PATHOPHYSIOLOGY OF ORGANS AND SYSTEMS

Methodical recommendations for teachers regarding preparation for students' practical classes (majoring in "Medicine" and "Dentistry")

МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
Харківський національний медичний університет

PATHOPHYSIOLOGY OF ORGANS AND SYSTEMS

Methodical recommendations for teachers regarding preparation for students' practical classes (majoring in "Medicine" and "Dentistry")

ПАТОФІЗІОЛОГІЯ ОРГАНІВ ТА СИСТЕМ

Методичні рекомендації для викладачів щодо підготовки до практичних занять студентів (спеціальність «Медицина» та «Стоматологія»)

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Патофізіологія органів та систем: методичні рекомендації для викладачів щодо підготовки до практичних занять студентів (спеціальність «Медицина» та «Стоматологія») / упоряд. М. С. Мирошниченко, В. О. Бібіченко, М. О. Кучерявченко, О. О. Павлова та ін. Харків : ХНМУ, 2023. 128 с.

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Substantial module 4. Pathophysiology of the blood system

Topic № 1. Changes in the total volume of the blood. Qualitative changes of erythrocytes and leukocytes. Erythrocytosis

Number of hours: 2 academic hours.

Relevance of the topic: Maintaining the stability of the internal environment of the body – homeostasis, provides the internal environment of the body, which unites all organs and systems at the humoral level and is represented by three components – blood, lymph and interstitial fluid, the composition and properties of which are closely related to each other.The blood system, as well as other systems of the human body, can change under the influence of environmental factors, or as a result of a violation of the activity of certain organs and systems that maintain the stability of morphological, protein, ion, electrolyte, gas and other blood components. As a result, the inherent functions of the blood are disturbed. The ability to interpret changes in the total volume (mass) of blood and its constituent elements (erythrocytes and leukocytes) is important for a doctor of any specialty.

Purpose of the lesson:

General: to be able to determine changes in the total volume of blood, as well as qualitative changes of erythrocytes and leukocytes.

Specifically:

Know:

1. Know the normal parameters of hematocrit in an adult.
2. Describe the main stages of erythrocyte formation.
3. Know the normal parameters of the number of reticulocytes in an adult.
4. Determine the number of reticulocytes in the blood.
5. Prepare and stain a blood smear according to Romanovsky-Giemsa and recognize the cellular elements of the erythrocyte series under its microscopy.

Be able to:

1. On the basis of hematocrit data, identify changes in the total volume of blood and give their characteristics.
2. To characterize the possible qualitative changes of erythrocytes occurring in anemia, to detect their presence during microscopy of a blood smear of an animal with experimental anemia.
3. Describe possible qualitative changes in leukocytes.

Practical experience:

1. Identify regenerative forms of erythrocytes in peripheral blood smears (stained according to Romanovsky or Pappenheim), interpret their presence or absence.
2. Identify the regenerative forms of erythrocytes in stained blood smears it is vital to evaluate the value of their presence or absence in the blood.
3. Describe the degenerative forms of erythrocytes in blood smears stained by Romanovsky or Pappenheim, to interpret their presence in the blood.

4. Determine the pathological forms of erythrocytes in a smear of peripheral blood, interpret their presence in the blood.

5. Calculate the content of erythrocytes in a unit of blood volume, draw a conclusion about violation of the quantitative composition of "red" blood (anemia, erythrocytosis).

Technological map of students' work on the topic "Changes in the total volume of the blood. Qualitative changes of erythrocytes and leukocytes. Erythrocytosis"

№	Stage lesson	Academic time, min	Educational guide		Place holding a class
			Educational tools	Equipment	
1	Determination of the basic level of knowledge	10	Written answer to test tasks	Test tasks	Study room
2	Analysis of theoretical material	35	Analysis of theoretical material based on control questions of the topic, situational tasks, tasks of KROK-1	Topic control questions, KROK-1 tasks, situational tasks	
3	Practical part (conduct experiment)	30	Introduction and preparation for setting up the experiment. Setting up the experiment. Discussion of the results of the experiment and formulation the conclusions	Rabbit, microscopes, immersion oil, subject slides, polish slides, hydrochloric acid solution, distillates for painting of smears, fixation, Romanovsky staining, injector, pins, Petri-dish, solution of brilliant cresyl blue	
4	Determination of the final level of knowledge and skills. Summarizing the results	15	Determination of the final level of knowledge and skills. Summarizing the results	KROK-1 tasks, situational tasks	

The graphological structure of the topic "Changes in the total volume of the blood. Qualitative changes of erythrocytes and leukocytes. Erythrocytosis." is attached.

Material and methodological support of the topic "Changes in the total volume of the blood. Qualitative changes of erythrocytes and leukocytes. Erythrocytosis":

1. Lectures;
2. Methodical instructions for teachers;
3. Methodical instructions for students;
4. Set of test tasks to determine the basic level of knowledge;
5. Set of situational tasks to determine the final level of knowledge;
6. Set of KROK-1 tasks;
7. Set of schemes and tables (presentation);
8. Set of forms with a clinical blood test;
9. Video films;

10. For the experiment (experimental animals – rabbit, microscopes, immersion oil, subject slides, polish slides, hydrochloric acid solution, distillates for painting of smears, fixation, Romanovsky staining, injector, pins, Petri-dish, solution of brilliant cresyl blue).

Lesson content:

1. Changes in the total volume of blood
2. Hypervolemia
3. Hypovolemia
4. Hemodynamic disturbances in hemorrhage
5. The qualitative changes of erythrocytes
6. The qualitative changes in leukocytes
7. Erythrocytosis

Setting up the experiment. Discussion the results and formulation the conclusions

- **Studying the qualitative changes of erythrocytes in experimental hemolytic anemia.**

The conduction of the experiment: take the blood from the marginal vein of the ear of the anemic and control rabbits. Prepare smears. Put the blood on the fatless subject slide. The polished slide place slopingly to the subject slide (angle of slopy is 45 degree) till the contact with the drop. When the drop of blood spills on the rim of polished slide, move it along the subject slide. The smear must be rather thin and pro-portional. Dry the smear, and then fix it in the mixture of spirit with the ether for 5 min., after that stain according to Romanovsky during 15 min. After that wash the smear by thin stream of the water and dry it. Microscope the smear. Put the drop of immersion oil on the smear and look at it under the microscope with immersion objective. Pay attention to hypochromic erythrocytes, anisocytosis, poikilocytosis, polychromatophils (erythrocytes stained by acid and basic stains, which have violet or violet-blue colour in destinction from mature erythrocytes stained in pink colour) and normoblasts erythrocytes with nuclears. Compare the content of qualitative changed erythrocytes in the blood of experimental and control rabbits.

Describe and draw the slides in details.

- **Studing the reticylocytes in experimental hemolytic anemia**

The conduction of the experiment: take the thin part of brilliant cresyl blue subject slide and mark this site by colour pencil. Make the smear of the blood and without drying quickly place it into a humid chamber (Petri-dish with humid filter paper). In 15 min. take the smear off, dry it in the air and microscope it. Erythrocytes are stained into green colour. In some of them there is a thin dark blue net which is placed either in all cells or in its center, where the denser ball is formed. This net is called substantia reticulogranulofilamentosa. Erythrocytes with such granules are called reticulocytes. Compare the amount of reticulocytes in the blood of experimental and control rabbits.

Describe and draw the slides in details.

Discussion of the results of the experiment

Changes on the part of red blood cells are expressed primarily in changes in form, that is, a change in the quality of erythrocytes – quantity, anisocytosis, a change in color – hypochromic, hyperchromic erythrocytes; possible pathological inclusions in erythrocytes – Cabot rings, Zolla bodies.

Changes in the structure of leukocytes are also possible. List again the degenerative forms of leukocytes.

With hemolytic anemia, the release of reticulocytes from the bone marrow into the peripheral blood stream increases.

Formulation the conclusions based on the experiment

A change in the shape of blood cells – erythrocytes and leukocytes, ultimately leads to a violation of their physiological functions. A change in structure entails a change in function, which, in turn, leads to the development of pathological conditions associated with a disturbance in the blood cells themselves.

An increase in the number of reticulocytes in the peripheral blood indicates hemlytic or posthemorrhagic anemia and is a compensatory reaction of the body aimed at normalizing the number of erythrocytes.

Terminology:

- Hypervolemia
- Hypovolemia
- Polycythemia
- Oligocythemia
- Anisochromic erythrocytes
- Anisocytes
- Poikilocytes

Tasks for independent work on the topic "Changes in the total volume of the blood. Qualitative changes of erythrocytes and leukocytes. Erythrocytosis"

The student is offered to investigate the results of a clinical blood analysis of a patient with a disorder in the blood system. It is necessary to determine the signs and type of violation. Be able to explain the mechanism of occurrence. Analysis the errors with an explanation of the correct answers.

List of questions and works to be studied:

1. Violations of total amount of blood. Normovolemia. Types of a normovolemia, causes of their development.
2. Hypervolemia. Types of a hypervolemia, causes and mechanisms of development. Pathogenetic value of a hypervolemia.
3. Hypovolemia. Types of a hypovolemia, causes and mechanisms of development. Pathogenetic value of a hypovolemia.
4. Definition of the terms "blood loss", "bleeding", "hemorrhage", "hematoma". Etiology and classification of blood loss.
5. Acute blood loss. Pathogenesis. The pathological changes happening at blood loss. Protective and compensatory reactions at blood loss.

6. Qualitative changes of erythrocytes, their causes.
7. Qualitative changes of leukocytes, their causes.

List of practical skills that must be mastered:

1. Identify the regenerative forms of erythrocytes in peripheral blood smears (stained according to Romanovsky or Pappenheim), interpret their presence or absence.
2. Identify the regenerative forms of erythrocytes in stained blood smears it is vital to evaluate the value of their presence or absence in the blood.
3. Describe the degenerative forms of erythrocytes in blood smears stained by Romanovsky or Pappenheim, to interpret their presence in the blood.
4. Determine the pathological forms of erythrocytes in a smear of peripheral blood, interpret their presence in the blood.
5. Calculate the content of erythrocytes in a unit of blood volume, draw a conclusion about violation of the quantitative composition of "red" blood (anemia, erythrocytosis).

Situational tasks KROK-1 to determine the final level of knowledge

1. A 32-year-old patient was admitted to the hospital with gross blood loss due to auto accident trauma. Ps – 110 Bpm, RR – 22 pm, BP – 100/60 mm Hg. What changes in the blood will occur in an hour after the blood loss?

A. Hypovolemia. *D. Leukopenia.*
B. Erythropenia. *E. Hypoproteinemia.*
C. Hypochromia of erythrocytes.

2. A 26-year-old pregnant woman is under treatment in hospital. After a continuous attack of vomiting she was found to have reduced volume of circulating blood. What kind of change in general blood volume is the case?

A. Polycythemic hypovolemia. *D. Simple hypovolemia.*
B. Oligocythemic hypervolemia. *E. Oligocythemic hypovolemia.*
C. Polycythemic hypervolemia.

3. In a car accident a man got injured and lost a lot of blood. What changes in peripheral blood are most likely to occur on the 2nd day after the injury?

A. Erythropenia. *C. Anisocytosis.* *E. Significant reticulocytosis.*
B. Microplania. *D. Hypochromia.*

4. On the fifth day after the acute blood loss a patient has been diagnosed with hypochromic anemia. What is the main mechanism of hypochromia development?

A. Release of immature red blood cells from the bone marrow.
B. Increased destruction of red blood cells in the spleen.
C. Increased excretion of body iron.
D. Impaired iron absorption in the intestines.
E. Impaired globin synthesis.

5. A 42 year old patient complains of pain in the epigastral area, vomiting; vomit masses have the colour of “coffee-grounds”, the patient has also melena.

Anamnesis records gastric ulcer. Blood formula: erythrocytes – 2.8×10^{12}/l, leukocytes – 8×10^{9}/l, Hb – 90 g/l. What complication is it?

A. Haemorrhage. *C. Perforation.* *E. Pyloric stenosis.*
B. Penetration. *D. Canceration.*

6. A 30-year-old patient's blood test revealed the following: erythrocyte count is 6×10^{12}/l, hemoglobin is 10.55 mmol/l. Vaquez disease was diagnosed. Name the leading part of pathogenesis:

A. B12-deficiency. *C. Neoplastic erythroid hyperplasia.* *E. Acidosis.*
B. Iron-deficiency. *D. Hypoxia.*

7. As a result of increased permeability of the erythrocyte membrane in a patient with microspherocytic anaemia (Minkowsky-Shauffard disease) cells receive sodium ions and water. Erythrocytes take form of spherocytes and can be easily broken down. What is the leading mechanism of erythrocyte damage in this case?

A. Electrolytic osmotic. *C. Acidotic.* *E. Nucleic.*
B. Calcium. *D. Protein.*

8. A 32-year-old patient was delivered to the clinic with massive blood loss as a result of an accident. Ps – 110 beats/min., RR – 22 per minute, blood pressure – 100/60 mm Hg. Art. Which of the following blood changes will be the most characteristic 1 hour after blood loss?

A. Erythropenia. *D. Hypoproteinemia.*
B. Hypochromia of erythrocytes. *E. Hypovolemia.*
C. Leucopenia.

9. In a person weighing 80 kg, after prolonged physical activity, the volume of circulating blood decreased, hematocrit – 50 %, total blood protein – 80 g/l. Such blood counts are the result, first of all:

A. Increased diuresis.
B. Increased plasma oncotic pressure.
C. Increase in the number of red blood cells.
D. Water loss through sweat.
E. Increase in blood proteins.

10. A week after massive blood loss, a patient has a large number (5 %) of regenerative forms of erythrocytes in the blood. Which ones?

A. Reticulocytes. *C. Megalocytes.* *E. Spherocyto.*
B. Megaloblasts. *D. Microcytes.*

Standards of correct answers to the task KROK-1

1	2	3	4	5	6	7	8	9	10
A	*A*	*A*	*A*	*A*	*C*	*A*	*E*	*D*	*A*

Recommendations for registration of work results

1. Written answer to test tasks (initial level of knowledge).
2. The results of the experiment are drawn up in the form of an experiment protocol with the determination of relevant conclusions.

3. Protocol for the study of the results of the patient's clinical blood analysis.

4. Protocol for solving situational tasks with an explanation of the correct answers.

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Topic № 2. Erythrocytosis. Anemias: definition of the concept, general clinical and hematological manifestations. Post-hemorrhagic anemias, anemias caused by erythropoiesis disorders, hemolytic anemias

Number of hours: 2 academic hours.

Relevance of the topic: The term "anemia" without details does not define a specific disease but only indicates the presence of a symptom that may be one of the manifestations of various pathological processes, that is, anemia can act as an independent nosological unit (for example, iron deficiency anemia, protein deficiency, thalassemia), and as symptoms diseases (leukemia, systemic lupus erythematosus, etc.). Under physiological conditions, the number of erythrocytes that are destroyed is equal to the number of newly formed ones, due to which their standard number is constantly maintained. Anemia develops when the synthesis of red blood cells in the bone marrow can no longer compensate for their shortened lifespan; The destruction of erythrocytes can result from the influence of various factors related to their recycling and the physicochemical properties of the environment. Under physiological conditions, aging erythrocytes are removed from the circulation and destroyed mainly in the spleen, liver, and to a lesser extent in the bone marrow by cells of the mononuclear system. Thus, knowledge of the complex pathophysiological mechanisms of development, course, and outcome of dyserythropoietic and hemolytic anemia is essential for diagnosing diseases in various fields of medicine. Knowledge of the main manifestations of anemia and the mechanisms of its development allows the doctor to determine the correct diagnosis, and choose adequate therapy, as well as preventive measures for the occurrence of this type of pathology.

Purpose of the lesson:

General – general - to be able to define, using data of quantitative and qualitative changes of erythrocytes, the existence of an anemia, wich duo to disturbances of erythropoiesis and/or hemolytic anemia.

Specifically:

Know:

1. According to number of erythrocytes, concentration of hemoglobin and a color indicator to resolve an issue of existence of anemia.
2. Characterize the possible qualitative changes of erythrocytes which are found at anemias to find their existence at blood dab microscopy of an animal with experimental anemia.
3. Classify anemias by their etiology, pathogenesis, quantitative and qualitative changes of erythrocytes and an erythropoesis, dynamics of a current.
4. Generalize the obtained data on quantitative and qualitative changes of erythrocytes at an animal and on the basis of it to give a conclusion concerning character of anemia.

Be able to:

1. To give the pathogenetic classification of anemia that arose as a result of impaired hematopoiesis and increased destruction of red blood cells (hemolytic).

2. To know the diagnostic features and the direction of changes in the indicators of the number of erythrocytes, hemoglobin concentration, color index, and the number of reticulocytes in anemia associated with impaired erythropoiesis and hemolytic anemia in children and adults.

3. To compare the normative indicators of the number of erythrocytes, hemoglobin concentration, color index, and the number of reticulocytes in a healthy with those in a patient with anemia associated with impaired erythropoiesis and/or hemolytic anemia.

4. To prepare and paint blood smear according to Romanovsky-Geimsa and to distinguish at its microscopy cellular elements of an erythrocyte row.

Practical experience:

1. Definition of hemoglobin amount in experimental hemolytic anemia.
2. Calculation of the number of erythrocytes in experimental hemolytic anemia.
3. Determination of anemia in a patient based on symptoms and data hemogram, description of peripheral blood smear.

Technological map of students' work on the topic "Erythrocytosis. Anemias: definition of the concept, general clinical and hematological manifestations. Post-hemorrhagic anemias, anemias caused by erythropoiesis disorders, hemolytic anemias."

№	Stage lesson	Academic time, min	Educational guide		Place holding a class
			Educational tools	Equipment	
1	Determination of the initial level of knowledge	10	Written answer to test tasks	Test tasks	Study room
2	Analysis of theoretical material	35	Analysis of theoretical material based on control questions of the topic, situational tasks, tasks of KROK-1	Topic control questions, KROK-1 tasks, situational tasks	
3	Practical part (conduct experiment)	30	Introduction and preparation for setting up the experiment. Setting up the experiment. Discussion of the results of the experiment and formulation of conclusions	Microscopes, Vidal's tubes, injectors, micropipets, 5,0 ml pipets, 10 % sol. Natrium chloridum, calculating chamber, hemometers, injectors, 0,1 % hydrochloric acid solution, distill water	
4.	Determination of the final level of knowledge and skills. Summarizing the results	15	Determination of the final level of knowledge and skills. Summarizing the results	KROK-1 tasks, situational tasks	

The graphological structure of the topic "Erythrocytosis. Anemias: definition of the concept, general clinical and hematological manifestations. Post-hemorrhagic anemias, anemias caused by erythropoiesis disorders, hemolytic anemias" is attached.

Material and methodological support of the topic "Erythrocytosis. Anemias: definition of the concept, general clinical and hematological manifestations. Post-hemorrhagic anemias, anemias caused by erythropoiesis disorders, hemolytic anemias":

1. Lectures;
2. Methodical instructions for teachers;
3. Methodical instructions for students;
4. Set of test tasks to determine the basic level of knowledge;
5. Set of situational tasks to determine the final level of knowledge;
6. Set of KROK-1 tasks;
7. Set of schemes and tables (presentation);
8. Set of forms with a clinical blood test
9. Video films;
10. For the experiment (experimental animals – rabbit, microscopes, immersion oil, Vidal's tubes, injectors, micropipets, 5,0 ml pipets, 10 % sol. Natrium chloridum, calculating chamber, hemometers, injectors, 0,1 % hydrochloric acid solution, distill water.).

Lesson content:

1. Classification of anemias
2. Etiology of posthemorrhagic anemia
3. Hematological signs of anemia
4. Clinical manifestations of anemia
5. Anemias witch duo to disturbances of erythropoiesis
6. Hemolytic anemia

Setting up the experiment. Discussion of results and formulation of conclusions

- **Calculation of the number of erythrocytes in experimental hemolytic anemia**

Take two rabbits before practical lesson daily one of them under control of hemoglobin was injected under skin 1 % sol. phenylhydrasin in increased dose: 1 injection – 0,25 ml; 2 – 0,5 ml; 3 – 0,75 ml; 4 – 1 ml.

Take 0,02 ml of blood by micropipete from marginal vein of ear and blow off into Vidal's tube with 3,98 ml one per cent of hydrochloric natrium solution.

After 3 times the washing of pipet shake up mixture carefully. By pipet for distill water from hemometer get the drop of mixture into calculating chamber. Calculate erythrocytes in 5 big (that is in 80 small) squares of Goryajev' net. By number of erythrocytes in 5 big squares calculate their numbers in 1 mkl of blood. Use the formula for calculation

$$X = \frac{A \times 4000 \times B}{C},$$

Where X – the number of erythrocytes;
A – the sum of erythrocytes, calculated in 5 big squares;
C – the number of calculated small squares (80);
B – the blood dilution (in 200 times);
4000 – multiplier, which brings the volium of the liquid in limits of small square (1/4000 mkl) to 1 mkl.

Calculate the number of erythrocytes for 1 L.

Compare the amount of erythrocytes of experimental and normal rabbits.

- **The definition of hemoglobin amount in experimental hemolytic anemia**

Investigate experimental and normal rabbits. Pour 0,1 N of hydrochloric acid into a middle tube of hemometer to the mark of 2 on the scale × 10 g/l. Then by the pipete from the hemometer fill 0,02 ml blood, wipe the end by cotton with great care, gently blow the blood into the tube, wash the pipete by solution twice. After 5 min. dilute the mixture by distill water till the color of solution in the tube will not corresponde with the colour of standart hydrochloric hematin solution.

Mark the figure on the lower level of meniskas on the tube which then must be multiplied by 10 to define hemoglobin in g/l.

Define the colour index according to the formula:

$$C.I. = \frac{\text{founded amount of hemoglobin}}{\text{normal amount of hemoglobin}} : \frac{\text{founded amount of erythrocytes}}{\text{normal amount of erythrocytes}}$$

Compare the amount of hemoglobin and colour index in anemia and normal rabbits.

Discussion of the results of the experiment

Changes on the part of blood in anemia include: a possible change in blood volume, a decrease in erythrocytes, a change in the composition of erythrocyte forms in connection with a possible compensatory change in erythropoiesis, changes in the quality of erythrocytes themselves; decrease in hemoglobin content, its qualitative changes.

Formulation the conclusions based on the experiment

Hemolytic anemias are erythroblastic, regenerative, normo- or hypochromic (rarely hyperchromic). Degenerative and regenerative forms are revealed. In hereditary forms of anemia, enhanced regeneration of the erythrocyte sprout is noted, but often with ineffective erythropoiesis, when the nuclear forms of erythrocytes are destroyed in the bone marrow. Hyporegenerative anemia may develop with frequent hemolytic crises.

Terminology:

- Erythrocytosis.
- Anemia.

- Diserythropoiesis
- Hemolisis
- Anisocytes
- Poikilocytes
- Hemoglobinemia,
- Hemoglobinuria,
- Methemalbuminemia,
- Jaundice,
- Hemosiderinuria.

Tasks for independent work on the topic "Erythrocytosis. Anemias: definition of the concept, general clinical and hematological manifestations. Post-hemorrhagic anemias, anemias caused by erythropoiesis disorders, hemolytic anemias".

The student is offered to investigate the results of a clinical blood analysis of a patient with a disorder in the blood system. It is necessary to determine the signs and type of violation. Be able to explain the mechanism of occurrence. Analysis of errors with an explanation of the correct answers.

List of questions and works to be studied:

1. Erythrocytosis. Definition of term, types, etiology, pathogenesis.
2. Clinical manifestations, changes in peripheral blood and marrow at an erythrocytosis.
3. Anemia. Definition of term. Classification of anemias.
4. General clinical and hematologic symptoms of anemias.
5. Posthemorrhagic anemia.
6. Iron deficiency anemia. Etiology, pathogenesis, leading clinical syndromes.
7. B_{12}-and anemia of folic acid deficiency. Etiology, pathogenesis, leading clinical syndromes.
8. Hypoplastic (aplastic) anemias. Etiology, pathogenesis, leading clinical syndromes.
9. Haemolytic anemias. Classification. Main clinical syndromes.
10. Intravascular hemolysis of erythrocytes. Causes, mechanisms. The changes developing in an organism owing to an intravascular hemolysis. Clinical and laboratory signs of an intravascular hemolysis.
11. Intracellular hemolysis of erythrocytes. Reasons, mechanisms. The changes developing in an organism owing to an intracellular hemolysis. Clinical and laboratory signs of an intracellular hemolysis.
12. Hereditary haemolytic anemias. Fermentopathy. Hemoglobinopathy. Membranopathy.
13. The acquired haemolytic anemias. Causes and mechanisms of development.

List of practical skills that must be mastered:

1. Definition of hemoglobin amount in experimental anemia.
2. Calculation of the number of erythrocytes in experimental anemia.
3. Determination of anemia in a patient based on symptoms and data hemogram, description of peripheral blood smear.
4. Determination of anemia in a patient based on symptoms and data hemogram, description of peripheral blood smear.

Situational tasks KROK-1 to determine the final level of knowledge

1. A 46-year-old female patient complaining of having alveolar haemorrhage for 6 hours after a tooth extraction, general weakness and dizziness was delivered to a hospital. The patient has a history of essential hypertension. Objectively: pale skin and mucous membranes. In blood: Hb – 80 g/l, Ht – 30%, bleeding and coagulation time is normal. What complication had been provoked by the haemorrhage?

A. Acute posthaemorrhagic anaemia. *D. Haemolytic anaemia.*
B. Iron deficiency anaemia. *E. Chronic posthaemorrhagic anaemia.*
C. Folic acid deficiency anaemia.

2. A 42 year old patient complains of pain in the epigastral area, vomiting; vomit masses have the colour of "coffee-grounds", the patient has also melena. Anamnesis records gastric ulcer. Blood formula: erythrocytes – $2{,}8\times10^{12}$/l, leukocytes – 8×10^{9}/l, Hb – 90 g/l. What complication is it?

A. Haemorrhage. *C. Perforation.* *E. Pyloric stenosis.*
B. Penetration. *D. Canceration.*

3. A 55 y.o. woman consulted a doctor about having continuous cyclic uterine hemorrhages for a year, weakness, dizziness. Examination revealed skin pallor. Hemogram: Hb – 70 g/l, erythrocytes – $3{,}2\times10^{12}$/l, color index – 0,6, leukocytes – $6{,}0\times10^{9}$/l, reticulocytes – 1 %; erythrocyte hypochromia. What anemia is it?

A. Chronic posthemorrhagic anemia. *D. B_{12}-folate-deficiency anemia.*
B. Hemolytic anemia. *E. Iron-deficiency anemia.*
C. Aplastic anemia.

4. A 30-year-old patient's blood test revealed the following: erythrocyte count is 6×10^{12}/l, hemoglobin is 10,55 mmol/l. Vaquez's disease was diagnosed. Name the leading part of pathogenesis:

A. B12-deficiency. *D. Hypoxia.*
B. Iron-deficiency. *E. Acidosis.*
C. Neoplastic erythroid hyperplasia.

5. A 38-year-old woman with gastric bleeding was brought to the admission and diagnostic department. What changes are most likely in the blood in a day?

A. Erythrocytosis. *C. Leukopenia.* *E. Leukocytosis.*
B. Decreased hematocrit. *D. Increased hematocrit.*

6. A 32-year-old patient was delivered to the clinic with massive blood loss as a result of an accident. Ps – 110 beats / min., RR – 22 per minute, blood pressure – 100/60 mm Hg. Art. Which of the following blood changes will be the most characteristic 1 hour after blood loss?

A. Erythropenia.
B. Hypochromia of erythrocytes.
C. Leucopenia.
D. Hypoproteinemia.
E. Hypovolemia.

7. Due to injury, the patient lost 25 % of the volume of circulating blood. Name the urgent mechanism for compensating for blood loss.

