FEDERAL STATE BUDGET EDUCATIONAL
HIGHER EDUCATION INSTITUTION
"ROSTOV STATE MEDICAL UNIVERSITY"
MINISTRY OF HEALTH OF THE RUSSIAN FEDERATION

FACULTY OF TREATMENT AND PREVENTION

Appraisal Fund
in the discipline "Patophysiology"

Specialty 05/31/01 General Medicine

2023

1. Interim certification form:test and exam.

2. Type of intermediate certification:intermediate certification students in the form of credit is implemented on the basis of the student's rating formed during the 5th semester. Interim certification of students in the form of an exam takes place at the end of the VI semester and consists of 2 stages. The first stage consists of writing test tasks and solving situational problems; the second stage is an oral interview.

3. List of competencies formed by the discipline or in the formation of which the discipline participates in

Code competencies	Content of competencies (results of mastering OOP)	Contents of elements competencies in the implementation of which the discipline participates
OPK 9	ability to assess morpho-functional, physiological states and pathological processes in the human body to solve professional problems	register an ECG and determine from its data the main types of arrhythmias, signs of ischemia and myocardial infarction; assess the cellular composition of the inflammatory exudate and phagocytic activity of leukocytes; analyze the leukocyte formula of neutrophils and, on this basis, formulate a conclusion about changes in it in adults and children of different ages; formulate a conclusion based on the hemogram about the presence and type of a typical form of pathology of the blood system; analyze coagulogram parameters and, on this basis, formulate a conclusion about changes in it; define typical forms disorders of gas exchange function of the lungs By indicators alveolar ventilation, blood gas composition and blood flow in the lungs; differentiate pathological types of breathing and explain the mechanisms of their development; characterize typical renal dysfunction according to blood, urine and clearance tests; differentiate various types of jaundice;

		evaluate indicators of acid-base state (ABS) and formulate conclusions regarding various O types of its violations; differentiate various types of hypoxia; define typical violations secretory function of the stomach and intestines according to gastric analysis And intestinal content; interpret results main diagnostic allergy tests;

4. Stages of formation of competencies in the process of developmenteducational program

Competence	Disciplines	Semester
OPK-9	Anatomy	1, 2, 3
	Topographical anatomy And operative surgery	5, 6
	Histology, embryology	2, 3
	Normal physiology	3, 4
	Microbiology, virology	3, 4
	Immunology	9
	Pharmacology	5, 6
	Pathological anatomy	5, 6
	Clinical pathological anatomy	12
	Clinical pharmacology	eleven
	Propaedeutics of internal diseases	5, 6
	Fundamental medicine	7

5. Stages of developing competencies inprocess of mastering the discipline

Sections of the discipline	Codes generated competencies
	OPK-9
Semester 5	
Section 1	+
Section 2	+
Section 3	+
Semester 6	
Section 4	+

6. Current control

Current monitoring of students' progress in the discipline "Patophysiology" is carried out during practical classes. The forms of current control are text test tasks, situational

tasks and oral interview. In a practical lesson, either one of the forms of assessing current performance, or a combination of them, can be used. In the latter case, the final grade for the lesson is given as the arithmetic average of the grades for the corresponding forms of control. Questions for current classes and test control questions, as well as the criteria for their assessment, coincide with similar tasks and criteria for intermediate certification for the corresponding section. Examples of situational tasks and criteria for their evaluation are given below.

7. Interim certification

Forms of control from discipline RPD	Sample (standard) tasks, quantity
Tests	500
Situational tasks	100
Oral survey, interview	230

Technology for issuing intermediate certification.

- Interim certification in the form of “pass”.

Interim certification in the form of a test in the discipline “Patophysiology” is carried out at the end of the 5th semester. The criterion for positive certification is that the student obtains points in the range from 60 to 100. The above points are formed according to the following criteria:

1. Attending lectures – 1 point (9*1=9);
2. Availability of a notebook for practical work with protocols for their implementation - 3 points - there is a notebook, 0 points - there is no notebook;
3. Grades in current classes: grade “2” - 0 points;
   score “3” - 2 points;
   score “4” - 3 points;
   rating “5” - 4 points;
4. Marks in “repetition classes”:
   score “2” - 0 points;
   score “3” - 6 points;
   score “4” - 7 points;
   rating “5” - 8 points;
5. Additional points (active participation in the department’s INC, presentations at conferences) – 6 points; prizes at all-Russian conferences – 12 points.

- Interim certification in the form of an “exam”.

Interim certification in the form of an exam in the discipline "Patophysiology" is carried out at the end of the VI semester.

