ASSESSMENT FUND

of the discipline «Pathological physiology»

Level of higher education

SPECIALTY

Field of study

31.05.01 - RF, 560001 -KR General medicine

2026

The assessment fund is intended for control of knowledge of students in the direction of training (specialty) «General medicine» on discipline «Pathological physiology».

The assessment fund was considered and approved at the meeting of the department
Pathology and forensic medicine

Protocol № 1 from "26" 08.2026 year

The Head of Department
Pathology and forensic medicine — Professor Kakeev B.A.

Developped by:
Associate professor — Abdukarimova E.E.

1. LIST OF COMPETENCES WITH INDICATION OF STAGES OF THEIR FORMATION IN THE PROCESS OF MASTERING A DISCIPLINE

Formed competencies	Planned results of training in the discipline that characterizes the stages of formation of competences	Types of assessment tools/ section code in this document
PC-5: Able to define pathological states in a patient, symptoms and syndromes of diseases, clinical entities in accordance with International Statistical Classification of Diseases and Related Health Problems of the X review.	**Knowledge of:** - procedures of identifying main pathological states in a patient, symptoms and syndromes of diseases, clinical entities; - specific features of identifying various types of pathological states, diseases symptoms and syndromes, clinical entities in accordance with ISCD of the X review; - basic syndromes of organ and system abnormalities and their features in differential exclusions of various nosological entities in compliance with ISCD of the X review.	**Block A, D** - reproductive level tasks - oral questioning; - baseline test questions
	Skills: - comprehend the obtained results of investigating the main clinical entitites of diseases; - analyze various types of pathological states, diseases symptoms and syndromes, clinical entities in accordance with ISCD of the X review; - indicate practical utility when comparing specific abnormal diseases' syndromes and symptoms.	**Block B, D** - reconstructive level tasks - solving situational tasks; - control questions of the final level.

	Expertise: - experience in identifying main pathological states in a patient, symptoms and syndromes of diseases; - methods of searching, identifying and classifying the main pathological states, diseases' syndromes and symptoms clinical entities in accordance with the ISCD of the X review; - experience in independent reasoning of aggregating various symptoms and syndromes into clinical entities in accordance with ISCD of the X review.	**Block C, D -** practice-oriented and/or research level tasks

2. FLOW CHART OF THE DISCIPLINE/PRACTICE

Pathological Physiology planning sheet

Discipline of Pathological Physiology

Field of study/specialization 560001 General medicine

Course/semester 2/4

Credit units (CU) 5

Scope of Testing Semester: credit

IV SEMESTER

Name of moduls of discipline according to course outline	Control	Forms of control	Credit minimum	Credit maximum	Graphic of control (weeks of semester)
Modul 1					
«General pathophysiology. General etiology, pathogenesis, damaging effects of external factors, cell injury»	Formative assessment	Attendance,activity, conspect (lectures and practical class), IWS report situational case, test	**3**	**6**	**3**
	Midterm examination	Written control work, oral interview, tests.	**3**	**6**	
Modul 2					
«Pathophysiology of reactivity and	Formative assessment	Attendance,activity, conspect (lectures and	**3**	**6**	

hypersensitivity. Allergy».		practical class), IWS report situational case, test			**6**
	Midterm examination	Written control work, oral interview, tests.	**4**	**6**	
Modul 3					
« Pathophysiology of local circulation, thrombosis, embolism, inflammation, fever, infection process».	Formative assessment	Attendance,activity, conspect (lectures and practical class), IWS report situational case, test	**6**	**12**	**10**
	Midterm examination	Written control work, oral interview, tests.	**7**	**12**	
Modul 4					
«Pathophysiology of Carbohydrate, Protein, Lipid metabolism».	Formative assessment	Attendance,activity, conspect (lectures and practical class), IWS report situational case, test	**4**	**6**	**13**
	Midterm examination	Written control work, oral interview, tests.	**4**	**6**	
Modul 5					
«Pathophysiology of Acid-base imbalance	Formative assessment	Attendance,activity, conspect (lectures and practical class), IWS	**3**	**5**	

Tumor, Hypoxia»		report situational case, test			**16**
	Midterm examination	Written control work, oral interview, tests.	**3**	**5**	
Total			**40**	**70**	
Midterm examination (credit without mark) (report/presentation)			**20**	**30**	
Semester rating			**60**	**100**	

Pathological Physiology planning sheet

Discipline of Pathological Physiology

Field of study/specialization 31.05.01. General medicine
Course/semester 3/5

Credit units (CU) 4

Scope of testing semester: examination

Name of moduls of discipline according to course outline	Control	Forms of control	Credit minimum	Credit maximum	Graphic of control (weeks of

					semester)
Modul 1					
«Modul 1 "Typical forms of pathology of system of external respiration and cardio-vascular system»	Formative assessment	Attendance,activity, conspect (lectures and practical class), IWS situational case, test, report	**5**	**7**	**28**
	Midterm examination	Written control work, oral interview, tests.	**5**	**9**	
Modul 2					
Modul 2 «Typical forms of blood pathology»	Formative assessment	Attendance,activity, conspect (lectures and practical class), IWS situational case, test, report	**5**	**7**	**33**
	Midterm examination	Written control work, oral interview, tests.	**5**	**9**	
Modul 3					
Modul 3 «Typical forms of liver pathology and gastro-intestinal tract pathology.	Formative assessment	Attendance,activity, conspect (lectures and practical class), IWS situational case, test, report	**6**	**10**	**37**

Typical forms of kidneys pathology»	Midterm examination	Written control work, oral interview, tests.	**6**	**14**	
Modul 4					
Modul 4 «Typical forms of endocrine and nervous system pathology»	Formative assessment	Attendance,activity, conspect (lectures and practical class), IWS situational case, test, report	**4**	**7**	**41**
	Midterm examination	Written control work, oral interview, tests.	**4**	**7**	
Total			**40**	**70**	
Midterm examination (exam) written answering by tickets.			**20**	**30**	
Semester rating			**60**	**100**	

3. STANDARD CONTROL TASKS AND OTHER MATERIALS NECESSARY FOR ASSESSING THE PLANNED LEARNING OUTCOMES OF THE DISCIPLINE/PRACTICUM (ASSESSMENT TOOLS)

Block A

A. 1. Survey questions

CONTROL QUESTIONS FOR IV SEMESTER:

UNIT 1: **General pathophysiology. General etiology, pathogenesis, damaging effects of external factors, cell injury**

TO KNOW:

1. The main historical stages of pathophysiology worldwide. Scientists who contributed to the development of pathophysiological as science.

2. The main sections of pathological physiology: General nosology, typical pathological processes, pathological physiology of body systems. Their characteristic.

3. Basic concepts and categories of General nosology: health, disease, disease periods.

4. Etiological factors of the disease: definition. Classification, their role at different stages of the disease. Conditions of occurrence of the disease: definition, types, meaning

5. Pathophysiological characteristics of the periods of overheating. The main changes in the function of organs and systems and metabolism during overheating.

6. The factors on which the damaging effect of the electric current depends.

7. Definitions of "cell damage". The main types of cell damage. Exogenous and endogenous factors (causes and conditions of damage) cells.

8. The outcomes of cell damage. Dystrophy, dysplasia, necrosis, apoptosis.

9. Definition of the concepts of reactivity and resistance of the organism. Types and forms of reactivity, their characteristics. Pathological reactivity.

10.The subject, the tasks of pathophysiology. Clinical pathophysiology, its goals and objectives. Its place among other medical sciences, the importance for the clinic.

11. Pathological process, typical pathological process, pathological condition, pathological reaction.

12. Etiology, definition. Preceding theories and modern general theses of etiology.

13. The concept of pathogenesis. The main link and the "vicious circle" in the pathogenesis of disease.

14. Adaptive (protective-adaptive, compensatory) mechanisms are an integral part of pathogenesis. Mechanisms of recovery.

15. External and internal causal factors of disease occurrence, their characteristics, general properties and features.

16. Overheating (hyperthermia) - factors that cause overheating of the body (causes and conditions), mechanisms for the development of hyperthermia.

17. Kinetosis: causes and clinical manifestations. Types of acceleration, the mechanism of action of accelerations on the body. Overloading views.

18. The mechanism of the action of electric current on the body.

19. Typical mechanisms of cell damage.

20. The role of free radicals in cell damage. Peroxide oxidation of lipids.

21. Adaptive and pathogenic value of cell death in normal and pathological conditions.

22. The subject, the tasks of pathophysiology. Clinical pathophysiology, its goals and objectives. Its place among other medical sciences, the importance for the clinic.

23. Methods of pathological physiology. Experiment as the main method of pathophysiology. Importance of the experiment. Types and stages of the experiment.

24. Signs of the disease. Nature of the course of the disease. Relapses, remissions. Outcomes and complications of the disease.

25. Pathogenic effect of the thermal factor. Burns, types and degrees of burns and their characteristics.

26. Burn disease, clinical stages and their pathophysiological characteristics. The pathogenesis of burn shock.

27. Pathogenesis of heat and sun stroke, pathogenetic principles of first aid.

Tests:

1.The term prognosis refers to the:

a. period of recovery and return to a normal state.

b. expected outcome of the disease.

c. mortality and morbidity rates for a given population.

d. typical collection of signs and symptoms.

2. When prolonged ischemia occurs to an area of the heart, the resulting damage is referred to as:

a. atrophy.

b. liquefactive necrosis.

c. apoptosis.

d. infarction.

3. During the evaluation process for a new therapy’s effectiveness and safety, a double blind study may be conducted during:

a. the first stage.

b. the second stage.

c. the third stage.

d. any of these stages.

4. Why are the predisposing factors for a specific disease important to health professionals?

a. To predict the prognosis

b. To determine treatments

c. To develop preventive measures

d. To develop morbidity statistics

5. Cell damage may be caused by exogenous sources such as:

a. abnormal metabolic processes.

b. certain food additives.

c. genetic defects.

d. localized hypoxia.

6. Which of the following is usually included in a medical history?

1. Past illnesses or surgeries

2. Current illnesses, acute and chronic

3. Prescribed medication or other treatments

4. Nonprescription drugs and herbal remedies

5. Current allergies

a. 1, 3

b. 2, 4, 5

c. 1, 3, 4

d. 1, 2, 3, 4, 5

7. A situation when there is a higher than expected number of cases of an infectious disease within a given area is called a/an:

a. epidemic.

b. exacerbation.

c. morbidity.

d. pandemic.

8. The term pathogenesis refers to:

a. the development of a disease or sequence of events related to tissue changes involved in the disease process.

b. the determination of the cause(s) involved in the development of a malignant neoplasm.

c. the specific signs and symptoms involved in the change from an acute disease to a chronic disease.

d. the changes in cells of affected tissue that result in necrosis.

9. When a group of cells in the body dies, the change is called:

a. ischemia.

b. gangrene.

c. hypoxia.

d. necrosis.

ANS: D

Which of the following is the best definition of epidemiology?

a. The science of tracking the occurrence and distribution of diseases

b. The relative number of deaths resulting from a particular disease

c. Identification of a specific disease through evaluation of signs and symptoms

d. The global search for emerging diseases

21. Which of the following can cause cell injury or death?

1. Hypoxia

2. Exposure to excessive cold

3. Excessive pressure on a tissue

4. Chemical toxins

a. 1, 2

b. 2, 4

c. 1, 3, 4

d. 1, 2, 3, 4

22. All of the following are part of the Seven Steps to Health EXCEPT:

a. follow cancer screening guidelines.

b. use sun block agents whenever exposed.

c. participate in strenuous exercise on a regular daily basis.

d. choose high fiber, lower fat foods.

23. The term disease refers to:

a. the period of recovery and return to a normal healthy state.

b. a deviation from the normal state of health and function.

c. the treatment measures used to promote recovery.

d. a basic collection of signs and symptoms.

24. A collection of signs and symptoms, often affecting more than one organ or system, that usually occur together in response to a

certain condition is referred to as a (an):

a. acute disease.

b. multiorgan disorder.

c. syndrome.

d. manifestation.

25. All of the following statements are correct about cell damage EXCEPT:

a. The initial stage of cell damage often causes an alteration in metabolic reactions.

b. If the factor causing the damage is removed quickly, the cell may be able to

recover and return to its normal state.

c. If the noxious factor remains for an extended period of time, the damage becomes

irreversible and the cell dies.

d. Initially, cell damage does not change cell metabolism, structure, or function.

26. Which of the following conditions distinguishes double blind studies used in health research?

a. Neither the members of the control group or the experimental group nor the

person administering the treatment knows who is receiving the experimental therapy.

b. Both groups of research subjects and the person administering the treatment know who is receiving the experimental therapy.

c. The research subjects do not know, but the person administering the treatment knows who is receiving placebo or standard therapy.

d. Only members of the control group know they are receiving standard therapy.

27. If the data collected from the research process confirm that the new treatment has increased effectiveness and is safe, this is called:

a. the placebo effect.

b. evidence-based research.

c. blind research studies.

d. approval for immediate distribution.

28. A short-term illness that develops very quickly with perhaps a high fever or severe pain is called:

a. acute.

b. latent.

c. chronic.

d. manifestation.

29. The term prognosis refers to the:

a. period of recovery and return to a normal state.

b. expected outcome of the disease.

c. mortality and morbidity rates for a given population.

d. typical collection of signs and symptoms.

30. When prolonged ischemia occurs to an area of the heart, the resulting damage is referred to as:

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c. the third stage.

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1. Past illnesses or surgeries

2. Current illnesses, acute and chronic

3. Prescribed medication or other treatments

4. Nonprescription drugs and herbal remedies

5. Current allergies

a. 1, 3

b. 2, 4, 5

c. 1, 3, 4

d. 1, 2, 3, 4, 5

35. A situation when there is a higher than expected number of cases of an infectious disease within a given area is called a/an:

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b. exacerbation.

c. morbidity.

d. pandemic.

36. The term pathogenesis refers to:

a. the development of a disease or sequence of events related to tissue changes involved in the disease process.

b. the determination of the cause(s) involved in the development of a malignant neoplasm.

c. the specific signs and symptoms involved in the change from an acute disease to a chronic disease.

d. the changes in cells of affected tissue that result in necrosis.

BLOCK B

SKILLS:

Task №1.

The child is 14 years old, who is in the clinic for tuberculous gonitis (inflammation of the knee joint) pain for two years. Onset of the disease is associated with injury of the knee joint in the fall. Often suffered from colds, complained of a constant lack of appetite. The fatness of the child is markedly reduced.

Questions:

What is the cause of the disease and what is the condition?

Task № 2

A young man, K., 18 years old, constantly performing excessive muscular loadings during trainings, was admitted to the surgical department to conduct a planned operation for hernia of the white line of the abdomen. During intubation of the trachea, trismus developed, in connection with which he was given a fluorotan anesthesia, and dithilin was administered as a muscle relaxant. 10 minutes after the start of the operation, the patient developed paroxysmal tachycardia, stiffness of the spinal muscles, marbled cyanosis. The skin has become hot to the touch. The temperature has risen sharply (42.0 ° C). The operation was discontinued, K. lined with ice. Biochemical rapid analysis of

blood: lactate 9.0 mmol / l (norm 0.6 - 1.5 mmol / l); pyruvate 0.3 mmol / l (normal 0.05 - 0.15 mmol); K + 6.0 mmol / l (norm 3.5-5.0 mmol / l); Mg2 + 1.5 mmol / l (norm 0.8 - 1.3 mmol / l).

Questions

1) What is the pathological condition developed in K.?

2) What are the mechanisms for the development of this state?

3) Why did S. have acidosis and hyperkalemia?

4) What is the prevention of this condition?

5) What are the principles of treatment of such conditions?

UNIT 2. **Pathophysiology of reactivity and hypersensitivity**
BLOCK A
TO KNOW

1. Definition of the concepts of reactivity and resistance of the organism. Types and forms of reactivity, their characteristics. Pathological reactivity.

2.

Directed change of individual and group reactivity as the most important means of prevention and therapy of diseases.

3. The resistance of the organism, types and their characteristics. Cellular and humoral factors that provide resistance of the body.

4. Definition and General characteristics of allergies. Classification of allergic conditions.

5. Anaphylaxis, stages, characteristics. Sensitization: active and passive.

6. Definition and classification of hereditary forms of pathology.

7. Factors determining reactivity: the role of the genotype, age, sex, constitution. The influence of the external environment, social and environmental factors on the reactivity of the organism.

8. Allergens. Types of allergens and their characteristics.

9. General pathogenesis and stages of allergic reactions.

10. Etiology of hereditary and congenital diseases. Mutagens and their types: exogenous (physical, chemical, biological), endogenous.

11. Mutations and their types. Characteristics of teratogenes.

12. The mechanism of the action of electric current on the body.

13. Typical mechanisms of cell damage.

14. The role of free radicals in cell damage. Peroxide oxidation of lipids.

15. Adaptive and pathogenic value of cell death in normal and pathological conditions

Etiology and pathogenesis of the development of type I allergic reactions by Gell and Coombs. Clinical forms.

16. Characteristics of allergens, mediators and mechanisms of development of cytotoxic and cytolytic allergic reactions of type II according to Gell and Coombs, their role in pathology. Clinical forms.

17. Characteristics of allergens, mediators and mechanisms of development of immunocomplex allergic reactions of type III according to Gell and Coombs, their role in pathology. Clinical forms.

18. Characteristics of allergens, mediators and mechanisms for the development of type IV allergic reactions according to Gell and Coombs, their role in pathology. Clinical forms.

19. Pathogenesis of anaphylactic shock in humans. Features of the course of experimental anaphylactic shock in guinea pigs, dogs and rabbits.

20. Desensitization, hyposensitization. General principles of diagnosis, prevention and treatment of allergic diseases.

21. Methods of diagnosis of hereditary diseases (demographic, genealogical, twin method, cytogenetic, biochemical, immunological method, dermatoglyphic, experimental).

1. Resistance is:
1.The ability of the living system to respond precisely to irritants.
2. The ability of the living system not to respond to irritants.
3. The ability of the living system to oppose irritants.
4. The ability of the living system of self – improvement.
5. 1, 4.

2. The most precise definition of reactivity is:
1. The ability of the living system to adapt.
2. The response of the living system to irritants.
3. The combination of reactions of the living system.
4. The ability of the living system to change its living activity
5. The continuous adaptive alterability of the organisms.

3. The external factors, influencing reactivity and resistance are:
1. Environment.
2. Ecology.
3. Nutrition.
4. Gender and age
5. 1, 2, 3.
6. 1, 3, 4.

4. The internal factors, influencing reactivity and resistance are:
1. Social conflicts.
2. Psychological factors.
3. Climate and geographic influence.
4. Heredity, gender and age.
5. Lifestyle of the individual.