A. Entry of interstitial fluid into the vessels.
B. Restoration of the protein composition of the blood.
C. Increase in the number of reticulocytes.
D. Recovery of red blood cells.
E. Erythropoiesis activation.

8. A general examination of the patient revealed hyperemia of all skin with a cyanotic tint. Attention is drawn to the patient's lethargy and slowing down movements. Blood analysis showed: erythrocytes 9×10^{12}/l, hematocrit 60 %. At what pathological condition is absolute erythrocytosis:

A. Megaloblastic anemia.
B. Lymphoma.
C. Hemodilution.
D. Hemoconcentration.
E. Vaquez's disease.

9. On the fifth day after acute blood loss, the patient was diagnosed with hypochromic anemia. What is the main mechanism in the development of hypochromia?

A. Bone marrow supply of immature erythrocytes.
B. Intestinal iron malabsorption.
C. Increased destruction of red blood cells in the spleen.
D. Impaired synthesis of globin.
E. Increased excretion of iron from the body.

10. An ambulance delivered a 46-year-old patient to the clinic with complaints of alveolar bleeding within 6 hours after tooth extraction, general weakness, dizziness. She has a history of hypertension. Objectively: pale skin and visible mucous membranes. In the blood: Hb – 80 g/l, Ht – 30 %, indicators of bleeding time and blood clotting are within normal limits. What complication developed in the patient due to bleeding?

A. Hemolytic anemia.
B. Iron deficiency anemia.
C. Chronic posthemorrhagic anemia.
D. Acute posthemorrhagic anemia.
E. Folate deficiency anemia.

11. A 58-year-old female patient complains of rapid fatigability, performance decrement, sleepiness, dyspnea during fast walking. In blood: RBCs – $4{,}0\times10^{12}$/l, Hb – 80 g/l, CI – 0,6; alurge number of annulocytes and microcytes. What anaemia are these presentations typical for?

A. Iron-deficient.
B. Posthemorrhagic.
C. Sickle-cell.
D. Pernicious.
E. Haemolytic.

12. A 15 year old girl has pale skin, glossitis, gingivitis. Blood count: erythrocytes – $3,3\times10^{12}$/l, hemoglobin – 70 g/l, colour index – 0,5. Examination of blood smear revealed hypochromia, microcytosis, poikilocytosis. What type of anemia is it?

A. B_{12}*-folic acid-deficient.* *C. Iron-deficient.* *E. Thalassemia.*
B. Sickle-cell. *D. Hemolytic.*

13. Patient with hypochromic anemia has splitting hair and loss of hair, increased nail brittling and taste alteration. What is the mechanism of the development of these symptoms?

A. Deficiency of iron-containing enzymes.
B. Deficiency of vitamin B_{12}*.*
C. Decreased production of parathyrin.
D. Deficiency of vitamin A.
E. Decreased production of thyroid hormones.

14. 2 years ago a patient underwent resection of pyloric part of stomach. He complains of weakness, periodical dark shadows beneath his eyes, dyspnea. In blood: Hb – 70 g/l, erythrocytes – $3,0\times10^{12}$/l, colour index – 0,7. What changes of erythrocytes in blood smears are the most typical for this condition?

A. Megalocytes. *C. Schizocytes.* *E. Macrocytes.*
B. Microcytes. *D. Ovalocytes.*

15. A patient is diagnosed with iron-deficiency sideroachrestic anemia, progression of which is characterized by skin hyperpigmentation, pigmentary cirrhosis, heart and pancreas affection. Iron level in the blood serum is increased. What disorder of iron metabolism causes this disease?

A. Excess*ive iron intake with food.*
B. Disorder of iron absorption in bowels.
C. Failure to assimilate iron leading to iron accumulation in tissues.
D. Increased iron assimilation by body.
E. –.

16. A 37-year-old female patient complains of headache, vertigo, troubled sleep, numbness of limbs. For the last 6 years she has been working at the gas-discharge lamp-producing factory in the lead-processing shop. Blood test findings: low hemoglobin and RBC level, serum iron concentration exceeds the norm by several times. Specify the type of anemia:

A. Iron-deficiency anemia. *D. Iron refractory anemia.*
B. Minkowsky-Shauffard disease. *E. Metaplastic anemia.*
C. Hypoplastic anemia.

17. A year after subtotal stomach resection on account of ulcer of lesser curvature the following blood changes were revealed: anemia, leukocytopenia and thrombocytopenia, color index – 1,3, megaloblasts and megalocytes. What factor deficiency caused the development of those pathology?

A. Castle's factor. *C. Mucin.* *E. Gastrin.*
B. Hydrochloride acid. *D. Pepsin.*

18. Blood test of a patient suffering from atrophic gastritis gave the following results: RBCs – $2,0\times10^{12}$/l, Hb – 87 g/l, colour index – 1,3, WBCs – $4,0\times10^{9}$/l, thrombocytes – 180×10^{9}/l. Anaemia might have been caused by the following substance deficiency:

A. Vitamin K. *B. Iron.* *C. Vitamin A.* *D. Zinc.* *E. Vitamin B_{12}.*

19. A 50-year-old patient has been examined by a dentist and found to have crimson smooth tongue. Blood analysis revealed a decrease in RBC level and hemoglobin concentration, colour index of 1,3, symptoms of megaloblastic hematopoiesis, degenerative changes in WBCs. What blood disorder was found in this patient?

A. Hemolytic anemia.
B. B_{12}-folic-acid-deficiency anemia.
C. Myeloid leukemia.
D. Iron deficiency anemia.
E. Aplastic anemia.

20. A 56 year old man was taken to the hospital with complaints of general weakness, pain and burning in the region of tongue, extremity numbness. In the past, he had resection of cardiac part of ventricle. Blood test: Hb – 80 g/L; RBC – $2,0\times10^{12}$/L; colour index of blood – 1,2; leukocytes – $3,5\times10^{9}$/L. What type of anemia is it?

A. B_{12} folic-deficient. *C. Posthemorrhagic.* *E. Iron-deficient.*
B. Hemolytic. *D. Aplastic.*

21. A 56 year old patient came to a hospital with complaints about general weakness, tongue pain and burning, sensation of limb numbness. In the past, he underwent resection of fore stomach. In blood: Hb – 80 g/l; erythrocytes – $2,0\times10^{12}$/l; colour index – 1,2, leukocytes – $3,5\times10^{9}$/l. What anemia type is it?

A. B_{12}-folate deficient. *C. Posthemorrhagic.* *E. Iron-deficient.*
B. Hemolytic. *D. Aplastic.*

22. Examination of a 52-year-old female patient has revealed a decrease in the amount of red blood cells and an increase in free hemoglobin in the blood plasma (hemoglobinemia). Color index is 0.85. What type of anemia is being observed in the patient?

A. Acquired hemolytic.
B. Hereditary hemolytic.
C. Acute hemorrhagic.
D. Chronic hemorrhagic.
E. Anemia due to diminished erythropoiesis.

23. A 19 year old patient was diagnosed with chronic acquired hemolytic anemia. What is the leading pathogenetic mechanism of this pathology's development?

A. Toxic hemolysis.
B. Intracellular hemolysis.
C. Autoimmune hemolysis.
D. Hyposmolarity of plasma.
E. Osmotic hemolysis.

24. Substitution of the glutamic acid on valine was revealed while examining initial molecular structure. For what inherited pathology is this typical?

A. Sickle-cell anemia. *C. Minkowsky-Shauffard disease.* *E. Hemoglobinosis.*
B. Thalassemia. *D. Favism.*

25. A 19-year-old female patient has had low haemoglobin rate of 90–95 g/l since childhood. Blood count results obtained after hospitalization are as follows: erythrocytes – $3,2\times10^{12}$/l, Hb – 85 g/l, colour index – 0,78; leukocytes – $5,6\times10^{9}$/l, platelets – 210×10^{9}/l. Smear examination revealed anisocytosis, poikilocytosis and target cells. Reticulocyte rate is 6 %. Iron therapy was ineffective. What blood pathology corresponds with the described clinical presentations?

A. Membranopathy. *C. Sickle-cell anemia.* *E. Enzymopathy.*
B. Favism. *D. Thalassemia.*

26. A 25 year old Palestinian woman complains of weakness, dizziness, dyspnea. In anamnesis: periodically exacerbating anemia. In blood: Hb – 60 g/l, erythrocytes – $2,5\times10^{12}$/l, reticulocytes – 35 ‰, anisocytosis and poikilocytosis of erythrocytes, a lot of target cells and polychromatophils. What type of anemia is it?

A. Sickle-cell anemia.
B. Thalassemia.
C. Minkowsky-Shauffard disease.
D. Addison-Biermer disease.
E. Glucose 6-phosphate dehydrogenase-defi-cient anemia.

27. A 34 year old woman was diagnosed with hereditary microspherocytic hemolytic anemia (Minkowsky-Shauffard disease). What mechanism caused haemolysis of erythrocytes?

A. Membranopathy. *D. Autoimmune disorder.*
B. Enzymopathy. *E. Bone marrow hypoplasia.*
C. Hemoglobinopathy.

28. As a result of increased permeability of the erythrocyte membrane in a patient with microspherocytic anaemia (Minkowsky-Shauffard disease) cells receive sodium ions and water. Erythrocytes take form of spherocytes and can be easily broken down. What is the leading mechanism of erythrocyte damage in this case?

A. Calcium. *C. Protein.* *E. Nucleic.*
B. Acidotic. *D. Electrolytic osmotic.*

Standards of correct answers to the task КROK-1

1	2	3	4	5	6	7	8	9	10	11	12	13	14
A	*A*	*A*	*C*	*B*	*E*	*A*	*E*	*A*	*D*	*A*	*C*	*A*	*B*
15	**16**	**17**	**18**	**19**	**20**	**21**	**22**	**23**	**24**	**25**	**26**	**27**	**28**
C	*D*	*A*	*E*	*B*	*A*	*A*	*A*	*C*	*A*	*D*	*B*	*A*	*D*

Recommendations for registration of work results

1. Written answer to test tasks (initial level of knowledge).
2. The results of the experiment are drawn up in the form of an experiment protocol with the determination of relevant conclusions.
3. Protocol for the study of the results of the patient's clinical blood analysis.
4. Protocol for solving situational tasks with an explanation of the correct answers.

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Topic № 3. Leukocytosis. Leukopenia

Number of hours: 2 academic hours.

Relevance of the topic: Changes in the leukocyte composition of peripheral blood play an important role in determining the effectiveness of treatment and the prognosis of the disease. Leukocytosis in most cases reflects the satisfactory reactivity of the bone marrow hematopoietic system in response to the action of external and internal stimulators of leukopoiesis. Accounting for these indicators in dynamics the development of the disease and its treatment very often plays an important role in determining the effectiveness of the treatment and the prognosis of the disease. Knowledge of the causes, mechanisms of the occurrence and development of symptomatic changes in the leukocyte composition of blood in various pathological processes and diseases is of great importance. An idea about the number of leukocytes, the ratio of their individual forms in peripheral blood, as well as the peculiarities of their qualitative changes are necessary for a doctor of any specialty

Purpose of the lesson:

General – to be able to understand the main symptomatic quantitative and qualitative changes of leucocyte composition of blood in the conditions of pathology, to know the possible causes and mechanisms of their emergence and development, to interpret changes of these data in diagnostic and predictive aspects of different types of pathology.

Specifically:

Know:

1. To distinguish from pathological positions symptomatic changes of leucocyte composition of peripheral blood at system forms of its pathology.
2. To give the characteristic to forms of changes of leucocyte composition of blood of symptomatic character, to explain their causes, the mechanism of development and to give classification.
3. To estimate data of changes of quantity of leukocytes and a leucocyte formula at various pathological processes and diseases, to be able to prove their diagnostic and predictive value.

Be able to:

1. To know the main stages of a leucopoesis and to have idea about normal leukocyte formula.
2. To make calculation of quantity of leucocytes and to know limits of normal fluctuations of this indicator in blood at the human.
3. To prepare and paint blood dab according to Romanovsky-Giemsa, nobility and distinguish (at its microscopy) different forms of leukocytes, to be able to count them and to count absolute number of each type of leucocytes.

Practical experience:

1. To prepare and color a blood smear according to Romanovsky-Giems, to know and recognize (with its microscopy) different forms of leukocytes, to be able to count them and calculate the absolute number of each type of leukocytes.

2. Count the number of leukocytes and know the limits of normal fluctuations of this indicator in the blood of a person.

3. Distinguish symptomatic changes in the leukocyte composition of peripheral blood from general pathological conditions in systemic forms of its pathology.

4. To characterize the forms of symptomatic changes in the leukocyte composition of blood, to explain their causes, the mechanism of development, and to give a classification.

5. To evaluate data on changes in the number of leukocytes and leukocyte formula in various pathological processes and diseases, to be able to justify their diagnostic and prognostic value.

Technological map of students work on the topic "Leukocytosis. Leukopenia"

№	Stage lesson	Academic time, min	Educational guide		Place holding a class
			Educational tools	Equipment	
1	Determination of the initial level of knowledge	10	Written answer to test tasks	Test tasks	Study room
2	Analysis of theoretical material	35	Analysis of theoretical material based on control questions of the topic, situational tasks, tasks of KROK-1	Topic control questions, KROK-1 tasks, situational tasks	
3	Practical part (conduct experiment)	30	Introduction and preparation for setting up the experiment. Setting up the experiment. Discussion of the results of the experiment and formulation of conclusions	White rat, micropipette (capilary tube) from haemometer, test-tubes, injection needles, pipettes, counting came-ras, 5 % solution of acetic acid, blood smears of sick people with different types of leukocytosis, microscopes with immersion objectives, immersion oil	
4	Determination of the final level of knowledge and skills. Summarizing the results	15	Determination of the final level of knowledge and skills. Summarizing the results	KROK-1 tasks, situational tasks	

The graphological structure of the topic "Leukocytosis. Leukopenia" is attached.

Material and methodological support of the topic "Leukocytosis. Leukopenia":

1. Lectures;
2. Methodical instructions for teachers;

3. Methodical instructions for students;
4. Set of test tasks to determine the basic level of knowledge;
5. Set of situational tasks to determine the final level of knowledge;
6. Set of KROK-1 tasks;
7. Set of schemes and tables (presentation);
8. Set of forms with a clinical blood test
9. Video films;
10. For the experiment (experimental animals – white rat, micropipette (capilary tube) from haemometer, test-tubes, injection needles, pipettes, counting came-ras, 5% solution of acetic acid, blood smears of sick people with different types of leukocytosis, microscopes with immersion objectives, immersion oil.).

Lesson content:

1. Stages of leucopoesis of pathology .
2. Absolute leukocytosis.
3. Absolute leucopenia
4. Clinical manifestations of leucopenia.
5. Clinical manifestations of leukocytosis.
6. Causes and mechanisms of agranulocytosis.

Setting up the experiment. Discussion the results and formulation the conclusions

- **Differential Leukocyte count (Leukocyte formula)**

From your teacher, collect a numbered blood smear with the amount of leukocytes in a unit volume indicated. Determine the differential leucocyte count and crosscheck with the standard figures Determine the kind of leukocytosis. Write the results in the table below:

Total of leucocytes	Basophils	Eosinophils	Neutrophils				Lymphocytes	Monocytes
			Mielocytes	Young	Band neutrophils	Segment neutrophils		

- **Count the number of white blood cells during experimental postradiational leukopenia.**

Take two rats, one of which five days Before the experiment was subjected to general X-ray radiation in doses of 600 roentgen. Study the external signs of radiation syndrome, noting haemorrhage of the eyes, nose, changes in the fur, diarrhea, weight loss, change in general state (languitidy). Count the number of white blood cells of the experimental and intact rats. Place the rat in a camera, lower the tail in water with a temperature of 38 ^{o}C, there by causing hyperemy. Wipe the tail with cotton balls, place. Pierce one of the tail veins with an in-jection needle. Wipe away the first drop of blood, the second collect to the marked point in a measuring pipette from a haemometer. Blow the blood from

the pipette to the bottom of the test-tube, where earlier was poured of 0,38 ml of a 3 % solution of acetic acid, dyed in hencianviolet. Rinse the pipette three times. Shake the mixture energetically for 3 minutes, transfer to the counting camera with the pipette for distelled water from a haemometer. Count the white blood cells in 100 big squares of Gorev's camera. The amount of white blood cells in 1 mkl of blood. Calculate by the formula:

$$X = \frac{A \times 4000 \times B}{C},$$

where

X – unknown number of white blood cells

A – sum of white blood cells, counted in 100 big squares

B – sum of counted small squares (1600)

C – dilution of the blood (20 times)

The volume of the small square = 1/4000 mcl (microlitre), therefore to transfer to 1 mkl, the formula has a multiplier of 4000. The amount of white blood cells calculate in 1l. Compare the amount of white blood cells of the radiated rat and the control rat.

- **Discussion of the results of the experiment**

During the discussion, indicate the reasons that cause the corresponding shifts in the leukocyte formula. Point out the features of the leukocyte shift in acute infectious diseases.

The research carried out in this lesson shows that ionizing treatment affects cells more strongly, the more their ability to reproduce, than a long path of the mitotic process, than cells are younger and less differentiated. In the discussion, pay attention to the appearance of immature forms of leukocytes in the peripheral blood.

The action of radiation is accompanied, in particular, by damage to hematopoietic organs, primarily bone marrow, as a result of which a hypoplastic state of hematopoiesis develops.

- **Formulation of conclusions based on the experiment**

In the set of smears there are drugs with neutrophilia, eosinophilia, lymphocytosis and indicate the reasons that cause the corresponding shifts in the leukocyte formula. Point out the features of the leukocyte shift in acute infectious diseases.

In the irradiated rat, the number of leukocytes decreases sharply compared to the control. Violation of hematopoiesis appears soon after exposure. The appearance of immature leukocytes in the peripheral blood, which are found in places of hematopoiesis, indicates a deep qualitative violation of the process of hematopoiesis, leukemia is an independent disease characterized by systemic damage to hematopoietic organs.

Terminology:

- Leukocytosis
- Leukopenia
- Leucopoesis
- Agranulocytosis

Tasks for independent work on the topic "Leukocytosis. Leukopenia."

The student is offered to investigate the results of a clinical blood analysis of a patient with a disorder in the blood system. It is necessary to determine the signs and type of violation. Be able to explain the mechanism of occurrence. Analysis of errors with an explanation of the correct answers

List of questions and works to be studied:

1. Leukocytosis. Emergence mechanisms, classification.
2. Physiological leukocytosis.
3. Pathological leukocytosis. Causes and mechanisms of development.
4. Jet and redistributive leukocytosis. Causes and mechanisms of development.
5. Types of a leukocytosis depending on a type of leucocytes.
6. Leucocyte formula. Relative and absolute leukocytosis and leucopenia.
7. Nuclear shift of neutrophile leucocytes. Types, causes, their predictive value.
8. Leucemoid reactions, their causes and hematologic characteristic.
9. Leucopenia, principles of classification.
10. Pathogenesis of the main clinical manifestations of leucopenia.
11. Leucopenia owing to reduction of time of stay of leukocytes in peripheral blood and redistributive leucopenia. Causes and mechanisms of development.
12. Leucopenia due to violation of receipt of leukocytes from red bone marrow in blood. Causes, development mechanisms.
13. Agranulocytosis. Causes and mechanisms of development.

List of practical skills that must be mastered:

1. Explain the causes of leukopoiesis disorders.
2. Explain the concepts of "physiological", "pathological", "reactive" and "redistributive" leukocytosis.
3. Determine changes in the leukocyte formula, relative and absolute leukocytosis and leukopenia.
4. Analyze the results of clinical blood analysis (standards and criteria disorders of leukocytes and their individual types).
5. Define leukocytosis and leukopenia and their separate types in blood analysis, explain possible causes and mechanisms of their development.
6. To be able to determine the nuclear shift in a blood test for various pathologies.
7. Determine the signs of agranulocytosis, explain its causes and mechanism development

Situational tasks KROK-1 to determine the final level of knowledge

1. Two hours after an exam a student had a blood count done and it was revealed that he had leukocytosis without significant leukogram modifications. What is the most probable mechanism of leukocytosis development?

A. Redistribution of leukocytes in the organism.
B. Leukopoiesis intensification.
C. Deceleration of leukocyte lysis.
D. Deceleration of leukocyte migration to the tissues.
E. Leukopoiesis intensification and deceleration of leukocyte lysis.

2. Examination of a patient admitted to the surgical department with symptoms of acute appendicitis revealed the following changes in the white blood cells: the total count of leukocytes is 16×10^9/l. Leukocyte formula: basophils – 0, eosinophils – 2 %, juvenile forms – 2 %, stabnuclear – 8 %, segmentonuclear – 59 %, lymphocytes – 25 %, monocytes – 4 %. The described changes can be classified as:

A. Neutrophilia with right shift.
B. Neutrophilia with degenerative left shift.
C. Neutrophilic leukemoid reaction.
D. Neutrophilia with hyperregenerative left shift.
E. Neutrophilia with regenerative left shift.

3. 24 hours after appendectomy blood of a patient presents neutrophilic leukocytosis with regenerative shift. What is the most probable mechanism of leukocytosis development?

A. Redistribution of leukocytes in the organism.
B. Amplification of leucopoiesis.
C. Decelerated leukocyte destruction.
D. Decelerated emigration of leukocytes to the tissues.
E. Amplification of leukopoiesis and decelerated emigration of leukocytes to the tissues.

4. After an attack of bronchial asthma a patient had his peripheral blood teste d. What changes can be expected?

A. Eosinophilia. *C. Erythrocytosis.* *E. Leukopenia.*
B. Lymphocytosis. *D. Thrombocytopenia.*

5. A 3-year-old child had eaten some strawberries. SeSon he developed a rash and itching. What was found in the child's leukogram?

A. Hypolymphemia. *C. Eosinophilia.* *E. Neutrophilic leukocytosis.*
B. Lymphocytosis. *D. Monocytosis.*

6. A 5 year old child is ill with measles. Blood analysis revealed increase of total number of leukocytes up to 13×10^9/l. Leukogram: basophils – 0, eosinophils – 1, myelocytes – 0, juvenile neutrophils – 0, band neutrophils – 2, segmented neutrophils – 41, lymphocytes – 28, monocytes – 28. Name this phenomenon:

A. Agranulocytosis. *C. Monocytosis.* *E. Neutropenia.*
B. Lymphocytosis. *D. Eosinopenia.*

7. As a result of a road accident a 26-year-old man is in the torpid phase of shock. Blood count: leukocytes – $3,2\times10^9$/l. What is the leading mechanism of leukopenia development?

A. Faulty release of mature leukocytes from the bone marrow into the blood.
B. Leukocyte destruction in the hematopietic organs.
C. Leukopoiesis inhibition.
D. Leukocyte redistribution in the bloodstream.
E. Increased excretion of the leukocytes from the organism.

8. A 26-year-old man is in the torpid shock phase as a result of a car accident. In blood: $3,2\times10^9$/l. What is the leading mechanism of leukopenia development?

A. Disturbed going out of mature leukocytes from the marrow into the blood.
B. Leukopoiesis inhibition.
C. Lysis of leukocytes in the blood-forming organs.
D. Redistribution of leukocytes in bloodstream.
E. Intensified elimination of leukocytes from the organism.

9. Parents of a 3-year-old child have been giving him antibiotics with purpose of preventing enteric infections for a long time. A month later the child's condition changed for the worse. Blood examination revealed apparent leukopenia and granulocytopenia. What is the most probable mechanism of blood changes?

A. Myelotoxic. *C. Redistributive.* *E. Hemolytic.*
B. Autoimmune. *D. Age-specific.*

10. A patient with atrophic gastritis developed a vitamin B12 deficiency. What change in the leukocyte formula is the most typical for hypovitaminosis B12?

A. Hyperregenerative shift to the left.
B. Degenerative shift to the left.
C. Nuclear shift to the right.
D. Regenerative-degenerative nuclear shift to the left.
E. Regenerative nuclear shift to the left.

Standards of correct answers to the task KROK-1

1	2	3	4	5	6	7	8	9	10
A	*E*	*B*	*A*	*C*	*C*	*D*	*D*	*A*	*C*

Recommendations for registration of work results

1. Written answer to test tasks (initial level of knowledge).
2. The results of the experiment are drawn up in the form of an experiment protocol with the determination of relevant conclusions.
3. Protocol for the study of the results of the patient's clinical blood analysis.
4. Protocol for solving situational tasks with an explanation of the correct answers.

LITERATURE

Main

1. Pathophysiology : textbook / N. V. Krishtal, V. A. Mikhnev, N. N. Zayko [et al.] ; ed.: N. V. Krishtal, V. A. Mikhnev. 2nd ed., corrected. Kyiv : AUS Medicine Publishing, 2018. 656 p.
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2. Essentials of Pathology / Ya. Bodnar, A. Romanyuk, V. Voloshyn [et al.]. Kharkiv : Planeta-Print LTD, 2020. 219 p.
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Topic № 4. Leukemia

Number of hours: 2 academic hours.

Relevance of the topic: Leukemia (leukosis) are a group of malignant tumor diseases, in which the processes of proliferation and differentiation of hematopoietic cells are disturbed, causing progressive infiltration of the bone marrow with displacement of normal hematopoietic cells. Leukemia refers to diseases of the blood system of a tumor nature and is one of the most dangerous forms of human pathology. In solving the issues of etiology, pathogenesis, diagnosis and treatment of leukemia, they have a high mortality rate, especially in childhood and younger age. Therefore, every doctor needs to know the symptoms of these diseases, the mechanisms of their development, and the features of the hematological condition in their various forms.

Purpose of the lesson:

General – to be able to give the characteristic to leukemia as kinds of hemoblastoses, to classify them, to explain blood formation violation mechanisms, quantitative and qualitative changes in composition of peripheral blood, to give the characteristic of leukemia at different forms of this disease. To be able to interpret the modern ideas of an etiology and pathogenesis of leukemia.

Specifically:

Know:

1. To characterize a leukemia as a system disease of the hematogenic system.
2. To give modern classification of leukemia by the hystogenetic principle, a course and a state of peripheral blood.
3. To characterize the main techniques of the hematologic researches which are used at diagnosis of leukemia.
4. To decide the possibility of a leucosis by hematograms data.
5. To understand a state of change of peripheral blood at a leukemia and to distinguish different forms of leukemia.
6. To call the main cytochemical indicators used for differential diagnosis of leukosis and to give by them the characteristic of the main forms of leukemia.
7. To explain mechanisms of changes of erythrocyte and platelet composition of blood at leukemia.
8. To explain mechanisms of violations of functions of organs and systems at leukemia.

Be able to:

1. To characterize the main stages of a haemopoesis.
2. To distinguish at blood dab microscopy forming elements of granulocyte, lymphocyte, monocyte and erythrocyte ranks.

Practical experience:

1. To characterize the violation of the qualitative and quantitative composition of "white blood" according to the leukogram of a patient with chronic myeloid leukemia.

2. Demonstrate immature granulocytes in a peripheral blood smear of a patient with chronic myeloid leukemia, interpret their presence in the blood.

3. To characterize the violation of the qualitative and quantitative composition of "white blood" in the leukogram of a patient with chronic lymphocytic leukemia.

4. To identify degeneratively changed lymphocytes in the peripheral blood smear of a patient with chronic lymphocytic leukemia.

5. Based on the study of the hemogram, characterize changes in the qualitative and quantitative composition of blood in patients with acute leukosis.

