**Questions for Pathophysiology final exam, SIMU test, semester V**

1. How do the resting membrane potential and neuromuscular excitability change in severe hyperkalemia (above 6,5 mEq/l)?
2. How pH and bicarbonate are changed in insulin deficiency?
3. What are the blood changes detected in metabolic acidosis?
4. What are the causes of hypotonic hyperhydration?
5. What change in the blood is found in hyperhydration?
6. What changes in the blood are found in isotonic hyperhydration?
7. What is the cause of absolute hyponatremia?
8. What is the change of hyperkalemia on ECG?
9. How does the cellular metabolism change in the ischemia?
10. What are the main mediators involved in type II allergic reactions?
11. For every vital function the cell needs energy. What is a consequence of energy depletion for cells?
12. What are the mechanisms of tissular injury in type IV allergic reactions?
13. What biological products can be DAMP (damage associated molecular pattern)?
14. What can be the consequences of cell dystrophy?
15. What change in the blood is found in hyperhydration?
16. What is the biological significance of leucocyte emigration in the inflammatory focus?
17. What is the cause of hypotonic hyperhydration?
18. What is the pathogenetic role of intracellular potassium dyshomeostasis in development of cell necrosis?
19. What mediators determine development of arterial hyperemia into the inflammatory focus?
20. What pathogenetic factors increase vascular permeability into the inflammatory focus?
21. Which factors promote adhesion and rolling of leukocytes at the endothelial level?
22. Cell hypoxia and activation of anaerobic glycolysis led to intracellular acidosis. What is a consequence of decompensated intracellular acidosis?
23. Cell metabolic disorders can affect one or several organs. What can be the causes of multiple cell metabolic disorders?
24. In what cases develops gaseous embolism?
25. What can be the consequence of cell dystrophy?
26. What does represent allergy?
27. What inflammatory mediators are derived from neutrophils?
28. What is characteristic for arterial hyperemia?
29. What is the consequence of venous hyperemia?
30. What is the external manifestation of ischemia?
31. What is the pathogenetic mechanism of neurotonic arterial hyperemia?
32. What pathological cellular processes can trigger cell dystrophy?
33. What pathological cellular processes can trigger cell dystrophy?
34. A homeostatic process precluding sclerosis is collagenolysis. What is the mechanism of collagen excess reducing in the organ?
35. A homeostatic process precluding sclerosis is collagenolysis. What is the mechanism of collagen excess reducing in the organ?
36. Accumulation of what products leads to metabolic acidosis in insulin lack?
37. Apoptosis is a programmed cell death, which can be initiated in physiological and pathological situations. What are the conditions for physiological apoptosis?
38. Apoptosis is a programmed cell death, which can be initiated in physiological and pathological situations. What are the conditions for physiological apoptosis?
39. Apoptosis is a programmed cell death, which can be initiated in physiological and pathological situations. What are the conditions for pathological apoptosis?
40. Apoptosis is a programmed cell death, which can be initiated in physiological and pathological situations. What are the conditions for pathological apoptosis?
41. By what is manifested prodromal period of the disease?
42. Cell hypoxia and activation of anaerobic glycolysis leads to intracellular acidosis. What are the consequences of decompensated intracellular acidosis?
43. Cell organelles are separated from hyaloplasm by membrane. What is the consequence of lysosomal membrane destabilization?
