Ministry of Education and Science of Ukraine
V. N. Karazin Kharkiv National University

PATHOPHYSIOLOGY

Methodical guidance
for the 3rd academic year students of the School of Medicine

Electronic resource

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Reviewers:

I. V. Sorokina – PhD, Sc.D., MD, Professor of the Pathological Anatomy Department of the Kharkiv National Medical University, Kharkiv, Ukraine;

S. O. Sherstiuk – PhD, Sc.D., MD, Professor, Head of the Department of Human Anatomy and Physiology, School of Medicine, V. N. Karazin Kharkiv National University, Kharkiv, Ukraine.

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Pathophysiology : methodical guidance for the 3rd academic year students of the School
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Methodical guidance contains basic recommendations for preparing third-year medical students for classes, tests and exams in the discipline “Pathophysiology”. Developed for applicants for higher medical education in higher medical educational institutions of Ukraine III-IV levels of accreditation: specialty «222 Medicine», field of knowledge «22 Health Care», professional qualification «Second (Master of Medicine)», qualification «Doctor».

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CONTENTS

GENERAL DESCRIPTION
OF THE «PATHOPHYSIOLOGY»DISCIPLINE

The «Pathophysiology» discipline occupies a very important place in the educational program of the medical faculty, because it studies the causes and mechanisms of physiological disorders that cause disease or arise as a result of it.

The *purpose* of the «Pathophysiology» discipline is the formation of students' ability to interpret the basic concepts of general nosology; interpret the causes, mechanisms of development and manifestations of typical pathological processes and the most common diseases; analyze and draw conclusions about the causes and mechanisms of functional, metabolic and structural disorders of organs and systems in diseases; to provide fundamental training and acquisition of practical skills for the subsequent professional activity of a doctor.

The knowledge that applicants receive from the «Pathophysiology» discipline is basic for a whole block of disciplines that provide scientific and professional training.

According to the National Qualifications Framework and the educational and professional program of the V. N. Karazin Kharkiv National University School of Medicine, ***Pathophysiology provides graduates with following competencies***:

General:

- ✓ Ability for abstract thinking, analysis and synthesis; ability to learn and master modern knowledge (3K01).
- ✓ Ability to apply knowledge in practical situations (3K02).
- ✓ Knowledge and understanding of the subject area and understanding of professional activity (3K03).
- ✓ Ability to adapt and act in a new situation (3K04).
- ✓ Ability to make informed decisions; team work; interpersonal skills (3K05).

- ✓ Skills in the use of information and communication technologies; ability to search, process and analyze information from various sources (ЗК07).
- ✓ Definiteness and persistence in terms of tasks and responsibilities (ЗК08).
- ✓ Ability to act socially responsible and consciously (ЗК09).

Professional:

- ✓ Ability to determine the required list of laboratory and instrumental studies and evaluate their results (ФК2).
- ✓ Ability to determine the principles and characteristic of the treatment of diseases (ФК5).
- ✓ Skills to perform medical manipulations (ФК9).
- ✓ Ability to keep medical records (ФК14).
- ✓ Ability to assess the impact of the environmental, socio-economic and biological determinants on the health of the individual, family, population (ФК16).

According to the National Qualifications Framework and the educational and professional program of V. N. Karazin Kharkiv National University School of Medicine, ***applicants who have completed the educational course «Pathophysiology», must to***:

- ✓ Have general and special fundamental and professionally-oriented knowledge, skills, abilities, competencies necessary to perform typical professional tasks related to activities in the medical field in the relevant position (ПРН 1).
- ✓ Have knowledge of human psychophysiological characteristics, human health, health support, disease prevention, human treatment, public health (ПРН 2).
- ✓ Apply the acquired knowledge, skills and understanding to solve typical problems of physician's activities, the scope applying of which is provided by the lists of syndromes and symptoms, diseases, emergencies, laboratory and instrumental studies, medical manipulations (ПРН 3).

- ✓ Determine the tactics of physiological childbirth and the postpartum period (ПРН 14).
- ✓ Apply the acquired knowledge about the existing health care system to optimize their own professional activities and participate in solving practical problems of the healthcare domain (ПРН 20).
- ✓ Adhere to the code of ethics of the physician, which ensures the formation of a specialist with appropriate personal qualities (ПРН 21).

TEACHING METHODS

Several classical and modern pedagogical approaches are used in teaching the discipline:

1. «Teacher-centered approach» or «direct instruction model» — conveying information to higher education students through lectures and instruction on learning.
2. «Student-centered approach» and «personalized learning», namely students' considerable freedom and independence in choosing topics for individual tasks and expressing their own opinions during its implementation.
3. «High-tech approach», namely the use of Web technologies to support access of students to the materials of the discipline, communication with the teacher and communication with other participants in the educational process.
4. Differentiated approach to methods of memorizing information, for in process of studying the material, students use both passive (reading and taking MCQ tests, viewing the presentations and videos) and active (answering the provided questions for self-check or control tests with written answers) methods of memorization.

THEMATIC PLAN OF THE DISCIPLINE

The discipline program is divided into two sections:

Section I. General pathophysiology.

Section II. Systemic pathophysiology.

The total amount of credits and hours
of the «Pathophysiology» discipline

Structural sections of the discipline	Number of hours				ECTS credits	Year and semester
	Lectures	Practical	ISW	Total		
1. General pathophysiology	20	34	36	90	3	3 year, V semester
2. Systemic pathophysiology	18	36	36	90	3	3 year, V,VI semester

Notes: 1 ECTS credit – 30 study hours; classroom load – 60%; ISW – 40%; average weekly load – 6 hours per week (including ISW); average classroom weekly load – 3.6 hours per week.

SECTION I. General pathophysiology

TOPIC №1. Subject and tasks of Pathophysiology. The main stages of the Pathophysiology development. Methods of pathophysiological research. Basic concepts of Pathophysiology. Cell pathology.

Equipment facilities: projector, educational video materials, clinical cases, MCQs.

Theory questions for self-control:

1. Causes of cell injury.
2. Mechanisms of cell injury.
3. ATP deficiency as a mechanism of cell injury.
4. Disruption of calcium homeostasis as a mechanism of cell injury.

5. Damage to mitochondria as a mechanism of cell injury.
6. Oxidative stress.
7. Non-enzymatic and enzymatic antioxidants. Mechanism of action.
8. Pathological effects of free radicals.
9. DNA damage as a mechanism of cell damage.
10. Necrosis: causes and outcome.
11. Apoptosis: causes, mechanism and outcome.

***Assignment questions* (ISW)**:

1. List the pathologic effects on the human fetus of in utero exposure to ionizing radiation, and discuss these in terms of dosage and timing of radiation required.
2. Discuss the various biochemical consequences of single gene defects.
3. Discuss the possible influence on oogenesis and spermatogenesis of maternal and paternal exposure to toxic agents.
4. Discuss radiation injury in terms of: sources of radiation, molecular effects and cellular effects.
5. Compare and contrast the free radical-induced and chemical-induced types of cell injury in terms of biochemical and molecular mechanisms.
6. Discuss the pathogenetic mechanisms that are crucial in the production of mutations.
7. Describe the pathogenesis of adverse effects of the microwave radiation, electromagnetic fields and ultrasound.
8. Compare rubella infection and thalidomide ingestion in pregnant women in terms of developmental effects on the embryo and fetus.
9. Describe the pathogenesis and morphological features of the embryopathies associated with ingestion of the alcohol and hydantoin during pregnancy.
10. Identify factors which influence the type and extent of congenital anomalies produced by teratogenic agents.

11. Discuss the pathogenetic mechanisms that are crucial in the production of acquired congenital anomalies.

12. Discuss the pathogenetic mechanisms that are crucial in the production of nondisjunction.

13. Discuss the usefulness of the following laboratory tests in regard to genetic disorders and congenital malformations: amniotic fluid analysis, tissue culture, buccal smear, and chromosomal analysis.

14. Compare and contrast the free radical-induced and reperfusion-induced types of cell injury in terms of biochemical and molecular mechanisms.

15. Discuss the mechanism of formation and use of chromatin (Barr) body identification in the recognition and diagnosis of chromosome disorders.

16. Compare chromosome analysis (karyotyping) and Barr body count (buccal smear) in terms of appropriateness in various types of clinical situations and accuracy.

17. Discuss the more common chromosomal abnormalities in terms of pathogenesis, classification and specific features.

18. Compare translocation and mosaic types of Down syndrome on the basis of karyotype, maternal factors and inheritance.

19. Compare pseudo- and true hermaphroditism on the basis of genetic and gonadal morphology.

20. Discuss the significance of intracellular accumulations of lipids, proteins, glycogen and pigments (exogenous and endogenous).

TOPIC №2. Disorders of acid-base balance.

Equipment facilities: projector, educational video materials, clinical cases, MCQs.

Theory questions for self-control:

1. Normal indices and mechanism of blood pH level control.
2. Buffer systems that regulate blood pH.
3. Mechanism and biological role of bicarbonate reabsorption.

4. Mechanism and biological role of ammoniogenesis.

5. Compensation of acid-base balance disorders by the kidneys.

6. Compensation of acid-base balance disorders by lungs.

7. Respiratory acidosis: causes, pathophysiology, clinical manifestations, complications, diagnosis, treatment.

8. Respiratory alkalosis: causes, pathophysiology, clinical manifestations, complications, diagnosis, treatment.

9. Metabolic acidosis: causes, pathophysiology, clinical manifestations, complications, diagnosis, treatment.

10. Metabolic alkalosis: causes, pathophysiology, clinical manifestations, complications, diagnosis, treatment.

***Assignment questions* (ISW)**:

1. Explain how to calculate the dose of sodium bicarbonate (in mmol) to reverse the metabolic acidosis. Give some examples of calculation.

2. Discuss the relationship between the bicarbonate gap and the anion gap.

3. Explain how the low level of albumin does affect the calculation of anion gap.

4. Explain the difference between type 1 and type 2 respiratory failure. What are their causes?

5. Explain the property of H^+ ions that is the reason for the need to regulate its concentration.

6. Explain what can be done to correct respiratory acidosis and improve alveolar ventilation.

7. What is the most complicated acid-base imbalance to treat? Why?

8. Explain how the hyperventilation does lead to perioral and peripheral paresthesia.

9. Most of the hydrogen ions in the body come from acidic substances in the foods we ingest. True or false? Explain your point of view.

10. Define the utility of anion gap in the diagnosis and how it relates to osmolality.

11. Explain what the Partial Pressure does mean. How to calculate it?

12. Discuss the differential diagnosis for a metabolic acidosis with raised anion gap.

13. Explain the mechanism of how antacids work and give relief from acidity. List the names of antacids.

14. Explain what a high base excess (BE) and a low base excess (BE) does mean on acid base balance.

15. Explain the purpose of using the anion gap.

16. Explain the goal of secreting more H^+ ions.

17. Discuss the most important sources of acids and bases in the body.

18. Explain the goal of secreting less H^+ ions.

19. Explain the difference between acidemia and acidosis. Why is it important to know?

20. Which blood is more acidic - venous or arterial? Why?

TOPIC №3. Disorders of water and salt balance.

Equipment facilities: projector, educational video materials, clinical cases, MCQs.

Theory questions for self-control:

1. Regulation of water and electrolyte balance.
2. Edema: mechanisms, examples.
3. Isotonic, hypertonic, hypotonic alterations of water and salt balance.
4. Hyponatremia: causes, signs and symptoms, treatment.
5. Hypernatremia: causes, signs and symptoms, treatment.
6. Hypokalemia: causes, signs and symptoms, treatment.
7. Hyperkalemia: causes, signs and symptoms, treatment.
8. Hypocalcemia: causes, signs and symptoms, treatment.
9. Hypercalcemia: causes, signs and symptoms, treatment.
10. Hypomagnesemia: causes, signs and symptoms, treatment.
11. Hypermagnesemia: causes, signs and symptoms, treatment.

12. Hypophosphatemia: causes, signs and symptoms, treatment.

13. Hyperphosphatemia: causes, signs and symptoms, treatment.

14. Hypovolemia: causes, pathophysiology, signs and symptoms, complications.

15. Estimating fluid loss: signs and symptoms.

16. Hypervolemia: causes, pathophysiology, signs and symptoms, complications.

Assignment questions **(ISW)**:

1. What is the primary concern regarding fluid & electrolytes when caring for the elderly patients?

2. How various diuretics can disrupt a person's fluid and electrolyte balance?

3. Blood plasma, glomerular filtrate and urine have different concentrations of solutes, such as glucose and proteins. Explain the processes occurring in the kidney that cause difference in the concentrations of these solutes between blood plasma, glomerular filtrate and urine.

4. Explain why and how hypertonic and hypotonic urine is produced in the medulla of the kidney.

5. The sodium/calcium exchanger (NCX) transports sodium into and calcium out of cardiac muscle cells. Describe why this transporter is classified as secondary active transport.

6. Reveal the relationship of ionic sodium with normal cardiovascular system functioning.

7. Imagine a man who has just landed on a desert island. Trace the course of events leading to his sensation of thirst. Can he satisfy his thirst by drinking seawater? Explain your answer.

8. Compare and contrast the overall osmotic effects of electrolytes and nonelectrolytes.

9. Discuss what is better for a patient to have: hypophosphatemia or hypochloremia? Why?

10. Discuss what is better for a patient to have: hypervolemia or hypovolemia? Why?

11. List the factors that determine the body water content and describe the effect of each factor

12. Explain the importance of obligatory water losses.

13. Discuss the feedback mechanisms that regulate water intake and water output.

14. Indicate the routes of electrolyte entry and loss from the body.

15. A cell develops a mutation in its potassium channels that prevents the ions from leaving the cell. If the cell's aquaporins are still active, what will happen to the cell? Be sure to describe the tonicity and osmolality of the cell.