Technological map of students' work on the topic "Leukemia"

№	Stage lesson	Academic time, min	Educational guide		Place holding a class
			Educational tools	Equipment	
1	Determination of the initial level of knowledge	10	Written answer to test tasks	Test tasks	Study room
2	Analysis of theoretical material	35	Analysis of theoretical material based on control questions of the topic, situational tasks, tasks of KROK-1	Topic control questions, KROK-1 tasks, situational tasks	
3	Practical part (conduct experiment)	30	Introduction and preparation for setting up the experiment. Setting up the experiment. Discussion of the results of the experiment and formulation of conclusions	Blood smears of the sick, microscopes with immersion objectives, immersion oil.	
4	Determination of the final level of knowledge and skills. Summarizing the results	15	Determination of the final level of knowledge and skills. Summarizing the results	KROK-1 tasks, situational tasks	

The graphological structure of the topic "Leukemia" is attached.

Material and methodological support of the topic "Leukemia":

1. Lectures;
2. Methodical instructions for teachers;
3. Methodical instructions for students;
4. Set of test tasks to determine the basic level of knowledge;
5. Set of situational tasks to determine the final level of knowledge;
6. Set of KROK-1 tasks;
7. Set of schemes and tables (presentation);
8. Set of forms with a clinical blood test
9. Video films;
10. For the experiment (Blood smears of the sick, microscopes with immersion objectives, immersion oil.).

Lesson content:

1. Stages of leucopoesis
2. Leukemoid reaction
3. Acute lymphocytic leukemia.
4. Chronic lymphocytic leukemia.

5. Acute myelogenous leukemia.
6. Chronic myelogenous leukemia.

Setting up the experiment. Discussion the results and formulation the conclusions

- **Microscopy of preparations of blood during aqute myeloid (myeloblastic) leukosis**

Count the different forms of leukocytes. Pay attention to the absence of transition forms between young (blastic) and matured neutrophils.

- **Microscopy of preparations of blood during chronic myeloid (myelocytic) leukosis.**

Count the different forms of leukocytes. Pay attention that during clinical form leucosis in blood smear at great quantity meats the cells of mieloid types at all stadies of development.

- **Microscopy of preparations of blood during chronic lymphoid leukosis**

While counting different forms of leukocytes, pay attention to the predominance of young lymphocyte forms (prolymphocytes, lymphoblasts).

- **Discussion of the results of the experiment**

Examine the blood smear and pay attention to the impossibility of differentiation of leukocytes according to morphological features. Note the absence of transitional forms between immature cells (blast forms) and mature segmented neutrophils. Pay attention to the fact that with this myeloid form of leukosis in a blood smear, there are a large number of myeloid cells at all stages of development.

- **Formulation of conclusions based on the experiment**

The appearance in the peripheral blood of immature leukocytes, which in physiological conditions are found in places of hematopoiesis, indicates a deep a qualitative violation of the process of hematopoiesis characteristic of leukosis. In contrast to pathological leukocytosis, which is a symptom of diseases (infections, intoxication, malignant neoplasms) leukemias are independent a disease characterized by a systemic lesion of the hematopoietic organs.

Terminology:

- Leukemic cells
- Myeloblasts
- Lymphoblasts
- Erythromyelosis
- Lymphogranulomatosis

Tasks for independent work on the topic "Leukemia"

The student is offered to investigate the results of a clinical blood analysis of a patient with a disorder in the blood system. It is necessary to determine the signs and type of violation. Be able to explain the mechanism of occurrence. Analysis of errors with an explanation of the correct answers**.**

List of questions and works to be studied:

1. Leukemia. Etiology and pathogenesis.
2. Modern classification of leukemia.
3. Acute leukemia. Clinical manifestations. Pathogenesis of the main clinical syndromes.
4. Chronic leukemia. Clinical manifestations.
5. Stages of an acute and chronic leukemia. Principles of diagnostics and treatment.
6. Changes in a hemogram and a myelogram in leukemia.

List of practical skills that must be mastered:

1. To characterize the violation of the qualitative and quantitative composition of "white blood" according to the leukogram of a patient with chronic myeloid leukemia.
2. Demonstrate immature granulocytes in a peripheral blood smear of a patient with chronic myeloid leukemia, interpret their presence in the blood.
3. To characterize the violation of the qualitative and quantitative composition of "white blood" in the leukogram of a patient with chronic lymphocytic leukemia.
4. To identify degeneratively changed lymphocytes in the peripheral blood smear of a patient with chronic lymphocytic leukemia.
5. Based on the study of the hemogram, characterize changes in the qualitative and quantitative composition of blood in patients with acute leukemia.

Situational tasks KROK-1 to determine the final level of knowledge

1. A 23 y.o. patient complains of weakness, temperature rise up to 38–40 °C. Objectively: liver and spleen are enlarged. Hemogram: Hb – 100 g/l, erythrocytes – $2{,}9\times10^{12}$/l, leukocytes– $4{,}4\times10^{9}$/l, thrombocytes – 48×10^{9}/l, segmentonuclear neutrophils – 17 %, lymphocytes – 15 %, blast cells – 68 %. All cytochemical reactions are negative. Make a hematological conclusion:

A. Undifferentiated leukosis.
B. Chronic myeloleukosis.
C. Acute myeloblastic leukosis.
D. Acute lymphoblastic leukosis.
E. Acute erythromyelosis.

2. The total number of leukocytes in patient's blood is 90×10^{9}/1. Leukogram: eosinophils – 0 %, basophils – 0 %, juvenile – 0 %, stab neutrophils – 2 %, segmentonuclear cells – 20 %, lymphoblasts – 1 %, prolymphocytes – 2 %, lymphocytes – 70 %, monocytes – 5 %, Botkin-Gumprecht cells. Clinical examination revealed enlarged cervical and submandibular lymph nodes. Such clinical presentations are typical for the following pathology:

A. Chronic myeloleukosis.
B. Acute lympholeukosis.
C. Chronic lympholeukosis.
D. Lymphogranulomatosis.
E. Infectious mononucleosis.

3. A patient suffering from chronic myeloleukemia has got the following symptoms of anemia: decreased number of erythrocytes and low haemoglobin

concentration, oxyphilic and polychromatophilic normocytes, microcytes. What is the leading pathogenetic mechanism of anemia development?

A. Substitution of haemoblast.
B. Reduced synthesis of erythropoietin.
C. Chronic haemorrhage.
D. Intravascular hemolysis of erythrocytes.
E. Deficiency of vitamin B_{12}.

4. A 39-year-old patient underwent hematologic tests. The following results were obtained: RBC – $2{,}8\times10^{12}$/L, Hb – 80 g/L, color index – 0,85, reticulocytes – 0,1 %, platelets – 160×10^{9}/L, WBC – 60×10^{9}/L. Basophils – 2, eosinophils – 8, promyelocytes – 5, myelocytes – 5, immature neutrophils – 16, stab neutrophils – 20, segmented neutrophils – 34, lymphocytes – 5, monocytes – 5. What form of blood pathology are these results indicative of?

A. Chronic myeloid leukemia.
B. Undifferentiated leukemia.
C. Hemolytic anemia.
D. Acute myeloid leukemia.
E. Hypoplastic anemia.

5. A patient who had a tooth removed due to acute purulent periostitis had long-term bleeding from the socket that did not stop with conventional methods. In the blood: erythrocytes – 2.9×10^{12}/l, Hb – 90 g/l; color index – 0.9; clot. – 60×10^{9}/l; leukocytes – 52×10^{9}/l.; basophils – 0, eosinophils – 1 %, monocytes – 0, neutrophils: young – 0, Band nuclear – 2 %, segmentonuclear – 18 %, lymphocytes – 8 %, monocytes – 1 %, myeloblasts – 70 %. What blood disease does the patient have?

A. Undifferentiated leukemia.
B. Promyelocytic leukemia.
C. Chronic myeloid leukemia.
D. Erythromyelosis.
E. Acute myeloid leukemia.

6. During the examination, leukocytosis, lymphocytosis, Botkin-Gumprecht cells against the background of anemia were found in the patient's blood. What disease should we think about?

A. Chronic lymphocytic leukemia.
B. Myeloma disease.
C. Acute myeloid leukemia.
D. Lymphogranulomatosis.
E. Infectious mononucleosis.

7. Hematologic study shows the following pattern: erythrocytes – $2{,}8\times10^{12}$/L, Hb – 80 g/L, color index – 0.85, reticulocytes – 0.1 %, platelets – 160 thousand per microliter, leukocytes – 60×10^{9}/L. Basocytes – 2 %, eosinophils – 8 %, promyelocytes – 5 %, myelocytes – 5 %, juvenile – 16 %, stab neutrophils – 20 %, segmented neutrophils – 34 %, lymphocytes – 5 %, monocytes – 5 %. This clinical presentation indicated the following blood pathology:

A. Undifferentiated leukemia.
B. Chronic myeloleukemia.
C. Acute myeloleukemia.
D. Hemolytic anemia.
E. Hypoplastic anemia.

8. In the patient's leukogram: leukocytes – 14×10^9/l; myeloblasts – 71 %, promyelocytes, myelocytes, metamyelocytes – 0 %, rod neutrophils – 6 %, segmentonuclear – 13 %; lymphocytes – 7 %, monocytes – 3 %. What pathology of the blood in sick?

A. Myeloblastic leukemia.
B. Neutrophil leukocytosis.
C. Chronic myeloleukosis.
D. Lymphoblastic leukemia.
E. Chronic lymphocytic leukemia.

9. Over the past year, the patient began to notice increased fatigue, general fatigue weakness Blood analysis: Er – 4.1×10^{12}/l, Hb – 119 g/l, CP – 0.87, leukocytes – 57×10^9/l, leukocyte formula: y–0, p–0, c–9%, e–0, b–0, lymphoblasts – 2 %, prolymphocytes – 5 %, lymphocytes – 81 %, m – 3 %, platelets – 160×10^9/l. In the smear: normochromia, large number of Botkin-Gumprecht shadows. What kind of pathology does the blood system indicate this hemogram?

A. Chronic myeloid leukemia.
B. Acute lymphoblastic leukemia.
C. Chronic lymphocytic leukemia.
D. Acute myeloblastic leukemia.
E. Chronic monoleukosis.

10. Patient P. has the following changes in peripheral blood: Er. 3.0×1/l, Hemoglobin – 80 g/l, Leukocytes – 21×1/l. Leukocyte formula: basophils – 0 %, eosinophils – 0 %, myeloblasts – 54 %, promyelocytes – 1 %; myelocytes – 0 %, metamyelocytes – 0 %, stab – 1 %, segmented – 28 %, lymphocytes – 13 %, monocytes – 3 %. Determine the most likely pathology according to the given description blood pictures:

A. Acute myeloid leukemia.
B. Chronic myeloid leukemia.
C. Erythromyelosis.
D. Leukemoid reaction.
E. Undefined leukemia.

Standards of correct answers to the task KROK-1

1	2	3	4	5	6	7	8	9	10
A	*C*	*A*	*A*	*E*	*A*	*B*	*A*	*C*	*A*

Recommendations for registration of work results

1. Written answer to test tasks (initial level of knowledge).
2. The results of the experiment are drawn up in the form of an experiment protocol with the determination of relevant conclusions.
3. Protocol for the study of the results of the patient's clinical blood analysis.
4. Protocol for solving situational tasks with an explanation of the correct answers.

LITERATURE

Main

1. Pathophysiology : textbook / N. V. Krishtal, V. A. Mikhnev, N. N. Zayko [et al.] ; ed.: N. V. Krishtal, V. A. Mikhnev. 2nd ed., corrected. Kyiv : AUS Medicine Publishing, 2018. 656 p.
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Secondary

1. Banasic, J. L. Pathophysiology : textbook / J. L. Banasic, L-E. C. Copstead. - 6th ed. St. Louis : Elsevier, 2019. XXII, 1177 p.
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Topic № 5. Disturbances in hemostasis

Number of hours: 2 academic hours.

Relevance of the topic: The anticoagulation system contains natural anticoagulants that block the clotting mechanisms at each stage. Their greatest activity is noted with excessive activity of clotting factors (coagulation). Hemostasis is a complex of factors and mechanisms aimed at preserving blood in the bloodstream, maintaining its physicochemical properties, which prevent bleeding and condition the restoration of blood flow in the event of occlusion of blood vessels with a blood clot. Hemostasis determines the stopping of bleeding or the restoration of blood flow. A complex of factors and mechanisms of hemostasis by closely interrelated coagulation and anticoagulation systems. Thus the range of manifestations of coagulapatias is extremely wide, from the hidden latent forms to life-threatening manifestations. Despite the fact that in most part of change of different indicators of physical and chemical properties of blood have nonspecific character, date about some of them, along with data of other laboratory researches, help a doctor make a diagnose, and also at judgment about efficiency of cure.

Purpose of the lesson:

General - to be able to characterize a condition of the coagulation system of blood by coagulogram indicators. To be able to interpret changes of ESR and osmotic resistance of erythrocytes at different pathological processes and diseases as the data of additional methods at diagnosis of diseases and criterion of efficiency of treatment.

Specifically:

Know:

1. To define the term "system of a hemostasis".
2. To characterize the mechanisms providing a stop of bleeding and restoration of integrity of the vascular course.
3. To define changes of ESR and osmotic resistance of erythrocytes.
4. To connect these changes with possible violations of physical and chemical properties of blood at pathology.
5. To explain mechanisms of change of ESR and osmotic resistance of erythrocytes at different pathological processes and diseases.

Be able to:

1. To characterize coagulation mechanisms.
2. To explain participation of a liver in coagulation mechanisms.
3. To characterize the main physical and chemical properties of blood and erythrocytes, to explain, than they are caused.
4. What shows the ESR indicator, what factors it depends on?
5. To determine value of ESR and to know normal borders of its fluctuations.

6. To characterize the cause of the osmotic and oncotic pressure of plasma of blood.

7. Know that represent iso-, hypo – and hypertensive solutions to characterize influence of these solutions on a cell.

8. To give definitions of the terms "maximum" and "minimum" resistance of erythrocytes, to specify normal borders of fluctuations of these sizes.

9. To explain, what properties of erythrocytes and plasma the size of osmotic resistance depends on.

10. To perform and consider reaction of determination of osmotic resistance of erythrocytes.

Practical experience:

1. Based on the study of the patient's hemogram, characterize changes in the number of thrombocytes and state the cause of this disorder

2. Based on the study of the patient's hemogram, characterize changes in ESR and state the cause of this disorder

3. To define changes of ESR and osmotic resistance of erythrocytes.

4. To perform and consider reaction of determination of osmotic resistance of erythrocytes.

Technological map of students' work on the topic "Disturbances in hemostasis"

№	Stage lesson	Academic time, min	Educational guide		Place holding a class
			Educational tools	Equipment	
1	Determination of the initial level of knowledge	10.	Written answer to test tasks	Test tasks	Study room
2	Analysis of theoretical material	35	Analysis of theoretical material based on control questions of the topic, situational tasks, tasks of KROK-1	Topic control questions, KROK-1 tasks, situational tasks	
3	Practical part (conduct experiment)	30	Introduction and preparation for setting up the experiment. Setting up the experiment. Discussion of the results of the experiment and formulation of conclusions	Rabbit, sets of hypotonic solutions of table salt with different concentrations (from 0,60–0,32 %), 1 ml. pipettes, micropipettes (capillary tubes), injection needles, centrifuge, Panchenkov's apparatus, porcelain (china) crucibles, injection needles	
4	Determination of the final level of knowledge and skills. Summarizing the results	15	Determination of the final level of knowledge and skills. Summarizing the results	KROK-1 tasks, situational tasks	

The graphological structure of the topic "Disturbances in hemostasis" is attached.

Material and methodological support of the topic "Disturbances in hemostasis":

1. Lectures;
2. Methodical instructions for teachers;
3. Methodical instructions for students;
4. Set of test tasks to determine the basic level of knowledge;
5. Set of situational tasks to determine the final level of knowledge;
6. Set of KROK-1 tasks;
7. Set of schemes and tables (presentation);
8. Set of forms with a clinical blood test
9. Video films;
10. For the experiment (experimental animals – rabbit, sets of hypotonic solutions of table salt with different concentrations (from 0,60–0,32 %), 1 ml. pipettes, micropipettes (capillary tubes), injection needles, centrifuge, Panchenkov's apparatus, porcelain (china) crucibles, injection needles).

Lesson content:

1. Mechanisms of coagulopathias.
2. Thrombocytic factors.
3. Activation of fibrinolytic system.
4. Disseminated intravascular clotting.
5. Mechanisms of hemorrhage disorders.
6. Increased vascular permeabili.
7. Hemorrhagic diatheses.
8. Willebrand's disease.

Setting up the experiment. Discussion of results and formulation of conclusions

- **Determination of the osmotic resistance of erythrocytes in experimental haemolitic anemia.**

Take two rabbits, in one of which experimentally was caused haemolytic phenylhydrase anemia. With the help of a pipette, transfer different concentrations of 1 ml. solutions of table salt. Carry out the transfer, beginning with lower concentrations and ending with the highest. Then add 0,02 ml. of blood taken from the marginal vein of the ear into the test-tubes. Addition of the blood should begin from the highest concentration to the lowest. The capillary for taking blood should be rinsed twice with the same solution to ensure complete removal of blood. In half an hour all the test-tubes should be centrifuged at a speed of 3000 RPM/min for 5 minutes. Estimate the degree of haemolysis. Note the maximum (complete haemolysis) and the minimum (traces of haemolysis) osmotic resistance of erythrocytes in the blood of the control rabbit and experimental rabbit.

- **Studing of reticylocytes in experimental hemolytic anemia**

Rinse the capillary tube from Panchenkov's apparatus with 5% solution of sodium citrate. Then fill the tube with the same solution to the point "P" (50 mm) and blow into the crucible. From the marginal ear vein take blood twice till point "K" (100 mm), blow into the crucible and mix throughly. By doing this the blood becomes stabilized . Fill up the capillary tube with the stabilized blood till point "O" and place it strictly upright in Panchenkov's apparatus. In an hour's time count in ml the height of the formed column of plasma. Compare the ESR of the anemic and control rabbit.

- **Discussion of the results of the experiment**

A rabbit with hemolytic anemia has a decrease in the osmotic resistance of erythrocytes (maximum – up to 0.48, minimum– up to 0.60), compared to a control rabbit (maximum 0.32–0.34, minimum – 0.44–0.46).

The rate of sedimentation of erythrocytes in a rabbit with hemolytic anemia is significantly accelerated (10–12 mm/h), while in a control rabbit – 1–2 mm/h.

- **Formulation of conclusions based on the experiment**

Indicate that the osmotic resistance of erythrocytes depends on the degree of their maturity, shape and on changes in plasma composition; young erythrocytes are more stable than mature ones; erythrocytes with a spherical shape are less resistant (normal sphericity index – 0.27–0.23).

In experimental animals, a decrease in the osmotic resistance of erythrocytes is noted, which is explained by a violation of erythrocyte production.

The rate of sedimentation of erythrocytes depends on changes in the protein fractions of blood, changes in blood viscosity, changes in the number of erythrocytes, the difference in the specific weights of erythrocytes and plasma, etc. In this case, with hemolytic phenylhydrazine anemia, ESR is accelerated due to a change in blood viscosity, a decrease in the number of erythrocytes.

Terminology:

- Coagulopathias
- Hemorrhagic diatheses
- Hypoproteinemia
- Hyperproteinemia
- Dysproteinemia
- Paraproteinemia

Tasks for independent work on the topic "Disturbances in hemostasis".

The student is offered to investigate the results of a clinical blood analysis of a patient with a disorder in the blood system. It is necessary to determine the signs and type of violation. Be able to explain the mechanism of occurrence. Analysis of errors with an explanation of the correct answers

List of questions and works to be studied:

1. Hemostasis. The structurally functional components enabling the realization of mechanisms of a hemostasis.
2. Violations of vascular platelet hemostasis. Causes, pathogenesis. Clinical manifestations, their pathogenesis.
3. Main mechanisms and pathological manifestations of vascular platelet hemostasis (primary hemostasis).
4. Main mechanisms and pathological manifestations of a plasma hemostasis (secondary hemostasis).
5. Groups of hemorrhagic diseases. Criteria of diagnostics of mechanisms of violation of a hemostasis.
6. Bleeding types, their main manifestations.
7. Coagulopathy due to the excess of anticoagulants and acute stimulation of a fibrinolisys.
8. Hereditary coagulopathy. Pathogenesis, manifestations. Hemophilia. Clinical and laboratory criteria.
9. Thrombocytopenia. Etiology, pathogenesis.
10. Clinical and laboratory criteria idiopathic trombocytepenic purple.
11. Thrombocytopathy. Mechanism of violations of adhesion, aggregation of platelets, releases of platelets granules.
12. Vazopathy: types, Causes, development mechanisms, pathogenesis of the main clinical manifestations.
13. Hyper coagulation. Thtombocytic syndrome (trombophylia).
14. Syndrome of a disseminated intravascular coagulation (DIC-syndrome).
15. Osmotic and oncotic pressure of blood. Causes of disorders.
16. Osmotic resistance of erythrocytes. Causes of disorders.
17. Speed of subsidence of erythrocytes. Causes of disorders.
18. Violations of proteinaceous composition of blood, their causes.

List of practical skills that must be mastered:

1. Based on the study of the patient's hemogram, characterize changes in the number of thrombocytes and state the cause of this disorder
2. Based on the study of the patient's hemogram, characterize changes in ESR and state the cause of this disorder
3. To define changes of ESR and osmotic resistance of erythrocytes.
4. To perform and consider reaction of determination of osmotic resistance of erythrocytes.

Situational tasks KROK-1 to determine the final level of knowledge

1. A patient with tissue trauma was taken a blood sample for the determination of blood clotting parameters. Specify the right sequence of extrinsic pathway activation.

A. III–IV–Xa. *C. IV–VIII: TF–Xa.* *E. III–VIII: TF–Xa.*
B. III–VIIa–Xa. *D. IV–VIIa–Xa.*

2. A patient is diagnosed with hereditary coagulopathy that is characterized by factor VIII deficiency. Specify the phase of blood clotting during which coagulation will be disrupted in the given case:

A. Thrombin formation. *D. Clot retraction.*
B. Thromboplastin formation. *E. –.*
***C.** Fibrin formation.*

3. A 3-year-old boy with pronounced hemorrhagic syndrome doesn't have antihemophilic globulin A (factor VIII) in the blood plasma. Hemostasis has been impaired at the following stage:

A. External mechanism of prothrombinase activation.
B. Conversion of prothrombin to thrombin.
C. Internal mechanism of prothrombinase activation.
D. Conversion of fibrinogen to fibrin.
E. Blood clot retraction.

4. A 12-year-old patient has been admitted 3 a hospital for hemarthrosis of the knee Dint. From early childhood he suffers from frequent bleedings. Diagnose the boy's disease:

A. B_{12} (folic acid)-deficiency anemia. *D. Thrombocytopenic purpura.*
B. Hemolytic anemia. *E. Hemorrhagic vasculitis.*
C. Hemophilia.

5. Tooth extraction in a patient with chronic persistent hepatitis was complicated by a prolonged bleeding. What is the cause of hemorrhagic syndrome?

A. Decreased production of thrombin.
B. Decreased production of fibrin.
C. Increased synthesis of fibrinogen.
D. Increased fibrinolysis.
E. Increased production of thromboplastin.

6. A patient underwent a surgery for excision of a cyst on pancreas. After this he developed haemorrhagic syndrome with apparent disorder of blood coagulation. Development of this complication can be explained by:

A. Insufficient fibrin production. *D. Activation of Christmas factor.*
B. Reduced number of thrombocytes. *E. Activation of fibrinolytic system.*
C. Activation of anticoagulation system.

7. After a tourniquet application a patient was found to have petechial haemorrhages. The reason for it is the dysfunction of the following cells:

A. Neutrophils. *C. Monocytes.* *E. Lymphocytes.*
B. Platelets. *D. Eosinophils.*

8. A 43-year-old patient has thrombopenia, reduction of fibrinogen, products of degradation of fibrin presented in the blood, petechial haemorrhage along with septic shock. What is the most likely cause of the changes?

A. Autoimmune thrombocytopenia. *D. Disorder of thrombocytes production.*
B. DIC-syndrome. *E. Exogenous intoxication.*
C. Haemorrhagic diathesis.

9. A patient was ill with burn disease that was complicated by DIC syndrome. What stage of DIC syndrome can be suspected if it is known that the patient's blood coagulates in less than 3 minutes?

A. Hypercoagulation. *C. Hypocoagulation.* *E. Terminal.*
B. Transition phase. *D. Fibrinolysis.*

10. A patient, who had been working hard under conditions of elevated temperature of the environment, has now a changed quantity of blood plasma proteins. What phenomenon is the case?

A. Absolute hyperproteinemia. *D. Paraproteinemia.*
B. Absolute hypoproteinemia. *E. Relative hyperproteinemia.*
C. Dysproteinemia.

Standards of correct answers to the task КРОК-1

1	2	3	4	5	6	7	8	9	10
B	*B*	*C*	*C*	*A*	*E*	*B*	*B*	*A*	*E*

Recommendations for registration of work results

1. Written answer to test tasks (initial level of knowledge).
2. The results of the experiment are drawn up in the form of an experiment protocol with the determination of relevant conclusions.
3. Protocol for the study of the results of the patient's clinical blood analysis.
4. Protocol for solving situational tasks with an explanation of the correct answers.

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SUBSTANTIAL MODULE 5.
Pathophysiology of systemic circulation, heart, blood vessels. The pathophysiology of external respiration

Topic № 6. Pathophysiology of the systemic blood circulation. Insufficiency blood circulation. Cardiac insufficiency

Number of hours: 2 academic hours.

Relevance of the topic. One of the main causes leading to disability and even death of patients suffering from diseases of the cardiovascular system is heart failure, in which the heart cannot provide tissues with enough blood for normal metabolism and leads to an increase in systemic venous pressure, plethora (stagnation) in peripheral organs, hypoxia and severe systemic damage. In this regard, the study of the etiology and pathogenesis of the disease In this regard, the study of the etiology and pathogenesis of the disease is necessary for the future doctor, not only to understand the strategy of patient management but also to choose rational and effective approaches to the treatment of each patient.

Purpose of the lesson:

General – to be able to characterize heart failure, and explain the main causes and mechanisms of development.

Specifically:

Know:

1. The concept of heart failure. Reasons, types.
2. Compensatory and adaptive mechanisms of heart failure. Violation of hemodynamics.
3. Clinical manifestations of heart failure.
4. Tonogenic and myogenic dilatation of the heart. Mechanisms of long-term adaptation of the heart to overloads. Myocardial hypertrophy.

Be able to:

1. To reveal the essence of the concept of "heart failure".
2. Classify the causes and mechanisms of heart failure
3. Identify the main manifestations of heart failure.
4. Explain the mechanisms of their occurrence and development.Graph of the logical structure of the topic "Pathophysiology of the circulatory system. Insufficiency of blood circulation. Heart failure" – added.

Technological map of student work topics «Pathophysiology of the systemic blood circulation. Insufficiency blood circulation. Cardiac insufficiency»

№	Stage lesson	Academic time, min	Educational guide		Place holding a class
			Educational tools	Equipment	
1	Determination of the initial level of knowledge	10	Written response to test tasks	Test tasks.	Learning room

№	Stage lesson	Academic time, min	Educational guide		Place holding a class
			Educational tools	Equipment	
2	Analysis of theoretical material	45	Analysis of the theoretical material is carried out on the basis of control questions of the topic and "KROK 1" tasks	Control questions of the topic, task " KROK 1"	
3	Determination of the final level of knowledge and skills. Summarizing the results.	35	Determination of the final level of formation of knowledge and skills	KROK 1 tasks, situational tasks	

The graphological structure of the topic "Pathophysiology of the circulatory system. Insufficiency of blood circulation. Cardiac insufficiency" is added.

