**Questions for final exam, SIMU test, MG (semester VI)**

1. What are hematological changes in the peripheral blood in aplastic anemia?
2. What are hematological changes in the peripheral blood in aplastic anemia?
3. What are hematological changes in the peripheral blood in aplastic anemia?
4. What are hematological changes in the peripheral blood in aplastic anemia?
5. What are clinical manifestations in aplastic anemia?
6. What types of anemia are considered macrocytic according to mean corpuscular volume (MCV)> 100 fl?
7. What type of anemia is considered macrocytic according to mean corpuscular volume (MCV)> 100 fl?
8. What types of anemias are considered microcytic according to mean corpuscular volume (MCV) ˂ 80 fl?
9. What types of anemias are considered microcytic according to mean corpuscular volume (MCV) ˂ 80 fl?
10. What types of anemias are considered hyperchromic according to mean corpuscular hemoglobin (MCH) > 35 pg?
11. What types of anemias are considered hyperchromic according to mean corpuscular hemoglobin (MCH) > 35 pg?
12. What types of anemias are considered hypochromic according to mean corpuscular hemoglobin (MCH) ˂ 27 pg?
13. What type of anemia is considered hypochromic according to mean corpuscular hemoglobin (MCH) ˂ 27 pg?
14. What type of anemia is considered hypochromic according to mean corpuscular hemoglobin (MCH) ˂ 27 pg?
15. What types of anemias are considered hyperregenerative according to reticulocyte count in the peripheral blood > 1,5%?
16. What types of anemia is considered hyperregenerative according to reticulocyte count in the peripheral blood > 1,5%?
17. What types of anemias are considered hyporegenerative according to reticulocyte count in the peripheral blood ˂ 1,5%?
18. What biochemical changes in the blood are found in B12 deficiency anemia?
19. What biochemical changes in the blood are found in folate deficiency anemia?
20. What can be possible causes leading to B12 vitamin deficiency in the patients?
21. What can be possible causes leading to B12 vitamin deficiency in the patients?
22. How mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) are changed in B12 deficiency anemia?
23. How mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) are changed in folate deficiency anemia?
24. How mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC) are changed in folate deficiency anemia?
25. How mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC) are changed in B12 deficiency anemia?
26. How mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) change in folate deficiency anemia?
27. How mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) change in B12 deficiency anemia?
28. What are hematological changes in B12 deficiency anemia?
29. What are hematological changes in folate deficiency anemia?
30. What is a hematological change in B12 deficiency anemia?
31. What are hematological changes in B12 deficiency anemia?
32. What are hematological changes in B12 deficiency anemia?
33. What are gastrointestinal manifestations in B12 deficiency?
34. What is the pathogenetic mechanism of neurologic syndrome in B12 deficiency?
35. What is the pathogenetic mechanism of neurologic syndrome in B12 deficiency?
36. What is the pathogenetic mechanism of anemic syndrome in B12 deficiency?
37. What is the pathogenetic mechanism of gastrointestinal syndrome in B12 deficiency?
38. What are pathogenetic mechanisms of neurologic syndrome in B12 deficiency?
39. What is the pathogenetic mechanism of atipical mitosis in B12 deficiency anemia?
40. What is the pathophysiological mechanism of clinical manifestations in folate deficiency anemia?
41. What is the pathophysiological mechanism of clinical manifestations in folate deficiency anemia?
42. What are pathophysiological mechanisms of clinical manifestations in B12 deficiency anemia?
43. What are pathophysiological mechanisms of clinical manifestations in B12 deficiency anemia?
44. What are etiological factors of folate deficiency anemia?
45. What is a cause of iron deficiency anemia?
46. What are causes of iron deficiency anemia?
47. What is the pathogenetic mechanism of iron deficiency anemia in chronic inflammation?
48. What is the pathogenetic mechanism of iron deficiency anemia in chronic inflammation?
49. What are pathogenetic factors involved in development of iron deficiency anemia?
50. What is a pathogenetic factor involved in development of iron deficiency anemia?
51. What is a pathogenetic factor involved in development of iron deficiency anemia?
52. What are hematological changes in iron deficiency anemia?
53. How mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) are changed in iron deficiency anemia?
54. How mean corpuscular hemoglobin concentration (MCHC) and mean corpuscular hemoglobin (MCH) are changed in iron deficiency anemia?