5. According to the reaction magnitude, reactivity is classified as:
1. Normergic, hyperergic, hypergic, super-allergic.

2. Normergic, hyperergic, hypergic, anergic.
3. Local, segment, diffuse, generalized.
4. Organelle, cellular, organ, organism.
5. Paralytic, kinetic, tonic.

6. Natural resistance is:
1. Acquired.
2. Postnatal.
3. Adaptively developed.
4. Neurogenic developed.
5.Genetically pre-programmed.

7. What type of resistance is acquired by vaccination:
1. Natural, absolute, active.
2. Natural, relative, passive.
3. Acquired, artificial, active.
4. Acquired, artificial, passive.
5. Acquired, natural, active.
6. Acquired, natural, passive.

8. The type of resistance, acquired by hyperimmune serum therapy:
1. Acquired, artificial, active.
2. Acquired, artificial, passive.
3. Natural, absolute, passive.
4. Natural, relative, active.
5. Acquired, natural, active.
6. Acquired, natural, passive.

9. What type of resistance is acquired following an infectious disease:
1. Acquired, artificial, active.
2. Acquired, artificial, passive.
3. Natural, absolute.
4. Acquired, natural, active.
5. Acquired, natural, passive.

10. Allergy is developed by the following mechanisms:
1. Non-immunological mechanisms.
2. Mechanisms of direct injury.
3. Mechanisms of disturbed reflex arc.
4. Immunological mechanisms .
5. 1, 2.

11. Allergy is:
1. Normergic immunological reactivity.
2. Hyperergic immunological reactivity.
3. Reaction of idiosyncrasy.
4. Explosive cellular – tissue distress.
5. Non – specific hypersensitivity.

12. The main types of allergic reactions are:
1. Of humoral type.
2. Of neuro – reflex type.

3. Of cellular type.
4. Of neuroendocrine type.
5. 1, 3. 6. 2, 4.

13. The main types of disturbed immune response are:
1. Immune hypersensitivity.
2. Immune idiosyncrasy.
3. Disturbed auto - tolerance.
4. Immune insufficiency.
5. 1, 3, 4. 6. 1, 2, 3, 4.

14. Humoral immunity deficiency is characterized:
1. Failure to recognise foreign antigens.
2. Lack of activation of T-helpers.
3. Suppressed proliferation and differentiation of B – Lymphocytes.
4. Disturbed synthesis of specific immunoglobulines.
5. 3, 4. 6. 1, 2, 3, 4.

15. The alteration of which mechanisms does not disturb the immune response:
1. Phagocytosis, processing and presentation of the antigen.
2. Activation of T – helper Lymphocytes.
3. Activation of cytotoxic T – cells and B – Lymphocytes.
4. Reflex activation of the HPA axis (hypothalamus – pituitary – adrenal)
5. Binding and clearance of antigens

BLOCK B

SKILLS:

TASK number 1

The control group of animals and adrenalectomics were subjected to prolonged exposure to low temperature, after which the degree of endurance on the treadmill was determined. Control animals ran 45 minutes, adrenalectomized - 10 minutes.

Questions:

1. To determine the type of investigated reactivity (species, group, individual).

2. To determine the type of reactivity studied (physiological, pathological).

3. Determine the form of reactivity in the subjects- (adrenalectomized) (normergia, hypergia, hyperergy, anergy, dysergy)

4. How did the reactivity and resistance in the experimental group change compared with the control group?

TASK number 2

In winter, under natural conditions, two groups of animals (gophers and rats) received an ultra-high dose of ionizing radiation. Mortality in rats was 95%, in gophers - 10%.

Questions:

1. To determine the type of investigated reactivity (species, group, individual).

2. To determine the type of reactivity studied (physiological, pathological).

3. Determine the form of reactivity in the subjects (normergia, hypergia, hypergia, anergia, dysergia).

4. How did the reactivity and resistance in the experimental group change compared with the control group?

UNIT 3. **Pathophysiology of local circulation, thrombosis, embolism, inflammation, fever, infection process**

BLOCK A

TO KNOW

1. Typical forms of peripheral circulatory disorders. Stasis, types, main causes and mechanisms of development and consequences.

2. Embolism, types of embolism and their characteristics embolism.

3. Definition and General characteristics of inflammation. The etiology of the inflammation. Inflammatory mediators (cellular and humoral) and their role in the development of the inflammatory process.

4. The reasons for the transition of arterial hyperemia in the venous inflammation. Local and common signs of inflammation and mechanisms of their development.

5. Types of exudates and their characteristics. The difference of exudate from transudate (Rivolt test).

6. Definition of the concept and general characteristics of fever as TPP. Etiology of fever. Characteristics of exo- and endogenous pyrogenic substances. Leukocytic (true) pyrogens.

7. Types of fever, depending on the cause, the degree of rise in body temperature and the type of temperature curves.

8. Infectious process, definition, types of infectious process. Forms of relationships between macro- and microorganisms.

9. Kinds of infectious agents and their properties. Conditions for the emergence of the infectious process: the entrance gate, the pathways of the spread of infectious agents, the mechanisms of anti-infectious protection.

10. Stages of infectious diseases, characteristic. Mechanisms of protection of the organism from pathogens of infections: nonspecific (bactericidal and bacteriostatic, cellular and humoral, reflex) and specific.

Arterial hyperemia, types, causes, mechanisms of development.

11. Venous hyperemia, the main causes and mechanism of development.

12. Ischemia, types, causes, mechanisms of development.

13. Thrombosis. The main causes and conditions of blood clot formation in blood vessels.

14. Pathogenetic features of air and gas embolism.

15. Sludge. Causes, mechanism of development and consequences.

16. Stages of the inflammatory process (pathogenesis of inflammation). Alteration, species and their characteristics. The significance of primary and secondary alteration in inflammation.

17. Features of metabolic disorders and physico-chemical changes in the focus of inflammation.

18. Phases of vascular reaction in inflammation and mechanisms of their development.

19. Exudation. The importance of vascular and tissue factors in the mechanism of development of exudation. Adaptive and pathogenic importance of exudation in the development of inflammation.

20. Factors on which the type, composition and properties of the exudate depends.

21. Pathogenetic features of acute and chronic inflammation.

22. Pathogenesis of fever (change of set point).

23. Stages of fever. The relationship between heat production and heat transfer in various stages of fever. Critical and lytic temperature decrease.

24. Difference of fever from overheating

BLOCK B

SKILLS:

TASK # 1

In the phase of alteration, a marked increase in highly active enzymes occurs in the inflammation focus: elastase, collagenase, hyaluronidase, phospholipase A2, myeloperoxidase, and others.

1. Which of the following enzymes induces increased formation of prostaglandins?

2. Describe the role of prostaglandins in the outbreak of inflammation.

3. What other inflammatory mediators are formed after activation of this enzyme? What are their basic properties?

4. How can I block the increased formation of this enzyme?

TASK # 2

Patient K. Patient turned to the doctor for three days. On the first day of the disease, I felt general malaise, headache, rapid physical and mental fatigue. On the second day, a slight increase in body temperature (37-37.2 °) and sore throat appeared. On the day of going to the doctor, the body temperature rose to 38 ° and pain appeared when swallowing saliva and food. On examination of the oral cavity and pharynx, the doctor found enlarged, loose and reddened tonsils, on the surface of which isolated lacunae filled with pus were found. Bacteriological examination of scrapings from the surface of the tonsils streptococci is inoculated.

Questions:

1. What disease has the patient developed?

2. Decipher the etiological factor that caused the disease.

3. Tell me, what typical pathological processes are we talking about?

4. In your opinion, single lacunae filled with pus are exudates or transudates (justify the answer).

5. What kind of therapy (etiotropic, pathogenetic, symptomatic) is appropriate in this case?

Tests:

1. Tears are considered to be part of the:

1. first line of defense.

2. second line of defense.

3. third line of defense.

4. specific defenses.

5. nonspecific defenses.

a. 1, 4

b. 1, 5

c. 3, 4

d. 2, 5

2. A specific defense for the body is:

a. phagocytosis.

b. sensitized T lymphocytes.

c. the inflammatory response.

d. intact skin and mucous membranes.

3. The inflammatory response is a nonspecific response to:

a. phagocytosis of foreign material.

b. local vasodilation.

c. any tissue injury.

d. formation of purulent exudates.

4. Chemical mediators released during the inflammatory response include:

a. albumin and fibrinogen.

b. growth factors and cell enzymes.

c. macrophages and neutrophils.

d. histamine and prostaglandins.

5. Which of the following result directly from the release of chemical mediators following a moderate burn injury?

1. Pain

2. Local vasoconstriction

3. Increased capillary permeability

4. Pallor

a. 1, 2

b. 1, 3

c. 2, 3

d. 2, 4

6. Granulation tissue is best described as:

a. highly vascular, very fragile, and very susceptible to infection.

b. an erosion through the wall of viscera, leading to complications.

c. a type of adhesion with no vascularization.

d. a form of stenosis, in a duct, that is extremely tough and resists attack by microbes.

7. Edema associated with inflammation results directly from:

a. increased fluid and protein in the interstitial compartment.

b. increased phagocytes in the affected area.

c. decreased capillary permeability.

d. general vasoconstriction.

8. The warmth and redness related to the inflammatory response results from:

a. increased interstitial fluid.

b. production of complement.

c. a large number of white blood cells (WBCs) entering the area.

d. increased blood flow into the area.

9. What is the correct order of the following events in the inflammatory response immediately after tissue injury?

1. Increased permeability of blood vessels

2. Dilation of blood vessels

3. Transient vasoconstriction

4. Migration of leukocytes to the area

5. Hyperemia

a. 5, 3, 2, 1, 4

b. 1, 2, 4, 5, 3

c. 2, 3, 5, 4, 1

d. 3, 2, 5, 1, 4

10. The process of phagocytosis involves the:

a. ingestion of foreign material and cell debris by leukocytes.

b. shift of fluid and protein out of capillaries.

c. formation of a fibrin mesh around the infected area.

d. movement of erythrocytes through the capillary wall.

11. Systemic effects of severe inflammation include:

a. erythema and warmth.

b. loss of movement at the affected joint.

c. fatigue, anorexia, and mild fever.

d. abscess formation.

12. The term leukocytosis means:

a. increased white blood cells (WBCs) in the blood.

b. decreased WBCs in the blood.

c. increased number of immature circulating leukocytes.

d. significant change in the proportions of WBCs.

13. Which of the following statements applies to fever?

a. Viral infection is usually present.

b. Heat-loss mechanisms have been stimulated.

c. It is caused by a signal to the thalamus.

d. It results from release of pyrogens into the circulation.

14. Mechanisms to bring an elevated body temperature down to the normal level include:

a. general cutaneous vasodilation.

b. generalized shivering.

c. increased heart rate.

d. increased metabolic rate.

15. Replacement of damaged tissue by similar functional cells is termed:

a. fibrosis.

b. regeneration.

c. resolution.

d. repair by scar tissue.

16. Scar tissue consists primarily of:

a. granulation tissue.

b. epithelial cells.

c. collagen fibers.

d. new capillaries and smooth muscle fibers.

17. Which of the following promotes rapid healing?

a. Closely approximated edges of a wound

b. Presence of foreign material

c. Exposure to radiation

d. Vasoconstriction in the involved area

18. Glucocorticoids are used to treat inflammation because they directly:

a. promote the release of prostaglandins at the site.

b. decrease capillary permeability.

c. mobilize lymphocytes and neutrophils.

d. prevent infection.

UNIT 4. Pathophysiology of Carbohydrate, Protein, Lipid metabolism

TO KNOW

1. Hyperproteinemia, hypoproteinemia - causes and consequences.

2. Factors affecting the duration of fasting. Fasting, as a method of treatment. Dietotherapy.

3. Hypoglycemia and hypoglycemic syndrome - types, causes, mechanisms of development and basic clinical manifestations.

4. Glucosuria - species, causes and mechanisms of development. Renal diabetes.

5. Etiology, pathogenesis and the main manifestations of diabetes insipidus.

6. Experimental forms of diabetes mellitus (pancreatic, alloxan, and floridazine).

7. Disturbance of interstitial metabolism of fat in diabetes mellitus. Hyperketonemia (ketosis), causes and mechanisms. Ketonuria.

8. The main causes and characteristics of endocrine obesity.

9. Etiology and pathogenesis of alimentary obesity.

10. Consequences of obesity. Violations of the functions of organs and systems for obesity.

11. Atherosclerosis, the definition of a concept, a general characteristic. General etiology and pathogenesis of atherosclerosis.

12. Gas acidosis and gas alkalosis. Causes and mechanisms of development.

13. Negative alkalosis and a negative acidosis. Causes and mechanisms of development.

14. Typical forms of water balance disturbance: hypo- and hyperhydration, species, etiology and pathogenesis.

15. Hypo-and hypernarthriemia. Causes and consequences.

16. Hypo-and hyperkalemia. Causes and consequences.

17. Hypo-and hypercalcemia. Causes and consequences.

18. Tolerance of the body to carbohydrates, types of tolerance disorders (sugar curves).

19. Hyperglycemia - types, causes, developmental mechanisms and main clinical manifestations.

20. Hypoglycemia - types, causes, mechanisms of development and main clinical manifestations.

21. Glycosuria - types, causes and mechanisms of development. Renal diabetes.

22. Etiology and pathogenesis of diabetes mellitus (type 1 DM and type 2 DM).

23. Metabolic disorders in diabetes mellitus.

24. The main clinical and biochemical manifestations of diabetes mellitus and their mechanism of development.

25. Pathogenesis of acute (early) complications of diabetes. Differentiation of lumps in diabetes.

26. Pathogenesis of chronic (late) complications of diabetes.

27. Etiology, pathogenesis and main manifestations of diabetes insipidus.

BLOCK B

SKILLS

TASK №1

1. Diabetes mellitus type I (insulin-dependent). Increased blood glucose (12 mmol / l), children's age (7 years), a viral infection (parotitis).

2. Etiology - a virus of mumps, tropic to β-cells of the pancreas. Pathogenesis consists in the production of antibodies to antigens of this virus, cross-linked with antigens of β-cells of the pancreas, which leads to the development of autoimmune lesions of the islets of Langerhans, accompanied by lysis of β-cells and a decrease in insulin secretion into the blood.

3. All symptoms - evidence of dehydration and energy deficiency (nocturnal enuresis - weakness of the bladder sphincter).

TASK №2

1. Glycosylation of peripheral nerve proteins, the formation of antibodies against these proteins and the development of autoimmune reactions. Hypoxia of the nervous structures with the development of energy deficiency and slowing the speed of impulses, therefore, a violation of the trophism of tissues innervated by these fibers, and the development of pain.

2. Late complications (diabetic foot syndrome).

3. Pathogenetic (lifelong administration of glucose-lowering drugs), symptomatic (antiseptics, analgesics).

UNIT 5. Pathophysiology of Acid-base imbalance Tumor, Hypoxia

TO KNOW

1. Classification of tumors. The difference between benign tumors and malignant tumors.

2. Stages of tumor development (carcinogenesis) and their characteristics. The concept of proto-oncogenes, oncogenes, oncoproteins and their role in the cellular and molecular mechanisms of carcinogenesis.

3. Disturbance of metabolism and physiological functions in acute and chronic hypoxia.

4. Hypoxia - as a universal mechanism of damage and cell death.

5. Urgent and long-term adaptation mechanisms for hypoxia.

6. Hypoxic hypoxia: hypobaric and normobaric, hyperbaric - causes and mechanisms of development.

7. Gas acidosis and gas alkalosis. Causes and mechanisms of development.

8. Negative alkalosis and a negative acidosis. Causes and mechanisms of development.

9. Typical forms of water balance disturbance: hypo- and hyperhydration, species, etiology and pathogenesis.

10. Hypo-and hypernarthriemia. Causes and consequences.

11. Hypo-and hyperkalemia. Causes and consequences.

12. Hypo-and hypercalcemia. Causes and consequences.

Tests:

1. Choose the correct proportion of water to body weight to be expected in a healthy male adult's body:

a. 30%

b. 45%

c. 60%

d. 70%

ANS: C

2. Choose the correct proportion of blood (to body weight) in an adult male's body:

a. 30%

b. 20%

c. 10%

d. 4%

ANS: D

3. Insensible fluid loss refers to water lost through:

a. perspiration only.

b. feces only.

c. perspiration and expiration.

d. urine and feces.

ANS: C

4. When the osmotic pressure of the blood is elevated above normal, water would shift from the:

a. blood into the cells.

b. interstitial compartment into the cells.

c. interstitial compartment into the blood.

d. cells into the interstitial compartment.

ANS: C

5. Which of the following would result from a deficit of plasma proteins?

a. Increased osmotic pressure

b. Decreased osmotic pressure

c. Increased hydrostatic pressure

d. Decreased hydrostatic pressure

ANS: B

6. Which of the following would cause edema?

a. Decreased capillary hydrostatic pressure

b. Increased capillary osmotic pressure

c. Decreased capillary permeability

d. Increased capillary permeability

ANS: D

7. Which of the following would likely be related to an elevated hematocrit reading?

a. Fluid excess

b. Fluid deficit

c. Increased sodium level

d. Decreased erythrocytes

ANS: B

8. Which of the following is a typical sign of dehydration?

a. Rapid, strong pulse

b. Low hematocrit

c. Increased urine output

d. Rough oral mucosa

ANS: D

9. Which of the following terms refers to a combination of decreased circulating blood volume combined with excess fluid in a body

cavity?

a. Dehydration

b. Third-spacing

c. Hypovolemia

d. Water retention

ANS: B

Which of the following is the primary cation in the extracellular fluid?

a. Sodium

b. Potassium

c. Calcium

d. Iron

ANS: A

11. Which of the following is a common cause of hyponatremia?

a. Loss of the thirst mechanism

b. Excessive sweating

c. Excessive aldosterone secretion

d. Prolonged period of rapid, deep respirations

ANS: B

12. Which of the following is a common effect of both hypokalemia and hyperkalemia?

a. Skeletal muscle twitch and cramps

b. Oliguria

c. Elevated serum pH

d. Cardiac arrhythmias

ANS: D

13. Choose the correct effect of increased parathyroid hormone.

a. Increased movement of calcium ions into the bones

b. Increased activation of vitamin D

c. Increased absorption of calcium from the digestive tract

d. Decreased reabsorption of calcium in the kidneys

ANS: C

14. Which of the following results from hypocalcemia?

1. Low serum phosphate levels

2. Nausea and constipation

3. Skeletal muscle twitch and spasms

4. Weak cardiac contractions

a. 1, 2

b. 1, 4

c. 2, 3

d. 3, 4

ANS: D

15. Which of the following causes tetany?

a. Increased permeability of nerve membranes due to low serum calcium

b. Excess calcium ions in skeletal muscle due to excess parathyroid hormone (PTH)

c. Excess calcium ions inside somatic nerves as a result of neoplasms

d. Increased stimulation of the nerves in the cerebral cortex

ANS: A

16. In which of the following processes is phosphate ion NOT a major component?

a. Bone metabolism

b. Metabolic processes involving adenosine triphosphate (ATP)

c. Blood clotting

d. Acid-base balance

ANS: C

17. Which of the following would be considered normal serum pH?

a. 4.5-8

b. 7.0

c. 7.4

d. 8

ANS: C

18. When many excess hydrogen ions accumulate in the blood, what happens to serum pH? The pH:

a. decreases.

b. increases.

c. remains constant.

d. varies based on metabolism.

