**QUESTIONS FOR THE FINAL EXAM OF PATHOPHYSIOLOGY (2020-2021)**

**MEDICINE 2. SEMESTER I**

**Topic 1. Etiology, pathogenesis, nosology, cell damage, hypoxia**

1. What is the general pathogenesis?

2. What is the role of the cause in the occurrence of the disease?

3. What is the role of the cause in the evolution of the disease?

4. What is the possible variant of the combination of general and local lesions within the disease?

5. What is the pathogenetic factor?

6. What is the main link of the pathogenesis?

7. What is the pathogenetic therapy of the disease?

8. What is nonspecific disease prophylaxis?

9. What is the characteristic of the physiological reaction of the organism?

10. What is the adaptive reaction?

11. What is the compensatory reaction?

12. What is the protective reaction?

13. What characterizes the prodromal period of the disease?

14. What characterizes the resolution period of the disease?

15. What is the pathological process?

16. What are the primary sanogenetic mechanisms?

17. What are the secondary sanogenetic mechanisms?

18. What is the characteristic of the vicious circle in pathogenesis?

19. What is a secondary cell lesion?

20. How does the enzymatic spectrum of blood change in bile duct epithelial lesions?

21. What is the effect of the action of electric current on excitable cells?

22. What endogenous enzymes can cause damage to the cytoplasmic membrane?

23. What extracellular dyshomeostasis causes cell damage?

24. What is the effect of increasing the concentration of potassium ions in the extracellular sector?

25. What process can cause the electrical resistance of the cytoplasmic membrane to decrease?

26. Which of the intracellular enzymes is activated to increase the concentration of Ca2 + in the hyaloplasm?

27. What is the effect of nonspecific activation of cellular endonucleases?

28. What is the consequence of decompensated cellular acidosis?

29. What is the effect of decoupling the oxidation and phosphorylation processes?

30. What are the effects of decoupling the oxidation and phosphorylation processes?

31. What is the consequence of the energy shortage in the cell?

32. What are the stabilizing factors that stabilize lysosomal membranes?

33. What processes lead to the generation of free radicals?

34. What substances are part of the endogenous antioxidant system?

35. What is the effect of free radical action?

36. In what conditions does histotoxic hypoxia develop?

36. In what conditions does blood hypoxia develop?

37. Why does the vulnerability of different organs to hypoxia depend?

38. How does the water balance of the cell change in hypoxia?

39. What are the vascular effects of hypoxia?

40. What are the vascular effects of hypoxia?

41. How does the oxyhemoglobin dissociation curve CHANGE in hypoxia?

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**Topic 2. Typical cellular and tissue processes**

1. Which cells are subject to apoptosis?

2. What causes apoptosis during the initiation period?

3. What condition is necessary for the final development of apoptosis?

4. What causes apoptosis in the middle period?

5. What are the patterns of physiological apoptosis?

6. What are the patterns of pathological apoptosis?

7. What are the negative apoptotic signals?

8. What are the caspases of the apoptosis initiation phase?

9. What are the mechanisms of caspase activation?

10. Which intracellular factors inhibit caspase 8?

11. What factors trigger apoptosis by activating caspase1?

12. What factors trigger apoptosis of the virus-infected cell?

13. What does the apoptosome contain?

14. What are the consequences of the insufficient expression of phosphatidylserine?

15. Which intracellular factors alter the expression of phosphatidylserine (its translocation on the membrane surface)?

16. What is the main link of the pathogenesis of necrosis in the cytoplasmic membrane lesion?

17. What are the consequences of necrosis?

18. What regeneration is pathological?

19. What regeneration is pathological?

20. Which cytokines have a profibrotic effect?

21. Which cytokines have antifibrotic effect?

22. What regenerative processes are possible at the molecular level?

23. What regenerative processes are possible in cellular organs?

24. What are the consequences of sclerosis?

25. What pathological processes lead to advanced sclerosis?

26. What is the pathogenesis of sclerosis?

27. What factors cause sclerosis?

28. What atrophy is pathological?

29. What atrophy is physiological?

30. What is organ atrophy?

31. What is hypertrophy?

32. Who are the intracellular messengers of hypertrophy?

33. Who are the intracellular messengers of hypertrophy?

34. What is the general cause of cellular dystrophies?

35. What is the general cause of cellular dystrophies?

36. What is the general cause of cellular dystrophies?

37. What are the consequences of dystrophies?

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**Topic 3. Disorder of the regional circulation**