55. How mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC) are changed in iron deficiency anemia?
56. How mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH) and seric ferritin are changed in iron deficiency anemia?
57. How hemoglobin (Hb), mean corpuscular hemoglobin (MCH) and seric ferritin are changed in iron deficiency anemia?
58. How hemoglobin (Hb), mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC) are changed in iron deficiency anemia?
59. How mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC) are changed in iron deficiency anemia?
60. What are hematological changes in the peripheral blood in absolute primary erythrocytosis?
61. What are hematological changes in the peripheral blood in absolute primary erythrocytosis?
62. What is a hematological change in the peripheral blood in absolute secondary erythrocytosis?
63. What are hematological changes in the peripheral blood in relative erythrocytosis?
64. What is the pathogenetic mechanism of absolute primary erythrocytosis?
65. How hematocrit (Ht) and mean corpuscular volume (MCV) of erythrocytes is changed in absolute primary erythrocytosis?
66. How hematocrit (Ht) and mean corpuscular volume (MCV) of erythrocytes is changed in absolute secondary erythrocytosis?
67. How hematocrit (Ht) and mean corpuscular volume (MCV) of erythrocytes is changed in relative erythrocytosis?
68. How hematocrit (Ht) and mean corpuscular hemoglobin concentration (MCHC) is changed in absolute primary erythrocytosis?
69. How hematocrit (Ht) and mean corpuscular hemoglobin concentration (MCHC) is changed in absolute secondary erythrocytosis?