ANS: A REF: 28

19. What is the slowest but most effective control for acid-base balance?

a. Respiratory system

b. Buffer systems in the blood

c. Kidneys

d. Brain

ANS: C

Which of the following is essential in order to maintain serum pH within normal range?

a. Carbonic acid and bicarbonate ion must be present in equal quantities.

b. All excess carbonic acid must be excreted by the kidneys.

c. The concentration of bicarbonate ion must remain constant.

d. The ratio of carbonic acid to bicarbonate ion must be 1:20.

ANS: D

21. Which is the correct effect on the body of abnormally slow respirations?

a. Increased carbonic acid

b. Decreased carbonic acid

c. Increased bicarbonate ion

d. Decreased bicarbonate ion

ANS: A

22. Which condition is likely to cause metabolic acidosis?

a. Slow, shallow respirations

b. Prolonged diarrhea

c. Mild vomiting

d. Excessive fluid in the body

ANS: B

23. What would a serum pH of 7.33 in a patient with kidney disease indicate?

a. Metabolic alkalosis

b. Metabolic acidosis

c. Respiratory alkalosis

d. Respiratory acidosis

ANS: B

24. Which serum value indicates decompensated metabolic acidosis?

a. pH is below normal range

b. pH is above normal range

c. Bicarbonate level decreases

d. Bicarbonate level increases

ANS: A

25. What is the effect on blood serum when excessive lactic acid accumulates in the body?

a. Bicarbonate ion levels decrease

b. Bicarbonate ion levels increase

c. Carbonic acid levels increase

d. pH increases

ANS: A

26. The direct effects of acidosis are manifested primarily in the functioning of the:

a. Digestive system

b. Urinary system

c. Nervous system

d. Respiratory system

ANS: C

27. Compensation mechanisms in the body for dehydration would include:

a. increased antidiuretic hormone (ADH).

b. decreased aldosterone.

c. slow, strong heart contraction.

d. peripheral vasodilation.

ANS: A

28. Which acid-base imbalance results from impaired expiration due to emphysema?

a. Metabolic acidosis

b. Metabolic alkalosis

c. Respiratory acidosis

d. Respiratory alkalosis

ANS: C

29. In patients with impaired expiration associated with emphysema, effective compensation for the acid-base imbalance would be:

a. increased rate and depth of respiration.

b. decreased rate and depth of respiration.

c. increased urine pH and decreased serum bicarbonate.

d. decreased urine pH and increased serum bicarbonate.

ANS: D

Case Study

A routine mammogram showed a large mass in the right breast of Mrs. H, age 42 years. A biopsy confirmed the presence of a malignant tumor. Mrs. H was concerned because her mother and an aunt had breast cancer. No metastases were detected at this time. A mastectomy was performed. Several number of axillary and mediastinal lymph nodes were removed. Pathologic examination showed that each of the nodes from each area contained malignant cells. Given that this case was considered to be stage II, it was recommended that Mrs. H have chemotherapy and radiation treatment following surgery and later have her ovaries removed to reduce her estrogen levels

Case Study

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ASSESSMENT FUND

ADVANCEMENT QUESTIONS FOR V SEMESTER:

UNIT 1. Typical forms of pathology of system of external respiration and cardio-vascular system

TO KNOW:

1. General etiology and pathogenesis of respiratory failure.
2. Main causes and mechanisms of impaired alveolar ventilation of the lungs (alveolar hypoventilation; alveolar hyperventilation).
3. Main causes and mechanisms of impaired diffusion and perfusion capacity of the lungs.
4. Etiology and pathogenesis of pulmonary hypertension (precapillary, postcapillary, mixed).
5. Primary pulmonary and primary extrapulmonary forms of respiratory failure.
6. Etiology and pathogenesis of obstructive and restrictive types of respiratory dysfunction.
7. Main indicators of respiratory failure and their characteristics.
8. Dyspnea and its types. Causes and mechanisms of development (Hering–Breuer reflex).
9. Periodic and terminal types of breathing (Biot's, Cheyne–Stokes, Kussmaul, etc.). Causes and mechanisms of development.
10. Etiology and pathogenesis of respiratory and circulatory disorders in bronchial asthma and pulmonary emphysema.
11. Etiology and pathogenesis of respiratory and circulatory disorders in pneumonia and pulmonary edema, including high-altitude pulmonary edema.
12. Etiology and pathogenesis of respiratory and circulatory disorders in various types of pneumothorax.
13. Sinus tachycardia, sinus bradycardia, sinus arrhythmia: types and causes.
14. Extrasystole (sinus, atrial, atrioventricular, ventricular). Its causes and ECG changes depending on the site of origin.
15. Pathogenesis and consequences of paroxysmal tachycardia.
16. Heart blocks: types and mechanisms of development. Wenckebach–Samoilov periods and features of grade IV atrioventricular block.
17. Pathogenesis of atrial fibrillation (ventricular fibrillation).
18. Main causes and types of coronary insufficiency (relative and absolute).
19. Stages of coronary insufficiency and their characteristics.
20. Myocardial infarction. Hemodynamic disorders and ECG changes depending on the area of myocardial damage.
21. Pathogenesis of major clinical manifestations of myocardial infarction: pain syndrome and resorptive–necrotic syndrome.
22. Complications and outcomes of angina pectoris and myocardial infarction. Pathogenesis of cardiogenic shock and its clinical manifestations.
23. The role of atherosclerosis and other risk factors in the development of coronary insufficiency (atherogenic and lipotropic factors).
24. Modern understanding of the pathogenesis of atherosclerosis.
25. Classification and general characteristics of hypertensive conditions.
26. Hypertensive disease. Etiology and pathogenesis, risk factors.
27. Stages and main clinical manifestations of hypertensive disease and mechanisms of their development.
28. Complications and consequences of arterial hypertension.

29. Secondary (symptomatic) arterial hypertension: types, causes, and mechanisms of development.
30. Pathogenesis of renal arterial hypertension (Goldblatt and Grollman theories).
31. Experimental models of arterial hypertension (renoprival, central, salt-induced, adrenaline-induced, disinhibition hypertension).
32. Pathophysiological characteristics of chronic arterial hypotension: primary and secondary (symptomatic). Etiology and pathogenesis.
33. Syncope, collapse, shock: types and mechanisms of development, manifestations, and consequences.
34. Definition and classification of forms of circulatory failure.
35. Main (cardiac and extracardiac) causes of chronic heart failure (CHF).
36. Pathogenetic classification of CHF (volume overload, resistance overload).
37. Heart defects: types and characteristics.
38. Compensation mechanisms in heart defects (immediate and delayed).
39. Compensatory cardiac hyperfunction: isotonic and isometric, mechanisms of development.
40. Tonogenic and myogenic dilatation and their characteristics.
41. Mechanism and adverse effects of prolonged tachycardia in heart defects.
42. Myocardial hypertrophy: stages and types — eccentric and concentric. Mechanisms of development.
43. Mechanism of transition from compensated to decompensated heart defects.
44. Main hemodynamic indicators and their changes in CHF.
45. Metabolic disturbances in CHF.
46. Clinical (subjective and objective) manifestations of CHF and their mechanisms.
47. Mechanisms of cardiac edema and their differences from renal edema.
48. Pathogenetic principles of prevention and therapy of respiratory and circulatory failure.
49. Neonatal respiratory distress syndrome and its differences from adults.
50. Neonatal asphyxia: causes and mechanisms of development.
51. Features of rhythm disturbances in children.
52. General etiology and pathogenesis of congenital heart defects and major vessel anomalies.
53. Types of congenital heart defects (CHD) and their characteristics.

BLOCK B

SKILLS

Task 1.

A 63-year-old woman has the sudden onset of 'knife-like' pain in the chest radiating to the back. She has been previously healthy except for a history of poorly controlled hypertension. She is transported to the hospital and on arrival she has a heart rate of 90/minute, respirations 20/minute, temperature 36.8°C, and blood pressure 150/100 mm Hg. No murmurs, rubs, or gallops are audible. A chest radiograph reveals a widened mediastinum. Laboratory findings include a total serum creatine kinase of 55 U/L, creatinine 0.9 mg/dL, and glucose 123 mg/dL.

1. Which of the following is the most likely diagnosis?
2. Explain pathogenesis of developed condition.

Task 2.

A 53-year-old man comes to the emergency department due to a few weeks of severe heartburn and difficulty swallowing He has had mild to moderate heartburn for several years and

has tried weight loss elevating the head of the bed while sleeping, and several months of proton pump inhibitor therapy. Other medical problems include hypertension and hypothyroidism Temperature is 36 7 C (98 1 F) blood pressure is 130:80 mm Hg pulse is 78/min and respirations are 16/min BMI is 25 kg/m2: Physical examination is unremarkable An upper gastrointestinal endoscopy is performed and esophageal biopsy shows columnar epithelium with interspersed goblet cells. A similar adaptive response can be seen in which of the following scenarios?

UNIT 2: Typical forms of blood pathology.

TO KNOW:

1. Changes in total blood volume: normo-, hypo-, and hypervolemia, their types, causes, and mechanisms of development.
2. Erythrocytosis: absolute and relative; causes; features of the etiology and pathogenesis of true (absolute) polycythemic hypervolemia; hemodynamic disturbances in hypervolemia.
3. Definition and classification principles of anemic conditions.
4. Causes of death and factors determining the consequences of acute blood loss.
5. Compensation mechanisms in acute blood loss (phases of blood loss).
6. Mechanism of the bone marrow phase of compensation in acute blood loss. Features of blood regeneration after hemorrhage under high-altitude conditions.
7. Pathological forms (regenerative and degenerative) of red blood cells.
8. Reticulocytes and their significance for understanding the pathogenesis of various types of anemia.
9. Blood picture in acute post-hemorrhagic anemia depending on the time elapsed after bleeding.
10. Hypercoagulation. Thrombotic syndrome: main causes, mechanisms of development, manifestations, and consequences.
11. Hypocoagulation. Hemorrhagic syndrome: main types, causes, mechanisms of development, manifestations, and consequences.
12. DIC syndrome (disseminated intravascular coagulation): etiology and pathogenesis, stages, manifestations, and consequences. Pathogenetic principles of therapy and prevention of DIC syndrome.
13. Types of hemorrhagic syndromes and diatheses.
14. Etiology, pathogenesis, general characteristics, and blood picture in Vaquez's disease (polycythemia vera).
15. Vasopathies caused by microvascular lesions (hemorrhagic vasculitis or Henoch–Schönlein purpura).
16. Hemorrhagic diatheses and syndromes caused by platelet hemostasis disorders (thrombocytopenic purpura or Werlhof's disease).
17. Coagulopathies caused by impaired blood clotting (hemophilia A and B).
18. Etiology and pathogenesis of iron deficiency anemias.
19. General characteristics of iron deficiency anemias: chlorosis (early and late), alimentary iron deficiency anemia.
20. Blood picture in iron deficiency anemias.
21. Etiology and pathogenesis of vitamin B12 and folate deficiency anemias.
22. The concept of the external and internal Castle factor (gastromucoprotein) and their role in the pathogenesis of vitamin B12 and folate deficiency anemias.
23. Pathogenesis of the triad of symptoms in vitamin B12 and folate deficiency anemias.
24. Blood picture and features of hematopoiesis in vitamin B12 and folate deficiency anemias.
25. Etiology and pathogenesis of acquired hemolytic anemias (HA).
26. Thalassemia: etiology, pathogenesis, blood picture, and main clinical manifestations.

27. Sickle cell anemia: etiology, pathogenesis, and main clinical manifestations.
28. Comparative characteristics of the blood picture in various forms of anemia (post-hemorrhagic, dyserythropoietic, and hemolytic).
29. Typical changes in the leukocyte system.
30. Differences between physiological and pathological leukocytosis.
31. Etiology and pathogenesis of pathological leukocytoses: by mechanism of development and by changes in the leukocyte formula.
32. Neutrophilic leukocytosis: causes and mechanisms of development.
33. Left and right shifts of the leukocyte formula in neutrophilic leukocytosis: pathophysiological characteristics and significance for understanding the body's reactivity in pathology.
34. Leukemoid reactions: types, causes, mechanisms of development, and their distinction from leukemias.
35. Eosinophilic and basophilic leukocytosis: causes and mechanisms of development.
36. Monocytosis and lymphocytosis (absolute and relative): causes and mechanisms of development.
37. Leukopenia: types, causes, and mechanisms of development.
38. Agranulocytosis: types, etiology, pathogenesis, blood picture, and consequences.
39. Definition and general characteristics of leukemias. Etiology of leukemias.
40. Pathogenesis of leukemias (clonal theory) according to modern concepts. Features of tumor cell metastasis in leukemias.
41. Principles of classification of leukemias and their characteristics. The concept of monoclonal and polyclonal leukemias.
42. Signs of tumor progression in leukemias. Concepts of “leukemic hiatus,” “blast crisis,” aleukemic and leukemic phases of leukemia.
43. Mechanisms of development of secondary clinical manifestations in leukemias: anemic, hemorrhagic, intoxication, infectious, and metastatic syndromes.
44. General characteristics and blood picture in acute myeloblastic leukemia (AML) and acute lymphoblastic leukemia (ALL).
45. General characteristics and blood picture in chronic myelogenous leukemia (CML).
46. General characteristics and blood picture in chronic lymphocytic leukemia (CLL).

BLOCK B

SKILLS

Task 1.

A 72-year-old man comes to the physician for a health maintenance examination. He has no personal or family history of major medical illnesses. He has never smoked cigarettes. Pulse oximetry on room air shows an oxygen saturation of 98%. Physical examination shows plethoric skin and splenomegaly. Laboratory studies show: Hemoglobin 21.1 g/dL Hematocrit 61% Leukocyte count 15,000/mm3 Segmented neutrophils 68% Basophils 4% Lymphocytes 28% Platelet count 501,000/mm3 Leukocyte alkaline phosphatase increased A peripheral blood smear shows occasional giant platelets. The primary hematologic defect in this patient most likely occurred in which of the following cells?

Task 2.

A 32-year-old woman with type 1 diabetes mellitus has had progressive renal failure during the past 2 years. She has not yet started dialysis. Examination shows no abnormalities. Her hemoglobin concentration is 9 g/dL, hematocrit is 28%, and mean corpuscular volume is 94 μm3

. A blood smear shows normochromic, normocytic cells. Which of the following is the most likely cause?

Task 3.

A 35-year-old man is brought to the emergency department 30 minutes after he sustained a cut on his hand while loading cargo at his job. He lives alone and takes most of his meals at a local restaurant. He eats mostly snack foods at the bar and fast food. He drinks four to six 12-oz beers daily and double that amount on weekends. He takes no medications. Physical examination shows a 3-cm laceration on the right hand that is bleeding steadily. Laboratory studies show a hemoglobin concentration of 11 g/dL, leukocyte count of 4000/mm3 , and platelet count of 150,000/mm3 . A photomicrograph of a peripheral blood smear is shown. A deficiency of which of the following is the most likely cause of this patient's anemia?

UNIT 3: Typical forms of liver pathology and gastro-intestinal tract pathology. Typical forms of kidneys pathology.

TO KNOW:

1. Main causes of liver failure and its clinical manifestations.
2. Impairment of the barrier and detoxification functions of the liver.
3. Pathogenesis of hepatic coma and its main manifestations.
4. Experimental models for reproducing and studying liver failure (total and partial hepatectomy, London's angiostomy, N. Eck – I.P. Pavlov fistula—direct and indirect).
5. Hepatitis: types, etiology, major manifestations, and consequences.
6. Mechanisms of viral liver injury (direct cytopathic, immune-mediated, apoptosis induction, autoimmune mechanisms).
7. Mechanisms of ethanol-induced liver damage.
8. Metabolic disturbances in liver failure.
9. Excretory (cholestatic) liver failure: causes and experimental reproduction.
10. Liver cirrhosis: types, etiology, pathogenesis, and consequences of cirrhosis development.
11. Etiology and pathogenesis of hepatic coma.
12. Jaundice: types and characteristics:
    • Prehepatic (hemolytic) jaundice – etiology, pathogenesis, and major manifestations;
    • Hepatic (hepatocellular) jaundice – etiology, pathogenesis, and major manifestations;
    • Posthepatic (obstructive/mechanical) jaundice – etiology, pathogenesis, and major manifestations.
13. Methods for differentiating types of jaundice (direct and indirect bilirubin, stercobilin, urobilin).
14. Enzymopathic jaundice: etiology and differential diagnosis.
15. Cholemia (bile poisoning): changes in the blood system, nervous system, and cardiovascular system.
16. Causes, manifestations, mechanisms of acholia and its consequences.
17. Portal hypertension: types, causes, major manifestations, and consequences.
18. General etiology and pathogenesis of common pathological forms of the digestive system.
19. Typical disorders of taste, appetite, salivation, and swallowing.
20. Types of gastric secretion disorders: hypersecretion, hyposecretion, and achylia. Main causes and consequences.
21. Pathological types of gastric secretion and their characteristics (inhibitory, excitable, inert, asthenic).