- Test control

List of test tasks for intermediate certification with standard answers:

1. Note the process in which the leukemoid reaction is most often recorded:

1. Heart attack.
2. Kidney failure.
3. Sepsis.

2. Absolute erythrocytosis is observed when:

1. Crisis with pneumonia and typhus.
2. Water fasting.
3. Heart failure.
4. Profuse diarrhea (cholera).

3. Mark the most important element of the anticoagulant system:

1. Fibrinogen.
2. Thrombin.
3. Plasmin.
4. Fibrin.
5. Thrombomodulin.

4. Poikilocytosis means:

1. Increase in leukocytes in the blood.
2. Increase in red blood cells in the blood.
3. An increase in red blood cells of various shapes in the smear.
4. Reduction of red blood cells in the blood.

5.*Mark the arteries most often affected by atherosclerosis:*

1. Coronary.
2. Subclavian.
3. Axillary.
4. Pulmonary.

6.Note the antiatherogenic factor:

1. Chylomicrons.
2. HDL.
3. VLDL.
4. LDL.

7. Resorption-necrotic syndrome during myocardial infarction includes:

1. Appearance of cardiomyocyte enzymes in the blood.
2. The appearance of changes on the ECG.
3. The appearance of cardiotropic autoantibodies in the blood.
4. Activation of thrombus formation.

8. Name the main hemodynamic indicator that determines the value of blood pressure:

1.**Vascular resistance**

2.Rheological properties of blood

3. Concentration of adrenaline in the blood

9. Specify the depressor mechanism of blood pressure regulation:

1.Cerebral ischemia.

2.**Baroreceptor mechanism.**

3.Renin-angeotensin-aldosterone mechanism.

4. Chemoreceptor.

10. Note the clinical manifestations of Morgagni-Edams-Stokes syndrome:

1. Increased blood pressure.

2. Loss of consciousness.

3. Facial hyperemia.

Correct answers to test questions are marked**"bold"**font.

Test control grading scale:

percentage of correct answers	Marks
100-91	Great
90-81	Fine
80-71	satisfactorily
Less than 71	unsatisfactory

- Situational tasks

Task 1.

A 35-year-old patient was admitted to the surgical department of a hospital with complaints of nausea, abdominal pain that worsened with changes in body position, coughing, walking, and lifting the limbs. From the anamnesis: pain in the lower right part of the abdomen appeared two days ago, was pulsating in nature, and later became diffuse and constant. Objectively: the patient's condition is of moderate severity, the skin is hot and moist to the touch, symptoms of peritoneal irritation are noted by palpation. Body temperature 39^0. In the emergency room, blood was urgently drawn for analysis.

1.What is the presumptive diagnosis? Explain the mechanisms development of exudation in the pathological focus.

2.What changes in the blood should be expected in the patient?

Answer.

1. A patient has acute appendicitis - inflammation of the vermiform process.

One of the signs of acute inflammation is exudation, which characterized by the release of the liquid part of the blood and formed elements into the site of inflammation. In the pathological process of inflammation, there are two phases of increasing the permeability of microvasculature vessels:**early or**

transient (instantly increasing) permeability vessels, caused by the action of vasoactive mediators (histamine, leukotrienes); **late prolonged**(within hours) associated with
the predominant action of leukotrienes and cytokines (IL1, TNF-α, γ-interferron). Exudation at the site of inflammation is caused by both direct damage to the microvasculature and the effects of inflammatory mediators. It is carried out in three ways: through interendothelial clefts, through the body of endothelial cells through specialized channels, and also by micropinocytosis in the form of active conduction of the smallest drops through the cell body. An increase in hydrostatic pressure during venous hyperemia, as well as at later stages an increase in osmotic and oncotic pressure in the tissue is also important. The vascular bed first leaves water, finely dispersed proteins - albumin, coarsely dispersed - globulins, and even fibrinogen. Then emigration of leukocytes occurs. PMN-leukocytes and monocytes exit through the interendothelial gaps, alymphocytes through the body of the endothelial cell. Chemotaxis, changes in pH at the site of inflammation and the electrical potential of leukocytes and endothelial cells of the vascular wall are important in the emigration of leukocytes. As a result of the release of water, proteins and blood cells from the vascular bed, an inflammatory exudate is formed.

2. With the development of acute inflammation in a patient in the general analysis blood should be expected: an increase in the total number of leukocytes, a shift in the leukocyte formula to the left with an increase in band neutrophils and the appearance of young forms of neutrophils, a significant increase in ESR, an increase in C-reactive protein.

Task 2.

A 30-year-old patient consulted an allergist with complaints of the appearance of red itchy spots on the skin of the face in cold weather. She also notes that when washing with cold water, she experiences itching and severe swelling of the skin at the site of contact with water.

Cold exposure test on the skin of the shoulder by applying an ice cube for 30 seconds. was strongly positive. Hyperemia and a blister appeared at the site where the piece of ice was applied.

1.What pathology does the patient have? What does the role do? allergen in this case?

2. Specify the type and pathophysiological mechanisms of allergic reactions that occur in this pathology.

Answer.