Material and methodological support of the topic "Pathophysiology of the circulatory system. Insufficiency of blood circulation. Cardiac insufficiency":

1. Lectures;
2. Methodical instructions for teachers;
3. Methodical instructions for students;
4. Set of test tasks to determine the basic level of knowledge;
5. Set of situational tasks to determine the final level of knowledge;
6. Set of KROK-1 tasks;
7. Set of schemes and tables (presentation);
8. Set of forms with a clinical blood test;
9. Video films;

Lesson content:

1. Circulatory insufficiency.
2. Acute and chronic insufficiency.
3. Heart failure.
4. Classification of heart failure types.
5. Mechanisms of heart function compensation.
6. Types of heart dysfunction.
7. Manifestations of heart failure**.**

Terminology:

- Hemodynamic
- Cardiogenic
- Nomotopic
- Heterotopic
- Heterometric
- Homeometric
- Hypertrophy

Tasks for independent work on the topic «Pathophysiology of the systemic blood circulation. Insufficiency blood circulation. Cardiac insufficiency»

The student is offered 2–3 ECGs with signs of heart failure. It is necessary to determine ECG signs of acute and chronic heart failure. Be able to explain the electrophysiological mechanisms of their occurrence. Analysis of errors with an explanation of the correct answers

List of questions and works to be studied:

1. The concept of heart failure. Reasons, types.
2. Compensatory and adaptive mechanisms.
3. Violation of hemodynamics.
4. Manifestations of heart failure.
5. Tonogenic and myogenic dilation of the heart.
6. Mechanisms of long-term adaptation of the heart to overloads.
7. Myocardial hypertrophy.

List of practical skills that must be mastered:

1. Explain compensatory reactions in heart failure.
2. Compare the features of systolic and diastolic dysfunction.
3. Identify on the ECG signs of accompanying disorders of systemic hemodynamics and heart function in acute and chronic heart failure,
4. Describe signs reflecting the presence of systolic and diastolic dysfunction, compensatory hypertrophy, nomotopic and heterotopic arrhythmias, conduction, excitability and contractility.

Situational tasks KROK-1 to determine the final level of knowledge

1. ECG of a 44-year-old patient shows signs of hypertrophy of both ventricles and the right atrium. The patient was diagnosed with the tricuspid valve insufficiency. What pathogenetic variant of cardiac dysfunction is usually observed in case of such insufficiency?

A. Heart overload by volume.
B. Heart overload by resistance.
C. Primary myocardial insufficiency.
D. Coronary insufficiency.
E. Cardiac tamponade.

2. In course of a preventive examination of a miner a doctor revealed changes of cardiovascular fitness, which was indicative of cardiac insufficiency at the compensation stage. What is the main proof of cardiac compensation?

A. Tachycardia .
B. Myocardium hypertrophy.
C. Rise of arterial pressure.
D. Dyspnea.
E. Cyanosis.

3. Dystrophic changes of the heart muscle are accompanied with cardiac cavity enlargement, decrease of the strength of heart contraction, increased amount of blood, which remains in the heart during systolic phase, overfilled veins. For what state of heart is it characteristic?

A. Myogenic dilatation.

B. Tonogenic dilatation.
C. Emergency stage of hyperfunction and hypertrophy.
D. Cardiosclerosis.
E. Tamponade of the heart.

4. A patient has a history of chronic heart failure. Which of the following hemodynamic parameters is a major symptom of cardiac decompensation development?

A. Tonogenic dilatation. *D. Increased peripheral vascular resistance.*
B. Decreased stroke volume. *E. Increased central venous pressure.*
C. Tachycardia development.

5. An animal with aortic valve insufficiency got hypertrophy of its left heart ventricle. Some of its parts have local contractures. What substance accumulated in the myocardiocytes caused these contractures?

A. Potassium. *C. Calcium.* *E. Sodium.*
B. Lactic acid. *D. Carbon dioxide.*

6. A patient with mitral valve insufficiency developed hypertrophy of the left ventricle of the heart. What is the starting mechanism in the development of hypertrophy?

A. Activation of the genetic apparatus.
B. Increasing consumption of fatty acids.
C. Increasing the intensity of cellular respiration.
D. Activation of glycolysis.
E. Increase in Ca2+ influx into the cell.

7. A patient who suffers from severe disorder of water-salt metabolism experienced cardiac arrest in diastole. What is the most probable mechanism of cardiac arrest in diastole?

A. Hyperkaliemia. *C. Organism dehydratation.* *E. Hyponatremia.*
B. Hypernatremia. *D. Hypokaliemia.*

8. In order to reproduce heart failure, the frog's heart was perfused with a solution of cadmium bromide, a blocker of sulfhydryl groups. What variant of heart failure occurs in this case?

A. Mixed form.
B. From volume overload.
C. From toxic damage to the myocardium.
D. Caused by a violation of coronary blood circulation.
E. From resistance overload.

9. Transmural myocardial infarction in the patient was complicated with progressive acute left ventricle insufficiency. What is the most typical for this state?

A. Edema of the lungs. *C. Cyanosis.* *E. Arterial hypertension.*
B. Edema of the extremities. *D. Ascites.*

10. A patient with extensive myocardial infarction has developed heart failure. What pathogenetic mechanism contributed to the development of heart failure in the patient?

A. Pressure overload.
B. Acute cardiac tamponade.
C. Myocardial reperfusion injury.
D. Volume overload.
E. Reduction in the mass of functioning myocardiocytes.

Standards of correct answers to the task КROK-1

1	2	3	4	5	6	7	8	9	10
A	*B*	*A*	*C*	*C*	*A*	*A*	*C*	*A*	*E*

Recommendations for registration of work results

1. Written answers to test tasks (initial level of knowledge).
2. Protocol of ECG analysis with acute and chronic heart failure.
3. Protocol for solving situational tasks with an explanation of the correct answers.

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https://accessmedicine.mhmedical.com/book.aspx?bookid=2748

Topic № 7. Heart arrhythmia

Number of hours: 2 academic hours.

Relevance of the topic: A complication of many diseases of the cardiovascular system is changes in heart rate, which are accompanied by a violation of the frequency, rhythm, sequence of excitation, and contraction of the heart muscle. Arrhythmia is based on a change in the conditions for the formation of excitation of the heart muscle or an anomaly in its distribution paths, which can significantly affect the normal contractile activity of the heart and lead to hemodynamic disorders and a number of serious complications. Modeling of arrhythmias in animals makes it possible to study in more detail the etiological factors that cause arrhythmias and to elucidate some of the missing links in their pathogenesis. This information, as well as an understanding of ECG changes, is necessary for the doctor to select the correct etiological and pathogenetic therapy and thus an effective treatment strategy.

Purpose of the lesson:

General – to be able to reproduce the model of the main forms of cardiac rhythm disturbances caused by dysfunction of the pathways of the heart, to explain the causes and mechanisms of their occurrence in order to develop the ability to use etiotropic and pathogenetic treatment of arrhythmias.

Specifically:

Know:

1. Give the definition of "cardiac arrhythmia" and classify them.
2. In the experiment on the frog modulate violations of automatism, excitability and conduction of the heart.
3. Write down kymograms arrhythmias and compare them with the normal rhythm to determine the main features of arrhythmias.
4. Be able to explain the mechanisms of arrhythmias appearance, their main manifestations on ECG and hemodynamic disturbances in the organism during arrhythmia.

To be able to:

1. Explain the basic properties of the heart muscle (automatism, excitability, conductivity and contractility).
2. Explain the ECG of a healthy person in the standard leads.
3. To determine the main ECG signs in cardiac arrhythmias caused by a violation of the function of automatism, excitability and conduction of the heart. Explain the mechanism of their occurrence.
4. Explain the mechanisms of neural and humoral regulation of cardiac activity.

Practical experience:

Determination of signs of heart rhythm disorders on the ECG:

1. Nomotopic rhythms (sinus tachycardia, sync bradycardia, sinus arrhythmia);
2. Heterotopic rhythms (atrial, nodal, ventricular, pacemaker migration);

3. Extrasystole, flutter and fibrillation of atria and ventricles;
4. Slowing down or blockage of conduction of impulses to the heart;
5. Syndrome of premature excitation of the ventricles.

Technological map of students' work on the topic «Heart Arrhythmia»

№ п\п	Stage of the lesson	Academic time, min	Educational guide		Place holding a class
			Educational tools	Equipment	
1	Determination of the initial level of knowledge	10	Written response to test tasks	Test tasks	Learning room
2	Analysis of theoretical material	35	Analysis of the theoretical material is carried out on the basis of control questions of the topic and "KROK 1" tasks	Control questions of the topic, task " KROK 1" situational tasks, ECG set	
3	Practical part (carrying out experiment)	30	Introduction and preparation for setting up the experiment. Setting up the experiment. Discussion of experiment results and formulation of conclusions	Kymograph, tripod, cork board for frog fixation, device for recording heart contractions, tweezers, scissors, Ringer's solution, ligatures	
4	Determination of the final level of knowledge and skills. Summarizing the results	15	Solution of situational tasks to determine the final level of knowledge and skills of the student	KROK-1 tasks, situational tasks	

The graphological structure of the topic "Heart Arrhythmia" is attached.

Material and methodological support of the topic "Heart Arrhythmia".

1. Lectures;
2. Methodical instructions for teachers;
3. Methodical instructions for students;
4. Set of test tasks to determine the basic level of knowledge;
5. Set of situational tasks to determine the final level of knowledge;
6. Set of KROK-1 tasks;
7. Set of schemes and tables (presentation);
8. Set of ECG with irregular heart rhythm.
9. Video films;
10. For the experiment (experimental animals - frogs; kymograph, tripod, cork board for fixing the frog, device for recording heart contractions, tweezers, scissors, Ringer's solution, ligatures).

Lesson content:

1. Arrhythmia definition
2. Causes of arrhythmias
3. Arrhythmia classification
4. Pathogenesis of arrhythmia
5. Manifestations of arrhythmia

6. ECG signs
7. Complication

Setting up the experiment. Discussion of results and formulation of the conclusions

- **Modeling of cardiac automatism disorders:**

1. **Sinus tachycardia** – irrigate the heart with 1–2 ml of Ringer's solution, heated to 30–35 °C, and observe how quickly the frequency of heart contractions occurs;

2. **Reflex sinus bradycardia** – several blows are applied to the frog's stomach with tweezers; at the same time, there is a short-term stop or a sharp slowing of heart contractions; the activity of the heart is soon restored.

3. **Simulation of extrasystole**: after turning on the current, touch the end of the electrode to the ventricles of the heart during diastole. Electrical irritation causes an additional contraction (extrasystole), followed by a prolonged pause (compensatory).

4. **Simulation of heart block**: the recording of atrial contractions is set up simultaneously with the recording of ventricular contractions. After recording the curve of normal contractions of the atria and ventricles, apply a ligature on the border between the atria and the ventricle and tighten it. After the complete cessation of conduction, the atria continue to contract in the former rhythm. The contractions of the ventricles first stop, and then they begin to contract, but at a much slower rate.

Discussion of the results of the experiment

• As the first experience shows, the effect of heat (heated Ringer's solution) on the heart causes an increase in its contractions (tachycardia).

• Bradycardia, which was observed when the animal was tapped on the stomach, occurs reflexively. Irritation is perceived by the receptors of the organs of the abdominal cavity, comes along the precentral fibers to the center of the vagus nerve in the medulla oblongata, and from there is transmitted along the branches of the vagus nerve to the heart.

A similar stoppage or slowing down of heart activity in humans can be observed with injuries.

• Additional contraction of the heart (extrasystole) occurs in connection with the occurrence of an additional source of excitation in any part of the conduction system of the heart. The extended pause (compensatory) observed after extrasystole is explained by the fact that another normal impulse coming from the sinus node finds the heart muscle in the refractory phase of extrasystole. Therefore, the next contraction of the heart falls out and the ventricle remains at rest until the next normal impulse reaches it from the sinus node.

• Imposition of a ligature in our experience caused a violation of the conduction between the atria and the ventricles (atrioventricular block) (Stannius

experiment). If you gradually tighten the ligature, you can sometimes observe incomplete blockade: for two or three contractions of the atria, one contraction of the ventricles is necessary. With the stronger tightening of the ligature, a complete blockade of the atrium is obtained, and the ventricles contract independently of each other. At the same time, the atria continue to receive impulses from the sinus node and contract in the same rhythm. The ventricle receives impulses from the lower parts of the conduction system. Due to the fact that the automatism of these departments is less pronounced than the automatism of the sinus node, the contractions of the ventricles occur at a much slower rate.

• Conduction disorders can occur as a result of changes of both functional and organic nature in the conducting system of the heart. I.I. For the first time, Pavlov experimentally induced neurogenic atrioventricular heart block by stimulating the heart's accelerating nerves. When the heart's reinforcing nerves were irritated, this phenomenon was eliminated.

Formulation of conclusions based on the experiment

1. **Sinus tachycardia**. As a result of applying heated Ringer's solution to the heart, an increase in heart rate occurs – sinus tachycardia. Mechanism: direct effect of the temperature factor on the sinus node.

2. **Sinus bradycardia.** As a result of blows to the stomach, damage to heart contractions occurs – sinus bradycardia. Mechanism: irritation of the vagus nerve, which in turn causes hyperpolarization, that is, there is a significant increase in the maximum diastolic potential.

3.**Ventricular extrasystole.** When an electric current is applied to the heart muscle, an extraordinary contraction occurs – extrasystole. Mechanism: additional contraction of the heart occurs due to the appearance of an additional source of excitation in the area of the heart's conduction system. An increased (prolonged) compensatory pause is observed after extrasystole and is explained by the fact that another normal impulse coming from the sinus node catches the heart muscle in the reflex phase of extrasystole.

4. **Complete atrioventricular block**. The imposition of a ligature caused a violation of conduction between the atria and ventricles (atrioventricular block). At the same time, the atria continue to receive impulses from the sinus node and contract in the former rhythm. The ventricles receive impulses from the lower parts of the conduction system. Because the automatism of these departments is less pronounced than the automatism of the sinus node, the contraction of the ventricles occurs in a more rarefied mode.

Terminology:

- Arrhythmia
- Tachycardia,
- Bradycardia
- Excitability

- Extrasystole
- Conductivity
- Blocade
- Contractility

Tasks for independent work on the topic "Heart Arrhythmia"

The student is offered 2–3 ECGs with rhythm and conduction disturbances. It is necessary to determine the ECG signs and the type of rhythm and conduction disturbance. Be able to explain the mechanism of occurrence. Analysis of errors with an explanation of the correct answers.

List of questions and works to be studied:

1. Definition of the term "arrhythmia". The main causes of arrhythmias. Signs of functional and organic heart rhythm disorders.
2. Arrhythmias caused by a violation of automatism. Reasons, types. ECG signs.
3. Sinus node weakness syndrome. Causes, electrophysiological mechanism, main ECG signs.
4. Heterotopic heart rhythm disorders. Types of ectopic rhythms.
5. Migration of the rhythm driver. Reasons, main ECG signs.
6. Arrhythmias as a result of impaired conduction of impulses to the heart. Kinds.
7. Arrhythmias as a result of slowing down or blocking the conduction of impulses to the heart. Reasons, types.
8. Syndrome of premature excitation of the ventricles. Reasons. Manifestations. Basic ECG signs.
9. Combined heart rhythm disorders. Causes, mechanisms, types.

List of practical skills that must be mastered:

Determination of signs of heart rhythm disorders on the ECG:

1. Nomotopic rhythms (sinus tachycardia, sync bradycardia, sinus arrhythmia, SNWS);
2. Heterotopic rhythms (atrial, nodal, ventricular, pacemaker migration);
3. Extrasystole, flutter and fibrillation of atria and ventricles;
4. Slowing down or blockage of conduction of impulses to the heart;
5. Syndrome of premature excitation of the ventricles

Situational tasks KROK-1 to determine the final level of knowledge

1. Processes of repolarisation are disturbed in ventricular myocardium in examined person. It will cause amplitude abnormalities of configuration and duration of the wave:

A. T. *B. Q.* *C. R.* *D. S.* *E. P.*

2. Person has stable HR, not more than 40 bpm. What is the pacemaker of the heart rhythm in this person?

A. Atrioventricular node.
B. Sinoatrial node.
C. His' bundle.
D. Branches of His' bundle.
E. Purkinye' fibers.

3. A patient has a first-degree atrioventricular block accompanied by the prolongation of P-Q interval up to 0,25 s. Under such conditions the following myocardial function will be disturbed:

A. Excitability. *C. –.* *E. Contractibility.*
B. Conduction. *D. Automatism.*

4. A patient has extrasystole. ECG shows no P wave, QRS complex is deformed, there is a full compensatory pause. What extrasystoles are these?

A. Atrial. *C. Atrioventricular.* *E. –.*
B. Ventricular. *D. Sinus.*

5. A 45-year-old patient was admitted to the cardiological department. ECG data: negative P wave overlaps QRS complex, diastolic interval is prolonged after extrasystole. What type of extrasystole is it?

A. Sinus. *C. Atrioventricular.* *E. Bundle-branch.*
B. Atrial. *D. Ventricular.*

6. A 67-year-old patient complains of periodic heart ache, dyspnea during light physical activities. ECG reveals extraordinary contractions of heart ventricles. Such arrhythmia is called:

A. Bradycardia. *C. Flutter.* *E. Fibrillation.*
B. Tachycardia. *D. Extrasystole.*

7. Analysis of the ECG revealed the missing of several PQRST cycles. The remaining waves and complexes are not changed. Specify the type of arrhythmia:

A. Sinoatrial block. *D. Intra-atrial block.*
B. Atrial fibrillation. *E. Atrial premature beat.*
C. Atrioventricular block.

8. In a 45-year-old patient on ECG it was revealed: sinus rhythm, the number of auricular complexes exceeds number of ventricular complexes; progressing extension of the P-Q interval from complex to complex; fallout of some ventricular complexes; P waves and QRST complexes are without changes. Name the type of heart rhythm dysfunction.

A. Synoauricular block.
B. Atrioventricular blockade of the I degree.
C. Intraatrial block.
D. Complete atrioventricular block.
E. Atrioventricular block of the II degree.

9. A 49 y.o. woman consulted a doctor about heightened fatigue and dyspnea during physical activity. ECG: heart rate is 50/min, PQ is extended, QRS is unchanged, P wave quanity exceeds quantity of QRS complexes. What type of arrhythmia does the patient have?

A. Extrasystole. *D. Sinoatrial block.*
B. Sinus bradycardia. *E. Atrioventricular block.*
C. Ciliary arrhythmia.

10. ECG of a patient shows such alterations: P-wave is normal, P-Q-interval is short, ventricular QRST complex is wide, R-wave is double-peak or two-phase. What form of arrhythmia is it?

A. WPW syndrome (Wolff-Parkinson-White).
B. Frederick's syndrome (atrial flutter).
C. Atrioventricular block.
D. Ventricular fibrillation.
E. Ciliary arrhythmia.

Standards of correct answers to the situational tasks

1	2	3	4	5	6	7	8	9	10
A	*A*	*B*	*B*	*C*	*D*	*A*	*E*	*E*	*A*

Recommendations for registration of work results

1. Written answer to test tasks (basic level of knowledge).
2. The results of the experiment are drawn up in the form of an experiment protocol with the determination of relevant conclusions.
3. Protocol of ECG analysis with heart rhythm disturbance.
4. Protocol for solving situational tasks with an explanation of the correct answers.

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Main

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Topic № 8. Coronary damage of the myocardium. Coronary insufficiency. Coronary heart disease. Myocardial infarction. Cardiogenic shock

Number of hours: 2 academic hours.

Relevance of the topic. The high level of morbidity and mortality from diseases of the cardiovascular system is largely determined in connection with their significant prevalence. Among them, an important place is occupied by the coronary insufficiency, due to spasm of coronary vessels, narrowing of the lumen due to atherosclerotic plaque or thrombus, and reduction of coronary blood circulation causes local ischemia of the myocardium. Occurrence of acute coronary insufficiency causes myocardial infarction, chronic – progresses to stable angina pectoris with subsequent development of myocardial hypertrophy and heart failure. In this regard, knowledge of the causes and mechanisms of the development of this pathology will contribute to the formation of clinical thinking and the rational choice of approaches to the treatment of each patient.

Purpose of the lesson:

General – to be able to characterize coronary insufficiency, explain the main causes and mechanisms of development.

Specifically:

Know:

1. To reveal the essence of the concept of "coronary heart damage", "acute heart failure"," acute coronary insufficiency", "cardiogenic shock",
2. To classify the causes and mechanisms of coronary damage
3. Coronary insufficiency. Reasons. Myocardial reperfusion.
4. Cardiogenic shock. Pathogenesis.
5. Ischemic heart disease, myocardial infarction. Causes, detection mechanism and hemodynamic disorders.
6. Pathogenesis of electrocardiogram changes in coronary artery disease, myocardial infarction.

To be able to:

1. To model acute heart failure on rats, to explain the mechanisms of compensation and decompensation during the experiment.
2. Identify the main manifestations of heart failure,
3. To explain the mechanisms of their occurrence and development.
4. Identify ECG signs of ischemia, damage, and necrosis of the myocardium.
5. Identify ECG signs of acute coronary insufficiency, stage of myocardial infarction, explain the mechanism of their occurrence.

Practical experience:

1. Determination of signs of ischemia, damage, necrosis of the myocardium on the ECG.
2. Determination on the ECG of signs of acute coronary insufficiency.
3. Myocardial infarction, its stage of development, depth, localization.

Technological map of students' work on the topic "Coronary damage of the myocardium. Coronary insufficiency. Coronary heart disease. Myocardial infarction. Cardiogenic shock"

№	Stage lesson	Academic time, min	Educational guide		Place holding a class
			Educational tools	Equipment	
1	Determination of the initial level of knowledge	10	Written answer to test task.	Test task	Learning room
2	Analysis of theoretical material	35	Analysis of theoretical material based on control questions of the topic, situational tasks, tasks of KROK-1	Control questions of the topic, task " KROK 1" Situational tasks	
3	Practical part (carrying out experiment)	30	Introduction and preparation for setting up the experiment. Setting up the experiment. Discussion of the results of the experiment and formulation the conclusions	Electrocardiograph, adrenaline, syringe. The experimental animal is rats.	
4	Determination of the final level of knowledge and skills. Summarizing the results	15	Determination of the final level of knowledge and skills. Summarizing the results	KROK-1 tasks, situational tasks	

The graphological structure of the topic "Coronary damage of the myocardium. Coronary insufficiency. Coronary heart disease. Myocardial infarction. Cardiogenic shock" is attached.

Material and methodical support of the topic "Coronary damage of the myocardium. Coronary insufficiency. Coronary heart disease. Myocardial infarction. Cardiogenic shock":

1. Lectures;
2. Methodical instructions for teachers;
3. Methodical instructions for students;
4. Set of test tasks to determine the basic level of knowledge;
5. Set of situational tasks to determine the final level of knowledge;
6. Set of KROK-1 tasks;
7. Set of schemes and tables (presentation);
8. Set of forms with a clinical blood test;
9. Video films;
10. For the experiment experimental animal – rats, electrocardiograph, adrenaline, syringe.

Lesson content:

1. Causes of coronary insufficiency
2. The leading pathogenetic factor of coronary insufficiency
3. Mechanisms of disorders of energy supply of cardiomyocytes
4. Myocardial reperfusion

5. Stable angina
6. Unstable angina
7. Acute Coronary Syndromes
8. Forms of myocardial infarction
9. ECG criterium of myocardial infarction

Setting up the experiment. Discussion of results and formulation of conclusions

- **Induction of myocardial necrosis in the rat.**

1. Fix the rat .

2. Strengthen the electrodes connected to the electrocardiograph through the transition bar.

3. Record the electrocardiogram in standard leads.

4. Administer intraperitoneally a 0.1% solution of adrenaline at the rate of 300 μg per 100 g of body weight.

5. Record the electrocardiogram after 15, 30, 45 minutes. after the injection of adrenaline.

6. Compare electrocardiograms with the original data. Pay attention to the change in the QRS complex, confusion of the S-T segment, the peculiarities of the T wave, as well as the change in the rhythm of cardiac activity.

- **Discussion of the results of the experiment.** One of the models of noncoronary myocardial necrosis is epinephrine. Noncoronarogenic experimental necrosis also includes hypoxic necrosis, electrolyte-steroid cardiopathy, immune and neurogenic heart damage.

Formulation of conclusions from the experiment.

1. Myocardial necrosis. Myocardial necrosis developed as a result of the introduction of adrenaline in the rat. Mechanism: cardiotoxic effect of catecholamines on the myocardium (increased myocardial oxygen demand due to positive chronotropic and inotropic effects, decrease in coronary blood flow, disruption of ATP resynthesis mechanisms, prooxidant effect).

2. ECG signs of damage (necrosis) of the myocardium are manifested by a shift in the ST segment, a change in the T wave and the ventricular QRS complex (decrease in its amplitude), as well as heart rhythm disturbances.

Terminology:

- Coronarogenic
- Ischemia.
- Atherosclerosis
- Catecholamines
- Reperfusion
- Angina
- Infarction

Tasks for independent work on the topic "Coronary damage of the myocardium. Coronary insufficiency. Coronary heart disease. Myocardial infarction. Cardiogenic shock":

The student is offered 2–3 ECGs with coronary damage of the myocardium. It is necessary to determine the ECG signs and the type of damage to the myocardium in case of coronary heart disease (ischemia, damage, necrosis, cardiosclerosis). To be able to explain the mechanism of the occurrence. Analysis of errors with an explanation of the correct answers

List of questions and works to be studied:

Coronary heart damage.

1. Ischemic heart disease, myocardial infarction.
2. Pathogenesis of electrocardiogram changes.
3. Coronary insufficiency. Definition of the concept. Reasons.
4. Myocardial reperfusion.
5. Reversible and irreversible disorders of coronary blood flow.
6. Cardiogenic shock. Pathogenesis.
7. Experimental models of myocardial necrosis.

List of practical skills that must be mastered:

1. Identify the main manifestations of heart failure,
2. Explain the mechanisms of their occurrence and development.
3. Determination of signs of ischemia.
4. Necrosis of the myocardium on the ECG.
5. Determination on the ECG of signs of acute coronary insufficiency – myocardial infarction, its stage of development, depth, localization.

Situational tasks KROK-1 to determine the final level of knowledge

1. After a serious psycho-emotional stress a 45-year-old patient suddenly felt constricting heart pain irradiating to the left arm, neck and left scapula. His face turned pale, the cold sweat stood out on it. The pain attack was stopped with nitroglycerine. What process has developed in this patient?

A. Myocardial infarction.
B. Stenocardia.
C. Stroke.
D. Psychogenic shock.
E. Stomach ulcer perforation.

2. After a serious psychoemotional stress a 48 year old patient suddenly developed acute heart ache irradiating to the left arm. Nitroglycerine relieved pain after 10 minutes. What is the leading pathogenetic mechanism of this process development?

A. Spasm of coronary arteries.
B. Dilatation of peripheral vessels.
C. Obstruction of coronary vessels.
D. Compression of coronary vessels.
E. Increase in myocardial oxygen consumption.

3. After a psychoemotional stress a 48 year old patient had a sudden attack of acute heart pain with irradiation to the left hand. Nitroglycerine suppressed pain in 10 minutes. What pathogenetic mechanism is principal for the pain development?

A. Spasm of coronary vessels.

B. Dilatation of peripheral vessels.

C. Coronary vessel occlusion.

D. Embarrassment of coronary vessels.

E. Increased need of myocardium in oxygen.

4. A patient is 59 years old and works as director of a private enterprise. After the inspection by tax authorities he developed intense burning retrosternal pain radiating to the left arm. After 15 minutes the patient returned to normal. What is the leading mechanism for the development of stenocardia in this patient?

