**Test SIMU for Medicine, totalisation II (2024-2025)**

* + - 1. What determines the clinical manifestations of secondary hypocorticism?

1. What are the pathogenetic principles of tertiary hypocorticism therapy?
2. What are the principles of feedback regulation of the hypothalamus-pituitary-adrenal cortical axis?
3. 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?
4. Lack of glucocorticoid hormones is clinically manifested by vascular disturbances. How does vascular tone change in glucocorticosteroid hyposecretion?
5. Lack of glucocorticoid hormones is clinically manifested by disturbances in heart function. How cardiac functions are altered in hypocorticism?
6. 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?
7. Primary and secondary hypocorticism have most analogous clinical manifestations. What is a characteristic clinical manifestation for primary hypocorticism?
8. Primary and secondary hypocorticism have most analogous clinical manifestations. What is a characteristic clinical manifestation for secondary hypocorticism?
9. What are the risks of stress for people with hypocorticism?
10. One of the vital risks of stress for people with hypocorticism is arterial collapse. What is the pathogenesis?
11. Glucocorticosteroid hormones are important in immunity and inflammation. How does the inflammatory reaction proceed in people with hypocorticism?
12. Glucocorticosteroid hormones are important in immunity and inflammation. What is the inflammatory reaction in people with hypercorticism?
13. Glucocorticosteroid hormones are important in immunity and inflammation. What is the inflammatory reaction in people with hypercorticism?
14. In patient C., suffering from primary objective hypocorticism, hyperpigmentation of the skin was observed. What is the pathogenesis?
15. In patient C., primary hypocorticism was found. What is the possible etiology?
16. What is the possible cause of secondary hypercorticism?
17. What determines the clinical manifestations of tertiary hypercorticism?
18. Glucocorticosteroids in physiologic and pharmacologic doses have ambiguous influence on immunity. How does the immune system change in glucocorticosteroid hypersecretion?
19. 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?
20. Glucocorticosteroids are also involved in protein metabolism. How does protein metabolism change in glucocorticosteroid hypersecretion?
21. Clinical examination of patient D. with hypercorticism demonstrates edema on the legs. What is the possible pathogenesis?
22. Hyperaldosteronism has several pathogenetic mechanisms, on which therapeutic strategies will depend. What is the pathogenesis of secondary hyperaldosteronism in liver failure?
23. Insulin simultaneously has effects on glycemia and peripheral glucose utilization. What is the mechanism of increased peripheral utilization of glucose by insulin?
24. In type I diabetes mellitus, the secretion of glucagon is increased along with insulin deficiency. What are the effects of glucagon hypersecretion?
25. Glucose is an osmotically active substance. What are the effects and consequences of excessive hyperglycemia in type I diabetes mellitus?
26. What are the effects and consequences of excessive hyperglycemia in type I diabetes mellitus?
27. The paradoxical clinical phenomenon for type I diabetes mellitus is weight deficit concomitant with increased appetite. What is the pathogenesis of hyperrexia?
28. Weight loss is characteristic for type I diabetes mellitus. What is the pathogenesis?
29. 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?
30. Patient S. with type I diabetes mellitus complains of erectile dysfunction. What is the pathogenesis?
31. 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?
32. What is the cause of polydipsia in type I diabetes mellitus?
33. 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?
34. 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?
35. What is the main pathogenetic link of secondary hypothyroidism?
36. What are the pathogenetic principles of secondary hypothyroidism therapy?
37. What is the hormonal parent in secondary hypothyroidism?
38. What is the clinical manifestation of tertiary hyperthyroidism?
39. A patient with hyperthyroidism has been diagnosed with Graves' disease. What type of allergic reaction is Graves' disease?
40. 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?