44. Define the correct notion of metaplasia?
45. For every vital function the cell needs energy. What is a consequence of energy depletion for cells?
46. How apoptosis is manifested in the initial period?
47. How do the blood circulatory volume and blood viscosity change in relative hyponatremia?
48. How do the blood viscosity and blood cell concentration change in absolute hyponatremia?
49. How do the circulatory blood volume and blood cell concentration change in absolute hyponatremia?
50. How do the intravascular volume, osmolarity of the plasma and cell volume change in relative hypernatremia?
51. How do the intravascular volume, osmolarity of the plasma and cell volume change in absolute hyponatremia?
52. How do the resting membrane potential and neuromuscular excitability change in mild hyperkalemia (5,5-6,0 mEq/l)?
53. How do the resting membrane potential and neuromuscular excitability change in hypokalemia?
54. How does carbohydrates metabolism change in liver failure?
55. How does carbohydrates metabolism change in liver failure?
56. How does glycemia change in liver insufficiency?
57. How does the blood protein content change in liver failure?
58. How does the blood protein content change in liver failure?
59. How does the blood viscosity and concentration of blood cells change in relative hypernatremia?
60. How does the circulatory blood volume and concentration of blood cells change in absolute hypernatremia?
61. How does the circulatory blood volume and concentration of blood cells change in relative hypernatremia?
62. How does the protein metabolism change in protein maldigestion?
63. How is explained the mechanisms of atrophy?
64. How oxyhemoglobin curve dissociation and hemoglobin affinity to O2 change in alkalosis?
65. How oxyhemoglobin curve dissociation and hemoglobin affinity to O2 change in acidosis?
66. How pH and bicarbonate change in the hypoxia?
67. How pH and PaCO2 change in the pulmonary hyperventilation?
68. How pH and PaCO2 change in the pulmonary hypoventilation?
69. How respiratory frequency (RF) and PaCO2 change in metabolic acidosis?
70. How respiratory frequency (RF) and PaCO2 change in metabolic alkalosis?
71. How the acidosis is defined?
72. How the apoptosis is manifested in the initial period?
73. How the blood glucose level, blood osmolarity and diuresis are changed in ketoacidosis triggered by insulin deficiency?
74. How the blood oncotic and osmotic pressure are changed in excessive intake of water?
75. How the blood oncotic and osmotic pressure are changed in massive infusion of isotonic NaCl solution?
76. How the blood osmolarity and cell volume are changed in vasopressin hypersecretion?
77. How the blood osmolarity and cell volume change in pulmonary hyperventilation?
78. How the blood osmolarity and cell volume change in vasopressin deficiency?
79. How the blood osmolarity and cell volume is changed in hypertonic dehydration?
80. How the blood osmolarity and intracellular hydric compartment are changed in hypertonic hyperhydration?
81. How the blood osmolarity and sodium blood level change in pulmonary hyperventilation?
82. How the initial period of apoptosis is manifested?
83. How the lipid metabolic reactions are changed in inanition?
84. How the lipid metabolism is changed in liver failure?
85. How the pH and NaHCO3 are changed in the metabolic acidosis?
86. How the pH and NaHCO3 are changed in the metabolic alkalosis?
87. How the pH and PaCO2 are changed in respiratory acidosis?
88. How the pH and PaCO2 are changed in the metabolic acidosis?
89. How the pH and PaCO2 are changed in the metabolic alkalosis?
90. How the pH and PaCO2 are changed in the respiratory alkalosis?
91. In condition of hyperosmolar environment in the cell are triggered multiple injuries: a. Intracellular hyperosmolarity b. Dehydration of cellular organelles c. Disintegration of mitochondria d. Dehydration of cellular hyaloplasm e. Cytolysis What is the pathogenetic chain of the cellular death to the action of hyperosmolar environment?
92. In condition of hypoosmolar environment in the cell are triggered multiple injuries: a. hypoosmolarity of cellular hyaloplasm b. increases mechanical intracellular pressure c. cellular swelling d. hyperhidration of cellular hyaloplasm e. cytolysis What is the pathogenetic chain of the cellular death to the action of hypoosmolar environment?
93. In condition of hypoxia in the cell are triggered multiple injuries: a. Cessation of ionic pumps b. Intracellular hyperosmolarity c. Decreases ATP synthesis d. Intra-extracellular electrolyte dysbalance e. Cytolysis What is the pathogenetic chain of cell death in hypoxia?
94. In condition of low temperature in the cell are triggered multiple injuries: a. mechanical breakdown of cytoplasmic membrane b. crystallization of intracellular water c. intracellular hyperosmolarity d. Intra-extracellular electrolyte dysbalance e. Cytolysis What is the pathogenetic chain of the cellular death to the action of low temperature?