70. How change circulatory blood volume (CBV) and serum iron in absolute primary erythrocytosis?
71. How change circulatory blood volume (CBV) and serum iron in relative erythrocytosis?
72. How change circulatory blood volume (CBV) and serum iron in absolute primary erythrocytosis?
73. How change circulatory blood volume (CBV) and serum iron in absolute secondary erythrocytosis?
74. How change circulatory blood volume (CBV) and serum iron in relative erythrocytosis?
75. On the basis of what cells there is increased hematocrit in patients with absolute primary erythrocytosis?
76. How the erythropoietin level is changed in absolute primary erythrocytosis?
77. How the erythropoietin level is changed in absolute secondary erythrocytosis?
78. How the erythropoietin level is changed in relative erythrocytosis?
79. What clinical manifestations are characteristic in patient with Vaquez disease?
80. What clinical manifestations are characteristic in patient with Vaquez disease?
81. What are pathophysiological mechanisms of autoimmune hemolytic anemia?
82. What are biochemical changes in intracellular hemolysis?
83. What are biochemical changes in intravascular hemolysis?
84. What biochemical change is found in both, intravascular and intracellular hemolysis?
85. What are biochemical changes characteristic for intravascular hemolysis?
86. What is a characteristic pathophysiological mechanism for intravascular hemolysis?
87. What are common biochemical changes which are found in both, intravascular and extravascular hemolysis?
88. What is the pathophysiological mechanisms of absolute leukocytosis?
89. What are the features of relative leukocytosis?
90. What is the pathophysiological mechanism of relative leukocytosis?
91. What conditions are associated with relative leukocytosis?
92. What leukocytosis are considered physiological?
93. What is the cause of neutrophilia?
94. What is the pathophysiological mechanism of neutrophilia?
95. What does represent neutrophilia with “left nuclear shift”?
96. What form of neutrophils is found in the peripheral blood in neutrophilia with “left nuclear shift”?
97. What does represent neutrophilia with “right nuclear shift”?
98. What is the other clinical term to define neutrophilia with “left nuclear shift” hyperregenerative type?
99. What are the signs of neutrophil degeneration?
100. What is the cause of neutrophilia with “left nuclear shift” hyperregenerative type?
101. What are the causes of eosinophilia?
102. In what conditions can be found absolute lymphocytosis?
103. In what conditions can be found absolute lymphocytosis?
104. What hematological disorders are associated with relative lymphocytosis?
105. What is the clinical significance of relative lymphocytosis in the patient?
106. What is the pathophysiological mechanism of absolute lymphocytosis?
107. What is the pathophysiological mechanism of relative lymphocytosis?
108. What does represent agranulocytosis?
109. What does represent agranulocytosis?
110. When can be found primary agranulocytosis?
111. When can be found secondary agranulocytosis?
112. What immunoglobulin are involved in development of secondary agranulocytosis?
113. What is the pathophysiological mechanism of primary agranulocytosis?
114. What is the pathophysiological mechanism of secondary agranulocytosis?
115. What is the pathophysiological mechanism of secondary agranulocytosis?
116. What is the pathophysiological mechanism of secondary agranulocytosis?
117. What is the pathophysiological mechanism of autoimmune neutropenia?
118. How is defined hemoblastosis?
119. What pathological processes at the level of hematopoietic bone marrow are present in acute leucosis?
120. What does mean hyperplasia of hematopoietic bone marrow in pathogenesis of acute leucosis?
121. What does mean metaplasia of hematopoietic bone marrow in pathogenesis of acute leucosis?
122. In what pathologic conditions can be attested overload of the heart with resistance?
123. In what pathologic conditions can be attested overload of the heart with resistance?
124. In what pathologic conditions can be attested overload of the heart with resistance?
125. In what pathologic conditions can be attested overload of the heart with volume?
126. What is a possible cause of right heart failure?
127. What are characteristic manifestations for left heart failure?
128. What are characteristic manifestations for right heart failure?
129. What are characteristic manifestations for right heart failure?
130. What is one of immediate cardiac compensatory reaction in heart failure?
131. What are immediate cardiac compensatory reactions in heart failure?
132. What are immediate cardiac compensatory reactions in heart failure?
133. What is one of late cardiac compensatory reaction in heart failure?
134. What is one of late cardiac compensatory reaction in heart failure?
135. What is one of immediate extracardiac compensatory mechanism in heart failure?
136. What is one of immediate extracardiac compensatory mechanism in heart failure?
137. What is one of immediate extracardiac compensatory mechanism in heart failure?
138. What are immediate extracardiac compensatory mechanism in heart failure?
139. What are late extracardiac compensatory mechanisms in heart failure?
140. What is one of late extracardiac compensatory mechanism in heart failure?
141. How is performed predominant homeometric hyperfunction of the myocardium?
142. How is performed predominant homeometric hyperfunction of the myocardium?
143. How is performed predominant heterometric hyperfunction of the myocardium?
144. How is performed predominant heterometric hyperfunction of the myocardium?
145. How does the structure of hypertrophic myocardium change?
146. How does the structure of hypertrophic myocardium change?
147. How does the systolic volume and cardiac output change in heart failure?
148. What are the causes of hypervolemia in chronic heart failure?
149. What are the causes of hypervolemia in chronic heart failure?
150. What is the cause of hypervolemia in chronic heart failure?
151. What are the causes of hypervolemia in chronic heart failure?
152. What is the cause of hypervolemia in chronic heart failure?
153. What are the consequences of venous stasis in circulatory insufficiency?
154. What are the consequences of venous stasis in circulatory insufficiency?
155. What are the consequences of venous stasis in circulatory insufficiency?
156. What is one of the consequences of portal hypertension in heart failure?
157. What are the consequences of portal hypertension in heart failure?
158. What are the consequences of portal hypertension in heart failure?
159. What are the pathogenic factors of cardiac edemas?
160. What are the pathogenic factors of cardiac edemas?
161. What is one of the pathogenic factors of cardiac edemas?
162. What are the causes of hyperventilation?
163. What are the causes of hyperventilation?
164. What are the causes of hypoventilation?
165. What are the causes of hypoventilation?
166. What does hypercapnia represent?
167. What does hypoxemia represent?
168. What does hyperpnea mean?
169. What does polypnea represent?
170. 170.What does bradypnea represent?
171. What does hyperventilation represent?
172. What ventilatory parameters of the lungs are changed in hyperventilation?
173. What does hypoventilation represent?
174. What does pulmonary restriction mean?
175. What are the causes of extrapulmonary restriction?
176. What does the intraparenchymatouse pulmonary restriction mean?
177. What are the causes of intraparenchymatouse restrictive lung diseases?
178. In what disorders is attested shallow and accelerated breathing?
179. In what disorders is attested shallow and accelerated breathing?
180. What does the pulmonary emphysema mean?
181. What are the sources of proteolytic enzymes which damage pulmonary alveoli?
182. What is characteristic for pulmonary emphysema?
183. What is the main pathogenetic link in pulmonary emphysema?
184. What is the main pathogenetic link in pulmonary emphysema?
185. One of the characteristic signs of pulmonary emphysema is the increasing of thoracic cavity volume (“barrel chest”). What is the explanation of this sign in pulmonary emphysema?