41. What is the mechanism of hyposecretion of antidiuretic hormone in pituitary foot trauma?
42. What is the pathogenetic mechanism of glomerular haematuria?
43. In which conditions is leukocyturia present?
44. In which conditions is lipiduria present?
45. What factors cause decreased water reabsorption in the proximal renal tubules?
46. What factors cause decreased water reabsorption in the distal and collecting tubules?
47. What factors cause decreased distal reabsorption of Na+ ions?
48. The result of which condition is tubular proteinuria?
49. Which factors cause decreased glucose reabsorption?
50. Which conditions cause aminoaciduria?
51. In which pathologies hyposthenuria is present?
52. In which pathologies hypersthenuria is present?
53. In which cases isosthenuria is present?
54. Which disorders include nephrotic syndrome?
55. What pathologic manifestations does nephritic syndrome include?
56. What processes cause proximal canalicular acidosis?
57. What processes cause distal canalicular acidosis?
58. Which factors stimulate renin secretion?
59. What are the endocrine functions of the kidney?
60. What are the prerenal causes of acute renal failure?
61. What are the intrinsic causes of acute renal failure?
62. What are the intrinsic causes of acute renal failure?
63. What are the postrenal cause of acute renal failure?
64. What are the main syndromes in acute renal failure?
65. What are the manifestations of urinary syndrome in acute renal failure?
66. What are the manifestations of the humoral syndrome in acute renal failure?
67. What are the manifestations of the clinical syndrome in acute renal failure?
68. What are the causes of chronic renal failure?
69. What is the sequence of acute renal failure?
70. How does the glomerular filtration rate change in glomerulopathies?
71. How does the glomerular filtration rate change in hypervolemia?
72. How does the glomerular filtration rate change in hypovolemia?
73. How does diuresis change in hypoproteinemia?
74. How does diuresis change in hyperproteinaemia?
75. How does diuresis change in conditions of decreased cardiac output?
76. What is the mechanism of hypercoagulability in nephrotic syndrome?
77. What is the mechanism of hyperlipidemia in nephrotic syndrome?
78. What is the mechanism of loss of renal filter size selectivity?
79. What is the mechanism of loss of electrostatic selectivity of the renal filter?
80. What are the pathogenetic mechanisms of decreased GFR in acute renal failure?
81. What are the consequences of urinary obstruction?
82. What are the consequences of atrophic gastritis?
83. What are the consequences of atrophic gastritis?
84. What are the mechanisms of pancreatic autoaggression?
85. What is the role of alcohol in the pathogenesis of pancreatitis?
86. What are the possible consequences of sialorrhea?
87. What are the possible consequences of sialorrhea?
88. What is one of the consequences of sialorrhea?
89. Which exogenous factor causes stomach hypersecretion?
90. Which endogenous factor causes stomach hypersecretion?
91. How does the evacuation function of the stomach change in hyperacidity hypersecretion?
92. How does stomach motor function change in hypersecretion with hyperacidity?
93. How does bowel transit change in hypersecretion with stomach hyperacidity?
94. What is *achlorhydria*?
95. What can cause achlorhydria?
96. What can cause achlorhydria?
97. What are the consequences of stomach hypoacidity?
98. What are the consequences of stomach hypoacidity?
99. What can be the consequences of vomiting?
100. What biochemical tests reflect cholestatic syndrome in liver failure?
101. Which biochemical tests reflect the protein-synthetic function of the liver?
102. Which biochemical test reflects hepatocyte injury?
103. Which pathologic processes lead to the development of venous hyperemia in the liver?
104. What are the mechanisms of hepatocyte injury induced by hemodynamic factors?
105. What are the mechanisms of alcohol-induced toxic hepatocyte injury?
106. Which cells are responsible for releasing pro-fibrinogenic cytokines and triggering hepatocyte fibrosis in toxic liver injury?
107. Which cells are responsible for the excess production of collagen fibers and extracellular matrix in toxic liver injury?
108. Which cells in the liver parenchyma can be transformed into myofibroblasts and trigger liver fibrosis by excessive collagenogenesis?
109. What is the pathogenic chain of liver fibrosis?
110. What is one of the main pathogenetic links of liver fibrosis?
111. What are the main pathogenetic links of liver fibrosis?
112. What blood biochemical changes reflect deregulated protein metabolism in liver failure?
113. What blood biochemical changes reflect disturbances of protein metabolism in liver failure?
114. How do the levels of branched and aromatic amino acids change in the blood of the liver failure patient?
115. What are the consequences of reduced amino acid transamination processes in liver failure?
116. What is the consequence of reduced amino acid transamination processes in liver failure?
117. What is the mechanism of increased serum levels of aromatic amino acids in liver failure?
118. What is the mechanism of reduced serum levels of branched amino acids in liver failure
119. How does carbohydrate metabolism change in liver failure?
120. What are the clinical manifestations that reflect imbalance of glucose metabolism in liver failure?
121. What blood biochemical changes reflect imbalance of glucose metabolism in liver failure?
122. What are the pathogenetic mechanisms of fasting hypoglycemia in liver failure?
123. What is the pathogenetic mechanism of postprandial hyperglycemia in liver failure?
124. Which pathogenetic factors contribute to development of fatty liver dystrophy in liver failure?
125. Accumulation of which products denotes metabolic acidosis in liver failure?
126. What are the pathogenetic mechanisms of metabolic acidosis in liver failure?
127. What is the pathogenetic mechanism of metabolic acidosis in liver failure?
128. What are the pathogenetic mechanisms of metabolic alkalosis in liver failure?
129. What are the pathogenetic factors responsible for excessive ketone body production in liver failure?
130. What is the mechanism of NADPH deficiency in hepatocytes that contributes to the development of ketonemia in liver failure?