16. Compare the composition of the blood arriving at the kidney via the renal artery with the composition of the blood carried away from it in the renal vein.

17. Both of the regular intravenous solutions administered in medicine, normal saline and lactated Ringer's solution, are isotonic. Why is this important?

18. Discuss the feedback mechanisms that regulate sodium intake and sodium output.

19. Discuss factors that determine fluid and ion shifts in the body.

20. What's the difference between diffusion and osmosis; crystalloid and colloid solutions?

TOPIC №4. Inflammation and Fever.

Equipment facilities: projector, educational video materials, clinical cases, MCQs.

Theory questions for self-control:

1. Acute inflammation: cardinal signs, major components, stimuli.
2. Reactions of blood vessels in acute inflammation.
3. Reactions of leukocytes in acute inflammation.
4. Mechanism of the cellular response to inflammation.
5. Mediators of inflammation: sources, actions.

6. Outcomes of acute inflammation.
7. Chronic inflammation: causes, types.
8. Cell regeneration: types of cells.
9. Connective tissue repair.
10. Pyrogens: types.
11. Fever patterns and their causes.
12. Pathophysiology and clinical manifestations of fever.

***Assignment questions* (ISW)**:

1. Discuss the factors affecting healing in a positive and negative way.
2. Describe the time course of the vascular and cellular events responsible for the acute inflammatory response to injury, and discuss the receptors and ligands that are responsible for these events.
3. Discuss the role soluble and stromal factors play in the process of angiogenesis.
4. Describe systemic changes seen in inflammation, including metabolic consequences of changes in levels of serum proteins (acute phase reactants).
5. Discuss factors that determine what outcomes of inflammation are seen under different circumstances.
6. List the important proteins of the extracellular matrix and discuss the importance of cell-matrix interactions in the regulation of cell growth and differentiation.
7. Provide examples of how structural alterations of matrix proteins produce disease.
8. Describe the regulation of angiogenesis, discussing receptors on vascular endothelium.
9. Discuss the factors delaying wound healing.
10. Discuss the role of growth factors involved in tissue regeneration and repair.
11. Discuss the mechanism of fracture healing in details.

12. Discuss the role of selectins and intergrins involved in the inflammatory response.

13. Discuss the role of vasoactive amines in acute inflammation.

14. Discuss various processes that participate in the formation of a scar.

15. Discuss the role of chemokines involved in the inflammatory response.

16. Discuss the role of eosinophils in parasitic infections.

17. Discuss the role of chemoattractants in acute inflammation.

18. Discuss the role of free radicals in acute inflammation.

19. Explain the mechanisms of leukocyte transmigration.

20. Explain the mechanism of the triple response of Lewis.

TOPIC №6. Alterations of the immune response.

Equipment facilities: projector, educational video materials, clinical cases, MCQs.

Theory questions for self-control:

1. Comparative characteristics of innate and acquired immunity.
2. Types and role of immune cells.
3. Humoral (B-cell) immunodeficiency: types, causes, manifestations.
4. Cellular (T-cell) immunodeficiency: types, causes, manifestations.
5. Combined B-cell and T-cell immunodeficiency: types, causes, manifestations.
6. Type I hypersensitivity reactions: mechanism, mediators, clinical examples.
7. Type II hypersensitivity reactions: mechanism, clinical examples.
8. Type III hypersensitivity reactions: mechanism, clinical examples.
9. Type IV hypersensitivity reactions: mechanism, clinical examples.
10. Host-versus-graft disease: causes, mechanism, clinical examples.
11. Graft-versus-host disease: causes, mechanism, clinical examples.
12. Mechanisms of autoimmune disease.

***Assignment questions* (ISW)**:

1. Discuss the peoples' concerns regarding vaccination and point out the scientific rationale for vaccination.

2. Discuss how a person’s immune system would be affected in the absence of the thymus gland.

3. Discuss the role of the major histocompatibility antigens in health and disease.

4. Discuss the various mechanisms of immune suppression.

5. What causes asthma/allergy in the metropolitan cities and in the small villages? Why?

6. Explain how the interferons do monitor the infection of new cells.

7. Explain what the ‘memory’ associated with immune system does mean. Explain the mechanisms of the immunologic memory formation.

8. Explain how and why the active immunity is differing from passive immunity.

9. Explain the mechanism of T cell selection, activation, cloning and memory.

10. Write the names of the entities from which the different vaccines are synthesized.

11. List the preventive barriers that protect the organism against microbial bacteria entering. Name the immunity type observed in those cases.

12. Discuss the significance of mother’s milk to a newborn infant.

13. Explain what the recombinant DNA vaccine is. Give examples of such vaccines. State their advantages and disadvantages.

14. Explain the principles of vaccination.

15. Is it advantageous to have an identical twin for an organ transplant? If yes/no, why?

16. Explain the significance of the primary and the secondary immune responses.

17. Define and give examples of the types of acquired immunity.

18. Discuss the role of IgE, mast cells, and eosinophils in allergy.

19. Discuss the different types of T cells in terms of thymic interactions.

20. Discuss the functions of lymph nodes in the immune response.

TOPIC №6. Neoplasia.

Equipment facilities: projector, educational video materials, clinical cases, MCQs.

Theory questions for self-control:

1. Molecular basis of cancer: oncogenesis.
2. Cancer cell transformation: stages, mechanism.
3. Causes of cancer: genetics.
4. Causes of cancer: viruses.
5. Causes of cancer: immunologic mechanisms.
6. Causes of cancer: hormones.
7. Causes of cancer: chemical carcinogens.
8. Causes of cancer: ultraviolet and ionizing radiation.
9. Comparative characteristics of benign and malignant tumors.
10. Cancer cells characteristics.
11. Mechanisms of tumor invasion.
12. Mechanisms of tumor metastasis.
13. Mechanism of tumor growth.
14. Clinical manifestations of tumors: seven warning signs.
15. Paraneoplastic syndrome: mechanisms, clinical examples.

***Assignment questions* (ISW)**:

1. Explain the role of Human Papillomavirus in the induction of cancer.
2. Explain the process of chemical carcinogenesis in details, giving examples.
3. What types of benign tumors have the greatest malignant potential? Why?
4. List the general cytologic, biochemical, antigenic, metabolic, karyotypic, and molecular genetic changes found in neoplastic cells.

5. Discuss metastasis of malignant neoplasms in terms of molecular genetics, cellular adhesion and mechanisms of invasion of extracellular matrix.

6. Compare and contrast tumors transmitted by dominant inheritance and recessive inheritance. Give examples of each.

7. Discuss the different types of escape mechanisms utilized by neoplasms to evade the immune surveillance system of an immunocompetent host.

8. Compare and contrast acquired cancer-causing genetic mutations and germline cancer-causing genetic mutations. Give examples of each.

9. Discuss chemical carcinogenesis in terms of dose dependency.

10. Discuss genetic disorders that are associated with malignancy illustrating your answer with examples.

11. Discuss the mechanisms involved in the induction of cancer by radiation. Illustrate your answer with examples.

12. Discuss tumor specific antigens in terms of presence on normal cells and importance in anti-tumor immunity.

13. Discuss metastasis of malignant neoplasms in terms of tissues and organs in which metastases are common or uncommon.

14. Discuss the prognostic factors of tumor behavior that can be applied in clinical oncological practice.

15. Discuss the role of the environment in the development of neoplasms.

16. Discuss the significance of p53 and other tumor suppressor genes in neoplasia development.

17. What do you think is more dangerous – active smoking or passive smoking? Explain why.

18. Discuss the relationship between immune deficiency and neoplasia.

19. Contrast the effects of benign and malignant tumors on the host.

20. List the relative incidence and mortality of cancer for each sex and decade of age.

CREDIT

Equipment facilities: MCQs.

List of questions for Credit preparation:

1. Causes of cell injury.
2. Mechanisms of cell injury.
3. ATP deficiency as a mechanism of cell injury.
4. Disruption of calcium homeostasis as a mechanism of cell injury.
5. Damage to mitochondria as a mechanism of cell injury.
6. Oxidative stress.
7. Non-enzymatic and enzymatic antioxidants. Mechanism of action.
8. Pathological effects of free radicals.
9. DNA damage as a mechanism of cell damage.
10. Necrosis: causes and outcome.
11. Apoptosis: causes, mechanism and outcome.
12. Normal indices and mechanism of blood pH level control.
13. Buffer systems that regulate blood pH.
14. Mechanism and biological role of bicarbonate reabsorption.
15. Mechanism and biological role of ammoniogenesis.
16. Compensation of acid-base balance disorders by the kidneys.
17. Compensation of acid-base balance disorders by lungs.
18. Respiratory acidosis: causes, pathophysiology, clinical manifestations, complications, diagnosis, treatment.
19. Respiratory alkalosis: causes, pathophysiology, clinical manifestations, complications, diagnosis, treatment.
20. Metabolic acidosis: causes, pathophysiology, clinical manifestations, complications, diagnosis, treatment.
21. Metabolic alkalosis: causes, pathophysiology, clinical manifestations, complications, diagnosis, treatment.
22. Regulation of water and electrolyte balance.
23. Edema: mechanisms, examples.

24. Isotonic, hypertonic, hypotonic alterations of water and salt balance.
25. Hyponatremia: causes, signs and symptoms, treatment.
26. Hypernatremia: causes, signs and symptoms, treatment.
27. Hypokalemia: causes, signs and symptoms, treatment.
28. Hyperkalemia: causes, signs and symptoms, treatment.
29. Hypocalcemia: causes, signs and symptoms, treatment.
30. Hypercalcemia: causes, signs and symptoms, treatment.
31. Hypomagnesemia: causes, signs and symptoms, treatment.
32. Hypermagnesemia: causes, signs and symptoms, treatment.
33. Hypophosphatemia: causes, signs and symptoms, treatment.
34. Hyperphosphatemia: causes, signs and symptoms, treatment.
35. Hypovolemia: causes, pathophysiology, signs and symptoms, complications.
36. Estimating fluid loss: signs and symptoms.
37. Hypervolemia: causes, pathophysiology, signs and symptoms, complications.
38. Acute inflammation: cardinal signs, major components, stimuli.
39. Reactions of blood vessels in acute inflammation.
40. Reactions of leukocytes in acute inflammation.
41. Mechanism of the cellular response to inflammation.
42. Mediators of inflammation: sources, actions.
43. Outcomes of acute inflammation.
44. Chronic inflammation: causes, types.
45. Cell regeneration: types of cells.
46. Connective tissue repair.
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48. Fever patterns and their causes.
49. Pathophysiology and clinical manifestations of fever.
50. Comparative characteristics of innate and acquired immunity.
51. Types and role of immune cells.

52. Humoral (B–cell) immunodeficiency: types, causes, manifestations.

53. Cellular (T–cell) immunodeficiency: types, causes, manifestations.

54. Combined B–cell and T–cell immunodeficiency: types, causes, manifestations.

55. Type I hypersensitivity reactions: mechanism, mediators, clinical examples.

56. Type II hypersensitivity reactions: mechanism, clinical examples.

57. Type III hypersensitivity reactions: mechanism, clinical examples.

58. Type IV hypersensitivity reactions: mechanism, clinical examples.

59. Host–versus–graft disease: causes, mechanism, clinical examples.

60. Graft–versus–host disease: causes, mechanism, clinical examples.

61. Mechanisms of autoimmune disease.

62. Molecular basis of cancer: oncogenesis.

63. Cancer cell transformation: stages, mechanism.

64. Causes of cancer: genetics.

65. Causes of cancer: viruses.

66. Causes of cancer: immunologic mechanisms.

67. Causes of cancer: hormones.

68. Causes of cancer: chemical carcinogens.

69. Causes of cancer: ultraviolet and ionizing radiation.

70. Comparative characteristics of benign and malignant tumors.

71. Cancer cells characteristics.

72. Mechanisms of tumor invasion.

73. Mechanisms of tumor metastasis.

74. Mechanism of tumor growth.

75. Clinical manifestations of tumors: seven warning signs.

76. Paraneoplastic syndrome: mechanisms, clinical examples.

Examples of MCQs for the Credit:

What can lead to hypoxia?

A. Decreased oxygen saturation of the blood

B. Increased oxygen saturation of the blood

C. Increased tissue oxygen demand

D. Decreased tissue oxygen demand

E. Decreased oxygen production in cells

Identify a physical factor of cell damage.

A. Critical temperatures

B. Ischemia

C. Car exhaust gases

D. Alcohol

E. Prions

What does ATP deficiency in a cell lead to?

A. Decreased energy metabolism of the cell

B. Reversible damage to intracellular structures

C. Activation of calcium pumps

D. Activation of protein synthesis

E. Increased activity of membrane ATP-dependent pumps

Indicate a non-enzymatic antioxidant.

A. Vitamin E

B. Superoxide dismutase

C. Catalase

D. Glutathione peroxidase

E. Hydrogen peroxide

Increased concentration of which ions in the cell can activate its apoptosis?

A. Calcium

B. Potassium

C. Sodium

D. Magnesium

E. Chlorine

Where does the bicarbonate buffer system operate?

A. In the blood

B. In the kidneys

C. In the cells

D. In the erythrocytes

E. In the brain

During acidosis in the cells of the proximal tubules of the kidneys:

A. Ammonia synthesis increases

B. Ammonia synthesis decreases

C. Hydrogen synthesis increases

D. Bicarbonate reabsorption decreases

E. Glutamine metabolism is suppressed

What can lead to respiratory acidosis?

A. Airway obstruction

B. Liver failure

C. Excessive mechanical ventilation

D. Hypoaldosteronism

E. Chronic vomiting

What indicates compensation for metabolic acidosis?