22. Common forms of impaired gastric motility: main causes and consequences.
23. Etiology and pathogenesis of peptic ulcer disease of the stomach: its major manifestations and complications.
24. Etiology and pathogenesis of dumping syndrome and its main manifestations.
25. Typical disorders of intestinal motility: diarrhea and constipation—main types, mechanisms, and consequences.
26. General etiology and pathogenesis of intestinal diseases: malabsorption syndrome, chronic enteritis, chronic colitis, and irritable bowel syndrome—main clinical manifestations.
27. Etiology and pathogenesis of peptic ulcer disease of the stomach and duodenum: major manifestations and complications.
28. Experimental methods for reproducing gastric ulcers.
29. Etiology and pathogenesis of pancreatitis.
30. Pathogenetic mechanisms of acute pancreatitis.
31. General etiology and pathogenesis of renal functional disorders.
32. Causes and mechanisms of impaired glomerular filtration and tubular reabsorption.
33. Renal and extrarenal symptoms in kidney pathology.
34. Urinary syndrome: causes and mechanisms of development.
35. Proteinuria: types and mechanisms of development.
36. Mechanism of hypoproteinemia in nephrotic syndrome.
37. Hematuria: types and mechanisms of development.
38. Pathogenesis of disturbances in major renal processes: filtration, excretion, reabsorption, and secretion.
39. Changes in daily urine output: polyuria, oliguria, and anuria—their causes and diagnostic significance.
40. Changes in urine specific gravity: hypo- and isosthenuria—their causes and diagnostic significance.
41. The endocrine (incretory) function of the kidneys and its role in maintaining blood pressure, erythropoiesis, and coagulation mechanisms.
42. Mechanisms of reduced glomerular filtration.
43. Main causes of impaired renal concentrating ability.
44. Mechanisms of impaired tubular reabsorption.
45. Polyuria: its types according to mechanisms of development (water diuresis, osmotic diuresis, hypertensive diuresis).
46. Nephritic syndrome: pathogenesis of its main symptoms.
47. Nephrosis and nephrotic syndrome (primary and secondary): main causes and manifestations.
48. Pathophysiological characteristics of chronic glomerulonephritis (CGN).
49. Immune and non-immune mechanisms in the pathogenesis of glomerulonephritis.
50. Pathophysiological characteristics of diffuse pyelonephritis.
51. Pathogenesis of renal arterial hypertension (pressor and depressor renal functions).
52. Mechanism of development of anemic syndrome in kidney diseases.
53. Mechanism of renal edema (nephrotic and nephritic) and their differences from cardiac edema
54. Experimental methods for reproducing renal pathology (kidney diseases).
55. Pathogenetic principles of prevention and treatment of kidney diseases.
56. Definition and key indicators of renal failure.
57. Etiology and pathogenesis of acute renal failure (ARF).
58. Acute renal failure (prerenal, renal, postrenal): causes.
59. Etiology and pathogenesis of chronic renal failure (CRF).
60. Stages of CRF and their pathophysiological characteristics.
61. Main links in the pathogenesis of the oligoanuric stage of CRF.

62. Uremia: causes, mechanisms of development, and main manifestations.
63. Pathogenesis of uremic (renal) coma and its main manifestations.
64. Pathogenetic principles of renal failure treatment: hemodialysis and kidney transplantation.
65. Nephrolithiasis (urolithiasis): etiology and pathogenesis.
66. Features of kidney pathology in children.

BLOCK B

SKILLS

Task 1.

A 3-week-old girl delivered at term with no complications is brought to the physician by her mother because of a 1-week history of yellow eyes and skin, tan-colored stools, and dark brown urine. The newborn has been breast-feeding without difficulty. She is alert and appears to be in no distress. She is at the 50th percentile for length and weight. Physical examination shows scleral icterus and jaundice. There is mild hepatomegaly; the spleen is not palpable. Laboratory studies show: Hemoglobin 14.4 g/dL Hematocrit 43% Leukocyte count 8000/mm3 Serum Albumin 3.5 g/dL Bilirubin, total 14 mg/dL Direct 12.5 mg/dL AST 50 U/L ALT 45 U/L. What is the diagnosis?

Task 2.

A 51-year-old man with a 10-year history of gastroesophageal reflux and suspected Barrett esophagus comes to the office because his omeprazole dose "doesn't work around the Christmas holidays." He states that he prides himself on having a large appetite and "holding his liquor" during the holidays. He currently takes the maximum dose of omeprazole. Which of the following is the most appropriate initial action by the physician?

Task 3.

A 5-year-old boy is brought to the emergency department by his mother because of an episode of bloody stool 3 hours ago. The mother says the stool was hard "like pebbles" and she noted bright red blood on the tissue when the patient cleaned himself. His previous bowel movement was 5 days ago. The patient has no abdominal or rectal pain now, but he did have abdominal pain during his bowel movement 5 days ago. He has no history of major medical illness and receives no medications. Vaccinations are up-to-date. The patient has no recent history of travel. He is at the 5th percentile for height and the 10th percentile for weight; BMI is at the 50th percentile. Vital signs are within normal limits. Abdominal examination shows hypoactive bowel sounds and a soft, slightly distended abdomen that is not tender to palpation. Rectal examination shows 1 cm of bright red rectal mucosa protruding from the right side of the anus; there is no rectal bleeding. The remainder of the examination shows no abnormalities. Which of the following is the most likely cause of this patient's physical findings?

Task 4.

A 60-year-old woman is brought to the emergency department because of a 4-day history of fever, joint aches, and rash. Three weeks ago, she was admitted to the hospital for treatment of Staphylococcal aureus endocarditis. She has received 21 days out of a prescribed 42-day course of intravenous oxacillin. Currently, she appears to be in mild distress. Temperature is 38.0°C

(100.4°F), pulse is 115/min, respirations are 24/min, and blood pressure is 120/70 mm Hg. Pulse oximetry on room air shows an oxygen saturation of 97%. Physical examination shows a diffuse maculopapular rash over the trunk and upper and lower extremities. There is no pus or erythema at the skin insertion site of the peripherally inserted central catheter line initially placed on the day of hospital discharge. Results of laboratory studies are shown: Hemoglobin 11.1 g/dL Hematocrit 33% Leukocyte count 12,100/mm3 Segmented neutrophils 78% Eosinophils 9% Lymphocytes 7% Monocytes 6% Platelet count 341,000/mm3 Serum Na+ 133 mEq/L K+ 6.5 mEq/L Cl− 100 mEq/L HCO3 − 15 mEq/L Urea nitrogen 65 mg/dL Glucose 96 mg/dL Creatinine 5.7 mg/dL Urine microscopy shows eosinophils and WBC casts. Which of the following is the most likely underlying cause of this patient's condition?

UNIT 4: Typical forms of endocrine and nervous system pathology.

TO KNOW:

1. General etiology and pathogenesis of endocrinopathies.
2. Main causes of disorders of the central mechanisms regulating peripheral endocrine glands (disturbance of trans-hypophyseal regulation; disturbance of para-hypophyseal regulation; the role of impaired feedback mechanisms).
3. Etiology of primary dysfunctions of peripheral endocrine glands (disturbances in hormone biosynthesis and secretion).
4. Etiology and pathogenesis of peripheral (extraglandular) forms of endocrine disorders.
5. Pathological characteristics of hypo- and hyperfunction of the anterior pituitary lobe.
6. Hypopituitarism: types, pathogenesis, and manifestations.
7. Hyperpituitarism: types, pathogenesis, and manifestations.
8. Mechanism of impaired protein, carbohydrate, and lipid metabolism in hypersecretion of growth hormone (GH).
9. Pathophysiology of the neurohypophysis: diabetes insipidus and syndrome of inappropriate ADH secretion (SIADH).
10. General etiology, pathogenesis, and major manifestations of hypothyroidism: myxedema and cretinism.
11. Endemic goiter: etiology and pathogenesis, pathophysiological characteristics of the main manifestations. The role of autoimmune and environmental factors in the development of endemic goiter.
12. General etiology and manifestations of hyperthyroidism.
13. Pathogenesis of metabolic disturbances and functional changes of organs and systems in diffuse toxic goiter (Graves' disease).
14. Etiology and pathogenesis of hyperaldosteronism: primary (Conn's disease) and secondary, major manifestations and mechanisms of their development.
15. Role of the adrenal glands in regulating blood pressure and in the development of hypertension.
16. Etiology and pathogenesis of hypercorticism. Major manifestations of Cushing's syndrome and Cushing's disease and mechanisms of their development.
17. Etiology and pathogenesis of adrenogenital syndrome (AGS), its main types and manifestations (virilization—masculinization—and feminization).
18. Etiology and pathogenesis of chronic adrenal cortex insufficiency (Addison's disease), its major manifestations and mechanisms of development.

19. General etiology and pathogenesis of disorders of the endocrine function of the gonads and their major manifestations: hypogonadism, eunuchoidism, hypergonadism.
20. Pathogenetic principles of prevention and therapy of endocrine gland dysfunctions.
21. General etiology and pathogenesis of disorders of the nervous system (neuronal damage; disturbances of interneuronal interactions; disruption of integrative activity).
22. Etiopathogenesis of locomotor dysfunctions of the nervous system: hypo- and hyperkinesias, hypodynamia, ataxias—coordination disorders.
23. Etiopathogenesis of neurogenic sensory disturbances: anesthesia and hypesthesia, hyperesthesia and dysesthesia.
24. Pathophysiology of pain: concept and general characteristics of pain—physiological and pathological.
25. Pathological pain of peripheral and central origin: thalamic, phantom, and causalgic pain.
26. Biological and pathogenic significance of pain. Pathogenetic principles of analgesia and acupuncture therapy.
27. Disturbances of the trophic function of the nervous system. Neurodystrophy and denervation syndrome: major manifestations and mechanism of development.
28. Consequences of complete and partial removal of the cerebral cortex.
29. Significance of protective inhibition and its clinical application.
30. Mechanisms of higher nervous activity (HNA) dysfunction. Neuroses: concept, types, characteristics.
31. Etiology of neuroses, their manifestations in humans, and their pathophysiological characteristics. The role of HNA features in the onset and development of neuroses.
32. Principles and methods of inducing experimental neuroses, their manifestations, and pathophysiological characteristics.
33. Disorders of the autonomic nervous system: damage to the hypothalamus, sympathetic, and parasympathetic innervation. Autonomic neuroses and their characteristics.
34. Pathogenetic principles of prevention and treatment of diseases of the nervous system and higher nervous activity.

BLOCK B

SKILLS

Task 1.

A 34-year-old man comes to the office because of a 1-month history of diarrhea. He has a history of pheochromocytoma treated 2 years ago. His mother is being treated for a tumor of her parathyroid gland. He has no other history of major medical illness and takes no medications. His temperature is 37.0°C (98.6°F), pulse is 84/min, respirations are 10/min, and blood pressure is 120/75 mm Hg. Pulse oximetry on room air shows an oxygen saturation of 97%. Vital signs are within normal limits. Physical examination shows a 3-cm, palpable mass on the right side of the neck. A biopsy specimen of the mass shows a neuroendocrine neoplasm of parafollicular cell origin. The most likely cause of the findings in this patient is a mutation in which of the following types of genes?

Task 2.

A previously healthy 52-year-old woman comes to the physician because of a 2-month history of fatigue, constipation, and frequent urination. Her temperature is 37.1°C (98.8°F), pulse is 80/min, respirations are 14/min, and blood pressure is 140/90 mm Hg. Diffuse crackles are heard bilaterally. Her serum calcium concentration is 11.1 mg/dL, and serum parathyroid hormone

concentration is decreased. A chest x-ray shows bilateral hilar lymphadenopathy and interstitial infiltrates. Which of the following is the most likely cause of this patient's hypercalcemia?

Task 3.

Newborn delivered at 36 weeks' gestation to a 22-year-old woman, gravida 1, para 1, has difficulty feeding and listlessness. The mother received no prenatal care. Spontaneous vaginal delivery was uncomplicated. The mother's only medication was a prenatal vitamin. The newborn's length is 49 cm (19 in; 39th percentile), and weight is 3100 g (6 lb 13 oz; 30th percentile); head circumference is 33 cm (13 in; 12th percentile). Temperature is 37.0°C (98.6°F), pulse is 134/min, respirations are 38/min, and blood pressure is 73/50 mm Hg. Physical examination shows ambiguous genitalia. 38 Results of serum studies are shown: Na+ 133 mEq/L K+ 5.0 mEq/L (N=3.2–5.5) Cl– 103 mEq/L HCO3 – 17 mEq/L Glucose 42 mg/dL (N=30–60) The most appropriate pharmacotherapy for this patient targets which of the following receptors?

Tests

1. What process can lead to respiratory failure?

1) decrease in blood oxygen capacity

2) violation of the diffusion of gases through the alveolar-capillary membrane

3) disruption of oxygen transport by blood from the lungs to the tissue

4) violation of tissue respiration

2. What system is not involved in external breathing?

1) respiratory

2) blood circulation

3) central nervous system

4) excretory

3. What caused the restrictive alveolar ventilation disorders?

1) a decrease in the respiratory surface of the lungs

2) narrowing of the airways

3) violation of the passage of gases through the alveolar-capillary membrane

4) impaired pulmonary blood flow

4. What causes obstructive alveolar ventilation?

1) reduction of the respiratory surface of the lungs

2) violation of the airway

3) violation of the passage of gases through the alveolar-capillary membrane

4) impaired pulmonary blood flow

5. Restrictive pulmonary ventilation disorders are observed with:

1) pulmonary hypertension

2) pleurisy

3) pharyngeal abscess

4) bronchial asthma

6. Restrictive pulmonary ventilation disorders are observed with:

1) pneumonia

2) aspiration vomit

3) emphysema

4) bronchial asthma

7. Restrictive pulmonary ventilation disorders are observed with:

1) retraction of the language

2) angioedema

3) tonsillitis

4) peritonitis

8. Obstructive disorders of pulmonary ventilation are noted when:

1) emphysema

2) pneumonia

3) tuberculosis

4) pleurisy

9. What is called perfusion pulmonary insufficiency?

1) insufficiency caused by a decrease in blood flow through the lungs

2) insufficiency due to decreased ventilation

3) insufficiency due to increased blood flow through the lungs

4) failure due to a decrease in gas exchange between the alveolar-capillary

10. A decrease in the Tiffno index, a decrease in lung capacity, an increase in residual lung volume and total lung capacity are characteristic of:

1) lower airway obstruction

2) pneumonia

3) pulmonary edema

4) pulmonary fibrosis

11. How does the residual volume change in obstructive pulmonary insufficiency?

1) not changed

2) increased

3) reduced

4) progressively decreases

12. How does the residual volume (RV) change with a restrictive type of pulmonary insufficiency?

1) not changed

2) increased

3) reduced

4) progressively increases

13. How does the lung capacity change (LC) in obstructive pulmonary insufficiency?

1) does not change

2) decreases

3) increases

4) changes arbitrarily

14. How does the vital capacity of the lungs (LC) change with a restrictive type of pulmonary insufficiency?

1) decreases

2) does not change

3) increases

4) first increases, then decreases

15. Under what pathologies does perfusion pulmonary insufficiency occur?

1) chronic bronchitis

2) bronchial asthma

3) pulmonary embolism (PE)

4) hemothorax

16. What is the characteristic of Kussmaul's breath?

1) the apnea alternates with respiratory movements of increasing and then decreasing depth

2) the apnea alternates with respiratory movements of the same depth

3) rare breathing with increased inhalation and exhalation

4) deep noisy breathing

17. What indicator changes in obstructive type of respiratory failure?

1) tidal volume (TV)

2) minute volume of respiration (MVR)

3) Tiffno index

4) lung capacity (LC)

18. What form of pathology develops with a decrease in the synthesis of lung surfactant?

1) pneumonia

2) pulmonary edema

3) pneumofibrosis

4) pulmonary atelectasis

19. Obstructive disorders of pulmonary ventilation are noted when:

1) peritonitis

2) tuberculosis

3) pneumothorax

4) drowning

20. The restrictive type of respiratory failure develops when:

1) emphysema

2) chronic bronchitis

3) pulmonary fibrosis

4) tracheal stenosis

21. What form of pathology leads to the appearance of a restrictive type of respiratory failure disorder?

1) bronchial asthma

2) bronchitis

3) pneumofibrosis

4) tonsillitis

22. What type of ventilation disorder develops with unilateral pneumothorax?

1) obstructive

2) restrictive

3) constructive

4) constrictive

23. For the restrictive type of respiratory failure is characterized by a decrease:

1) straightening the lungs

2) upper airway patency

3) low airway

4) tissue respiration

24. What dyspnea occurs with a restrictive type of violation?

1) inspiratory

2) expiratory

3) mixed

4) central genesis

25. What dyspnea occurs with bronchiolospasm?

1) inspiratory

2) mixed

3) expiratory

4) central genesis

26. What kind of shortness of breath occurs when stenosis of the trachea, larynx?

1) inspiratory

2) expiratory

3) mixed

4) central genesis

27. What type of dyspnea occurs with emphysema?

1) inspiratory

2) expiratory

3) mixed

4) central genesis

28. What is the characteristic of Cheyne-Stokes breathing?

1) the apnea alternates with the respiratory movements of increasing and then decreasing depth

2) the apnea alternates with respiratory movements of the same depth

3) rare breathing with increased inhalation and exhalation

4) deep noisy breathing

29. What is the characteristic of breathing biota?

1) the apnea alternates with respiratory movements of increasing and then decreasing depth

2) the apnea alternates with respiratory movements of the same depth

3) rare breathing with increased inhalation and exhalation

4) deep noisy breathing

30. What is the characteristic of Kussmaul's breath?

1) the apnea alternates with the respiratory movements of increasing and then decreasing depth