1. What is the main pathogenetic link of arterial hyperemia?

2. What is the correlation between blood flow and reflux in hypertension?

3. What is the pathogenetic mechanism of neurotonic arterial hyperemia?

4. What is the pathogenetic mechanism of neuroparalytic arterial hyperemia?

5. What is the pathogenetic mechanism of neuromyoparalytic arterial hyperemia?

6. What is the pathogenetic mechanism of functional arterial hyperemia?

7. What causes arterial hyperemia?

8. What is venous hyperemia?

9. What are the causes of venous hyperemia?

10. What is the main pathogenetic link of venous hyperemia?

11. What are the external manifestations of venous hyperemia?

12. What is the cause of organ enlargement in venous hyperemia?

13. What is the cause of the decrease in local temperature in venous hyperemia?

14. What is one of the consequences of venous hyperemia?

15. What are the consequences of venous hyperemia?

16. What are the local pathogenetic mechanisms of ischemia?

17. How does local hemodynamics change in ischemia?

18. How does cellular metabolism change in ischemia?

19. What are the outward manifestations of ischemia?

20. In which organs are the collaterals absolutely functionally insufficient?

21. Which emboli are of endogenous origin?

22. What is retrograde embolism?

23. In whose trauma is air embolism possible?

24. In which case does gas embolism occur?

25. Which vessel is blocked in case of embolism with amniotic fluid?

26. What are the consequences of arterial embolism?

27. What factors damage the rheological properties of the blood?

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**Topic 4. Inflammation**

1. From which cells do inflammatory mediators come?

2. What are the effects of mast cell tryptase on inflammation:

3. What chemotactic factors release mast cells?

4. What is the biological effect of prostaglandins in the inflammatory focus?

5. What is the biological effect of thromboxanes in the inflammatory focus?

6. What is the biological effect of leukotrienes in the inflammatory focus?

7. What effects does interleukin 1 (IL-1) have on the inflammatory focus?

8. What inflammatory mediators come from neutrophil leukocytes?

9. What oxygen-dependent bactericidal factors are generated by neutrophil leukocytes?

10. What bacteriostatic factor is generated by neutrophil leukocytes?

11. What inflammatory mediators come from eosinophils?

12. What inflammatory mediators come from lymphocytes?

13. What is the effect of C3a and C5a in the inflammatory focus?

14. What are the effects of the activated Hageman contact factor?

15. What is the general effect of kinins on inflammation?

16. What is the sequence of vascular reactions in the inflammatory focus?

17. What mediators cause inflammatory hypertension?

18. What are the peculiarities of inflammatory arterial hyperemia?

19. What is the pathogenesis of vascular hyperpermeability in inflammation?

20. What is the pathogenesis of inflammatory venous hyperemia?

21. What is the biological importance of venous hyperemia and inflammatory stasis?

22. What is the pathogenesis of exudation in the inflammatory focus?

23. What is the distinguishing mark of purulent exudate?

24. What are the mechanisms of leukocyte migration in the inflammatory focus?

25. What is the biological importance of neutrophil leukocyte migration in the inflammatory focus?

26. What is the biological importance of eosinophilic leukocyte migration in the inflammatory focus?

27. What is the biological importance of lymphocyte migration in the inflammatory focus?

28. What are the cellular sources of proliferation in the inflammatory focus?

29. What processes does physiological regeneration include in the inflammatory focus?

30. What is one of the general manifestations in the body in the inflammatory reaction?

31. What are the general manifestations in the body in the inflammatory reaction?

32.Which hormone has direct anti-inflammatory action?