1. The patient has one of the types of allergic reactions -**cold allergies (cold urticaria)**. Urticaria is a common allergic skin disease that is caused by external (including weather) and internal factors (foci of infection,

intoxication, neurovegetative dysfunction).Urticaria involves the superficial dermis and is characterized by limited blisters with raised, circumscribed borders and pale centers; the blisters may coalesce. In a particular case, the role of a trigger for the development of urticaria is played by low external temperature, which affects the skin and mucous membranes of the face. At temperatures below 4^0C in the body Class M immunoglobulins are produced, which are called cold autoantibodies. These immunoglobulins interact with antigens fixed on the membranes of the patient's red blood cells, which leads to the launch of an allergic reaction when exposed to low t^o.

2.Hives occur according to the immediate type of allergy. General the pathogenetic link of these reactions is an increase in vascular permeability and the development of acute edema in the surrounding area. The leading mechanism for the development of urticaria is the reagin mechanism of damage, in some cases - the immune complex. When sensitized by an allergen (cold factor), IgE, IgG, and IgM accumulate in the body. Upon repeated contact with the allergen, IgE and IgG are fixed on cell membranes (reagin allergy mechanism) or deposition on the surface of the vascular wall IgG immune complexes, IgM+ antigen (immune complex mechanism of allergy) formed in the blood. With the reagin type of allergic reactions, as a result of the interaction of the allergen with antibodies (IgE and IgG) fixed on the membranes of mast cells and basophils, they degranulate with the formation of a large number of mediators of immediate allergy. In the immunocomplex type, the process of degranulation of target cells occurs under the influence of anaphylotoxin, which is the C3a and C5a components of complement. The mediators of the immediate type of allergy formed as a result of degranulation initiate the development of an acute inflammatory reaction, the manifestations of which determine the clinical picture of the disease.

Task 3.

"Patient S., 20 years old. For three days I have been bothered by a cough, runny nose, and low-grade fever. I took amidopyrine. On the fourth day I noticed subcutaneous hemorrhages on the skin of the chest and limbs, and there were repeated nosebleeds.

Objectively: common petechial And subcutaneous new hemorrhages. Positive symptoms of pinch and tourniquet. Otherwise, no special features."

Exercise:

1. What hemostasis disorder developed in patient S.?
2. Give a pathogenetic explanation for the clinical and laboratory manifestations.

Answer.

1. Patient S. probably developed hemorrhagic diathesis.

2. The appearance of hemorrhage may be associated with the use amidopyrine. Amidopyrine leads to thrombocytopenia, which is associated with increased platelet destruction as a result of an immunological conflict. It is known that one of the side effects of amidopyrine is the development of an allergic reaction of the cytotoxic type. Thus, patient S. apparently developed thrombocytopenic purpura. A decrease in the number of platelets, as well as their damage (by complexes Ig M + G + AG + complement) leads to both disruption of the formation of platelet thromboplastin (aggregation and lysis processes are disrupted) and to a decrease in blood clot retraction, which is the final stage of the blood coagulation process (deficiency of retractoenzymes in platelets). The consequence of these changes is an increase in bleeding time, which is clinically manifested by spontaneous hemorrhages.

Task 4.

"Patient G., 43 years old. Examination revealed a single peptic ulcer in the proximal duodenum. From the anamnesis IN the medical record contains information that patient G. underwent surgery for a stomach ulcer 1.5 years ago and has a concomitant disease - hyperparathyroidism. ***Objectively:*** increase in basal and stimulated gastric secretion, blood gastrin level is 1000 g/ml (normal is up to 150 g/ml). Patient G. complains of diarrhea.

Exercise:

1. What is the possible mechanism for the development of recurrent ulcers in the duodenum in patient G.?
2. Can hyperparathyroidism affect the development of ulcers?
3. What is the mechanism of diarrhea in patient G.?

Answer

1. Surgery for peptic ulcer disease does not always relieve patients from this disease, because the possibility of relapse of peptic ulcer disease is quite high. It is obvious that simple gastroenterostomy and suturing of the ulcer do not change the activity of aggressive factors; the influence of the parasympathetic nervous system and the formation of gastrin in the antrum of the stomach remain.

2. Hyperparathyroidism and, as a consequence, hypercalcemia, have a direct, stimulating effect on acid secretion in the stomach, which

is also found in patients with Zollinger-Ellison syndrome. It is known that intravenous administration of calcium also stimulates acid secretion in healthy people. Calcium stimulates the release of gastrin from G cells because it is an intracellular regulator of secretory function and realizes its action through $_{C}$AMF. Intracellular Ca complex^{2+}with calmodulin activates cell responses to cholinergic impulses and histamine. It is known that hyperparathyroidism is often accompanied by the development of peptic ulcers in the stomach. Elimination of hypercalcemia by removal of the parathyroid glands leads to a decrease in both basal acid secretion and gastrin levels in the blood.