2) the apnea alternates with respiratory movements of the same depth

3) rare breathing with increased inhalation and exhalation

4) deep noisy breathing

31. Impaired lung ventilation is:

1) violation of gas exchange between atmospheric and alveolar air

2) violation of gas exchange between alveolar air and blood

3) violation of gas transportation with blood

4) violation of gas exchange between blood and tissues

32. When alveolar hypoventilation develops:

1) gas acidosis

2) metabolic acidosis

3) metabolic alkalosis

4) gas alkalosis

33. Violation of gas diffusion is:

1) violation of gas exchange between atmospheric and alveolar air

2) violation of gas exchange between alveolar air and blood

3) violation of gas transport

4) violation of gas exchange between blood and tissues

34. When alveolar hyperventilation develops:

1) gas acidosis

2) gas alkalosis

3) metabolic acidosis

4) metabolic alkalosis

35. Respiratory failure is a pathological syndrome in which:

1) O2 voltage in the blood does not change

2) O2 voltage in the blood falls

3) CO2 voltage in the blood does not change

4) CO2 voltage in the blood falls

36. Obstructive type of respiratory failure develops when:

1) bronchial asthma

2) pneumothorax

3) high aperture

4) pulmonary fibrosis

37. Restrictive type of respiratory failure develops when:

1) emphysema

2) chronic bronchitis

3) pulmonary fibrosis

4) tracheal stenosis

38. The excitability of the respiratory center increases as a result of:

1) cerebral hemorrhage

2) sleeping pills poisoning

3) increase of O2 voltage in the blood

4) increase the voltage of CO2 in the blood

39. The distance for diffusion of gases may increase with:

1) hyperventilation

2) violation of the mechanics of breathing

3) increasing the number of functioning alveoli

4) fibrotic changes in the lungs

40. Dyspnea (dyspnea) is:

1) frequent deep breathing

2) frequent shallow breathing

3) rare deep breathing

4) feeling of lack of air

41. Bradipnoe observed when:

1) lowering blood pressure

2) thyrotoxicosis

3) fever

4) inhibition of the respiratory center

42. Centrogenic ventilation failure occurs when:

1) pulmonary pathology

2) pathology of the respiratory system

3) pathology of the respiratory center

4) pathology of the respiratory muscles

43. When depression of the respiratory center occurs:

1) diffusion form of respiratory failure

2) ventilation form of respiratory failure

3) perfusion form of respiratory failure

4) valve mechanism of bronchial obstruction

44. The reason for Kussmaul breathing is:

1) the effect of ketone bodies on the respiratory center

2) hypocapnia

3) hypercapnia

4) hypoxemia

45. Tachypnea is:

1) frequent, shallow breathing

2) frequent, deep breathing

3) irregular breathing

4) rare, shallow breathing

46. For the restrictive form of respiratory failure characterized by:

1) increase in VC, decrease in Tiffno index

2) reduction of VC, OOL, OEL

3) decrease in VC and Tiffno index

4) FEV1 reduction, Tiffno index 50%

47. Periodic is breathing:

1) fabric

2) Kussmaul

3) Gasping breath

4) Cheyne-Stokes

48. In the pathogenesis of periodic respiration matters:

1) decrease in sensitivity of the respiratory center to CO2

2) increased sensitivity of the respiratory center to CO2

3) stimulation of the respiratory center

4) reduction of the respiratory surface of the lungs

49. To the terminal carry breathing:

1) Biota

2) Cheyne-Stokes

3) gasping

4) bradypnea

50. Obstructive disorders of pulmonary ventilation are noted when:

1) pneumothorax

2) asbestosis

3) angioedema

4) alveococcosis

51. Absolute coronary insufficiency is observed when:

1) anemia

2) cardiac muscle hypertrophy

3) prolonged tachycardia (thyrotoxicosis)

4) coronary embolism

52. In myocardial infarction, the ischemia zone on the ECG is characterized by:

1) change of a P wave

2) negative T wave

3) ST interval rise

4) deep Q wave

53. In case of myocardial infarction, it is typical for the ECG damage zone:

1) change of a P wave

2) negative T wave

3) ST interval rise

4) deep Q wave

54. In myocardial infarction, the necrosis zone on the ECG is characterized by:

1) change of a P wave

2) negative T wave

3) ST interval rise

4) deep Q wave

55. Atherosclerosis is a chronic disease:

1) arterial vessels of large and medium size

2) capillaries

3) venous vessels

4) lymphatic vessels

56. Atherogenic factors include:

1) HDL - high density lipoproteins

2) vegetable fatty acids

3) Vit. C

4) animal fatty acids

57. At what disease of the endocrine glands does atherosclerosis develop faster?

1) with thyrotoxicosis

2) with hypergonadism

3) with pituitary hyperfunction

4) with diabetes

58. Non-coronary factor causing cardiac muscle necrosis:

1) excessive accumulation of catecholamines in the heart muscle

2) coronary thrombosis

3) atherosclerosis of the coronary vessels

4) long spasm of the coronary arteries

59. Coronary insufficiency is:

1) inadequate blood supply to the brain

2) inadequate blood supply to the liver

3) inadequate blood supply to the lung

4) inadequate blood supply to the heart muscle

60. The characteristic violations of metabolic processes in cardiomyocytes during ischemia include:

1) reducing the formation of ATP

2) increase in ATP

3) potassium accumulation

4) hypohydration

6**1. Coronarogenic coronary insufficiency is:**

1) atherosclerosis of the coronary vessels

2) catecholamine necrosis

3) toxic and inflammatory necrosis

4) autoimmune myocardial damage

62. Non-coronary factor causing cardiac muscle necrosis:

1) atherosclerosis of the coronary vessels

2) coronary thrombosis

3) autoimmune myocardial damage

4) long spasm of coronary vessels

63. A possible consequence of acute coronary insufficiency is:

1) arterial hypertension

2) heart failure

3) increase in cardiac output

4) hypervolemia

64. What are the hemogram indicators characteristic of acute myocardial infarction:

1) lymphopenia

2) neutrophilic leukocytosis with a shift to the left

3) eosinophilia

4) neutropenia with a nuclear shift to the right

65. Relative coronary insufficiency is observed when:

1) excessive physical exertion

2) coronary spasm

3) atherosclerosis of the coronary vessels

4) thrombosis of coronary vessels

66. The main clinical manifestations of coronary insufficiency do not include:

1) myocardial infarction

2) angina pectoris

3) cardiosclerosis

4) hypertension

67. The basis of acute myocardial infarction is:

1) sclerosis of the heart muscle

2) necrosis of cardiomyocytes

3) ischemia of cardiomyocytes

4) pathological arterial hyperemia of the heart muscle

68. Angina is based on:

1) sclerosis of the heart muscle

2) necrosis of cardiomyocytes

3) ischemia of cardiomyocytes

4) pathological arterial hyperemia of the heart muscle

69. The basis of cardiosclerosis is:

1) sclerosis of the heart muscle

2) necrosis of cardiomyocytes

3) ischemia of cardiomyocytes

4) pathological arterial hyperemia of the myocardium

70. Complications of acute myocardial infarction include:

1) hypertensive crisis

2) cardiogenic shock

3) myocarditis

4) diabetes

71. Coronary insufficiency is:

1) inadequate pumping function of the heart

2) myocardial automaticity violation

3) myocardial conduction disturbance

4) inadequate supply of myocardium with oxygen

72. The compensatory mechanism for myocardial ischemia is:

1) development of arrhythmias

2) increased sympathetic effects on the heart

3) increased collateral circulation

4) accumulation of lipid peroxidation products

73. The characteristic violations of metabolic processes in cardiomyocytes during ischemia include:

1) reducing the formation of ATP

2) increase in ATP

3) potassium accumulation

4) hypohydration

74. Myocardial hibernation is:

1) cardiac muscle necrosis

2) reversible myocardial dysfunction

3) myomalacia cardiac muscle

4) myocardial rupture

75. With which disease of the endocrine glands does atherosclerosis develop faster?

1) with thyrotoxicosis

2) with hypergonadism

3) with pituitary hyperfunction

4) with diabetes

76. At what disease of the endocrine glands does atherosclerosis develop more slowly?

1) with myxedema

2) with diabetes

3) in hypogonadism

4) with goiter's disease

77. Coronarogenic causes of coronary insufficiency include:

1) electrolyte steroid necrosis

2) catecholamine necrosis

3) autoimmune myocardial damage

4) coronary thrombosis

78. Coronarogenic coronary insufficiency is:

1) electrolyte steroid necrosis

2) prolonged spasm of the coronary vessels

3) catecholamine necrosis

4) toxic and inflammatory necrosis

79. Non-coronary factor causing cardiac muscle necrosis:

1) excessive accumulation of catecholamines in the heart muscle

2) coronary thrombosis

3) atherosclerosis of the coronary vessels

4) long spasm of the coronary arteries

80. Coronary insufficiency is:

1) inadequate blood supply to the brain

2) inadequate blood supply to the liver

3) inadequate blood supply to the lung

4) inadequate blood supply to the heart muscle

81. Coronary insufficiency may result from:

1) stenotic coronary sclerosis

2) accumulation of adenosine in the myocardium

3) myocarditis

4) hypercapnia

82. The basis of acute myocardial infarction is:

1) sclerosis of the heart muscle

2) necrosis of cardiomyocytes

3) ischemia of cardiomyocytes

4) pathological arterial hyperemia of the heart muscle

83. Clinical equivalents of transient myocardial ischemia are:

1) angina pectoris

2) hibernation of the myocardium

3) sudden cardiac death

4) all answers are correct

84. Atherogenic factors include:

1) HDL - high density lipoproteins

2) vegetable fatty acids

3) Vit. C

4) animal fatty acids

85. A possible consequence of acute coronary insufficiency is:

1) arterial hypertension

2) heart failure

3) increase in cardiac output

4) hypervolemia

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1) lymphopenia

2) neutrophilic leukocytosis with a shift to the left

3) eosinophilia

4) neutropenia with a nuclear shift to the right

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4) with diabetes

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3) venous vessels

4) lymphatic vessels

9**1. In myocardial infarction, ischemia on an ECG is characterized by:**

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2) negative T wave

3) ST interval rise

4) deep Q wave

92. Clinical equivalents of transient myocardial ischemia are:

1) angina pectoris

2) hibernation of the myocardium

3) sudden cardiac death

4) all answers are correct

93. Non-coronary factors that cause necrosis of the heart muscle:

1) excessive accumulation of catecholamines in the heart muscle

2) coronary thrombosis

3) atherosclerosis of the coronary vessels

4) long spasm of the coronary arteries

94. Coronary insufficiency is:

1) inadequate blood supply to the brain

2) inadequate blood supply to the liver

3) inadequate blood supply to the lung

4) inadequate blood supply to the heart muscle

95. When myocardial infarction increases the activity of enzymes:

1) creatine phosphokinase (CPK)

2) aspartate aminotherase (AST)

3) lactate dehydrogenase (LDH)

4) all answers are correct

96.In myocardial infarction, the necrosis zone on the ECG is characterized by:

1) change of a P wave

2) negative T wave

3) ST interval rise

4) deep Q wave

97. The compensatory mechanism for myocardial ischemia is:

1) development of arrhythmias

2) increased sympathetic effects on the heart

3) increased collateral circulation

4) accumulation of lipid peroxidation products

98. Relative coronary insufficiency is observed when:

1) excessive physical exertion

2) coronary spasm

3) atherosclerosis of the coronary vessels

4) thrombosis of coronary vessels

99. The main clinical manifestations of coronary insufficiency do not include:

1) myocardial infarction

2) angina pectoris

3) cardiosclerosis

4) hypertension

100. The basis of cardiosclerosis is:

1) ischemia and necrosis of cardiomyocytes

2) apoptosis of cardiomyocytes

3) hyperplastic processes in the myocardium

4) pathological arterial hyperemia of the myocardium

101. What factor plays a leading role in the occurrence of increased bleeding in leukemia?

1) thrombocytopenia

2) decrease in the content of procoagulants in the blood plasma

3) increased activity of the fibrinolytic system

4) increased plasma anticoagulant activity

102. What cells are most characteristic of acute myeloblastic leukemia?

1) myeloblasts

2) myelocytes

3) metamyelocytes

4) stab neutrophils

103. For which leukemia is the formation of the "Philadelphia" chromosome characteristic?

1) chronic monocytic

2) chronic megakaryocytic

3) chronic myeloid leukemia

4) chronic lymphocytic leukemia

104. The bodies of Botkin-Humprecht in a blood smear are:

1) hypochromic erythrocytes

2) “severed shoulder” of the Philadelphia chromosome

3) destroyed lymphocytes

4) neutrophils with toxic granularity

105. Eosinophilic-basophilic association occurs with:

1) chronic lymphocytic leukemia

2) chronic myeloid leukemia

3) acute myeloid leukemia

4) acute lymphocytic leukemia

106. What is the difference between leukemia and leukemoid reaction (choose the wrong statement)?

1) higher leukocytosis in leukemia

2) leukemia is an independent disease, and the leukemoid reaction is a symptom of another disease

3) in case of leukemia, blast forms of cells appear in the blood, and in case of a leukemoid reaction there is no

4) leukopenia

107. Blood smears of patients with acute myeloblastic leukemia mainly contain:

1) lymphoblasts

2) erythroblasts

3) megakaryoblasts

4) myeloblasts

108. Leukoses most often develop after the action:

1) hypoxic factor

2) thermal factor

3) mechanical injury

4) ionizing radiation

109. In acute leukemia, the tumor tissue mainly consists of:

1) class 6 mature cells

2) class 5 maturing cells

3) a class 1 potent stem cell

4) "blast" cells of 2-3-4 classes

110. "Leukemic failure" is most characteristic of:

1) acute lymphoblastic leukemia

2) acute myeloid leukemia

3) chronic myeloid leukemia

4) chronic lymphoblastic leukemia

111. The term "leukemic failure" means:

1) severe anemia

2) leukocyte shift to the left

3) the absence in the leukocyte formula of maturing neutrophils in the presence of blasts

4) a sharp increase in ESR

112. A significant increase in cells in the peripheral blood is characteristic of:

1) leukemic leukemia

2) subleukemic leukemia

3) aleukemic form of leukemia

4) chronic leukemia

113. In chronic lymphoblastic leukemia, blood smears are most common:

1) myeloblasts

2) monoblasts

3) erythroblasts

4) the shadows of Botkin-Humprecht

114. Severe hemorrhagic syndrome in leukemia caused by:

1) lack of vitamin K

2) a decrease in blood leukocytes

3) thrombocytopenia

4) reduction of reticulocytes

115. The bodies of Botkin-Humprecht in a blood smear are:

1) hypochromic erythrocytes

2) “severed shoulder” of the Philadelphia chromosome

3) destroyed lymphocytes

4) neutrophils with toxic granularity

116. The difference between acute and chronic leukemia is determined by:

1) the degree of differentiation of tumor cells

2) the duration of the disease

3) the severity of clinical manifestations

4) the cause of the disease

117. Anemia in leukemia is associated with _____

1) a decrease in circulating blood volume

2) inhibition of erythropoiesis

3) inhibition of spleen function

4) leukopenia

118. In acute leukemia, the main substrate of the tumor is:

1) undifferentiated "blast" elements or progenitor cells of class I or II

2) mature cells

3) more mature cells differentiated in the direction of certain hemopoiesis sprouts

4) mature cells

119. "Philadelphia" chromosome - the most characteristic feature:

1) acute lymphoblastic leukemia

2) acute myeloid leukemia

3) chronic lymphoblastic leukemia

4) chronic myeloid leukemia

120. The most common cause of death for leukemia is:

1) reduced nutrition

2) dysfunction of the respiratory system

3) dysfunction of the cardiovascular system

4) total oppression of blood formation

121. The main symptom of acute leukemia is:

1) the presence of a large number of blast cells in the peripheral blood and red bone marrow

2) Botkin-Humprecht shadows

3) eosinophilic-basophilic association

4) the presence of the Philadelphia chromosome

122. Indicate the change in the body with leukemia:

1) myocardial infarction

2) secondary immunodeficiency

3) increase in blood pressure

4) retinopathy

123. The predominance in the blood of blast forms of granulocytes and the absence of intermediate forms of maturation with a small number of mature cells ("leukemic failure") is characteristic of:

1) chronic leukemia

2) acute leukemia

3) leukemoid reaction

4) leukocytosis

124. For leukemoid reactions characteristic is:

1) basophilic-eosinophilic association

2) hyperregenerative leukocyte shift to the left

3) leukemic failure

4) pancytopenia

125. Indicate the correct definition of leukemia:

1) increase in the number of leukocytes per unit volume of blood

2) the extremely pronounced degree of activation of the immune system, resulting in a massive release into the blood of certain forms of leukocytes

3) activation of proliferation of normal cells of leukopoietic tissue

4) tumor of hematopoietic tissue with primary localization in the bone marrow

126. The general pathogenesis of leukemia includes the following steps (select the wrong statement):

1) proliferation

2) initiation

3) promotion

4) progression

127. Clinical and laboratory indicators of chronic lymphocytic leukemia include:

1) reticulocytosis

2) basophilic-eosinophilic association

3) Philadelphia chromosome

4) Botkin's Taurus - Humprecht

128. Chronic leukemia include:

1) lymphoblastic leukemia

2) erythremia

3) myeloblastic leukemia

4) all answers are correct

129. What changes in peripheral blood are not characteristic of chronic myeloid leukemia?

1) the presence of myelocytes

2) “leukemic failure”

3) thrombocytopenia

4) the appearance of isolated myeloblasts

130. Leukoses most often develop after the action:

1) hypoxic factor

2) thermal factor

3) mechanical injury

4) ionizing radiation

131. At which leukemia in the blood cells and bone marrow is the Philadelphia chromosome detected?

1) in acute myeloid leukemia

2) with chronic myeloid leukemia

3) with acute lymphocytic leukemia

4) with chronic lymphocytic leukemia

132. Acute leukemia is:

1) a tumor, the substrate of which consists of mature cells that have not lost their ability to differentiate

2) a tumor, the substrate of which is immature cells that have lost their ability to differentiate

3) a tumor that is characterized by the appearance of a basophilic-eosinophilic association

4) a tumor that is characterized by the appearance of the Philadelphia chromosome

133. Leukemia progression is characterized by:

1) inhibition of normal sprouts of hematopoietic tissue with the development of anemia, thrombocytopenia, leukopenia

2) activation of proliferation of normal cells of leukopoietic tissue

3) increased release of leukocytes from the blood-forming organs, including immature forms

4) absence of blast cells in the peripheral blood

134. For the leukemic form of leukemia is always characteristic:

1) leukopenia

2) pronounced leukocytosis

3) erythrocytosis

4) disappearance of blood blast cells

135. Severe hemorrhagic syndrome in leukemia is caused by:

1) lack of vitamin K

2) a decrease in blood leukocytes

3) thrombocytopenia

4) reduction of reticulocytes

136. Eosinophilic-basophilic association occurs with:

1) chronic lymphocytic leukemia

2) chronic myeloid leukemia

3) acute myeloid leukemia

4) acute lymphocytic leukemia

137. The most common cause of death for leukemia is:

1) reduced nutrition

2) dysfunction of the respiratory system

3) dysfunction of the cardiovascular system

4) total oppression of blood formation

138. The tumor substrate (main cells) for acute leukemia are:

1) mature blood cells

2) blast cells

3) stem cells

4) lymphopoiesis precursor cells

139. Acute leukemia include

1) lymphocytic leukemia

2) erythremia

3) myeloblastic leukemia

4) all answers are correct

140. Thrombocytopenia in acute leukemia is associated with:

1) with the destruction of platelets in the bloodstream

2) destruction of platelets in the spleen

3) with inhibition of megakaryocytic germ

4) insufficient function of megakaryocytes

141. What blood cells are most characteristic of acute myeloblastic leukemia?

1) Botkin - Gumprecht calf

2) basophils, eosinophils

3) myeloblasts

4) lymphoblasts

142. What is the most common cause of death for acute leukemia:

1) bleeding

2) development of respiratory failure

3) liver function disorder

4) renal dysfunction

143. The general pathogenesis of leukemia includes the following steps (select the wrong statement):

1) proliferation

2) initiation

3) promotion

4) progression

144. Leukoses most often develop after the action:

1) hypoxic factor

2) thermal factor

3) mechanical injury

4) ionizing radiation

145. The difference between acute and chronic leukemia is determined by:

1) the degree of differentiation of tumor cells

2) the duration of the disease

3) the severity of clinical manifestations

4) the cause of the disease

146. Anemia in leukemia is associated with ...

1) a decrease in circulating blood volume

2) inhibition of erythropoiesis

3) inhibition of spleen function

4) leukopenia

147. The main symptom of acute leukemia is:

1) the presence of a large number of blast cells in the peripheral blood and red bone marrow