186. What are the causes of transudate accumulation into the pleural cavity?
187. What are the causes of exudate accumulation into the pleural cavity?
188. What does the pneumosclerosis mean?
189. What are the pathogenetic mechanisms of pneumosclerosis?
190. What are the manifestations of pneumosclerosis?
191. What are the manifestations of pneumosclerosis?
192. What does pulmonary atelectasis mean?
193. What does pulmonary atelectasis mean?
194. What types of atelectasis are?
195. What does pulmonary obstruction mean?
196. What factors provoke obstruction of respiratory superior airways?
197. What factors provoke obstruction of inferior respiratory airways?
198. What factors provoke obstruction of inferior respiratory airways?
199. What does represent pulmonary edema?
200. What factors can trigger development of pulmonary edema?
201. What factors can trigger development of pulmonary edema?
202. What determines the clinical manifestations of secondary hypocorticism?
203. What are the pathogenetic principles of tertiary hypocorticism therapy?
204. What are the principles of feedback regulation of the hypothalamus-pituitary-adrenal cortical axis?
205. Patient C., suffering from chronic non-specific polyarthritis for a long time, was treated with high-dose glucocorticosteroids. Subsequently radiography revealed atrophy of both adrenals. What is the pathogenesis?
206. Lack of glucocorticoid hormones is clinically manifested by vascular disturbances. How does vascular tone change in glucocorticosteroid hyposecretion?
207. Lack of glucocorticoid hormones is clinically manifested by disturbances in heart function. How cardiac functions are altered in hypocorticism?
208. All three forms of hypocorticism (primary, secondary and tertiary) represent damage to the hypothalamus-pituitary-adrenal axis at different levels. The level of the disorder can be determined by measuring hormones in the blood. What is the hormonal background in primary hypocorticism?
209. Primary and secondary hypocorticism have most analogous clinical manifestations. What is a characteristic clinical manifestation for primary hypocorticism?
210. Primary and secondary hypocorticism have most analogous clinical manifestations. What is a characteristic clinical manifestation for secondary hypocorticism?
211. What are the risks of stress for people with hypocorticism?
212. One of the vital risks of stress for people with hypocorticism is arterial collapse. What is the pathogenesis?
213. Glucocorticosteroid hormones are important in immunity and inflammation. How does the inflammatory reaction proceed in people with hypocorticism?
214. Glucocorticosteroid hormones are important in immunity and inflammation. What is the inflammatory reaction in people with hypercorticism?
215. Glucocorticosteroid hormones are important in immunity and inflammation. What is the inflammatory reaction in people with hypercorticism?
216. In patient C., suffering from primary objective hypocorticism, hyperpigmentation of the skin was observed. What is the pathogenesis?
217. In patient C., primary hypocorticism was found. What is the possible etiology?
218. What is the possible cause of secondary hypercorticism?
219. What determines the clinical manifestations of tertiary hypercorticism?
220. Glucocorticosteroids in physiologic and pharmacologic doses have ambiguous influence on immunity. How does the immune system change in glucocorticosteroid hypersecretion?
221. Specific clinical features for hypercorticism are excessive lipid deposits in certain areas of the body - "moon face", "buffalo hump", deposits on the trunk. What is the pathogenesis of fatty tissue hypertrophy in these areas?
222. Glucocorticosteroids are also involved in protein metabolism. How does protein metabolism change in glucocorticosteroid hypersecretion?
223. Clinical examination of patient D. with hypercorticism demonstrates edema on the legs. What is the possible pathogenesis?
224. Hyperaldosteronism has several pathogenetic mechanisms, on which therapeutic strategies will depend. What is the pathogenesis of secondary hyperaldosteronism in liver failure?
225. Insulin simultaneously has effects on glycemia and peripheral glucose utilization. What is the mechanism of increased peripheral utilization of glucose by insulin?