A. Functional overload of heart.

B. Increased level of blood catecholamines.

C. Coronary thrombosis.

D. Coronary atherosclerosis.

E. Intravascular aggregation of blood corpuscles.

5. A patient suffering from stenocardia was taking nitroglycerine which caused restoration of blood supply of myocardium and relieved pain in the cardiac area. What intracellular mechanism provides restoration of energy supply of insulted cells?

A. Reduction of ATP resynthesis.

B. Increased permeability of membranes.

C. Intensification of ATP resynthesis.

D. Intensification of oxygen transporting into the cell.

E. Intensification of RNA generation.

6. During fighting a man had a cardiac arrest as a result of a hard blow to the upper region of anterior abdominal wall. Which of the described mechanisms might have provoked the cardiac arrest?

A. Sympathetic unconditioned reflexes.

B. Peripheric reflexes.

C. Parasympathetic unconditioned reflexes.

D. Sympathetic conditioned reflexes.

E. Parasympathetic conditioned reflexes.

7. A 59 year old patient is a plant manager. After the tax inspection of his plant he felt intense pain behind his breastbone irradiating to his left arm. 15 minutes later his condition came to normal. Which of the possible mechanisms of stenocardia development is the leading in this case?

A. Coronary atherosclerosis.

B. Intravascular aggregation of blood corpuscles.

C. Coronary thrombosis.

D. High catecholamine concentration in blood.

E. Functional heart overload.

8. The patient with acute myocardial infarction was given intravenously different solutions during 8 hours with medical dropper 1 500 ml and oxygen intranasally. He died because of pulmonary edema. What caused the pulmonary edema?

A. Decreased oncotic pressure due to hemodilution.

B. Allergic reaction.

C. Neurogenic reaction.

D. Inhalation of the oxygen.

E. Volume overload of the left ventricular.

9. Since a patient has had myocardial infarction, atria and. ventricles contract dependently from each , other with a sequence of 60–70 and 35–40 per minute. Specify the type of heart block in this case:

A. Sino-atrial.

B. Intraventricular.

C. Intra-atrial.

D. Complete atrioventricular.

E. Partial atrioventricular.

10. A 47 year old man with myocardium infarction was admitted to the cardiological department. What changes of cellular composition of peripheral blood are induced by necrotic changes in the myocardium?

A. Neutrophilic leukocytosis.

B. Monocytosis.

C. Eosinophilic leukocytosis.

D. Thrombocytopenia.

E. Lymphopenia.

Standards of correct answers to situational problems

1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
B	*A*	*A*	*B*	*C*	*C*	*D*	*E*	*D*	*A*

Recommendations for registration of work results.

1. Written answer to test tasks (initial level of knowledge)

2. The results of the experiment are drawn up in the form of an experiment protocol with the determination of relevant conclusions.

3. Protocol for the ECG analysis with coronary myocardial damage.

4. Protocol for solving situational tasks with an explanation of the correct answers.

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Topic № 9. Pathophysiology of blood vessels. Arterial Hypertension and hypotension. Vascular insufficiency. Atherosclerosis

Number of hours: 2 academic hours.

Relevance of the topic: Mechanisms of regulation of vascular tone are of urgent importance for theoretical and clinical medicine. The study of the causes and mechanisms of regulation of vascular tone is relevant for theoretical and clinical medicine, since these disorders underlie hypertensive disease and symptomatic hypertension observed in numerous diseases. Knowledge of the causes and mechanisms of violations is important and to understand hypotension, especially acute vascular insufficiency. The creation of adequate models is important for elucidating the causes and mechanisms of its disorders in humans and, thus, for improving the prevention and treatment of hyper- and hypotension. To improve preventive measures and treatment of hypertension and hypotension, it is important to study the cause of cardiovascular diseases, including atherosclerosis. Pathogenesis and consequences of atherosclerosis are an urgent problem of modern medicine.

Purpose of the lesson:

General:

– to study common laws and features of different forms of disorders of vascular tone, particularly etiological different types of hypertension;

– to study the common laws of beginning, development and results of atherosclerosis.

Specifically:

Know:

1. To determine the blood pressure in rabbits.
2. To explain the mechanisms of centrogenic, reflexogenic and renal hypertension.
3. Differ the hyper- and hypotension states, and acute vascular insufficiency.
4. Form the knowledge about atherosclerosis.
5. Explain the mechanisms of atherosclerosis development.
6. Know the results and value atherosclerosis in pathology.

To be able to:

1. Classify the types of division of ships according to their purpose.
2. Explain the features of neurohumoral regulation of various forms of vascular tone disorders.
3. Know the features of the manifestation of hypertension of different types.
4. Explain the mechanisms of development of atherosclerosis

Practical experience:

1. To analyze the mechanisms of regulation of vascular tone and the mechanisms of impaired regulation.
2. To find out the pathogenesis of hypotensive and hypertensive conditions.
3. To analyze the consequences of a persistent increase in blood pressure.

Technological map of the topic "Pathophysiology of blood vessels. Arterial hypertension and hypotension. Vascular insufficiency. Atherosclerosis"

№	Stage lesson	Academic time, min	Educational guide		Place holding a class
			Educational tools	Equipment	
1	Determination of the basic level of knowledge	10	Written answer to test tasks	Test tasks	Study room
2	Analysis of theoretical material	35	Analysis of theoretical material based on control questions of the topic, situational tasks, tasks of KROK-1	Topic control questions, KROK-1 tasks, situational tasks	
3	Practical part (conduct experiment)	30	Introduction and preparation for setting up the experiment. Setting up the experiment. Discussion of the results of the experiment and formulation the conclusions	Rabbit, device for measuring blood pressure	
4	Determination of the final level of knowledge and skills. Summarizing the results	15	Determination of the final level of knowledge and skills. Summarizing the results	KROK-1 tasks, situational tasks	

The graphological structure of the topic«Pathophysiology of blood vessels. Arterial hypertension and hypotension. Vascular insufficiency. Atherosclerosis» is attached.

Material and methodical provision of the topic "Pathophysiology of blood vessels. Arterial hypertension and hypotension. Vascular insufficiency. Atherosclerosis":

1. Lectures;
2. Methodical instructions for teachers;
3. Methodical instructions for students;
4. Set of test tasks to determine the basic level of knowledge;
5. Set of situational tasks to determine the final level of knowledge;
6. Set of KROK-1 tasks;
7. Set of schemes and tables (presentation);
8. Video films;
9. For the experiment: an experimental animal – a rabbit (intact, with experimental renal and reflexogenic hypertension), a device for measuring blood pressure.

Lesson content:

1. Definition of the concepts of "arterial hypertension and hypotension".
2. Hypertensive disease.
3. Vascaulr insufficiency
4. Hypertensive crisis, risk factors.
5. Pathogenesis of atherosclerosis.
6. Experimental models of atherosclerosis.

Setting up the experiment. Discussion the results and of conclusions.

- **Mastering the method of determining blood pressure according to the Grand Rothschild method.**

1. Put a cuff on the central artery of the rabbits ear so that the artery crossing the cuff in its central part is clearly visible through the outer surface of the cuff.
2. Connect the cuff with the injection balloon and the device for measuring blood pressure.
3. With the fingers of the left hand, fix the cuff, with the right hand, squeezing the balloon, inject air into the system until the blood flow stops in the center of the artery.
4. At this moment, note the value of the blood pressure according to the manometer reading (measure the pressure at least three times and calculate the average value).
5. Set the value of blood pressure in the rabbit in the norm.
6. Blood pressure on the day of the class is mmHg.

- **Study of blood pressure in experimental renal hypertension.**

1. Before the lesson, an incision was made in the skin and subcutaneous adipose tissue in the place of the projection of the left kidney in a rabbit under general anesthesia (mask ether anesthesia).
2. Tissue stratification up to the paranephric tissue was produced by a blunt method.
3. The kidney is brought out into the operating hole.
4. The renal pedicle is compressed by muscles on both sides.
5. The wound is sutured in layers.
6. Blood pressure on the day of the class is mmHg.
7. Draw a diagram of the pathogenesis of renal (renovascular) hypertension.

- **Studing of blood pressure in experimental reflexogenic hypertension.**

1. Before the lesson, an incision was made in the skin and subcutaneous fascia of the rabbit under local anesthesia (10 ml of 0.25 % novocaine solution) along the middle line of the neck.
2. Bluntly selected vascular-nerve bundle.
3. The depressor nerve is taken with ligatures and brought out into the operating hole.
4. Two ligatures are placed on the nerve at a distance of 1 cm from each other and tightened tightly.
5. We dissect the nerve between these ligatures.
6. The area of the carotid sinus is treated with ethyl alcohol.
7. The wound is sutured in layers.
8. Blood pressure on the day of the class is mmHg.
9. Draw a scheme of pathogenesis of reflexogenic hypertension.

Discussion of the results of the experiment

- **Discussion of the results of the experiment.** There are various methods of causing a persistent increase in blood pressure – hypertension in an experiment by influencing the animals nervous system. Such experimental neurogenic hypertension includes reflexogenic hypertension. Receptors of blood vessels, in particular, receptors in the ascending part of the aortic arch and in the region of the carotid sinus, are important in the reflex regulation of blood circulation. In these places of the vascular system, there are sensitive endings of afferent nerves – the depressor (in the arch of the aorta) and the sinus nerve (in the carotid sinus). Bilateral transection of these nerves has an inhibitory effect on the vascular-motor center and causes a long-term increase in blood pressure in the animal. The second type of experimental neurogenic hypertension demonstrated in this session is caused by a direct effect on the anima central nervous system. For this purpose, a suspension of kaolin (20 mg/kg) is injected into the cysterna cerebello-medularis, as a result of which an inflammatory process develops, which leads to difficulty in draining the cerebrospinal fluid and increasing intracranial pressure. It is believed that hypertension develops as a result of brain ischemia.
- **Formulation the conclusions based on the experiment**

1. Experimental renal hypertension is caused by applying clamps to both renal arteries, due to which their lumen is narrowed. Kidney ischemia leads to the appearance of renin in the blood, under the influence of which angiotensinogen is split into the decapeptide angiotensin I, which, interacting with enzymes of the lungs and other tissues, turns into the octapeptide angiotensin II, which is the most powerful of the known pressor substances.

2. Experimental reflexogenic hypertension (refers to neurogenic hypertension) is caused by bilateral cutting of the afferent nerves of the aortic arch and carotid sinus, which has an inhibitory effect on the vasomotor center and causes a long-term increase in blood pressure in the animal.

3. Experimental hypertension is important for studying the pathogenesis of neugenic (reflexogenic) and renal (nephrogenic) hypertension in humans. Tasks for independent work on the topic. Pathophysiology of blood vessels.

Terminology:

- Hypertension.
- Hypotension.
- Dystonia.
- Hypertensive crisis.
- Vascular insufficiency.
- Atherosclerosis.

Tasks for independent work on the topic "Pathophysiology of blood vessels. Arterial hypertension and hypotension. Vascular insufficiency. Atherosclerosis".

The student needs to determine the signs of arterial hypertension and is asked to explain the mechanism of increased blood pressure during stenosis of the renal arteries. Analysis the errors with an explanation of the correct answers.

List of questions and works to be studied:

1. Definition of the concepts hypertension; hypotension.
2. Classification of disorders of vascular tone.
3. Hypertensive disease, its etiology and pathogenesis.
4. Symptomatic hypertension, their types, pathogenesis.
5. Experimental models of hypertension.
6. Acute and chronic vascular insufficiency, its types.
7. Shock. Causes, pathogenesis, stages, changes in the body.
8. Collapse. Reasons, mechanism of development.

List of practical skills that must be mastered:

1. Explain the mechanisms of centrogenic, reflexogenic hypertension.
2. Describe the mechanisms of renal hypertension
2. Determine hypo- and hypotonic states; acute vascular insufficiency

Situational tasks KROK-1 to determine the final level of knowledge

1. A 43-year-old-patient has arterial hypertension caused by an increase in cardiac output and general peripheral resistance. Specify the variant of hemodynamic development of arterial hypertension in the given case:

A. Hyperkinetic. *C. –.* *E. Eukinetic.*
B. Hypokinetic. *D. Combined.*

2. Prophylactic medical examination of a 36-year-old driver revealed that his AP was 150/90 mm Hg. At the end of working day he usually hears ear noise, feels slight indisposition that passes after some rest. He was diagnosed with essential hypertension. What is the leading pathogenetic mechanism in this case?

A. Neurogenic. *C. Humoral.* *E. Reflexogenic.*
B. Nephric. *D. Endocrinal.*

3. Arterial pressure of a surgeon who performed a long operation rised up to 140/110 mm Hg. What changes of humoral regulation could have caused the rise of arterial pressure in this case?

A. Activation of formation and excretion of aldosterone.
B. Activation of sympathoadrenal system.
C. Activation of renin angiotensive system.
D. Activation of kallikrein kinin system.
E. Inhibition of sympathoadrenal system.

4. A patient has the following diagnosis: renal hypertension. What is the initial pathogenetic factor of arterial hypertension development in this case?

A. Hypernatremia.
B. Renal ischemia.
C. Hyperaldosteronism.
D. Intensified renin synthesis.
E. Intensified angiotensin synthesis.

5. A patient with constant headaches, pain the occipital region, tinnitus, dizziness has in admitted to the cardiology department, objectively: AP – 180/110 mm Hg, heart rate 5/min. Radiographically, there is a stenosis one of the renal arteries. Hypertensive condition in this patient has been caused by in activation of the following system:

A. Renin-angiotensin.
B. Sympathoadrenal.
C. Kinin.
D. Immune.
E. Hemostatic.

6. Arterial hypertension is caused by the stenosis of the renal arteries in the patient. Activation of what system is the main link in the pathogenesis of this form of hypertension?

A. Sympathoadrenal.
B. Parasympathetic.
C. Renin-angiotensin.
D. Kallikrein-kinin.
E. Hypothalamic-pituitary.

7. A month after surgical constriction of rabbit's renal artery the considerable increase of systematic arterial pressure was observed. What of the following regulation mechanisms caused the animal's pressure change?

A. Serotonin.
B. Noradrenaline.
C. Angiotensin-II.
D. Adrenaline.
E. Vasopressin.

8. A patient ill with essential arterial hypertension had a hypertensic crisis that resulted in an attack of cardiac asthma. What is the leading mechanism of cardiac insufficiency in this case?

A. Heart overload caused by increased blood volume.
B. Heart overload caused by high pressure.
C. Absolute coronary insufficiency.
D. Myocardium damage.
E. Blood supply disturbance.

9. A 50 year old patient suffers from essential hypertension. After a physical stress he experienced muscle weakness, breathlessness, cyanosis of lips, skin and face. Respiration was accompanied by distinctly heard bubbling rales. What mechanism underlies the development of this syndrome?

A. Acute left-ventricular failure.
B. Chronic right-ventricular failure.
C. Chronic left-ventricular failure.
D. Collapse.
E. Cardiac tamponade.

10. An 18-year-old patient complains of general weakness, fatigue, low spirits. The patient is of the asthenic constitution type. Ps – 68/min., AP – 90/60 mm Hg. She has been found to have primary neurocirculatory hypotension. What is the leading factor of the arterial pressure drop in this patient?

A. Decreased minute blood volume.

B. Hypovolemia.

C. Decreased tonus of resistive vessels.

D. Deposition of blood in the veins of the systemic circulation.

E. Decreased cardiac output.

Standards of correct answers to the situational tasks

1	2	3	4	5	6	7	8	9	10
E	*A*	*B*	*B*	*A*	*C*	*C*	*B*	*A*	*C*

Recommendations for registration of work results

1. Written answer to test tasks (basic level of knowledge).

2. The results of the experiment are drawn up in the form of an experiment protocol with the determination of relevant conclusions.

3. Protocol for the study of the results determining blood pressure in experimental renal hypertension and experimental reflexogenic hypertension

4. Protocol for solving situational tasks with an explanation of the correct answers (final level of knowledge).

LITERATURE

Main

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Topic № 10. Pathophysiology of external respiration. Respiratory failure

Number of hours: 2 academic hours.

Relevance of the topic. The function of external respiration is vital since it ensures the continuous supply of oxygen to the living organism with the inhaled air and the release of carbon dioxide formed in it and, if necessary, other toxic gaseous metabolic products. The respiratory system provides protection from the action of environmental factors, being an important internal barrier, and is also involved in providing thermoregulation, voice production, smell, and humidification of the inhaled air. Lung tissue plays an important role in such processes as hormone synthesis, regulation of water-salt and lipid metabolism, and blood deposition. Taking into account that respiratory failure can occur with various diseases of the respiratory system and be a consequence of dysfunction of various organs and systems, the study of the etiology and pathogenesis of respiratory disorders is necessary for the development of clinical thinking of the future doctor, since without understanding the cause and mechanisms of the process, it is impossible to choose an effective method of treating this pathology.

Purpose of the lesson:

General – to be able to characterize the dyspnea as a manifestation of expiration, explain the common causes and mechanisms of its development.

Specifically:

Know:

1. Define the “pathological breathing”, “dyspnea”.
2. Classify the pathological types of breathing, types of dyspnea.
3. To model the different types of dyspnea on rabbits.
4. Show the role of reflect influences and disorders of functions of upper respiratory trance in forming of the dyspnea.
5. To distinguish the main signs and manifestations of dyspnea, explain the main mechanisms of those forming and development.

Be able to:

1. Explain the role of mechanoreceptors (Hering-Beuer reflex) in expiration regulation
2. Interpret chemoreceptor regulation of expiration.
3. Explain the influence of changes of frequency and depth of expiration on iteffectiveness.

Practical experience:

1. Identify the possible options for changing the frequency, depth and rhythm of breathing that occur in the animal in the experiment when reproducing stenosis of the upper respiratory tract.
2. Describe the mechanisms in accordance with which the above changes occur.
3. Determine the features of changes in breathing during acute strangulation of the upper respiratory tract, leading to asphyxia.

4. Describe the sequence and duration of inhalation and exhalation asta violation from the beginning of asphyxia to its outcome

Technological map of students' work on the topic "Pathophysiology of external respiration. Respiratory failure"

№	Stage lesson	Academic time, min	Educational guide		Place holding a class
			Educational tools	Equipment	
1	Determination of the initial level of knowledge	10	Written answer to test tasks	Test task	Learning room
2	Analysis of theoretical material	35	Analysis of the theoretical material is carried out on the basis of control questions of the topic and situational tasks, tasks of KROK-1	Topic control questions, KROK-1 tasks, situational tasks	
3	Practical part (carrying out experiment)	30	Introduction and preparation for setting up the experiment. Setting up the experiment. Discussion of experiment results and formulation of conclusions	Board for fixation of animals, pneumograph, kymograph, tripod, device for recording of breathing movements, scissors, tweezers, surgical needles, threads, ether, syringe, 20 % Na nitrite solution	
4	Determination of the final level of knowledge and skills. Summarizing the results	15	Determination of the final level of knowledge and skills. Summarizing the results	KROK-1 tasks, situational tasks	

The graphological structure of the topic "Pathophysiology of external respiration. Respiratory failure" is attached.

Material and methodological support of the topic "Pathophysiology of external respiration. Respiratory failure":

1. Lectures;
2. Methodical instructions for teachers;
3. Methodical instructions for students;
4. Set of test tasks to determine the basic level of knowledge;
5. Set of situational tasks to determine the final level of knowledge;
6. Set of KROK-1 tasks;
7. Set of schemes and tables (presentation);
8. Set of forms with a clinical blood test;
9. Video films;
10. For the experiment: a board for animal's fixing, a pneumograph, a kymograph, a tripod, a device for recording respiratory movements, scissors, tweezers, surgical needles, threads, ether, a syringe, 20 % sodium nitrite solution. Experimental animals – rabbit, mice.

Lesson content:

1. Respiratory failure
2. Typical disorders of external breathing
3. Alveolar hypoventilation
4. Alveolar hyperventilation
5. Obstractive disorders of alveolar ventilation
6. Restrictive disorders of alveolar ventilation
7. Violation of lung perfusion
8. Violation of the diffusion lungs capacity
9. Asphyxia.

Setting up the experiment. Discussion of the results and formulation of conclusions

- **Causing breathing disorders with gradual narrowing of the upper respiratory tract.**

1. The rabbit is tied to the machine.

2. The sensors of the pneumograph are attached to the chest and the recording of respiratory movements is set up on the kymograph tape.

3. Observe the nature of breathing in the initial state.

4. Observe the nature of breathing at different degrees of closure of the nasal passages, paying attention to changes in the frequency and depth of respiratory movements.

- **Causing breathing disorders with a sharp narrowing of the upper respiratory tract (asphyxia).**

1. The mouse is fixed to the board on the back.

2. An incision of 1 cm length is made along the midline of the neck, the muscles are bluntly pushed aside and the trachea is exposed.

3. Carefully place a silk thread under the trachea with tweezers.

4. The board with the mouse is fixed on a tripod.

5. I apply a small clamp connected to a pen to the skin of the chest.

6. Adjust the recording on the kymograph drum.

7. Record the initial respiratory movements.

8. Bandage the trachea and observe the nature of changes in breathing during asphyxia.

9. After stopping breathing, open the thorax, expose the heart of the animal and observe heart contractions.

Discussion of the results of the experiment.

Discussing the results of the experiments, remind the students of the main mechanisms of breathing regulation. Explain the features of stenotic breathing and the mechanism of its occurrence. It should be noted that with slight narrowing of the upper airways, the increase in the volume of pulmonary ventilation occurs due to an increase in the depth of breathing, and a sharper

stenosis is accompanied by a decrease in the depth of respiratory movements, which remain rare. In the explanation of the mechanism of stenotic breathing, note the delay of the Hering-Breuer reflex.

Identify 3 periods of asphyxia. Explain the mechanism of changes in breathing, blood circulation and heart activity during asphyxia. Explain the mechanism of terminal respiratory movements. Note the importance of maintaining cardiac activity after respiratory arrest.

Point out the importance of the research of domestic scientists in the development of the main issues of respiratory pathology (I.M. Sechenov, V.V. Pashutin, N.M. Syrotinin, etc.).

Formulation of conclusions from the experiment.

1. With a gradual narrowing of the upper respiratory tract (incomplete closure of the rabbit's nasal passages), an increase in the volume of pulmonary ventilation is observed due to an increase in the depth of breathing while simultaneously slowing it down (stenotic breathing). Mechanism: delay of the Hering-Breyer reflex and associated inhibition of inhalation.

2. With rapid narrowing of the upper respiratory tract (asphyxia), 3 periods of breathing disturbance are observed:

1) excitement, characterized by increased frequency and deepening of breathing with a predominance of the inhalation phase over the exhalation phase (inspiratory dyspnea);

2) depression, characterized by a decrease in the frequency and depth of breathing with difficult exhalation (expiratory dyspnea);

3) terminal, characterized by agonal or terminal breathing after short-term apnea (gasping breathing) and preservation of cardiac activity after stopping breathing.

Terminology:

- Intrapulmonary
- Extrapulmonary
- Hypoventilation
- Hyperventilation
- Perfusion
- Diffusion
- Asphyxia
- Dyspnea

Tasks for independent work on the topic "Pathophysiology of external respiration. Respiratory failure"

The student is invited to determine the causes of violations of the external respiration system and identify possible consequences. To be able to explain the mechanism of occurrence. Analysis of errors with an explanation of the correct answers

List of questions and works to be studied:

1. Definition of the concepts of "breathing", "external breathing", "cellular (tissue) breathing. Factors determining the effectiveness of the external breathing system.

2. Indicators of the functional state of the external breathing system (lung volumes and capacities) and their changes in the pathology of external breathing.

3. Respiratory insufficiency. Types. Causes. Typical disorders of external breathing.

4. Alveolar hypoventilation. Definition of the concept. The main reasons. Pathogenesis. Forms Manifestations

5. Alveolar hyperventilation. Reasons. Forms Manifestations

6. Impaired lung perfusion. Pulmonary hypertension: forms, main causes, pathogenesis. Pulmonary hypotension: forms, main causes, pathogenesis.

7. Violation of ventilation-perfusion ratios. Reasons, options.

8. Violation of the diffusion capacity of the lungs. Reasons. Mechanisms.

9. Manifestations of insufficiency of external breathing. Shortness of breath: types, mechanisms. Periodic breathing: types, mechanisms. Asphyxia: causes, mechanisms, types, mechanisms.

10. Features of the pathology of external breathing in children.

List of practical skills that must be mastered:

1. Explain the role of reflex effects and disorders of the function of the upper respiratory tract in the origin of shortness of breath.

2. Identify the main signs and manifestations of shortness of breath.

3. Explain the main mechanisms of their occurrence and development.

Situational tasks KROK-1 to determine the final level of knowledge

1. An unconscious young man with signs of morphine poisoning entered admission office. His respiration is shallow and infrequent which is caused by inhibition of respiratory centre. What type of respiratory failure is it?

A. Ventilative dysregulatory. *D. Perfusive.*
B. Ventilative obstructive. *E. Diffusive.*
C. Ventilative restrictive.

2. A patient with bronchial asthma has developed acute respiratory failure. What kind of respiratory failure occurs in this case?

A. Obstructive disturbance of alveolar ventilation. *D. Diffusion.*
B. Restrictive ventilatory defect. *E. Perfusion .*
C. Dysregulation of alveolar ventilation.

3. A 12 y.o. boy who suffers from bronchial asthma has an acute attack of asthma: evident expiratory dyspnea, skin pallor. What type of alveolar ventilation disturbance is it?

A. Restrictive. *C. Central.* *E. Obstructive.*
B. Throracodiaphragmatic. *D. Neuromuscular.*

4. A patient has a history of chronic obstructive bronchitis. Blood gas analysis revealed the development of hypoxemia and hypercapnia on the background of dyspnea, tachycardia and cyanosis. What disorder of external respiration is observed in the patient?

A. Hyperperfusion. *C. Hyperdiffusion.* *E. Hypoventilation.*
B. Hyperventilation. *D. Hypoperfusion.*

5. A patient with marked pneumofibrosis that developed after infiltrating pulmonary tuberculosis has been diagnosed with respiratory failure. What is its pathogenetic type?

A. Restrictive. *C. Obstructive.* *E. Apneustic.*
B. Dysregulatory. *D. Reflex.*

6. A patient with evident pneumosclerosis that developed after infiltrative pulmonary tuberculosis presents with respiratory failure. What is its pathogenetic type?

A. Restrictive. *C. Obstructive.* *E. Dysregulative.*
B. Apneustic. *D. Reflectory.*

7. A 50-year-old male patient suffers from chronic bronchitis, complains about dyspnea during physical activity, sustained cough with sputum. After examination he was diagnosed with pulmonary emphysema. This complication is caused by:

A. Decrease in lung compliance. *D. Decrease in lung elasticity.*
B. Decrease in lung perfusion. *E. Ventilation-perfusion disbalance.*
C. Decrease in alveolar ventilation.

8. Examination of a miner revealed pulmonary fibrosis accompanied by disturbance of alveolar ventilation. What is the main mechanism of this disturbance?

A. Constriction of superior respiratory tracts.
B. Disturbance of neural respiration control.
C. Limitation of breast mobility.
D. Limitation of respiratory surface of lungs.
E. Bronchi spasm.

9. A patient staying in the pulmonological department was diagnosed with pulmonary emphysema accompanied by reduced elasticity of pulmonary tissue. What type of respiration is observed?

A. Expiratory dyspnea. *D. Infrequent respiration.*
B. Inspiratory dyspnea. *E. Periodic respiration.*
C. Superficial respiration.

10. A 23-year-old patient has been admitted to a hospital with a craniocerebral injury. The patient is in a grave condition. Respiration is characterized by prolonged convulsive inspiration followed by a short expiration. What kind of respiration is it typical for?

A. Apneustic. *C. Kussmaul's.* *E. Biot's.*
B. Gasping breath. *D. Cheyne-Stokes.*

11. A 23 year patient was admitted to the hospital in grave condition with craniocerebral trauma. His respiration is characterized by a spasmodic long inspiration interrupted by a short expiration. What respiration type is it typical for?