95. In cytoplasmatic membrane injury there is equilibration of intra-extracellular electrolytes levels. What is the effect of equilibration of intra-extracelular levels of K+?
96. In what cases is attested negative nitrogen balance?
97. In what cases is attested positive nitrogen balance?
98. In what pathological process there is found protein maldigestion?
99. In what pathological processes is attested hyperkalemia?
100. In what pathological processes is attested hypernatremia?
101. In which organ the irreparable cell lesions obligatory provoke sclerosis?
102. In which organ the irreparable cell lesions obligatory provoke sclerosis?
103. Inflammation leads to development of acute-phase response. What are the changes in the body during acute phase-response?
104. Liver is the parenchymatous organ frequently affected by lipid dystrophy. What is one of the causes of liver steatosis?
105. Lysosomal membrane traps the lysosomal enzymes within organelle. What is the factor which can destabilize lysosomal membrane?
106. Lysosomal membrane traps the lysosomal enzymes within organelles. What factor works as endogenous stabilization factor for lysosomal membrane?
107. Lysosomal membrane traps the lysosomal enzymes within organelles. What factor works as endogenous stabilization factor for lysosomal membrane?
108. Maintenance of intra-extracellular ionic gradient is energy-dependent. What is the consequence of cell ATP depletion?
109. Many cellular pathological processes lead to generation of reactive oxygen species. What are negative effects of ROS?
110. Many cellular pathological processes lead to generation of reactive oxygen species. What is negative efect of ROS?
111. On what depends the vulnerability of different organs to the hypoxia?
112. 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?
113. Some intracellular metabolic reactions are energy-dependent. What is the consequence of cell ATP deficiency?
114. The cell injury results in release of intracellular enzymes in the blood. What enzymatic changes are characteristic for bile duct epithelial injury?
115. The inanition period is followed by hypoglycemia. How do change the insulin secretion and glucagon secretion in this period?
116. Under the action of high temperature in the cell appear multiple injuries. What primary injury is caused by high temperature?
117. Under the action of hypoxia in the cell appeared multiple injuries. What primary injury is caused by hypoxia?
118. Under the action of low temperature in the cell appear multiple injuries. What primary injury is caused by low temperature?