3. After gastrectomy, changes develop associated with limited absorption of iron and calcium ions. When a gastroduodenal anastomosis is applied, the rhythmic phase interaction of the digestive organs is disrupted, pancreobiliary asynchrosia develops, and the digestion process suffers. In the post-resection period, latent lactose deficiency may appear as a result of the rapid evacuation of food from the stomach into the intestine. Clinically, this fermentopathy is manifested by flatulence, abdominal pain and diarrhea.

Task 5.

Patient V., 35 years old. Complaints of weakness, dizziness, bruising for no apparent reason. Sick for 4 months.

Objectively: moderate condition. The skin is pale with petechiae and ecchymoses. The tonsils are not enlarged, no pathology of the lungs or heart was detected, the liver and spleen are not palpable.

Additional research: blood analysis: Hb - 80g/l, Er. - 2.4 x 10_{12}/l, leukocytes - 2 x 10_{9}/l, platelets - 20.0 x10_{9}/l, ESR - 42 mm/hour.

Exercise:

1. What type of hemostasis disorder is observed in patient V.? Explain
2. the mechanism of hemorrhage and pancytopenia in sick V.

Answer.

1. Patient V. has a tendency to increased bleeding or hemorrhagic diathesis.

2. Hemorrhages in patient V. are associated with a decrease in the amount platelets in peripheral blood, i.e. thrombocytopenia.$_{IN}$The formation of thrombocytopenia is based on three important mechanisms; disruption of the formation of platelets in the bone marrow, increased destruction of platelets as a result of an immunological conflict of the cytotoxic type and increased their breakdown in the spleen due to splenomegaly. In patient V., along with thrombocytopenia, there is a decrease in the amount of hemoglobin and red blood cells, which indicates the development of anemia. Moreover, the absence of reticulocytes in a blood test indicates the hyporegenerative nature of anemia. In combination with thrombocytopenia and anemia, a sharp decrease in the number of leukocytes is observed. Such changes in the blood of patient V.

indicate pancytopenia, i.e. a decrease in the amount of all formed elements in the blood. Therefore, we can make the assumption that the damage occurs at the level of the pluripotent stem cell of the hematopoietic system, as a result of which the observed changes, including hemorrhages, are probably associated with direct damage to the bone marrow.

Evaluation criteria:

Mark	Description
Great	Demonstrate a thorough understanding of the problem. Ability to analyze a situation and draw conclusions Demonstrate confident situation-solving skills Demonstration of professional thinking
Fine	Demonstrate significant understanding of the problem. Ability to analyze a situation Demonstration of situation-solving skills Demonstration of professional thinking
satisfactorily	Demonstration of partial understanding of the problem. Demonstration of insufficient ability to analyze a situation Demonstrating poor problem solving skills
unsatisfactory	Demonstrating a lack of understanding of the problem. There was no attempt to solve the problem.

- Oral survey, interview

List of questions for intermediate certification:

1. Subject and tasks of pathophysiology. The role of pathophysiology in the system of medical knowledge.
2. Basic methods of pathophysiology. Pathophysiological experiment and its characteristics. Moral and ethical aspects of experimentation on animals.
3. The main stages of development of pathophysiology. The leading role of domestic scientists in its formation and development.
4. Basic concepts of general nosology: etiology, pathogenesis, pathological process, pathological condition, typical pathological processes.
5. Modern ideas about etiology. Etiotropic therapy.

6. The concept of pathogenesis. Causal relationships in pathogenesis. Leading links in pathogenesis, “vicious circles”. Pathogenetic therapy.

7. Types of reactivity. The structure of individual reactivity.

8. The role of gender and age in the reactivity and resistance of the body.

9. The role of heredity and constitution in the reactivity of the body.

10. Basic principles of classification of constitutional types.

11. Acquired reactivity: biological and social factors influencing its formation.

12. Mechanisms of formation of acquired reactivity. The role of the initial functional state in the reactivity of the body.

13. The role of the nervous system in the mechanisms of the body’s reactivity.

14. The role of the endocrine system and metabolism in the mechanisms of the body’s reactivity.

15. The role of immunological mechanisms in the reactivity of the body.

16. The concept of heredity. Methods for studying hereditary diseases.

17. Classification of hereditary diseases: a) according to the relative role of heredity and environment; b) depending on the level of damage to the heredity apparatus.

18. Congenital diseases. Phenocopying and its causes.

19. Etiology of hereditary diseases. Mutations and their causes.

20. Patterns of inheritance of dominant, recessive and sex-linked diseases.

21. General pathogenesis of hereditary diseases at the molecular level.

22. Hereditary enzymopathies and mechanisms of their development.

23. Mechanisms of hereditary predisposition to the development of diseases.

24. Chromosomal diseases and mechanisms of their development.

25. Isolates, inbreeding and their role in the pathology of heredity. Principles of prevention and possible methods of treatment of hereditary disease.