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**Topic 5. Allergy**

1. What characterizes type I (anaphylactic) allergic reactions?

2. What characterizes type II (cytotoxic) allergic reactions?

3. What characterizes type III allergic reactions (Arthus)?

4. What characterizes type IV (delayed) allergic reactions?

5. What characterizes allergic reactions of type V (stimulator)?

6. How long does the latent period of the anaphylactic reaction last after the first contact with the allergen?

7. What antigens cause anaphylactic allergic reactions?

8. Which cells perform anaphylactic allergic reactions?

9. What immunoglobulins are involved in anaphylactic allergic reactions?

10. Where are antibodies of IgE located in anaphylactic reactions?

11. Which mediators are in stored mast cells?

12. Which mediators are synthesized in mast cells by the lipoxygenase pathway?

13. What is one of the local pathophysiological processes in anaphylactic reactions?

14. What pathophysiological process develops in the lungs in anaphylactic reactions?

15. What pathophysiological process develops in the cardiovascular system in anaphylactic reactions?

16. What pathophysiological process develops in the digestive tract in anaphylactic reactions?

17. What antigens participate in type II allergic reactions (cytotoxic, cytolytic)?

18. What are the mechanisms of cytolysis in type II allergic reactions (cytotoxic, cytolytic)?

19. What is the clinical manifestation of type II allergic reactions?

20. What antigens initiate type III allergic reactions?

21. Who are the mediators of the type III allergic reaction?

22. What structures are affected in the type III allergic reactions?

23. Which cells are frequently involved in the allergic reactions of type V?

24. Who are the mediators of the pathochemical phase of type IV allergic reactions?

25. What is the final manifestation of type IV allergic reactions?

26. What is nonspecific hypersensitivity?

27. What stages are characteristic for nonspecific hypersensitivity include?

28. What are the causes of autoimmune reactions?

29. What are the consequences of developing antibodies against postsynaptic striatal myocyte receptors?

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**Topic 6. Hydro-electrolytic disorders**

1. How do the osmolarity, protein and sodium content in the blood change with the deprivation of drinking water?

2. How do the osmolarity, sodium and protein content in the blood change in heavy sweating?

3. How do the osmolarity, chlorine and hydrogen ions in the blood change in uncontrollable vomiting?

4. How do the osmolarity, sodium and hydrogen ions in the blood change in diarrhea?

5. How do the osmolarity, Na concentration and blood volume change in acute hemorrhage in the first 2 hours?

6. How do the blood volume and cell concentration change in intravascular dehydration?

7. What are the compensatory reactions in intravascular dehydration?

8. How does capillary-interstitial and interstitial-cell exchange change in isoosmolar dehydration?

9. How does capillary-interstitial and interstitial-cell exchange change in hypersmolar dehydration?

10. How does capillary-interstitial and interstitial-cell exchange change in hypoosmolar dehydration?

11. How do the volume, osmolarity of the blood, sodium concentration and cell volume change with excessive drinking water consumption?

12. How does the volume of interstitial and intracellular fluid change with excessive drinking water consumption?

13. How does the oncotic and osmotic blood pressure change with excessive drinking water consumption?

14. How does the oncotic and osmotic blood pressure change with massive infusions of isotonic NaCl solutions?

15. How does the volume of interstitial and intracellular fluid change with massive infusions of isotonic NaCl solutions?

16. What are the immediate changes in oncotic and osmotic blood pressure in the case of massive infusions of 5% glucose solutions?

17. What are the compensatory reactions in intravascular hyperhydration?

18. What are the consequences of hypoosmolar hyperhydration in the cell?

19. What do we call edema?

20. What is the pathogenesis of cardiac edema?

21. What is the pathogenesis of nephritic edema?

22. What is the pathogenesis of nephrotic edema?

23. What is the pathogenesis of cachectic edema?

24. What is the pathogenesis of allergic edema?

25. In which pathological processes is hypernatremia found?

26. What are the causes of relative hypernatremia?

27. What are the compensatory mechanisms in absolute hypernatremia?

28. What is the final consequence of hypernatremia for cells?

29. What is the pathogenesis of edema in primary hyperaldosteronism?

30. In what pathological processes is secondary hyperaldosteronism encountered?

31. What is the cause of absolute hyponatremia?

32. What are the causes of relative hyponatremia?

33. In which pathological processes is absolute hyperkalemia found?

34. In which pathological processes is absolute hyperkalemia found?

35. What is the cause of hypokalemia?

36. ​​What are the consequences of hypocalcemia?

37. What are the causes of secondary hypercalcemia?

38. What is the cause of hypocalcemia?

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**Topic 7. Acid-base disorders**

