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Учреждение образования
«ГРОДНЕНСКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ»

Кафедра патологической физиологии

ПАТОЛОГИЧЕСКАЯ ФИЗИОЛОГИЯ

в 2-х частях

Часть 1

ОБЩАЯ ПАТОФИЗИОЛОГИЯ

Пособие для студентов факультета иностранных учащихся с английским языком обучения

GENERAL PATHOPHYSIOLOGY

in 2 parts

Part 1

GENERAL PATHOPHYSIOLOGY

Manual for the faculty of foreign students

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Manual «Pathophysiology. Manual in 2 parts for the faculty of foreign students. Part 1. General Pathophysiology» is intended for the faculty of foreign students with English medium of instruction for successful studying the subject. It contains the program on Pathophysiology, list of practical skills, and information on purposes and questions to topics. It contains the information on purposes and questions to the topics, the description of the laboratory functioning, tests, tasks, literature, questions to the tests.
PROGRAMME ON PATHOPHYSIOLOGY

I. GENERAL PATHOPHYSIOLOGY

SUBJECT OF PATHOPHYSIOLOGY


Health and disease. Periods of a disease (latent, beginning of disease, manifestations of a disease, outcomes of diseases).

DISORDERS OF MICROCIRCULATION


INFLAMMATION


Leukocyte recruitment: margination, adherence to vascular wall, emigration through vascular wall, chemotaxis. Phagocytosis. Stages: chemotaxis, adherence to bacteria, absorption (phagosome formation), digestion (phagolysosome formation). Pus. Composition. Proliferation and regeneration. Types to regenerations. Categorization of inflammation: according to velocities of development (acute, subacute, chronic), according to dominating stage (alterative, exudative, proliferate), according to the exudate type (serous, festering, fibrinous, hemorrhagic and others).

Local signs of inflammation (rubor, tumor, dolor, color, functio laesae) and mechanisms their development. General reactions at the inflammation (fever, leukocytosis, increase growing of settling an red blood cells and others). Inflammation mediators (histamine, bradykinine, complement system, prostaglandins, leukotrienes). Role of mediators. Outcomes of inflammation. Factors, influencing upon the current of inflammation. Biological role of inflammation.
THERMAL REGULATORY DYSFUNCTION.
FEVER. HYPERTHERMIA. HYPOTHERMIA

Fever. Causes and mechanisms of fever development, its biological role, etiopathogenesis and consequences of hyperthermia and hypothermia influence on the organism.


REACTIVITY AND RESISTANCE. IMMUNOLOGICAL DISORDERS. CATEGORIZATION. AIDS.

Reactivity. Types of reactivity (typical, group, individual). Physiological and pathophysiological reactivity. Specific and nonspecific mechanisms of reactivity.

Degree of reactivity (normoergic, hyperergic and hypergic). Resistance. Local and common resistance, hereditary and acquired resistance. Factors, which determine the reactivity and resistance (heredity, constitution, sex, environmental and social factors). Immunological disorders. Categorization. AIDS. Etiology. Clinical symptoms and mechanisms of their development.

ALLERGY

Allergy. Causes. Allergens, their categorization and description. Types of allergic reactions. Pathogenesis of
allergic reactions of different types. Stages of allergy: (immunological stage, pathochemical stage, pathophysiological stage). Allergic mediators. Sensibilization. Experimental model of anaphylactic shock on guinea pig. Description of allergic disorders (allergic rhinitis (pollinosis), bronchial asthma, e.g.). Desensibilization (hyposensibilization).

PATHOPHYSIOLOGY OF METABOLISM.

FASTING


Consequences of total and partial fasting (carbohydrates, lipids and protein deficite). Protein-calorie malnutrition. Particularities in children (Kwashiorkor).

PATHOLOGY OF CARBOHYDRATE METABOLISM


Causes and consequences of carbohydrate intermediary metabolism. Causes and consequences of lipid intermediary metabolism.

PATHOLOGY OF LIPID METABOLISM


PATHOLOGY OF PROTEIN METABOLISM.


Gout. Pathogenesis. Symptoms and therapy.

PATHOLOGY OF WATER-MINERAL BALANCE

Edema, classification. Factors influencing edema development. Pathogenesis of cardiac, renal, hepatic, inflammatory, allergic, toxic edema.

Disturbances of mineral metabolism (Na\(^+\), K\(^+\), Ca\(^{+2}\)).

CLASSIFICATION OF ACID-BASE DISTURBANCES


PATHOLOGY OF VITAMINES METABOLISM

Deficiency of water-soluble vitamins (B\(_1\), B\(_2\), B\(_6\), B\(_{12}\), C, PP). Deficiency of fat-soluble vitamins (A, D, E, K).

CELL PATHOLOGY


**HYPOXIA. HYPEROXIA**


**ROLE OF HEREDITY IN PATHOLOGY**

EXTREME CONDITIONS. STRESS. SHOCK. COMA. COLLAPSE


TUMOR GROWING

The tumor growth. Malignant and nonmalignant tumors. Description.


Pathogenesis of tumor growth. Stages: transformation, promotion, progression.


Antitumor activity of human organism.

RADIATION SICKNESS

Radiation. Issues and power of different radiation rays. Units of radiation assay.

II. PATHOPHYSIOLOGY OF ORGANS AND SYSTEMS

PATHOPHYSIOLOGY OF BLOOD
DISORDERS OF CIRCULATIVE BLOOD VOLUME (HYPERVOLEMIA, HYPOVOLEMIA). BLEEDING


Erythrocytosis. Polycythemia vera or Wakes’s disease.


ANEMIAS

Clinical symptoms of anemia and mechanisms of their development. Categorization of anemias by: etiopathogenesis, color parameter, severity of anemia, regenerative possibility, type of hematopoiesis, erythrocyte’s size.

Post-hemorrhagic anemia. Description, the picture of the blood in acute and chronic post-hemorrhagic anemia.

Vitamin B₁₂ and folic acid deficiency anemia. Etiology, pathogenesis. The picture of the blood. Addison-Biermer disease.

Hemolytic anemia. Types (congenital, autoimmune e.g.). The picture of the blood. Clinical symptoms. Newborn hemolytic anemia.

**LEUKOCYTOSIS. LEUCOPENIA**

Stages of leucopoiesis. Leukocyte’s description on different stages of leucopoiesis. Leukocyte’s functions (neutrophils, eosinophils, basophils, monocytes, lymphocytes).


Leukocyte’s formula. Nuclear shift of leukocyte’s formula to the left, to the right. Clinical importance of leukocyte’s formula calculation.

**LEUKEMIA**


Characteristic of morphological picture of blood in acute and chronic myeloid and lymphoid leukemia. Leukemia clinical syndromes.

Clinical syndromes in leukemia: anemic, hemorrhagic, infective, metastatic, and intoxicative. Leukemic reactions and pathogenesis.
PATHOLOGY OF HEMOSTASIS

Components of hemostasis (blood vessel, platelets, plasma coagulation factors), primary and secondary hemostasis. Disorders of hemostasis (thrombophylic, hemorrhagic, thrombohemorrhagic hemostasiopathias).

Thrombosis
Outcomes and consequences. Mechanism of white and red thrombosis development. Causes and conditions of thrombosis development. Virchow’s triad (vascular injury, blood coagulant system activation, blood flow decrease).

Hemorrhagic disorders of hemostasis


Disseminated intravascular coagulation (DIG). Causes. Stages, mechanisms of development.

PATHOPHYSIOLOGY OF NERVOUS SYSTEM

PATHOPHYSIOLOGY OF HIGH NERVOUS SYSTEM ACTIVITY

Classification of types of high nervous system activity. A role of the generator of pathologically enhanced excitation, pathological system in occurrence of a pathology of the excitatory system. Common etiology of high nervous system activity disorders.


Influence of alcohol on the organism. The characteristic of manifestations of acute intoxication. Severity levels.


Infringements of intelligence, memory, emotions, behaviour, etc.

The characteristic of manifestations and causes of sleep-wakefulness cycle disorders.

**PATHOPHYSIOLOGY OF ENDOCRINE SYSTEM**


**PATHOPHYSIOLOGY OF CARDIOVASCULAR SYSTEM**
Kinds of blood circulation insufficiency (acute and chronic, it is left - and right heart). Etiology and pathogenesis and main symptoms of acute heart failure.


Causes of death at a myocardium heart attack. Cardiogenic shock and acute pulmonary edema. Etiology and pathogenesis.

Clinical symptoms of insufficiency of blood circulation and substantiation of mechanisms of their development.

The characteristic of changes of heart hemodynamics in insufficiency of mitral valves and foramens stenoses.

**ARRHYTHMIAS**


**HYPERTENSION. HYPOTENSION**

Factors involved in regulation of blood pressure. Severity levels of an arterial hypertensia. Stages.


Hypotension. Causes.

**PATHOLOGY OF RESPIRATORY SYSTEM**

Pulmonary volumes and capacities. Respiratory failure (etiology, causes, symptoms). Types of alveolar ventilation disturbances. Obstructive and restrictive disturbances.


Pathogenesis of periodic breathing (Cheyne-Stokes, Biote, Cussmaul).
**PATHOPHYSIOLOGY OF KIDNEY**

Role of kidney in the organism. Etiology and pathogenesis of kidney disease.


Consequences of chronic glomerulonephritis.

Pyelonephritis. Ethiopathogenesis. The characteristic of infringements.

Nephrotic syndrome, Clinical manifestations. Pathogeny of hypostases in nephrotic syndrome.

Pathogenesis of nephritic and nephrotic oedema.

Renal insufficiency. Uraemia. Concept of hemodialysis (an artificial kidney).

Urolithic illness. Factors and the mechanisms promoting formation of stones.

**PATHOPHYSIOLOGY OF THE LIVER**

Studying of liver functions in experiments and clinic. Functional trials.


Insufficiency of the liver. Causes. Development stages. Their characteristic. Hepatic coma. Symptoms and
mechanisms of their occurrence. Concept of haemosorption. Liver transplantation.


Jaundice. Kinds. The characteristic of infringements of a pigmental exchange at separate kinds of jaundices (haemolytic, liver, mechanical).

Cholelithiasis. The basic mechanisms and manifestations of cholelithiasis. Cholestasis. The causes of cholestasis. Disturbances in an organism at cholestatic syndrome.


PATHOPHYSIOLOGY OF GASTROINTESTINAL TRACT

Role of digestive organs. Aetiology of gastrointestinal disorders. A role of alcohol, smoking and other factors in their occurrence.


Digestion distresses in oral cavity (mastications, salivations, swallowings). The causes and consequences. Dental caries. Ethiopathogenesis, preventive maintenance.


Pancreatitis. Etiology and pathogenesis. Consequences.
LIST OF PRACTICAL SKILLS

List of practical skills:
The students have to:

1. **Use:**
   - obtained knowledge in clinical practice;
   - electrocardiogram in cardiac arrhythmia diagnosis;
   - parameters of pulmonary ventilation for revealing of disorders of lung functions;
   - blood gas parameters to determine hypoxia type;
   - parameters of pigment metabolism to determine jaundice type;
   - data of stomach juice for eliciting of pathological gastric secretion type;
   - obtained practical skills during experiments on the animals, anesthesia, taking of investigative material, analysis of obtained results and conclusions according *Principles for the Care and Use of Animals* recommended by the Ethical Committee

2. **Master** the methods of:
   - calculation and estimation of value in blood:
     - red blood cells (erythrocytes) level;
     - white blood cells (leucocytes) level;
     - platelets (thrombocytes) level;
     - reticulocytes level;
     - leukocytemic formula;
     - hematocrit
     - hemoglobin;
     - color index
   - determining of pathological blood cells in blood samples;
   - differentiation of leukemia by blood picture;
   - performing in experimental animals:
     - anesthesia;
     - subcutaneous, intravenous, intramuscular infusions
     - taking of blood samples;
     - registration of temperature
     - registration of electrocardiogram
LESSON № 1

Topic:
SUBJECT AND PURPOSE OF PATHOPHYSIOLOGY

Aim of the lesson: to study problems and methods of pathophysiology, to get an idea of etiology, pathogenesis, and disease

STUDY QUESTIONS:

1. Subject of Pathophysiology. Main parts:
   a. General Pathophysiology:
      - Study of disease (nosology)
      - Study of typical (standard) pathological processes
   b. Systems of Pathophysiology (pathophysiology of organs and systems)
2. Purpose of Pathophysiology
4. Etiology. Description of main etiological factors.
5. Pathogenesis.
6. Importance of etiology and pathogenesis study.
7. Pathological reactions.
8. Pathological process.

LITERATURE:
1. Lecture material.
Fig. 1. Characteristic of disease.

The relationship between aetiology, pathogenesis, morphological and functional manifestations and sequelae is exemplified by four diseases. A. Boil. B. Lung cancer. C. Cirrhosis. D. Hypertension
Fig. 2. Common causes of death in developed countries
LESSON № 2

Topic:  
DISORDERS OF PERIPHERAL CIRCULATION

Aim of lesson: to study the causes, mechanisms and manifestations of microcirculation disturbances.

QUESTIONS:

1. Central and peripheral circulation, their correlation.
3. Classification of microcirculation disturbances.
4. Arterial hyperemia: causes, symptoms, and consequences.
5. Venous hyperemia: causes, symptoms, and consequences.
6. Ischemia: causes, symptoms, and consequences.
7. Stasis: causes, symptoms, and consequences.
11. Embolism by a blood clot.
Fig. 3. Structure of microcirculatory unit

Fig. 4. Fat emboli in arterial vessels of frog peritoneum

Students analyze, draw and make conclusions.
Fig. 5. Pulmonary embolism and infarction

LABORATORY WORKS

Laboratory work 1. Modulation of arterial hyperemia on the rabbit ear.

Description of the work: Students perform the control of skin color and analyze the vasculature structure of rabbit ear. Then students massage one of a rabbit’s ear and fix the changes.

Fig. 4. Arterial hyperemia on the rabbit ear
Laboratory work 2. Compressive ischemia on the rabbit’s ear.

Description of the work: Students perform the control of skin color and analyze the vasculature structure of rabbit ear. Then students compress the main vessels of the ear and fix the symptoms of ischemia. After decompression we observe the development of reactive arterial hyperemia.

Laboratory work 3. Modulation of peripheral circulation disorders on a human’s arm (Konchalovsky test).

Description of the work: The cuff is put on a volunteer’s shoulder and arterial pressure is estimated. The pressure in the cuff should make 5-10 mm Hg less then maximal during 5 min. The students look for venous hyperemia development and fix the symptoms. After cuff removal students fix the vasculature changes on the shoulder.