119. What are biological characeristics of immediate hypersensibility?
120. What are biological characteristics of active sensitisation?
121. What are biological characteristics of delayed hypersensibility?
122. What are biological characteristics of passive sensitisation?
123. What are biological characteristics of passive sensitisation?
124. What are biological effects of anaphylatoxins in inflammatory focus?
125. What are effects of increased sodium level in the cell hyaloplasm?
126. What are effects of reduced sodium ions concentration in the interstitium?
127. What are eventual causes of the primary cell injuries?
128. What are eventual causes of the secondary cell injuries?
129. What are hallmarks of the immunological stage of type II allergic reactions?
130. What are mediators involved in type II allergic reactions?
131. What are pathogenetic factors of hypoxic cell injuries?
132. What are pathogenetic factors of hypoxic cell injuries?
133. What are pathogenetic factors of hypoxic cell injuries?
134. What are pathogenetic factors of hypoxic cell injuries?
135. What are pathogenetic mechanisms of exudation in the inflammatory focus?
136. What are pathogenetic mechanisms of exudation in the inflammatory focus?
137. What are pathogenic mechanisms which explain autophagy in atrophy?
138. What are pathogenic mechanisms which explain autophagy in atrophy?
139. What are pathophysiologic factors of reduced apoptosis?
140. What are plasma-derived inflammatory mediators?
141. What are plasma-derived inflammatory mediators?
142. What are the biologic effects of fragments C3a and C5a into the inflammatory focus?
143. What are the biologic effects of kinin in inflammation?
144. What are the biological characteristics of active sensitisation?
145. What are the biological characteristics of acute inflammation?
146. What are the biological characteristics of antigen presenting cells?
147. What are the biological characteristics of chronic inflammation?
148. What are the biological effects of anti-inflammatory interleukins?
149. What are the biological effects of pro-inflammatory interleukins (IL-1, IL-6)?
150. What are the biological effects of pro-inflammatory interleukins (IL-1, IL-6)?
151. What are the blood changes detected in metabolic acidosis?
152. What are the blood changes detected in metabolic alkalosis?
153. What are the blood changes detected in respiratory acidosis?
154. What are the blood changes detected in respiratory alkalosis?
155. What are the causes of lipid maldigestion?
156. What are the causes of parenchymatous lipid dystrophy?
157. What are the causes of retention hyperlipidemia?
158. What are the causes of retention hyperlipidemia?
159. What are the cellular chemotactic substances that are important in emigration of leukocytes?
160. What are the cellular chemotactic substances that are important in emigration of leukocytes?
161. What are the changes of hyperkalemia on ECG?
162. What are the characteristics of apoptosis?
163. What are the characteristics of apoptosis?
164. What are the characteristics of apoptosis?
165. What are the characteristics of necrosis?
166. What are the characteristics of necrosis?
167. What are the characteristics of necrosis?
168. What are the compensatory reactions in hypercalcemia?
169. What are the compensatory reactions in hyperglycemia?
170. What are the compensatory reactions in hyperglycemia?
171. What are the consequences of exaggerated apoptosis?
172. What are the consequences of excessive alimentary consumption of proteins?
173. What are the consequences of excessive protein intake?
174. What are the consequences of lipid malabsorption?
175. What are the consequences of lipid malabsorption?
176. What are the consequences of lysosomal membrane destabilization?
177. What are the consequences of sclerosis?
178. What are the consequences of vomiting?
179. What are the effector immune cells in delayed hypersensitivity?
180. What are the effects of leukotriens realised by mast cells in allergic reaction type I?
181. What are the effects of prostaglandins realised from mast cells in allergic reaction type I?
182. What are the features of apoptosis?
183. What are the features of apoptosis?
184. What are the features of apoptosis?
185. What are the features of immunologic stage of immediate type allergic reactions?
186. What are the features of immunologic stage of immediate type allergic reactions?
187. What are the general causes of energy depletion that trigger cell dystrophy?
188. What are the general causes of energy depletion that trigger cell dystrophy?
189. What are the general consequences of apoptosis for the organism?
190. What are the general consequences of necrosis for the organism?
191. What are the general consequences of necrosis?
192. What are the humoral chemotactic substances that are important in emigration of leukocytes?
193. What are the humoral chemotactic substances that are important in emigration of leukocytes?