131. What is the mechanism of oxaloacetate deficiency in hepatocytes that contributes to the development of ketonemia in liver failure?
132. What is the pathogenetic role of NADPH deficiency in the development of ketonemia in liver failure?
133. What is the pathogenetic role of oxaloacetate deficiency in the development of ketonemia in liver failure?
134. What causes galactosemia in liver failure?
135. What are the specific clinical manifestations of fat-soluble vitamin D deficiency in liver failure?
136. What is one of the specific clinical manifestations for fat-soluble vitamin A deficiency in liver failure?
137. What are the pathogenetic mechanisms of hypocalcemia in liver failure?
138. What is one of the pathogenetic mechanisms of hypocalcemia in liver failure?
139. Patients with liver failure may have skin changes such as acne, hirsutism and full moon facies. What is the pathogenesis of these symptoms?
140. Which hematologic changes are characteristic of hypersplenism in liver failure?
141. What is the osmotic mechanism of ascites?
142. What is the oncotic mechanism of ascites?
143. What is the hydrostatic mechanism of ascites?
144. What causes activation of the renin- angiotensin- aldosterone system in patients with portal hypertension?
145. What is one of the causes of activation of the renin- angiotensin- aldosterone system in patients with portal hypertension?
146. in hepatic encephalopathy caused by hyperammonaemia?
147. What is one of the central nervous system changes in hepatic encephalopathy caused by hyperammonaemia?
148. What is the pathogenetic factor of astrocyte edema in ammonia encephalopathy?
149. What are the prehepatic causes of portal hypertension?
150. What are the hepatic causes of portal hypertension?
151. What are the posthepatic causes of portal hypertension?
152. What is cholestasis?
153. Changes in which biochemical test reflect moderate cholestasis?
154. What is cholemia?
155. What is an acholia?
156. What is the mechanism of jaundice caused by increased blood levels of unconjugated bilirubin?
157. What is the mechanism of jaundice caused by increased blood levels of conjugated bilirubin?
158. What are the properties of unconjugated bilirubin?
159. What is one of the properties of conjugated bilirubin?
160. What are the causes of prehepatic jaundice?
161. What are the causes of hepatic jaundice?
162. What are the causes of posthepatic jaundice?
163. What is the pathogenetic chain of prehepatic jaundice?
164. What are the features of prehepatic jaundice?
165. What are the features of prehepatic jaundice?
166. What are the features of prehepatic jaundice?
167. What are the features of prehepatic jaundice?
168. How does bile metabolism change in hemolytic jaundice?
169. What is one of the blood biochemical changes seen in prehepatic jaundice in intracellular hemolytic anemia?
170. What is the mechanism of intense faeces colour in prehepatic jaundice?
171. What blood biochemical changes are seen in liver jaundice?
172. What are the features of liver jaundice?
173. What are the features of liver jaundice?
174. What are the features of liver jaundice?
175. What are the features of liver jaundice?
176. What is the mechanism of faeces discoloration in patients with hepatic jaundice?
177. What are the mechanisms of intense urine staining in patients with hepatic jaundice?
178. What is the mechanism of hyperbilirubinemia in patients with hepatic jaundice?
179. What is the mechanism of hyperbilirubinemia in patients with hepatic jaundice?
180. Which process of bilirubin metabolism is affected in posthepatic jaundice?
181. What are the features of posthepatic jaundice?
182. What are the features of posthepatic jaundice?
183. What is one of the features of posthepatic jaundice?
184. What is the cause of dark urine color in patients with posthepatic jaundice?
185. What is the pathogenetic chain that explain the faeces discoloration in posthepatic jaundice?
186. What is the pathogenetic factor of faeces discoloration in posthepatic jaundice?
187. What is one of the features of posthepatic jaundice?
188. What blood biochemical changes are seen in posthepatic jaundice?
189. How do faeces change in patients with posthepatic jaundice?
190. What are the mechanisms of bleeding syndrome in patients with posthepatic jaundice?
191. What is the cause of bleeding syndrome in patients with hepatic and posthepatic jaundice?
192. What is the cause of bleeding syndrome in patients with hepatic and posthepatic jaundice?
193. What are the cardiovascular changes and their mechanisms in cholemic syndrome?
194. What is one of the cardiovascular changes and mechanism in cholemic syndrome?
195. What is one of the cardiovascular changes and mechanism in cholemic syndrome?
196. What is the pathophysiologic mechanism of pruritus in patients with cholemic syndrome?
197. What is the pathophysiologic mechanism of pruritus in patients with cholemic syndrome?
198. What biochemical changes are seen in the blood in cholemic syndrome?
199. What is the mechanism of cholestatic syndrome in posthepatic jaundice?