Tasks:

A patient's left leg is cool, pale, pulseless and painful. His right leg is warm, purple, swollen and painful, but still has a pulse. Which leg is more likely to have an arterial thrombus, and which one – a venous thrombus? Explain. What are causes (your differential diagnoses) for each type of occlusion?
Tests:

1. *Arterial hyperemia is:*
   a) organ augmented blood flow due to increase of blood supply from the arterial vessels
   b) organ augmented blood flow as a result of impaired venous return from a tissue
   c) increased amount of blood in the organism
   d) hematocrit increase

2. *Clinical signs of arterial hyperemia are:*
   a) redness
   b) cyanosis
   c) skin temperature increase
   d) skin temperature decrease

3. *Types of physiological arterial hyperemia are:*
   a) functional
   b) inflammatory
   c) postischemic
   d) after action of some physical and chemical factors (mustard plaster, heat)
   e) conditioned by reflex reaction (shame blush)

4. *The causes of pathological arterial hyperemia are:*
   a) inflammation
   b) organ reperfusion
   c) action of vacuum
   d) exercises
   e) elimination of vessels sympathetic innervation influence
5. **Postischemic hyperemia occurs due to:**
   a) organ reperfusion
   b) quick removing of ascetic fluid
   c) paralysis of vasoconstrictive nerves or their centers

6. **The causes of neuroparalytic arterial hyperemia are:**
   a) transection of vasoconstrictive vegetative nerves
   b) temporal stagnation of the circulation
   c) action of vacuum

7. **How does microcirculation change in arterial hyperemia?**
   a) dilation of arterioles
   b) constriction of venes
   c) opening of nonoperation capillaries

8. **The mechanisms of increased temperature in arterial hyperemia are:**
   a) increased warmth of arterial flow
   b) intensification of oxidative processes
   c) inhibition of respiratory enzymes activity
   d) decreased heat emission

9. **The causes of pathological arterial hyperemia are:**
   a) action of pathogenic factors
   b) in physical work
   c) injury of soft tissues

10. **The causes of venous hyperemia are:**
    a) increased arterial flow
    b) impediment outflow of venous blood
    c) thrombosis of abdominal veins
11. **The causes of venous hyperemia are:**
   a) thrombosis of veins  
   b) exercise stress  
   c) compression of veins by tumor  
   d) heart failure  

12. **The causes of venous hyperemia in organs of abdominal cavity are:**
   a) thrombosis of the portal vein  
   b) thrombosis of mesenteric arteries  
   c) left-sided heart failure  
   d) right-sided cardiac failure  
   e) hepatic cirrhosis  

13. **Clinical signs of venous hyperemia are:**
   a) cyanosis  
   b) redness  
   c) reduced organ temperature  
   d) increased organ temperature  
   e) organ reduction  

14. **The causes of reduction of the skin temperature in venous hyperemia are:**
   a) intensification of heat loss  
   b) disconnection of oxidation and phosphorilation in mitochondria  
   c) heat production decrease  
   d) feebleness circulation  

15. **The causes of stasis are:**
   a) impediment blood outflow in veins  
   b) increased blood flow
c) decreased blood flow
d) increased blood outflow from organs
e) increased blood viscosity

16. The causes of capillary stasis are:
a) erythrocyte aggregation (sludge)
b) venous thrombosis
c) reduced blood flow to capillaries
d) decreased hematocrit

17. Ischemia is the decreasing of blood flow due to:
a) decreasing blood flow in arteries
b) anemia
c) low count of erythrocytes in blood
d) decreased hematocrit

18. The clinical signs of ischemia are:
a) skin paleness
b) cyanosis
c) redness
d) decreased temperature of the organ
e) increased temperature of the organ

19. Peculiarities of microcirculation in ischemia are:
a) feebleness circularity
b) increased blood flow
c) reduction of capillaries diameter
d) extension of capillaries diameter

20. The organs with functionally absolute sufficient collaterals are:
a) distal part of lower extremities
b) brain
c) heart

d) upper extremities

21. **Following vasodilators are synthesized in endothelium:**
a) prostacyclin
b) prostaglandin F2α
c) NO
d) acetylcholine
e) endothelin

22. **Following vasoconstictors are synthesized in endothelium:**
a) prostacyclin
b) prostaglandin F2α
c) NO
d) acetylcholine
e) endothelin

23. **What form of NO-synthase takes part in vasodilatation:**
a) cerebral
b) endothelial
c) induced (macrophage)

24. **Glutamate toxicity participates in:**
a) neuroparalitic arterial hyperemia
b) brain ischemia
c) portal hypertension
d) true capillary stasis in the heart

25. **Reperfusion syndrome occurs due to:**
a) resuscitation
b) intra-arterial infusion of blood
c) removing of ascetic fluid from the abdominal cavity
d) thrombolysis in arteries
e) intravenous infusion of plasma substitutes

26. The main pathogenic mechanism of reperfusion syndrome is:
   a) development of edema
   b) oxidative stress
   c) deficiency of energy
   d) action of lysosomal enzymes

   Answers:

   1a, 2ac, 3ade, 4abce, 5ab, 6a, 7ac, 8ab, 9ac, 10bc, 11acd, 12ade, 13ac, 14acd, 15ace, 16a, 17a, 18ad, 19ac, 20ad, 21ac, 22be, 23b, 24b, 25 acd, 26b.

LITERATURE:
1. Lecture material.
LESSON № 3

Topic: INFLAMMATION. ALTERATION AND EXUDATION

Aim of the lesson: to study causes and mechanisms of main inflammation signs development.

QUESTIONS:

1. Inflammation (flogosis).
3. Inflammation stages (alteration, exudation and emigration, proliferation).
4. Alteration. Primary and secondary alteration.
5. Inflammation mediators (histamine, bradykinin, complement system, prostaglandins, leukotrienes). Role of mediators.
6. Particularities of metabolism at alteration.
7. Physicochemical changes at alteration.
8. Description of microcirculation disorders in the area of inflammation (short ischemia, arterial hyperemia, venous hyperemia, stasis) and mechanisms of their development.
10. Leukocyte recruitment:
   - margination, adherence to vascular wall,
   - emigration through vascular wall,
   - chemotaxis.
Fig. 5. The components of typical light microscope

1. Base
2. Support column
3. Body tube
4. Nosepiece
5. Stage
6. Coarse focus knob
7. Fine focus knob
8. Condenser focus knob
9. Eyepiece
10. Objective lens
11. Stage rotation knob
Fig. 6. Disturbances of microcirculation in inflammation

LABORATORY WORKS

Laboratory work 1. *Alteration and vasculature reactions on frog tongue during inflammation.*

**Description of the work:** The frog is fixated on the laboratory table. The frog’s tongue should be isolated. The students analyze the normal microcirculation in the organ under microscope. Then salt (AgNO$_2$) should be put on the center of the tongue and students fix the changes, draw inflammatory zones and make conclusions.
Fig. 7. Fixation of a frog

Fig. 8. Frog’s tongue preparation
Laboratory work 2. *The vasculature reactions on frog mesentery during inflammation (Kogeim experiment).*

**Description of the work:** The frog is fixated on the laboratory table.

The mesentery should be isolated and during 60 min students watch the microcirculative disturbances and leukocytes recruitment during inflammation. The students pay attention to white blood cells marginating and passing through the vascular wall, draw emigrated leukocytes and make conclusions.

Fig. 9. Fixation of frog’s mesentery
Fig. 10. Frog’s mesentery

Fig. 11. Adhesion and emigration of neutrophils in response to chemotactic agents
Fig. 12. Migration of white blood cells from the vessels
1 – leukocytes standing outside of the vessel;
2 – transition of leukocytes through the vascular wall;
3 – erythrocytes.

LITERATURE:
1. Lecture material.
LESSON № 4

Topic:  
INFLAMMATION. PROLIFERATION AND REGENERATION

Aim of the lesson: to study proliferate and regenerative process in inflammation area, systemic manifestations of inflammation, local inflammation signs, outcomes, categorization and biological role of inflammation.

QUESTIONS:

1. Phagocytosis. Stages:
   • chemotaxis,
   • adherence to bacteria,
   • absorption (phagosome formation),
   • digestion (phagolysosome formation).
2. Pus. Composition.
3. Proliferation and regeneration.
4. Types to regenerations.
5. Categorization of inflammation:
   - on velocities of development (sharp, subsharp, chronic),
   - on dominating stage (alterative, exudative, proliferate),
   - on the exudate type (serous, festering, fibrinous, hemorrhagic and others.).
Local signs of inflammation (- rubor, tumor, - dolor,- color,- functio laesae) and mechanisms of development.
6. General reactions in inflammation (fever, leukocytosis, increase growing of settling of red blood cells and others).
7. Outcomes of inflammation. Factors, influencing the inflammation outcome.
8. Biological role of inflammation.
Fig. 13. Approach of leukocytes to a foreign substance, adhesion

Fig. 14. entrap (absorption), killing and digestion
Fig. 15. Oxygen-dependent mechanisms of leucocytes

Fig. 16. Intravascular cells, connective tissue cells and extravascular matrix in the inflammatory response
Fig. 17. Local signs of inflammation

- Redness (rubor);
- Heat (calor);
- Swelling (tumor);
- Pain (dolor);
- Lack of function (functio laesa)

LABORATORY WORKS

Laboratory work 1. Main clinical symptoms of inflammation on the rabbit ear.

Description of the work: The main clinical symptoms of inflammation occur on the rabbit ear after 5-30 min of ksyslol application. The students watch the redness (rubor), eodema (tumor), heat (calor), painless (dolor) and disfunction (functio laesa) development on the rabbit’s ear, analyze, draw and make conclusions.
Laboratory work 2. *Microscopy of smears with phagocytosis.*

**Description of the work:** 5 ml of 10% peptone solution should be administrated intraperitoneally to rat of peritonitis induction. After 24-48 hr bird’s red blood cells should be administrated intraperitoneally too. 1-3 days later the peritoneal fluid should be taken for smear preparation. The students watch the stages of phagocytosis, draw and make conclusions.

![Diagram of phagocytosis stages](image)

**Fig. 18. The stages of phagocytosis (approach (A), adhesion (Б), Absorption (В), digestion (Г))**

1– Macrophages of guinea pig;
2 – Red blood cells of pegion;
3 – Macrophage lysosomes.
**Fig.19. Components of purulent exudate:**
1 – pyocytes; 
2 – staphylococcus; 
3 – neutrophil; 
4 – red blood cells, 
5 – collagen fibers

**Laboratory work 3. Calculation of white blood cell (WBC) account in the rabbit blood during inflammation.**

**Description of the work:** The inflammation should be induced in a rabbit by ksylol administration. The rabbit blood should be dissolved 20 times by 3% solution of acetic acid. After 5 min of exposition the suspension should be put to Goryaev camera. The students perform calculation of WBC in 100 big non-shade quadrants. The sum of WBC should be added to the formula:
\[ X = \frac{A \times 20 \times 4000}{1600} \times 10^6, \text{ where} \]

\( X \) – WBC amount per 1 liter of the blood,

\( A \) – sum of WBC in 100 big non-shade quadrants,

20 – dissolving degree,

4000 – total level of small quadrants in Goryaev camera.

1600 – sum of small quadrants in 100 big non-shade quadrants.

The students calculate WBC amount in the rabbit blood and make conclusions.

Tests:

1. *The pH in the focus of inflammation:* 
   a) reduces 
   b) increases 
   c) is unaffected

2. *Osmotic pressure in the focus of inflammation is:* 
   a) reduced 
   b) increased 
   c) unaffected
3. Disturbances of microcirculation in the focus of inflammation have following succession:
   a) arterial hyperemia, ischemia, venous hyperemia, stasis
   b) ischemia, arterial hyperemia, venous hyperemia, stasis
   c) venous hyperemia, arterial hyperemia, stasis, ischemia

4. Kinine system in the focus of inflammation causes:
   a) increased arterioles tone
   b) decreased arterioles tone
   c) no changes with arterioles tone

5. Prostaglandin E and prostacyclin in the focus of inflammation causes:
   a) arteriolar constriction
   b) arteriolar dilation
   c) no changes with arterioles tone

6. Vascular permeability under the action of histamine and serotonin in the focus of inflammation is:
   a) increased
   b) reduced
   c) unaffected

7. Vascular permeability under the action of bradykinine in the focus of inflammation is
   a) increased
   b) reduced
   c) unaffected

8. The chemical structure of prostaglandins derived from the metabolism of:
   a) amino acids
   b) peptides
   c) arachidonic acid by cyclooxygenase pathway
d) of arachidonic acid by lipoxygenase pathway

9. The chemical structure of leukotrienes derived from the metabolism of:
   a) amino acids
   b) peptides
   c) arachidonic acid by cyclooxygenase pathway
   d) arachidonic acid by lipoxygenase pathway

10. Diphtheria is characterized by:
   a) serous exudate
   b) catarrhal exudate
   c) fibrinogenous exudate
   d) purulent exudate

11. What substances inhibit scar formation after operation?
   a) heparin
   b) IL-1
   c) γ-interferon
   d) thrombin

12. Emigration of leukocytes occurs in:
   a) arteriole
   b) arteriolar end of capillary
   c) venous end of the capillary
   d) postcapillary venule

13. How does the concentration of C-reactive protein change in inflammation?
   a) it is increased
   b) it is reduced
   c) it is unaffected
14. The most important mediator of the acute-phase reaction (fever) is:
   a) histamine
   b) IL-1
   c) bradykinine
   d) serotonin

15. The cause of inflammation is:
   a) local action of damage factor
   b) apoptosis

16. Inflammation is the process intended to:
   a) damage
   b) destruction of damage factor
   c) restoration of injury

17. What processes occur in inflammation?
   a) alteration
   b) transudation
   c) fibrosis
   d) embolism

18. Emigration of leukocytes is the process of:
   a) leucocytes transit from the intravascular space to the focus of inflammation
   b) leucocytes transit from the tissue to the blood
   c) spreading of leukocytes in blood

19. The causes of primary alteration are:
   a) active forms of oxygen
   b) disturbance of microcirculation
   c) microorganisms
   d) mediators of inflammation
20. **The causes of secondary alteration are the action of:**
   a) active forms of oxygen
   b) disturbance of microcirculation
   c) microorganisms
   d) mediators of inflammation
   e) circulating immune complexes

21. **Local signs of acute inflammation are:**
   a) redness
   b) swelling
   c) pain
   d) heat in the focus of inflammation
   e) leucocytosis

22. **Systemic signs of acute inflammation are:**
   a) redness
   b) swelling
   c) pain
   d) fever
   e) leukocytosis

23. **Physicochemical changes in the focus of alteration are:**
   a) increased anaerobic glycolysis
   b) increased hydrolysis
   c) hyperoncia
   d) hyperosmria
   e) acidosis

24. **Metabolic changes in the focus of alteration are:**
   a) hyperoncia
b) increased anaerobic glycolysis
c) increased hydrolysis
d) hyperosmia
e) activation of peroxidation

25. **Cellular mediators are:**
a) histamine
b) kinins
c) complement
d) serotonine
e) thromboxane

26. **Plasma-derived mediators are:**
a) histamine
b) kinins
c) the complement system
d) serotonine
e) thromboxane

27. **Primary alteration occurs:**
a) under the influence of disturbing factor
b) during inflammatory process itself

28. **Secondary alteration occurs:**
a) under the influence of disturbing factor
b) during inflammatory process itself

29. **Exudation is characterized by:**
a) amount of protein more than 3%
b) amount of protein content less than 3%
c) the content of the cells more than 3000/mm³
d) the content of the cells less than 3000/mm³
30. **Exudation is characterized by:**
   a) specific gravity is above 1018
   b) specific gravity is below 1018
   c) high concentration of hydrogen ions
   d) low concentration of hydrogen ions

31. **Transudation is characterized by:**
   a) amount of protein more than 3%
   b) amount of protein less than 3%
   c) the content of the cells more than 3000/mm\(^3\)
   d) the content of the cells less than 3000/mm\(^3\)

32. **Transudation is characterized by:**
   a) specific gravity is above 1018
   b) specific gravity is below 1018
   c) high concentration of hydrogen ions
   d) low concentration of hydrogen ions

33. **Positive role of exudation consists in:**
   a) decreased concentration of microbes, their toxins and biological active substances
   b) exacerbate of alteration
   c) it causes pain
   d) it prevents the spread of microbes and toxins throughout the organism
   e) worsening of blood supply

34. **Negative role of exudation consists in:**
   a) decreased concentration of microbes, their toxins and biological active substances
   b) exacerbate of alteration
   c) it causes pain
d) it prevents the spread of microbes and toxins throughout the organism  
e) worsening of blood supply

35. **Oxygen-dependent bactericidal system of leucocytes includes:**  
a) lactoferrin  
b) nonenzymatic cation proteins  
c) hypochlorid  
d) superoxide  
e) lysozyme

36. **Oxygen-independent bactericidal system of leucocytes includes:**  
a) lactoferrin  
b) nonenzymatic cation proteins  
c) hypochloride  
d) superoxide

37. **Composition of the pus:**  
a) purulent corpuscles  
b) fibrin  
c) collagen fibers  
d) microorganisms  
e) platelets

38. **Abscess is a suppurative inflammation:**  
a) limited  
b) diffused  
c) in cavities and hollow organs

39. **Phlegmon is a suppurative inflammation:**  
a) limited
b) diffused
c) in cavities and hollow organs

40. **Empyema is a suppurative inflammation:**
   a) limited
   b) diffused
   c) in cavities and hollow organs

41. **What pathogenic factors lead to edema in inflammation?**
   a) the high hydrostatic pressure at the arteriolar end of capillaries
   b) reduced hydrostatic pressure at the arteriolar end of capillaries
   c) decreased vascular permeability
d) increased vascular permeability

42. **What pathogenic factors lead to edema in inflammation?**
   a) decreasing of colloid-osmotic pressure in the focus of inflammation
   b) increasing of oncotic pressure in blood
   c) increasing of colloid-osmotic pressure in the focus of inflammation
d) decreased of the lymph outflow

43. **Acute purulent inflammation is characterized by prevalence in infiltrate of:**
   a) neutrophils
   b) lymphocytes
   c) epithelial cells
d) plasmatic cells
44. **Alterative inflammation is characterized by:**
   a) prevalence of dystrophic, necrotic and necrobiotic processes  
   b) migration of eosinophills in focus of alteration  
   c) accumulation of water in focus of alteration  

45. **Next components take part in phagocytosis:**
   a) mitochondrions  
   b) lysosomes  
   c) ribosomes  
   d) Golgi complex  

46. **Vascular response in inflammation occurs under the influence of:**
   a) increased osmotic pressure in the focus of inflammation  
   b) increased count of leucocytes  
   c) action of mediators  
   d) activation of phagocytosis  

47. **High level of lymphocytes, histiocytes, plasmatic cells, macrophages in the punctate occurs in:**
   a) acute allergic inflammation  
   b) acute exudative inflammation  
   c) chronic inflammation  
   d) aseptic inflammation  

48. **High level of erythrocytes, macrophages, lymphocytes, neutrophills in the punctate occurs in:**
   a) cataral inflammation  
   b) putrid inflammation  
   c) hemorrhagic inflammation  
   d) purulent inflammation
Answers:
1a, 2b, 3b, 4b, 5b, 6a, 7a, 8c, 9d, 10c, 11ac, 12d, 13a, 14b, 15a, 16bc, 17ac, 18a, 19ce, 20abd, 21abcd, 22de, 23cde, 24bce, 25ade, 26bc, 27a, 28b, 29ac, 30ac, 31bd, 32bd, 33ad, 34bce, 35cd, 36ab, 37acd, 38a, 39b, 40c, 41ad, 42cd, 43a, 44a, 45b, 46c, 47c, 48c.