A. pCO_2 in the blood < 35 mmHg

B. pCO_2 in the blood > 45 mmHg

C. pCO_2 in the blood < 45 mmHg

D. pCO_2 in the blood > 35 mmHg

E. pCO_2 in the blood < 25 mmHg

What confirms the diagnosis of metabolic alkalosis?

A. $[HCO_3^-]$ in the blood > 29 mmol/L

B. $[HCO_3^-]$ in the blood < 22 mmol/L

C. $[HCO_3^-]$ in the blood > 22 mmol/L

D. $[HCO_3^-]$ in the blood < 29 mmol/L

E. $[HCO_3^-]$ in the blood > 26 mmol/L

What acid-base disturbance can be caused by chronic vomiting?

A. Metabolic alkalosis

B. Metabolic acidosis

C. Respiratory alkalosis

D. Respiratory acidosis

E. There will be no disturbance

What does vitamin C cause in terms of promoting edema formation?

A. Increases capillary permeability

B. Decreases colloidal osmotic pressure

C. Increases capillary pressure

D. Obstructs lymphatic flow

E. Decreases colloidal osmotic pressure

What can cause isotonic fluid volume deficit?

A. Polyuria

B. Increased salt intake

C. Heart failure

D. Corticosteroid excess

E. «Water intoxication»

During hypovolemic shock:

A. Venous return to the heart decreases

B. Cardiac output increases

C. Mean arterial pressure increases

D. Tissue perfusion increases

E. The body's need for fluid decreases

What leads to the distention of the neck veins during hypervolemia?

A. Increased blood volume

B. High plasma tonicity

C. Activation of the sympathetic nervous system

D. Decreased blood pressure

E. Tissue dehydration

What can cause hyperkalemia?

A. Massive burns

B. Vomiting

C. Drinking seawater

D. Water diarrhea

E. Lithium medications

What can cause hypocalcemia?

A. Vitamin D deficiency

B. Water diarrhea

C. Vomiting

D. Excessive vitamin D intake

E. Lithium medications

What increases the vascular permeability during inflammation?

A. Damage to the endothelium

B. Swelling of endothelial cells

C. Tightening of the endothelium

D. Blocking of transcytosis

E. Margination of leukocytes

What event is mediated by the Sialyl–Lewis X molecule during acute inflammation?

A. Rolling of leukocytes

B. Pavementing of leukocytes

C. Emigration of leukocytes

D. Chemotaxis of leukocytes

E. Phagocytosis

Which mediator of inflammation causes pain?

A. Bradykinin

B. Histamine

C. Serotonin

D. Leukotriene

E. Interleukin-1

During chronic inflammation, activated macrophages produce:

A. Interleukin-1

B. Vascular endothelial growth factor

C. Nerve growth factor

D. Transforming growth factor β

E. γ-interferon

What happens during the proliferative phase of repair?

A. Formation of granulation tissue

B. Migration of phagocytes to the site of damage

C. Hemostatic processes

D. Removal of fibroblasts from the site of damage

E. Remodeling of the scar

What will the temperature pattern be like in a patient with acute respiratory viral infection?

A. Remittent

B. Intermittent

C. Continuous

D. Recurrent

E. Distorted

Th1-helper lymphokines:

A. Stimulate phagocytosis by macrophages

B. Inhibit potentially dangerous inflammatory reactions

C. Remove neutrophils and monocytes from the area of injury

D. Suppress the production of prostaglandins

E. Inhibit the production of T-killers

Indicate the feature of type IV hypersensitivity reaction.

A. Sensitized T-lymphocytes destroy antigenic cells

B. The antigen and antibody are free and form complexes

C. The antigen is located on the surface of the cell

D. The antigen binds to IgE on the membrane of the mast cell

E. Sensitized T-lymphocytes activate the antigen

What type of hypersensitivity reaction is bronchial asthma associated with?

A. Anaphylactic

B. Cytotoxic

C. Immune complex

D. T-cell mediated

E. Distorted

Indicate a disease that is associated with a type II hypersensitivity reaction.

A. Hemolytic disease of the newborn

B. Bronchial asthma

C. Serum sickness

D. Contact dermatitis

E. Graft-versus-host reaction

In a child's blood, decreased levels of IgA, IgE, IgG_2 and a low CD_4^+/CD_8^+ ratio were found. What type of immunodeficiency is this?

A. Louis-Bar syndrome

B. Wiskott-Aldrich syndrome

C. Bruton's disease

D. Nezelof syndrome

E. DiGeorge syndrome

Indicate a sign of Wiskott-Aldrich syndrome in the blood.

A. Decreased level of IgM and increased levels of IgA and IgE

B. Lack of mature B-lymphocytes, plasma cells, and IgG, IgA, IgM

C. Decreased level of IgG without a decrease in IgA and IgM levels

D. Decreased levels of IgG, IgA, IgM with normal B-lymphocyte counts

E. Decreased levels of IgA, IgE, and IgG_2

Indicate an oncogene.

A. MYC

B. p53

C. Rb

D. APC

E. BRCA

What does hepatitis B virus cause?

A. Hepatocellular carcinoma

B. B-cell lymphoma

C. Squamous cell carcinoma of the cervix

D. Nasopharyngeal carcinoma

E. Kaposi's sarcoma

Indicate an indirect carcinogen.

A. Aflatoxin B1

B. Chlorambucil

C. Nitrosourea

D. Cyclophosphamide

E. β-propiolactone

How can the intake of cytotoxic drugs contribute to tumor development? These drugs:

A. Destroy circulating lymphocytes

B. Suppress the thymus and T-cell immunity

C. Divert the immune system's «attention» to themselves

D. Exhaust the immune system and lead to anergy

E. Cause errors in the copying of genetic material

What happens immediately after the formation of a metastatic subclone during the process of hematogenous tumor metastasis?

A. Adhesion and invasion of the basement membrane

B. Passage through the extracellular matrix

C. Intravasation

D. Interaction with host lymphocytes

E. Formation of a tumor embolus

Activation of which hormone causes cachexia in patients with malignant tumors?

A. Cortisol

B. Aldosterone

C. Gastrin

D. Testosterone

E. Somatotropin

SECTION II. Systemic pathophysiology

TOPIC №1. Red blood cells disorders.

Equipment facilities: projector, educational video materials, clinical cases, MCQs.

Theory questions for self-control:

1. Classification of anemia according to underlying mechanism.
2. Blood loss anemia: types, causes, manifestations.
3. Extravascular and intravascular hemolysis: causes, mechanisms, manifestations.
4. Hereditary spherocytosis: cause, mechanism, signs and symptoms, complications.
5. Sickle cell anemia: cause, mechanism, signs and symptoms, complications.
6. Thalassemia: cause, mechanisms, signs and symptoms, complications.
7. Hemolytic disease of the newborn: cause, mechanism, signs and symptoms, complications.
8. Iron-deficiency anemia: causes, mechanism, signs and symptoms, complications.
9. Megaloblastic anemia: causes, mechanism, signs and symptoms, complications.

10. Aplastic anemia: causes, mechanism, signs and symptoms, complications.

11. Polycythemia: types, causes, mechanism, signs and symptoms, complications.

Assignment questions **(ISW)**:

1. Compare and contrast warm vs. cold antibody immunohemolytic anemias in terms of pathogenesis and associated risks-diseases.

2. Identify and explain the various conditions that can change the oxygen dissociation curve.

3. Discuss components of the mature red cell that are essential for its survival and function.

4. List and explain the pathogenesis of conditions with increased erythropoietin production.

5. Discuss the enzyme deficiency in RBCs that leads to ATP depletion and hemolytic anemia.

6. Discuss the enzyme deficiency in RBCs that leads to hemolytic anemia due to oxidative stress.

7. Discuss how iron-deficiency anemia does affect teenagers and old people.

8. Discuss the four mechanisms of drug-induced hemolytic anemias.

9. Which of these groups – men, women, teenagers, old people – are the most likely to have anemia? Why?

10. Define the major clinical signs and symptoms and abnormal laboratory tests results that are typically associated with homo and hetero conditions of HbS, HbC, HbD and HbE, and compound heterozygous conditions involving HbS and other variant hemoglobins.

11. Discuss the difference between sickle cell disease and sickle cell trait. Give some examples.

12. Describe the purpose of the metabolic pathways used by erythrocytes: Embden-Meyerhof, Hexose monophosphate shunt and Methemoglobin reductase.

13. Discuss and differentiate nonmegaloblastic macrocytosis from megaloblastic anemia.

14. Explain the pathogenesis of clinical signs and symptoms of Fanconi anemia.

15. Explain the pathogenesis of hematological signs and symptoms Shwachman-Diamond syndrome.

16. Describe nutritional and regulatory factors that are associated with erythropoiesis.

17. Describe the mechanisms of blood volume restoration after hemorrhage.

18. Which gases can and cannot bind to hemoglobin? Why?

19. Discuss the factors that contribute to the Bohr Effect.

20. Summarize the mechanisms by which normal hemoglobin is structured and synthesized in the developing red cell.

TOPIC №2. White blood cells disorders.

Equipment facilities: projector, educational video materials, clinical cases, MCQs.

Theory questions for self-control:

1. Neutrophil leukocytosis: causes.
2. Eosinophil leukocytosis: causes.
3. Basophil leukocytosis: causes.
4. Monocytosis: causes.
5. Lymphocytosis: causes.
6. Leukopenia: types, causes.
7. Clinical manifestations of leukemia and their pathologic basis.
8. Acute leukemias: types, causes, signs and symptoms, complications.
9. Chronic leukemias: types, causes, signs and symptoms, complications.

Assignment questions **(ISW)**:

1. Discuss factors associated with the prognosis in AML and ALL.

2. Discuss the purpose, advantages, and concerns related to allogeneic hematopoietic-cell transplantation.

3. Discuss the etiology, characteristic abnormalities and clinical features for the qualitative/functional disorders of neutrophils: Pelger-Huet anomaly and Alder-Reilly anomaly.

4. Discuss the use of various diagnostic techniques used to assess neoplastic disorders of blood and bone marrow cells.

5. Discuss the significance of the discovery of the human T-cell leukemia virus (HTLV) family and describe associated disorders.

6. List and describe the major systems used to classify neoplastic disorders of leukocytes.

7. Discuss the etiology, characteristic abnormalities and clinical features for the qualitative/functional disorders of neutrophils: Chediak-Higashi syndrome and May-Hegglin anomaly.

8. Discuss qualitative and quantitative alterations of monocytes, and indicate their causes and clinical significance.

9. Discuss the etiology, characteristic abnormalities and clinical features for the qualitative/functional disorders of neutrophils: Chronic granulomatous disease (CGD) and Myeloperoxidase deficiency.

10. Compare and discuss the principles of various cytochemical stains and the cell lineages they react with.

11. Explain the appearance, etiology and pathogenesis of the various morphological abnormalities in the mature granulocytes.

12. Correlate diagnostic blood and bone marrow findings to various sub-types of acute myeloid leukemia.

13. Discuss the mechanisms and characteristics of severe congenital neutropenia and cyclic neutropenia.

14. Discuss the causes and mechanisms of reactive lymphocytosis conditions.

15. Compare the clinical findings and laboratory features of various plasma cell disorders.

16. Compare the characteristics of leukemia, lymphoma, and myeloma.

17. Describe the clinical and laboratory findings commonly found in myelodysplastic syndrome.

18. Discuss factors related to epidemiology and long-term survival of AML patients.

19. Describe the Philadelphia chromosome. Why and how this mutation does occur? Does CML run in families?

20. Discuss the length of time the neutrophils, eosinophils, basophils, mast cells, monocytes, macrophages, B- and T-lymphocytes spend in tissues and circulating pool.

TOPIC №3. Disorders of hemostasis

Equipment facilities: projector, educational video materials, clinical cases, MCQs.

Theory questions for self-control:

1. Formation of platelet plug and blood coagulation.
2. Thrombocytopenia: causes, manifestations.
3. Thrombocytopathy: causes, manifestations.
4. Impaired synthesis of coagulation factors: causes, manifestations.
5. Hemophilia and von Willebrand disease: causes, signs and symptoms, complications.
6. Bleeding disorders: vascular disorders: causes, signs and symptoms, complications.
7. Conditions associated with hypercoagulability states: causes, manifestations.
8. Disseminated intravascular coagulation (DIC): causes, mechanism, signs and symptoms, complications.

***Assignment questions* (ISW)**:

1. Compare types and explain the etiology, pathogenesis and clinical manifestations of congenital amegakaryocytic thrombocytopenia.

2. Compare and contrast idiopathic thrombocytopenic purpura (ITP) and thrombotic thrombocytopenic purpura (TTP) in terms of etiology, pathogenesis and clinical manifestation.

3. Compare and contrast idiopathic thrombocytopenic purpura (ITP) and hemolytic-uremic syndrome (HUS) in terms of etiology, pathogenesis and clinical manifestation.

4. Categorize and discuss acquired disorders of platelet function in terms of etiology, pathogenesis and clinical manifestation.

5. Compare and contrast the Glanzmann thrombasthenia and Chediak-Higashi syndrome in terms of definition, genetics, laboratory features (including platelet aggregation patterns) and clinical manifestations.

6. Compare and contrast the Hermansky-Pudlak syndrome and von Willebrand disease in terms of genetics, laboratory features (including platelet aggregation patterns) and clinical manifestations.

7. Compare and contrast the Bernard-Soulier disease and gray platelet syndrome disease in terms of genetics, laboratory features (including platelet aggregation patterns) and clinical manifestations.