226. In type I diabetes mellitus, the secretion of glucagon is increased along with insulin deficiency. What are the effects of glucagon hypersecretion?
227. Glucose is an osmotically active substance. What are the effects and consequences of excessive hyperglycemia in type I diabetes mellitus?
228. What are the effects and consequences of excessive hyperglycemia in type I diabetes mellitus?
229. The paradoxical clinical phenomenon for type I diabetes mellitus is weight deficit concomitant with increased appetite. What is the pathogenesis of hyperrexia?
230. Weight loss is characteristic for type I diabetes mellitus. What is the pathogenesis?
231. Glucose uptake from the blood depends on the type of membrane transporters (GLUT-1-4), which are insulin-dependent or insulin-independent. Which cells have insulin-dependent Glut-4 receptors?
232. Patient S. with type I diabetes mellitus complains of erectile dysfunction. What is the pathogenesis?
233. Primary symptoms of patients with type I diabetes mellitus are thirst, frequent and excessive water consumption, excessive urination. What is the cause of polydipsia in type I diabetes mellitus?
234. What is the cause of polydipsia in type I diabetes mellitus?
235. Hematologic examination of patient C., 24 years old with type I diabetes mellitus revealed: erythrocytes - 6.1012/L, hematocrit - 60%. What is the pathogenesis of these disorders?
236. Patient C., 24 years old, addressed to the endocrinologist with the presumptive diagnosis "type II diabetes mellitus". What are the signs that differentiate type II from type I diabetes?
237. What is the main pathogenetic link of secondary hypothyroidism?
238. What are the pathogenetic principles of secondary hypothyroidism therapy?
239. What is the hormonal parent in secondary hypothyroidism?
240. What is the clinical manifestation of tertiary hyperthyroidism?
241. A patient with hyperthyroidism has been diagnosed with Graves' disease. What type of allergic reaction is Graves' disease?
242. Patient D., 45 years old, addressed to the endocrinologist because of enlargement of the thyroid gland ("goiter"). Biochemical investigations showed: thyroid hormone - increased; TSH in the blood - increased. Scintigraphy demonstrates exaggerated uptake of radioactive iodine uniformly throughout the thyroid parenchyma. What is the pathogenesis of this pathology?