LITERATURE:
1. Lecture material.
LESSON № 5

Topic: THERMAL REGULATORY DYSFUNCTION. FEVER. HYPERTHERMIA. HYPOTHERMIA

Aim of the lesson: to study causes and mechanisms of fever development, its biological role, etiopathogenesis and consequences of hypertermia and hypothermia influence on the organism.

QUESTIONS:

2. Fever categorization.
4. Stage of fevers, their features.
5. Metabolism changes in the organism in fever.
6. Functional Changes in the organism in fever.
7. Types of temperature curves.
8. Biological role of fever.
10. Differentiation hyperthermia from the fever.
Fig. 20. Chronology of events required for the induction of fever

Fig. 21. Mechanisms of exopyrogen action
LABORATORY WORKS

Laboratory work 1. Experimental fever in rats.

Description of the work: The students estimate rats rectal temperature by electric thermometer. Then rats taking pyrogenal (Salmonella typhi endotoxin) 100 Un/100g intramuscularly. Students measure rectal temperature every 15 min during 1,5 hr, make graph of rat rectal temperature curve. Students analyze, draw and make conclusions.

<table>
<thead>
<tr>
<th>Temperature curves</th>
<th>The name of fever</th>
<th>Daily fluctuation °C</th>
</tr>
</thead>
<tbody>
<tr>
<td>f. continua</td>
<td>Day variation</td>
<td>&lt; 1 °C</td>
</tr>
<tr>
<td>f. remittens</td>
<td>Day variation</td>
<td>1.5-2 °C</td>
</tr>
<tr>
<td>f. intermittens</td>
<td>Fever after 1-2  days</td>
<td></td>
</tr>
<tr>
<td>f. hectica</td>
<td>Day variation</td>
<td>&gt; 3 °C</td>
</tr>
<tr>
<td>f. athypica</td>
<td>Unstable changing</td>
<td></td>
</tr>
</tbody>
</table>

Fig. 22. Types of temperature curves
Tests:

1. **The causes of noninfectious fever are:**
   a) aseptic injury of tissues
   b) protein injection
   c) bacteria synthesized products
   d) immune complexes

2. **Exogenous pyrogens are:**
   a) gram-negative bacteria endotoxins
   b) leucocyte synthesized substances
   c) glucose

3. **Endogenous pyrogens are:**
   a) bacterial lipopolysaccharides
   b) bacteria exotoxins
   c) leucocyte synthesized substances (IL-1)

4. **Endopyrogenes are:**
   a) histamin
   b) IL-1
   c) bradykinin
   d) platelet-activating factor, PAF

5. **Exopyrogenes stimulate synthesis of:**
   a) IL-1
   b) thromboxan A₂
   c) prostacyclin
   d) TNF

6. **Endopyrogenes stimulate synthesis of:**
   a) IL-1
   b) thromboxan A₂
c) prostacyclin
d) prostaglandin E₂

7. **What cells are the sources of endogenous pyrogens?**
a) lymphocytes
b) macrophages
c) eosinophills
d) endoteliocytes

8. **Pyrogenic effect due to gram-negative bactéries is determined by:**
a) lipoid A
b) peptides
c) nucleic acids
d) polysaccharides

9. **What mechanisms promote increasing of temperature in the first stage of fever?**
a) muscular quiver
b) increased heat emission
c) reduction of heat emission
d) skin vessels dilation

10. **The mechanisms of increasing temperature in the first stage of fever are:**
a) vasoconstriction
b) reduced perspiration
c) vasodilation
d) increased perspiration

11. **Heat regulation in the first stage of fever in infants is characterized by:**
a) increased heat production
b) reduction of heat emission
c) absence of muscular quiver

12. The mechanisms of temperature decrease at the third stage of fever are:
a) increase of perspiration
b) reduction of perspiration
c) decrease of diuresis
d) increase of diuresis

13. The heat regulation at the third stage of fever:
a) is unaffected
b) is characterized by decreased of heat production
c) remains at higher level
d) is characterized by intensification of heat emission

14. The third stage of fever is characterized by:
a) increased perspiration
b) dilatation of skin blood vessels
c) increased synthesis of aldosterone
d) increased blood volume

15. Temperature homeostasis point in the third stage of fever:
a) does not change
b) stays at a higher heat regulation level
c) returns to starting position

16. What is critical decrease of temperature?
a) rapid reduction of temperature
b) gradual reduction of temperature
17. **What lytic decrease of temperature means?**
a) reduction of temperature in few hours  
b) gradual reduction of temperature  

18. **What type of temperature decrease is more dangerous?**
a) critical  
b) lytic  

19. **How does the function of respiratory system change in fever?**
a) tachypnoe with reduced respiration depth  
b) bradipnoe with increased respiration depth  

20. **How does the secretory and motoric function of gastrointestinal system change in fever?**
a) inhibits  
b) intensifies  

21. **Nitrogen balance in fever is:**
a) negative  
b) positive  

22. **Diuresis in stadium fastigii is:**
a) decreased  
b) increased  

23. **Diuresis in stadium decrementi is:**
a) decreased  
b) increased  

24. **What is the mechanism of collapse during fever?**
a) reduction of cardiac work  
b) vasodilation
25. **In what stage of fever does heat production exceed heat emission?**
   a) st. incrementi  
   b) st. fastigii  
   c) st. decrementi

26. **Subfebrile fever is characterized by:**
   a) rise of temperature to 38 °C  
   b) rise of temperature to 38-39 °C  
   c) rise of temperature to 39-41 °C

27. **Moderate fever is characterized by:**
   a) rise of temperature to 38°C  
   b) rise of temperature to 38-39°C  
   c) rise of temperature to 39-41°C  
   d) rise of temperature to 41-42°C

28. **High fever is characterized by:**
   a) rise of temperature to 38°C  
   b) rise of temperature to 38-39°C  
   c) rise of temperature to 39-41°C  
   d) rise of temperature to 41-42°C

29. **Hyperpyretic fever is characterized by:**
   a) Rise of temperature to 38°C  
   b) Rise of temperature to 38-39°C  
   c) Rise of temperature to 39-41°C  
   d) Rise of temperature to 41-42°C

30. **MPD is:**
   a) maximal pyrogenic dose  
   b) minimal purulentic dose  
   c) minimal pyrogenic dose
31. **Properties of exogenous pyrogens are:**
   a) thermostable
   b) carbohydrate
   c) peptide
   d) thermolabile
   e) lypopolysaccharide

32. **Endogenous pyrogens are characterized by:**
   a) thermostable
   b) carbohydrate
   c) peptide
   d) thermolabile
   e) lypopolysaccharide

33. **Metabolism in the first stage of fever is characterized by:**
   a) decrease of glycogenolysis
   b) decrease of anaerobic glycolysis
   c) increase of glycogenolysis
   d) increase of anaerobic glycolysis

34. **Hyperthermia is the type of heat regulation disturbance characterized by:**
   a) progressing temperature increase
   b) unaffected mechanisms of heat regulation
   c) affected mechanisms of heat regulation

35. **The causes of hyperthermia are:**
   a) insufficient heat inflow in organism
   b) increased heat production
   c) reduced heat production
   d) insufficient emission of heat
Answers:
1abd, 2a, 3c, 4b, 5ad, 6d, 7bd, 8a, 9ac, 10ab, 11ac, 12ad, 13bd, 14ab, 15c, 16a, 17b, 18a, 19a, 20a, 21a, 22a, 23b, 24b, 25b, 26a, 27b, 28c, 29d, 30c, 31ae, 32cd, 33cd, 34ac, 35bd.

LITERATURE:
1. Lecture material.
LESSON № 6

Topic: IMMUNOLOGICAL DISORDERS. CATEGORIZATION. AIDS

Aim of the lesson: to get knowledge on the reactivity and resistance, to study mechanisms of nonspecific reactivity and resistance, implored factors on the human reactivity, etiopathogenesis of immunological disorders and mechanisms of clinical symptoms of AIDS.

QUESTIONS:

1. Reactivity. Types of reactivity (typical, group, individual).
2. Physiological and pathophysiological reactivity.
3. Specific and nonspecific mechanisms of reactivity.
4. Degree of reactivity (normergic, hyperergic and hypergic).
5. Resistance. Local and common resistance, hereditary and acquired.
6. Factors, which determine the reactivity and resistance (heredity, constitution, sex, environmental and social factors).
8. AIDS. Etiology. Clinical symptoms and mechanisms of their development.
Fig. 23. Classical pathway complement activation

Fig. 24. Alternative pathway is activated by microorganisms including bacteria
Fig. 25. Effects of the components complement system action
Fig. 26. Stages of the development of the T- and B-systems of immunity with genetic levels of realization of the genetic defects

Fig. 27. The multiple effects of loss of CD4+ T cells by HIV infection
LABORATORY WORKS

Laboratory work 1. Estimation of vascular resistance by Nesterov test.

Description of the work: The pot of Nesterov apparatus should be applied on the middle one third of internal part of forearm. Experimenter maintains the pressure in the pot about 0.4 atm. during 3 minutes. After
pot removing students calculate amount of petechias (small red points which develop due to hemorrhage) on the forearm. The vascular resistance is estimated in 4 degrees:

0 degree – less than 20 petechias
1 degree – 20-40 petechias
2 degree – more than 40 petechias
3 degree – total hemorrhage bruise

The first two degree (0-1) suggest about normal vascular resistance, but next degree (2-3) show decreasing vascular resistance and increasing permeability of vessels. It can be during different pathological conditions like thrombocytopenia, deficiency of vitamin C, vasculitis and other.

Tests

1. Nonspecific mechanisms of the immune defense:
   a) system of the phagocytic cells
   b) antibodies (Ig A, G, M, E, D)
   c) cytotoxic lymphocytes-killers
   d) complement system

2. Nonspecific mechanisms of the immune defense:
   a) system of the phagocytic cells
   b) antibodies (Ig A, G, M, E, D)
   c) cytotoxic lymphocytes-killers
   d) complement system

3. Immunodeficiency – is the condition of:
   a) insufficiency of the immune system
   b) insufficiency of the all forms of reactivity
   c) antigen overload
4. *During immunodeficiency suffers:*
   a) resistance to infectious
   b) resistance to noninfectious agents
   c) resistance to stressors

5. *Causes of the acquired immunodeficiency states:*
   a) lack of the gene, which is necessary for immunoglobulin synthesis
   b) genetic defect in blood stem cell maturation
   c) poor nutrition
   d) contamination by human immunodeficiency virus

6. *The humoral immunity realizes defense against:*
   a) bacterias
   b) fungies
   c) viruses
   d) protozoas

7. *Cell immunity realizes defense against:*
   e) bacterias
   f) fungies
   g) viruses
   h) protozoas

8. *Signs of insufficiency of the humoral immunity are:*
   a) decreased B-lymphocytes level
   b) presence of antivirus immunity
   c) development of the virus infections
   d) development of the pneumocystosis

9. *Signs of insufficiency of the humoral immunity are:*
   a) presence of antivirus immunity
   b) decreased resistance to fungies
c) tumor development
d) decreased resistance to pneumocystosis

10. **Signs of humoral immunity insufficiency are:**
a) disturbances in the specific antibodies production
b) absence of the lymph nodes reaction
c) decrease of the antitransplantat immunity
d) fungies induced infections

11. **Signs of cellular immunity insufficiency are:**
a) disturbances in specific antibodies production
b) absence of the lymph nodes reaction
c) normal antivirus immunity
d) absence of immune reaction against transplantat

12. **Two signs of cellular immunity insufficiency are:**
a) disturbances in specific antibodies production
b) absence of the lymph nodes reaction
c) normal antivirus immunity
d) tumors development

13. **Syndroms of cellular immunity insufficiency are:**
a) bruton syndrom
b) selective IgA immunodeficite
c) di George syndrom
d) chediak-Chigashi syndrom
e) nezelof syndrom

14. **Syndroms of humoral immunity insufficiency are:**
a) bruton syndrom
b) selective IgA immunodeficite
c) chediak-Chigashi syndrom
d) nezelof syndrom
e) dysgammaglobulinemia

15. **Syndroms of phagocytosis insufficiency are:**
a) bruton syndrom
b) selective IgA immunodeficiency
c) DiGeorge syndrom
d) chediak-Chigashi syndrom
e) nezelof syndrom

16. **Syndroms of complement system insufficiency are:**
a) bruton disease
b) immunocomplex pathology
c) chediak-Chigashi syndrome
d) angioneurotic oedema
e) dysgammaglobulinemia

17. **Which part of the immune system is affected in Di George syndrome?**
a) disturbances in the cellular and humoral immunity
b) disturbances in the humoral immunity
c) disturbances in the cellular immunity
d) phagocytosis disturbances

18. **What mechanism determines disturbances in the organism defence in Bruton syndrome?**
a) disturbances in the cellular immunity
b) disturbances in the humoral immunity
c) disturbances in the cellular and humoral immunity
d) phagocytosis disturbances
19. Which biological fluids can contaminate organism by AIDS?
   a) blood
   b) sperm
   c) sweat
   d) breast milk
   e) saliva

20. What are the target cells for human immunodeficiency virus?
   a) neurons
   b) macrophages
   c) t-killers
   d) t-helpers
   e) t-suppressors

21. What signs present at AIDS?
   a) Kaposi sarcoma
   b) memory decreasing
   c) icheterus
   d) lymphoma
   e) pneumocystic pneumonia

**Answers:**
1ad, 2bc, 3ac, 4ab, 5cd, 6a, 7bcd, 8ab, 9a, 10ab, 11bd, 12bd, 13ce, 14abe, 15d, 16bd, 17a, 18b, 19abd, 20abd, 21abde.

**LITERATURE:**
LESSON № 7

Topic: ALLERGY

Aim of lesson: to study causes and mechanisms of allergic reactions of different types, to take opinion of the sensibilization and desensibilization.

STUDY QUESTIONS:

1. Allergy. Causes.
2. Allergens, their categorization and description.
3. Types of allergic reactions.
4. Pathogenesis of allergic reactions of different types.
5. Immunological stage.
7. Pathophysiological stage.
9. Description of allergic disorders (allergic rhinitis (pollinosis), bronchial asthma, e.g.)
10. Desensibilization (hyposensibilization).
**Fig. 29. Normal mast cell and mast cell after degranulation**

<table>
<thead>
<tr>
<th>Effects of histamine</th>
</tr>
</thead>
<tbody>
<tr>
<td>✷ Dilation of arterial vessels</td>
</tr>
<tr>
<td>✷ Bronchoconstriction</td>
</tr>
<tr>
<td>✷ Hypersecretion of mucous</td>
</tr>
<tr>
<td>✷ Increased musculare tone in gastrointestinal tract</td>
</tr>
<tr>
<td>✷ Stimulation of gastric secretion</td>
</tr>
<tr>
<td>✷ Release of catecholamines from adrenals</td>
</tr>
<tr>
<td>✷ Irritation of pain receptors</td>
</tr>
<tr>
<td>✷ Vasodilation, tachycardia, headache</td>
</tr>
</tbody>
</table>

**Fig. 30. Pathophysiological effects of histamine during allergy**
Type I of hypersensitivity
Anaphylactic type

Type II hypersensitivity
Cytotoxic type

Type III of hypersensitivity
(immune complex)

IV type of hypersensitivity
Cell-mediated type

Fig. 31. Types of hypersensitivity (Gell, Coombs, 1968)
LABORATORY WORKS

Laboratory work 1. Anaphylactic reactions on the frog peritoneum.

Description of the work: 1. Sensibilization. The horse serum 0.3 ml was administrated in frog lymphatic sack twice with 2-3 day interval. 2. Sensibilizated frog is fixated on the laboratory table. The peritoneum should be isolated. Students analyze normal blood flow in the peritoneal vessels. Then experimenter adds 1-2 drops of horse serum and students watch the changes in the blood circulation in peritoneum (vasodilatation, emigration of WBC, red blood cells (RBC) aggregation and other.

Students analyze, draw and make conclusions.