8. Compare and contrast the HIV-associated thrombocytopenia and drug-induced thrombocytopenia in terms of genetics, laboratory features (including platelet aggregation patterns) and clinical manifestations.

9. Discuss thrombocytopoesis in terms of the mechanisms of megakaryocytes formation, the life span of platelets and factors which activate and inhibit thrombocytopoesis.

10. Describe the mechanism(s) by which the aspirin, coumadin (warfarin) and heparin affect the hemostasis and discuss the methods by which each is monitored.

11. Explain the mechanisms of procoagulant and anticoagulant activity of the endothelium.

12. Discuss thrombocytopenia in terms of clinical manifestations, differential diagnosis, bone marrow morphology and laboratory findings.

13. Discuss thrombocytosis in terms of clinical manifestations, differential diagnosis, bone marrow morphology and laboratory findings.

14. List and discuss the laboratory diagnostic procedures used to approach patients with bleeding disorders and thrombotic disorders.

15. Discuss disseminated intravascular coagulopathy (DIC) in terms of the pathogenesis of morphologic features, laboratory diagnosis, complications and prognosis.

16. Outline the process for stepwise evaluation of a bleeding patient, a patient with suspected platelet disorder and a patient with suspected hypercoagulability.

17. Describe abnormal morphological forms of platelets and megakaryocytes, and indicate their causes, pathogenesis and clinical significance.

18. Distinguish between venous thrombi and arterial thrombi on the basis of etiologic and precipitating factors, mechanisms and common sites of occurrence, type and size of vessel involved, morphologic appearance.

19. Distinguish between venous thrombi and arterial thrombi on the basis of organs commonly involved, local and distant effects, clinical and laboratory features, prognosis.

20. Discuss thrombi in terms of types of thrombotic material, factors conditioning the development of thrombi and possible fate of thrombi.

TOPIC №4. Disorders of cardiovascular system.

Equipment facilities: projector, educational video materials, clinical cases, MCQs.

Theory questions for self-control:

1. Principal mechanisms of the cardiovascular dysfunction.

2. General causes of heart failure.

3. Right-sided heart failure: causes, mechanism, signs and symptoms, complications.

4. Left-sided heart failure: causes, mechanism, signs and symptoms, complications.

5. Systolic and diastolic dysfunction: causes, mechanism, signs and symptoms, complications.

6. Cardiogenic shock: causes, mechanism, signs and symptoms, complications.

7. Sinus tachycardia, sinus bradycardia and paroxysmal supraventricular tachycardia (PSVT): ECG features.

8. Atrial flutter, atrial fibrillation and ventricular fibrillation: ECG features.

9. AV block: degrees, ECG features.

10. Premature ventricular contraction (PVC) and ventricular tachycardia: ECG features.

11. Angina pectoris: causes, types, signs and symptoms, complications.

12. Myocardial infarction: causes, signs and symptoms, complications.

13. Essential (primary) hypertension: causes, mechanism, signs and symptoms, complications.

14. Secondary hypertension: causes, mechanism, signs and symptoms, complications.

15. Orthostatic hypotension: causes, mechanisms.

16. Atherosclerosis: causes, pathogenesis, complications.

***Assignment questions* (ISW)**:

1. List and explain the mechanisms of the short- and long-term complications associated with prosthetic heart valves.

2. Discuss the causes and mechanisms of congestive heart failure exacerbations.

3. Indicate the causes and explain the mechanisms of a prolonged QT interval.

4. How to distinguish the ECG findings of pericarditis from myocardial infarction and early repolarization?

5. Indicate the causes and explain the mechanisms of AV dissociation on ECG.

6. Can you diagnose a myocardial infarction on ECG if there is a left bundle branch block (LBBB)?

7. Describe the mechanisms of common LQTS, Brugada, short-QT, and J-wave syndromes.

8. What are the ECG changes of hyperkalemia and what can be done to resolve them?

9. How does the heart work and what are the consequences in patients that have AV dissociation?

10. Discuss the role and methods of interventional cardiology and bypass surgery and why these treatments are used.

11. What are the important teaching points for the patient receiving a heart transplant?

12. Discuss congenital heart disease in terms of types which come to medical attention in: a) infancy, b) childhood and c) adulthood.

13. List and explain the pathogenesis of the peripheral signs of aortic regurgitation.

14. List the cardiac biomarkers. Which cardiac biomarker elevates first? Why? Which cardiac biomarker stays elevated the longest? Why?

15. Discuss the causes, mechanisms of the primary heart overload pattern and complications in patients with Hypertrophic and Dilated cardiomyopathy.

16. Discuss the causes, mechanisms of the primary heart overload pattern and complications in patients with Congenital atrial septal defect and Congenital ventricular septal defect.

17. Discuss the causes, mechanisms of the primary overload pattern and complications in patients with Mitral insufficiency and Tricuspid insufficiency.

18. Discuss the genetic and environmental factors which result in: a) left-to-right vs. right-to-left shunts; and b) cyanotic vs. acyanotic disease.

19. Discuss sudden cardiac death in terms of causes, pathogenesis, relationship to arrhythmias and cardiac morphology.

20. What causes an S_3 heart sound versus an S_4 heart sound? Compare the 3rd and 4th heart sounds and indicate their clinical significance.

TOPIC №5. Disorders of respiratory system.

Equipment facilities: projector, educational video materials, clinical cases, MCQs.

Theory questions for self-control:

1. Pathological basis of respiratory signs and symptoms.
2. Pathological breathing: types, causes, manifestations.
3. Dyspnea: causes, mechanisms.
4. Restrictive lung diseases: causes, pathogenesis.
5. Atelectasis: types, causes, signs and symptoms.
6. Pneumothorax: types, causes, signs and symptoms.
7. Bronchial asthma: causes, pathogenesis, signs and symptoms, complications.
8. Emphysema: causes, pathogenesis, signs and symptoms, complications.
9. Interstitial lung diseases: causes, pathogenesis, manifestations.
10. Diffusion abnormalities: causes, pathogenesis.
11. Pulmonary hypertension: types, causes, signs and symptoms, complications.
12. Pulmonary edema: causes, pathogenesis, signs and symptoms, complications.

***Assignment questions* (ISW)**:

1. Discuss drug-induced lung disease, enumerating drugs most commonly associated with the following pulmonary reactions: bronchospasm, pulmonary edema, hypersensitivity pneumonitis (HP) and pulmonary fibrosis.

2. Discuss respiratory bronchiolitis of smokers (small airways disease) in terms of pathogenesis, morphology, clinical manifestation, complications and prognosis.

3. Compare and contrast primary and secondary pulmonary hypertension in terms of predisposing factors/associated conditions, pathogenesis, age and sex distribution, size and type of vessels involved, morphologic features (including reversible vs. irreversible lesions), hemodynamic consequences and prognosis.

4. Compare and contrast neonatal and adult respiratory distress syndrome in terms of predisposing factors/associated conditions, pathogenesis, clinical course, complications and prognosis.

5. Compare and contrast the etiology, pathogenesis, morphology, clinical manifestations, complications and prognosis of diffuse alveolar damage (DAD) and bronchiolitis obliterans-organizing pneumonia (BOOP).

6. Compare and contrast the etiology, pathogenesis, morphology, clinical manifestations, complications and prognosis of usual interstitial pneumonia (UIP) and desquamative interstitial pneumonia (DIP).

7. Compare and contrast the etiology, pathogenesis, morphology, clinical manifestations, complications and prognosis of lymphoid interstitial pneumonia (LIP) and nonspecific interstitial pneumonia (NSIP).

8. Discuss the acute and chronic stages of radiation lung injury in terms of pathogenesis, temporal features, morphology, complications and prognosis.

9. Compare and contrast the etiology, pathogenesis, morphology, clinical manifestations, complications and prognosis of coal workers' pneumoconiosis, silicosis, asbestosis and berylliosis.

10. Discuss the indications and contraindications for lung transplantation and explain the mechanisms of its various complications.

11. Compare etiology, pathogenesis, clinical manifestations and prognosis of pulmonary involvement in systemic lupus erythematosus (SLE), rheumatoid arthritis (RA) and progressive systemic sclerosis (PSS).

12. Differentiate among tuberculosis, sarcoidosis, granulomatous and fungal lung diseases (etiology, pathogenesis, organs involved, radiologic features, clinical manifestation, diagnostic tests, laboratory findings and prognosis).

13. Discuss pulmonary edema, embolism and infarction in terms of predisposing factors and etiology, pathogenesis, radiologic features, clinical manifestations and prognosis.

14. Contrast obstructive and restrictive pulmonary disease, in terms of pathogenesis, morphological features, radiologic manifestations and pulmonary function test results.

15. Compare and contrast the etiologies and effects of airflow obstruction that occur in lesions involving the airways with those that involve the alveolar parenchyma.

16. Compare and contrast the etiology, pathogenesis, clinical and pathologic features of bronchial asthma: atopic, non-atopic, drug-induced and occupational.

17. Differentiate pathogenically and clinically: a) atelectasis and pleural effusion; b) transudative and exudative pleural effusion.

18. Discuss the etiology, pathogenesis, morphology and clinical manifestation of pulmonary circulatory disease associated with congenital heart disease and persistent fetal circulation.

19. Describe the mechanisms by which the following pulmonary defense mechanisms accomplish their functions: nasal clearance, laryngeal (including epiglottis) action, tracheobronchial clearance and alveolar clearance.

20. Discuss the following pulmonary congenital anomalies in terms of genetics, pathophysiology and clinical consequences: agenesis, hypoplasia, congenital lobar overinflation ("emphysema"), congenital cyst and bronchopulmonary sequestration.

TOPIC №6. Disorders of hepatic and biliary function.

Equipment facilities: projector, educational video materials, clinical cases, MCQs.

Theory questions for self-control:

1. Functions of the liver and manifestations of altered function.
2. The mechanism of bilirubin elimination.
3. Prehepatic jaundice: causes, pathogenesis, manifestations.
4. Hepatic jaundice: causes, pathogenesis, manifestations.
5. Posthepatic jaundice: causes, pathogenesis, manifestations.
6. Cirrhosis: causes and pathogenesis.
7. Cirrhosis: signs and symptoms, complications.
8. Portal hypertension: causes and pathogenesis.
9. Portal hypertension: signs and symptoms, complications.
10. Liver failure: causes and pathogenesis.
11. Liver failure: signs and symptoms, complications.
12. Cholelithiasis: causes, signs and symptoms, complications.

***Assignment questions* (ISW)**:

1. Compare and contrast the etiology, pathogenesis, morphology, clinical manifestation, laboratory findings, complications and prognosis of Crigler-Najjar syndrome type 1, Crigler-Najjar syndrome type 2 and Gilbert syndrome.
2. Describe the pathogenic mechanisms whereby the acetaminophen, carbon tetrachloride and halothane cause liver injury.
3. Compare and contrast the etiology, pathogenesis, morphology, clinical manifestations, laboratory findings, complications and prognosis of Dubin-Johnson syndrome, Rotor syndrome and Crigler-Najjar syndrome.
4. Describe the pathogenic mechanisms whereby the phenothiazines, tetracyclines and paracetamol cause liver injury.
5. Compare and contrast biliary atresia and neonatal hepatitis in terms of etiology, pathogenesis, morphology, clinical manifestations, laboratory findings, complications and prognosis.

6. Compare and contrast the etiology, pathogenesis, morphology, clinical manifestations, laboratory findings, complications and prognosis of alcohol hepatitis, non-alcoholic steatohepatitis and viral hepatitis.

7. Compare and contrast the etiology, pathogenesis, morphology, clinical manifestations, laboratory findings, complications and prognosis of granulomatous hepatitis, drug-induced hepatitis and toxic hepatitis.

8. Compare and contrast the etiology, pathogenesis, morphology, clinical manifestations, laboratory findings, complications and prognosis of α-1-antitrypsin deficiency, alcoholic cirrhosis and secondary biliary cirrhosis.

9. Compare and contrast the etiology, pathogenesis, morphology, clinical manifestations, laboratory findings, complications and prognosis of non-alcoholic steatohepatitis, postnecrotic cirrhosis and Wilson disease.

10. Compare predictable and unpredictable drug induced liver diseases.

11. Compare and contrast the etiology, pathogenesis, morphology, clinical manifestations, laboratory findings, complications and prognosis of autoimmune hepatitis and primary biliary cirrhosis.

12. Compare and contrast the etiology, pathogenesis, morphology, clinical manifestations, laboratory findings, complications and prognosis of secondary biliary cirrhosis and primary sclerosing cholangitis.

13. Discuss the indications, benefits, and complications of the liver transplantation.

14. Discuss the pathogenesis, morphology, clinical manifestations and complications of liver transplant rejection.

15. Compare and contrast the etiology, pathogenesis, morphology, clinical manifestations, laboratory findings, complications and prognosis of hepatic vein thrombosis and portal hypertension.

16. Compare and contrast empyema and hydrops of the gallbladder in terms of etiology, epidemiology, pathogenesis, relationship to cholelithiasis, morphology, clinical manifestations, complications and prognosis.

17. Discuss carcinoma of the gallbladder and carcinoma of the extrahepatic bile ducts, in terms of etiology, epidemiology, pathogenesis, relationship to cholelithiasis, morphology, clinical manifestations, complications and prognosis.

18. Compare and contrast acute and chronic cholecystitis in terms of etiology, epidemiology, associated diseases, morphology, clinical manifestations, complications (including complications of therapy) and prognosis.

19. Discuss typical infectious liver diseases caused by bacteria, protozoa and helminths in terms of pathogenesis, morphology, clinical manifestations, laboratory findings, complications and prognosis.

20. Discuss the causes and pathogenesis of fatty change (steatosis) of the liver in terms of size of fat vacuoles and zonal distribution of fat.