243. What is the mechanism of hyposecretion of antidiuretic hormone in pituitary foot trauma?
244. What is the pathogenetic mechanism of glomerular haematuria?
245. In which conditions is leukocyturia present?
246. In which conditions is lipiduria present?
247. What factors cause decreased water reabsorption in the proximal renal tubules?
248. What factors cause decreased water reabsorption in the distal and collecting tubules?
249. What factors cause decreased distal reabsorption of Na+ ions?
250. The result of which condition is tubular proteinuria?
251. Which factors cause decreased glucose reabsorption?
252. Which conditions cause aminoaciduria?
253. In which pathologies hyposthenuria is present?
254. In which pathologies hypersthenuria is present?
255. In which cases isosthenuria is present?
256. Which disorders include nephrotic syndrome?
257. What pathologic manifestations does nephritic syndrome include?
258. What processes cause proximal canalicular acidosis?
259. What processes cause distal canalicular acidosis?
260. Which factors stimulate renin secretion?
261. What are the endocrine functions of the kidney?
262. What are the prerenal causes of acute renal failure?
263. What are the intrinsic causes of acute renal failure?
264. What are the intrinsic causes of acute renal failure?
265. What are the postrenal cause of acute renal failure?
266. What are the main syndromes in acute renal failure?
267. What are the manifestations of urinary syndrome in acute renal failure?
268. What are the manifestations of the humoral syndrome in acute renal failure?
269. What are the manifestations of the clinical syndrome in acute renal failure?
270. What are the causes of chronic renal failure?
271. What is the sequence of acute renal failure?
272. How does the glomerular filtration rate change in glomerulopathies?
273. How does the glomerular filtration rate change in hypervolemia?
274. How does the glomerular filtration rate change in hypovolemia?
275. How does diuresis change in hypoproteinemia?
276. How does diuresis change in hyperproteinaemia?
277. How does diuresis change in conditions of decreased cardiac output?
278. What is the mechanism of hypercoagulability in nephrotic syndrome?
279. What is the mechanism of hyperlipidemia in nephrotic syndrome?
280. What is the mechanism of loss of renal filter size selectivity?
281. What is the mechanism of loss of electrostatic selectivity of the renal filter?
282. What are the pathogenetic mechanisms of decreased GFR in acute renal failure?
283. What are the consequences of urinary obstruction?
284. What are the consequences of atrophic gastritis?
285. What are the consequences of atrophic gastritis?
286. What are the mechanisms of pancreatic autoaggression?
287. What is the role of alcohol in the pathogenesis of pancreatitis?
288. What are the possible consequences of sialorrhea?
289. What are the possible consequences of sialorrhea?
290. What is one of the consequences of sialorrhea?
291. Which exogenous factor causes stomach hypersecretion?
292. Which endogenous factor causes stomach hypersecretion?
293. How does the evacuation function of the stomach change in hyperacidity hypersecretion?
294. How does stomach motor function change in hypersecretion with hyperacidity?
295. How does bowel transit change in hypersecretion with stomach hyperacidity?
296. What is *achlorhydria*?
297. What can cause achlorhydria?
298. What can cause achlorhydria?
299. What are the consequences of stomach hypoacidity?
300. What are the consequences of stomach hypoacidity?
301. What can be the consequences of vomiting?
302. What biochemical tests reflect cholestatic syndrome in liver failure?
303. Which biochemical tests reflect the protein-synthetic function of the liver?
304. Which biochemical test reflects hepatocyte injury?
305. Which pathologic processes lead to the development of venous hyperemia in the liver?
306. What are the mechanisms of hepatocyte injury induced by hemodynamic factors?
307. What are the mechanisms of alcohol-induced toxic hepatocyte injury?
308. Which cells are responsible for releasing pro-fibrinogenic cytokines and triggering hepatocyte fibrosis in toxic liver injury?
309. Which cells are responsible for the excess production of collagen fibers and extracellular matrix in toxic liver injury?
310. Which cells in the liver parenchyma can be transformed into myofibroblasts and trigger liver fibrosis by excessive collagenogenesis?
311. What is the pathogenic chain of liver fibrosis?
312. What is one of the main pathogenetic links of liver fibrosis?
313. What are the main pathogenetic links of liver fibrosis?
314. What blood biochemical changes reflect deregulated protein metabolism in liver failure?
315. What blood biochemical changes reflect disturbances of protein metabolism in liver failure?
316. How do the levels of branched and aromatic amino acids change in the blood of the liver failure patient?
317. What are the consequences of reduced amino acid transamination processes in liver failure?
318. What is the consequence of reduced amino acid transamination processes in liver failure?
319. What is the mechanism of increased serum levels of aromatic amino acids in liver failure?
320. What is the mechanism of reduced serum levels of branched amino acids in liver failure
321. How does carbohydrate metabolism change in liver failure?
322. What are the clinical manifestations that reflect imbalance of glucose metabolism in liver failure?
323. What blood biochemical changes reflect imbalance of glucose metabolism in liver failure?
324. What are the pathogenetic mechanisms of fasting hypoglycemia in liver failure?
325. What is the pathogenetic mechanism of postprandial hyperglycemia in liver failure?
326. Which pathogenetic factors contribute to development of fatty liver dystrophy in liver failure?
327. Accumulation of which products denotes metabolic acidosis in liver failure?
328. What are the pathogenetic mechanisms of metabolic acidosis in liver failure?
329. What is the pathogenetic mechanism of metabolic acidosis in liver failure?
330. What are the pathogenetic mechanisms of metabolic alkalosis in liver failure?
331. What are the pathogenetic factors responsible for excessive ketone body production in liver failure?
332. What is the mechanism of NADPH deficiency in hepatocytes that contributes to the development of ketonemia in liver failure?
333. What is the mechanism of oxaloacetate deficiency in hepatocytes that contributes to the development of ketonemia in liver failure?
