism?
40. What are the causes of hypercoagulation?
41. What are the cellular sources of proliferation in the inflammatory focus?
42. What are the characteristics of allergic reactions type II?
43. What are the characteristics of complete antigen?
44. What are the characteristics of incomplete antigen?
45. What are the characteristics of inflammatory stasis?
46. What are the characteristics of normoergic inflammation?
47. What are the characteristics of type I allergic reactions?
48. What are the characteristics of type II allergic reactions?
49. What are the characteristics of type III allergic reaction?
50. What are the characteristics of type III allergic reactions?
51. What are the consequences of annihilation of intra - and extracellular Na+ ions gradient?
52. What are the consequences of annihilation of intra - and extracellular K+ ions gradient?
53. What are the consequences of annihilation of the transmembrane Ca2+ ions gradient?
54. What are the consequences of cell dystrophy?
55. What are the consequences of exaggerated apoptosis?
56. What are the consequences of long-lasting stasis in the inflammatory focus?
57. What are the consequences of the intracellular ATP-ases activation?
58. What are the consequences of the intracellular nucleoproteases activation?
59. What are the consequences of the intracellular proteases activation?
60. What are the consequences of venous hyperemia?
61. What are the disorders that could lead to teeth attrition?
62. What are the effects of interleukins in inflammation?
63. What are the effects of mediators involved in the allergic reaction type III?
64. What are the effects of mediators released from neutrophils?
65. What are the end-effectors in anaphylactic allergic reactions?
66. What are the endogenous causes of diseases?
67. What are the etiological factors of lipid dystrophy?
68. What are the exogenous causes of diseases?
69. What are the external changes of arterial hyperemia?
70. What are the external manifestations of ischemia?
71. What are the external manifestations of venous hyperemia?
72. What are the features of inflammatory arterial hyperemia?
73. What are the final effects in type II allergic reactions?
74. What are the general causes of energy depletion that trigger cell dystrophy?
75. What are the general consequences of apoptosis for the organism?
76. What are the general consequences of embolism?
77. What are the general consequences of necrosis for the organism?
78. What are the general consequences of venous hyperemia?
79. What are the hallmarks of serous exudate?
80. What are the kinin effects in the inflammatory focus?
81. What are the local consequences of necrosis?
82. What are the manifestations of cell necrosis?
83. What are the manifestations of cellular alterations in the inflammatory focus?
84. What are the manifestations of stasis?
85. What are the mechanisms of activation of thermogenesis in fever?
86. What are the mechanisms of increased thermogenesis in fever?
87. What are the mechanisms of phagocytosis?
88. What are the mechanisms of primary haemostasis?
89. What are the mechanisms of reduced thermolysis in the initial period of fever?
90. What are the mechanisms of secondary haemostasis?
91. What are the mediators of pathochemical phase of allergic reactions type IV?
92. What are the metabolic changes in arterial hyperemia?
93. What are the metabolic changes in ischemia?
94. What are the metabolic changes in the second stage of fever?
95. What are the metabolic changes in venous hyperemia?
96. What are the pathogenetic factors involved in pathochemical phase of allergic reactions type III?
97. What are the pathogenetic factors of capillary stasis?
98. What are the pathogenetic mechanisms of edema in venous hyperemia?
99. What are the pathogenetic mechanisms of external changes in venous hyperemia?
100. What are the pathogenetic mechanisms of inflammatory venous hyperemia?
101. What are the pathogenetic mechanisms of ischemia?
102. What are the pathogenetic mechanisms of necrosis due to cytoplasmic membrane damage?
103. What are the pathogenetic mechanisms of necrosis due to mitochondrial injury?
104. What are the pathogenetic mechanisms of scleroderma in oral cavity?
105. What are the pathogenetic mechanisms of sclerosis?
106. What are the pathogenic mechanisms of lipid dystrophy?
107. What are the possible variants of relationship between general and local injuries?
108. What are the primary endogenous pyrogenic factors?
109. What are the secondary endogenous pyrogenic factors?
110. What are the trigger factors for development of hypertrophy?
111. What atrophy is considered as physiological?
112. What cells are involved in apoptosis?
113. What conditions are considered favorable for the organism?
114. What conditions are considered unfavorable for the organism?
115. What conditions are necessary for apoptosis?
116. What disorders can lead to ulcerative changes of oral mucosa?
117. What disorders underlie on the basis of delayed hypersensibility?
118. What do pathogenetic factors represent?
119. What does a physiological reaction mean?
120. What does clinical pathophysiology study?
121. What does general pathophysiology study?
122. What does injury represent?
123. What does involve the microcirculatory system?