TOPIC №7. Disorders of gastrointestinal and exocrine pancreas function.

Equipment facilities: projector, educational video materials, clinical cases, MCQs.

Theory questions for self-control:

1. Anorexia, nausea and vomiting: causes, mechanisms.
2. Gastrointestinal tract bleeding: causes, manifestations.
3. Dysphagia: causes, manifestations.
4. Gastroesophageal reflux: causes, pathogenesis, signs and symptoms, complications.
5. Gastritis: types, causes, mechanisms.
6. Peptic ulcer: causes, pathogenesis, signs and symptoms, complications.
7. Stress ulcers: types, causes, mechanisms.
8. Diarrhea: types, causes, mechanisms.
9. Constipation: causes, mechanisms.
10. Intestinal obstruction: types, causes, mechanisms.
11. Malabsorption: causes.

12. Malabsorption: signs and symptoms of impaired absorption of dietary constituents.

13. Acute pancreatitis: causes, pathogenesis, signs and symptoms, complications.

***Assignment questions* (ISW)**:

1. Discuss islet cell tumors of the pancreas in terms of incidence, benignity vs. malignancy, immunohistochemical characteristics, endocrine function, clinical manifestations and prognosis.

2. Discuss indications, benefits and complications of pancreatic islet cell transplantation.

3. Explain how celiac disease, sprue, gastroenteritis and inflammatory bowel disease lead to malabsorption.

4. Compare and contrast acute (erosive), autoimmune, atrophic and chronic gastritis in terms of etiology, pathogenesis, morphology, clinical manifestations, laboratory findings, complications and prognosis.

5. Compare and contrast the etiology, pathogenesis, morphology, clinical manifestations, laboratory findings, complications and prognosis of celiac sprue, tropical sprue and Whipple disease.

6. Contrast and compare diarrheal disease caused by enterotoxin-producing bacteria and diarrhea due to enteroinvasive microbes.

7. Discuss carcinoid tumors of the colon, rectum, and appendix in terms of pathogenesis, clinical manifestations (including extra-colonic manifestations), complications and prognosis.

8. List and discuss the clinical situations in which stool examination may be helpful in the diagnosis of alimentary diseases.

9. Compare and contrast the etiology, pathogenesis, morphology, clinical manifestations, laboratory findings, complications and prognosis of necrotizing enterocolitis and infectious enterocolitis.

10. Discuss the pathogenesis and clinical manifestations for disorders of the GI tract that arise from hypoxia or ischemia.

11. Outline the precursor lesions, risk factors and hereditary cancer syndromes that lead to GI neoplasia.

12. Compare and contrast the etiology, pathogenesis, morphology, clinical manifestations, laboratory findings, complications and prognosis of pseudomembranous colitis and ischemic colitis.

13. Compare and contrast the etiology, pathogenesis, morphology, clinical manifestations, laboratory findings, complications and prognosis of collagenous colitis and lymphocytic colitis.

14. Discuss esophageal varices, their pathogenesis and typical complications.

15. Compare and contrast the pathogenesis and clinical manifestations of inflammatory bowel disease.

16. Describe the pathogenesis and clinical manifestations of disorders presenting with dysphagia.

17. Compare and contrast the pathophysiology of gastrointestinal disorders that present with GI obstruction.

18. Explain the pathogenesis, clinical manifestations, laboratory findings, complications and prognosis tracheoesophageal fistula, pyloric stenosis, intestinal atresia and Hirschprung disease.

19. Compare and contrast the pathogenesis, clinical manifestations, laboratory findings, complications and prognosis of adenocarcinoma of the pancreatic head, pancreatic body/tail and ampulla of Vater.

20. Compare and contrast the pathogenesis and clinical manifestations of exocrine and endocrine pancreatic insufficiency.

TOPIC №8. Disorders of renal function.

Equipment facilities: projector, educational video materials, clinical cases, MCQs.

Theory questions for self-control:

1. Pathological basis for urinary tract symptoms and signs.

2. Acute glomerulonephritis: causes, pathogenesis, signs and symptoms, complications.

3. Rapidly progressive glomerulonephritis: causes, pathogenesis, signs and symptoms, complications.

4. Chronic glomerulonephritis: causes, pathogenesis, signs and symptoms, complications.

5. Nephrotic syndrome: causes, pathogenesis, signs and symptoms, complications.

6. Acute pyelonephritis: causes, signs and symptoms, complications.

7. Chronic pyelonephritis: causes, signs and symptoms, complications.

8. Acute renal failure: types, causes.

9. Acute renal failure: phases, signs and symptoms, complications.

10. Chronic renal failure: causes, pathogenesis, signs and symptoms, complications.

11. Urinary tract obstruction: causes.

12. Nephrolithiasis: causes, pathogenesis, signs and symptoms, complications.

Assignment questions **(ISW)**:

1. Compare and contrast renal diseases that involve the glomerular basement membrane and explain their etiology, pathogenesis, complications and prognosis.

2. Compare the basic medical and surgical complications after deceased and living donor kidney transplantation.

3. Compare and contrast ischemic and nephrotoxic forms of acute tubular injury, including typical clinical contexts, pathogenesis of renal failure, microscopic appearance, and expected outcome.

4. How the dietary and lifestyle changes might help reduce the damage of the kidney in patients with various types of renal pathologies?

5. Compare and contrast the etiology, pathogenesis, clinical manifestations, morphology, complications and prognosis of autosomal dominant

(adult) polycystic kidney disease and autosomal recessive (childhood) polycystic kidney disease.

6. Discuss the proliferative and pro-inflammatory conditions presenting with nephritic syndrome.

7. Compare and contrast typical hemolytic uremic syndrome (HUS), atypical HUS, and thrombotic thrombocytopenic purpura (TTP) in terms of etiology, pathogenesis, clinical manifestations, renal histopathology, complications, and prognosis.

8. List the three major thrombotic microangiopathies and describe the renal effects and pathogenesis in terms of clinical manifestation, complications and prognosis.

9. Compare and contrast the mechanisms of immune complex- and antibody-mediated glomerulonephritis.

10. Compare and contrast acute pyelonephritis, drug-induced interstitial nephritis, and lupus nephritis in terms of pathogenesis, clinical manifestation, morphology, complications and prognosis.

11. Discuss the genetic mutations and the pathogenesis of relevant benign and malignant tumors of kidneys and urinary tract.

12. What is the "neurogenic bladder"? Describe the etiology, pathogenesis, clinical manifestations, complications, differential diagnosis and prognosis.

13. Compare thrombotic and embolic causes of renal arterial occlusions in terms of underlying pathogenesis, gross and microscopic morphology and clinical manifestation.

14. Discuss the pathogenesis and clinical manifestations of tubulointerstitial diseases and discuss how their pathogenesis relates to treatment and outcomes.

15. Discuss the significance of unilateral renal artery disease, including usual causes, mechanisms, clinical manifestations, morphological changes in contralateral kidney, tests used for detection and localization, complications and prognosis.

16. Compare and contrast benign and malignant nephrosclerosis in terms of etiology, pathogenesis, clinical manifestation, complications and prognosis.

17. Discuss how the hypertension leads to structural changes in the renal vasculature and how those vascular lesions cause renal dysfunction.

18. List the different chemical types of kidney stones, explain the mechanisms related to their development and describe therapy/prevention of kidney stones.

19. Discuss the pathogenesis of diabetic nephropathy and the associated clinical manifestations.

20. Discuss the proper use of the following laboratory tests in the evaluation of urinary tract disease and interpret abnormalities of these parameters in clinical context: creatinine, urea (blood urea nitrogen, BUN) and urinalysis.

TOPIC №9. Disorders of endocrine function, part 1.

Equipment facilities: projector, educational video materials, clinical cases, MCQs.

Theory questions for self-control:

1. Sources and mechanism of action of hormones.
2. Control of hormone levels.
3. The causes of the decrease and increase in the level of hormones.
4. Hypofunction of an endocrine gland: causes.
5. Hyperfunction of an endocrine gland: causes.
6. Primary, secondary, and tertiary disorders.
7. Hypopituitarism: causes, pathogenesis, signs and symptoms, complications.
8. Growth hormone deficiency: causes, pathogenesis, signs and symptoms, complications.
9. Growth hormone excess: causes, pathogenesis, signs and symptoms, complications.
10. Manifestations of hypothyroid and hyperthyroid states.

11. Hypothyroidism in adults: causes, signs and symptoms, complications.

12. Hypothyroidism in children: causes, signs and symptoms, complications.

13. Hyperthyroidism: causes, signs and symptoms, complications.

***Assignment questions* (ISW)**:

1. Compare and contrast infectious and subacute (granulomatous) thyroiditis in terms of age and sex distribution, etiology, pathogenesis, clinical manifestations, laboratory findings, complications and prognosis.

2. Compare and contrast subacute lymphocytic and Hashimoto's thyroiditis in terms of age and sex distribution, etiology, pathogenesis, clinical manifestations, laboratory findings, complications and prognosis.

3. Compare and contrast Graves' disease and Riedel's thyroiditis in terms of age and sex distribution, etiology, pathogenesis, clinical manifestations, laboratory findings, complications and prognosis.

4. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of Sheehan syndrome and empty sella syndrome.

5. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of diabetes mellitus and diabetes insipidus.

6. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of Waterhouse-Friderichsen syndrome and Addisonian crisis.

7. Describe methods of screening and following patients with diabetes mellitus using laboratory tests (7 tests).

8. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of anterior lobe pituitary neoplasms and craniopharyngioma.

9. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of pheochromocytoma and neoplasms of extra-adrenal paraganglia.

10. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of thyroid adenomas and thyroid carcinomas.

11. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of adrenal cortical adenomas and adrenal cortical carcinomas.

12. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of neuroblastoma and ganglioneuroma.

13. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of pancreatic islet cell neoplasms.

14. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of pinealomas.

15. Discuss the biomarkers of the various types of endocrine gland tumors.

16. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of multiple endocrine neoplasms (MEN) syndromes.

17. Discuss the tests for evaluating adrenal androgens, their indications, and their interpretation.

18. Discuss the biosynthesis of adrenal steroids and describe the relevant enzymatic defects leading to adrenal hyperplasia.

19. Discuss the utilization of fine-needle aspiration (FNA) of the thyroid in terms of basic methodology, indications, sensitivity and specificity.

20. Discuss tests used in evaluating plasma adrenal gland hormones, their indications and interpretation.

TOPIC №10. Disorders of endocrine function, part 2.

Equipment facilities: projector, educational video materials, clinical cases, MCQs.

Theory questions for self-control:

1. Primary adrenal cortical insufficiency (Addison's disease): causes, signs and symptoms, complications.
2. Secondary adrenal cortical insufficiency: causes, signs and symptoms, complications.
3. Cushing's syndrome: causes, forms, signs and symptoms, complications.
4. Adrenogenital syndrome: causes, pathogenesis, signs and symptoms, complications.
5. Hyperparathyroidism: causes, signs and symptoms, complications.
6. Hypoparathyroidism: causes, signs and symptoms, complications.
7. Type 1 diabetes mellitus: causes, pathogenesis, signs and symptoms, complications.
8. Type 2 diabetes mellitus: causes, pathogenesis, signs and symptoms, complications.
9. Features distinguishing type I from type II diabetes mellitus.
10. Male hypogonadism: causes, signs and symptoms, complications.
11. Female hypergonadism: causes, pathogenesis, signs and symptoms, complications.
12. Female hypogonadism: causes, pathogenesis, signs and symptoms, complications.

***Assignment questions* (ISW)**:

1. Compare and contrast infectious and subacute (granulomatous) thyroiditis in terms of age and sex distribution, etiology, pathogenesis, clinical manifestations, laboratory findings, complications and prognosis.

2. Compare and contrast subacute lymphocytic and Hashimoto's thyroiditis in terms of age and sex distribution, etiology, pathogenesis, clinical manifestations, laboratory findings, complications and prognosis.

3. Compare and contrast Graves' disease and Riedel's thyroiditis in terms of age and sex distribution, etiology, pathogenesis, clinical manifestations, laboratory findings, complications and prognosis.

4. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of Sheehan syndrome and empty sella syndrome.

5. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of diabetes mellitus and diabetes insipidus.

6. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of Waterhouse-Friderichsen syndrome and Addisonian crisis.

7. Describe methods of screening and following patients with diabetes mellitus using laboratory tests (7 tests).

8. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of anterior lobe pituitary neoplasms and craniopharyngioma.

9. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of pheochromocytoma and neoplasms of extra-adrenal paraganglia.

10. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of thyroid adenomas and thyroid carcinomas.

11. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of adrenal cortical adenomas and adrenal cortical carcinomas.

12. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of neuroblastoma and ganglioneuroma.

13. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of pancreatic islet cell neoplasms.

14. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of pinealomas.

15. Discuss the biomarkers of the various types of endocrine gland tumors.

16. Compare and contrast the etiology, pathogenesis, hormonal changes, clinical manifestations, laboratory findings, complications and prognosis of multiple endocrine neoplasms (MEN) syndromes.

17. Discuss the tests for evaluating adrenal androgens, their indications, and their interpretation.

18. Discuss the biosynthesis of adrenal steroids and describe the relevant enzymatic defects leading to adrenal hyperplasia.

19. Discuss the utilization of fine-needle aspiration (FNA) of the thyroid in terms of basic methodology, indications, sensitivity and specificity.

20. Discuss tests used in evaluating plasma adrenal gland hormones, their indications and interpretation.

TOPIC №11. Disorders of nervous system.

Equipment facilities: projector, educational video materials, clinical cases, MCQs.