2) Botkin-Humprecht shadows

3) eosinophilic-basophilic association

4) the presence of the Philadelphia chromosome

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3) myeloblastic leukemia

4) all answers are correct

149. What changes in peripheral blood are not characteristic of chronic myeloid leukemia?

1) the presence of myelocytes

2) “leukemic failure”

3) thrombocytopenia

4) the appearance of isolated myeloblasts

150. What white blood cells are most often found in a smear of a patient with acute myeloid leukemia?

1) prolymphocytes

2) lymphocytes

3) lymphoblasts

4) myeloblasts

151. What changes are characteristic of obstructive (mechanical) jaundice:

1) direct bilirubin increased in blood

2) there is a lot of urobilin in the urine

3) in feces much sterkobilin

4) there are no bile acids in the blood

152. When obstructive jaundice occur:

1) intestinal digestive disorders

2) hypervolemia

3) violation of the antitoxic function of the liver

4) tachycardia

153. What changes are characteristic of hepatic (parenchymal) jaundice:

1) in feces many sterkobilin

2) indirect bilirubin increased in blood

3) there are no bile acids in the blood

4) there is a lot of urobilin in the urine

154. Characteristic changes developing in cholemia:

1) increase in blood pressure

2) BP does not change

3) pruritus

4) hypovolemia

155. What changes are characteristic of suprahepatic (hemolytic) jaundice:

1) steatorrhea

2) there is no urobilin in the urine

3) indirect bilirubin increased in blood

4) many bile acids in the blood

156. The cause of hemolytic jaundice is:

1) intestinal digestive disorders

2) anemia

3) increased antitoxic function of the liver

4) bradycardia and hypotension

157. When parenchymal jaundice occurs the following violation:

1) intestinal digestive disorders

2) anemia

3) violation of the antitoxic function of the liver

4) tachycardia

158. Characteristic change developing with cholemia:

1) bradycardia

2) increase in blood pressure

3) BP does not change

4) tachycardia

159. Characteristic changes developing in cholemia:

1) increase in blood pressure

2) BP does not change

3) tachycardia

4) pruritus

160. Changes in the blood composition that occurs after complete removal of the liver in experimental animals:

1) increase in urea content

2) increase in ammonia content

3) hyperglycemia

4) increase in the content of prothrombin

161. Characteristic changes developing in cholemia:

1) increase in blood pressure

2) decrease in blood pressure

3) BP does not change

4) tachycardia

162. When obstructive jaundice occurs the following disorders:

1) intestinal digestive disorders

2) hypervolemia

3) violation of the antitoxic function of the liver

4) tachycardia

163. In chronic liver failure, the following changes in the protein composition of the blood are observed:

1) hypoproteinemia

2) increase in urea

3) increase in the content of prothrombin

4) increase in uric acid content

164. Changes in the protein composition of blood plasma corresponding to the term "paraproteinemia":

1) the change in the ratio between protein fractions

2) change in total protein content

3) appearance of qualitatively altered globulins

4) increase in the amount of albumin

165. Name the carbohydrate metabolism disorder observed in liver failure:

1) inhibition of glycogenesis

2) carbohydrate metabolism does not change

3) hyperglycemia

4) activation of gluconeogenesis

166. What is the violation of fat metabolism observed in liver failure?

1) increased formation of liver lipoproteins

2) increased triglyceride formation

3) increased formation of ketone bodies

4) reducing the formation of ketone bodies

167. Factor causing the development of hemolytic jaundice:

1) obturation of stones of the bile duct

2) incompatible blood transfusion

3) violation of the innervation of the biliary tract

4) changes in the neurohumoral regulation of the liver

168. Factor causing the development of parenchymal jaundice:

1) blood transfusions incompatible

2) the introduction of alloxan

3) phloridzin poisoning

4) phosphorus poisoning

169. Factor causing the development of obstructive jaundice:

1) blood transfusions incompatible

2) phenylhydrazine poisoning

3) obturation with stones of the common bile duct

4) phosphorus poisoning

170. After complete removal of the liver occurs:

1) increase in blood sugar

2) increase in NH3 in the blood

3) hyperlipidemia

4) increase in albumin in the blood

181. What is the carbohydrate metabolism disorder observed in liver failure?

1) inhibition of glycogenesis

2) carbohydrate metabolism does not change

3) hyperglycemia

4) activation of gluconeogenesis

182. When imposing direct fistula Ekka-Pavlov's observed:

1) cessation of blood flow from the intestinal vessels to the liver

2) liver ischemia

3) hypoglycemic coma

4) hyperglycemia

183. The overlapping of Ekka-Pavlov's fistula leads to:

1) liver ischemia

2) liver plethora

3) hypoglycemic coma

4) hyperglycemia

184. When hepatic coma accumulates in the blood:

1) NH3

2) glucose

3) urea

4) albumin

185. When liver failure bleeding develops due to:

1) insufficiency of vit. A

2) insufficiency of vit. E

3) insufficiency of vit. K

4) insufficiency of vit. D

186. What pigments give dark color to urine in obstructive jaundice?

1) conjugated bilirubin

2) unconjugated bilirubin

3) urobilin

4) stercobilin

187. What type of jaundice in the urine can cause indirect bilirubin?

1) with hemolytic

2) with none of the listed

3) with hepatocellular

4) with mechanical

188. One way to prevent the development of coma in liver failure is to limit the diet:

1) carbohydrates

2) liquids

3) proteins

4) fat

189. What type of jaundice is hepatic transaminase blood production typical for?

1) hepatocellular

2) hemolytic

3) enzymopathic

4) for any type

190. The following factors are involved in the formation of ascites with portal hypertension of hepatic origin:

1) hypoproteinemia

2) increase in proteins in the blood

3) decrease in hydrostatic pressure in v. porta

4) inhibition of the renin-angiotensin-aldosterone system

191. The cause of suprahepatic jaundice may be:

1) poisoning with hepatotropic poisons

2) rhesus conflict between the mother and fetus

3) cholelithiasis

4) hepatitis A virus

192. Bradycardia in obstructive jaundice may be associated with:

1) increased intracranial pressure

2) decreased intracranial pressure

3) irritation of tissue receptors by bile acids

4) the Ashner reflex

193. What causes a change in the rhythm of cardiac activity in obstructive jaundice?

1) indirect bilirubin

2) direct bilirubin

3) bile acids

4) cholesterol

194. What syndrome is characterized by an increase in blood bile acids, itchy skin, bradycardia, a decrease in blood pressure?

1) cholemia

2) hepatolienal

3) acholias

4) cytolytic

195. What syndrome is characterized by a decrease in blood clotting, intestinal auto-intoxication, meteorism, steatorrhea:

1) cholemia

2) hepatolienal

3) acholias

4) cytolytic

196. What etiological factors cause primary liver damage?

1) Botkin's disease virus

2) circulatory failure

3) ionizing radiation

4) obesity

197. Portal hypertension may occur due to:

1) left ventricular heart failure

2) right ventricular heart failure

3) overlay porto-caval anastomosis

4) hypovolemia

198. The leading link in the pathogenesis of subhepatic jaundice is:

1) damage to hepatocytes

2) sialolithiasis

3) enhanced red blood cell hemolysis

4) violation of the outflow of bile

199. The yellow color of the skin and mucous membranes in hemolytic jaundice is due to

1) an excess of direct bilirubin in the blood

2) an excess of indirect bilirubin in the blood

3) the appearance of bile acids in the blood

4) increase in blood cholesterol

200. The leading pathogenesis of hepatic jaundice:

1) enhanced red blood cell hemolysis

2) violation of the outflow of bile

3) damage to hepatocytes

4) violation of the outflow of bile

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1) the change in the ratio between protein fractions

2) change in total protein content

3) appearance of qualitatively altered globulins

4) increase in the amount of albumin

202. For which jaundice is characteristic the appearance in the blood of hepatic transaminases?

1) hepatocellular

2) hemolytic

3) enzymopathic

4) for any type

203. The leading pathogenesis of hepatic jaundice:

1) enhanced red blood cell hemolysis

2) violation of the outflow of bile

3) damage to hepatocytes

4) violation of the outflow of bile

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2) liver plethora

3) hypoglycemic coma

4) hyperglycemia

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1) NH3

2) glucose

3) urea

4) albumin

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2) insufficiency of vit. E

3) insufficiency of vit. K

4) insufficiency of vit. D

210. Characteristic changes developing in cholemia:

1) increase in blood pressure

2) BP does not change

3) tachycardia

4) pruritus

211. Helicobacter pylori refers to

1. viruses

2. Gram-negative bacteria

3. Gram-positive bacteria

4. the simplest

212. The complications of peptic ulcer include

1. penetration

2. jaundice

3. dolichocolon

4. cholecystocholangitis

213. Achalasia accompanied

1. vomiting with admixture of bile

2. spit up after 1 hour

3. vomit undigested food

4. vomit digested food

214. The most objective method of determining the acidity of gastric juice is

1. one-time study on Boas-Ewald

2. fractional study followed by titration

3. pH-metry

4. acidosis

215. For verification of pancreatitis it is necessary to investigate

1. serum amylase level

2. blood sugar

3. clinical analysis of blood

4. serum calcium level

216. The breakdown products of fats, which are absorbed in the digestive tract of children and adults, mainly represent

1. phospholipids

2. triglycerides

3. glycerin

4. fatty acids

217. The consequences of stopping or dramatically reducing the flow of bile into the intestine include:

1. increased protein decay in the intestines

2. decrease in the absorption of vitamins B1, B2, C

3. enhancement of parietal fat splitting

4. decrease in carbohydrate absorption

218. Indicate a factor that is not involved in the burp pathogenesis:

1. fermentation and rotting in the stomach

2. cardiac spasm

3. pyloric spasm

4. reflex contraction of the muscles of the stomach and diaphragm

219. Indicate the possible cause of gastric hyposecretion:

1. increased parasympathetic stimulation of the stomach

2. damage to the occipital cells of the gastric mucosa

3. an increase in the production and release of histamine

4. reducing the production and secretion of secretin

220. In the splitting of fats in children and adults involved.

1. trypsinogen

2. enterokinase

3. amylase

4. lipase

221. Helicobacter pylori is primarily parasitic in

1. antrum of the stomach

2. body of the stomach

3. duodenal bulb

4. in the floor of the stomach

222. The most informative method for diagnosing the exacerbation of chronic pancreatitis is:

1. ultrasound examination of the pancreas

2. scatological

3. endoscopy of the upper gastrointestinal tract

4. determination of the level of pancreatic enzymes in the blood

223. The most characteristic sign of achalasia is

1. refusal to eat

2. vomiting while eating

3. vomiting of stagnant stomach contents

4. vomiting after eating

224. The most objective method of determining the acidity of gastric juice is

1. one-time study on Boas-Ewald

2. fractional study followed by titration

3. acidosis

4. pH-metry

225. The cause of antral gastritis is

1. duodenogastric reflux

2. injury of the gastric mucosa

3. anacid

4. helicobacteriosis

226. Pathogenetic agents for the treatment of acute pancreatitis include:

1. inhibitors of enzymes (kontikal, trasilol, etc.)

2. antibiotics

3. Enzymes (Creon, Pancreatin)

4. anticholinergics (atropine, platifillin)

227. The consequences of stopping or dramatically reducing the flow of bile into the intestine include:

1. a decrease in the absorption of vitamins B1, B2, C

2. increased protein decay in the intestines

3. enhancement of parietal fat splitting

4. decrease in carbohydrate absorption

228. Indicate the possible cause of gastric hyposecretion:

1. increased parasympathetic stimulation of the stomach

2. reduced production and secretion of gastrin

3. an increase in the production and release of histamine

4. reducing the production and secretion of secretin

229. Pathological increased appetite is indicated by the term:

1. hyperrexia

2. polyphagia

3. aphagia

4. anorexia

230. Indicate that NOT is a consequence of Acholia:

1. deterioration of the digestion and absorption of fat

2. violation of the activity of the intestinal microflora

3. deterioration of water and electrolyte absorption

4. weakening of intestinal motility

231. Indicate the possible cause of the development of gastric hyposecretion:

1. increased parasympathetic stimulation of the stomach

2. damage to the occipital cells of the gastric mucosa

3. an increase in the production and release of histamine

4. reducing the production and secretion of secretin

5. reduction of cholecystokinin secretion

232. Specify gastrointestinal hormone, the excess of which causes pancreatic juice hypersecretion:

1. gastrin

2. cholecystokinin

3. motilin

4. histamine

233. Specify the possible reasons for the decrease in secretory activity of the pancreas:

1. increased parasympathetic gland stimulation

2. weakening of parasympathetic gland stimulation

3. increased production and release of cholecystokinin

4. increasing the production and secretion of secretin

234. When indomitable vomiting arise

1. Hyperkalemia

2. Hypernatremia

3. Hypochloremia

4. Metabolic acidosis

235. What vitamins are absorbed significantly with Acholia?

1. Vitamin A, D, E, K

2. Vitamin B1, B2, B6

3. Vitamin C

4. Vitamin B12, folic acid

236. Specify the possible cause of gastric hypersecretion:

1. an increase in the production and release of somatostatin

2. deficiency of gastrin production

3. an increase in the formation and release of histamine in the wall of the stomach

4. increase in histaminase activity

237. Atonic constipation occurs when:

1. intestinal spasms

2. lead intoxication

3. malabsorption

4. fiber deficiency in food

238. Mechanical intestinal obstruction occurs

1. with spasm or paralysis of the intestinal muscles;

2. with thrombosis of the vessels of the intestinal wall;

3. with paresis of intestinal muscles;

4. with intestinal tumors and helminthiases.

239. Pathogenesis of intestinal auto-intoxication due to toxic effects.

1. products of protein rotting in the intestines and biogenic amines (cadaverine, putrescine)

2. indirect bilirubin

3. ketone bodies

4. bile acids

240. What are the manifestations of intestinal malabsorption syndrome:

1. weight loss, asthenia, polyhypovitaminosis, edema, anemia

2. vomiting, belching, heartburn,

3. arterial hypertension,

4. obesity, hypercholesterinemia.

241. The excitatory postsynaptic potential of a neuron corresponds to:

1. Depolarization of the membrane

2. Repolarization of the membrane

3. Hyperpolarization of the membrane

4. No change in the potential difference of the membrane

242. Neurotropic viruses and bacteria do not apply to:

1) streptococcal exotoxin

2) tetanus toxin

3) botulinum toxin

4) polio virus

243. Neurotropic toxic effect has:

1) aldosterone

2) adenosine

3) meteonin

4) alcohol

244. Antinociceptive system is

1) a generator of pathologically enhanced excitation

2) pain control system

3) pain relief system

4) system for the formation of epicritical pain

245. Mediators of the antinociceptive system include

1) bradykinin

2) endorphin

3) histamine

4) substance p

246. Hypoxia is more sensitive:

1) brain stem

2) spinal cord

3) the cerebral cortex

4) all answers are correct

247. The acute phase of neuron damage is associated with:

1) increasing the concentration of Ca2 + in the cytosol

2) a decrease in the entry of Ca2 + into the neuron

3) the flow of Na + into the neuron

4) Na + release from neuron

248. "Loss of function" of the nervous system may occur when (specify the wrong answer):

1. significant damage to the nerve centers

2. deep inhibition of the nerve centers

3. superficial inhibition of nerve centers

4. epileptic seizure

249. Slow antegrade axonal transport provides:

1. delivery of substances for synaptic activity

2. delivery of trophogen to tissue

3. delivery of trophogens to the neuron

4. Antibody penetration into the central nervous system

250. For denervation syndrome characterized by:

1. the presence of trophic disorders in the zone of reservation

2. the absence of trophic disorders in the zone of reservation

3. distorting the sensitivity of the denervated structure to biologically active substances

4. Decreased sensitivity of the denervated structure to biologically active substances.

251. Axis retrograde transport may cause (specify an incorrect answer):

1. delivery of substances for synaptic activity

2. delivery of trophogen to neuron

3. virus penetration into the central nervous system

4. Antibody penetration into the central nervous system

252. How do conditioned reflexes change when the cerebral cortex is completely removed in dogs:

1) conditioned reflexes do not change

2) conditioned reflexes are enhanced

3) conditioned reflexes are reduced

4) conditioned reflexes disappear completely

253. The generator of pathologically enhanced excitation of the nervous system results from:

1. sustainable neuron excitation

2. sustainable hyperpolarization of neurons

3. disinhibition of neurons

4. 4. deep inhibition of neurons

254. The value of education GPUV

1) is the determinant of the pathological system

2) promotes the formation of the physiological system

3) enhances the trophic effect of the neuron on the innervated structures

4) is the basis for the development of neuropathological processes

255. Neurogenic dystrophy is

1) autoimmune brain damage

2) impaired endorphin synthesis

3) metabolic disorders in tissues with the breakdown of their innervation

4) parabiosis of nerve cells

256. General pathology of the nervous system studies:

1) patterns and typical mechanisms of development of pathological processes in the nervous system

2) participation of the nervous system in the regulation of physiological and pathological processes in the body

3) methods of neuroengineering

4) manifestations of mental illness

257. The neural route of entry of the pathogenic agent into the central nervous system is characteristic of:

1) ethyl alcohol

2) influenza virus

3) polio virus

4) staphylococcus

258. For spinal shock is characterized by:

1. irreversible loss of reflexes

2. reversible loss of reflexes

3. violation of reflexes above the place of brain break

4. activation of inhibitory effects on the brain

259. Treat hypokinesias (specify the wrong answer):

1) clonic convulsions

2) paresis

3) triplegia

4) paralysis

260. Epilepsy is caused by the occurrence of a generator of pathologically enhanced excitation in:

1) cerebral cortex

2) thalamus

3) the cerebellum

4) spinal cord

261. The endogenous factor of damage to the nervous system is:

1) ionizing radiation

2) botulinum toxin

3) excessive activation of lipid peroxidation (LPO)

4) polio virus

262 Changes in stage I of partial removal of the cerebral cortex:

1) there are no major changes in the central nervous system

2) the excitation process is enhanced

3) limiting inhibition occurs

4) conditional reflexes are activated

263. What is phantom pain?

1) pain projected on amputated limbs

2) excruciating pain arising after injury to a large nerve

3) pain that occurs in certain areas on the surface of the skin during the development of pathology in the internal organs

4) all answers are correct

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2) promotes the formation of the physiological system

3) enhances the trophic effect of the neuron on the innervated structures

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1) increasing the concentration of Ca2 + in the cytosol

2) a decrease in the entry of Ca2 + into the neuron

3) the flow of Na + into the neuron

4) Na + release from neuron

268. What signs are characteristic of peripheral paralysis?

1) increased spinal reflexes

2) the appearance of pathological reflexes

3) muscle wasting

4) muscular hypotonia

269. In contrast to the "physiological", pathological pain is characterized by:

1) the occurrence of damage, excessive irritation or destruction of nerves and / or receptors

2) the occurrence of damage or irritation of the thalamic zone of the nervous system

3) decrease in resistance of the organism to pathogenic effects

4) all answers are correct

270.Mediators of the antinociceptive system are not:

1) Met-Enkephalin

2) leu-enkephalin

3) endorphin

4) substance P

271. The endogenous factor of damage to the nervous system is:

1) ethyl alcohol

2) herpes virus

3) increase in osmotic pressure of interstitial fluid

4) traumatic situations

272. The antinociceptive system is

1) a generator of pathologically enhanced excitation

2) pain control system

3) pain relief system

4) system for the formation of epicritical pain

273. What opiate system mediator is enkephalin?

1) hormonal

2) neural

3) neuronal neuropathic

4) hormonal non-opiate

274. The supraspinal PAS level includes:

1) aggregates of hyperactive neurons

2) sensitized receptors

3) thalamic nuclei

4) damaged nerves

275. Neurotropic toxic effect has:

1) aldosterone

2) adenosine

3) meteonin

4) alcohol

276. The second phase of neuron damage is associated with:

1) Ca2 + input to the neuron

2) a decrease in the entry of Ca2 + into the neuron

3) the flow of Na + into the neuron

4) Na + release from neuron

277. Reperfusion post-ischemic neuron damage is associated with:

1) the termination of receipt of Ca2 +

2) a decrease in O2 entry into the neuron

3) enhanced FLOOR

4) all answers are correct

278. What is hyperpathy?

1) intense pain with mild nociceptive irritation

2) maintaining a feeling of intense pain after the cessation of provoking irritation

3) an attack of pain when exposed to projection zones by non-nociceptive stimuli

4) all answers are correct

279. What processes can lead to excessive inhibition of the neuron?

1. hyperactivation of asparagine secretion into the sympathetic gap

2. increase the release of glycine in the sympathetic gap; hyperactivating glutanic acid receptor

3. moderate oxygenation

4. deprivation of afferent influences.