Fig. 32. Technique of subcutaneous injections
Fig. 33. Technique of intraperitoneal injection

**Laboratory work 2.** The allergic slide-show

**Tests:**

1. *The first stage of allergy is:*
   
   a) pathochemical  
   b) immunological  
   c) pathophysiological

2. *The second stage of allergy is:*
   
   a) pathochemical  
   b) immunological  
   c) pathophysiological
3. *The third stage of allergy is:*
   a) pathochemical  
   b) immunological  
   c) pathophysiological

4. *The main component of the immune system in allergy reaction I type is:*
   a) immunoglobulin E  
   b) immunoglobulins M, G  
   c) sensibilized T-lymphocyte

5. *The main component of the immune system in allergy reaction II type (cytotoxic) is:*
   a) immunoglobulin E  
   b) immunoglobulins M, G  
   c) sensibilized T-lymphocytes  
   d) complement

6. *The main component of the immune system in allergy reaction III type is:*
   a) immunoglobulin E  
   b) immunoglobulins M, G  
   c) sensibilized T-lymphocytes  
   d) complement  
   e) phagocytes

7. *The main component of the immune system in allergy reaction IV type is:*
   a) immunoglobulin E  
   b) immunoglobulin M  
   c) sensibilized T-lymphocytes  
   d) complement
8. *What cells have receptors with high affinity to Ig E?*
   a) macrophages
   b) eosinophils
   c) mast cells
   d) platelets

9. *Primary mediators of allergic reaction 1 type are:*
   a) noradrenaline
   b) histamine
   c) thromboxane
   d) prostacycline
   e) serotonin

10. *Secondary mediators of allergic reaction 1 type are:*
    a) noradrenaline
    b) histamine
    c) heparin
    d) neutrophylls chemotaxic factor
    e) leucotriene D

11. *What cells can release factors for inactivation of allergic mediators?*
    a) neutrophils
    b) lymphocytes
    c) eosinophils
    d) macrophages

12. *The role of eosinophils in allergy is:*
    a) activation of histamine synthesis
    b) activation of histamine excretion
    c) intensification of histamine biological action
    d) inactivation of histamine
13. Anaphylactic shock is the allergy of:
   a) type I
   b) type II
   c) type III
   d) type IV

14. Anaphylactic shock leads to:
   a) pneumonia
   b) lung oedema
   c) bronchoconstriction
   d) lung atelectasis

15. How does the tonus of arterioles change in anaphylactic shock?
   a) it is raised
   b) it is reduced
   c) it is unaffected

16. The examples of the I type of allergic reaction are:
   a) hives
   b) autoimmune anemia
   c) autoimmune glomerulonephritis
   d) reaction against transplant

17. The examples of the II type of allergic reaction are:
   a) hives
   b) autoimmune anemia
   c) autoimmune glomerulonephritis
   d) reaction against transplant

18. The examples of the III type of allergic reaction are:
   a) hives
   b) autoimmune anemia
c) autoimmune glomerulonephritis
d) reaction against transplant

19. The examples of the IV type of allergic reaction are:
a) hives  
b) autoimmune anemia  
c) autoimmune glomerulonephritis  
d) reaction against transplant

20. What mediator of allergy leads to bronchospasm?
a) heparin  
b) chymase  
c) leukotrien C  
d) triptase

21. The release of histamine occurs in:
a) immunological stage of allergy  
b) biochemical stage of allergy  
c) pathophysiological stage of allergy

22. What type of allergy is the leader in non-infectious bronchial asthma?
a) I  
b) II  
c) III  
d) IV

23. What type of allergy is the leader in angioneurotic edema?
a) I  
b) II  
c) III  
d) IV
24. What type of allergy is the leader in allergic rhinitis?
   a) I  
   b) II  
   c) III  
   d) IV

25. What type of allergy is the hives (urticaria)?
   a) I  
   b) II  
   c) III  
   d) IV

26. What type of allergic reaction is complement-dependent?
   a) I  
   b) II  
   c) III  
   d) IV

27. What type of allergic reaction is dependent on antibodies and is caused by immune complexes?
   a) I  
   b) II  
   c) III  
   d) IV

28. What elements of the blood prevent adjournment of immune complexes in a vascular wall?
   a) neutrophils  
   b) eosinophils  
   c) erythrocytes  
   d) basophils
29. What cells phagocyted immune complexes?
   a) neutrophils
   b) eosinophils
   c) macrophages
   d) basophils

30. What type of allergy is the leader in serum sickness?
   a) I
   b) II
   c) III
   d) IV

31. What type of allergy is Arstus-Sacharov phenomeon?
   a) I
   b) II
   c) III
   d) IV

32. What cells mediate the IV type of allergy?
   a) t-lymphocytes-killers
   b) t- lymphocytes-suppressors
   c) b-lymphocyte
   d) nk- cells

33. The cause of pollinosis is:
   a) pollen of plants
   b) house dust
   c) street dust
   d) library dust
   e) wool of animals
Ansewss:
1b, 2a, 3c, 4a, 5bd, 6bde, 7c, 8c, 9be, 10e, 11c, 12d, 13a,
14c, 15b, 16a, 17b, 18c, 19d, 20c, 21b, 22ac, 23a, 24a, 25a,
26b, 27c, 28b, 29c, 30c, 31c, 32a, 33a.

LITERATURE:
1. Lecture material.
LESSON № 8

Topic: DISORDERS OF CARBOHYDRATE METABOLISM

Aim of the lesson: to study disorders of carbohydrate metabolism, mechanisms of diabetes mellitus symptoms development and its complications.

STUDY QUESTIONS:

7. Clinical symptoms of diabetes mellitus and mechanisms of there development.
Fig. 34. Glucose intake enhances glycogen storage

Fig. 35. Pancreatic Islets hormones
Fig. 36. Effect of insulin on target cells

Fig. 37. Metabolism in diabetes mellitus
Fig. 38. Consequences of hyperglycemic states

Fig. 39. Oral glucose tolerance curves
Fig. 40. Pathogenesis of type II diabetes mellitus
Laboratory work 1. Modulation of hypoglycemic coma and it

**Fig. 41. Angiopathias in diabetes mellitus**

**Laboratory work 1. Experimental therapy of the diabetes mellitus on the mouse.**

**Description of the work:** Insulin (10 U/kg) is administrated subcutaneously for two 1day-starved white mice. Students watch the changing in the animal behavior.
After convulses appearance the 20% solution of glucose should be injected intraperitoneously to 1 mouse (0,5 ml/10g). Students continue to observe. Students analyze, draw and make conclusions.

Tests

1. **The most difficult consequences for organism are:**
   a) carbohydrate starvation
   b) proteinic starvation
   c) fat starvation
   d) vitamin starvation

2. **Which carbohydrate absorbs in the intestine?**
   a) mucopolysaccharide
   b) fructose
   c) glycogen
   d) starch

3. **Which enzyme hereditary deficiency leads to von Gierke disease?**
   a) hexokinase
   b) glucose-6-phosphatase
   c) acid α-1,4-glucosidase
   d) amylo-1,6-glucosidase

4. **In children very often occurs deficiency of:**
   a) maltase
   b) amylase
   c) isomaltase
   d) lactase
   e) saccharase
5. In what diabetes is high risk of ketoacidosis development?
   a) type 1
   b) type 2

6. Insulin stimulates:
   a) synthesis of lipid
   b) lipolysis
   c) synthesis of ketone bodies
   d) synthesis of cholesterol
   e) synthesis of lactic acid

7. Insulin inhibits:
   a) synthesis of lipids
   b) lipolysis
   c) synthesis of ketone bodies
   d) synthesis of cholesterol

8. Consequences of carbohydrate starvation is increasing in blood level of:
   a) acetone oxaloacetic acid
   b) pyruvic acid
   c) glucose

9. Reasons of increased level of glucosae in blood after its intake are:
   a) suction in intestine
   b) increased glycogenolysis
   c) activation of gluconeogenesis

10. The causes of hyperglycemia are the increased blood level of:
    a) insulin
b) glucagon

c) glucocorticoid

d) adrenaline

11. The causes of hyperglycemia are the decreased blood level of:
   a) glucagon
   b) glucocorticoid
   c) adrenaline
   d) insulin

12. Reasons of increased sensitivity of nerve cells to deficiency of glucosae are:
   a) absence of reserve of glicogen
   b) insulin-dependent tissue
   c) impossibility usage of fatty acids as source of energy

13. The causes of diabetes mellitus type 2 are:
   a) insufficient synthesis of proinsulin
   b) raised catabolism of insulin
   c) lack of receptors to insulin on target organs

14. Clinical onset of mild hypoglycemia is:
   a) disturbances coordination of movements
   b) convulsions
   c) movement exitement
   d) rise of appetite

15. Risk factors of diabetes mellitus are overconsumption of:
   a) sugar
   b) alcohol
   c) proteins
16. **Hypoglycemic coma can occur in:**
   a) thyrotoxicosis
   b) deficiency in insulin
   c) insuloma

17. **Diabetes mellitus is characterized by the increased blood level of:**
   a) glucose
   b) maltose
   c) saccharose
   d) urea

18. **Deficiency of what enzyme leads to disturbance digestion of carbohydrate in the intestine?**
   a) hexokinase
   b) alkaline phosphatase
   c) amylase
   d) phospholipase

19. **Clinical onset of ketoacidic coma expresses in:**
   a) soft eye-bulbes
   b) muscular trembling
   c) hunger
   d) aceton-like smell

20. **Reactions of intermediary carbohydrate metabolism are:**
   a) transamination
   b) gluconeogenesis
   c) β-oxidation
   d) anaerobic glycolysis
21. Clinical onset of lactacidic coma expresses in:
   a) muscular trembling
   b) hunger
   c) aceton-like smell
   d) decrease of blood pH

22. Causes of hyperglycemia in diabetes mellitus are:
   a) inadequate synthesis of glycogen
   b) increased synthesis of glycogen
   c) disorders in utilization of glucosae by cells
   d) increased suction of glucosae in the intestine

23. Normal level of glucosae in blood is:
   a) 2,2-3,3 mM
   b) 3,3-5,5 mM
   c) 5,1-6,4 mM
   d) 2,7-3,6 mM

24. Easily absorbted carbohydrate is:
   a) mucopolysaccharide
   b) glucose
   c) glycogen
   d) starch

25. Causes of intermediary carbohydrate metabolism disorder are:
   a) lack of insulin
   b) hypoxia
   c) deficiency in bile
   d) hypercholesterolemia
26. **Reason of glucose malabsorption is hereditary deficiency of:**
   a) hexokinase
   b) glucose-6-phosphatase
   c) acid α-1,4-glucosidase
   d) amylo-1,6-glucosidase

27. **Causes of Pompe’s disease are hereditary deficiency of:**
   a) hexokinase
   b) glucose 6-phosphatase
   c) acid α-1,4-glucosidase
   d) amylo-1,6-glucosidase

28. **Insulin stimulates:**
   a) aerobic glycolysis
   b) proteolysis
   c) krebs cycle
   d) gluconeogenesis

29. **What kind of coma in diabetes mellitus is characterized by very high level of glucooe?**
   a) ketoacidotic
   b) hyperosmolarity
   c) lactatic
   d) hypoglycemic

30. **Insulin inhibits:**
   a) aerobic glycolysis
   b) lipolysis
   c) gluconeogenesis
   d) glycogenolysis
31. Deficiency of insulin is characterized by increased blood level of:
   a) glucose
   b) phospholipids
   c) ketone bodies
   d) urea

32. Gierke disease is characterized by accumulation of:
   a) sphingomyelin
   b) triglyceride
   c) proteins
   d) glycogen

33. The kidney barier for glucosae is the level of glucosae:
   a) in blood in which glucosae appear in primary urine
   b) in primary urine, which enters to secondary urine
   c) in blood, in which glucose can’t be fully absorbed

34. Hypoglycemia is caused by increased level of:
   a) insulin
   b) glucagon
   c) glycocorticoid
   d) adrenalin

35. The causes of diabetes mellitus type 1 are:
   a) insuffitient synthesis of proinsulin
   b) heightened catabolism of insulin
   c) lack of receptors to insulin on target tissues

36. Insulin promotes glucose uptake by:
   a) fatty tissue
   b) liver
   c) blood cells
d) brain

37. The clinical features of severe hypoglycemic coma are:
   a) disorder of coordination of movement
   b) convulsions
   c) acetone-like smell
   d) loss of consciousness

38. The clinical onset of diabetes mellitus type 1 expresses in:
   a) thirst
   b) oliguria
   c) weight loss
   d) obesity

39. Acetone-like smell is the clinical feature of:
   a) hypoglycemic coma
   b) renal diabetes
   c) ketoacidosis
   d) hyperosmolar coma

40. The complications of diabetes mellitus are:
   a) hypoglycemic coma
   b) hyperosmolar coma
   c) nephropathy
   d) gangrene

41. The complications of diabetes mellitus are:
   a) anemia
   b) retinopathy
   c) thirst
   d) hyperglycemia
42. **In diabetes mellitus urine analysis detects:**
   a) erythrocytes
   b) bilirubin
   c) glucose
   d) ketone bodies

43. **Carbohydrate starvation leads to:**
   a) increased synthesis of ketone bodies
   b) weight loss
   c) increase synthesis of proteins
   d) no consequences

44. **Diabetes mellitus leads to disorder:**
   a) only of protein metabolism
   b) only of lipid metabolism
   c) of all metabolism kinds
   d) only of carbohydrate metabolism
   e) only of water-salt metabolism

45. **The causes of intermediate carbohydrate metabolism disorder are:**
   a) B\textsubscript{1} hypovitaminosis
   b) hypoxia
   c) deficiency in bile
   d) hypercholesterolemia

46. **Clinical manifestations of hypoglycemic coma are:**
   a) acetone in urine
   b) kussmaul’s respiration
   c) soft eye-bulbes
   d) decreased level of glucose in blood
Answers:

1bd, 2b, 3b, 4d, 5a, 6a, 7bcd, 8ac, 9a, 10bcd, 11d, 12ac, 13bc, 14cd, 15ab, 16c, 17ad, 18c, 19ad, 20bd, 21d, 22ac, 23b, 24b, 25ab, 26a, 27c, 28ac, 29b, 30bcd, 31acd, 32d, 33c, 34a, 35a, 36ab, 37bd, 38ac, 39c, 40bcd, 41b, 42cd, 43ab, 44c, 45ab, 46d.

LITERATURE:

1. Lecture material.
LESSON № 9

Topic: DISORDERS OF LIPID METABOLISM

Aim of the lesson: to study disorders of lipid metabolism, mechanisms of hyperlipidemia, obesity and atherosclerosis development and its complications.

QUESTIONS:

1. Biological role of lipids in the human body.
3. Causes and consequences of lipid intermediary metabolism.
4. Pulmonary and hepatic role in lipid metabolism.
Laboratory work 1. Calculation of the body weight index.

**Description of the work.** After weight and growth estimation students calculate their body weight indexes according to the formula:

\[
BWI = \frac{BW(kg)}{H^2(m^2)}, \text{where}
\]

BWI - body weight index;
BW - body weight, kg
H – height, m
Students analyze the results and make conclusions.
Table 1. Analysis of body weight index

<table>
<thead>
<tr>
<th>№</th>
<th>Result</th>
<th>Conclusion</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&lt; 18,5</td>
<td>Deficiency of BW</td>
<td>Increased food intake, consultation of endocrinologist</td>
</tr>
<tr>
<td>2</td>
<td>18,5-24,9</td>
<td>Normal BW</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>25,0-29,9</td>
<td>Excess of BW (1° of obesity)</td>
<td>Diet, physical exercises</td>
</tr>
<tr>
<td>4</td>
<td>30,0-39,9</td>
<td>2° of obesity</td>
<td>Diet, consultation of endocrinologist</td>
</tr>
<tr>
<td>5</td>
<td>&gt; 40</td>
<td>3° of obesity</td>
<td>Diet, consultation of endocrinologist</td>
</tr>
</tbody>
</table>

Fig. 43. Metabolism and transport of lipoproteins
Fig. 22-1  The composition of plasma lipoproteins. VLDL = very-low-density lipoprotein; LDL = low-density lipoprotein; HDL = high-density lipoprotein.