1. How does the acid-base balance change in hyperoxia?

2. Which endogenous substances can lead to acidosis?

3. Accumulation of which endogenous substances can lead to alkalosis?

4. What are the criteria for acidosis?

5. What are the criteria for alkalosis?

6. What do we call compensated acidosis?

7. What do we call decompensated acidosis?

8. What do we call compensated alkalosis?

9. What do we call decompensated alkalosis?

10. In what processes does respiratory acidosis occur?

11. In what process does metabolic acidosis occur?

12. What are the pathogenetic factors of alkalosis?

13. What are the compensatory renal reactions in acidosis?

14. What is the compensatory reaction of the respiratory system in acidosis?

15. What are the consequences of acidosis?

16. What are the effects of acidosis on basal vascular tone in the systemic circuit?

17. What are the compensatory reactions in alkalosis?

18. What is the pathogenetic factor of acid-base dyshomeostasis in starvation?

19. What is the pathogenetic factor of acid-base dyshomeostasis in insulin hyposecretion?

20. Explain the mechanisms of headache in alkalosis?

21. Explain the mechanisms of headache in acidosis?

22. How does the volume of intracellular water change in acidosis?

23. How does pH and bicarbonate change in starvation?

24. How do respiratory rate (RR) and PaCO2 change in metabolic alkalosis?

25. What are the attested changes in the blood in respiratory alkalosis?

26. What are the attested changes in the blood in metabolic acidosis?

27. What are the changes in the blood in respiratory acidosis?

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**SEMESTER II**

**Topic 1. Endocrine pathophysiology**

1. Why are the clinical manifestations of secondary hypocorticism determined?

2. What are the pathogenetic principles of tertiary hypocorticism therapy?

3. What are the principles of retroregulation (ascending) of the hypothalamic - pituitary - adrenal cortex axis?

4. Patient C., who suffers from chronic nonspecific polyarthritis for a long time, was treated with glucocorticoids in high doses. Subsequently, the radiograph found atrophy of both adrenals. What is the pathogenesis?

5. Lack of glucocorticoid hormones is clinically manifested by vascular disorders. How does vascular tone change in glucocorticoid hyposecretion?

6. Lack of glucocorticoid hormones is clinically manifested by disorders of heart function. How does cardiac function change in hypocorticism?

7. All three forms of hypocorticism (primary, secondary and tertiary) represent the involvement of the hypothalamic - pituitary - adrenal axis at different levels. The level of the condition can be determined by measuring the hormones in the blood. What is the hormonal pattern in primary hypocorticism?

8. Primary and secondary hypocorticism have most analogous clinical manifestations. What is a characteristic clinical manifestation for primary hypocorticism?

9. Primary and secondary hypocorticism have most analogous clinical manifestations. What is a characteristic clinical manifestation for secondary hypocorticism?

10. What are the risks of stress for people with hypocorticism?

11. One of the vital risks of stress for people with hypocorticism is arterial collapse. What is the pathogenesis?

12. Glucocorticoid hormones are important in immunity and inflammation. How does the inflammatory reaction in people with hypocorticism work?

13. In patient C. suffering from primary objective hypocorticism, skin hyperpigmentation was found. What is the pathogenesis?

14. In primary patient C. primary hypocorticism was found. What is the possible etiology?

15. Is it a possible cause of secondary hypercorticism?

16. Why are the clinical manifestations of tertiary hypercorticism determined?

17. Glucocorticoids in physiological and pharmacological doses have an ambiguous influence on severe immunity. How does the immune system change in the hypersecretion of glucocorticoids?

18. Specific clinical features for hypercorticism are excessive lipid deposits in certain areas of the body - "full moon figure", "bison hump", deposits on the body trunk. What is the pathogenesis of adipose tissue hypertrophy in these areas?

19. Glucocorticoids are also involved in protein metabolism. How does protein metabolism change in hypersecretion of glucocorticoids?

20. Clinical examination of patient D. with hypercorticism demonstrates edema on the legs. What is the possible pathogenesis?

21. Insulin simultaneously alters blood glucose and peripheral glucose utilization. What is the mechanism of insulin intensification of peripheral carbohydrate use?

22. In type I diabetes, the lack of insulin intensifies glucagon secretion. What are the effects of glucagon hypersecretion?

23. Glucose is an osmotically active substance. What are the effects and consequences of exaggerated hyperglycemia in type I diabetes?

24. The paradoxical clinical phenomenon for type I diabetes is the weight deficit simultaneously with the increase of appetite. What is the pathogenesis of polyphagia?

25. Weight loss is characteristic of type I diabetes. What is the pathogenesis?

26. The assimilation of blood glucose depends on the character of the membrane transporters (GLUT-1-4), which on different cells are insulin-independent or insulin-independent. Which cells are endowed with insulin-independent Glut-4 receptors?

27. Patient S. with type I diabetes has erectile dysfunction. What is the pathogenesis?

28. Symptoms with which patients with type I diabetes are primarily addressed thirst, frequent and excessive water consumption, excessive elimination of urine. What causes polydipsia in type I diabetes?

29. Hematological examination of patient C., 24 years old with type I diabetes showed: erythrocytes - 6.1012 / L, hematocrit - 60%. What is the pathogenesis of these disorders?