A. Gasping.
B. Kussmaul's respiration.
C. Apneustic.
D. Cheyne-Stokes respiration.
E. Biot's respiration.

12. A 62-year-old patient was admitted to the neurological department due to cerebral haemorrhage. Condition is grave. There is observed progression of deepness and frequency of breathe that turnes into reduction to apnoea, and the cycle repeates. What respiration type has developed in the patient?

A. Kussmaul's respiration.
B. Biot's respiration.
C. Cheyne-Stockes respiration.
D. Gasping respiration.
E. Apneustic respiration.

13. A 62-year-old patient with cerebral haemorrhage was admitted to the neurological department in grave condition. Objectively: increase of respiration depth and rate with its following reduction to apnoea, thereafter respiration cycle restores. What respiration type is it?

A. Cheyne-Stokes.
B. Biot's.
C. Gasping.
D. Apneustic.
E. Kussmaul's.

14. A patient with a craniocerebral injury presents with respiration characterized by progressively deeper respiratory movements followed by a gradual decrease that results in a temporary stop in breathing. What pattern of abnormal respiration are these features typical for?

A. Cheyne-Stokes.
B. Biot's.
C. Kussmaul's.
D. Gasping.
E. Apneustic.

15. While having the dinner the child choked and aspirated the food. Meavy cough has started, skin and mucosa are cyanotic, rapid pulse, rear breathing, expiration is prolonged. What disorder of the external breathing developed in the child?

A. Stage of inspiratory dyspnea on asphyxia.
B. Stage of expiratory dyspnea on asphyxia.
C. Stenotic breathing.
D. Alternating breathing.
E. Biot's breathing.

16. A 30-year-old man has sustained an injury to his thorax in a traffic incident, which caused disruption of his external respiration. What type of ventilatory difficulty can be observed in the given case ?

A. Restrictive extrapulmonary ventilatory impairment.
B. Obstructive ventilatory impairment.
C. Impair ventilation regulation dysfunction.
D. Restrictive pulmonary ventilatory impairment.
E. Cardiovascular collaps.

Standards of correct answers to the situational tasks

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
A	*A*	*E*	*E*	*A*	*A*	*D*	*D*	*A*	*A*	*C*	*C*	*A*	*A*	*B*	*A*

Recommendations for registration of work results

1. Written answer to test tasks (basic level of knowledge).

2. The results of the experiment are drawn up in the form of an experiment protocol with the determination of relevant conclusions.

3. Protocol for solving situational tasks with an explanation of the correct answers (final level of knowledge).

LITERATURE

Main

1. Pathophysiology : textbook / N. V. Krishtal, V. A. Mikhnev, N. N. Zayko [et al.] ; ed.: N. V. Krishtal, V. A. Mikhnev. 2nd ed., corrected. Kyiv : AUS Medicine Publishing, 2018. 656 p.

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Substantial module 6.
Pathophysiology of the digestive system, liver and kidneys. Pathophysiology of the regulatory systems (endocrine, nervous). Pathophysiology of the extreme states

Topic № 11. Pathophysiology of the digestive system. Insufficiency of digestion

Number of hours: 2 academic hours

Relevance of the topic: One of the most important and earliest disorders of the functions of the stomach is a disorder of gastric secretion, which can develop as a result of a violation of neuroendocrine regulation, with pathological processes in the stomach, in other organs and systems, with various external influences on the body. Secretory disorders of the stomach are characterized by quantitative and qualitative changes. Knowledge of the basic regularities of gastric secretion disorders, quantitative and qualitative changes in gastric juice, at the same time, makes it possible to correctly carry out prevention and rational therapy of gastric secretory disorders. The main functions of the liver are the formation of bile secretion, which takes part in all types of metabolism, cholesterol secretion, maintenance of vascular tone and hematopoietic processes, and others. This is a pathological syndrome that can be observed in diseases of a therapeutic, infectious and surgical profile.

Purpose of the lesson:

General: to be able to determine and characterize the acidity of gastric juice in various disorders of gastric secretion, the essence of jaundice as a pathological syndrome, which is a consequence of impaired bile formation and bile secretion, to interpret the main laboratory parameters. Differential diagnosis of various types of jaundice. To be able to interpret the cholemic syndrome as a set of functional changes in the body in diseases of the liver and biliary tract, accompanied by mechanical or parenchymal jaundice.

Specifically:

Know:

1. Explain normal and pathological types of gastric secretion.
2. To analyze the mechanism of hypo- and hypersecretion, hypo- and hyperacidity. 3. Explain the effect of secretion disorders on the mechanism of gastric motor function disorders.
4. Use the knowledge of these mechanisms to correctly understand their role in the pathogenesis of diseases of the digestive tract.
5. Define the concept of jaundice as a pathological syndrome.
6. Classify jaundice taking into account their etiology and pathogenesis.
7. To justify the use of the used laboratory indicators, the study of which is necessary for the differential diagnosis of jaundice.

8. Carry out a qualitative determination of direct and indirect bilirubin in blood serum.

9. Conduct differential diagnosis of various types of jaundice, using knowledge of the mechanisms of biochemical disorders.

10. List the main symptoms of cholemic syndrome.

11. Explain the mechanism of emergence and development of the main manifestations of cholemic syndrome.

Be able to:

1. Describe the content and acidity of gastric juice.

2. Interpret and explain the mechanisms of gastric secretion regulation.

3. To analyze the mechanism of development of hypo- and hypersecretion.

4. To explain the effect of secretory disorders on the mechanism of disturbances in the motor function of the stomach.

5. To apply the received ideas about the mechanisms of digestive disorders in the stomach for a correct understanding of their role in the pathogenesis of diseases of the gastrointestinal tract (GI).

6. Visualize the anatomical and histological structure of the liver and biliary tract.

7. To know the main stages of the exchange of bile pigments in the human body.

8. Explain the main physiological mechanisms of the biliary function of the liver.

9. Characterize the processes of bile formation and bile secretion and the chemical composition of bile.

10. Explain the importance of bile in the digestion process.

Practical experience:

1. Define the terms digestion, digestive insufficiency, dyspepsia syndrome, dyspepsia.

2. Determine the main causes of taste disturbance, appetite, thirst. Types. Causes.

3. Find out what are the signs of the type of esophageal dysfunction, their causes.

4. Kokava reasons for the development of dyspepsia in the stomach. What is a violation of the secretory function of the stomach.

5. Explain what is a violation of the digestive function of the intestine.

6. Name the signs of malabsorption syndrome, the main causes and manifestations. Nonspecific ulcerative colitis. Causes, manifestations

7. Name the main pathological syndromes of liver damage, their biochemical markers.

8. Explain the consequences of functional liver failure.

9. Define the concept of jaundice. Types of jaundice. Hemolytic jaundice. Causes, pathogenesis and manifestations.

Technological map of students' work on the topic "Pathophysiology of the digestive system. Insufficiency of digestion"

No n\n	Stage lesson	Study time, min	Study Materials		Place holding a class
			learning Tools	Equipment	
1	Definition of initial knowledge	20	Control of theoretical training of students programmable by using structural answers to the issue of tickets	Test control issue tickets	Study room
2	Analysis of theoretical material	45	Analysis of theoretical material on the basis of control conduct by question topic	Control question topic	Study room
3	The experiment	5	Introduction to the formulation and preparation of the experiment. Setting experiment	Burettes, pipettes, erlenmeyerovskie cones, 1 % phenolphthalein alcohol solution, 0.5 % alcohol solution dimetilamid 0,1 N sodium hydroxide solution	Study room
4	The final stage of determining the level of knowledge and skills. Summing up.	20	Determining the source of formation of knowledge and skills	The decision of situational problems	Study room

The graphological structure of the topic "Pathophysiology of the digestive system. Insufficiency of digestion" is attached.

Material and methodological support of the topic "Pathophysiology of the digestive system. Insufficiency of digestion".

1. Lectures;
2. Methodical instructions for teachers;
3. Methodical instructions for students;
4. Set of test tasks to determine the basic level of knowledge;
5. Set of situational tasks to determine the final level of knowledge;
6. Set of KROK-1 tasks;
7. Set of schemes and tables (presentation);
8. Set of forms with a clinical blood test;
9. Video films;
10. For the experiment (experimental animals – rabbit, microscopes, immersion oil, subject slides, polish slides, hydrochloric acid solution, distillates for painting of smears, injector, pins, Petri-dish).

Lesson content

1. Disorders of mastication.

2. Disturbances in salivary secretion.
3. Hypersecretion.
4. Gastric motor function disorders.
5. Jaundice as a pathological syndrome.
6. Biochemical disorders types of jaundice.
7. Disorders cholemic syndrome.

Setting up the experiment. Discussion the results and formulation the conclusions.

Experiment 1 Determination of the acidity of gastric juice of patients with hypo-, hyper- and normo-secretion.

1. Fill burettes with 0.1 N caustic sodium solution.

2. Pour 5 ml of gastric juice into an Erlenmeyer flask with a pipette, add 1–2 drops of a 0.5 % alcoholic solution of dimethylamidoazobenzene and 1–2 drops of a 1 % alcoholic solution of phenolphthalein. The juice turns crimson.

3. Note the initial level of alkali in the burette and titrate the juice until the appearance of a brick color, which corresponds to the end of neutralization of free hydrochloric acid with caustic soda.

4. Determine the number of milliliters of caustic soda solution that was needed to neutralize free hydrochloric acid, and continue the titration until the appearance of a persistent pink color, which corresponds to the neutralization of all acids of the gastric juice with alkali, that is, the total acidity. Note once again the amount of spent caustic soda solution.

5. Calculate the total and free acidity of gastric juice in milliliters of caustic soda solution required for titration of 100 ml of gastric juice, as well as in mmol/l.

6. Determine the content of hydrochloric acid in gastric juice by multiplying the amount of free hydrochloric acid by 0.00365.

7. Using the obtained experimental data and applying knowledge of the theoretical material, formulate and record the conclusions of the conducted experiment. Enter the obtained results in the table.

- **Discussion of the results of the experiment**

With gastric hypersecretion, there is an increase in the amount of gastric juice both after eating and on an empty stomach, correspondingly, an increase in total acidity and the content of free hydrochloric acid in gastric juice.

- **Formulation the conclusions based on the experiment.**

Based on the experiment, it can be explained that gastric hypersecretion can be simulated in the experiment a) by stimulating mucosal receptors; b) a decrease in the inhibitory effect of the cerebral cortex on the vagus centers; c) electrical stimulation of the centers of the vagus nerve or its efferent fibers innervating the stomach; d) the introduction of histamine or pharmacological agents that stimulate its formation in the gastric mucosa.

Tasks for independent work on the topic "Pathophysiology of the digestive system. Insufficiency of digestion"

The student is invited to examine the results of the content of gastric juice to determine the violation of its formation in the gastric mucosa. It is necessary to determine the signs and type of violation. Be able to explain the mechanism of occurrence. Analysis of errors with an explanation of the correct answers.

Terminology:

- Hyperacidity
- Hypoacidity
- Jaundice
- Hepatitis
- Portal hypertension
- Malabsorption

Tasks for independent work on the topic "Pathophysiology of the digestive system. Insufficiency of digestion"

The student is invited to examine the results of the content of gastric juice to determine the violation of its formation in the gastric mucosa. It is necessary to determine the signs and type of violation. Be able to explain the mechanism of occurrence. Analysis of errors with an explanation of the correct answers.

List of questions and works to be studied:

1. Definition of the terms "digestion", "digestive insufficiency", "syndrome of insufficiency of digestion", "dyspepsia". Main reasons for insufficiency of digestion.
2. Violations of taste, appetite, feeling of thirst. Types. Reasons. Consequences.
3. Violation of digestion in oral cavities and violations of swallowing. Types. Reasons. Consequences.
4. Gullet dysfunction. Types. Reasons. Consequences.
5. Violations of digestion in a stomach. Disorders of secret, motor, absorptive, barrier and protective function of a stomach.
6. Violation of digestion in intestines. Disorders of the digesting, motor, barrier and protective function of intestines.
7. Stomach ulcer of a stomach and duodenum. Etiology. Pathogenesis. Manifestations. Complications.
8. Malabsorption syndrome. Main reasons and manifestations. Coeliacs disease. Nonspecific ulcer colitis. Reasons. Manifestations.
9. Violation of excretory function of a liver. Frustration of a bile production and bile excretion. Reasons. Manifestations.
10. Jaundice. Definition of concept. Types of jaundice. Haemolytic jaundice. Reasons. Pathogenesis. Manifestations.
11. Parenchymatous (hepatic) jaundice. Reasons. Pathogenesis. Manifestations.
12. Mechanical (subhepatic) jaundice. Reasons. Holemic syndrome. Pathogenesis. Manifestations.

13. Violation of haemodynamic function of a liver. Syndrome of portal hypertensia. Etiology. Pathogenesis. Manifestations.

14. Hepatic coma. Etiology. Pathogenesis.

List of practical skills that must be mastered:"Pathophysiology of the digestive system. Insufficiency of digestion"

1. Explain the types of gastric secretion, hypo- and hypersecretion, hypo- and hyperacidity.

2. Determine the mechanism of disruption of the motor function of the stomach, understanding their role in the pathogenesis of diseases of the digestive tract

3. Define the concept of jaundice as a pathological syndrome, classify jaundice according to their etiology and pathogenesis.

4. Justify the use of laboratory parameters, the study of which is necessary for the differential diagnosis of jaundice.

5. Explain the mechanism of occurrence and development of manifestations of cholemic syndrome. List the main symptoms of cholemic syndrome. List the main symptoms of cholemic syndrome.

6. Determine direct and indirect bilirubin in blood serum.

Situational tasks KROK-1 to determine the final level of knowledge

1. A 42-year-old patient complains of pain in the epigastral area, vomiting; vomit masses have the colour of "coffee-grounds", the patient has also melena. Anamnesis records gastric ulcer. Blood formula: erythrocytes – $2{,}8\times10^{12}$/l, leukocytes – 8×10^{9}/l, Hb – 90 g/l. What complication is it?

A. Pyloric stenosis. *C. Penetration.* *E. Perforation.*
B. Haemorrhage. *D. Canceration.*

2. During an acute experiment some of diluted solution of hydrochloric acid was injected into the duodenal cavity of an experimental animal. This will result in hypersecretion of the following hormone:

A. Motilin. *B. Histamine.* *C. Neurotensin.* *D. Secretin.* *E. Gastrin.*

3. A 57-year-old patient was admitted to the gastroenterological department with suspicion on Zollinger-Ellison syndrome because of rapid increase of gastrin level in the blood serum. What disorder of the secretory function of the stomach is the most likely?

A. Hyperacid hyposecretion. *D. Hypoacid hypersecretion.*
B. Achylia. *E. Hyperacid hypersecretion.*
C. Hypoacid hyposecretion.

4. Roentgenologically confirmed obstruction of common bile duct resulted in preventing bile from inflowing to the duodenum. What process is likely to be disturbed?

A. Fat emulgation. *D. Protein absorption.*
B. Hydrochloric acid secretion in stomach. *E. Salivation inhibition.*
C. Carbohydrate hydrolysis.

5. Hepatitis has led to the development of hepatic failure. Mechanism of edemata formation is activated by the impairment of the following liver function:

A. Barrier. *C. Antitoxic.* *E. Protein-synthetic.*
B. Chologenetic. *D. Glycogen-synthetic.*

6. A tooth extraction in a patient with chronic persistent hepatitis was complicated with prolonged hemorrhage. What is the reason for the haemorrhagic syndrome?

A. Decrease in thrombin production.
B. Fibrinolysis intensification.
C. Increase in thromboplastin production.
D. Increase in fibrinogen synthesis.
E. Decrease in fibrin production.

7. As a result of dysfunction of protein synthesis in liver a patient with hepatic insufficiency has disturbed synthesis of procoagulants, prothrombin, fibrinogen. Which of the listed syndromes can be expected in this patient?

A. Acholia syndrome. *D. Haemorrhagic.*
B. Portal haemorrhagic syndrome. *E. Cholaemia syndrome.*
C. Hepatolienal syndrome.

8. A patient who has been treated for viral hepatitis B developed symptoms of hepatic insufficiency. What changes indicating disorder in protein metabolism are likely to be observed in this case?

A. Absolute hyperalbuminemia. *D. Absolute hyperglobulinemia.*
B. Absolute hypoalbuminemia. *E. Protein rate in blood will stay unchanged.*
C. Absolute hyperfibrinogenemia.

9. A patient being treated for viral hepatitis type B got symptoms of hepatic insufficiency. What blood changes indicative of protein metabolism disorder will be observed in this case?

A. Absolute hyperalbuminemia. *D. Proteinic blood composition is unchanged.*
B. Absolute hyperfibrinogenemia.
C. Absolute hypoalbuminemia. *E. Absolute hyperglobulinemia.*

10. A patient presents with icteritiousness of skin, scleras and mucous membranes. Blood plasma the total bilirubin is increased, stercobilin is increased in feces, urobilin is increased in urine. What type of jaundice is it?

A. Haemolytic. *C. Parenchymatous.* *E. Cholestatic.*
B. Gilbert's disease. *D. Obturational.*

11. A patient with jaundice has high total bilirubin that is mainly indirect (unconjugated), high concentration of stercobilin in the stool and urine. The level of direct (conjugated) bilirubin in the blood plasma is normal. What kind of jaundice can you think of?

A. Parenchymal (hepatic). *D. Hemolytic.*
B. Mechanical. *E. Neonatal jaundice.*
C. Gilbert's disease.

12. Examination of a chemical plant worker who had had a poisoning revealed an increase in total bilirubin concentration at the expense of indirect fraction. Feces and urine are characterized by high stercobilin concentration. The level of direct bilirubin in blood plasma is normal. What type of jaundice is the case?

A. Mechanical. *C. Obstructive.* *E. Hemolytic.*
B. Parenchymatous. *D. Hepatic.*

13. A 53-year-old male patient complains of acute pain in the right hypochondrium. Objective examination revealed scleral icterus. Laboratory tests revealed increased ALT activity, and stercobilin was not detected in the stool. What disease is characterized by these symptoms?

A. Hemolytic jaundice. *C. Chronic colitis.* *E. Chronic gastritis.*
B. Hepatitis. *D. Cholelithiasis.*

14. An infectious disease unit admitted a patient with signs of jaundice caused by hepatitis virus. Select an indicator that is specific only for parenchymatous jaundice:

A. Increase in ALT and AST rate. *C. Bilirubinuria.* *E. Urobilinuria.*
B. Hyperbilirubinemia. *D. Cholaemia.*

15. A 48 y.o. patient was admitted to the hospital with complaints about weakness, irritability, sleep disturbance. Objectively: skin and scleras are yellow. In blood: conjugated bilirubin, cholalemia. Feces are acholic. Urine is of dark colour (bilirubin). What jaundice is it?

A. Hemolytic. *D. Gilbert's syndrome.*
B. Parenchymatous. *E. Crigler-Najjar syndrome.*
C. Mechanic.

16. Blood analysis of a patient with jaundice reveals conjugated bilirubinemia, increased concentration of bile acids. There is no stercobilinogen in urine. What type of jaundice is it?

A. Hepatocellular jaundice. *D. Hemolytic jaundice.*
B. Obstructive jaundice. *E. Cythemolytic jaundice.*
C. Parenchymatous jaundice.

17. A patient ill with jaundice has increased content of conjugated bilirubin and bile acids in blood, no stercobilinogen in urine. What jaundice are these symptoms typical for?

A. Hepatic. *B. Hepatocellular.* *C. Hemolytic.* *D. Obstructive.* *E. Cythemolytic.*

18. A patient with a pronounced icteritiousness of skin, sclera and mucous membranes has urine of dark beer colour and colourless feces. Direct bilirubin in blood is elevated, urine contains bilirubin. What type of jaundice is it?

A. Hemolytic. *C. Excretory.* *E. Obstructive.*
B. Parenchymatous. *D. Conjugation.*

19. A coprological survey revealed light-colored feces containing drops of neutral fat. The most likely reason for this condition is the disorder of:

A. Bile inflow into the bowel. *D. Intestinal absorption.*
B. Pancreatic juice secretion. *E. Gastric juice acidity.*
C. Intestinal juice secretion.

20. A 35-year-old man with peptic ulcer disease has undergone antrectomy. After the surgery secretion of the following gastrointestinal hormone will be disrupted the most:

A. Gastrin. *C. Secretin.* *E. Histamine.*
B. Cholecystokinin. *D. Neurotensin.*

21. A 43-year-old patient suffers from acute pancreatitis with disrupted common bile duct patency. What condition can develop in this case ?

A. Hepatocellular jaundice. *D. Hemolytic jaundice.*
B. Portal hypertension. *E. Hepatic coma.*
C. Mechanical jaundice.

22. A 50-year-old man, who has been suffering from chronic hepatic failure for several years, has developed ascites. What is the main mechanism of this disorder development ?

A. Decrease of albumin and globulin synthesis in liver.
B. Neurotoxins appearing in blood.
C. Increased pressure in portal vein system.
D. Increased content of low-density and very low-density lipoproteins in blood.
E. Increase of blood oncotic pressure.

23. A patient visited a dentist to extract a tooth. After the tooth had been extracted, bleeding from the tooth socket continued for 15 minutes. Anamnesis states that the patient suffers from active chronic hepatitis. What phenomenon can extend the time of hemorrhage ?

A. *Thrombocytopenia.*
B. Decrease of fibrinogen content in blood.
C. Decrease of albumine content in blood.
D. Increased activity of anticoagulation system.
E. Hypocalcemia.

Standards of correct answers to the task KROK-1

1	2	3	4	5	6	7	8	9	10	11	12	13
B	*D*	*E*	*A*	*E*	*A*	*D*	*B*	*A*	*A*	*D*	*E*	*D*

14	15	16	17	18	19	20	21	22	23
A	*C*	*B*	*D*	*E*	*A*	*A*	*C*	*C*	*B*

Recommendations for registration of work results

1. Written answer to test tasks (initial level of knowledge).
2. The results of the experiment are drawn up in the form of an experiment protocol with the determination of relevant conclusions.

3. The protocol of the experiment analysis of various forms of impaired vascular tone, in particular, hypertensive disease

4. Protocol for solving situational tasks with an explanation of the correct answers.

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Topic № 12. Pathophysiology of the kidneys. Renal failure

Number of hours: 2 academic hours.

Relevance of the topic: The kidneys are an actively functioning organ that takes an active part in the water-electrolyte and acid-base exchanges of the body. The main functions of the kidneys ensure the maintenance of the volume of circulating blood, ensure the constancy of the osmotic pressure of the blood, the piston concentration in the blood of certain ions, in particular hydrogen ions. The renin-angiotensin mechanism is involved in the regulation of blood pressure and maintenance of circulating blood volume, and erythropoietin stimulates the formation of red blood cells. The nephron is the structural unit of the kidneys and consists of a glomerulus in which blood is filtered and a tubular component. Arterial hypertension is usually an early manifestation of chronic renal failure. The kidneys are sensitive to circulatory disorders and lead to diseases of various etiologies that cause pathological processes and various clinical manifestations.

Purpose of the lesson:

General – to be able to characterize the main causes and mechanisms of urinary disorders and the urinary function of the kidneys, the pathogenesis of changes in diuresis and urine composition.

Specifically:

Know:

1. To characterize the main causes and pathogenetic mechanisms of impaired filtration, reabsorption and secretion, their manifestations.
2. Explain the mechanism of changes in the qualitative composition of urine.
3. Using biochemical methods of research, explain the pathological components of urine: protein, glucose, acetone, bilirubin to interpret their diagnostic value.

Be able:

1. Explain the mechanism of urine formation
2. To evaluate the main indicators characterizing urine production and the urinary function of the kidneys.
3. Determine the procedure for carrying out qualitative reactions on the content of protein, sugar, bile pigments, acetone in urine, as well as be able to determine the specific gravity of urine
4. Quantitative and qualitative changes in urine can be characterized in case of kidney damage.

Practical experience:

1. Explain the main functions of the kidneys. Causes of kidney disease.
2. Describe the violations of glomerular filtration, reabsorption, secretion.
3. Determine changes in the relative density and composition of urine.
4. Explain the causes and pathologies of the kidneys in which they are observed.

5. Analyze extrarenal manifestations of renal dysfunction. Types of kidney pathology by origin.

6. Give a classification and manifestations of acute glomerulonephritis.

7. Explain the causes of chronic glomerulonephritis.

8. Determine risk factors for pyelonephritis. Pathogenesis and manifestations.

9. Definition of the concept of "nephrotic syndrome".

10. Definition of the concept. kidney failure. Acute renal failure. Causes. Pathogenesis. manifestations

11. Criteria for the diagnosis of chronic renal failure.

12. Determination of the mechanism of uremia formation. The main factors of tissue and organ damage in uremia and renal coma.

13. Find out the basic principles of the **t**reatment of renal dysfunction. lusion about violation of the quantitative composition of "red" blood

Technological map of students' work on the topic "Pathophysiology of the kidneys. Renal failure"

No n\n	Stage lesson	Study time, min	Study Materials		Place holding a class
			Learning Tools	Equipment	
1	Definition of initial knowledge	10	Control of theoretical training of students programmable by using structural answers to the issue of tickets	Test control issue tickets.	Study room
2	Analysis of theoretical material	45	Analysis of theoretical material on the basis of control conduct by question topic	Control question topic.	Study room
3	The experiment	20	Introduction and preparation for setting up experiments. Setting experiment	Samples of patients, measuring cylinders, urometr, vidalevski tubes, Pasteur pipettes, concen-trated nitric acid, supports a set of chemical test tubes and pipettes, reagent Benedict microscope, subject and cover glass	Study room
4	The final stage of determining the level of knowledge and skills. Summing up	15	Determining the source of formation of knowledge and skills.	The decision of situational problems	Study room

The graphological structure of the topic "Pathophysiology of the kidneys. Renal failure" is attached.

Material and methodological support of the topic "Pathophysiology of the kidneys. Renal failure":

1. Lectures;
2. Methodical instructions for teachers;
3. Methodical instructions for students;
4. Set of test tasks to determine the basic level of knowledge;
5. Set of situational tasks to determine the final level of knowledge;
6. Set of KROK-1 tasks;
7. Set of schemes and tables (presentation);
8. Set of forms with a clinical blood test;
9. Video films;
10. For the experiment. Microscopes, immersion oil, glass slides, cover slip, ,Pasteur pipette, test strips (determination of sugar concentration in urine), hydrometer and hydrometer (determination of urine density), urometer (determination of specific gravity of urine), distillates for staining smears, injector, pins.

Lesson content:

1. Pathophysiology of kidneys
2. Disorders of function of kidneys
3. Types of pathology of kidneys by origin
4. Acute glomerulonephritis
5. Chronic glomerulonephritis
6. Pyelonephritis
7. Nephrotic syndrome
8. Renal failure
9. Uraemia
10. Nephrolithiasis

Setting up the experiment. Discussion the results and formulation the conclusions

The conduction of the experiment:

1. Experiment. 1. Investigation of the physical and chemical properties of urine and microscopy of urine sediment in case of impaired kidney function Determination of the specific gravity of urine: pour urine into a cylinder, then slowly immerse the urometer and note the number on the scale on the lower meniscus. The urometer should not come into contact with the walls of the cylinder.

2. Determination of protein: a reagent consisting of 1 volume of concentrated nitric acid and 4 volumes of a saturated solution of magnesium sulfate is poured into a narrow test tube. Carefully pour urine over the wall of the test tube so that it does not mix with the reagent. In the presence of protein in the urine, a grayish-white ring forms on the border of both liquids (Heller's test).