Fig. 44.  The composition of plasma lipoproteins

Fig. 45.  Vessel wall at atherosclerosis
Fig. 46. The major complications of obesity

Tests

1. *Unsaturated fatty acid is also called vitamin:*  
a) F  
b) E  
c) U  
d) A  
e) B
2. **Leptin is hormone of:**
   a) hypothalamus
   b) hypophysis
   c) gastrointestinal tract
   d) adipocytes

3. **Emulsification of lipids is:**
   a) splitting of triglyceride into glycerine and fatty acid
   b) dividing of big lipid drops to small by bile acids
   c) synthesis of chylomicrons

4. **What factors lead to development of cholelithiasis?**
   a) lack of low density lipoproteins (LDL)
   b) lack of high density lipoproteins (HDL)
   c) deficiency of apoprotein A
   d) excess of apoprotein C-II

5. **What factors lead to development of obesity?**
   a) inhibition of sympathetic nervous system
   b) activation of sympathetic nervous system
   c) increased level of insulin
   d) increased level of glucocorticoids

6. **Obesity leads to:**
   a) elevated blood pressure
   b) decreased blood pressure
   c) increased blood coagulation
   d) reduction of blood coagulation

7. **What lipoproteins carry cholesterol to the cells?**
   a) chylomicrons
   b) LDL
   c) HDL
8. What substances are necessary for digestion of triglycerides in the intestine?
   a) hydrochloric acid  
   b) amylasa  
   c) apoprotein C  
   d) bile

9. Main transport form of exogenous fat is:
   a) LDL  
   b) VLDL  
   c) HDL  
   d) chylomicrons

10. The consequences of lipid starvation are:
    a) absent  
    b) increased proteolysis  
    c) deficiency of all vitamins  
    d) deficiency of fatsoluble vitamins

11. Unsaturated fatty acids are:
    a) butyric acid  
    b) palmitinic acid  
    c) stearic acid  
    d) arachidonic acid

12. The atherogenic lipoproteins are:
    a) HDL  
    b) LDL  
    c) IDL  
    d) chylomicrons
13. Antiatherogenic properties of HDL are determined by:
   a) acetilcholesterolaciltransferase
   b) endothelial lipoprotein lipase
   c) hepatic lipoprotein lipase
   d) ornithincarbamamialtransferase

14. Lipopectic function in lungs is realized by:
   a) acetilcholesterolaciltransferase
   b) endothelial lipoprotein lipase
   c) hepatic lipoprotein lipase
   d) lipokain

15. Disorder of lipid metabolism leads to:
   a) ischaemic heart disease
   b) diabetes insipidus
   c) kwashiorkor
   d) stroke

16. The causes of hyperchylomicronemia are:
   a) the absence of receptors to apoprotein B
   b) lack of lipoprotein lipase
   c) deficiency in apoprotein C-II
   d) synthesis of abnormal VLDL

17. Arachidonic acid is the precursor of:
   a) leukotrienes
   b) prostaglandins
   c) bradykinins
   d) thromboxane A₂

18. Triglycerides are digested in the intestin to:
   a) valine
   b) chylomicrons
c) fatty acids
d) ketone bodies

19. Lipotropic factors are:
a) methionine
b) lipokain
c) leptin
d) phospholipids

20. Deficiency of unsaturated fatty acids leads to:
a) disturbance of spermatogenesis
b) dermatitis
c) obesity
d) fatty degeneration of liver

21. Disturbances in lipid digestion and absorption are caused by deficiency of:
a) lactase
b) leptine
c) lipase
d) aminopeptidase

22. Increased level of ketone bodies is not specific for:
a) hypoglycemic coma
b) ketoacidic coma
c) hyperosmolar coma
d) lactacidic coma

23. What factors prevent obesity?
a) increased level of androgens hormones
b) increased level of thyroxine
c) activation sympathetic nervous system
d) inhibition sympathetic nervous system
24. **Biliary obstruction leads to:**
   a) hyperchylomicronemia
   b) steatorrhea
   c) polyuria
   d) deficiency of vit D

25. **The lipid mobilization hormones are:**
   a) insuline
   b) thyroxine
   c) aldosterone
   d) oxytocin

26. **Saturated fatty acids are:**
   a) docosahexaenoic
   b) arachic
   c) palmitic
   d) arachidonic

27. **Reaction of intermediary lipid metabolism is:**
   a) transamination
   b) β-oxidation
   c) decarboxylation
   d) synthesis of ketone bodies

28. **Normal cholesterol level in blood is:**
   a) 3,9-6,2 mM
   b) 2,2-3,8 mM
   c) 2,8-4,7 mM
   d) 6,3-9,5 mM

29. **What substances are needed for digestion of triglycerides in the intestine?**
   a) hydrochloric acid
b) amylase
c) lipase
d) bile

30. **Antiatherogenic lipoproteins are:**
   a) HDL
   b) LDL
   c) chylomicrones
   d) VLDL

31. **Transport form of endogenous fat is:**
   a) HDL
   b) LDL
   c) chylomicrones
   d) VLDL

32. **Cholesterol mobilization from tissue realizes by:**
   a) LDL
   b) chylomicrones
   c) HDL
   d) VLDL

33. **What factors are promoted formation of stones during holelithiasis?**
   a) increased level of cholesterol in bile
   b) increased level of bile acids in bile
   c) decreased level of bile acids in bile
   d) increased etherification of cholesterol

34. **What activate endothelial lipoproteine lipase?**
   a) apoprotheine C-I
   b) apoprotheine C-II
   c) apoprotheine C-III
d) apoprotheine A-I

35. **The reasons of dys-β «floating» hyperlipoproteinemia are:**
   a) absence of receptors to apoprothein B
   b) lack of lipoprotheine lipase
   c) deficit of apoprotheine C-II
   d) synthesis of abnormal VLDL

36. **The amount of adipocytes after puberty is:**
   a) increased
   b) decreased
   c) don’t change

37. **The causes of adipose infiltration of liver are lack of:**
   a) tryptophan
   b) arginine
   c) methionine
   d) valine

38. **Lack of what substances lead to fatty degeneration of liver?**
   a) histidine
   b) glutamine
   c) lipokaine
   d) taurine

39. **What factors lead to atherosclerosis?**
   a) increased cholesterol level in blood
   b) obesity
   c) damage of endothelium
   d) increased synthesis of prostacycline
40. What factors lead to atherosclerosis?
a) decreased synthesis of nitric oxide (NO) in endothelium
b) increased level of chylomicrones in blood
c) increased level of cholesterol in blood
d) increased synthesis of NO in endothelium

41. What blood cells take part in pathogenesis of atherosclerosis?
a) basophils
b) eosinophils
c) monocytes

42. What vascular cells turn into foam cells during atherosclerosis?
a) endothelials
b) smooth muscle
c) fibroblasts
d) macrophages

Ansews:
1a, 2d, 3b, 4bc, 5acd, 6ac, 7b, 8d, 9d, 10d, 11d, 12b, 13a, 14b, 15ad, 16bc, 17abd, 18c, 19abd, 20abd, 21c, 22acd, 23abc, 24bd, 25b, 26bc, 27bd, 28a, 29cd, 30a, 31d, 32c, 33ac, 34b, 35d, 36c, 37c, 38c, 39abc, 40ac, 41c, 42bcd.

LITERATURE:
1. Lecture material.
LESSON № 10

Topic: DISORDERS OF PROTEIN METABOLISM. GOUT

**Aim of the lesson:** to study disorders of protein metabolism, mechanisms of protein insufficiency and gout development and their complications.

**QUESTIONS:**

5. Causes and consequences of intermediary amino acid metabolism.
6. 
8. Types of residual plasma nitrogen level increasing. Mechanisms of their development.
Fig. 47. Digestion of proteins
Fig. 48. Protein synthesis
Deamination

Transamination

Decarboxilation

Fig. 49.
Tests

1. Disorder of tyrosine metabolism leads to:
   a) tyrosinosis
   b) alkaptonuria
   c) albinism
   d) phenylketonuria

2. The causes of positive nitrogen balance are:
   a) growth
   b) pregnancy
   c) fever
   d) burn

3. What substances are inhibitory transmitters?
   a) aspartate
   b) glutamate
   c) glycine
   d) Gamma-aminobutyric acid (GABA)

4. What substances are excitatory neurotransmitters?
   a) aspartate
   b) glutamate
   c) glycine
   d) gamma-aminobutiric acid (GABA)

5. Increased basal metabolic rate is the result of increased level of:
   a) adrenocorticotropic hormone (ACTH)
   b) insuline
   c) parathormone
   d) thyroxine
6. **Which protein fraction prevails in blood?**
   a) \( \alpha_1 \)-globuline
   b) \( \beta \)-globuline
   c) albumen
   d) \( \alpha_2 \)-globuline

7. **Which protein fraction acts as an antibody?**
   a) \( \alpha_1 \)-globuline
   b) \( \gamma \)-globulins
   c) \( \alpha_2 \)-globuline

8. **Phenilketonuria is characterized by increased synthesis of:**
   a) homogentisic acid
   b) dioxyphenylalanine
   c) melanin
   d) phenylpyruvic acid

9. **The cause of positive nitrogen balance is the increased synthesis of:**
   a) somatotropin hormone
   b) insuline
   c) glucocorticoid
   d) thyroxine

10. **The causes of positive nitrogen balance are:**
    a) lack of androgens
    b) lack of insuline
    c) pregnancy
    d) childhood

11. **Positive nitrogen balance can be observed in:**
    a) fever
b) starvation

c) recovery
d) hyperthyreosis

12. The reason of lipokaine synthesis deficiency is deficit of:

a) histidine
b) glutamin
c) methionine
d) taurine

13. The precursors of catecholamines are:

a) arginine
b) phenylalanine
c) tyrosine
d) lysine

14. The precursor of nitric oxide:

a) asparagine
b) glutamine
c) L-arginine
d) alanin

15. The types of hyperazotemia are:

a) lactacidic
b) ketoacidic
c) hypochloremic
d) hyperglycemic

16. What process leads to synthesis of new aminoacids?

a) transamination
b) deamination
c) decarboxylation
17. What enzymes digest the proteins in stomach?
   a) pepsin  
   b) chemotrypsin  
   c) rennin  
   d) phospholipase

18. Celiac disease is a disease which is characterized by deficiency in digestion of:
   a) meat proteins  
   b) lipids  
   c) saccharose  
   d) protein from cereals

19. The result of deamination is synthesis of:
   a) ammonia  
   b) uric acid  
   c) histamine  
   d) urea

20. Lack of vit B₆ leads to disorder of:
   a) deamination  
   b) decarboxylation  
   c) transamination

21. Decreased nonessential amino acid synthesis is caused by:
   a) disorder of deamination  
   b) disorder of decarboxylation  
   c) disorder of transamination

22. The components of the residual nitrogen are:
   a) proteins  
   b) peptides
c) amino acids  
d) ammonia

23. **The types of hyperazotemia are:**  
a) retentive  
b) hypochloremic  
c) hyperchloremic  
d) hypoammonia

24. **The causes of retentive azotemia are:**  
a) nephropathy  
b) shock  
c) thrombosis of venae porta  
d) fever

25. **What type of hyperazotemia leads to disorder in urea synthesis?**  
a) retentive  
b) hypochloremic  
c) hyperammonia

26. **The cause of gout is breakup of:**  
a) guanine  
b) uracil  
c) thymidine  
d) adenine

27. **Hyperammaniemia is the result of decreasing action of:**  
a) ornithine-carbomailtransferase  
b) phosphodiesterase  
c) acetylcholinesterase  
d) carbonic anhydrase
28. **Final product of breakup of protein is:**
   a) lactic acid  
   b) glutamic acid  
   c) ammonia  
   d) pyruvic acid

29. **What biologically active substances are synthesized in decarboxylation?**
   a) Thyroid hormones  
   b) melanin  
   c) histamin  
   d) serotonin

30. **Phenylketonuria is the disease which is characterized by metabolism disorder of:**
   a) tyrosine  
   b) phenylalanine  
   c) homogentisic acid

31. **What enzymes digest proteins in the intestine?**
   a) pepsin  
   b) chemotrypsin  
   c) renin  
   d) dipeptidase

32. **Abnormality proteins are:**
   a) albumens  
   b) γ-globulins  
   c) cryoglobulins  
   d) α-globulins

33. **The type of hyperazotemia is:**
   a) hyperosmolaric
b) ketoacidic

c) retentive

d) hypoglycemic

34. **Reactions of intermediary amino acid’s metabolism are:**
   a) transamination
   b) deamination
   c) gluconeogenesis
   d) β-oxydation

35. **The reasons of increasing deamination are:**
   a) starvation
   b) increased consumption of amino acids
   c) lack of vit PP
   d) lack of vit B₆

36. **Consequences of decreased deamination are:**
   a) decreased synthesis of proteins
   b) decreased synthesis of nonessential amino acids
   c) increased level of ammonia

37. **What reaction leads to synthesis of histamine?**
   a) deamination
   b) decarboxylation
   c) transamination

38. **The main component of residual plasma nitrogen level is/are:**
   a) amino acids
   b) ammonia
   c) ammonia ions
   d) urea
39. **Predisposing factors for gout are:**
   a) meat
   b) male sex
   c) female sex
   d) acidosis

40. **The productive hyperazothemia is characterized by increased plasma level of:**
   a) ammonia
   b) protein
   c) amino acids
   d) creatinine

41. **Gout is the disease which is characterized by disorder in metabolism of:**
   a) protamines
   b) histone
   c) urea
   d) purine bases

42. **Gout is the disease which is characterized by involvement of:**
   a) bones
   b) cartilages
   c) rens
   d) cor

43. **The causes of negative nitrogen balance are:**
   a) obesity
   b) emaciation
   c) excess of insuline
   d) hemorrhage
44. Most common reasons of hereditary enzymopathies are disorders in metabolism of:

a) valine
b) phenylalanine
c) isoleucine
d) leucine

Answers:

1abc, 2ab, 3cd, 4ab, 5d, 6c, 7b, 8d, 9ab, 10ab, 11c, 12c, 13bc, 14c, 15c, 16a, 17ac, 18d, 19a, 20bc, 21ac, 22bcd, 23ab, 24ab, 25c, 26ad, 27a, 28c, 29cd, 30b, 31bd, 32c, 33c, 34ab, 35ab, 36ab, 37b, 38d, 39abd, 40c, 41d, 42bc, 43bd, 44b.
FASTING

1. In the 1st period of complete fasting respiratory coefficient is?
   a) 0,7
   b) 0,8
   c) 1,0

2. In the 2nd period of complete fasting respiratory coefficient is:
   a) 0,7
   b) 0,8
   c) 1,0

3. In the 3rd period of complete fasting respiratory coefficient is:
   a) 0,7
   b) 0,8
   c) 1,0

4. The first period of complete fasting is characterized by disturbances of:
   a) proteins
   b) glicogene
   c) triglycerides

5. The second period of complete fasting is characterized by disturbances of:
   a) proteins
   b) starch
   c) glicogene
   d) triglycerides
   e) nucleoproteins
6. **What period of complete fasting is most prolonged?**
   a) the first
   b) the second
   c) the third

7. **Which two organs (tissue) lose mass less than others in complete fasting?**
   a) liver
   b) cor
   c) fatty tissue
   d) muscles
   e) nervous tissue

8. **What organ (tissue) loses mass more than others in complete fasting?**
   a) liver
   b) cor
   c) lung
   d) fatty tissue
   e) nervous tissue

9. **What nutrients are not used by an organism in the first period of the full fasting?**
   a) proteins
   b) lipids
   c) carbohydrates

10. **The 2nd period of complete fasting is characterized by disturbance of:**
    a) proteins
    b) lipids
    c) carbohydrates
11. The 3\textsuperscript{rd} period of complete fasting is characterized by disturbance of:
   a) proteins
   b) lipids
   c) carbohydrates

12. Basal metabolism in the 1\textsuperscript{st} period of complete fasting:
   a) rises
   b) reduces
   c) doesn’t change

13. Basal metabolism in the 2\textsuperscript{nd} period of complete fasting:
   a) rises
   b) reduces
   c) doesn’t change

14. Glycogen reserve at fasting:
   a) increases
   b) doesn’t change
   c) decreases

15. The cause of increased synthesis of ketone bodies in fasting is:
   a) Disorder of mineral metabolism
   b) increased splitting of lipids
   c) Disorder in function of ren
   d) Deficit of substrates of Krebs cycle

16. In what diseases fast is indicated:
   a) allergy
   b) obesity
   c) cachexia
d) neuropsychic disease

17. What tissues lose mass more than others in fasting?
   a) fatty tissue
   b) muscles
   c) bones
   d) nervous tissue

   **Answers:**
   1c, 2a, 3b, 4b, 5d, 6b, 7be, 8d, 9ab, 10b, 11a, 12a, 13b, 14c, 15bd, 16 ab, 17ab.

   **LITERATURE:**
   1. Lecture material.
LESSON № 11

Topic: DISORDERS OF WATER-MINERAL METABOLISM

Aim of the lesson: to study disorders of water-mineral metabolism, mechanisms of dehydration and edema development, and their complications.