194. What are the important pathogenetic factors for the initiation of intrinsic pathway of apoptosis?
195. What are the important pathogenetic factors for the initiation of extrinsic pathway of apoptosis?
196. What are the important pathogenetic factors for the initiation of intrinsic pathway of apoptosis?
197. What are the local pathogenetic mechanisms of ischemia?
198. What are the main mediators involved in type II allergic reactions?
199. What are the main pathogenetic mechanisms of intrinsic pathway of apoptosis?
200. What are the main pathogenetical mechanisms of fatty liver in malnutrition?
201. What are the main pathogenetical mechanisms of fatty liver?
202. What are the main pathogenic mechanisms of fatty liver?
203. What are the manifestations of cell necrosis?
204. What are the manifestations of immediate response (pathophysiological stage) in allergic reaction type I?
205. What are the manifestations of immediate response (pathophysiological stage) in allergic reaction type I?
206. What are the manifestations of persistent hyperglycemia?
207. What are the mechanisms of collagen excess reducing in the organ?
208. What are the mechanisms of cytotoxicity in type II allergic reactions?
209. What are the mechanisms of cytotoxicity in type II allergic reactions?
210. What are the mechanisms of hyperplasia?
211. What are the mechanisms of tissular injuries in type III allergic reactions?
212. What are the mechanisms of tissular injuries in type III allergic reactions?
213. What are the mechanisms of tissular injury in type IV allergic reactions?
214. What are the necrosis consequences?
215. What are the necrosis consequences?
216. What are the oxygen – dependent bactericide mechanisms that destroy the pathogenic agent in phagolysosome?
217. What are the oxygen – dependent bactericide products that destroy pathogenic agents in phagolysosome?
218. What are the oxygen – independent bactericide products that destroy pathogenic agents in phagolysosome?
219. What are the oxygen – independent bactericide products that destroy pathogenic agents in phagolysosome?
220. What are the pathogenetic factors of immunodeficiency in protein inanition?
221. What are the pathogenetic factors responsible for initiation of apoptosis?
222. What are the pathogenetic factors responsible for initiation of apoptosis?
223. What are the pathogenetic mechanisms of necrosis due to mitochondrial injury?
224. What are the pathogenetic mechanisms of protein maldigestion in protein inanition?
225. What are the pathogenic factors of cell dystrophy in condition of catecholamine excess?
226. What are the pathogenic factors of hypocalcemia in alkalosis?
227. What are the pathogenic factors of hypokalemia in alkalosis?
228. What are the pathophysiologic mechanisms of defective apoptosis?
229. What are the pathophysiologic mechanisms of defective apoptosis?
230. What are the pathophysiologic mechanisms of reduced apoptosis?
231. What are the pathophysiologic mechanisms of reduced apoptosis?
232. What atrophy can be estimated as endocrine?
233. What atrophy can be estimated as post-hypertrophic?
234. What atrophy is considered as hypofunctional?
235. What atrophy is considered as patholgical?
236. What atrophy is considered as physiological?
237. What biological product can be DAMP (damage associated molecular pattern)?
238. What biological products can be PAMP (pathogen associated molecular pattern)?
239. What can be the consequence of cell dystrophy?
240. What cell is exposed to apotosis in a matur organism?
241. What cell is exposed to apotosis in a matur organism?
242. What cells are exposed to apoptosis in a mature organism?
243. What clinical manifestations can be considered as resolution of the disease?
244. What condition is necessary for complete apoptosis evolution?
245. What condition is necessary for complete apoptosis evolution?
246. What disorders are related with increased apoptosis?
247. What disorders are related with increased apoptosis?
248. What disorders are related with reduced apoptosis?
249. What disorders are related with reduced apoptosis?
250. What does a physiological reaction mean?
251. What does a real atrophy mean?
252. What does represent paradoxical embolism?
253. What does represent the opsonisation process?
254. What does represent “point of no return“ in the course of cell injury?
255. What does represent “point of no return“ in the course of cell injury?
256. What does the aplasia mean?
257. What does the disease represent?
258. What does the dysplasia mean?
259. What does the hyperplasia mean?
260. What does the metaplasia mean?
261. What does the neurotrophic hypertrophy mean?
262. What does the physiological regeneration represent?
263. What does the sclerosis of organ mean?
264. What does the sclerosis of organ mean?
265. What does the vicious cycle in pathogenesis represent?
266. What dyshomeostasis of sodium develops in vasopressin hyposecretion?