3. Determination of sugar: use a reagent containing sodium citric acid (17.3 %), sodium carbon dioxide 10 %, copper sulfate 1.73 %. Add 8 drops of urine to

5 ml of this reagent and boil for 1–2 minutes. Observe the color change during cooling. Pea-green color corresponds to 0.08–0.1 % sugar, brown-greenish 0.5 %, brown – 0.5–0.6 %, yellow – 1 %, red – more than 2 %. (Benedict's trial).

4. Microscopy of urine sediment: pour urine into centrifuge tubes and centrifuge for 10 minutes. Drain the urine, the sediment remains on the narrow bottom of the test tube. Take a drop of sediment with a Pasteur pipette, transfer it to a glass slide and cover it with a cover glass, while avoiding air getting between the glass and cover glass. The sediment should be examined under a microscope first under low magnification, then under high magnification, with a narrowed aperture and lowered illuminator. Pay attention to organized (cylinders, fat droplets, erythrocytes, leukocytes, epithelial cells, etc.) and unorganized (different forms of uric acid crystals, urates, triple phosphates, calcium sulfate, calcium carbonate, triple phosphate crystals in the form of coffin lids and etc.) elements in urine sediment.

5. Make sketches and explain in which pathological conditions the specified changes can be detected.

6. Using the obtained experimental data and applying knowledge of the theoretical material, formulate and write down the conclusions of the conducted experiment.

- **Discussion of the results of the experiment.**

The conduction of the experiment: Research of physical and chemical properties of urine and urinary sediment microscopy with impaired renal function. The specific gravity of urine, urine is poured into the cylinder, then slowly dip urometr and note the number on the scale at the bottom of the meniscus. Urometr should not collide with the walls of the cylinder. Definition of protein, poured into a narrow tube reagent consisting of 1 part concentrated nitric acid and 4 volumes of a saturated solution of magnesium sulfate. Carefully pour urine on the wall of the tube so as not mixed with the reagent. In the presence of protein in urine on the border of the two fluids produced grayish-white ring. Determination of sugar, using a reagent containing sodium citrate (17.3 %), sodium carbonate 10 %, 1.73 % copper sernokysluyu. To 5 ml of this reagent add 8 drops of urine and boil for 1-2 minutes. Watch for the color change cooling. Pea green color corresponds 0,08–0,1 % sugar, brown-greenish 0.5 %, brown – 0, 5–0.6 %, 1 %-yellow, red-bolee 2 %.

- **Formulation the conclusions based on the experiment**

On the basis of the experiment, it can be explained that with normally functioning kidneys, there are wide fluctuations in the specific gravity of urine and a change in its concentration during the day, which is associated with periodic intake of food, water and fluid loss by the body (sweating, breathing). The kidneys under various conditions can excrete urine with a relative density of 1.001 to 1.040. Microscopy of the urinary sediment allows you to determine

under the microscope the elements of the urinary sediment (cylinders, drops of fat, erythrocytes, leukocytes, epitheliocytes, etc.) and unorganized (various forms of uric acid crystals, urates, tripel phosphates, calcium sulfide, calcium carbonate, tripel phosphate crystals in grave covers and etc.).

Terminology:

• Tubular necrosis.
• Glomerulonephritis.
• Pyelonephritis.
• Nephrolithiasis.
• Uremia.

Tasks for independent work on the topic" Pathophysiology of the kidneys. Renal failure"

The student is invited to examine the results of a clinical analysis of the urine of a patient with impaired renal function. It is necessary to determine the signs and type of violation. Be able to explain the mechanism of occurrence. Analysis of errors with an explanation of the correct answers.

List of questions and works to be studied:

1. Main functions of kidneys. The reasons of pathology of kidneys (by the nature, an origin, level of primary realization of action).
2. Violations of a glomerular filtration, reabsorption, secretion. Reasons.
3. Frustration of a urine production and urination. Manifestations.
4. Changes of relative density and composition of urine. The **reasons** and pathology of kidneys at which they are observed.
5. Extrarenal manifestations of disorders of function of kidneys.
6. Types of pathology of kidneys by origin. Standard forms of pathology of kidneys.
7. Sharp glomerulonephritis. Etiology. Pathogenesis. Classification. Manifestations.
8. Chronic glomerulonephritis. Etiology. Pathogenesis. Classification. Manifestations.
9. Pyelonephritis. Etiology. Risk factors. Pathogenesis. Manifestations.
10. Nephrotic syndrome. Definition of concept. Reasons. Pathogenesis. Manifestations.
11. Renal failure. Definition of concept. Sharp renal failure. Reasons. Pathogenesis. Manifestations. Criteria of diagnostics.
12. Chronic renal failure. Etiology. Pathogenesis. Manifestations.
13. Chronic Illness of Kidneys (CIK). Definition of concept. Modern criteria of CIK. Classification. Markers of injury of kidneys (laboratory, visual). Assessment of function of kidneys.
14. Uraemia. Definition of concept. Reasons. Major factors of damage of tissues and bodies at uraemia and a kidney coma.

15. Nephrolithiasis, urolithiasis. Definition of concepts. Reasons, conditions and mechanisms of development. Consequences.

16. Principles of treatment of disorders of functions of kidneys.

List of practical skills that must be mastered:

1. Explain what are the main causes and types of kidney pathology.

2. Find out the pathogenetic mechanisms of filtration, reabsorption and secretion disorders, their manifestations.

3. Determine the main mechanisms that underlie the violations of the formation and excretion of urine.

4. Explain what types of kidney pathology exist and how they affect the change in the qualitative composition of urine.

5. Justify the main factors that can cause an increase in glomerular filtration?

6. Identify the main methods by which kidney function can be assessed.

7. Explain what causes hematuria, leukocyturia, cylindruria?

8 Determine the most characteristic symptoms of pyelonephritis.

Situational tasks KROK-1 to determine the final level of knowledge

1. A man has a considerable decrease in diuresis as a result of 1,5 l blood loss. The primary cause of such diuresis disorder is the hypersecretion of the following hormone:

A. Parathormone. *C. Vasopressin.* *E. Natriuretic.*
B. Corticotropin. *D. Cortisol.*

2. Shock and signs of acute renal failure (ARF) developed in the patient due to permanent injury. What is the leading cause of development of ARF in the case?

A. Urine excretion violation.
B. Increased pressure in the nephron capsule.
C. Decreased arterial pressure.
D. Increased pressure in the renal arteries.
E. Decreased oncotic BP.

3. A child has an acute renal failure. What biochemical factor found in saliva can confirm this diagnosis?

A. Increase in urea concentration.
B. Increase in concentration of higher fatty acids.
C. Decrease in nucleic acid concentration.
D. Increase in glucose concentration.
E. Decrease in glucose concentration.

4. On the 6th day of treatment a patient with acute renal insufficiency developed polyuria. Diuresis intensification at the beginning of polyuria stage of acute renal insufficiency is caused by:

A. Volume expansion of circulating blood.
B. Renewal of filtration in nephrons.
C. Growth of natriuretic factor.

D. Reduction of aldosterone content in plasma.
E. Reduction of vasopressin content in plasma.

5. Diabetic nephropathy with uremia has developed in a patient with pancreatic diabetes. The velocity of glomerular filtration is 9 ml/min. What mechanism of a decrease in glomerular filtration velocity and chronic renal failure development is most likely in the case of this patient?

A. Decrease in systemic arterial pressure.
B. Obstruction of nephron tubules with hyaline casts.
C. Tissue acidosis.
D. Arteriolar spasm.
E. Reduction of active nephron mass.

6. A patient with a history of chronic glomerulonephritis presents with azotemia, oliguria, hypo- and isosthenuria, proteinuria. What is the leading factor in the pathogenesis of these symptoms development under chronic renal failure?

A. Intensification of glomerular filtration.
B. Tubular hyposecretion.
C. Disturbed permeability of glomerular membranes.
D. Mass decrease of active nephrons.
E. Intensification of sodium reabsorption.

7. Injection of an anaesthetic before the tooth extraction resulted in development of anaphylactic shock accompanied by oliguria. What pathogenetic mechanism caused a decrease in diuresis in this case?

A. Decrease in hydrostatic pressure in the renal corpuscle capillaries.
B. Increase in hydrostatic pressure in the Bowman's capsule.
C. Damage of glomerular filter.
D. Increase in vasopressin secretion.
E. Increase in oncotic pressure of blood plasma.

8. A driver who got a trauma in a road accident and is shocked has reduction of daily urinary output down to 300 ml. What is the main pathogenetic factor of such diuresis change?

A. Drop of oncotic blood pressure.
B. Drop of arterial pressure.
C. Increased vascular permeability.
D. Decreased number of functioning glomerules.
E. Secondary hyperaldosteronism.

9. A patient with massive burns developed acute renal insufficiency characterized by a significant and rapid deceleration of glomerular filtration. What is the mechanism of its development?

A. Damage of glomerular filter.
B. Reduction of renal blood flow.
C. Reduction of functioning nephron number.

D. Rise of pressure of tubular fluid.
E. Renal artery embolism.

10. Due to the use of poor-quality measles vaccine for preventive vaccination, a 1-year-old child developed an autoimmune renal injury. The urine was found to contain macromolecular proteins. What process of urine formation was disturbed?

A. Filtration.
B. Secretion and filtration.
C. Reabsorption.
D. Reabsorption and secretion.
E. Secretion.

11. Chronic glomerulonephritis was diagnosed in a 34-year-old patient 3 years ago. Edema has developed in the last 6 months. What caused it?

A. Hyperproduction of vasopressin.
B. Disorder of albuminous kidneys function.
C. Proteinuria.
D. Hyperosmolarity of plasma.
E. Hyperaldosteronism.

12. Two weeks after lacunar tonsillitis a 20-year-old man started complaining about general weakness, lower eyelid edemata. After examination the patient was diagnosed with acute glomerulonephritis. What are the most likely pathological changes in the urine formula?

A. Cylindruria.
B. Presence of fresh erythrocytes.
C. Pyuria.
D. Natriuria.
E. Proteinuria.

13. A patient suffering from glomerulonephritis was found to have anasarca, AP of 185/105 mm Hg, anaemia, leukocytosis, hyperazotemia, hypoproteinemia. What factor indicates that glomerulonephritis has been complicated by the nephrotic syndrome?

A. Hypoproteinemia.
B. Anaemia.
C. Arterial hypertension.
D. Hyperazotemia.
E. Leukocytosis.

14. A patient with primary nephrotic syndrome has the following content of whole protein: 40 g/l. What factor caused hypoproteinemia?

A. Transition of protein from vessels to tissues.
B. Proteinuria.
C. Reduced protein synthesis in liver.
D. Increased proteolysis.
E. Disturbance of intestinal protein absorption.

15. A patient with nephrotic syndrome has massive edemata of his face and limbs. What is the leading pathogenetic mechanism of edemata development?

A. Drop of oncotic blood pressure.
B. Increase of vascular permeability.
C. Rise of hydrodynamic blood pressure.
D. Lymphostasis.
E. Increase of lymph outflow.

16. A 30 year old woman has face edemata. Examination revealed proteinuria (5,87 g/l), hypoproteinemia, dysproteinemia, hyperlipidemia. What condition is the set of these symptoms typical for?

A. Nephritic syndrome. *D. Acute renal failure.*
B. Chronic pyelonephritis. *E. Chronic renal failure.*
C. Nephrotic syndrome.

17. Violation of safety rules resulted in calomel intoxication. Two days later the daily diuresis was 620 ml. A patient experienced headache, vomiting, convulsions, dyspnea, moist rales in lungs. What pathology is it?

A. Chronic renal insufficiency. *D. Acute renal insufficiency.*
B. Uraemic coma. *E. Pyelonephritis.*
C. Glomerulonephritis.

18. 14 days after quinsy a 15-year-old child presented with morning facial swelling, high blood pressure, "meat slops" urine. Immunohistological study of a renal biopsy sample revealed deposition of immune complexes on the basement membranes of the capillaries and in the glomerular mesangium. What disease developed in the patient?

A. Necrotizing nephrosis. *D. Lipoid nephrosis.*
B. Acute interstitial nephritis. *E. Acute glomerulonephritis.*
C. Acute pyelonephritis.

19. A patient has insufficient blood supply to the kidneys, which has caused the development of pressor effect due to constriction of arterial resistance vessels. This condition results from the vessels being strongly affected by the following substance:

A. Norepinephrine. *C. Renin.* *E. Angiotensinogen.*
B. Angiotensin II. *D. Catecholamines.*

20. Poisoning caused by mercury (II) chloride (corrosive sublimate) occurred in the result of safety rules violation. In 2 days the patient's diurnal diuresis became 620 ml. The patient developed headache, vomiting, convulsions, dyspnea; moist crackles are observed in the lungs. Name this pathology:

A. Acute renal failure. *C. Glomerulonephritis.* *E. Pyelonephritis.*
B. Chronic renal failure. *D. Uremic coma.*

Standards of correct answers to the task KROK-1

1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**
C	*C*	*A*	*B*	*E*	*D*	*A*	*B*	*B*	*A*	*C*	*E*	*A*

14	**15**	**16**	**17**	**18**	**19**	**20**
B	*A*	*C*	*D*	*E*	*C*	*A*

Recommendations for registration of work results

1. Written answer to test tasks (initial level of knowledge).
2. The results of the experiment are drawn up in the form of an experiment protocol with the determination of relevant conclusions.

3. Protocol for the study of the results of the patient's clinical blood analysis.

4. Protocol for solving situational tasks with an explanation of the correct answers.

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Main

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Topic № 13. Pathophysiology of the endocrine system

Number of hours: 2 academic hours.

Relevance of the topic: The endocrine system of the human body is responsible for changes in anatomically, histologically and cytologically differentiated structures that produce hormones. Certain endocrine cells are synthesized in endocrine glands, or endocrine glands. These include the pituitary gland, the pineal gland, the thyroid gland, the parathyroid glands, and the adrenal glands. Changes in the activity of the endocrine system occur in response to any physiological or pathological stimuli. The great role of hormones in the pathology of the pituitary gland and adrenal glands. As regulators, they participate in the pathogenesis of diseases and pathological processes. With hypo- or hyperfunction, numerous changes are possible due to the pathology of the endocrine glands, which are important in the non-specific resistance of the body.

Purpose of the lesson:

General – to know the main mechanisms of disruption of the functional activity of the endocrine glands, the importance of the hypothalamic-pituitary-adrenal system in the non-specific resistance of the body and its disorders, manifestations of hypo- or hyperfunction of these glands.

Specifically:

Know:

1. To characterize the endocrine system, to determine its place in the system of functional interaction of a whole organism
2. To interpret the principles of regulation of the functional activity of endocrine glands.
3. Describe the pituitary and parahypophyseal regulatory pathway and the principle of feedback.
4. Explain the physiological role of various adrenal hormones in the body.

Be able:

1. Describe the principles of regulation of the functional activity of endocrine glands.
2. Describe pituitary and parahypophyseal regulatory pathways and principles of feedback.
3. Explain the physiological role of various adrenal hormones in the body.

Practical experience:

1. Determine examples of modern hormone classification principles that are most relevant for practical use?
2. Explain the mechanisms of regulation of endocrine disorders.
3. Determine what principles of regulation underlie endocrine functions.
4. Substantiate the main reasons for the violation of hormone biosynthesis.
5. Explain what can cause a violation of hormone secretion.

6. Determine the main mechanism of hormone transport in the body, as well as endocrine dysfunction.

7. Explain the importance of the hypothalamus in the regulation of endocrine functions?

8. Describe what is the essence of the endocrine function of the pineal gland and what can be its violations?

Technological map of students' work on the topic "Pathophysiology of the endocrine system"

No n\n	Stage lesson	Study time, min	Study Materials		Place holding a class
			Learning Tools	Equipment	
1	Definition basic level of knowledge	10	Control of theoretical training of students programmable by using structural answers to the issue of tickets	Test control issue tickets	Study room
2.	Analysis of theoretical material	35	Analysis of theoretical material on the basis of control conduct by question topic	Situational tasks, tasks KROK-1	Study room
3	Practical part (conduct experiment)	30	Practical part (conduct experiment) 30 min. Introduction and preparation for productions experiment Setting experiment Discussion results e	Experimental animals are rats. Microscope, immersion oil, tripod, Blood smears stained according to Romanivskyi-Giemzi.	Study room
4	Determination of the final level of knowledge and skills. Beating results	15	Determining the source of formation of knowledge and skill	The decision of situational problems, tasks KROK-1	Study room

The graphological structure of the topic"Pathophysiology of the endocrine system" is attached.

Material and methodological support of the topic"Pathophysiology of the endocrine system":

1. Lectures;
2. Methodical instructions for teachers;
3. Methodical instructions for students;
4. Set of test tasks to determine the basic level of knowledge;
5. Set of situational tasks to determine the final level of knowledge;
6. Set of KROK-1 tasks;
7. Set of schemes and tables (presentation);
8. Set of forms with a clinical blood test;
9. Video films;
10. Experimental part of the lesson.

11. For the experiment. For an experiment. Experimental animals – rats, microscope, tripod, glass slide, cover glass, test strips, Pasteur pipettes, blood smears stained according to Romanivsky-Giemz, smear distillate, injector, needles.

Lesson content:

1. Endocrine disorders
2. Pathology of the pituitary gland
3. Hypothalamic disease
4. Hyperthyroidism
5. Myxedema
6. Primary hyperparathyroidism
7. Secondary hyperparathyroidism
8. Hypoparathyroidism
9. Addison disease
10. Cushing syndrome
11. Thymus gland pathology

Setting up the experiment. Discussion the results and formulation the conclusions.

The conduction of the experiment: Study of the functional state of the adrenal cortex by counting the number of eosinophils in peripheral blood under the influence of a stressor on the rat body.

To study the functional state of the adrenal cortex, it is necessary to determine the reaction of the stress factor on the rat's body. The number of eosinophils in the peripheral blood of a rat is determined with the help of the leukocyte formula, which is carried out by immersion microscopy of smears stained according to Romanivskyi-Giemz. Compare the number of eosinophils in the blood of a rat. Describe in detail and draw the slides.

- **Discussion of the results of the experiment.**

The experimental rat showed a significant decrease in the number of eosinophils.

Make sketches and explain under what pathological conditions these changes can be detected. Using the obtained experimental data and applying the knowledge of theoretical material, formulate and write down the conclusions of the experiment.

- **Formulation the conclusions based on the experiment.**

Based on the conducted experiment, it can be concluded that 1. A significant decrease in the number of eosinophils is found in the experimental rat. 2. In the development of the observed eosinopenia, the leading importance belongs to the breakdown of eosinophils under the influence of an excess of glucocorticoids. Make sketches and explain under which pathological conditions these changes can be detected.

Terminology:

- Hypopituitarism
- Hyperpituitarism

- Hyperthyroidis
- Hypothyroidism
- Thyrotoxicosis
- Hypoparathyroidism
- Aldosteronism

Tasks for independent work on the topic "Pathophysiology of the endocrine system."

The student is invited to investigate laboratory tests, blood sugar level, biochemical blood test (with a complete lipid profile, determine the level of cholesterol, including high-density lipids, low-density lipids, triglyceride levels). When examining the concentration of hormones, it is recommended to conduct a blood test, determine the concentration of glycosylated hemoglobin, and conduct a glucose tolerance test. It is necessary to determine the signs and type of hormonal disorders, to be able to explain the mechanism of occurrence.

List of questions and works to be studied:

1. Endocrine system. Definition of the concept. Hormones, their main functions. Variants of the effect of hormones on target cells. Causes of endocrine disorders.
2. Pathogenetic mechanisms of endocrine disorders.
3. Disorders of the hypothalamic-pituitary system.
4. Hypo- and hypopituitarism. Reasons. Kinds Pathogenesis. Manifestations
5. Neurohypophysis pathology. Diabetes insipidus. Pathogenesis. Manifestations.
6. Typical forms of pathology of the adrenal glands.
7. Hyper- and hypofunctional states of the adrenal glands. Reasons. Pathogenesis.
8. Acute insufficiency of the adrenal cortex. Reasons. Manifestations
9. Typical forms of thyroid pathology. Assessment of thyroid status.
10. Hyper- and hypothyroidism. Kinds Reasons. Pathogenesis. Manifestations.
11. Mechanisms of calcium and phosphorus homeostasis regulation.
12. Typical forms of parathyroid gland pathology.
13. Pathophysiology of gonads.

List of practical skills that must be mastered:

1.Determine the main causes of endocrine disorders.

2. Explain the most typical forms of disorders of the hypothalamic-pituitary regulation.

3. Justify the classification of the pathogenesis of hypo- and hyperpituitarism.

4. Determine the pathogenesis and manifestations of the pathology of the neurohypophysis.

5. Explain the causes of the main most typical forms of adrenal pathology.

6. Explain the modern classification of acute adrenal insufficiency.

7. Perform an assessment of the condition in a typical form of thyroid disease.

8. Substantiate examples of evaluation of the mechanisms of development of endocrine disorders.

Situational tasks KROK-1 to determine the final level of knowledge

1. A patient complains of hydruria (7 liters per day) and polydipsia. Examination reveals no disorders of carbohydrate metabolism. These abnormalities might be caused by the dysfunction of the following endocrine gland:

A. Adenohypophysis. *D. Islets of Langerhans (pancreatic islets).*
B. Neurohypophysis. *E. Adrenal medulla.*
C. Adrenal cortex.

2. A woman after labor lost 20 kg of body weight, her hair and teeth fall out, she has muscle atrophy (hypophysial cachexia). Synthesis of what hypophysis hormone is disturbed?

A. Somatotropic. *C. Thyreotropic.* *E. Prolactin.*
B. Corticotrophic. *D. Gonadotropic.*

3. Examination of a patient revealed enlargement of some body parts (jaw, nose, ears, feet, hands), but body proportions were conserved. It might be caused by intensified secretion of the following hormone:

A. Somatostatin. *C. Somatotropin.* *E. Cortisol.*
B. Tetraiodothyronine. *D. Triiodothyronine.*

4. Examination of a 42 year old patient revealed a tumour of adenohypophysis. Objectively: the patient's weight is 117 kg, he has moon-like hyperemic face, red-blue striae of skin distension on his belly. Osteoporosis and muscle dystrophy are present. AP is 210/140 mm Hg. What is the most probable diagnosis?

A. Cushing's disease. *D. Diabetes mellitus.*
B. Cushing's syndrome. *E. Essential hypertension.*
C. Conn's disease.

5. A 38-year-old female patient complains of general weakness, cardiac pain, increased appetite, no menstruation. Objectively: the height is 166 cm, weight 108 kg, the patient has moon-shaped face, subcutaneous fat is deposited mainly in the upper body, torso and hips. There are also blood-red streaks. Ps – 62/min, AP – 160/105 mm Hg. Which of the following diseases is the described pattern of obesity most typical for?

A. Alimentary obesity. *D. Cushing pituitary basophilism.*
B. Myxedema. *E. Babinski-Frohlich syndrome.*
C. Insulinoma.

6. A 46-year-old patient suffering from the diffuse toxic goiter underwent resection of the thyroid gland. After the surgery the patient presents with appetite loss, dyspepsia, increased neuromuscular excitement. The body weight remained unchanged. Body temperature is normal. Which of the following has caused such a condition in this patient?

A. Increased production of thyroxin.
B. Reduced production of parathormone.
C. Increased production of calcitonin.

D. Increased production of thyroliberin.
E. Reduced production of thyroxin.

7. A 5-month-old boy was hospitalized for tonic convulsions. He has a life-time history of this disease. Examination revealed coarse hair, thinned and fragile nails, pale and dry skin. In blood: calcium – 1,5 millimole/l, phosphor – 1,9 millimole/l. These changes are associated with:

A. Hypoaldosteronism. *C. Hypothyroidism.* *E. Hypoparathyroidism.*
B. Hyperparathyroidism. *D. Hyperaldosteronism.*

8. A child has abnormal formation of tooth enamel and dentin as a result of low concentration of calcium ions in blood. Such abnormalities might be caused by deficiency of the following hormone:

A. Thyroxi. *D. Somatotropic hormone.*
B. Triiodothyronine. *E. Thyrocalcitonin.*
C. Parathormone.

9. A patient is followed up in an endocrinological dispensary on account of hyperthyreosis. Weight loss, tachycardia, finger tremor are accompanied by hypoxia symptoms – headache, fatigue, eye flicker. What mechanism of thyroid hormones action underlies the development of hypoxia?

A. Inhibition of respiratory ferment synthesis.
B. Disjunction, oxydation and phosphorylation.
C. Competitive inhibition of respiratory ferments.
D. Intensification of respiratory ferment synthesis.
E. Specific binding of active centres of respiratory ferments.

10. A 45-year-old woman has been diagnosed with endemic goiter. What mechanism has caused hyperplasia of thyroid gland in this patient?

A. Increased thyroxine production.
B. Increased thyrotropin production.
C. Increased catecholamine production.
D. Increased hydration of derma and hypodermic cellulose.
E. Increased iodine absorption.

11. A patient from Prykarpattia (at the foot of the Carpathian mountains) with endemic goiter consulted a doctor about suppuration of gingival angles and loosening of teeth. What is a major factor of periodontitis development in this case?

A. Endocrine disorders. *C. Violation of swallowing.* *E. Malnutrition.*
B. Hypersalivation. *D. Stress effects.*

12. A 29-year-old female patient has moon face, upper body obesity, striae on her anterior abdominal wall, hirsutism; urine shows an increased rate of 17-oxy ketosteroids. What disease are these presentations typical for?

A. Itsenko-Cushing syndrome. *D. Conn's syndrome.*
B. Secondary aldosteronism. *E. Pheochromocytoma.*
C. Primary aldosteronism.

13. A 44 year old woman complains of general weakness, heart pain, significant increase of body weight. Objectively: moon face, hirsutism, AP is 165/100 mm Hg, height – 164 cm, weight – 103 kg; the fat is mostly accumulated on her neck, thoracic girdle, belly. What is the main pathogenetic mechanism of obesity?

A. Reduced production of thyroid hormones.
B. Increased mineralocorticoid production.
C. Increased insulin production.
D. Reduced glucagon production.
E. Increased production of glucocorticoids.

14. To prevent the transplant rejection after organ transplantation it is required to administer hormonotherapy for the purpose of immunosuppression. What hormones are used for this purpose?

A. Mineralocorticoids. *C. Catecholamines.* *E. Thyroid.*
B. Sexual hormones. *D. Glucocorticoids.*

15. A 41-year-old male patient has a history of recurrent attacks of heartbeats (paroxysms), profuse sweating, headaches. Examination revealed hypertension, hyperglycemia, increased basal metabolic rate, and tachycardia. These clinical presentations are typical for the following adrenal pathology:

A. Hypofunction of the medulla. *D. Hypofunction of the adrenal cortex.*
B. Hyperfunction of the medulla. *E. Primary aldosteronism.*
C. Hyperfunction of the adrenal cortex.

16. A patient suffering from pheochromocytoma complains of thirst, dry mouth, hunger. Blood test for sugar revealed hyperglycemia. What type of hyperglycemia is it?

A. Hypercorticoid. *C. Adrenal.* *E. Hypoinsulinemic.*
B. Alimentary. *D. Somatotropic.*

17. A girl is diagnosed with adrenogenital syndrome (pseudohermaphroditism). This pathology was caused by hypersecretion of the following adrenal hormone:

A. Androgen. *C. Aldosterone.* *E. Adrenalin.*
B. . Estrogen *D. . Cortisol*

18. A female patient presents with endocrine dysfunction of follicular cells of the ovarian follicles resulting from an inflammation. The synthesis of the following hormone will be inhibited:

A. Lutropin. *C. Follicle stimulating hormone.* *E. Estrogen.*
B. Follistatine. *D. Progesterone.*

19. A 40 year old man who took part in disaster-management at a nuclear power plant fell sick with paradontitis. What etiological agent is the most important for the development of this pathology?