QUESTIONS:

5. Pathogenesis of cardiac, renal, hepatic, inflammatory, allergic, toxic edema.
Fig. 50. Daily water transfer

Fig. 51. Daily water balance of a healthy person
Fig. 52. Daily water balance of a healthy person

Fig. 53. Normal formation and drainage of interstitial fluid
Fig. 54. The renin-angiotensin-aldosterone cascade

Fig. 55. Atrial and Brain Natriuretic Peptides
Fig. 56. Chemical structure of waters sectors

Fig. 57. Dehydration
Fig. 58. Overhydration

Fig. 59. Volume of erythocytes in isotonic, hypotonic and hypertonic solutions
Tests

1. **Which water compartment is the biggest one?**
   a) intracellular
   b) interstitial
   c) plasma
   d) transcellular

2. **The highest quantity of the water lost from the human body by:**
   a) evaporation
   b) ventilation
   c) excrement
   d) urinary excretion

3. **The smallest amount of the water is in the:**
   a) fat
   b) nervous tissue
   c) bones
   d) teeth

4. **The largest amount of the water is in the:**
   a) lung
   b) liver
   c) fat
   d) nervous tissue
   e) bones

5. **Hormone that increases sodium reabsorption in kidney is:**
   a) aldosterone
   b) antidiuretic hormone
   c) corticosterone
d) natriuretic hormone

6. *Hormone that increases sodium excretion by kidney is:*
   a) aldosterone
   b) antidiuretic hormone
   c) corticosterone
   d) natriuretic hormone

7. *Which transfusion can lead to water poisoning?*
   a) 3,0 % NaCl
   b) 0,4 % NaCl
   c) 0,9 % NaCl

8. *Which changing in plasma can lead to increased water accamulation in vessels?*
   a) increased osmotic pressure
   b) decreased osmotic pressure
   c) increased oncotic pressure
   d) decreased of oncotic pressure

9. *Which changing in plasma can lead to increased water accamulation in tissues?*
   a) increased osmotic pressure
   b) decreased osmotic pressure
   c) increased oncotic pressure
   d) decreased oncotic pressure

10. *The reasons of increased synthesis of vasopressin are:*
    a) increased content of water in the organism
    b) reduced osmotic pressure in plasma
    c) reduced of oncotic pressure in plasma
    d) increased osmotic pressure in plasma
11. Clinical onset of hyperhydration includes:
   a) increased blood pressure
   b) decreased blood pressure
   c) increased size of heart
   d) decreased size of heart

12. Diabetes insipidus is characterized by:
   a) hyperosmolar hyperhydration
   b) hypoosmolar hyperhydration
   c) isosmolar hyperhydration
   d) hyperosmolar hypohydration

13. What kind of urine osmolarity is often observed in diabetes insipidus?
   a) hypertonic
   b) hypotonic
   c) isotonic

14. The symptoms of hypotonic (hypoosmolaric) overhydration are:
   a) vomiting
   b) thirst
   c) dryness of skin
   d) convulsions

15. The signs of hypotonic (hypoosmolaric) dehydration are:
   a) vomiting
   b) thirst
   c) increase blood pressure
   d) decrease diuresis
16. Which medicine should be used for treatment of hypotonic dehydration?
   a) 0,9 % NaCl
   b) 2 % NaCl
   c) 5 % glucosae
   d) 10 % albumen

17. The symptoms of hypertonic overhydration are:
   a) vomiting
   b) thirst
   c) convulsion
   d) increase diuresis

18. Hypertonic overhydration is the increased concentration of electrolytes in:
   a) cells
   b) plasma
   c) intercellular sector
   d) all water sectors

19. Hypotonic overhydration – is decreased concentration of electrolytes in:
   a) plasma
   b) cells
   c) intercellular sector
   d) all water sectors

20. Which medicine should be used for treatment of isotonic dehydration?
   a) 0,9 % NaCl
   b) plasma
   c) 3 % NaCl
   d) 10 % albumen
21. Which medicine should be used for treatment of hyperosmolar dehydration?
   a) 0,9% NaCl  
   b) plasma  
   c) 2 % NaCl  
   d) 5 % glucosae  
   e) 10 % albumen

22. Lack of aldosterone leads to:
   a) increase of potassium concentration in blood  
   b) increase of sodium concentration in blood  
   c) decrease of potassium concentration in blood  
   d) decrease of sodium concentration in blood

23. How is water content changed during hypoaldosteronism?
   a) it is increased  
   b) it is decreased  
   c) it isn’t changed

24. How is water content changed during increased synthesis of vasopressin?
   a) it is increased  
   b) it is decreased  
   c) it isn’t changed

25. What disorders are characterized by tetany?
   a) hypomagnesemia  
   b) hypercalcemia  
   c) hypocalcemia
26. What influence does parathormone perform on the calcium and phosphorus content in the blood?
   a) calcium increasing
   b) phosphorus increasing
   c) calcium decreasing
   d) phosphorus decreasing

27. Excess of aldosterone leads to:
   a) hyperpotassemia
   b) hypopotassemia
   c) hypernatremia
   d) hyponatremia

28. Natriuretic hormone promotes:
   a) dehydration
   b) excretion of sodium and water
   c) lowering of blood pressure

29. Which hormones can regulate water-electrolyte metabolism in organism?
   a) aldosterone
   b) vasopressin
   c) tymaline
   d) natriuretic hormone

30. Which hormones can save electrolytes in organism?
   a) aldosterone
   b) antidiuretic hormone
   c) natriuretic hormone
   d) desoxicorticosteron

31. Which hormones can save water in organism?
   a) testosterone
b) antidiuretic hormone
c) corticosterone
d) natriuretic hormone

32. **Water poisoning – is a consequence of:**
a) hyperosmolar overhydration
b) hypoosmolar overhydration
c) isoosmolar overhydration

33. **What reasons can lead to increased synthesis of aldosterone?**
a) increased blood pressure
b) increased synthesis of angiothensine
c) increased osmolar pressure in blood
d) decreased blood pressure

34. **Diabetes mellitus leads to:**
a) hyperosmic overhydration
b) hypoosmic overhydration
c) isoosmic overhydration
d) hyperosmic dehydration

35. **The causes of diabetes insipidus are:**
a) low level of sugar in blood
b) lack of vasopressin
c) lack of insuline
d) excess of insuline

36. **What osmolarity in urine is generally observed during diabetes mellitus?**
a) hypertonic
b) hypotonic
c) isotonic
37. **What factors lead to oedema?**
   a) increased oncotic pressure in blood  
   b) increased vascular permeability  
   c) increased hydrostatic pressure in capillaries  
   d) increased outflow of lymph

38. **The water content in organism during hyperaldosteronism is:**
   a) increased  
   b) decreased  
   c) not changed

39. **In diabetes insipidus the concentration of electrolytes is:**
   a) increased  
   b) decreased  
   c) not changed

40. **Thirst is a symptom of:**
   a) hyperosmic overhydration  
   b) hypoosmic overhydration  
   c) hyperosmic dehydration  
   d) hypoosmic dehydration

41. **What disorders lead to muscular hypotonia?**
   a) hypernatremia  
   b) hyponatremia  
   c) hypercalcemia  
   d) hypocalcemia

42. **What kind of oedema is caused by lower oncotic pressure?**
   a) cardiac
b) starvating

c) hypothyroid

d) hepatic

**Answers:**

1a, 2d, 3e, 4d, 5a, 6d, 7b, 8ac, 9bd, 10d, 11ac, 12d, 13b, 14ad, 15ad, 16bd, 17bd, 18bc, 19ac, 20ab, 21d, 22ad, 23b, 24a, 25ac, 26ad, 27bc, 28bc, 29abd, 30ad, 31ab, 32b, 33bd, 34d, 35b, 36a, 37bc, 38a, 39a, 40ac, 41bc, 42bd.

**LITERATURE:**

1. Lecture material.

LESSON № 12

Topic: DISORDERS OF ACID-BASE BALANCE

Aim of the lesson: to study disorders of acid-base balance, mechanisms of acidosis and alkalosis, and their complications.

QUESTIONS:

3. Classification of acid-base disturbances. Compensatory and decompensatory, absolute and relative, respiratory and metabolic disorders.
5. Causes and parameters of acid-base balance in metabolic acidosis.
7. Causes and parameters of acid-base balance in metabolic alkalosis.
8. Consequences of acid-base disturbances.
Fig. 60. Combine data with case history and common sense.

Fig. 61. Respiratory acidosis
**Fig. 62. Metabolic acidosis**

Metabolic Acidosis (pH<sub>2</sub> & negative BE)

- \[ \text{CO}_2 + \text{H}_2\text{O} \rightarrow \text{H}_2\text{CO}_3 \rightarrow \text{H}^+ + \text{HCO}_3^- \]
- \[ \text{HB} \rightarrow \text{H}^+ + \text{B}^- \]

Both BE (\(\text{HCO}_3^-\) and \(\text{B}^-\)) falls

\(\text{BB-NBB} = \text{negative BE}\)

**Fig. 63. Metabolic alkalosis**

Respiratory Alkalosis (\(\text{pH}_2\) & \(P_{\text{aco}_2}\))

- \[ \text{CO}_2 + \text{H}_2\text{O} \rightarrow \text{H}_2\text{CO}_3 \rightarrow \text{H}^+ + \text{HCO}_3^- \]
- \[ \text{HB} \rightarrow \text{H}^+ + \text{B}^- \]

Protein + \(\text{Ca}^{2+}\) → Co-proteneate + 2\(\text{H}^+\)

Fall in plasma-[\(\text{Ca}^{2+}\)] opens Na⁺-channels, increases excitability and leads to tetanic cramps

**Fig. 17-11**
**Tasks**

1. The patient was on the high altitude (3000 m) about 3 days. After arterial blood gas analysis the parameters of acid-base balance were following:

   \[
   \begin{align*}
   \text{pH} &= 7,47 \\
   \text{pCO}_2 &= 33,4 \text{ mm Hg} \\
   \text{HCO}_3^- &= 26,1 \text{ mM} \\
   \text{BB} &= 41,4 \text{ mM} \\
   \text{BE} &= 2,0 \text{ mM}
   \end{align*}
   \]

   Make a conclusion about the type of acid-base balance disorder and about the cause of this disturbance.

2. The patient with peptic ulcer was treated by sodium bicarbonate. After arterial blood gas analysis the parameters of acid-base balance were following:

   \[
   \begin{align*}
   \text{pH} &= 7,48 \\
   \text{pCO}_2 &= 53,4 \text{ mm Hg} \\
   \text{HCO}_3^- &= 29,1 \text{ mM} \\
   \text{BB} &= 44,4 \text{ mM} \\
   \text{BE} &= - 2,5 \text{ mM}
   \end{align*}
   \]

   Make a conclusion about type of disorder of acid-base balance and about the cause of this disturbance.

3. The patient with diabetes mellitus presents in reanimation in coma state. Arterial blood gas analysis reveals changing in the acid-base parameters. Make a conclusion about the type of this disorder.

   \[
   \begin{align*}
   \text{pH} &= 7,28 \\
   \text{pCO}_2 &= 31,4 \text{ mm Hg}
   \end{align*}
   \]
HCO$_3^-$ = 24,1 mM  
BB = 36,4 mM  
BE = - 8,5 mM

4. The patient with bronchial asthma was treated in pulmonic department of hospital. The arterial blood gas analysis revealed the following changes in the acid-base parameters:

pH = 7,29  
pCO$_2$ = 51,4 mm Hg  
HCO$_3^-$ = 29,1 mM  
BB = 44,4 mM  
BE = - 2,5 mM

Make a conclusion about the type of acid-base disorder.

**Fig. 64. Metabolic alkalosis**
Tests

1. The normal pH range of venous blood is:
   a) 7,32 – 7,42
   b) 7,0 – 7,45
   c) 7,35 – 7,7
   d) 6,9 – 7,35

2. Acid-base balance is regulated by:
   a) buffer systems
   b) kidneys function
   c) lungs function
   d) heart function
   e) brain function

3. Buffer system consists of:
   a) weak acid and weak base
   b) weak acid and strong base
   c) weak acid and salt that synthesized by this acid and strong base

4. What buffer takes part in acidogenesis?
   a) protein
   b) phosphate
   c) hemoglobin
   d) acetate

5. In increased [H+] in blood lungs regulate acid base balance by:
   a) hyperventilation
   b) hypoventilation
   c) irregular ventilation
6. Increased pCO₂ in the blood is the cause of:
   a) respiratory acidosis
   b) respiratory alkalosis
   c) metabolic acidosis
   d) metabolic alkalosis

7. Decreased pCO₂ in the blood is the cause of:
   a) respiratory acidosis
   b) respiratory alkalosis
   c) metabolic acidosis
   d) metabolic alkalosis

8. Decreased buffer base (BB) is the cause of:
   a) respiratory acidosis
   b) respiratory alkalosis
   c) metabolic acidosis
   d) metabolic alkalosis

9. BB and base excess (BE) have no changes in:
   a) respiratory acidosis
   b) respiratory alkalosis
   c) metabolic acidosis
   d) metabolic alkalosis

10. The causes of respiratory acidosis are:
    a) hypoventilation
    b) hyperventilation
    c) expansion of dead space
    d) increased concentration of CO₂ in air

11. The causes of metabolic acidosis are:
    a) hypoventilation
b) hyperventilation
c) expansion of dead space
d) vomiting
e) diarrhoea

12. The causes of metabolic acidosis are:
a) diabetes mellitus
b) hypoxia
c) diabetes insipidus
d) administration of chlorides

13. The causes of absolute metabolic alkalosis are:
a) diabetes insipidus
b) vomiting
c) intake of bacing soda
d) intravenous administration of sodium bicarbonate

14. Excess of aldosterone results in:
a) respiratory acidosis
b) respiratory alkalosis
c) metabolic acidosis
d) metabolic alkalosis

15. Lack of aldosterone leads to:
a) respiratory acidosis
b) respiratory alkalosis
c) metabolic acidosis
d) metabolic alkalosis

16. What disorder of actual buffer base (ABB) is characterized by tetany due to decreased level of Ca++ in blood:
a) respiratory acidosis
b) respiratory alkalosis
c) metabolic acidosis
d) metabolic alkalosis

17. Acidosis is lowering of venous blood pH below:
a) 7,05
b) 7,15
c) 7,25
d) 7,32

18. Phosphate buffer consists of:
a) carbonic acid
b) carbonic gas
c) acetic acid
d) monosubstituted sodium phosphate
e) twice-substituted sodium phosphate

19. Enzyme carbonic anhydrase is necessary in kidneys for:
a) acidogenesis
b) ammoniagenesis
c) reabsorption of bicarbonate
d) reabsorption of glucosae

20. Main mechanisms in ABB regulation in kidneys are:
a) acidogenesis
b) reabsorption of bicarbonate
c) reabsorption of chlorides
d) excretion of bicarbonate
e) ammoniagenesis

21. Reaction of deamination of amino acids takes part in:
a) acidogenesis
b) ammoniagenesis

c) reabsorption of bicarbonate

22. The increase of pCO₂ is a compensation of:

a) respiratory acidosis
b) respiratory alkalosis
c) metabolic acidosis
d) metabolic alkalosis

23. Decreased pCO₂ is a compensation of:

a) respiratory acidosis
b) respiratory alkalosis
c) metabolic acidosis
d) metabolic alkalosis

24. Increased level of BB is the sign of:

a) respiratory acidosis
b) respiratory alkalosis
c) metabolic acidosis
d) metabolic alkalosis

25. The cause of respiratory alkalosis is:

a) hypoventilation
b) hyperventilation
c) extension of dead space
d) increased level of CO₂ in air

26. The causes of metabolic alkalosis are:

a) hyperventilation
b) vomiting
c) diarrhoea
d) intake baking soda
27. The causes of metabolic acidosis are:
   a) diarrhoea
   b) diabetes insipidus
   c) hypoxia
   d) vomiting

28. The causes of relative metabolic alkalosis are:
   a) diarrhoea
   b) diabetes mellitus
   c) excess intake of baking soda
   d) vomiting

29. Metabolic disorders of ABB result in the changing of:
   a) pCO₂
   b) actual bicarbonate
   c) BB
   d) BE, BD

Answers:
1a, 2abc, 3c, 4b, 5a, 6a, 7b, 8c, 9ab, 10acd, 11e, 12abd, 13cd, 14d, 15c, 16bd, 17d, 18de, 19abc, 20abe, 21b, 22d, 23c, 24d, 25b, 26bd, 27ac, 28d, 29cd.

LITERATURE:
1. Lecture material.
Deficiency of water-soluble vitamins (B₁, B₂, B₆, B₁₂, C, PP).
Deficiency of fat-soluble vitamins (A, D, E, K).