26. Pathogenic effect of low temperature on the body. Characteristics of forms and stages of overheating.

27. Pathogenesis of hyperthermia. Thermoregulation disorders.

28. Pathogenic effect of high temperature on the body. Stages of hyperthermia.

29. Pathogenic effect of electric current on the body. Factors determining the outcome of electrical injury. Causes of death due to electrical injury.

30. Damaging effects of ionizing radiation at the cellular level.
31. The concept of radioresistance and radiodamage to tissues. Mechanisms of action of ionizing radiation at the systemic and organismal levels.
32. Forms of radiation sickness. Pathogenesis of the acute (hemic) form. Pancytopenic syndrome.
33. Long-term effects of ionizing radiation.
34. Typical peripheral circulatory disorders. Their types, etiology, manifestations.
35. General phenomena of microcirculation disorders.
36. Hyperemia. Types, causes, mechanisms of development.
37. Arterial hyperemia. Types, features of microcirculation, development mechanisms, general manifestations, consequences.
38. Venous hyperemia. Causes of occurrence, mechanisms of development, external manifestations, features of microcirculation, consequences.
39. Ischemia. Causes of occurrence, classification, mechanisms of development, manifestations, features of microcirculation, outcomes.
40. Thrombosis and embolism. Causes, development mechanisms, outcomes.
41. The concept of inflammation. Etiology of inflammation.
42. Classification of inflammation (according to etiology, duration, nature of exudate, prevalence of processes at the site of inflammation).
43. Signs of inflammation: morphological, physicochemical, clinical.
44. Leading links in the pathogenesis of acute inflammation.
45. Primary and secondary alteration, development mechanisms.
46. Inflammatory mediators, their general biological effects.
47. Exudation and its mechanisms.
48. Emigration of leukocytes at the site of inflammation and its mechanisms. The role of phagocytosis in the pathogenesis of inflammation.
49. Violation of microcirculation and hemorheology in the area of inflammation.
50. Proliferation at the site of inflammation and its mechanisms. Outcomes of inflammation.
51. Conditions necessary for the development of purulent inflammation. Composition of pus.
52. Conditions necessary for the development of chronic inflammation.
53. Types of chronic inflammation. Composition of granuloma.
54. General reactions during inflammation.

55. Biological significance of inflammation. General principles of pathogenetic therapy of inflammation and their rationale.

56. Definition of “fever”. Etiology of fever.

57. Pyrogens. Their types and mechanisms of action.

58. Features of heat exchange at different stages of fever. Mechanisms of increased body temperature during fever.

59. Mechanisms of trembling during fever.

60. Mechanisms of heat during fever.

61. Mechanisms of sweating during fever.

62. The main differences between the mechanisms of development of fever and endogenous overheating.

63. Changes in metabolism and physiological functions during fever.

64. Biological significance of the feverish reaction. Pathophysiological principles of antipyretic therapy. The concept of pyrotherapy.

65. Energy metabolism disorders. Causes, development mechanisms, consequences.

66. Disturbances in the breakdown and absorption of carbohydrates. Causes, development mechanisms, consequences.

67. Disorders of interstitial carbohydrate metabolism. Causes, development mechanisms, consequences.

68. Etiology and pathogenesis of hyperglycemia.

69. Etiology and pathogenesis of hypoglycemia.

70. Disturbances in the breakdown and absorption of proteins. Causes, development mechanisms, consequences.

71. Disorders of interstitial protein metabolism. Disorders of protein synthesis and catabolism, disturbances in the conversion of amino acids.

72. Disorders of the final stages of protein metabolism. Disorders of binding and excretion of toxic products of nitrogen metabolism.

73. The main manifestations of nitrogen balance disturbances, their mechanisms.

74. Disturbances in the breakdown and absorption of fats. Causes, development mechanisms, consequences.

75. Hyperlipemia. Types, mechanisms of development.

76. Disorders of interstitial fat metabolism (general obesity, increased mobilization of fat from the depot, fatty liver, impaired conversion of fatty acids).

77. The role of free radical oxidation of lipids in pathology.

78. Cholesterol metabolism disorder. Hypercholesteremia. The role of lipid metabolism disorders in the pathogenesis of atherosclerosis.

79. Acidosis. Types, causes, development mechanism, compensatory reactions in the body, consequences for the body.

80. Alkalosis. Types, causes, development mechanism, compensatory reactions in the body, consequences for the body.

81. Electrolyte metabolism disorders.

82. Basic mechanisms of edema formation.

83. Fasting and its causes. Social causes of hunger. Changes in metabolism and physiological functions during different periods of fasting.