334. What is the pathogenetic role of NADPH deficiency in the development of ketonemia in liver failure?
335. What is the pathogenetic role of oxaloacetate deficiency in the development of ketonemia in liver failure?
336. What causes galactosemia in liver failure?
337. What are the specific clinical manifestations of fat-soluble vitamin D deficiency in liver failure?
338. What is one of the specific clinical manifestations for fat-soluble vitamin A deficiency in liver failure?
339. What are the pathogenetic mechanisms of hypocalcemia in liver failure?
340. What is one of the pathogenetic mechanisms of hypocalcemia in liver failure?
341. Patients with liver failure may have skin changes such as acne, hirsutism and full moon facies. What is the pathogenesis of these symptoms?
342. Which hematologic changes are characteristic of hypersplenism in liver failure?
343. What is the osmotic mechanism of ascites?
344. What is the oncotic mechanism of ascites?
345. What is the hydrostatic mechanism of ascites?
346. What causes activation of the renin- angiotensin- aldosterone system in patients with portal hypertension?
347. What is one of the causes of activation of the renin- angiotensin- aldosterone system in patients with portal hypertension?
348. in hepatic encephalopathy caused by hyperammonaemia?
349. What is one of the central nervous system changes in hepatic encephalopathy caused by hyperammonaemia?
350. What is the pathogenetic factor of astrocyte edema in ammonia encephalopathy?
351. What are the prehepatic causes of portal hypertension?
352. What are the hepatic causes of portal hypertension?
353. What are the posthepatic causes of portal hypertension?
354. What is cholestasis?
355. Changes in which biochemical test reflect moderate cholestasis?
356. What is cholemia?
357. What is an acholia?
358. What is the mechanism of jaundice caused by increased blood levels of unconjugated bilirubin?
359. What is the mechanism of jaundice caused by increased blood levels of conjugated bilirubin?
360. What are the properties of unconjugated bilirubin?
361. What is one of the properties of conjugated bilirubin?
362. What are the causes of prehepatic jaundice?
363. What are the causes of hepatic jaundice?
364. What are the causes of posthepatic jaundice?
365. What is the pathogenetic chain of prehepatic jaundice?
366. What are the features of prehepatic jaundice?
367. What are the features of prehepatic jaundice?
368. What are the features of prehepatic jaundice?
369. What are the features of prehepatic jaundice?
370. How does bile metabolism change in hemolytic jaundice?
371. What is one of the blood biochemical changes seen in prehepatic jaundice in intracellular hemolytic anemia?
372. What is the mechanism of intense faeces colour in prehepatic jaundice?
373. What blood biochemical changes are seen in liver jaundice?
374. What are the features of liver jaundice?
375. What are the features of liver jaundice?
376. What are the features of liver jaundice?
377. What are the features of liver jaundice?
378. What is the mechanism of faeces discoloration in patients with hepatic jaundice?
379. What are the mechanisms of intense urine staining in patients with hepatic jaundice?
380. What is the mechanism of hyperbilirubinemia in patients with hepatic jaundice?
381. What is the mechanism of hyperbilirubinemia in patients with hepatic jaundice?
382. Which process of bilirubin metabolism is affected in posthepatic jaundice?
383. What are the features of posthepatic jaundice?
384. What are the features of posthepatic jaundice?
385. What is one of the features of posthepatic jaundice?
386. What is the cause of dark urine color in patients with posthepatic jaundice?
387. What is the pathogenetic chain that explain the faeces discoloration in posthepatic jaundice?
388. What is the pathogenetic factor of faeces discoloration in posthepatic jaundice?
389. What is one of the features of posthepatic jaundice?
390. What blood biochemical changes are seen in posthepatic jaundice?
391. How do faeces change in patients with posthepatic jaundice?
392. What are the mechanisms of bleeding syndrome in patients with posthepatic jaundice?
393. What is the cause of bleeding syndrome in patients with hepatic and posthepatic jaundice?
394. What is the cause of bleeding syndrome in patients with hepatic and posthepatic jaundice?
395. What are the cardiovascular changes and their mechanisms in cholemic syndrome?
396. What is one of the cardiovascular changes and mechanism in cholemic syndrome?
397. What is one of the cardiovascular changes and mechanism in cholemic syndrome?
398. What is the pathophysiologic mechanism of pruritus in patients with cholemic syndrome?
399. What biochemical changes are seen in the blood in cholemic syndrome?
400. What is the mechanism of cholestatic syndrome in posthepatic jaundice?