30. Laboratory investigations of patient C., 24 years old with type I diabetes mellitus demonstrated hyperlipidemia with unesterified fatty acids. What is the pathogenesis?

31. Patient C., 24 years old, went to the endocrinologist with the presumptive diagnosis "type II diabetes". What are the signs that differentiate type II from type I diabetes?

32. What is the main pathogenetic link of secondary hypothyroidism?

33. What are the pathogenetic principles of secondary hypothyroidism therapy?

34. What is the hormonal pattern in secondary hypothyroidism?

35. What is the clinical manifestation of tertiary hyperthyroidism?

36. The diagnosis of Graves' disease has been established in a patient with hyperthyroidism. What type of allergic reaction does Graves’ disease mean?

37. Patient D., 45 years old, went to the endocrinologist due to the enlargement of the thyroid gland ("goiter"). Biochemical investigations have shown: the concentration of thyroid hormones in the blood - increased; TSH concentration in the blood - increased. Scintigraphy demonstrates the abnormal uptake of uniform radioactive iodine throughout the thyroid parenchyma. What is the pathogenesis of this pathology?

38. Patient D., 45 years old, went to the endocrinologist due to the enlargement of the thyroid gland ("goiter"). Hormonal investigations have shown: the concentration of thyroid hormones in the blood - low; TSH concentration in the blood - increased. Scintigraphy demonstrates decreased uptake of uniform radioactive iodine throughout the thyroid parenchyma. What is the pathogenesis of this pathology?

39. Patient D., 45 years old, went to the endocrinologist due to the enlargement of the thyroid gland ("goiter"). The diagnosis of Hashimoto's autoimmune hypothyroidism was subsequently established. What is the pathogenesis of this disease?

40. Patient D., 45 years old, went to the endocrinologist due to the enlargement of the thyroid gland ("goiter"). The diagnosis of Graves' disease was subsequently established. What is the pathogenesis of this disease?

Patient C., 32 years old with hyperthyroidism. Accusation: weight loss (5 kg / 3 months) despite excessive appetite. 41. What is the possible pathogenesis of appetite stimulation in hyperthyroidism?

What is the mechanism of hyposecretion of antidiuretic hormone in pituitary trauma?

42. What are the causes of hypersecretion of prolactin?

43. What are the manifestations of prolactin hypersecretion in women?

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**Topic 2. Blood pathophysiology**