A. Iron deficit. *D. Increased load of dentoalveolar apparatus.*
B. Emotional stress. *E. Streptococcus.*
C. Malnutrition.

20. In course of an experiment a white rat was being stimulated with a stress factor (electric current). The researchers could observe muscle hypotonia, arterial hypotension, hypothermia. What period of general adaptation syndrome is it?

A. Exhaustion stage. *C. Antishock phase.* *E. –.*

B. Shock phase. *D. Resistance stage.*

21. Rats being under stress have muscular hypertonia and high arterial pressure, high glucose concentration in blood and intensified secretion of corticotropin and corticosteroids. In what stress phase are these animals?

A. Exhaustion. *C. Erectile.* *E. Terminal .*

B. Shock phase. *D. Antishock phase.*

22. A 41-year-old man has a history of recurrent attacks of heartbeats (paroxysms), profuse sweating, headaches. Examination revealed hypertension, hyperglycemia, increased basal metabolic rate, and tachycardia. These clinical presentations are typical for the following adrenal pathology:

A. Hypofunction of the medulla. *D. Primary aldosteronism.*

B. Hyperfunction of the adrenal cortex. *E. Hyperfunction of the medulla.*

C. Hypofunction of the adrenal cortex.

Standards of correct answers to the task КROK-1

1	2	3	4	5	6	7	8	9	10	11	12	13
B	*A*	*C*	*A*	*D*	*B*	*E*	*C*	*B*	*B*	*A*	*A*	*E*

14	15	16	17	18	19	20	21	22
D	*B*	*C*	*A*	*E*	*B*	*B*	*D*	*E*

Recommendations for registration of work results

1. Written answer to test tasks (initial level of knowledge).

2. The results of the experiment are drawn up in the form of an experiment protocol with the determination of relevant conclusions.

3. The protocol of the experiment analysis of various forms of impaired vascular tone, in particular, hypertensive disease

4. Protocol for solving situational tasks with an explanation of the correct answers.

LITERATURE

Main

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Topic № 14. Pathophysiology of the nervous system

Number of hours: 2 academic hours.

Relevance of the topic: The nervous system controls the functions of all organs and systems, ensuring perfect adaptation of the body to the environment. The central nervous system refers to the brain and spinal cord. The peripheral nervous system includes the cervical, thoracic, lumbar, and sacral nerve trunks that run from the spine to the extremities. The autonomic nervous system controls involuntary functions in the body such as sweating, blood pressure, heart rate, or heartbeat. Diseases of the nervous system can cause a violation of the physiological system of the body.

Purpose of the lesson:

General – be able to describe disorders of higher nervous (conditional reflex) activity, motor and other functions of the nervous system, explain their causes, main forms and manifestations. The general goal is to be able to describe disorders of higher nervous activity, motor and other functions of the nervous system, to explain their causes, main forms and manifestations.

Specifically:

Know:

1. Describe the etiology and pathogenesis of disorders of higher nervous activity and neuroses.
2. Classify disorders of the motor function of the nervous system.
3. Describe the concepts of "hyperkinesis", "paralysis and paresis", "ataxia".
4. Explain the causes and mechanisms of hyperkinesis, paralysis and ataxia.
5. To reveal the main features and manifestations of disorders of the motor function of the nervous system in hyperkinesis, ataxia and paralysis, to explain the mechanism of their development.
6. Distinguish between central and peripheral paralysis.

Be able:

1. Describe the structure and functions of different departments of the central nervous system.
2. Explain the motor function of the nervous system, by which nervous structures it is carried out. What is a motor analyzer?
3. Describe the main motor nerve pathways
4. To assess the influence of the extrapyramidal system and the cerebellum on the body's motor function.
5. To evaluate the structural organization of connections of the spinal cord and other departments of the central nervous system

Practical experience:

1. Explain what is the main role of the nervous system and its main functions.
2. Justify the main risk factors for damage to the nervous system and the degree of its dysfunction.

3. Identify typical examples of various forms of disorders of the nervous system.

4. Explain the pathogenesis of neuronal dysfunctions and systemic pathological manifestations.

5. Justify the classification and main types of sensory disorders.

6. Substantiate the main mechanism of formation and development of pain, its causes and types of pain.

7. Explain the pathogenesis of the development of neurosis

Technological map of students' work on the topic "Pathophysiology of the nervous system"

No n\n	Stage lesson	Study time, min	Study Materials		Place holding a class
			Learning Tools	Equipment	
1	Definition of initial knowledge	10	Control of theoretical training of students programmable by using structural answers to the issue of tickets	Test control issue tickets	Study room
2	Analysis of theoretical material	35	Parsing theoretical material on based on control questions of the topic, situational tasks, tasks KROK-1	Control topic question, task KROK-1 situational tasks	Study room
3	Practical part (carrying out experiment)	30	Introduction and preparation for productions experiment Setting experiment Discussion results experiment and formulation of conclusions	10 % camphor solution, 1 % sodium amytal solution, syringes, injection needles; electric bell Experimental animals – white rats	
4	Determination of the final level of knowledge and skills. Beating results.	15	Determining the source of formation of knowledge and skills	Determination of the initial level of formation of knowledge and skills.	Study room

The graphological structure of the topic "Pathophysiology of the nervous system" is attached.

Material and methodological support of the topic "Pathophysiology of the nervous system":

1. Lectures;
2. Methodical instructions for teachers;
3. Methodical instructions for students;
4. Set of test tasks to determine the basic level of knowledge;
5. Set of situational tasks to determine the final level of knowledge;
6. Set of KROK-1 tasks;
7. Set of schemes and tables (presentation);
8. Set of forms with a clinical blood test;
9. Video films;

10. For the experiment (experimental animals – rabbit, microscopes, immersion oil, slides, polishing slides, distillates for staining smears, fixation, Romanovsky staining, injector, pins, Petri dish, 10 % camphor solution, 1 % amytal solution – sodium, syringes, injection needles, electric bell.

Lesson content:

1. Diseases of the nervous system
2. Sensory disorders
3. Cerebrovascular diseases
4. Encephalopathy
5. Extrapyramidal disorders
6. Cerebellar disorders
7. Cranial neuropathies

Setting up the experiment. Discussion the results and formulation the conclusions

The conduction of the experiment:

Reproduction of camphor epilepsy.

1. Inject 0.5 ml of 10 % camphor solution into the rat's abdominal cavity and observe the animal's condition.

2. Inject sodium amytal at the rate of 20 mg per 100 g of body weight under the skin of another rat.

3. After 10–15 minutes, when the animal becomes narcosis, inject 0.5 ml of 10 % camphor solution and observe the animal's condition.

4. Compare the condition of rats.

Reproduction of epilepsy caused by a strong audiogenic stimulus.

1. Place two previously selected rats with high audiogenic sensitivity in the cage.

2. After studying the initial state, turn on a strong electric bell for 3–5 minutes.

3. To note the phasic nature of the epileptoid response to a sound stimulus.

Study of the effect of sodium amytal on the convulsive state

1. Use rats from previous experience.

2. Inject sodium amytal at the rate of 15 mg per 100 g of body weight under the skin of one of the animals.

3. After 10 minutes, subject both animals to a sound stimulus according to the above method.

4. Describe the differences in condition

- **Discussion of the results of the experiment**

For the first time, reproduction of conditioned reflexes to irritation of the central nervous system by toxic substances was obtained by Podkopaev (1914 – apomorphine) and V. A. Krylov. (1922 – morphine). The experience spent in this session proves that any indifferent stimulus can acquire the meaning of a disease-causing agent due to the formation of pathological conditional connections. This experience expands our ideas about the etiology of human diseases. In addition, this experience testifies to the powerful effect of the bark through the

subcortical centers, on the vegetative processes in the body, and the possibility of the occurrence of various vegetative disorders when cortical activity changes.

- **Formulation the conclusions based on the experiment**

1. An epileptic attack caused by the intra-abdominal injection of 0.5 ml of a 10 % camphor solution as an audiogenic stimulus manifested itself in the form of successive phases of harbingers, tonic convulsions and post-seizure catalepsy. The mechanism of the epileptic action of camphor: the direct effect of camphor on brain tissue and the flow of impulses from the peritoneum (from the site of initial injection) and from vascular receptors, which forms many foci of epileptic activity in the brain, which are accompanied by a violation of the functional balance of intracerebral systems and the development of the function of neurons in the focus of epileptic activity.

2. The phasic nature of an episeizure is caused by a sequential change in the bioelectric potentials of the brain: synchronous high-amplitude potentials (phase of tonic seizures) and the development of inhibition in the central nervous system (phase of post-seizure catalepsy).

3. In experimental rats, which were previously injected with sodium amytal (amobarbital), the action of camphor and a sound stimulus did not cause the development of a convulsive syndrome, which was due to the inhibitory effect of sodium amytal on the central nervous system, preventing the formation of foci of epileptic activity in the brain. Describe and draw the slides in detail.

Terminology:

- Hyperkinesia
- Hypokinesia
- Parakinesia
- Paralysis
- Convultion
- Paraplegia
- Tetraplegia
- Monoplegia
- Dissociation
- Hemiplegia
- Phantom pain
- Causalgia

Tasks for independent work on the topic "Pathophysiology of the nervous system".

The student is invited to familiarize himself with the results of the study of the pathological effect of rats on an epileptic attack caused by the intra-abdominal injection of a solution of 0.5 ml of a 10 % solution of camphor. The mechanism of the epileptic effect of camphor: the direct effect of camphor on the brain tissue and the flow of impulses. It is necessary to determine the signs

and type of disorder. Be able to explain the mechanism of occurrence. Analysis of errors with an explanation of the correct answers. In experimental rats that were previously injected with sodium amytal (amobarbital), the action of camphor and a sound stimulus did not cause the development of a convulsive syndrome.

List of questions and works to be studied:

1. In contrast to physiological pain, what is pathological pain characterized.
2. What properties characterize epicritic pain.
3. Specify the features of protopathic pain.
4. What are the characteristic manifestations of central paralysis in the affected limbs.
5. What signs characterize peripheral paralysis.
6. State the signs of Brown-Sécart syndrome.
7. What is the characteristic development of syndromes for neuroses.
8. Specify the characteristic features of neurosis in a person.

List of practical skills that must be mastered:

1. Explain the method of experimental modulation of disturbances in motor functions of the nervous system.
2. Identify examples of the main syndromes that characterize disorders of the motor function of the nervous system.
3. Substantiate the causes and mechanisms of development of neuromuscular transmission disorders.
4. Explain the most effective methods used for experimental modeling of disorders of the autonomic nervous system.
5. Explain the causes and mechanisms of violations of electrophysiological processes in the central nervous system.
6. Determine the main mechanisms for the development of pathological excitation and inhibition in the nerve centers.
7. Formulate functional changes in the CNS that may be associated with the development of pathological excitation and inhibition.

8 Determine the mechanism of damage to brain neurons.

9. Explain the cause of intracranial hypertension and stroke.

Situational tasks KROK-1 to determine the final level of knowledge

1. A patient caught a cold after which there appeared facial expression disorder. He cannot close his eyes, raise his eyebrows, bare his teeth. What nerve is damaged?

A. Facial. *C. Trigeminus.* *E. Infraorbital.*
B. Vagus. *D. Glossopharyngeal.*

2. A 68-year-old woman can't move by the upper and lower right extremities due to insult. Muscle tone of these extremities and reflexes are increased. There are pathological reflexes. What form of the paralysis is it?

A. Paraplegia. *C. Monoplegia.* *E. Hemiplegia.*
B. Tetraplegia. *D. Dissociation.*

3. After a car accident a patient has been diagnosed with a fracture of spine. He is unable to move his lower extremities. This form of motor disorder is called:

A. Paralysis. *C. Hemiplegia.* *E. Quadriplegia.*
B. Paresis. *D. Paraplegia.*

4. After poisoning with an unknown drug a 37 year old patient has stereotypical face muscle contractions that imitate blinking and squinting. What form of motor function disorder of nervous system is it?

A. Hyperkinesia. *B. Hypokinesia.* *C. Akinesia.* *D. Ataxy.* *E. –.*

5. An experimental rat with extremity paralysis has no tendon and cutaneous reflexes, muscle tone is decreased, but muscles of the affected extremity maintain their ability to react with excitation to the direct action of continuous current. What type of paralysis is it?

A. Flaccid central. *C. Spastic peripheral.* *E. Extrapyramidal.*
B. Flaccid peripheral. *D. Spastic central.*

6. A patient complaining of pain in the left shoulder-blade region has been diagnosed with myocardial infarction. What kind of pain does the patient have?

A. Visceral. *B. Phantom.* *C. Protopathic.* *D. Radiating.* *E. Epicritic.*

7. After the traumatic tooth extraction a patient is complaining of acute, dull, poorly-localized pain in gingiva, body temperature rise up to 37,5 °C. The patient has been diagnosed with alveolitis. Specify the kind of pain in this patient:

A. Epicritic. *B. Visceral.* *C. Heterotopic.* *D. Protopathic.* *E. Phantom.*

8. A patient got a gunshot wound of hip which damaged the sciatic nerve. Any impact on the affected limb causes severe, excruciating pain. What mechanism of pain is most likely in this case?

A. Reflex. *D. Enkephalin hypofunction .*
B. Phantom. *E. Causalgic.*
C. Endorphin hypofunction.

9. Four months ago a 43 year old patient had a traumatic amputation of his lower extremity. Now he complains of sensing the amputated extremity and having constantly grave, sometimes unbearable pain in it. What type of pain does he have?

A. Causalgia. *C. Neuralgia.* *E. Reflex.*
B. Phantom. *D. Thalamic.*

10. A patient complains of toothache. On examination he has been diagnosed with pulpitis. Which factor played a main pathogenic role in the development of pain syndrome in this case?

A. Interleukin action.
B. Vasospasm.
C. Inadequate stimulation of a mandibular nerve branch.
D. Activation of one of the components of the complement system.
E. Increased intratissular pressure in the dental pulp.

11. A 28 year old man had a gunshot wound of shin that resulted in an ulcer from the side of the injury. What is the main factor of neurodystrophy pathogenesis in this case?

A. Traumatization of peripheral nerve.
B. Psychical stress.
C. Microcirculation disturbance.
D. Infection .
E. Tissue damage.

12. A patient who takes a blocker of membrane cytoreceptors of efferent conductor synapses of autonomic nervous system complains about dry mouth. What receptors are blocked?

A. Nicotinic cholinoreceptors.
B. H_2-receptors.
C. Alpha-adrenoreceptors.
D. Muscarinic cholinoreceptors.
E. Deta-adrenoreceptors.

Standards of correct answers to the task КROК-1

1	2	3	4	5	6	7	8	9	10	11	12
A	*E*	*D*	*A*	*B*	*D*	*D*	*E*	*B*	*E*	*A*	*D*

Recommendations for registration of work results

1. Written answer to test tasks (initial level of knowledge).
2. The results of the experiment are drawn up in the form of an experiment protocol with the determination of relevant conclusions.
3. Protocol for the study of the results of the patient's clinical blood analysis.
4. Protocol for solving situational tasks with an explanation of the correct answers.

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Topic № 15. Pathophysiology of extreme conditions: collapse, shock, coma. Pathophysiology of modern warfare injuries

Number of hours: 2 academic hours.

Relevance of the topic: Throughout life a person is exposed to the influence of various exogenous and endogenous factors of extreme strength, duration, or an unusual nature. The action of extreme factors leads to the development of either adaptation to this factor, or an extreme (critical, urgent) state. Extreme conditions are leading to development of severe states of the body that develop under the influence of extreme factors and are characterized by significant disorders of the body's vital functions, often leading to death. The most common and clinically significant extreme states include collapse, shock, and coma.

Modern warfare injuries are more severe than peacetime polytrauma, due to the difference in damaging factors, conditions of injury, timing of assistance to the victims, mass lesions. Thus, it is extremely important to study the pathological mechanisms of development of such pathological conditions as crunch syndrome, burn and traumatic disease, shock, coma, as well as wounded diseases of internal organs. Special role in the pathogenesis of internal pathology belongs to infection process of wounds, posthemorrhagic anemia, changes in the general reactivity under affection of the body by combat environment (overfatigue, nervous tension, cooling, etc.).

Purpose of the lesson:

General - to study the main types of extreme conditions, their causes and mechanisms of their development, pathogenetic methods of treatment.

Specifically:

Know:

1. Concepts of "extreme states", "terminal state"; their criteria.
2. Types of extreme states, their features.
3. Collapse: definition, classification, etiology, pathogenesis, clinical manifestations.
4. Shock: definition, classification, etiology, pathogenesis, clinical manifestations.
5. Violations of hemodynamics and microcirculation in the pathogenesis of shock states.
6. Coma: definition, classification, etiology, pathogenesis, clinical manifestations.
7. Etiology and pathogenesis of hepatic, uremic, diabetic coma.
8. Principles of treatment of extreme conditions.
9. Definition, classification, pathogenesis of traumatic disease, burn disease, tranche syndrome; basic principles of their treatment.

Be able to:

1. Define the concept of "extreme states" and differentiate their individual types – collapse, shock, coma.

2. Explain the causes, conditions and mechanisms of the development of collapse, shock, coma in order to develop in clinical practice the ability to give a correct assessment of these phenomena.

3. Violations of hemodynamics and microcirculation in the pathogenesis of shock states.

4. Be able to explain the etiology and pathogenesis of various types of coma (hepatic, uremic, diabetic).

Technological map of students work on the topic topic "Pathophysiology of extreme states: collapse, shock, coma. Pathophysiology of modern combat trauma"

No	Stage lesson	Academic time, min	Educational guide		Place holding a class
			Educational tools	Equipment	
1	Definition of the initial level of knowledge	10	Written answer to test tasks	Test cards	Study room
2	Analysis theoretical material	35	Analysis of theoretical material is carried out on the basis of control questions of the topic and "Krok-1" tasks	Control questions of the topic, "Krok-1" tasks	
3	Conducting an experiment	30	Introduction and preparation for setting up the experiment Setting up the experiment	Electrocardioscope, microscope, scissors, tweezers, clamps, napkins, planks with a hole for fixing the animal, hexanal	
4	The final stage of determining the level of knowledge and skills. Summing up.	15	Determination of the final level of formation of knowledge and skills	"Krok-1" tasks, situational tasks	

The graphological structure of the topic "Pathophysiology of extreme states: collapse, shock, coma. Pathophysiology of modern combat trauma" is attached.

Material and methodical support of the topic "Pathophysiology of extreme states: collapse, shock, coma. Pathophysiology of modern combat trauma":

1. Lectures;
2. Methodical developments for teachers;
3. Methodical instructions for students;
4. A set of test tasks to determine the basic level of knowledge;
5. A set of situational problems to determine the final level of knowledge;
6. A set of "KROK-1" tasks;
7. A set of diagrams and tables (presentation);
8. Video films.
9. Experiment equipment: vector electrocardioscope, microscope, scissors, tweezers, clamps, napkins, capitals with a hole (viewing window) for fixing the animal, hexanal for anesthesia; the experimental animals are white rats.

Lesson content:

1. Extreme conditions overview
2. Collapse
3. Shock
4. Comatosis states
5. Pathophysiology of modern combat trauma
6. Special types of coma during hostilities
7. Recirculation syndrome
8. Traumatic disease
9. Burn shock

Setting up the experiment. Discussion of results and formulation of conclusions.

Study of microcirculation disorders and heart rhythm during reproduction of hemorrhagic collapse.

1. The rat is previously anesthetized by intraperitoneal injection of hexenal at a dose of 10 mg/kg.
2. The rat is fixed in a supine position on a table with an opening (viewing window).
3. The abdominal cavity of the rat is opened on the lateral surface of the abdomen.
4. The small intestine is pulled out and the mesentery is fan-shaped above the inspection window (by pricking the intestine with pins).
5. Microcirculation vessels are looked for under the minimum magnification of the microscope and the movement of blood in the initial state is observed.
6. At the same time, the electrodes of the electrocardioscope vector are placed on the paws of the rat in standard leads and the nature of cardiac activity according to the data of the electrocardiogram (ECG) is observed on the oscilloscope screen.
7. Then the femoral artery is opened with scissors and acute blood loss is caused (blood loss is determined by the difference in the weight of the napkins that drain the place where the femoral artery is crossed).
8. Violations of hemodynamics in microvessels are observed.
9. At the same time, we register ECG changes on the oscilloscope.
10. The data are entered into the protocol and analyzed.

Discussion of the results of the experiment.

Collapse is a general condition that occurs as a result of a significant discrepancy in the circulating blood volume capacity of the vascular bed and is characterized by insufficient blood circulation, primary circulatory hypoxia, a disorder of the functions of tissues, organs and their systems. The direct cause of the collapse is the rapid development of a significant excess of the capacity of the vascular bed in comparison with circulating blood volume.

Hypovolemic collapse – collapse that develops when the circulating blood volume decreases due to: acute massive bleeding; rapid and significant dehydration of the body; loss of a large volume of blood plasma; redistribution of blood with deposition of a significant amount of it in venous vessels (shock).

Posthemorrhagic collapse – one of the types of hypovolemic collapse. The initial pathogenetic factor is a rapid and significant decrease in circulating blood volume (hypovolemia). The increase in vascular tone does not eliminate the discrepancy in their capacity, which significantly decreased circulating blood volume. As a result, hypoperfusion of organs and tissues develops, which leads to the development of first circulatory, and then mixed (hemic, tissue) hypoxia. The consequence of hypoxia is a progressive disorder of the functions of the central nervous system, lungs, kidneys, etc., which led to the cessation of cardiac activity and the death of the animal.

Formulation of conclusions from the experiment.

1. In the experiment, after acute blood loss, a rat developed signs of a microcirculation disorder (slowing down of blood flow, pendulum-like oscillation, stasis in individual microcirculation vessels) and at the same time rhythm disturbances were registered on the ECG (at first tachycardia, then bradycardia and fibrillation followed by cardiac arrest).
2. All the mentioned violations indicated the development of posthemorrhagic (from etiology) hypovolemic (from pathogenesis).
3. Disturbance of the cardiac system function is the initial and main pathogenetic link of collapse and is characterized by inadequate blood supply to organs and tissues.

Terminology:

- Shock
- Collapse
- Coma
- Extreme state
- Terminal state
- Combat trauma
- Warfare injury

Tasks for independent work on the topic "Pathophysiology of extreme conditions: collapse, shock, coma. Pathophysiology of modern warfare injuries".

It is necessary to characterize the different extreme states, such as shock, collapse and coma, mechanisms of their development, to explain the adaptive mechanisms of the organism developing in such conditions. Be able to explain the mechanisms of occurrence warfare injury and combat trauma. Analyze the errors with an explanation of the correct answers.

List of questions and works to be studied:

1.Extreme conditions. Similarities and differences of extreme and terminal states.
2. Etiology and pathogenesis of extreme conditions.

3. Collapse. Risk factors. Types. Reasons.
4. Peculiarities of the pathogenesis of various types of collapse.
5. General manifestations of collapse.
6. Shock. Reasons. Risk factors. Types of shock.
7. Pathogenesis and stages of shock. Clinical manifestations of shock
8. Coma. Changes and suppression of consciousness.
9. Causes of coma.
10. Classification of commas.
11. Pathogenesis of comatose states.
12. Clinical manifestations, course and exit from comatose states (hepatic, uremic, diabetic).
13. Principles of treatment of extreme conditions
14. Definition, classification, pathogenesis of traumatic disease, burn disease, trench syndrome; basic principles of their treatment.

List of practical skills that need to be explained:

1. Concepts of "extreme states", "terminal state"; their criteria.
2. Collapse: definition, classification, etiology, pathogenesis, clinical manifestations.
3. Shock: definition, classification, etiology, pathogenesis, clinical manifestations.
4. Coma: definition, classification, etiology, pathogenesis, clinical manifestations.
5. Etiology and pathogenesis of uremia, hepatic, hypoglycemic and diabetic tumors.
6. Combat trauma: definition, classification, etiology, pathogenesis, clinical manifestations.
7. Warfare injury: definition, classification, etiology, pathogenesis, clinical manifestations.

List of practical skills that must be mastered:

1. Define the concept of "extreme states" and differentiate their individual types - collapse, shock, coma;
2. Explain the causes, conditions and mechanisms of the development of collapse, shock, coma, in order to develop in clinical practice the ability to give a correct assessment of these phenomena.
3. Violations of hemodynamics and microcirculation in the pathogenesis of shock states.
4. Be able to explain the etiology and pathogenesis of various comas (hepatic, uremic, diabetic).

Situational tasks KROK-1 to determine the final level of knowledge

1. Patient with diabetes didn't get insulin injection in time that caused hyperglycemic coma (glucose in the blood 50 mmol/L). What mechanism is prevalent in the development of the coma?

A. Hyperosmia. *C. Hypoxia.* *E. Acidosis.*
B. Hypokaliemia. *D. Hyponatremia.*

2. A 12-year-old teenager has significantly put off weight within 3 months; glucose concentration rose up to 50 millimole/l. He fell into a coma. What is the main mechanism of its development?

A. Hyperosmolar. *D. Lactacidemic.*
B. Hypoglycemic. *E. Hypoxic.*
C. Ketonemic.

3. An experimental rat got intra-abdominal injection of 10 ml of 40 % glucose solution. 60 minutes later the rat passed into a comatose state as a result of dehydratation. What is the mechanism of development of this state?

A. Rise of osmotic pressure of extracellular fluid.
B. Rise of oncotic pressure of extracellular fluid.
C. Reduction of vasopressin secretion.
D. Loss of salts and water.
E. Acid-base disbalance.

4. An unconscious patient had been delivered to a hospital by the ambulance. Objectively: absent reflexes, occasional convulsions, irregular breathing. After a laboratory examination he was diagnosed with hepatic coma. What metabolite accumulation is essential for the development of the central nervous system disorders?

A. Ammonia. *C. Glutamine.* *E. Histamine.*
B. Urea. *D. Bilirubin.*

5. A 62-year-old patient has been hospitalized due to massive cerebral hemorrhage. Blood pressure is 70/30 mm Hg, heart rate is 120/min., respiratory rate is 4/min., unconscious, no response to external stimuli. Such condition can be determined as:

A. Collapse. *C. Shock.* *E. Stress.*
B. Coma. *D. Agony.*

6. A patient suffers from posttraumatic hemorrhage that resulted in development of hemorrhagic shock. What volume of circulating blood was lost by the patient?

A. 25–40 %. *C. 50–75 %.* *E. 3–20 %* .
B. 40–50 %. *D. 12–25 %.*

7. A victim of a traffic accident was received by the intensive care unit. The patient is in a grave condition that can be characterized as a severe pathologic process that leads to exhaustion of vital functions and puts the patient into the marginal state between life and death due to critical reduction of capillary circulation in the affected organs. The patient is in the state of:

A. Shock. *C. Preagony.* *E. Agony.*
B. Collapse. *D. Coma.*

8. The doctor started the absence of respiration and cardiac activity in a traffic accident victim. This condition lasts for 1 minute already. This clinical presentation corresponds with the following terminal state:

A. Traumatic shock, torpid phase.

B. Clinical death.

C. Agony.

D. Traumatic shock, erectile phase.

E. Preagony.

Standards of correct answers to the task КROК-1

1	2	3	4	5	6	7	8
A	*A*	*A*	*A*	*B*	*A*	*A*	*B*

Recommendations for registration of work results.

1. Written answer to test tasks (basic level of knowledge).

2. The results of the experiment are drawn up in the form of an experiment protocol with the determination of relevant conclusions.

3. Protocol for solving situational tasks with an explanation of the correct answers.

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Subject plan

Навчальне видання

ПАТОФІЗІОЛОГІЯ ОРГАНІВ ТА СИСТЕМ

Методичні рекомендації для викладачів
щодо підготовки до практичних занять студентів
(спеціальність «Медицина» та «Стоматологія»)

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