280. The neural route of entry of the pathogenic agent into the central nervous system is characteristic of:

1) ethyl alcohol

2) influenza virus

3) polio virus

4) staphylococcus

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1) bradykinin

2) endorphin

3) histamine

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1) Ca2 + input to the neuron

2) a decrease in the entry of Ca2 + into the neuron

3) the flow of Na + into the neuron

4) sodium release from neuron

283. The reperfusion posterior ischemic neuron damage is associated with:

1) termination of the entry of Ca2 +

2) a decrease in O2 entry into the neuron

3) enhanced FLOOR

4) all answers are correct

284. Pathological pain of peripheral origin occurs when:

1) the formation of a generator in the posterior horns of the spinal cord

2) damage to nocireceptors

3) formation of a generator in the nucleus of the trigeminal nerve

4) formation of a generator in the nuclei of the thalamus

285. The main substrate of oxidation in the neuron are:

1) proteins

2) lipids

3) glucose

4) all answers are correct

286. The acute phase of neuron damage is associated with:

1) increasing the concentration of Ca2 + in the cytosol

2) a decrease in the entry of Ca2 + into the neuron

3) the flow of Na + into the neuron

4) Na + release from neuron

287. Neurotropic toxic effect has:

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3) meteonin

4) alcohol

288. The value of education GPUV

1) is the determinant of the pathological system

2) promotes the formation of the physiological system

3) enhances the trophic effect of the neuron on the innervated structures

4) is the basis for the development of neuropathological processes

289. Neurogenic dystrophy is

1) autoimmune brain damage

2) impaired endorphin synthesis

3) metabolic disorders in tissues with the breakdown of their innervation

4) parabiosis of nerve cells

290. The supraspinal PAS level includes:

1) aggregates of hyperactive neurons

2) sensitized receptors

3) thalamic nuclei

4) damaged nerves

291. Neurotropic viruses and bacteria do not include:

1) streptococcal exotoxin

2) tetanus toxin

3) botulinum toxin

4) polio virus

292. Neurotropic toxic effect has:

1) aldosterone

2) adenosine

3) meteonin

4) alcohol

293. Neurosis occurs when:

1) synaptic transmission disorder

2) violation of the functions of GNI

3) spinal cord transections at various levels

4) partial decortication of the animal in the experiment

294. The following manifestation is characteristic of neurosis:

1) pathological changes in the cerebral cortex

2) locomotor and sensory disorders

3) peripheral nerve damage

4) peripheral paralysis

295. The following manifestation is characteristic of neurosis:

1) pathological changes in the cerebral cortex

2) neuro-trophic disorders

3) peripheral nerve damage

4) vestibular disorders

296. Neurosis is characterized by the following manifestation:

1) pathological changes in the cerebral cortex

2) disorders of the vegetative functions

3) peripheral nerve damage

4) vestibular disorders

297. The antinociceptive system is

1) a generator of pathologically enhanced excitation

2) pain control system

3) pain relief system

4) system for the formation of epicritical pain

298. Mediators of the antinociceptive system include

1) bradykinin

2) endorphin

3) histamine

4) substance p

299. Hypoxia is more sensitive:

1) brain stem

2) spinal cord

3) the cerebral cortex

4) all answers are correct

300. The acute phase of neuron damage is associated with:

1) increasing the concentration of Ca2 + in the cytosol

2) a decrease in the entry of Ca2 + into the neuron

3) the flow of Na + into the neuron

4) Na + release from neuron

301. Neurosis with a predominance of the excitation process is characterized by:

1) sleepiness

2) depression

3) decrease in motor activity

4) increased motor activity, fussiness

302. Neurosis with a predominance of the process of inhibition is characterized by:

1) aggressiveness

2) motor activity

3) depression and drowsiness

4) inadequate excitement, viciousness

303. In dogs with a strong unbalanced type of GNI, neurosis can be caused by:

1) increased excitatory process

2) reduced excitation process

3) strengthening the braking process

4) overstrain the process of internal braking

304. The main cause of neurosis in humans is:

1) increased muscular load

2) enhanced mental activity

3) decrease in physiological activity

4) psycho-emotional stress

305. How do conditioned reflexes change with complete removal of the cerebral cortex in dogs:

1) conditioned reflexes do not change

2) conditioned reflexes are enhanced

3) conditioned reflexes are reduced

4) conditioned reflexes disappear completely

306. Changes occurring in stage I of partial removal of the cerebral cortex:

1) there are no major changes in the central nervous system

2) the excitation process is enhanced

3) limiting inhibition occurs

4) conditional reflexes are activated

307. The value of education GPUV (3)

1) is the determinant of the pathological system

2) promotes the formation of the physiological system

3) enhances the trophic effect of the neuron on the innervated structures

4) is the basis for the development of neuropathological processes

308. Neurogenic dystrophy is

1) autoimmune brain damage

2) impaired endorphin synthesis

3) metabolic disorders in tissues with the breakdown of their innervation

4) parabiosis of nerve cells

309. The general pathology of the nervous system studies:

1) patterns and typical mechanisms of development of pathological processes in the nervous system

2) participation of the nervous system in the regulation of physiological and pathological processes in the body

3) methods of neuroengineering

4) manifestations of mental illness

310. The neural route of entry of the pathogenic agent into the central nervous system is characteristic of:

1) ethyl alcohol

2) influenza virus

3) polio virus

4) staphylococcus

311. The endogenous factor of damage to the nervous system is:

1) ionizing radiation

2) botulinum toxin

3) excessive activation of lipid peroxidation (LPO)

4) polio virus

312. The endogenous factor of damage to the nervous system is:

1) ethyl alcohol

2) herpes virus

3) increase in osmotic pressure of interstitial fluid

4) traumatic situations

313. Disruption of higher nervous activity is called:

1) nervousness

2) neurosis

3) neuralgia

4) epilepsy

314. The social factors of the development of neurosis include:

1) information overload

2) types of higher nervous activity

3) transferred diseases

4) endocrine organ conditions

315. The value of education GPUV (3)

1) is the determinant of the pathological system

2) promotes the formation of the physiological system

3) enhances the trophic effect of the neuron on the innervated structures

4) is the basis for the development of neuropathological processes

316. Neurogenic dystrophy is

1) autoimmune brain damage

2) impaired endorphin synthesis

3) metabolic disorders in tissues with the breakdown of their innervation

4) parabiosis of nerve cells

317. The disease, the development of which may be pathogenetically directly associated with neurosis

1) arterial hypertension

2) anemia

3) pneumonia

4) myxedema

318. Hypoxia is more sensitive:

1) brain stem

2) spinal cord

3) the cerebral cortex

4) all answers are correct

319. The acute phase of neuron damage is associated with:

1) increasing the concentration of Ca2 + in the cytosol

2) a decrease in the entry of Ca2 + into the neuron

3) the flow of Na + into the neuron

4) Na + release from neuron

320. Obsessive neurosis is:

1) "conflict" between the inflated demands of the individual to others and the inability to implement or achieve them

2) "conflict" between the inflated demands on himself and the inability to implement them

3) violation of mental activity, which is reflected in the disorder of the perception of the real world and disorganization of behavior.

4) “conflict” between desires, aspirations, personal needs and the inability to implement them for social or other reasons

321. Hysteria is:

1) “conflict” between desires, aspirations, the needs of the individual and the inability to implement them for social or other reasons

2) "conflict" between the inflated demands of the individual to others and the inability to implement or achieve them

3) "conflict" between the inflated demands on himself and the inability to implement them

4) violation of mental activity, which is reflected in the disorder of the perception of the real world and disorganization of behavior

322. The antinociceptive system is

1) a generator of pathologically enhanced excitation

2) pain control system

3) pain relief system

4) system for the formation of epicritical pain

323. In dogs with a strong unbalanced type of GNI, neurosis can be caused by:

1) increased excitatory process

2) reduced excitation process

3) strengthening the braking process

4) overstrain the process of internal braking

324. The main cause of neurosis in humans is:

1) increased muscular load

2) enhanced mental activity

3) decrease in physiological activity

4) psycho-emotional stress

325. What opiate system mediator is enkephalin?

1) hormonal

2) neural

3) neuronal neuropathic

4) hormonal non-opiate

326. The supraspinal PAS level includes:

1) aggregates of hyperactive neurons

2) sensitized nociceptors

3) thalamic nuclei

4) damaged nerves

327. Neurotropic toxic effect has:

1) aldosterone

2) adenosine

3) meteonin

4) alcohol

328. Neurosis occurs when:

1) synaptic transmission disorder

2) violation of the functions of GNI

3) spinal cord transections at various levels

4) partial decortication of the animal in the experiment

329. The second phase of neuron damage is associated with:

1) Ca2 + input to the neuron

2) a decrease in the entry of Ca2 + into the neuron

3) the flow of Na + into the neuron

4) Na + release from neuron

330. Post-ischemic reperfusion injury of a neuron is associated with:

1) the termination of receipt of Ca2 +

2) a decrease in O2 entry into the neuron

3) enhanced FLOOR

4) all answers are correct

331. Phobias are:

1) sustainable manifestation of various fears

2) "conflict" between the inflated demands of the individual to others and the inability to implement or achieve them

3) "conflict" between the inflated demands on himself and the inability to implement them

4) violation of mental activity, which is reflected in the disorder of the perception of the real world and disorganization of behavior

332. Mediators of the antinociceptive system include

1) bradykinin

2) endorphin

3) histamine

4) substance p

333. Neurosis with a predominance of the process of inhibition is characterized by:

1) aggressiveness

2) motor activity

3) depression and drowsiness

4) inadequate excitement, viciousness

334. The second phase of neuron damage is associated with:

1) Ca2 + input to the neuron

2) a decrease in the entry of Ca2 + into the neuron

3) the flow of Na + into the neuron

4) Na + release from neuron

335. Post-ischemic reperfusion injury of a neuron is associated with:

1) termination of the entry of Ca2 +

2) a decrease in O2 entry into the neuron

3) enhanced FLOOR

4) all answers are correct

336. Pathological pain of peripheral origin occurs when:

1) the formation of a generator in the posterior horns of the spinal cord

2) damage to nocireceptors

3) formation of a generator in the nucleus of the trigeminal nerve

4) formation of a generator in the nuclei of the thalamus

337. The main substrate of oxidation in the neuron are:

1) proteins

2) lipids

3) glucose

4) all answers are correct

338. The acute phase of neuron damage is associated with:

1) increasing the concentration of Ca2 + in the cytosol

2) a decrease in the entry of Ca2 + into the neuron

3) the flow of Na + into the neuron

4) Na + release from neuron

339. Disruption of higher nervous activity is called:

1) nervousness

2) neurosis

3) neuralgia

4) epilepsy

340. Neurotropic toxic effect has:

1) aldosterone

2) adenosine

3) meteonin

4) alcohol

Block D

List of questions and tasks for final examination:

Questions to check the level of training TO KNOW:

Questions to test the level of learning to KNOW:

1. The main historical stages of pathophysiology worldwide. Scientists who contributed to the development of pathophysiological as science.

2. The main sections of pathological physiology: General nosology, typical pathological processes, pathological physiology of body systems. Their characteristic.

3. Basic concepts and categories of General nosology: health, disease, disease periods.

4. Etiological factors of the disease: definition. Classification, their role at different stages of the disease. Conditions of occurrence of the disease: definition, types, meaning

5. Pathophysiological characteristics of the periods of overheating. The main changes in the function of organs and systems and metabolism during overheating.

6. The factors on which the damaging effect of the electric current depends.

7. Definitions of "cell damage". The main types of cell damage. Exogenous and endogenous factors (causes and conditions of damage) cells.

8. The outcomes of cell damage. Dystrophy, dysplasia, necrosis, apoptosis.

9. Definition of the concepts of reactivity and resistance of the organism. Types and forms of reactivity, their characteristics. Pathological reactivity.

10. Directed change of individual and group reactivity as the most important means of prevention and therapy of diseases.

11. The resistance of the organism, types and their characteristics. Cellular and humoral factors that provide resistance of the body.

12. Definition and General characteristics of allergies. Classification of allergic conditions.

13. Anaphylaxis, stages, characteristics. Sensitization: active and passive.

14. Definition and classification of hereditary forms of pathology.

15. Typical forms of peripheral circulatory disorders. Stasis, types, main causes and mechanisms of development and consequences.

16. Heart attack, species, outcomes. Collateral circulation, types of collaterals, mechanism of development. The value in pathology.

17. Embolism, types of embolism and their characteristics embolism.

18. Definition and General characteristics of inflammation. The etiology of the inflammation. Inflammatory mediators (cellular and humoral) and their role in the development of the inflammatory process.

19. The reasons for the transition of arterial hyperemia in the venous inflammation. Local and common signs of inflammation and mechanisms of their development.

20. Types of exudates and their characteristics. The difference of exudate from transudate (Rivolt test).

21. Definition of the concept and general characteristics of fever as TPP. Etiology of fever. Characteristics of exo- and endogenous pyrogenic substances. Leukocytic (true) pyrogens.

22. Types of fever, depending on the cause, the degree of rise in body temperature and the type of temperature curves.

23. Infectious process, definition, types of infectious process. Forms of relationships between macro- and microorganisms.

24. Kinds of infectious agents and their properties. Conditions for the emergence of the infectious process: the entrance gate, the pathways of the spread of infectious agents, the mechanisms of anti-infectious protection.

25. Stages of infectious diseases, characteristic. Mechanisms of protection of the organism from pathogens of infections: nonspecific (bactericidal and bacteriostatic, cellular and humoral, reflex) and specific.

26. Indicators characterizing the violation of protein metabolism. Nitrogen balance and its disturbances in pathology.

27. Types of starvation and their characteristics.

28. Hyperglycemia - types, causes, mechanisms of development and basic clinical manifestations.

29. The reasons for the violation of digestion and absorption of fats in the gastrointestinal tract. Steatorrhea. Hyperlipidemia, types and mechanisms of development (biochemical indicators).

30. Obesity, species and their characteristics. Factors contributing to the development of alimentary obesity.

31. The main forms of disorder ABB and their characteristics.

32. Types of edema. Etiology and pathogenesis.

33. Definitions of the concept of "tumor growth" and general characteristics of tumors. Etiology of tumors - types of carcinogens and their characteristics.

34. The concept of metastasis. Stages and ways of metastasizing in the body.

35. Definition of the concept and general characteristics of hypoxia and hypoxemia. Etiology and pathogenesis of the main types of endogenous hypoxia: respiratory, circulatory, hemic, tissue, overload, mixed.

36. General etiology and pathogenesis of insufficiency of external respiration.

37. The main causes and mechanisms of impaired diffusion and perfusion ability of the lungs. The main indicators of insufficiency of external respiration and their characteristics.

38. Sinus tachycardia, sinus bradycardia, sinus arrhythmia, types and causes of their occurrence.

39. Extrasystolia (sinus, atrial, atrioventricular, ventricular). Its causes and features of ECG changes depending on the place of origin.

40. The main causes and types of coronary insufficiency (relative and absolute). Stages of coronary insufficiency and their characteristics.

41. Classification and general characteristics of hypertensive states.

42. Fainting, collapse, shock. Types and mechanisms of development, manifestations and consequences.

43. Definition of the concept and classification of forms of circulatory insufficiency.

44. Heart defects, their types and characteristics.

45. Hypertrophy of the myocardium, stages and species: eccentric, concentric. Mechanisms of their development.

46. The main indicators of hemodynamics and their changes in CHF.

47. Changes in total blood: normo-; hypo- and hypervolemia, their types, causes and mechanisms of development.

48. Erythrocytosis: absolute and relative, the causes of the specificity of the etiology and pathogenesis of the true (absolute) polycythemic hypervolemia, a violation of hemodynamics.

49. Definitions of the concept and principles for the classification of anemic states.

50. General characteristics of iron deficiency anemia: chlorosis (early and late), alimentary iron deficiency anemia.

51. The concept of the external and internal factor of Castle (gastromukoprotein) and its role in the pathogenesis of Vit. In 12 and folic deficiency anemia.

52. Etiology and pathogenesis of acquired hemolytic anemia (HA).

53. The main causes of liver failure and signs of its manifestation.

54. Hepatitis, species. Etiology and basic manifestations and consequences.

55. Cholemia. Change in the system of blood, nervous system and cardiovascular system with cholemia.

56. General etiology and pathogenesis of typical forms of pathology of the digestive system. Typical disorders of taste, impaired appetite, salivation and swallowing.