1. What are the RDW reference values ​​(width of the erythrocyte distribution)?

2. Determine the type of anemia if RDW is high and VEM (mean erythrocyte volume) is low?

3. Determine the type of anemia if RDW is normal and VEM (mean erythrocyte volume) is low?

4. What are the mechanisms responsible for disrupting Fe blood transport?

5. What are the peculiarities of chronic granulocytic leukemia?

6. What are the changes in iron metabolism in the intestine to increase hepsidin?

7. What are the changes in iron metabolism in the intestine to increase hepsidin?

8. What factors facilitate the blood absorption of the non-blood fraction of Fe?

9. In what conditions is eosinophilic leukocytosis found?

10. What is the condition of oligocythemic hypovolemia?

11. In what conditions is polycythemic hypervolemia found?

12. What are the causes of absolute primary erythrocytosis?

13. What processes are disrupted in B12 deficiency anemia?

14. What is agranulocytosis?

15. What is the nuclear deviation "to the left"?

16. How does the blood count change in B12-deficient anemia?

17. Which anemia is characterized by a decrease in the average hemoglobin content?

18. Hematological picture of subleukemic myeloid leukosis?

19. Hematological picture of subleukemic myeloid leukosis?

20. What are the signs of intravascular hemolysis by the action of drugs and chemicals?

21. What are the basic hematological signs of myeloid myeloid leukosis?

22. What are the signs of relative leukocytosis?

23. What are the signs of erythrocyte hypochromia?

24. What are the signs of cell proliferation and maturation disorder in the erythroblastic series?

25. What is the pathogenetic substrate of hemoblastoses?

26. What are the general features of aplastic anemia?

27. What are the general features of acute leukemia?

28. What are hemogram changes in red marrow hyperproliferation?

29. What anemia is characterized by the presence of Heinz corpuscles?

30. What are the manifestations of gastrointestinal syndrome in anemia due to vitamin B 12 deficiency?

31. What are the typical manifestations of intravascular hemolysis?

32. What are the typical manifestations of extravascular hemolysis?

33. What are the disorders characteristic of vitamin B12 deficiency?

34. What are the pathogenetic forms of anemia by mechanism of erythropoiesis reduction?

35. In which type of anemia is the osmotic resistance of erythrocytes maximum?

36. What is the pathogenetic mechanism of pernicious anemia?

37. What is the picture of peripheral blood in aplastic anemia?

38. For which types of anemia is anisocytosis characteristic?

39. What are the signs of acute posthemorrhagic anemia <24 hours?

40. What are the extrinsic causes of hemolytic anemias?

41. What are the intrinsic causes of hemolytic anemias?

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**Topic 3. Cardiovascular pathophysiology**

1. What is the role of NO deficiency in the vascular remodeling of arterial hypertension?

2. What is the role of excess endothelin 1 (ET-1) in increasing peripheral vascular resistance?

3. What is the role of excess angiotensin II (Ang II) in increasing peripheral vascular resistance?

4. What factors reduce NO content and lead to essential hypertension?

5. What are the mechanisms of aldosterone in the pathogenesis of essential hypertension?

6. What are the mechanisms of increased neointima in essential hypertension?

7. What factors induce hyperhomocysteinemia?

8. What is the role of angiotensin 1-7 in the pathogenesis of essential hypertension?

9. What are the mechanisms of Ang II in the pathogenesis of essential hypertension?

10. What is the main pathogenetic link in renovascular hypertension?

11. What causes bradycardia?

12. What mechanisms are compensatory in ischemic systolic heart failure?

13. What mechanisms are compensatory in systolic heart failure?

14. In heart failure the production of BNP (natriuretic peptide B) increases. What are its effects?

15. What is the pattern of heart failure in hyperthyroidism?

16. What mechanisms are compensatory in diastolic heart failure?

17. What factors affect the length-to-strength ratio of the heart (heterometric mechanism)?

18. What factors lead to concentric hypertrophy of the myocardium?

19. What are the consequences of left ventricular failure?

20. What are the mechanisms of peripheral cardiac edema?

21. What is the cause of nocturia in patients with heart failure?

22. What are the consequences of myocardial ischemia?

23. What are the extracardiac mechanisms of compensation in heart failure?

24. What is the role of asymmetric dimethylarginine (DAS) in the pathogenesis of hypertension?

25. What is the pathophysiological entity of tachycardia?

26. What factors increase peripheral vascular resistance?

27. What is the role of NO deficiency in the pathogenesis of essential hypertension?

28. What is the basic pathogenetic link in renal nephrogenic hypertension?

29. What are the effects of hypoxia-inducible transcription factor (HIF) in the myocardium?

30. In what situations does the heart activate the Starling mechanism?

31. What can be the changes in central hemodynamic indices in a hypertensive patient with concentric hypertrophy?

32. Which cardiovascular reflexes facilitate the evolution of right heart failure?

33. What reflex is the basis of the homeometric adjustment of the heart in case of increased pregnancy?

34. What are the extracardiac mechanisms of compensation in heart failure?

35. What are the signs of ECG in hyperkalemia?

36. By what mechanisms does norepinephrine induce sinus tachycardia?

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**Topic 4. Respiratory pathophysiology**

1. What are the causes of respiratory distress?

2. What is the manifestation of the lesion (sectioning) of a branch of the phrenic nerve?

3. What is the Kretschmar respiratory reflex?

4. What is the Herring-Breuer reflex?

5. What changes in the composition of the alveolar air are found in hyperventilation?

6. What is hypoventilation?

7. What changes in the gaseous composition of arterial blood are found in hypoventilation?

8. What changes in the acid-base balance are found in hypoventilation?

9. What do we call pulmonary restriction?

10. In what conditions is superficial and accelerated breathing encountered?

11. What are the mechanisms of shallow and accelerated breathing?

12. What are the characteristic changes of the pneumogram for restrictive disorders?

13. What do we call pulmonary emphysema?

14. What characterizes pulmonary emphysema?

15. What is the main pathogenetic link of pulmonary emphysema?

16. What changes in the lung parenchyma are characteristic of emphysema?

17. One of the characteristic signs of pulmonary emphysema is shortness of breath. What explains the shortening of expiration in emphysema?