Theory questions for self-control:

1. Arousal disorders: causes, levels, clinical manifestations.

2. Cognition disorders: types, causes, clinical manifestations.

3. Movement disorders: hyperkinesia: types, mechanism, clinical manifestations.

4. Movement disorders: hypokinesia: types, clinical manifestations.

5. Muscle tone disorders: types, clinical manifestations.

6. Mechanisms of increased intracranial pressure and cerebral edema.

7. Classification and characteristics of acute and chronic pain.

***Assignment questions* (ISW)**:

1. How is the nervous system protected?

2. Define the essential underlying abnormalities of amyloid and tau proteins in the most common causes of dementia.

3. Describe the mechanisms of pathologic findings seen in the traumatic brain injury.

4. Discuss congenital conditions involving the central nervous system in terms of their etiology and mechanisms.

5. How does mental health impact the pathology of the nervous system?

6. Name several diseases that involve the basal ganglia and describe how to distinguish among the diseases in terms of gross, microscopic, and clinical pathology.

7. Why do our emotions change when we become a teenager?

8. How stress levels affect the nervous system?

9. What emotions and how do they affect the nervous system?

10. How is information transmitted through the nervous system?

11. List common opportunistic infections that involve the CNS of immunocompromised individuals and describe their pathologic features.

12. What is neurogenic pathology? Give examples and describe mechanisms.

13. What is the pathology of higher nervous activity? Give examples and describe mechanisms.

14. Outline the mechanism and clinicopathologic features of Progressive Multifocal Leukoencephalopathy (JC virus).

15. Describe the autoimmune mechanism and clinicopathologic features of multiple sclerosis.

16. Describe the gross and microscopic features of acute suppurative meningitis and brain abscess; and name the organisms most commonly associated with each.

17. Describe the etiology, clinical features, gross pathology, histopathology of Alzheimer disease and the regions of the brain that are usually involved.

18. Compare and contrast the clinical, gross, and microscopic manifestations of common bacterial, viral, and fungal infections of the central nervous system.

19. Compare and contrast the clinicopathologic findings seen in two types of traumatic vascular injury – epidural and subdural hematoma.

20. What disease affects the nervous system? Give examples and describe mechanisms.

FINAL EXAM

Equipment facilities: MCQs.

List of questions for Final Exam preparation:

1. Causes of cell injury.
2. Mechanisms of cell injury.
3. Oxidative stress.
4. Cell death: types, causes.
5. Respiratory acidosis: causes, pathophysiology, signs and symptoms.
6. Respiratory alkalosis: causes, pathophysiology, signs and symptoms.
7. Metabolic acidosis: causes, pathophysiology, signs and symptoms.
8. Metabolic alkalosis: causes, pathophysiology, signs and symptoms.
9. Edema: mechanisms, examples.
10. Isotonic, hypertonic, hypotonic alterations of water and salt balance.
11. Hyponatremia: causes, signs and symptoms, treatment.
12. Hypernatremia: causes, signs and symptoms, treatment.
13. Hypokalemia: causes, signs and symptoms, treatment.

14. Hyperkalemia: causes, signs and symptoms, treatment.

15. Hypocalcemia: causes, signs and symptoms, treatment.

16. Hypercalcemia: causes, signs and symptoms, treatment.

17. Hypomagnesemia: causes, signs and symptoms, treatment.

18. Hypermagnesemia: causes, signs and symptoms, treatment.

19. Hypophosphatemia: causes, signs and symptoms, treatment.

20. Hyperphosphatemia: causes, signs and symptoms, treatment.

21. Hypovolemia: causes, pathophysiology, signs and symptoms, complications.

22. Estimating fluid loss: signs and symptoms.

23. Hypervolemia: causes, pathophysiology, signs and symptoms, complications.

24. Acute inflammation: cardinal signs, major components, stimuli.

25. Reactions of blood vessels in acute inflammation.

26. Reactions of leukocytes in acute inflammation.

27. Mechanism of the cellular response to inflammation.

28. Mediators of inflammation: sources, actions.

29. Outcomes of acute inflammation.

30. Chronic inflammation: causes, types.

31. Cell regeneration: types of cells.

32. Connective tissue repair.

33. Pyrogens: types. Fever patterns and their causes.

34. Pathophysiology and clinical manifestations of fever.

35. Humoral (B–cell) immunodeficiency: types, causes, manifestations.

36. Cellular (T–cell) immunodeficiency: types, causes, manifestations.

37. Combined B–cell and T–cell immunodeficiency: types, causes, manifestations.

38. Type I hypersensitivity reactions: mechanism, mediators, clinical examples.

39. Type II hypersensitivity reactions: mechanism, clinical examples.

40. Type III hypersensitivity reactions: mechanism, clinical examples.

41. Type IV hypersensitivity reactions: mechanism, clinical examples.

42. Host–versus–graft disease and Graft–versus–host disease: causes, mechanism, clinical examples.

43. Mechanisms of autoimmune disease.

44. Molecular basis of cancer: oncogenesis, cancer cell transformation.

45. Causes of cancer: genetics, viruses, immunologic mechanisms.

46. Causes of cancer: hormones, chemical carcinogens, ultraviolet and ionizing radiation.

47. Cancer cells characteristics.

48. Mechanisms of tumor invasion and metastasis.

49. Mechanism of tumor growth.

50. Paraneoplastic syndrome: mechanisms, clinical examples.

51. Classification of anemia according to underlying mechanism.

52. Blood loss anemia: types, causes, manifestations.

53. Extravascular and intravascular hemolysis: causes, mechanisms, manifestations.

54. Hereditary spherocytosis: cause, mechanism, signs and symptoms, complications.

55. Sickle cell anemia: cause, mechanism, signs and symptoms, complications.

56. Thalassemia: cause, mechanisms, signs and symptoms, complications.

57. Hemolytic disease of the newborn: cause, mechanism, signs and symptoms, complications.

58. Iron–deficiency anemia: causes, mechanism, signs and symptoms, complications.

59. Megaloblastic anemia: causes, mechanism, signs and symptoms, complications.

60. Aplastic anemia: causes, mechanism, signs and symptoms, complications.

61. Polycythemia: types, causes, mechanism, signs and symptoms, complications.

62. Leukocytosis: types, causes.

63. Leukopenia: types, causes.

64. Clinical manifestations of leukemia and their pathologic basis.

65. Acute leukemias: types, causes, signs and symptoms, complications.

66. Chronic leukemias: types, causes, signs and symptoms, complications.

67. Thrombocytopenia and thrombocytopathia: causes, manifestations.

68. Impaired synthesis of coagulation factors: causes, manifestations.

69. Hemophilia and von Willebrand disease: causes, signs and symptoms, complications.

70. Bleeding disorders: vascular disorders: causes, signs and symptoms, complications.

71. Conditions associated with hypercoagulability states: causes, manifestations.

72. Disseminated intravascular coagulation (DIC): causes, mechanism, signs and symptoms, complications.

73. Principal mechanisms of the cardiovascular dysfunction.

74. General causes of heart failure.

75. Right–sided heart failure: causes, mechanism, signs and symptoms, complications.

76. Left–sided heart failure: causes, mechanism, signs and symptoms, complications.

77. Systolic and diastolic dysfunction: causes, mechanism, signs and symptoms, complications.

78. Cardiogenic shock: causes, mechanism, signs and symptoms, complications.

79. Sinus tachycardia, sinus bradycardia and paroxysmal supraventricular tachycardia (PSVT): ECG features.

80. Atrial flutter, atrial fibrillation and ventricular fibrillation: ECG features.

81. AV block: degrees, ECG features.

82. Premature ventricular contraction (PVC) and ventricular tachycardia: ECG features.

83. Angina pectoris: causes, types, signs and symptoms, complications.

84. Myocardial infarction: causes, signs and symptoms, complications.

85. Essential (primary) hypertension: causes, mechanism, signs and symptoms, complications.

86. Secondary hypertension: causes, mechanism, signs and symptoms, complications.

87. Orthostatic hypotension: causes, mechanisms.

88. Atherosclerosis: causes, pathogenesis, complications.

89. Pathological basis of respiratory signs and symptoms.

90. Pathological breathing: types, causes, manifestations.

91. Dyspnea: causes, mechanisms.

92. Restrictive lung diseases: causes, pathogenesis.

93. Atelectasis: types, causes, signs and symptoms.

94. Pneumothorax: types, causes, signs and symptoms.

95. Bronchial asthma: causes, pathogenesis, signs and symptoms, complications.

96. Emphysema: causes, pathogenesis, signs and symptoms, complications.

97. Interstitial lung diseases: causes, pathogenesis, manifestations.

98. Diffusion abnormalities: causes, pathogenesis.

99. Pulmonary hypertension: types, causes, signs and symptoms, complications.

100. Pulmonary edema: causes, pathogenesis, signs and symptoms, complications.

101. Functions of the liver and manifestations of altered function.

102. Jaundice: types, causes, pathogenesis, manifestations.

103. Cirrhosis: causes, pathogenesis, signs and symptoms, complications.

104. Portal hypertension: causes, pathogenesis, signs and symptoms, complications.

105. Liver failure: causes, pathogenesis, signs and symptoms, complications.

106. Cholelithiasis: causes, signs and symptoms, complications.

107. Anorexia, nausea and vomiting: causes, mechanisms.

108. Gastrointestinal tract bleeding: causes, manifestations.

109. Dysphagia: causes, manifestations.

110. Gastroesophageal reflux: causes, pathogenesis, signs and symptoms, complications.

111. Gastritis: types, causes, mechanisms.

112. Peptic ulcer: causes, pathogenesis, signs and symptoms, complications.

113. Stress ulcers: types, causes, mechanisms.

114. Diarrhea: types, causes, mechanisms.

115. Constipation: causes, mechanisms.

116. Intestinal obstruction: types, causes, mechanisms.

117. Malabsorption: causes.

118. Malabsorption: signs and symptoms of impaired absorption of dietary constituents.

119. Acute pancreatitis: causes, pathogenesis, signs and symptoms, complications.

120. Pathological basis for urinary tract symptoms and signs.

121. Acute glomerulonephritis: causes, pathogenesis, signs and symptoms, complications.

122. Rapidly progressive glomerulonephritis: causes, pathogenesis, signs and symptoms, complications.

123. Chronic glomerulonephritis: causes, pathogenesis, signs and symptoms, complications.

124. Nephrotic syndrome: causes, pathogenesis, signs and symptoms, complications.

125. Acute pyelonephritis: causes, signs and symptoms, complications.

126. Chronic pyelonephritis: causes, signs and symptoms, complications.

127. Acute renal failure: types, causes.

128. Acute renal failure: phases, signs and symptoms, complications.

129. Chronic renal failure: causes, pathogenesis, signs and symptoms, complications.

130. Urinary tract obstruction: causes.

131. Nephrolithiasis: causes, pathogenesis, signs and symptoms, complications.

132. Hypopituitarism: causes, pathogenesis, signs and symptoms, complications.

133. Growth hormone deficiency: causes, pathogenesis, signs and symptoms, complications.

134. Growth hormone excess: causes, pathogenesis, signs and symptoms, complications.

135. Manifestations of hypothyroid and hyperthyroid states.

136. Hypothyroidism in adults: causes, signs and symptoms, complications.

137. Hypothyroidism in children: causes, signs and symptoms, complications.

138. Hyperthyroidism: causes, signs and symptoms, complications.

139. Primary adrenal cortical insufficiency (Addison’s disease): causes, signs and symptoms, complications.

140. Secondary adrenal cortical insufficiency: causes, signs and symptoms, complications.

141. Cushing’s syndrome: causes, forms, signs and symptoms, complications.

142. Adrenogenital syndrome: causes, pathogenesis, signs and symptoms, complications.

143. Hyperparathyroidism: causes, signs and symptoms, complications.

144. Hypoparathyroidism: causes, signs and symptoms, complications.

145. Type 1 diabetes mellitus: causes, pathogenesis, signs and symptoms, complications.

146. Type 2 diabetes mellitus: causes, pathogenesis, signs and symptoms, complications.

147. Features distinguishing type I from type II diabetes mellitus.

148. Male hypogonadism: causes, signs and symptoms, complications.

149. Female hypergonadism: causes, pathogenesis, signs and symptoms, complications.

150. Female hypogonadism: causes, pathogenesis, signs and symptoms, complications.

151. Classification and characteristics of acute and chronic pain.

Examples of MCQs for the Final Exam:

What does ATP deficiency in a cell lead to?

A. Decreased energy metabolism of the cell

B. Reversible damage to intracellular structures

C. Activation of calcium pumps

D. Activation of protein synthesis

E. Increased activity of membrane ATP-dependent pumps

What indicates compensation for metabolic acidosis?

A. pCO_2 in the blood < 35 mmHg

B. pCO_2 in the blood > 45 mmHg

C. pCO_2 in the blood < 45 mmHg

D. pCO_2 in the blood > 35 mmHg

E. pCO_2 in the blood < 25 mmHg

During hypovolemic shock:

A. Venous return to the heart decreases

B. Cardiac output increases

C. Mean arterial pressure increases

D. Tissue perfusion increases

E. The body's need for fluid decreases

During chronic inflammation, activated macrophages produce:

A. Interleukin-1

B. Vascular endothelial growth factor

C. Nerve growth factor

D. Transforming growth factor β

E. γ-interferon

Indicate a sign of Wiskott-Aldrich syndrome in the blood.

A. Decreased level of IgM and increased levels of IgA and IgE

B. Lack of mature B-lymphocytes, plasma cells, and IgG, IgA, IgM

C. Decreased level of IgG without a decrease in IgA and IgM levels

D. Decreased levels of IgG, IgA, IgM with normal B-lymphocyte counts

E. Decreased levels of IgA, IgE, and IgG_2

What happens immediately after the formation of a metastatic subclone during the process of hematogenous tumor metastasis?