84. Definition of the concept of “hypoxia”. Hypoxia as a pathogenetic factor of various diseases.

85. Etiology, pathogenesis and types of hypoxia.

86. Blood oxygenation during various types of hypoxia.

87. Disorders of metabolism and physiological functions during hypoxia.

88. Mechanisms of immediate and long-term adaptation to hypoxia. Pathophysiological basis for the prevention and treatment of hypoxia.

89. Disturbances in the system of natural resistance factors and their connection with immunological reactivity. Pathology of phagocytosis.

90. Immunodeficient state (primary, secondary). Pathogenetic classification of acquired immunodeficiencies.

91. The concept of allergies. Classification of allergic reactions.

92. Classification of exo- and endoallergens.

93. Sensitization and its mechanisms in allergies of immediate and delayed types.

94. Stages of allergic reactions. Their brief description.

95. Pathogenesis of immediate type allergic reactions.

96. General biological effects of immediate allergy mediators.

97. Pathogenesis of delayed-type allergic reactions.

98. General biological effects of delayed allergy mediators.

99. Mechanisms of development of autoallergy.

100. The concept of shock as a typical pathological process. Etiology and types of shock.

101. Stages of shock. Leading links in the pathogenesis of shock conditions. Pathogenetic classification of shock stages.

102. Neuro-endocrine disorders in shock.

103. Disturbances of systemic hemodynamics and microcirculation during shock.

104. Metabolic disorders during shock. Irreversible changes. The concept of “traumatic illness”.

105. General principles of pathogenetic therapy of shock.

106. Stress. Etiology, stages, general pathogenesis of stress.

107. Disorders in the body under stress.

108. The role of stress in the occurrence of pathological processes and diseases.

109. Concept of tumors. Etiology of tumors.

110. The role of disturbances in the body’s reactivity in the occurrence and development of tumors (the significance of hereditary factors, changes in the nervous, endocrine and immune systems).

111. Initial (cellular) mechanisms of carcinogenesis.

112. Biological features of tumors.

113. Autonomy of tumor growth and its mechanisms.

114. Invasiveness of tumor growth, its mechanisms. Metastasis, pathways, mechanisms.

115. Tumor progression. Systemic effect of the tumor on the body (effect on the nervous, endocrine, immune systems and metabolism).