18. What do we call pneumothorax?

19. What do we call pneumosclerosis?

20. What are the causes of pneumosclerosis?

21. What are the pathogenetic links of pneumosclerosis?

22. What are the manifestations of pneumosclerosis?

23. What do we call pulmonary atelectasis?

24. What are the causes of nonobstructive atelectasis?

25. What are the consequences of atelectasis?

26. What do we call pulmonary obstruction?

27. What factors can cause upper airway obstruction?

28. What factors can cause lower airway obstruction?

29. What are the causes of deep and slow (stenotic) breathing?

30. What are the mechanisms of stenotic respiration?

31. What are the pathophysiological mechanisms of bronchial obstruction?

32. What are the basic manifestations of asthma?

33. Which respiratory indices are usually determined to assess the degree of bronchial obstruction?

34. What is one of the diagnostic criteria for asthma?

35. Which biologically active substances have a bronchoconstrictor effect?

36. Patient D. 48 years old was hospitalized with the following accusations: dyspnea attacks and excruciating cough that occurs unexpectedly, with sputum in small quantities at the end of the attacks. Breathing is wheezing and audible especially on exhalation. What is the type of dyspnea in the given patient?

37. Patient D. 48 years old was hospitalized with the following accusations: dyspnea attacks and excruciating cough that occurs unexpectedly, with small sputum at the end of the attacks. Breathing is wheezing and audible especially on exhalation. What is the main pathogenetic link of respiratory disorders in this patient?

38. A 7-year-old boy was hospitalized with the following accusations: difficult breathing, numbness, pain when swallowing, dry cough. Body temperature 38.6 С. Oral organs are hyperemiad, edematous, tonsils enlarged with deposits. Breathe 10-11 per minute. An inspiring hissing sound is heard, with the collapse of the soft tissues in the supra-, subclavicular, intercostal space. What is the type of dyspnea (breathing) in the given patient?

39. What is pulmonary edema?

40. What factors cause pulmonary edema?

41. What do we call acute respiratory distress syndrome?

42. What processes diminish the diffusion of gases through the alveolo-capillary barrier?

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**Topic 5. Digestive pathophysiology**

1. What is the role of prostaglandins in the mucosa of the gastrointestinal tract (GIT)?

2. What is the role of oxidative stress in the pathogenesis of gastric ulcer?

3. What are the peculiarities of the GIT epithelium?

4. What factors contribute to the loss of immune tolerance of the GIT mucosa?

5. What regulatory factors increase gastric secretion?

6. What regulatory factors decrease gastric secretion?

7. What regulatory factors decrease gastric secretion?

8. What regulatory factors increase gastric secretion?

9. What is the pathogenesis of gastric ulcer in the invasion with H. pylori (HP)?

10. What factors contribute to the induction of the inflammatory response in the GIT mucosa?

11. What is the pathogenetic factor of nonerosive chronic gastritis?

12. What is the pathogenetic factor of reactive chronic gastritis?

13. What is the pathogenetic factor of acute erosive gastritis?

14. What are the consequences of atrophic gastritis?

15. What are the consequences of atrophic gastritis?

16. What factors facilitate the colonization of the small intestine with bacteria?

17. What are the consequences of atrophic gastritis?

18. What are the consequences of atrophic gastritis?

19. What deficiency metabolic disorders are characteristic of atrophic gastritis?

20. What are the mechanisms of pancreatic self-aggression?

21. What are the mechanisms of pancreatic self-aggression?

22. What is the role of heat shock proteins (HSPs) in the pathogenesis of pancreatitis?

23. What is the role of exosomes in the pathogenesis of pancreatitis?

24. What is the pathogenesis of lung diseases in pancreatitis?

25. What is the role of alcohol in the pathogenesis of pancreatitis?

26. What is the role of alcohol in the pathogenesis of pancreatitis?

27. What is the role of alcohol in the pathogenesis of pancreatitis?

28. What is the role of pancreatic star cells (CSPs) in the pathogenesis of pancreatitis?

29. What is the role of alcohol in the pathogenesis of pancreatitis?

30. What is the role of nicotine in the pathogenesis of pancreatitis?

31. What is the role of nicotine in the pathogenesis of pancreatitis?

32. What is the role of nicotine in the pathogenesis of pancreatitis?

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**Topic 6. Liver pathophysiology**