A. Adhesion and invasion of the basement membrane

B. Passage through the extracellular matrix

C. Intravasation

D. Interaction with host lymphocytes

E. Formation of a tumor embolus

Indicate the cause of normocytic normochromic anemia.

A. Sepsis

B. Erythropoietin deficiency

C. Chemotherapy

D. Iron deficiency

E. Lead poisoning

What can stimulate the sickle cell crisis of erythrocytes in patients with sickle cell anemia?

A. Fever

B. Splenomegaly

C. Jaundice

D. Iron deficiency

E. Spectrin deficiency

Indicate the sign that confirms the presence of megaloblastic anemia.

A. Serum vitamin B12 level < 0.1 mg/mL

B. Mean corpuscular volume (MCV) < 120 fL

C. Hypoplasia of the red bone marrow without megaloblasts

D. Microcytosis and hypochromia of erythrocytes

E. Decreased MCH in erythrocytes

Indicate the clinical manifestation of iron deficiency anemia.

A. Rough, soft, spoon-shaped nails

B. Pain of varying intensity

C. Small body and large head

D. Coordination disorders

E. Bruises and petechiae on the skin

What type of leukocytosis is caused by rickettsioses?

A. Monocytosis

B. Eosinophilic

C. Neutrophilic

D. Basophilic

E. Lymphocytosis

What is the cause of night sweats and weight loss in patients with leukemias?

A. Increased metabolic rate of leukemic cells

B. Leukopenia

C. Anemia

D. Thrombocytopenia

E. Immaturity of the white cells

Activation of which coagulation factor initiates the extrinsic mechanism of coagulation hemostasis?

A. Tissue thromboplastin

B. Hageman factor

C. Prothrombinase

D. Proaccelerin

E. Fibrinogen

Indicate the cause of thrombocytopenia due to decreased platelet production.

A. Chemotherapy

B. Uremia

C. Hypersplenism

D. Thrombocytopenic purpura

E. DIC syndrome

What does arterial hypertension lead to?

A. Increased resistance to blood ejection from the left ventricle

B. Increased blood volume in the left ventricle

C. Decreased filling of the left ventricle with blood

D. Damage to the heart muscle

E. Increased oxygenation of the blood

During left-sided heart failure, there is:

A. Increased end-diastolic pressure in the left ventricle

B. Decreased pressure in the left atrium

C. Increased systemic venous pressure

D. Increased end-diastolic pressure in the right ventricle

E. Dilation of the right atrium

What leads to weakness and fatigue in patients with myocardial infarction?

A. Decreased perfusion of skeletal muscles

B. Cardiac conduction disturbances

C. Release of catecholamines into the blood

D. Reduced oxygen supply to the myocardium

E. Stimulation of the vasovagal reflexes

Indicate the sign of paroxysmal supraventricular tachycardia.

A. Additional P waves that are difficult to differentiate from preceding T wave

B. Sawtooth P-wave configuration (F-waves)

C. No P waves, or P waves are erratic, irregular, fibrillatory

D. No relation between P waves and QRS complexes

E. P waves are not discernible

On the ECG of a patient, premature QRS complexes are found that are followed by compensatory pause. What is this arrhythmia?

A. Premature ventricular contraction

B. Ventricular flutter

C. Atrial fibrillation

D. Junctional rhythm

E. Sinus tachycardia

What is the cause of wheezing during exhalation in patients with respiratory system pathology?

A. Narrowing of distal bronchi

B. Narrowing of the larynx, trachea, or proximal bronchi

C. Dilation of the larynx, trachea, or proximal bronchi

D. Dilation of distal bronchi

E. Increased gas content of thorax

Indicate the sign of lung collapse in patients with atelectasis.

A. Absent breath sounds on the affected side

B. Barrel-shaped chest

C. Prolonged expiration and grunting

D. Hyperresonance on chest percussion

E. Crackling beneath the skin on palpation

How does emphysema cause impaired gas diffusion in the lungs?

A. Decreases diffusion area

B. Increases cardiac output

C. Increases diffusion distance

D. Decreases permeability of lung capillaries

E. Decreases cardiac output

Pain with breathing in patients with pulmonary hypertension is a consequence of:

A. Lactic acid accumulation in the muscles

B. Left-sided heart failure

C. Right-sided heart failure

D. Increased cardiac output

E. Increased oxygen saturation of tissues

Indicate the consequence of impaired fat metabolism in the liver.

A. Increased cholesterol level in the blood

B. Hypoglycemia

C. Hypernatremia and hypokalemia

D. Cushing's syndrome

E. Increased ammonia level in the blood

Indicate the sign of prehepatic jaundice.

A. Increased unconjugated bilirubin in the blood

B. Increased conjugated bilirubin in the blood

C. Increased conjugated and unconjugated bilirubin in the blood

D. Increased alkaline phosphatase in the blood

E. Increased bile acid levels in the blood

What causes caput medusae in patients with portal hypertension?

A. Development of collateral channels

B. Increased pressure in the peritoneal capillaries

C. Accumulation of ammonia and toxins in the blood

D. Splenomegaly

E. Thrombocytopenia

Indicate a sign of impaired synthetic and storage functions of the liver in patients with liver failure.

A. Steatorrhea

B. Hepatic encephalopathy

C. Menstrual disorders

D. Jaundice

E. Drug intoxication

Indicate a sign of gastroesophageal reflux.

A. Burning epigastric pain radiating to the chest

B. Epigastric pain that worsens with eating

C. Nausea and anorexia

D. «Hunger like» epigastric pain

E. Epigastric pain that relieved by food

What increases the secretion of H^+ and hydrochloric acid in the stomach in patients with peptic ulcer?

A. Smoking

B. Bile salts

C. Pancreatic enzymes

D. Shock

E. Massive burns

What type of diarrhea develops as a result of bowel obstruction?

A. Motility

B. Secretory

C. Osmotic

D. Paralytic

E. Mechanical

What causes the activation of prothrombin in patients with acute pancreatitis?

A. Pancreatic gangrene

B. Fat necrosis

C. Shock

D. Hypoxia

E. Anuria

What can cause proteinuria?

A. Increased permeability of the glomerular basement membrane

B. Bladder tumors or trauma

C. Renal ischemia releasing renin

D. Renal failure

E. Impaired tubular concentration

Indicate a sign of hematuria in patients with glomerulonephritis.

A. Coffee-colored urine

B. Red-colored urine

C. Black-colored urine

D. Yellow-colored urine

E. Colorless urine

Indicate a sign that does NOT belong to nephrotic syndrome.

A. Oliguria

B. Massive proteinuria

C. Hypoalbuminemia

D. Generalized edema

E. Hyperlipidemia

Indicate a cause of acute prerenal kidney failure.

A. Embolism of the renal arteries

B. Crush injuries

C. Transfusion of incompatible blood

D. Bilateral renal vein thrombosis

E. Bladder obstruction

Which hormone stimulates contractions of the pregnant uterus?

A. Oxytocin

B. Prolactin

C. Follicle-stimulating

D. Estrogen

E. Progesterone

Indicate the cause of hyperfunction of the endocrine gland.

A. Ectopic hormone production by tumors

B. Production of biologically inactive hormone

C. Premature destruction of hormones

D. Absence of receptors in target cells

E. Autoimmune damage to the gland

Indicate a sign of hypothyroidism in children.

A. Dull facial expression

B. The infant is active and sleeps little

C. Increased body temperature

D. Diarrhea

E. Early tooth eruption

Indicate a sign of thyroid storm.

A. Vomiting

B. Extreme hypothermia

C. Bradycardia

D. Decreased blood pressure

E. Severe depression

Indicate a sign of aldosterone deficiency in patients with primary adrenal cortex insufficiency.

A. Hyponatremia

B. Fasting hypoglycemia

C. Decreased stress tolerance

D. Bronze skin color

E. Weight loss

What causes diabetes mellitus in patients with Cushing's syndrome?

A. Cortisol-induced insulin resistance

B. Aldosterone

C. Neurotransmitter effect of cortisol

D. Pharmacologic level effect of cortisol

E. Androgens

Indicate a sign of type I diabetes mellitus.

A. Associated with HLA

B. Obesity

C. No antibodies against β-cells

D. No need treatment with insulin

E. Presence of insulin resistance

In the case of female hormonal imbalance, what does early atherosclerosis indicate?

A. Estrogen deficiency

B. Progesterone deficiency

C. Estrogen excess

D. Progesterone excess

E. Deficiency of both estrogen and progesterone

Indicate a sign of acute pain.

A. Increased heart rate

B. No autonomic nervous system response

C. Continuous onset

D. Duration more than 6 months

E. Associated depression

Indicate a sign of chronic pain.

A. Decreased strength of relationships

B. Decreased salivation

C. Pupil dilation

D. Increased muscle tension

E. Increased heart rate

INDEPENDENT STUDENTS WORK (ISW)

The program provides for the implementation of 20 individual tasks on given topics in the form of a short essay (10 essays for each section of the discipline). The volume of the finished work (essay) should be 2-3 pages.

№	Content of assignment	Form of execution	Expected volume of finished work (pages)	Expected amount of hours for execution
1.	Work with scientific sources			3-4
2.	Essay writing	Essay	2-3	2

To complete an individual *assignment*, each higher education applicant is given one question on a topic that corresponds to the topic of the practical class, the material of which is not included in the part of the lecture or this practical class. The aim is to: read and analyze the text of scientific sources (articles, monographs); compare the information from different sources; synthesize of information sources; create a short essay that fully answers the question, describes the experience gained during the assignment writing, as well as shows the own thoughts on a given topic.

The deadline of one assignment is 2 weeks. If the applicant for higher education is unable to complete the assignment on time, then: assignments that were provided within one week after the deadline, receive a deduction of 10% mark; assignments that were sent with a delay of more than one week and less than 2 weeks will receive a deduction of 20% mark; assignments that were submitted 2 weeks or more late will not be accepted at all (except in special circumstances [e.g., death in the family, serious health problems]).

All essays are required to be designed according to an academic standard. To complete each task, applicants are provided with brief information on exactly how

to work with a particular source, what results are expected and what criteria will be used for assessment of their work.

The goals of independent individual assignments are to:

1. In-depth study of the topic of classes.
2. Acquire or improve skills in searching, processing and critical analysis of information from sources of various types.
3. Acquire or improve skills to clearly and concisely express own opinion in academic text.
4. Acquire or improve the skills of designing an academic text to meet standard requirements.

Where to find the questions for Assignments. You can find the List of Assignment questions in your Moodle. How would you know which question from this list is yours?

The question of your Assignment. In the course «Pathophysiology» on Moodle, the lists of students from all groups have been published in the E-register section. Each student in this list has their own serial number, which is assigned to him/her and does not change until the end of the academic year. Look at that list and remember *your serial number*, because that is the *number of your questions* in the List of Assignment questions. Thus, if you have a serial number - 3, that means that for each class you have to write an answer to the Assignment question number 3.

Where to send the completed Assignment. The completed Assignments must be sent to your teacher *by email*. When sending a completed assignment to your teacher by email, name your file as shown in the example below:

303-Adeleke-1.docx

- 303 is your group number
- Your last name
- 1 is a class number

CONTROL METHODS

There are two main forms of knowledge control:

Current control — is carried out at all practical classes in the form of an interview with the teacher or MCQs on the topic, as well as in the form of checking the individual work.

Final control (credit, exam) — is carried out at the last class of the relevant section of the discipline in the MCQs format: 80 MCQs [in each test question applicants need to choose one correct answer out of five; passing score – 50 correct answers [60%], time for taking the MCQs – 90 minutes]. A grade for a credit or exam is considered positive if it is 50 or more points.

SCORING SCHEME

According to the Regulations on the organization of the educational process in V. N. Karazin Kharkiv National University, «the maximum number of points that student can score as a result of studying a particular credit is ***200 points***, including for studying during the semester — ***120 points*** and for the final test work — **80 *points***»[1].

Grades for ***current control*** are given on a 20-point scale for the «General pathophysiology» section (12 points are considered as passing – 60%) and on a 12-point scale for the «Systemic pathophysiology» section (7 points – 60% are considered as passing). The maximum amount of points for all current classes is 120 points for each section.

[1] Положення про організацію освітнього процесу в V. N. Karazin Kharkiv National University (нова редакція) (Наказ ректора № 0211-1/342 від 10.07.2018 р.). – С. 75.

Score for 20-point scale	Percentage (%)	Score for 12-point scale	Percentage (%)
18–20	90–100	11–12	90–100
15–17	75–89	9–10	75–89
12–14	60–74	7–8	60–74
0-11	0–59	0-6	0–59

Grades for ***credit*** and ***exam*** are given on an 80-point scale (50 points – 60% are considered as passing). The maximum amount of points for credit and exam is 80 points, respectively.

Score for 80-point scale	Percentage (%)	Credit	Exam
72–80	90–100	Credited	Excellent
60–71	75–89	Credited	Good
50–59	60–74	Credited	Satisfactory
0-49	0–59	Not credited	Not satisfactory

The ***final score*** for the discipline is given on a 200-point scale (120 points are considered as passing – 60%). The maximum amount of points is 200 points.

Score for 200-point scale	Percentage (%)	Status
180–200	90–100	Excellent
150–179	75–89	Good
120–149	60–74	Satisfactory
0–119	0–59	Not satisfactory

ASSESSMENT CRITERIA

Assessment criteria of academic achievements of applicants in practical classes

Grades for *current control* are given on a 20-point scale for the «General pathophysiology» section (12 points are considered as passing – 60%) and on a 12-point scale for the «Systemic pathophysiology» section (7 points – 60% are

considered as passing). The maximum amount of points for all current classes is 120 points for each section.