116. The concept of erythron. Dysregulation of erythropoiesis. Pathological forms of red blood cells.

117. Erythrocytosis and erythremia. Etiology, types and mechanisms of development.

118. Anemia. Definition, pathogenetic classification. General manifestations of anemia.

119. Acute and chronic posthemorrhagic anemia. Etiology, pathogenesis, blood picture.

120. Iron deficiency anemia. Etiology, pathogenesis, blood picture.

121. Anemia due to lack of vitamin $B_{12}$and folic acid. Etiology, pathogenesis, blood picture.

122. Hypo- and aplastic anemia. Etiology, pathogenesis, blood picture.

123. Disorders of physiological functions and compensatory-adaptive reactions in anemia.

124. Leukocyte formula and its disorders in pathological conditions.

125. Leukocytoses and their types. Causes. Phases of the leukocyte reaction during the infectious process.

126. Leukopenia. Causes and mechanisms of development.

127. Concept of leukemia. Classification and etiology of leukemia.

128. General pathogenesis of leukemia. General manifestations of leukemia. The mechanism of their development.

129. Features of hematopoiesis and cellular composition of peripheral blood in different types of leukemia.

130. Mechanism of hemostasis. Classification of hemostasis disorders.

131. Angiopathy. Types, etiology, pathogenesis.

132. Thrombocytopathies. Types, etiology, pathogenesis.

133. Coagulopathies. Types, etiology, pathogenesis.

134. Fibrinolytic system. Fibrinolysis disorders.

135. Disseminated intravascular coagulation blood (thrombohemorrhagic syndrome).

136. Changes in total blood volume: hypo- and hypervolemia. Types, etiology, pathogenesis.

137. Acute blood loss and posthemorrhagic syndrome. Leading links in pathogenesis.

138. Protective-adaptive and compensatory reactions during blood loss. Principles of pathogenetic therapy.

139. Cardiac arrhythmias. Their types, etiology.

140. Arrhythmias due to disorders of cardiac automaticity. Types, mechanisms of development.

141. Arrhythmias due to combined excitability and conduction disorders. Types, general pathogenesis.

142. Extrasystole and paroxysmal tachycardia. Types, mechanisms of development.

143. Atrial flutter. Etiology, mechanism of development.

144. Atrial and ventricular fibrillation. Development mechanism. consequences for the body.

145. Cardiac conduction block. Types, mechanisms of development.

146. Cardiac contractility disorders. Manifestations, development mechanisms.

147. Circulatory failure. Its forms, manifestations.

148. Heart failure. Etiology, developmental mechanisms and main manifestations.

149. Mechanisms of compensation for heart failure.

150. Myocardial hypertrophy. Mechanisms of development. failure of a hypertrophied heart.

151. Coronary circulatory disorders. Etiology and pathogenesis of myocardial infarction. Experimental reproduction.

152. Heart failure due to pericardial pathology. Mechanisms of development, manifestations.

153. Arterial hypertension. Pathogenetic classification of hypertensive conditions.

154. Hemodynamic parameters and forms of arterial hypertension.

155. The role of nervous regulation disorders in the pathogenesis of arterial hypertension.

156. The role of renal pressor and depressor mechanisms in the pathogenesis of arterial hypertension.

157. The role of changes in the state of the vascular wall in the mechanisms of development of arterial hypertension.

158. The role of the renin-angiotensin-aldosterone system in the mechanism of development of arterial hypertension.

159. Experimental reproduction of arterial hypertension. Principles of treatment of hypertension.

160. Arterial hypotension (acute and chronic). Etiology, pathogenesis.

161. The concept of respiratory failure. Forms and etiological factors of respiratory failure.

162. Indicators of respiratory failure.

163. Pathological forms of breathing and their mechanisms: shortness of breath, periodic and terminal breathing.

164. Disorders of alveolar ventilation. Obstructive and restrictive types of ventilation disorders. Alveolocapillary diffusion disorders.

165. Impaired pulmonary perfusion. Uneven ventilation-perfusion relationships. Insufficient pulmonary perfusion. Pulmonary edema.

166. Insufficiency of the digestive systems. Concept. Etiology. Basic mechanisms and manifestations.

167. Digestive disorders in the oral cavity.

168. Digestive disorders in the stomach. Etiology, pathogenesis, manifestations.

169. Digestive disorders in the duodenum and small intestine.

170. Disorders of the exocrine function of the pancreas.

171. Concept of liver failure. Etiology, general manifestations.

172. Metabolic disorders in hepatocellular failure.

173. Violations of the barrier and detoxification functions of the liver.

174. Portal circulation disorders (portal hypertension syndrome).

175. Hepato-cerebral insufficiency.

176. Hepatic coma. Basic mechanisms of development.

177. Prehepatic jaundice. Etiology, pathogenesis of bilirubin metabolism disorders.

178. Hepatic jaundice. Etiology, pathogenesis of bilirubin metabolism disorders.

179. Subhepatic jaundice. Etiology, pathogenesis of bilirubin metabolism disorders.

180. Extrarenal and renal causes of renal dysfunction. Disorders of glomerular filtration, tubular reabsorption, secretion and excretion.

181. Basic renal dysfunctions and their manifestations. Changes in diuresis and urine composition.

182. Renal and extrarenal manifestations of kidney pathology.

183. Etiology and pathogenesis of nephrotic syndrome. Mechanisms of formation of the main manifestations of nephrotic syndrome.

184. Etiology and pathogenesis of acute renal failure (ARF). Mechanisms of formation of the main manifestations of acute renal failure.

185. Etiology and pathogenesis of chronic renal failure (CRF). Mechanisms of formation of the main manifestations of chronic renal failure.

186. Experimental modeling of kidney pathology.

187. Characteristics of the neurohumoral regulatory mechanism as a functional system and its disturbances in endocrine and non-endocrine pathologies.

188. Disorders of the hypothalamic-pituitary regulation of the function of the endocrine glands. Damage to the self-regulation mechanism in the neuroendocrine system.

189. Trans- and parapituitary mechanisms of regulatory disorders.

190. Primary disorders of hormone synthesis in peripheral endocrine glands. Genetically determined defects in hormone biosynthesis.

191. Peripheral (extraglandular) forms of endocrine disorders, disturbances in the binding, utilization and metabolism of hormones. Changes in the reactive properties of hormonal receptors.

192. Pathology of the hypothalamic-adenopituitary system. Partial and total hypo- and hyperfunction of the anterior pituitary gland.

193. Pathology of the hypothalamic-neurohypophyseal system.

194. Pathology of the adrenal cortex. Partial and total hyperfunction.

195. Hypercortisolism. Etiology, pathogenesis, main manifestations.

196. Aldosteronism. Etiology, pathogenesis, main manifestations.

197. Adrenogenital syndromes. Etiology, pathogenesis, main manifestations.

198. Acute and chronic insufficiency of the adrenal cortex.

199. Hyperthyroidism. Etiology, pathogenesis, main manifestations.

200. Hypothyroidism. Etiology, pathogenesis, main manifestations.

201. Pathology of the parathyroid glands: hyper- and hypoparathyroidism. Etiology, pathogenesis, main manifestations.

202. Pathology of the gonads: hypo- and hypergonadism in women. Etiology, pathogenesis, main manifestations.

203. Pathology of the gonads: hypo- and hypergonadism in men. Etiology, pathogenesis, main manifestations.

204. Etiology and pathogenesis of diabetes mellitus.

205. Metabolic disorders in diabetes mellitus. Pathogenesis of diabetic coma.

206. Possible manifestations of diabetes mellitus associated with disorders of carbohydrate metabolism.

207. Possible manifestations of diabetes mellitus associated with protein metabolism disorders.

208. Possible manifestations of diabetes mellitus associated with lipid metabolism disorders.

209. The mechanism of formation of atherosclerosis in diabetes mellitus.

210. The mechanism of formation of angiopathy in diabetes mellitus.

211. Possible complications of diabetes.

212. General principles of treatment of diabetes mellitus.

213. General etiology and general pathogenesis of disorders of the nervous system. Functional disorders of neurons, glia, and synaptic transmission.