1. **What vitamins are water-soluble ones?**
   a) B₁  
   b) B₂  
   c) A  
   d) D  

2. **What vitamins are liposoluble ones?**
   a) A  
   b) B₆  
   c) B₁₂  
   d) D  
   e) E  

3. **Vitamines with antioxidative properties are:**
   a) B₁  
   b) B₁₂  
   c) A  
   d) E  
   e) C  

4. **The reason of disease beriberi is deficiency of vitamin:**
   a) A  
   b) D  
   c) B₁
5. The cause of pellagra is deficiency of vitamin:
   a) A
   b) D
   c) B₁
   d) B₅
   e) PP

6. Lack of what vitamin leads to megaloblastic anemia?
   a) A
   b) D
   c) B₁
   d) C
   e) B₁₂

7. The cause of rachitis is deficiency of vitamin:
   a) A
   b) D
   c) B₁
   d) C
   e) B₁₂

8. The reason of haemorrhagic syndrome is deficit of vitamin:
   a) B₁
   b) B₆
   c) E
   d) C
   e) K

9. The cause of scurbutus is deficiency of vitamin:
10. **The signs of scorbutus are:**
   a) bruise  
   b) trombosis  
   c) infectious disease  
   d) hyperglycemia  
   e) hyperazotemia

11. **The signs of disease beriberi are:**
   a) neuritis  
   b) trombosis  
   c) cardiac failure  
   d) hyperglicemia  
   e) hyperazotemia

12. **Deficiency of vitamin B₂ is characterized by:**
   a) angular stomatitis  
   b) trombosis  
   c) cardiac failure  
   d) conjunctivitis  
   e) hyperazotemia

13. **Clinical manifestations of deficiency of vitamin B₆ are:**
   a) dermatitis  
   b) deficit of nicotinic acid  
   c) cardiac failure  
   d) conjunctivitis
14. Deficiency of vitamin B₆ is characterized by the disorder of:
   a) transamination
   b) decarboxylation
   c) formation of prothrombinase
   d) fibrinolysis
   e) glicogenesis

15. Clinical manifestations of vitamin B₁₂ deficit are:
   a) anemia
   b) cholelythiasis
   c) disorder movements coordination
   d) involvement of gastrointestinal tract
   e) icterus

16. Coenzyme forms of vitamin B₁₂ are:
   a) methylcobalamin
   b) adenosilcobalamin
   c) tetrahydrobiopterin

17. The reason of nervous system disorder in vitamin B₁₂ deficiency is the deficit of:
   a) methylcobalamin
   b) adenosilcobalamin
   c) tetrahydrobiopterin

18. The cause of anemia in vitamin B₁₂ deficiency is the deficit of:
   a) methylcobalamin
   b) adenosilcobalamin
   c) tetrahydrobiopterin

19. The reason of gastrointestinal tract disturbance in vitamin B₁₂ deficiency is the deficit of:
20. The signs of folic acid insufficiency are:
   a) anemia
   b) cholelythiasis
   f) disorder movements coordination
   c) involvement of gastrointestinal tract
   d) icterus

21. Nicotinic acid deficiency leads to:
   a) pellagra
   b) anemia
   c) scorbutus
   d) beriberi
   e) homocysteinemia

22. Signs of pellagra are (3):
   a) icterus
   b) diarrhea
   c) dermatitis
   d) steatorrhea
   e) dementia

23. Coenzyme form of nicotinic acid is:
   a) nicotinamide adenine dinucleotide
   b) coenzyme A
   c) methylcobalamin

24. Coenzyme form of pantothenic acid is:
   a) nicotinamide adenine dinucleotide
   b) coenzyme A
c) methylcobalamin

25. Lack of vitamin D leads to:
   a) pellagra
   b) rachitis
   c) scorbutus
   d) beriberi
   e) homocysteinemia

26. Deficit of vitamin A leads to:
   a) pellagra
   b) hyperkeratosis
   c) visual impairment
   d) beriberi
   e) homocysteinemia

27. Where does the synthesis of the most active metabolite of vitamin D$_3$ occur:
   a) liver
   b) kidney
   c) scin

28. The signs of rachitis are:
   a) muscular hypotonia
   b) muscular hypertonia
   c) osteomalacia
   d) bones fragility

29. Clinical onset of vitamin E deficit is:
   a) icterus
   b) ketonemia
   c) abnormalities of spermatogenesis
   d) spontaneous abortions
e) haemorrhagic disease of newborn

30. **Signs of vitamin K deficiency are:**
   a) icterus
   b) haemolytic disease of newborn
   c) sterility
   d) haemorrhagic disease of newborn

**Answers:**

1ab, 2ade, 3cde, 4c, 5e, 6e, 7b, 8de, 9b, 10ac, 11ac, 12ad, 13ab, 14 ab, 15 acd, 16ab, 17b, 18a, 19a, 20ad, 21a, 22bce, 23a, 24b, 25b, 26bc, 27b, 28ac, 29cd, 30d.

**LITERATURE:**

1. Lecture material.
Topic: CELL PATHOLOGY

Aim of the lesson: to study causes and mechanisms of cell damage, consequences of subcellular structure injury and cell compensative reaction.

QUESTIONS:

2. Causes and consequences of cell energy production disturbance.
4. Ion distribution in extra- and intracellular space. Role of ions in the cell function during pathological conditions.
5. The mechanisms of transcellular communication (eicosanoids, hormones and cellular growth factors). Role of calcium in cell function and injury.
6. Consequences of organelles injury (membrane, nuclear, mitochondria, ribosome, ect.)
7. Necrosis as general mechanism of accidental cell death. Role of calcium, lysosomal ferments and reactive oxygen spaces in necrosis development.
Fig. 65. Model of a cell membrane

Fig. 66. Cell components
Fig. 67. The critical role of oxygen in injury

Fig. 68. Mechanisms of cellular injury
Fig. 69. Reversible and nonreversible cells injury
Fig. 70.  Cell necrosis and apoptosis

Fig. 71.  Scheme of apoptosis (by Mullauer et al., 2002)
Tests:

1. Shortage of cytochromes c is the cause of:
   a) Damages in genetic system of a cell
   b) Disturbances of implementation of genetic program in a cell
   c) Abnormalities of ATP synthesis
   d) Disorder of ATP transport
   e) Disorder of ATP using

2. The cause of abnormalities in supply of energy is damage of:
   a) mitochondrione
   b) lysosome
   c) peroxisome
   d) rough endoplasmatic reticulum (ER)
   e) agranular endoplasmatic reticulum ER

3. The deficit of kreatine leads to:
   a) damage of ATP synthesis
   b) damage of ATP transport
   c) disorder of using ATP

4. Shortage of carnitine leads to:
   d) damages of ATP synthesis
   e) damages of ATP transport
   f) disorder of using ATP

5. The causes of insufficiency in synthesis of ATP are lack of:
   a) oxygen
   b) kreatine
   c) glucosae
d) G protein

6. The causes of insufficiency in ATP transport are lack of:
   a) oxygen
   b) kreatine
   c) carnitine
   d) G protein

7. Damage of mitochondrions leads to:
   a) disturbances of energy synthesis
   b) disturbances of the mechanisms of genetic programme realization
   c) transcription abnormalities
   d) translation abnormalities

8. Inhibition of oxidative phosphorylation leads to disorder of:
   a) synthesis of energy
   b) storage of genetic information
   c) cellular interaction

9. Injury of mitochondria leads to:
   a) activation of apoptosis
   b) disturbances of the mechanisms of the realization of the genetic program
   c) abnormalities of replication
   d) disorder of cellular interaction

10. Injury of cell membrane leads to:
    a) disturbances of energy synthesis
    b) activation of apoptosis
    c) imbalance of ion and water in the cell
11. The cause of disturbance of genetic program of the cell is damage of:
   a) nuclei
   b) lysosomes
   c) peroxysome
   d) rough ER
   e) agranular ER

12. The cause of water and ions imbalance in the cell is injury of:
   a) nuclei
   b) ribosome
   c) peroxysome
   d) cell membrane
   e) agranular ER

13. The cause of abnormalities reception is damage of:
   a) mitochondrion
   b) lysosome
   c) peroxysome
   d) cell membrane
   e) agranular ER

14. The cause of oxydative stress is prevalence of:
   a) oxydantes in antioxydantes
   b) antioxydantes in oxydantes

15. Oxydative stress leads to:
   a) disorder of replication
16. **Damage of DNA leads to:**
   a) edema of the cells
   b) abnormalities reception
   c) genetic apparatus of the cell
   d) realization of genetic program

17. **The cause of intracellular edema is:**
   a) activation of apoptosis
   b) disturbances of the mechanisms of the realization of the genetic programme
   c) disorder of replication
   d) damage of ion channel

18. **Injury of rough ER leads to:**
   a) decreased synthesis of protein
   b) decreased synthesis of lipids
   c) disturbances of fluid balance of the cell
   d) decreased carbohydrate synthesis

19. **Injury of rough ER leads to:**
   a) abnormalities level of calcium in the cytosole
   b) water imbalance in the cell
   c) reduction of antioxidant protection in the cell

20. **Damage of the agranular ER leads to:**
   a) disturbances of the calcium level in the cytosol
   b) decreasing of the detoxication processes
   c) water imbalance in the cell

21. **Injury of the agranular ER leads to:**
a) decrease of protein synthesis
b) water imbalance in the cell
c) decrease of carbohydrate synthesis

22. Consequences of the damage of Golgi complex are:
a) abnormalities of synthesis
b) disorder of transport of substances
c) disorder of detoxication

23. «Storage diseases» is the result of injury of the:
a) nuclei
b) lysosome
c) peroxysome
d) cell membrane
e) agranular ER

24. Decreased activity of katalase is the result of injury of the:
a) nuclei
b) lysosome
c) peroxysome
d) cell membrane
e) agranular ER

25. Zellweger syndrom is the result of damage of:
a) nuclei
b) lysosome
c) peroxysome
d) cell membrane
e) agranular ER

26. The cause of chronic inflammation of respiratory tracts is the damage of:
a) nuclei  
b) microtubule  
c) peroxysome  
d) cell membrane  
e) agranular ER

27. The cause of non-insulin-dependent diabetes mellitus is abnormalities of:
   a) processes of cellular signalling  
b) synthesis of insuline  
c) cellular distribution of water and ions  
d) cell receptors

28. The cause of disturbances of realization of postreceptors mechanisms is imbalance of:
   a) calcium ions  
b) diacylglycerol  
c) interferone  
d) epidermal growth factor

29. The main effector molecules in apoptosis are:
   a) cytochrome c  
b) domain of death  
c) caspase  
d) endonuclease

30. Insufficiency of apoptosis leads to:
   a) tumors  
b) AIDS  
c) alzheimer's disease  
d) autoimmune disease
31. The excess of apoptosis leads to:
   a) tumors
   b) AIDS
   c) alzheimer's disease
   d) autoimmune disease

Answers:
1c, 2a, 3b, 4a, 5ac, 6b, 7a, 8a, 9a, 10ce, 11a, 12d, 13d, 14a, 15ac, 16cd, 17d, 18a, 19a, 20b, 21c, 22ab, 23b, 24c, 25c, 26bc, 27ad, 28ab, 29cd, 30a, 31bcd.

LITERATURE:
1. Lecture material.
LESSON № 13

CONTROL QUESTIONS TO PATHOPHYSIOLOGY OF METABOLISM

2. Consequences of total and partial fasting (carbohydrate’s, lipid’s and protein deficit). Protein-calorie malnutrition. Particularities in children (Kwashiorkor).
13. Clinical symptoms of diabetes mellitus and mechanisms of their development.


17. Plasma lipoprotein’s composition and functions. Apoproteins.


25. Types of residual plasma nitrogen level increasing. Mechanisms of there development.


28. Cell pathology


31. Edema, classification. Factors leading to edema development.
32. Pathogenesis of cardiac, renal, hepatic, inflammatory, allergic, toxic edema.
33. Disturbances of mineral metabolism.
35. Parameters of acid-base balance at respiratory and metabolic acidosis.
36. Parameters of acid-base balance at respiratory and metabolic alkalosis.
37. Deficiency of water-soluble vitamins (B₁, B₂, B₆, B₁₂, C, PP).
38. Deficiency of fate-soluble vitamins (A, D, E, K).
LESSON № 14

Topic: HYPOXIA. HYPEROXIA

**Aim of the lesson:** to study causes and mechanisms of hypoxia and hyperoxia, its classification and pathogenesis.

**QUESTIONS:**

1. Normal air, alveoli and blood gas parameters. Nervous and humoral regulation of respiration and blood gas parameters.
2. Definition of hypoxia. Classification.
3. Causes and changes of blood gas parameters in
   a. hypoxic hypoxia.
   b. respiratory hypoxia.
   c. blood-depended hypoxia.
   d. circulative hypoxia.
   e. tissue hypoxia.
   f. hyperoxic hypoxia.
   g. hypoxia of owerload.
4. Acute and chronic compensatory mechanisms of hypoxia.
Fig. 72. Oxy-hemoglobin dissociation curve

Fig. 73. Effects of pH, pCO₂ temperature, DPG on the oxygen-hemoglobin dissociation curve

Figure 6-8.
1. **Hypoxia is a condition, in which tissues:**
   a) receive insufficient amount of oxygen
   b) do not receive oxygen at all
   c) receive increased amount of oxygen

2. **Hypoxemia – is a reduction:**
   a) of oxygen in tissues
   b) $P_aO_2$ in blood
   c) alveolar ventilation

3. **Blood oxygen capacity is:**
   a) maximal amount of oxygen, which can connect 100 ml of blood in oxygen complete Hb saturation
   b) contained in blood oxygen amount
   c) dissolved in plasma oxygen amount

4. **Arterial blood oxygen capacity is:**
   a) 19-20 vol.%
   b) 15-17 vol.%
   c) 25-30 vol.%

5. **To determinate blood oxygen capacity we have Hb amount in g% multiply in:**
   a) 1,34
   b) 2,34
   c) 3,4

6. **Healthy man arterio-venous difference in oxygen is:**
   a) 5 – 6 vol.%
   b) 8 – 10 vol.%
   c) 10 – 12 vol.%
d) 2 – 4 vol.%

7. *Arterial blood oxyhemoglobin content is:*
   a) 96 %
   b) 65-70 %
   c) 80 %

8. *Venous blood oxyhemoglobin content is*
   a) 96 %
   b) 65-70 %
   c) 80 %

9. *Blood oxygen capacity value depends on:*
   a) Hb quantity and quality
   b) red blood cells amount
   c) alveolar air oxygen content

10. *Oxygen sufficiency of organism is characterized by:*
    a) O₂ amount absorbed during **period of unit time**
    b) partial pressure of O₂ in arterial blood
    c) partial pressure of O₂ in venous blood

11. *Arterial blood oxygen content is:*
    a) 18-20 vol.%
    b) 10-14 vol.%
    c) 25-30 vol.%

12. *Venous blood oxygen content is:*
    a) 19 vol.%
    b) 14 vol. %
    c) 25 vol. %
13. **Cyanosis appears in arterial blood when content of oxygen is:**
   a) 10 vol.%
   b) 12 – 13 vol.%
   c) 18 – 20 vol.%

14. **Cyanosis appears in blood increase of:**
   a) reduction of Hb
   b) methemoglobin
   c) oxyhemoglobin
   d) carboxihemoglobin

15. **Hypercapnia – is:**
   a) increase of $p_{a}CO_{2}$ more than 44 mmHg
   b) decrease of $p_{ac}O_{2}$ less than 40 mmHg
   c) increase of $p_{v}CO_{2}$ more than 42 mmHg

16. **Hypocapnia – is:**
   a) increase of $p_{a}CO_{2}$ more than 40 mmHg
   b) decrease of $p_{a}CO_{2}$ less than 36 mmHg
   c) decrease of $p_{v}CO_{2}$ less than 44 mmHg

17. **Standart arterial blood $pCO_{2}$ is:**
   a) 40 mmHg
   b) 90 mmHg
   c) 50 mmHg
   d) 60 mmHg

18. **What kind of hypoxia caused by pulmonary gas exchange disturbance:**
   a) exogenic
   b) respiratory
   c) hemic
d) tissue

19. **What pathogenic factor leads to exogenous hypoxia?**
   a) decreased $p_aO_2$ in inspiratory air
   b) respiratory failure
   c) poisoning of carbon monoxide
   d) cyanide poisoning

20. **Circulatory hypoxia reasons are:**
   a) heart failure
   b) circulatory collapse
   c) respiratory failure
   d) cyanide poisoning

21. **The mechanism of circulatory hypoxia is:**
   a) decreased blood velocity
   b) blood oxygen capacity decrease
   c) inspiratory air oxygen partial pressure decrease

22. **The reasons of hemic hypoxia:**
   a) decreased blood velocity
   b) blood oxygen capacity decrease
   c) inspiratory air oxygen partial pressure decrease
   d) hemorrhage
   e) nitrits poisoning

23. **The reason of primary tissue hypoxia is decrease of:**
   a) blood oxygen capacity
   b) inspiratory air oxygen partial pressure
   c) respiratory enzymes activity

24. **Oxygen arterio-venous difference in primary tissue hypoxia is:**
a) unchanged
b) increased
c) decreased

25. **Dissociation of HbO₂ in right-side shift of dissociation curve is:**
   a) increased
   b) decreased
c) unchanged

26. **Dissociation of HbO₂ in left-side shift of dissociation curve is:**
   a) increased
   b) decreased
c) unchanged

27. **Factors of right-sided shift of dissociation curve are:**
   a) temperature decrease
   b) alkalosis
c) temperature increase
d) acidosis
e) hypercapnia

28. **Factors of left-sided shift of dissociation curve are:**
   a) temperature decrease
   b) hypocapnia
c) temperature increase
d) acidosis