Points	Criteria of assessment
20/ 12	It is given to the applicant, when he himself, competently and consistently, with full completeness, using the data of additional literature, answered to all questions with the manifestation of the ability to characterize a variety of biological phenomena and processes; clearly and correctly defines and discloses the content of scientific terms and concepts; independently and correctly performs individual work without any mistakes.
18-19/ 11	It is given to the applicant when he reveals complete knowledge of the actual material; is able to analyze, evaluate and disclose the essence of biological phenomena and processes; is able to establish causal relationships; is able to reach conclusions; independently and correctly performs individual work, but makes minor mistakes while using scientific terms and concepts.
15-17/ 9-10	It is given to the applicant when he understands the main content of the material; gives complete definitions of biological concepts and terms, makes minor mistakes in the sequence of presentation; independently, with the knowledge of the method, performs a individual work, but demonstrates some inaccuracies in the sequence of work.
13-14/ 8	It is given to the applicant when he understands basic material; makes significant mistakes; gives simple examples; the definition of biological concepts is not sufficient; characterizes the general features of biological objects; never finishes the individual work completely.
12/ 7	It is given to the applicant when he reveals the fragmentary understanding of the material; never finishes the individual work completely.
0	It is given to the applicant when he does not understand the basic material at all; never performs the individual practical work.

Assessment criteria of assignments (IWS)

Grades for *individual work of dtudents* (assignments) are given on a 20-point scale for the «General pathophysiology» section (12 points are considered as passing – 60%) and on a 12-point scale for the «Systemic pathophysiology» section (7 points – 60% are considered as passing). The maximum amount of points for all current classes is 120 points for each section.

Independence (absence of plagiarism), correctness and completeness of the assignment are taken into account. According to the «V.N. Karazin University Code

of Values», the university adheres to ***academic integrity***, so assignments in which the share of plagiarism exceeds 20% are returned without assessment; assignments with more than 60% of plagiarism are returned without permission to rework; and assignments that are completely copying another student's work are returned with prohibition to perform any other assignments.

Points	Criteria of assessment
20/ 12	It is given to the applicant when the he independently, competently and consistently, with knowledge of methods has performed the assignment, correctly applying scientific terms and concepts.
18-19/ 11	It is given to the applicant when the he independently, with knowledge of methods has performed the assignment, but has admitted small inaccuracies in sequence of the completed work.
15-17/ 9-10	It is given to the applicant when he independently, with knowledge of methods has performed the assignment, but has made a number of mistakes in sequence of the completed work.
13-14/ 8	It is given to the applicant when he is at the middle level in the method of work, the assignment was performed incompletely with significant mistakes.
12/ 7	It is given to the applicant when he badly revealed the essence of assignment, showed the initial idea of the study subject, did not complete the assignment.
0	It is given to the applicant when he has not written the assignment at all or has no idea about the subject of study, or the work is performed with a share of plagiarism over 60%.

Assessment criteria of academic achievements of applicants for the credit and exam

Grades for ***credit*** and ***exam*** are given on an 80-point scale (50 points – 60% are considered as passing). The maximum amount of points for credit and exam is 80 points, respectively.

To be permitted to the credit or exam, the applicant must complete all missed classes (lectures and practical).

The credit and the exam take place in a MCQs format: 80 MCQs [in each test question applicants need to choose one correct answer out of five; passing score –

50 correct answers [60%], time for taking the MCQs – 90 minutes]. A grade for a credit or exam is considered positive if it is 50 or more points.

MCQ questions for assessment contain lecture and theoretical material of the «General Pathophysiology» section. Test questions for the exam contain lecture and theoretical material from the «General Pathophysiology» and «Systemic Pathophysiology» sections. All theoretical material from the «Pathophysiology» course is provided to all applicants at the beginning of the academic year in electronic format. All lecture material (video lectures) from the «Pathophysiology» course» is provided to all applicants on the Moodle platform.

Refusal of an applicant of higher education to complete a written assignment during a credit or exam is considered as an unsatisfactory answer.

Applicants for higher education are notified of the results of the credit or exam written work no later than five working days after it was written. The applicant of higher education has the right to see the checked work and to receive an explanation of the received assessment.

In case of disagreement with the assessment, the applicant of higher education has the right to submit a written appeal to the head of the department on the day of the announcement of the assessment or the next working day, specifying the reasons for disagreement with the assessment. The head of the department together with the examiners (or, if necessary, other specialists) considers the appeal within three days and verbally notifies the higher education applicant about the results of the review.

If, based on the results of the credit or exam, the level of knowledge of the applicant of higher education is less than 50 points, they have the right to retake the credit or exam twice before the end of the examination session according to the official timetable. The points of a successful retake are summed with the previous attempts, and the average score is calculated, which cannot be less than 50 points. If the applicant of higher education missed the credit or exam without an officially confirmed valid reason, the maximum score for the credit or exam after retaking is 50 points.

Assessment criteria

of personal educational achievements of applicants

Points	Criteria of assessment
2	Selection of two video or two audio materials from the particular sections of the discipline.
4	Speech at a meeting of a student scientific circle.
6	Participation in a student's Olympiad on discipline or work on a student's scientific forum in the form of publication of abstracts.
8	Work on student's scientific forum in the form of a poster presentation.
10	Work on a student's scientific forum in the form of an oral report.
12	Prize-winning place for participation in the student's Olympiad on discipline or prize-winning place for participation in the work of the scientific forum.

RECOMMENDED LITERATURE

Main:

1. General pathophysiology: guidance for students [for the 3rd year medical students] / compiled by Protsenko E. S., Remnyova N. A. – V.N. Karazin Kharkov National University, 2022 – 140 p.;

2. Systemic pathophysiology. Part I: guidance for students [for the 3rd year medical students] / compiled by Protsenko E. S., Remnyova N. A. – V.N. Karazin Kharkov National University, 2022 – 124 p.;

3. Systemic pathophysiology. Part II: guidance for students [for the 3rd year medical students] / compiled by Protsenko E. S., Remnyova N. A. – V.N. Karazin Kharkov National University, 2022 – 136 p.;

4. Step-1. Pathophysiology / Protsenko E. S., Remnyova N. A. – V.N. Karazin Kharkov National University, 2021 – 122 p.

Additional:

1. Cohen, B. J., & Taylor, J. J. (2015) Memmler's The human body in health & disease (10th ed.). Philadelphia: Lippincott Williams & Wilkins.

2. Despopoulos, A., Silbernagl, S. (2015). Color Atlas of Physiology (7th ed). Stuttgart–New York: Thieme.

3. Guyton, A.C., Hall, J.E. (2020). Textbook of medical physiology (14th ed). Elsevier Health Sciences.

4. Kumar, V., Abbas, A., Fausto, N., Aster, J. (2020). Robbins and Cotran Pathologic Basis of Disease (10th ed). Elsevier Health Sciences.

5. Lazenby, R. B., & Corwin, E. J. (2013). Handbook of pathophysiology (4th ed). Philadelphia, Wolters Kluwer/Lippincott Williams & Wilkins Health.

6. Porth, C.M. (2018). Pathophysiology: Concepts of altered health states (10th ed.). Philadelphia: Lippincott Williams & Wilkins.

7. Silbernagl, S., Lang F. (2016). Color Atlas of Pathophysiology (3rd ed). Stuttgart–New York: Thieme.

8. Underwood, J.C.E. (2019). General and Systematic Pathology (7th ed). Elsevier Health Sciences.

USEFUL WEB SOURCES

1. **MedicalStudent.com** - provides links to online medical textbooks, medical journals, continuing education/board exam information, and more. https://www.medicalstudent.com

2. **MedicalMatrix** – offers both paid and free links to online textbooks, CME, and medical journals. https://www.healio.com/

3. **WebPath** -pathology images and text for medical education. This resource is designed for students and workers in the health care sciences studying pathology. https://webpath.med.utah.edu

4. **FreeBookCenter** - provides free e-books for medical students. http://www.freebookcentre.net/medical_text_books_journals/medical_text_books_online.html

5. **Omni Medical Search** – this search engine is dedicated to gathering information from some of the top medical professional websites. The information provided by the search engine is primarily from peer level sources. http://www.omnimedicalsearch.com/

6. **Bright Knowledge** – students are provided with essential guides for their career, education, and general student life. https://www.brightknowledge.org/

7. **PubMed** - provides access to citations going back for the past 40 years. https://www.ncbi.nlm.nih.gov/pubmed/

8. **eMedicine** - offers over 6,500 clinical articles written by contributing physicians. https://emedicine.medscape.com/

9. **Merriam Webster Online Dictionary** – famous medical dictionary trusted by many. You can also go to the pronunciation and explanation guides for more help. https://www.merriam-webster.com/

10. **Medexam** - prepares for many of your med school exams. http://www.medexam.net/

11. **Supercourse** - almost 3500 lectures from scientists around the world in this repository of information on public health and prevention. http://www.pitt.edu/~super1/

ONLINE STUDYING

Teaching platform. Online teaching is carried out on the *Moodle* platform – "Pathophysiology" Course.

Preparation for classes. Preparation for classes is carried out by students independently on the basis of textbooks *General Pathophysiology*, *Systemic*

Pathophysiology (parts 1 and 2), *Lectures* (video lectures), *Krok-1 questions book* and training videos. All books are free to download on our Moodle platform.

Classes. The classes and knowledge monitoring are carried out by your teacher in the *Google Meet* video chat according to your official Timetable (see The rules of conduct for online Classes, Credit and Final Exam below).

Rework of the classes. You have to rework your classes to your teacher in the *Google Meet* video chat according to the Rework Timetable (every teacher has his/her own Timetable of reworks, so they will announce it to you separately).

Academic honesty. Students found cheating in any form, will receive an automatic failing grade for the course and their names will be sent to the Dean of the Medical School for further disciplinary action.

Communication with teachers. For communication with teachers, email or a convenient messenger can be used by agreement.

RULES OF ONLINE CONDUCT

General rules:

1. The class, module and final exam will be video recorded.
2. The student taking a video proctored online class, module and final exam must prove their identity prior to the examination by showing their ID card, passport and credit book.
3. The student is obliged to show their room by making a 360° film of the room with the webcam.
4. The student must be dressed and behave decently at all times.
5. The student's room must be quiet and tranquil. There may not be sounds from music, television or any other sounds.

6. The webcam and microphone required for the class, module and final exam must be enabled and running.

7. The webcam must be focused on the student taking the class, module and final exam at all times.

8. The student's face must be positioned in the center of the webcam view and must be visible throughout the duration of the class, module and final exam.

9. Nothing may cover the lens of the webcam at any time during the class, module and final exam.

10. The student must face the webcam during the class, module and final exam constantly.

11. Wearing ear plugs or headphones is not allowed.

12. There may not be any other people in the student's room.

13. There may not be other computers or similar devices running in the student's room.

14. Lighting must be "daylight" quality and overhead. If overhead lighting is not possible, the source of light should not be behind the student.

15. On the desk or other workplace, there may not be anything except a computer/tablet/cell phone and, in case the computer/tablet/cell phone does not have an internal webcam, an external web camera. All other materials have to be removed, unless explicitly permitted (ID card, passport and credit book).

Credit and Final Exam rules:

16. The teacher doesn't have to interpret given questions for the student as class, module and final exam questions require the student to make his/ her own interpretations or assumptions.

17. The question review is not allowed, providing no opportunity to change student answers.

18. The student doesn't have to receive assistance from the teacher, or anyone else, during the class, module and final exam.

19. The student doesn't have to repeat questions of the teacher out loud.

20. The student cannot access any resources during the class, module and final exam. This means no resources of any kind are allowed, including external websites, other software applications, or any other hardcopy resources. All other web browser tabs must be closed during the class, module and final exam. Cell phones and all other electronic devices must be turned off for the class, module and final exam.

21. The student may not leave the room after starting the class, module and final exam. Everything must be completed in one sitting. Restroom or other breaks aren't allowed.

22. The class, module and final exam has a specific amount of time allotted (max 15 minutes for each student) and a specific number of questions (2 questions for class and 3 questions for module and final exam).

23. Questions must be answered orally. No keyboard is required and typing is not allowed.

24. The student has to answer the teacher's questions immediately without any hesitation.

Cheating:

25. Cheating is a serious offence and subject to disciplinary action under Code of Ethics. Any evidence of cheating that occurs during the class, module and final exam would be noted in detail by the teacher. The teacher, who becomes aware that the student may have cheated or have failed to follow the rules in any way, is obligated to notify the Head of Department and Dean Office.

Examples of the cheating are:

- ✓ Any recording of the screens, including taking screenshots, pictures, or video.
- ✓ Copying the questions or answers.

- ✓ Leaving mobile devices/smart phones, other web browsers, software applications, or other computers on during the class, module and final exam.
- ✓ Having access to or consulting notes or books during the class, module and final exam.
- ✓ Allowing other individuals to be present, to come in and out of the room during the class, module and final exam.
- ✓ Talking or otherwise communicating with another person during the class, module and final exam.
- ✓ Arranging to have another person take a class, module and final exam for the student.

Breaking the class, credit and final exam rules:

26. If the student breaks the Rules, the student can be excluded from participating or continuing the class, module and final exam. The teacher may also decide to nullify, not assess and/or not establish a result for the (partially) completed class, module and final exam.

Електронне навчальне видання комбінованого використання
Можна використовувати в локальному та мережному режимі

Проценко Олена Сергіївна
Ремньова Наталія Олексіївна

ПАТОФІЗІОЛОГІЯ

Методичні рекомендації
для студентів ІІІ курсу медичного факультету

В авторській редакції

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Харківський національний університет імені В. Н. Каразіна,
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