267. What dyshomeostasis of sodium develops in vasopressin hypersecretion?
268. What effect exert unfavorable conditions for the body?
269. What effects exert favorable conditions for the body?
270. What effects exert unfavorable conditions for the body?
271. What electrolytic dyshomeostasis of internal environment leads to cell injury?
272. What emboli are endogenous?
273. What endogenous conditions could influence the action of harmful agents?
274. What exogenous conditions could influence the action of harmful agents?
275. What factors can contribute to pathogenesis of diabetic ketoacidosis?
276. What factors induce sclerosis?
277. What general dyshomeostasis leads to cell dystrophy?
278. What hemodynamic changes are characteristic for ischemia?
279. What hemodynamic changes are characteristic for ischemia?
280. What intracellular dyshomeostasis is found in ischemic cells?
281. What intracellular dyshomeostasis is found in ischemic cells?
282. What intracellular electrolytic dyshomeostasis is found in cells which started necrosis?
283. What intracellular electrolytic dyshomeostasis is found in cells which started necrosis?
284. What is a compensatory reaction in hyperglycemia?
285. What is a consequence of lipid malabsorbtion?
286. What is a consequence of lipid malabsorbtion?
287. What is a consequence of protein metabolic changes in liver failure?
288. What is adaptive reaction?
289. What is characteristic arterial hyperemia?
290. What is characteristic for arterial hyperemia?
291. What is characteristic for the immunological stage of type I allergic reactions?
292. What is characteristic for vicious cycle in pathogenesis?
293. What is compensatory reaction?
294. What is one compensatory reaction in hypoglycemia?
295. What is one compensatory reaction in hypoglycemia?
296. What is one compensatory reaction in hypoglycemia?
297. What is pathogenetic therapy?
298. What is reparative reaction?
299. What is symptomatic therapy?
300. What is the cause of increased organ volume in venous hyperemia?
301. What is the cause of venous hyperemia?
302. What is the consequence of carbohydrate malabsorption?
303. What is the consequence of hyperammonaemia in liver failure?
304. What is the definition of cell injury?
305. What is the difference between purulent exudate and transudate?
306. What is the effect of action of continue electrical current on the cell?
307. What is the effect of electric current action on the excitable cells?
308. What is the electrophysiological change of hypokalemia in myocardium?
309. What is the external manifestation of ischemia?
310. What is the external manifestation of ischemia?
311. What is the main pathogenic link of metabolic acidosis installed in aldosterone hyposecretion?
312. What is the mechanism of firm adhesion of leukocytes to the vessel wall?
313. What is the mechanism of hypocalcemia in kidney failure?
314. What is the mechanism of ketogenesis in carbohydrate inanition?
315. What is the mechanism of leukocytes rolling along vessel wall?
316. What is the pathogenesis of acid-base disorder in starvation?
317. What is the pathogenesis of calcium dyshomeostasis in kidney failure?
318. What is the pathogenesis of calcium dyshomeostasis in liver failure?
319. What is the pathogenesis of neuromuscular excitability disorders in hypocalcemia?
320. What is the pathogenesis of neuromuscular excitability disorders in hypercalcemia?
321. What is the pathogenesis of potassium dyshomeostasis in hyperglycemia?
322. What is the pathogenesis of potassium dyshomeostasis in treatment with insulin?
323. What is the pathogenesis of relative hyponatremia?
324. What is the pathogenesis of sodium dyshomeostasis that is found in chronic liver failure?
325. What is the pathogenetic factor that inhibits extrinsic pathway of apoptosis?
326. What is the pathogenetic factor that inhibits extrinsic pathway of apoptosis?
327. What is the pathogenetic mechanism of cell dystrophy in condition of intracellular acidosis?
328. What is the pathogenetic mechanism of neuromyoparalytic arterial hyperemia?
329. What is the pathogenetic mechanism of neuroparalitic arterial hyperemia?
330. What is the pathogenetic role of calcium in necrosis?
331. What is the pathogenetic role of endothelial cell injury in development of white thrombus (primary thrombus)?
332. What is the pathogenetic role of endothelial cell injury in development of white thrombus (primary thrombus)?
333. What is the pathogenetic role of intracellular calcium dyshomeostasis in development of cell necrosis?
334. What is the pathogenetic role of intracellular calcium dyshomeostasis in development of cell necrosis?
335. What is the pathogenetic role of intracellular sodium dyshomeostasis in development of cell necrosis?
336. What is the pathogenetic role of mitochondrial damage in development of cellular pathological processes?
337. What is the pathogenetic role of mitochondrial damage in development of cellular pathological processes?