1. How does carbohydrate metabolism change in liver failure?

2. How does protein metabolism change in liver failure?

3. How does blood biochemistry change in liver failure?

4. How does the biochemistry of blood change in cholestasis?

5. What liver biochemical test shows the synthetic function of the liver?

6. What liver biochemical test can indicate the liver lesion?

7. What are the consequences of choledochal duct obturation?

8. What is cholestasis?

9. Which liver biochemical test is specific for cholestasis?

10. What is cholemia?

11. What cardiac changes are attested in cholemia?

12. What are the pathogenetic mechanisms of metabolic acidosis in liver failure?

13. What is acholia?

14. What is the mechanism of jaundice caused by increased blood levels of unconjugated bilirubin?

15. What is the mechanism of jaundice caused by increased blood levels of conjugated bilirubin?

16. What are the biochemical characteristics of unconjugated bilirubin?

17. What are the biochemical characteristics of conjugated bilirubin?

18. What causes prehepatic jaundice?

19. What causes hepatic jaundice?

20. What are the causes of posthepatic jaundice?

21. What are the characteristics of prehepatic jaundice?

22. What biochemical changes are attested in the blood in prehepatic jaundice from hemolytic anemias?

23. What biochemical changes occur in the blood in hepatic jaundice?

24. What are the characteristics of hepatic jaundice?

25. Which stage of bilirubin metabolism is affected in posthepatic jaundice?

26. What are the characteristics of posthepatic jaundice?

27. What is the mechanism of cholestatic syndrome in posthepatic jaundice?

28. What are the consequences of acolyte in patients with mechanical jaundice?

29. Which cells in the liver parenchyma are responsible for the release of pro-fibrinogenic cytokines and the onset of hepatocyte fibrosis in the liver lesion?

30. Which cells in the liver parenchyma are responsible for the excess production of liver fibers and extracellular matrix in the liver lesion?

31. Which cells in the liver parenchyma can be converted to myofibroblasts and trigger liver fibrosis by excessive collagenogenesis?

32. What are the pathogenetic links of liver fibrosis?

33. Whose products accumulation underlines the metabolic acidosis in liver failure?

34. What is the pathogenetic mechanism of metabolic acidosis in the liver failure?

35. What are the pathogenetic mechanisms of metabolic alkalosis in the liver failure?

36. What are the manifestations of vitamin D deficiency in the liver failure?

37. What are the manifestations of vitamin A deficiency in the liver failure?

38. What are the electrolyte changes triggered by secondary hyperaldosteronism in the liver failure?

39. What hematological changes are characteristic for hypersplenism due to liver failure?

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**Topic 7. Kidney pathophysiology**

1. What is the pathogenetic mechanism of glomerular hematuria?

2. In what conditions is leukocyturia attested?

3. Is what diseases the lipiduria is attested?

4. What factors cause decreased reabsorption of water in the proximal renal tubules?

5. What factors cause decreased water reabsorption in the distal tubules and collectors?

6. What factors cause decreased distal reabsorption of Na ions?

7. The result of which disease is tubular proteinuria?

8. What factors cause decreased glucose reabsorption?

9. What conditions cause amino acids?

10. In which pathologies is hyposthenuria attested?

11. In which pathologies is hypersthenuria attested?

12. In which cases is isosthenuria attested?

13. What disorders does nephrotic syndrome include?

14. What pathological phenomena does nephritic syndrome include?

15. What processes cause proximal canalicular acidosis?

16. What processes cause distal canalicular acidosis?

17. What factors stimulate renin secretion?

18. What are the endocrine functions of the kidney?

19. What are the prerenal causes of acute renal failure?

20. What are the causes of intrinsic acute renal failure?

22. What is the cause of acute renal failure of postrenal origin?

23. What are the main syndromes in acute renal failure?

24. What are the manifestations of urinary syndrome in acute renal failure?

25. What are the manifestations of humoral syndrome in acute renal failure?

26. What are the manifestations of the clinical syndrome in acute renal failure?

27. What are the causes of chronic kidney failure?

28. What is the succession of the acute renal failure development?

29. How does the rate of glomerular filtration in glomerulopathies change?

30. How does the rate of glomerular filtration in hypervolemia change?

31. How does the rate of glomerular filtration in hypovolemia change?

32. How does diuresis change in hypoproteinemia?

33. How does diuresis change in hyperproteinemia?

34. How does diuresis change with a decrease in cardiac output?

35. What is the mechanism of hypercoagulability in nephrotic syndrome?

36. What is the mechanism of hyperlipidemia in nephrotic syndrome?

37. What is the mechanism of loss of renal filter size selectivity?

38. What is the mechanism of loss of electrostatic selectivity of the renal filter?

39. What are the pathogenetic mechanisms of the decrease in GFR in acute renal failure?

40. What are the consequences of urinary tract obstruction?

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