124. What does represent allergic reactions type IV?
125. What does represent regeneration in the inflammatory focus?
126. What does represent the autoimmune reactions?
127. What does represent the haemorrhagic syndrome of plasmatic origin?
128. What does represent fever?
129. What does represent the endogenous antigen?
130. What does represent the process of thermoregulatory center restructuration in the first period of fever?
131. What does special pathophysiology study?
132. What does the chain of cause-effect in pathogenesis represent?
133. What does the disease represent?
134. What does “vicious circle” in pathogenesis represent?
135. What etiological factors are responsible for developing of vinous hyperemia?
136. What factor induces sclerosis?
137. What inflammatory mediator is released from thrombocytes?
138. What inflammatory mediators are released from eosinophils?
139. What inflammatory mediators are released from lymphocytes?
140. What inflammatory mediators are released from neutrophils?
141. What intracellular dyshomeostasis results from cessation of membrane ionic pumps function?
142. What ions have vasoconstrictive effect?
143. What ions have vasodilatory effect?
144. What is a primary endogenous pyrogenic factor?
145. What is a secondary endogenous pyrogenic factor?
146. What is adaptive reaction?
147. What is adaptive regeneration?
148. What is an effect of IL-1?
149. What is an exogenous non-infectious pyrogenic factor?
150. What is characteristic for neurotonic mechanism of arterial hyperemia?
151. What is characteristic for the final period of apoptosis?
152. What is characteristic for the first period of apoptosis?
153. What is characteristic for the latent period of the disease?
154. What is characteristic for the period of complete disease manifestation?
155. What is characteristic for the prodromal period of the disease?
156. What is compensatory reaction?
157. What is compensatory regeneration?
158. What is general etiology?
159. What is homeostatic regeneration?
160. What is normoergic reaction?
161. What is one of the consequences of sclerosis?
162. What is pathogenetic therapy?
163. What is pathological regeneration?
164. What is protective reaction?
165. What is protective regeneration?
166. What is reparative regeneration?
167. What is the antigen which can trigger type II allergic reaction?
168. What is the biological significance of allergic reactions?
169. What is the biological significance of apoptosis?
170. What is the biological significance of fever?
171. What is the biological significance of inflammatory venous hyperemia?
172. What is the biological significance of leukocyte emigration in the site of inflammation?
173. What is the definition of fever?
174. What is the feature of delayed hypersensibility?
175. What is the feature of immediate hypersensibility?
176. What is the final effect of allergic reactions type IV?
177. What is the hallmark of purulent exudate?
178. What is the main link of pathogenesis?
179. What is the mechanism of cytolysis in allergic reactions type II (cytotoxic, cytolytic)?
180. What is the mechanism of leukocyte emigration in the inflammatory focus?
181. What is the non-specific prophylaxis of the disease?
182. What is the origin of the digestive enzymes circulating in the blood?
183. What is the pathogenesis of exudation in the inflammatory focus?
184. What is the pathogenesis of inflammatory venous hyperemia?
185. What is the pathogenesis of pathophysiological phase in allergic reactions type IV?
186. What is the pathogenesis of proliferation in the inflammatory focus?
187. What is the pathogenetic factor of fatty liver?
188. What is the pathogenetic mechanism of decreased volume of the ischemic organ?
189. What is the pathogenetic role of ATP depletion in necrosis?
190. What is the pathogenetic role of calcium in necrosis?
191. What is the pathogenetic role of free radicals in necrosis?
192. What is the pathogenetic role of hypoxia in necrosis?
193. What is the physiological role of fibrinolytic system?
194. What is the programmed cell death?
195. What is the result of proliferation in the inflammatory focus?
196. What is the role of conditions in the disease appearance?
197. What is the role of the cause in the disease appearance?
198. What is the sequence of leukocyte emigration into the inflammatory site?
199. What is the sequence of processes in the synthesis of prostaglandins?
200. What is the sequence of vascular reactions in the inflammatory focus?
201. What is the significance of the increased intracellular enzymes activity in the blood?
202. What is the specific prophylaxis of the disease?
203. What is the symptomatic therapy?
204. What mechanisms are specific for functional arterial hyperemia?
205. What mediators are produced in the mast cells via cyclooxygenase pathway?
206. What mediators are produced in the mast cells via lipoxygenase pathway?
207. What mediators lead to development of inflammatory arterial hyperemia?
208. What structures of cytoplasmic membrane are damaged and lead to disintegration of the cell?
209. What substances are complete antigens?
210. What substances are incomplete antigens?
211. When can develop hyperergic inflammation?
212. When can develop hypoergic inflammation?
213. When the antipyretic therapy is justified?