338. What is the pathogenetic role of reactive oxygen species (ROS) in development of cellular pathologic processes?
339. What is the pathogenetic role of reactive oxygen species (ROS) in development of cellular pathologic processes?
340. What is the pathogenetic role of reactive oxygen species (ROS) in development of cell necrosis?
341. What is the pathogenetic role of reactive oxygen species (ROS) in development of cell apoptosis?
342. What is the pathogenetic role of the complement system activation in type III allergic reactions?
343. What is the pathogenic factor of acid-base disorder in hypoxia?
344. What is the pathogenic factor of acid-base disorder in pulmonary hyperventilation?
345. What is the pathogenic factor of acid-base disorder in pulmonary hypoventilation?
346. What is the pathogenic factor of decreased neuromuscular excitability in acidosis?
347. What is the pathogenic factor of potassium disorder in alkalosis?
348. What is the pathogeny of hypoxic cell injury?
349. What is the pathogeny of hypoxic cell injury?
350. What is the role of C reactive protein in pathogeny of acute inflammation?
351. What is the role of C3b fraction of the complement system in acute inflammation?
352. What is the role of C5a-C9a fraction of the complement system in acute inflammation?
353. What is the role of Hageman factor in pathogeny of acute inflammation?
354. What is the scheme of allergic reaction type I?
355. What is the scheme of allergic reaction type II?
356. What is the scheme of allergic reaction type III?
357. What is the scheme of allergic reaction type IV?
358. What is the sequence of the processes during phagocytosis?
359. What kind of stimuli accelerate proteolysis leading to atrophy?
360. What kind of stimuli activate ubiquitin ligases leading to atrophy?
361. What mediators are involved in developmet of inflammatory reaction in type III allergic reactions?
362. What mediators are released in the result of mast cell degranulation?
363. What mediators with pro-inflammatory effects are produced in the result of activation of Hageman factor in type III allergic reaction?
364. What organ develops insufficient absolute functional collaterals?
365. What oxigendependent bactericide factors are generated by neutrophils?
366. What pathogenetic factor contributes to development of thrombosis?
367. What pathogenetic factor contributes to development of thrombosis?
368. What pathological conditions are characterized by deviation of oxyhemoglobin dissociation curve to the right?
369. What pathological conditions are characterized by deviation of oxyhemoglobin dissociation curve to the left?
370. What pathological conditions are characterized by deviation of oxyhemoglobin dissociation curve to the left?
371. What pathological processes are associated with hypotonic hyperhydration?
372. What pathological processes are associated with isotonic dehydration?
373. What pathological processes are associated with transport hyperlipidemia?
374. What pro-iflammatory mediators are involved in pathogeny of delayed hypersensitivity?
375. What proteins are elevated in the blood in acute phase-response?
376. What types of the physiological hypertrophy are considered as compensatory?
377. What types of the physiological regeneration are considered as qualitatively inadequate?
378. What types of the physiological regeneration are considered as quantitative inadequate?
379. Which acid –base imbalance develops in hypokalemia?
380. Which acid-base imbalance develops in hyperkalemia?
381. Which arachidonic acid metabolites have vasoconstrictive effect?
382. Which arachidonic acid metabolites have vasodilatory effect?
383. Which are pathogenetic factors that contribute to development of thrombosis?
384. Which are pathogenetic factors that contribute to development of thrombosis?
385. Which are the primary sanogenetic mechanisms?
386. Which are the secondary sanogenetic mechanisms?
387. Which blood cells mainly will migrate to the tissue in acute coccic infection?
388. Which blood cells mainly will migrate to the tissue in parasite invasion?
389. Which endogenous enzymes could lead to cell injuries?
390. Which is one of the inflammatory mediators has anti-inflammatory effect?
391. Which is one of the variants of disease’s resolution?
392. Which newly synthesized mediators derived from mast cells and basophils in allergic reaction type I?
393. Which presynthetized mediators are realised from mast cells and basophils during degranulation process in allergic reaction type I?
394. Which regeneration is considered as homeostatical?
395. Which regenerative process is possible in the cell on molecular level?
396. Which regenerative process is possible in the cell on organelle level?
397. Which regenerative processes are possible in the cell on organelle level?
398. Which types of leukocytes have ability to make phagocytosis?
399. Which vessels damage lead to air embolism?
400. Which vessel’s damage leads to air embolism?