57. Proteinuria, hematuria, types and mechanisms of development.

58. The mechanism of development of renal edema (nephrotic and nephritic) and their difference from cardiac.

59. General etiology and pathogenesis of endocrinopathies.

60. Hypopituitarism, hyperpituitarism, species, pathogenesis and manifestations:

Questions for testing the level of training TO LEARN:

61. The subject, the tasks of pathophysiology. Clinical pathophysiology, its goals and objectives. Its place among other medical sciences, the importance for the clinic.

62. Pathological process, typical pathological process, pathological condition, pathological reaction.

63. Etiology, definition. Preceding theories and modern general theses of etiology.

64. The concept of pathogenesis. The main link and the "vicious circle" in the pathogenesis of disease.

65. Adaptive (protective-adaptive, compensatory) mechanisms are an integral part of pathogenesis. Mechanisms of recovery.

66. External and internal causal factors of disease occurrence, their characteristics, general properties and features.

67. Overheating (hyperthermia) - factors that cause overheating of the body (causes and conditions), mechanisms for the development of hyperthermia.

68. Kinetosis: causes and clinical manifestations. Types of acceleration, the mechanism of action of accelerations on the body. Overloading views.

69. The mechanism of the action of electric current on the body.

70. Typical mechanisms of cell damage.

71. The role of free radicals in cell damage. Peroxide oxidation of lipids.

72. Adaptive and pathogenic value of cell death in normal and pathological conditions.

73. Factors determining reactivity: the role of the genotype, age, sex, constitution. The influence of the external environment, social and environmental factors on the reactivity of the organism.

74. Allergens. Types of allergens and their characteristics.

75. General pathogenesis and stages of allergic reactions.

76. Etiology of hereditary and congenital diseases. Mutagens and their types: exogenous (physical, chemical, biological), endogenous.

77. Mutations and their types. Characteristics of teratogenes.

78. Arterial hyperemia, types, causes, mechanisms of development.

79. Venous hyperemia, the main causes and mechanism of development.

80. Ischemia, types, causes, mechanisms of development.

81. Thrombosis. The main causes and conditions of blood clot formation in blood vessels.

82. Pathogenetic features of air and gas embolism.

83. Sludge. Causes, mechanism of development and consequences.

84. Stages of the inflammatory process (pathogenesis of inflammation). Alteration, species and their characteristics. The significance of primary and secondary alteration in inflammation.

85. Features of metabolic disorders and physico-chemical changes in the focus of inflammation.

86. Phases of vascular reaction in inflammation and mechanisms of their development.

87. Exudation. The importance of vascular and tissue factors in the mechanism of development of exudation. Adaptive and pathogenic importance of exudation in the development of inflammation.

88. Factors on which the type, composition and properties of the exudate depends.

89. Pathogenetic features of acute and chronic inflammation.

90. Pathogenesis of fever (change of set point).

91. Stages of fever. The relationship between heat production and heat transfer in various stages of fever. Critical and lytic temperature decrease.

92. Difference of fever from overheating.

93. Violation of basal metabolism in diseases of the thyroid gland, anemia and chronic heart failure.

94. Hyperproteinemia, hypoproteinemia - causes and consequences.

95. Factors affecting the duration of fasting. Fasting, as a method of treatment. Dietotherapy.

96. Hypoglycemia and hypoglycemic syndrome - types, causes, mechanisms of development and basic clinical manifestations.

97. Glucosuria - species, causes and mechanisms of development. Renal diabetes.

98. Etiology, pathogenesis and the main manifestations of diabetes insipidus.

99. Experimental forms of diabetes mellitus (pancreatic, alloxan, and floridazine).

100. Disturbance of interstitial metabolism of fat in diabetes mellitus. Hyperketonemia (ketosis), causes and mechanisms. Ketonuria.

101. The main causes and characteristics of endocrine obesity.

102. Etiology and pathogenesis of alimentary obesity.

103. Consequences of obesity. Violations of the functions of organs and systems for obesity.

104. Atherosclerosis, the definition of a concept, a general characteristic. General etiology and pathogenesis of atherosclerosis.

105. Gas acidosis and gas alkalosis. Causes and mechanisms of development.

106. Negative alkalosis and a negative acidosis. Causes and mechanisms of development.

107. Typical forms of water balance disturbance: hypo- and hyperhydration, species, etiology and pathogenesis.

108. Hypo-and hypernarthriemia. Causes and consequences.

109. Hypo-and hyperkalemia. Causes and consequences.

110. Hypo-and hypercalcemia. Causes and consequences.

111. Classification of tumors. The difference between benign tumors and malignant tumors.

112. Stages of tumor development (carcinogenesis) and their characteristics. The concept of proto-oncogenes, oncogenes, oncoproteins and their role in the cellular and molecular mechanisms of carcinogenesis.

113. Disturbance of metabolism and physiological functions in acute and chronic hypoxia.

114. Hypoxia - as a universal mechanism of damage and cell death.

115. Urgent and long-term adaptation mechanisms for hypoxia.

116. Hypoxic hypoxia: hypobaric and normobaric, hyperbaric - causes and mechanisms of development.

117. The main causes and mechanisms of violation of alveolar ventilation of the lungs (alveolar hypoventilation, alveolar hyperventilation).

118. Etiology and pathogenesis of pulmonary hypertension (precapillary, postcapillary, mixed).

119. Shortness of breath and its kinds. Causes and mechanisms of their development (Goring-Breyer reflex).

120. Periodic and terminal types of respiration (Biots, Chain-Stokes, Kussmaul, etc.). Causes and mechanism of their development.

121. Pathogenesis and consequences of paroxysmal tachycardia.

122. Pathogenesis of the main clinical manifestations of myocardial infarction: pain and resorptive-necrotic syndrome.

123. The significance of atherosclerosis and other risk factors in the mechanism of development of coronary insufficiency (atherogenic and lipotropic factors).

124. Hypertensive disease. Etiology and pathogenesis, risk factors of hypertensive disease.

125. Stages and main clinical manifestations of hypertension, and mechanisms for their development. Complications and consequences of hypertension.

126. Secondary (symptomatic) arterial hypertension, their types, causes and mechanisms of development.

127. Pathophysiological characteristics of chronic arterial hypotension: primary, secondary (symptomatic). Etiology and pathogenesis.

128. Pathogenetic classification of CHF (overload of blood volume, resistance overloading).

129. Compensation mechanisms for heart defects (urgent and non-urgent): isotonic and isometric hyperfunction of the heart, tonogenic and myogenic dilatation and their characteristics.

130. The mechanism of the transition of compensated defects to decompensated ones.

131. Metabolic disorders in CHF.

132. Causes of death and factors that determine the consequences of acute blood loss.

133. Compensation mechanisms for acute blood loss (blood loss phase). Mechanism of bone marrow phase of compensation in acute blood loss. Features of blood regeneration in case of blood loss in high altitude conditions.

134. Hypercoagulation. Thrombotic syndrome. The main causes and mechanisms of development, manifestations and consequences.

135. Hypocoagulation. Hemorrhagic syndrome. The main types, causes, mechanisms of development, manifestations and consequences.

136. Etiology and pathogenesis of iron deficiency anemia.

137. Etiology and pathogenesis vit.B12 and folic deficiency anemia.

138. The pathogenesis of the triad of symptoms in Vit.B12 and folic deficiency anemia.

139. Thalassemia. Etiology and pathogenesis. The picture of blood and the main clinical manifestations.

140. Sickle-cell anemia. Etiology and pathogenesis and basic clinical manifestations.

141. Etiology and pathogenesis, types of pathological leukocytosis: the mechanism of development, the change in the leukocyte formula.

142. Neutrophilous leukocytosis. Causes and mechanisms of development.

143. Eosinophilic and basophilic leukocytosis. Causes and mechanisms of development.

144. Leukopenia, types, causes and mechanisms of development. Agranulocytosis, species. Etiology and pathogenesis. The picture of blood and the consequences.

145. Definition of concept and common characteristic and principles of leukemia classification. The concept of monoclonal and polyclonal leukemia.

146. Signs of tumor progression in leukemia. The concept of "leukemia gaping", "blast crisis", aleukemic and leukemia leukemia phases.

147. General characteristics and pattern of blood in acute myeloid (AML) and lymphoblastic leukemia (ALL).

148. General characteristics and pattern of blood in chronic myelocytic leukemia (CML).

149. General characteristics and pattern of blood in chronic lymphocytic leukemia (CLL).

150. Etiology and pathogenesis, general characteristic and picture of blood in Vaquez disease.

151. Violation of the barrier and detoxification of the liver.

152. Cirrhosis of the liver, species. Etiology and pathogenesis. The consequences of the development of liver cirrhosis.

153. Disturbance of metabolism in liver failure.

154. Jaundice. Types and their characteristics:

155. Portal hypertension. Types, causes, the main manifestations of its consequences.

156. Types of disorders of gastric secretion: hypersecretion, hyposelection and achilles. The main causes and consequences.

157. Typical forms of impaired motor function of the stomach. The main causes and consequences.

158. Typical forms of impaired motor function of the intestine: diarrhea and constipation. The main types and mechanisms of occurrence and consequences.

159. General etiology and pathogenesis of intestinal diseases: malabsorption syndrome, chronic enteritis, chronic colitis and irritable bowel syndrome. Its main clinical manifestations.

160. Etiology and pathogenesis of peptic ulcer of the stomach and duodenum. Its main manifestations and complications.

161. Nephrosis and nephrotic syndrome (primary and secondary). The main causes and manifestations.

162. Pathophysiological characteristics of chronic diffuse glomerulonephritis (CDH), and pyelonephritis.

163. Definition of the concept and basic indicators of renal failure.

164. Etiology and pathogenesis of acute renal failure (ARF).

165. Etiology and pathogenesis of chronic renal failure (CRF).

166. Stages of chronic renal failure and their pathophysiological characteristics.

167. The main causes of violations of the central mechanisms of regulation of peripheral endocrine glands (violation of transhypophysial regulation, violation of parahypophyseal regulation, the role of disturbances in the feedback mechanism.

168. Etiology of primary disorders of peripheral endocrine gland function (disruption of biosynthesis and hormone secretion).

169. Etiology and pathogenesis of peripheral (extragranular) forms of endocrine disorders.

170. Pathological characteristics of the hypo- and hyperfunction of the anterior lobe of the pituitary gland. The mechanism of the violation of protein, carbohydrate and fat metabolism in the case of hypersecretion of growth hormone (STH).

171. General etiology, pathogenesis and the main manifestations of hypothyroidism: myxedema, cretinism.

172. Endemic goiter. Etiology and pathogenesis, pathophysiological characteristics of the main manifestations. The role of autoimmune and environmental factors in the development of endemic goiter.

173. General etiology and manifestations of hyperthyroidism. Pathogenesis of metabolic disorders and changes in the functions of organisms and systems in diffuse toxic goiter (Basedova disease).

174. Etiology pathogenesis of adrenogenital (corticogenital) syndrome , and its main types and manifestations (virilism-masculinization and feminization).

175. The general etiology and pathogenesis of violations of the endocrine function of sexual glands and their main manifestations: hypogonadism, eunuchoidism, hypergonadism.

176. General etiology, pathogenesis of disorders of the nervous system (damage to neurons, violation of inter-neuronal interactions, disorder of integrative activity).

177. Etiopathogenesis of disorders of the locomotor function of the nervous system: hypo- and hyperkinesia, hypodynamia, ataxia - disorders of coordination of movements.

178. Etiopathogenesis of neurogenic sensitivity disorders: anesthesia and hypesthesia, hyperesthesia and dysesthesia.

179. Pathological pain of peripheral and central origin: thalamic, phantom and causal.

180. Disturbance of the autonomic nervous system, damage to the hypothalamus, sympathetic and parasympathetic innervation. Vegetative neuroses and their characteristics.

Questions (tasks) for testing the level of training TO BE ABLE:

181. Methods of pathological physiology. Experiment as the main method of pathophysiology. Importance of the experiment. Types and stages of the experiment.

182. Signs of the disease. Nature of the course of the disease. Relapses, remissions. Outcomes and complications of the disease.

183. Pathogenic effect of the thermal factor. Burns, types and degrees of burns and their characteristics.

184. Burn disease, clinical stages and their pathophysiological characteristics. The pathogenesis of burn shock.

185. Pathogenesis of heat and sun stroke, pathogenetic principles of first aid.

186. Etiology and pathogenesis of the development of type I allergic reactions by Gell and Coombs. Clinical forms.

187. Characteristics of allergens, mediators and mechanisms of development of cytotoxic and cytolytic allergic reactions of type II according to Gell and Coombs, their role in pathology. Clinical forms.

188. Characteristics of allergens, mediators and mechanisms of development of immunocomplex allergic reactions of type III according to Gell and Coombs, their role in pathology. Clinical forms.

189. Characteristics of allergens, mediators and mechanisms for the development of type IV allergic reactions according to Gell and Coombs, their role in pathology. Clinical forms.

190. Pathogenesis of anaphylactic shock in humans. Features of the course of experimental anaphylactic shock in guinea pigs, dogs and rabbits.

191. Desensitization, hyposensitization. General principles of diagnosis, prevention and treatment of allergic diseases.

192. Methods of diagnosis of hereditary diseases (demographic, genealogical, twin method, cytogenetic, biochemical, immunological method, dermatoglyphic, experimental).

193. Signs and consequences of arterial and venous hyperemia.

194. Signs and consequences of ischemia

195. Stages and mechanisms of thrombus formation in blood vessels. The fate of the thrombus and the consequences of thrombosis.

196. Features of the etiology, pathogenesis and course of embolism of the pulmonary artery and portal vein. Thromboembolic disease, etiology and pathogenesis.

197. Stages and mechanisms of emigration of leukocytes to the focus of inflammation. The role of leukocyte emigration in the development of the inflammatory process.

198. Features of metabolism, changes in the function of organs and systems in various stages of fever.

199. Meaning of a febrile reaction for the body. Pathogenetic principles of antipyretic therapy. Pyrotherapy.

200. Mechanism of development of protein-free edema (Crogh-Starling scheme).

201. Hypazotemia - types and mechanisms of development.

202. Disturbance of metabolism and changes in the functions of organs and systems during fasting, depending on the stage and periods of fasting.

203. Clinical forms of protein-energy deficiency - alimentary dystrophy (alimentary marasmus) and kwashiorkor.

204. Etiology and pathogenesis of diabetes mellitus (type 1 diabetes and diabetes mellitus type 2).

205. Disturbance of metabolism in diabetes mellitus. Main clinical and biochemical manifestations of diabetes mellitus and their mechanism of development.

206. Pathogenesis of acute (early) and chronic (late) complications of diabetes mellitus. Differentiation of com with diabetes mellitus.

207. The role of violations of fat metabolism in the development of atherosclerosis. Modern ideas about the pathogenesis of atherosclerosis.

208. Modern theories of carcinogenesis.

209. Features of growth and metabolism of tumor tissue. Effect of tumor on the body. The concept of paraneoplastic syndrome. Pathogenesis of cancer cachexia.

210. Etiology and pathogenesis of mountain sickness. The difference between mountain sickness and altitude.

211. Primary pulmonary and primary-extrapulmonary forms of external respiratory failure. Etiology and pathogenesis of obstructive and restrictive types of disturbance.

212. Etiology and pathogenesis of respiratory and circulatory disorders in bronchial asthma and pulmonary emphysema.

213. Etiology and pathogenesis of respiratory and circulatory disorders in pneumonia and pulmonary edema, including alpine ones.

214. Etiology and pathogenesis of respiratory and circulatory disorders in various types of pneumothorax.

215. Blockade of the heart, types and mechanisms of development. The period of Venkenbach-Samoilov and the peculiarities of the IV degree of atrioventricular blockade.

216. Pathogenesis of atrial fibrillation (ventricular fibrillation).

217. Complications and outcomes of angina pectoris and myocardial infarction. Pathogenesis of cardiogenic shock, clinical manifestations.

218. Pathogenesis of renal arterial hypertension (the theory of Goldblatt and Grollman).

219. Clinical (subjective and objective) manifestations of Chronic renal failure and mechanisms of their development.

220. Mechanisms of cardiac edema development and their difference from renal.

221. Pathological forms (regenerative and degenerative) of red blood. Reticulocytes and their importance for understanding the pathogenesis of various forms of anemia.

222. Picture of blood in acute posthemorrhagic anemia, depending on the period of blood loss.

223. DIC is a syndrome. Etiology and pathogenesis, stages, manifestations and consequences. Pathogenetic principles of therapy and prevention of DIC syndrome.

224. The picture of blood in iron deficiency anemia.

225. The picture of blood and features of hematopoies in Vit.B12 and folic deficiency anemia.

226. Shifts of the leukocyte formula to the left and to the right with neutrophilic leukocytosis, pathophysiological characteristics and significance for understanding the reactivity of the organism in pathology.

227. Leukemoid reactions, types, causes and mechanisms of development and their difference from leukemia.

228. The pathogenesis of leukemia (clone theory) in the light of modern concepts. Features of metastasis of tumor cells in leukemia.

229. The mechanism of development of secondary clinical manifestations in leukemia: anemic, hemorrhagic, intoxication, infectious, metastatic syndromes.

230. Pathogenesis of the hepatic coma and its main manifestations.

231. Experimental reproduction and study of liver function insufficiency (complete and partial removal of the liver, angiostomy by London, fistula Ekka -Pavlova (direct and indirect)).

232. Methods of differentiation of jaundice (direct and indirect bilirubin, stercobilin and urobilin).

233. Change in daily diuresis and relative density of urine: polyuria, oliguria and anuria, hypo- and isostenuria of their cause and diagnostic significance.

234. Pathogenesis of uremia and uremic (kidney) coma and its main manifestations.

235. Etiology and pathogenesis of hypercortisolism. The main manifestations of the syndrome and Diseases of Itsenko-Cushing and the mechanisms of their development.

236. Etiology and pathogenesis of chronic insufficiency of the adrenal cortex (Addison's disease), its main manifestations and mechanisms of development.

237. Pathophysiology of pain. The concept, a general characteristic of pain: physiological and pathological. Biological and pathogenic significance of pain.

238. Violation of the trophic function of the nervous system. Neurodystrophy and denervation syndrome. Main manifestations and mechanism of development.

239. Consequences of complete and partial removal of the cerebral cortex. The value of protective braking and its application in the clinic.

240. Mechanisms of the occurrence of the functions of CNS. Neuroses, concept, types, characteristics. Etiology of neuroses, manifestations in humans. The role of features of CNS in the emergence and developme