214. Sensory impairments. Various levels of sensitivity disorders. Their types and mechanisms of development.

215. Pathophysiology of the reticular formation.

216. Pathophysiology of the hypothalamus. The concept of diencephalic syndromes.

217. Dysfunction of the autonomic nervous system. Types, mechanisms. The concept of vegetative dystonia.

218. Disorders of the motor function of the nervous system. Pyramidal and extrapyramidal disorders.

219. Pathophysiology of extrapyramidal disorders.

220. Disorders of the trophic function of the nervous system. Neurodystrophies. Mechanisms of their development.

221. Spinal shock. Etiology, disorders in the body.

222. Brown-Séquard syndrome. Etiology, main manifestations.

223. The main manifestations of vago-insular crisis.

224. The main manifestations of sipathic-adrenal crisis.

225. Basic theories of nervous dystrophies and their rationale.

226. Morphological manifestations of nervous dystrophies at the cell and organ level.

227. Disorders of higher nervous activity. The concept of neuroses. Biological and social objects of the etiology and pathogenesis of neuroses.

228. Etiology of pain.

229. Pathogenesis of acute pain.

230. Pathogenesis of chronic pain.

231. Changes in the body during pain. General principles of pain treatment.

Interview assessment criteria:

Mark	**Description**
Great	The mark "EXCELLENT" is given to an answer that shows a solid knowledge of the basic processes of the subject area being studied and is distinguished by the depth and completeness of the topic; mastery of terminology; the ability to explain the essence of phenomena, processes, events, draw conclusions and generalizations, give reasoned answers, give examples; fluency in monologue speech, logic and consistency of response.
Fine	The mark “GOOD” evaluates an answer that reveals a solid knowledge of the basic processes of the subject area being studied, and is distinguished by the depth and completeness of the topic; mastery of terminology; the ability to explain the essence of phenomena, processes, events, draw conclusions and generalizations, give reasoned answers, give examples; fluency in monologue speech,

	logic and consistency of the answer. However, one or two inaccuracies in the answer are allowed.
satisfactorily	The mark “SATISFACTORY” evaluates an answer that mainly indicates knowledge of the processes of the subject area being studied, characterized by insufficient depth and completeness of the topic; knowledge of the basic issues of theory; poorly developed skills in analyzing phenomena and processes, insufficient ability to give reasoned answers and give examples; insufficient fluency in monologue speech, logic and consistency of response. There may be several errors in the content of the answer.
unsatisfactory	The mark "UNSATISFACTORY" evaluates an answer that reveals ignorance of the processes of the subject area being studied, characterized by a shallow disclosure of the topic; ignorance of the basic issues of theory, unformed skills in analyzing phenomena and processes; inability to give reasoned answers, poor command of monologue speech, lack of logic and consistency. Serious errors in the content of the answer are allowed.

8. Description of indicators and criteria for assessing competencies at the stages of their formation, description of assessment scales

Criteria	Levels of competency development		
	Threshold	***Sufficient***	***High***
	Competence formed. Demonstrated threshold, satisfactory sustainable level practical skill	Competence formed. Demonstrated enough level independence, sustainable practical skill	Competence formed. Demonstrated high level independence, high adaptability practical skill

Competency assessment indicators and rating scales

Grade "unsatisfactory o" (not counted) or absence formation competencies	Grade "satisfactorily" (passed) or satisfactory (threshold) level of development competencies	Rated "good" (passed) or sufficient level development competencies	Excellent rating (passed) or high level development competencies
failure to student on one's own will demonstrate b knowledge at solving tasks, absence independence in use skills. Absence confirmation availability formation competencies indicates negative results mastering educational disciplines	student demonstrates independence in use knowledge, skills and skills to solve educational assignments in complete According to sample given teacher, on assignments, the solution of which was shown teacher, should be considered what competence formed on satisfactorily m level.	student demonstrates on one's own e application knowledge, skills and skills at decision tasks, tasks similar samples that confirms Availability formed th competence for more high level. Availability of such competencies for sufficient level testify t about stable fixed practical skill	student demonstrates ability to full independence and in choice way to solve non-standard assignments within disciplines with using knowledge, skills and skills, received as in development progress given disciplines and adjacent disciplines should be considered competence formed on high level.