29. **Frequent mechanisms of hypoxia compensation are:**
   a) Hyperventilation
   b) Tachycardia
c) Blood coming out from depo
30. What kind of breathlessness occurs during bronchial asthma?
   a) expiratory
   b) inspiratory

31. Hypobaric hypoxic hypoxia is characterized by:
   a) decrease of general atmospheric pressure
   b) decrease of partial pressure of oxygen in closed rooms

32. Hypoxia development mechanism in CO poisoning:
   a) methemoglobin formation
   b) carboxyhemoglobin formation
   c) cytochrome oxidase inhibition

33. Hypoxia development mechanism at nitrite/nitrate poisoning:
   a) Methemoglobin formation
   b) Carboxyhemoglobin formation
   c) Cytochrome oxidase inhibition

34. Hypoxia development mechanism at cyanide poisoning:
   a) Methemoglobin formation
   b) Carboxyhemoglobin formation
   c) Cytochrome oxidase inhibition

35. Hypoxia type at cyanide poisoning is:
   a) respiratory
   b) hemic
   c) tissuer
   d) hypoxic
36. *Hypoxia type at CO poisoning is:*
   a) respiratory
   b) hemic
   c) tissue
   d) circulatory

37. *During adaptation to hypoxic hypoxia the breathing is:*
   a) deep and frequent
   b) deep and rare
   c) superficial and frequent
   d) superficial and rare

38. *During adaptation to hypoxia haemodynamic changes are:*
   a) tachycardia
   b) increased cardiac output
   c) decreased cardiac output
   d) bradycardia

39. *Long term mechanisms of adaptation to hypoxic hypoxia include:*
   a) increased hemoglobin oxygen affinity
   b) decreased hemoglobin oxygen affinity
   c) intensification of hemopoiesis
   d) suppression of hemopoiesis

40. *Which hypoxia is characterized by increased oxygen arterio-venous difference?*
   a) hemic
   b) circulatory
   c) respiratory
41. **What kind of hypoxia is characterized by essentially decreased arterio-venous difference by oxygen?**
   a) hemic (blood-depended)
   b) circulatory
   c) respiratory
   d) tissue

42. **What disturbance of acid-base balance occurs in initial phase of mountain illness?**
   a) respiratory acidosis
   b) respiratory alkalosis
   c) metabolic acidosis
   d) metabolic alkalosis

43. **What is the mechanism of carbon monoxide poisoning?**
   a) inactivation of respiratory chain ferments
   b) damages of respiration
   c) reduces affinity of haemoglobin to oxygen
   d) carbon monoxide inactivates haemoglobin

44. **Formation of what substance is faster?**
   a) oxyhemoglobin
   b) carboxyhemoglobin

45. **Which gas is used in gas mixture with oxygen for prevention of kessone disease?**
   a) carbon monoxide
   b) helium
   c) carbon dioxide
   d) nitrogen
46. What happens during kessone disease (decompression)?
   a) cytochrome oxidase inhibition
   b) formation of nitrogen bubbles in the vessels
   c) formation of carboxyhemoglobin
   d) methemoglobin formation

47. How is the blood oxygen capacity changed during chronic exogenous hypoxia?
   a) it is decreased
   b) it is unchanged
   c) it is increased

48. How is called hypoxia caused by blood circulation disturbances?
   a) circulatory hypoxia
   b) hemic hypoxia
   c) respiratory hypoxia

Answers:
1ab, 2b, 3a, 4a, 5a, 6a, 7a, 8b, 9ab, 10a, 11a, 12b, 13a, 14a, 15a, 16b, 17a, 18b, 19a, 20ab, 21a, 22bde, 23c, 24c, 25a, 26b, 27cde, 28ab, 29abc, 30a, 31a, 32b, 33a, 34c, 35c, 36b, 37a, 38ab, 39a, 40b, 41ac, 42b, 43d, 44b, 45b, 46bc, 47c, 48a

LITERATURE:
   1. Lecture material.
LESSON № 15

Topic: ROLE OF HEREDITY IN PATHOLOGY

Aim of lesson: to study causes and mechanisms of congenital diseases, there categorization and to meet with phenotypic realizing of congenital pathology.

STUDY QUESTIONS:

2. Congenital diseases.
3. Etiology and pathogenesis of congenital diseases.
4. Mutations and mutates (alcohol, nicotine, radiation e.g.).
5. Categorization of congenital diseases.
7. Gene diseases:
   - metabolic diseases (glycogenosis, phenylketonuria, galactosemia e.g.),
   - blood diseases (hemoglobinosis S, elliptocytosis, hemophilia e.g.).
8. Investigative methods for congenital diseases:
   - genealogical, - population-statistical,- cytogenetical,- biochemical,
   - dermatoglyphical
9. Diseases of congenital supports.
11. Medical genetic consulting.
Fig. 74. The periods of the most sensibility to action of the teratogenic lesion factors
- 1-st – end 1-st – begining 2-nd week, implantation)
- 2-nd – 3-6 weeks – neurolation and beginning periods of organogenesis

Fig. 75. The sperm decides the genetic sex
Fig. 76. Mechanisms of change the quantity of chromosomes

Fig. 77. Change of the chromosomes quantity
Fig. 78. Type of chromosomal rearrangements

Fig. 79. Type of chromosomal rearrangements
Fig. 80. Intersex syndrom

Fig. 81. Clinical features and karyotypes of Turners syndrome
Fig. 82. Clinical features of Klinefelter syndrome

Fig. 83. Consequences of Robertsorian translocation (14-21) on gametogenesis and production of Down syndrome
Fig. 84. Influence of the mother’s age on appearance of Down syndrome

Fig. 85. Down syndrome
Fig. 86. Trisomy of 13-15 chromosomes (Patau syndrome)

Fig. 87. Edwards syndrome (trisomy 17-18)

**Fallo tetrades:**
- Stenosis of the truncus pulmonalis
- The right ventricle of the heart hypertrophy
- The right position of the aorta
- High defect of interventricle septum
Fig. 88. Gen disease
Fig. 89. Methods of investigation of diseases inheritance:

Tasks:

Each of the following 5 statements have True/False options:

A. Phenylketonuria (PKU) is an autosomal dominant disorder.

B. Inside the ribosome the particular sequence of the mRNA is read, and the particular sequence of nucleotides is build into a polypeptide.

C. Sex chromosome trisomy (XXX) is called Klinefelter's syndrome.

D. When carcinogens cause point mutations in genomic DNA inside the coding region, they are often pathogenic.

E. The first nucleotides on the mRNA form a regulatory sequence called the 5’untranslated region, and does not code for amino acids.
Case History A
A newborn girl suffers from almost continuous coughing. The mother contacts her doctor, who - at the third consultation - suspects cystic fibrosis and arrange a sweat test to be performed. Pilocarpine iontophoresis facilitates the collection of sweat. The NaCl concentration in sweat is found to be 70 mM, which is several fold the normal value. The patient suffers from bronchopulmonary infection with mucoviscidosis of the exocrine gland ducts, malabsorption, fatty stools (steatorrhoea), and deficiency of fat soluble vitamins (A, D, K). - The incidence of cystic fibrosis is approximately 1 out of 1600 live births.
1. What is cystic fibrosis?

Case History B
A female, whose father is an albino, plan to marry a male albino (genotype aa). They wish to know the probability of having albino children and albino carriers.
1. What is albinism?
2. What is the probability of having an albino child?
3. What is the probability of having albino carriers among their children?

Case History C
The brother of a Phenylketonuria (PKU) patient seeks genetic advice before marriage. The brother is normal and cases of PKU are excluded for generations in the family of the female.
1. What is PKU?
2. What are the probability that the brother is heterozygous or normal?
3. What are the probability of the couple having a PKU child?
4. PKU patients rarely reproduce. How can the gene persist in the population?

Answers:

Multiple Choice Questions
Answers B, D, and E are true statements, whereas A and C are false.

Case History A
1. **Pancreatic cystic fibrosis** is a recessive genetic disease caused by dysfunction of exocrine glands. The defect is in a transmembrane regulator protein called the **cystic fibrosis transmembrane conductance regulator (CFTR)**. The CFTR represents a β-adrenergic gated chloride channel, which is normally opened by elevated intracellular cAMP. The patients have a minimal chloride excretion and thus as minimal excretion of salt and water into the duct systems. This is what makes all exocrine secretions viscid, the duct systems are occluded and dilated, and finally the ducts are destroyed (eg. chronic respiratory disease and pancreatic insufficiency).

Case History B
1. **Albinism or amelanosis** is inherited as an autosomal recessive disorder of melanin synthesis. The biosynthesis of the enzyme tyrosinase is defective, which results in lack of melanin. Amelanosis is manifest by white hair, pink-white skin, blue eyes and photophobia.

   2. The genotype of the female is a0 and of the male aa. Thus the 4 gene combinations are: aa, a0, a0, and aa. Accordingly, the probability of having an **albino child** is
3. As seen above this probability is also 50%.

Case History C
1. Phenylketonuria (PKU) is also an autosomal recessive disorder. There is a defect conversion of phenylalanine to tyrosine and thus hypopigmentation. The genetic defect results in lack of the enzyme phenylalanine 4-hydroxylase. PKU must be diagnosed and treated soon after birth in order to avoid severe mental retardation. PKU patients almost never reproduce. PKU occurs once in 25000 live births in the population.

2. There are 4 possibilities: AA, Aa, aA, and aa. The normal phenotype of the brother eliminates the possibility of being homozygous recessive for PKU (aa). Thus the probability is 2/3 – of the remaining 3 possibilities.

3. It appears likely that the female is normal (eg, homozygous dominant). Accordingly, the probability of having PKU children is close to zero (1/300).

4. The PKU gene is maintained in and transmitted by heterozygous, who occasionally get a PKU child. The loss of recessive genes in PKU patients is obviously balanced by mutations as long as the incidence is constant in the population.

Tests:

1. **Karyotype is:**
   a) complex of all genes in the organism
   b) full number of chromosomes in somatic cells
   c) complex of all signs in the organism

2. **Factors causing heredity diseases are called:**
a) teratogens
b) mutagens
c) carcinogens
d) flogogens

3. **Biological mutagens are:**
a) viruses
b) microorganisms
c) food supplements
d) oil products
e) organic solvent

4. **Chemical mutagens are:**
a) microorganisms
b) pesticides
c) nitrites, nitrates
d) radioactive elements
e) ultraviolet radiation

5. **Genomic mutations are characterized by:**
a) numeric abnormalities of chromosome
b) changing of chromosome structure
c) changing of structure of gens

6. **Genomic mutations lead to:**
a) duplication of chromosome number
b) appearance of additional chromosomes in genotype
c) duplication of the chromosome segment
d) deletion of the chromosome segment

7. **Consequences of gens mutations are:**
a) abnormal protein synthesis
b) malformation of heart
c) disorder of the structure of erythrocyte membrane
d) decreasing of chromosome number

8. **Mutations that lead to mosaicism occur in:**
a) somatic cells
b) gametes

9. **What mutations lead to chromosomal diseases?**
a) gene
b) chromosomal
c) genomic

10. **What mutations lead to gene diseases?**
a) gene
b) chromosomal
c) genomic

11. **Chromosome mutations are:**
a) deletion
b) polyploidy
c) trisomy
d) translocation

12. **Autosomal dominant disorders are:**
a) phenylketonuria
b) marfan syndrome
c) haemophilia A
d) huntington chorea

13. **Autosomal dominant disorders are characterized by:**
a) the fact that every child has one chance per two to have the disease
b) presence of affected children in every generation
c) the fact that affected are only females

d) the fact that the recurrence risk is 25% for each birth

14. **Autosomal recessive disorders are:**
a) alkaptonuria
b) hemophilia A
c) marfan syndrome
d) daltonism

15. **Autosomal recessive disorders are characterized by:**
a) the fact that the recurrence risk is 100% for each birth
b) phenotypically healthy parents
c) the fact that siblings have one chance per four to be affected
d) the fact that affected are only females

16. **X-linked recessive disorders are:**
a) daltonism
b) alkaptonuria
c) tyrosinosis
d) hemophilia A

17. **X-linked recessive disorders are characterized by:**
a) the fact that both males and females are affected
b) healthy parents
c) Ill parents
d) the fact that affected are only females

18. **Codominant disorders are:**
a) hemophilia B
b) tyrosinosis
c) lactase deficiency
d) sickle-cell anemia
19. **Y-linked disorders are characterized by:**
   a) transmission of the disease to sons
   b) transmission of the disease to daughters
   c) the fact that both males and females are affected

20. **Sex-linked disorders are:**
    a) hemophilia B
    b) galactosemia
    c) six-fingered
    d) hypertrichosis of ears

21. **Diseases caused by mutation in mitochondrial genes are characterized by:**
    a) the fact that both males and females are affected
    b) the fact that only mothers transmit the mitochondrial genes
    c) the fact that only fathers transmit the mitochondrial genes
    d) the fact that the mutation is transmitted only to daughters

22. **Karyotype 45, XO is:**
    a) down syndrome
    b) turner syndrome
    c) cri du cat syndrome
    d) patau syndrome

23. **Karyotype 48, XXXY is:**
    a) patau syndrome
    b) edwards syndrome
    c) down syndrome
    d) klinfelter syndrome
    e) turner syndrome
24. Karyotype 47, XXX characterized by:
   a) male phenotype
   b) female phenotype
   c) mental retardation
   d) normal mentation
   e) sexual retardation

25. The karyotype of Down syndrome is:
   a) 47, XX (13)
   b) 47, XY (18)
   c) 45, XO
   d) 47, XY (21)

26. The karyotype for Patau syndrome is:
   a) 47, XX (13)
   b) 47, XY (18)
   c) 45, XO
   d) 47, XY (21)

27. The karyotype for Edwards syndrome is:
   a) 47, XX (13)
   b) 47, XY (18)
   c) 45, XO
   d) 47, XY (21)

28. Down syndrome is characterized by:
   a) the fact that only males are affected
   b) mental retardation
   c) tall stature
   d) pterygium colli

29. Patau syndrome is characterized by:
a) tall stature
b) internal malformations
c) the fact that patients die before the age of 1
d) increased level of phenyl pyruvate in the blood

30. **Turner syndrome is characterized by:**
   a) Pterygium colli
   b) short stature
   c) male phenotype
   d) simian crease in palm

31. **Klinefelter syndrome is characterized by:**
   a) Pterygium colli
   b) Tall stature
   c) Male phenotype
   d) Gonadal dysgenesis

32. **Klinefelter syndrome is characterized by:**
   a) congenital heart defects
   b) short stature
   c) gynecomastia
   d) low posterior hairline

33. **Down syndrome results due to:**
   a) gen mutation
   b) nondisjunction of sex chromosomes during meiosis
   c) nondisjunction of 21 pair of chromosome during meiosis
   d) polyploidy

34. **Turner syndrome results from:**
   a) gen mutation
   b) nondisjunction of sex chromosomes during meiosis
   c) nondisjunction of 21 pair of chromosome during meiosis
d) polyploidy

**Answers:**

1b, 2b, 3ab, 4bc, 5a, 6ab, 7ac, 8a, 9bc, 10a, 11ad, 12bd, 13ab, 14a, 15bc, 16ad, 17b, 18d, 19a, 20ad, 21ab, 22b, 23d, 24be, 25d, 26a, 27b, 28b, 29bc, 30ab, 31bc, 32c, 33c, 34b.

**LITERATURE:**

1. Lecture material.
LESSON № 16

Topic: EXTREME CONDITIONS. STRESS. SHOCK. COMA. COLLAPSE

Aim of the lesson: to study the pathogenesis of stress, stroke, coma, and collapse.

QUESTIONS:

2. Pathophysiology of stress. The role of sympatoadrenal and hypothalamohypophysadrenal systems in stress.
8. Collapse, it causes and development. Difference between collapse and shock.
Fig. 90. Stages of the stress-reaction
   I-st stage – initial
   II-nd – resistance
   III-d – exaustity

Fig. 91. Shock pathogenesis
Tests:

1. What conditions pertain to extremal state?
   a) collapse
   b) paralysis
   c) coma

2. What changes characterize hypovolemic shock?
   a) increased blood volume
   b) decreased blood volume
   c) decreased cardiac output
   d) increased vascular resistance

3. What changes characterize cardiogenic shock?
   a) rapid, weak pulse
   b) decrease of cardiac output
   c) increase of vascular resistance
   d) decrease of vascular resistance

4. Torpid phase of shock is characterized by:
   a) fall down of arterial blood pressure
   b) increased diuresis
   c) loss of consciousness
   d) crackles in the lungs

5. Burn shock is characterized by:
   a) sharp pain reaction
   b) intoxication by waste products
   c) reduced arterial blood pressure
   d) polyuria

6. What disturbances can be registered in anaphylactic shock?
a) expiratory dyspnea
b) reduced of blood pressure
c) increased blood pressure
d) inspiratory dyspnea

7. How does aggregation of erythrocytes change in shock?
   a) decreased
   b) increased
   c) unaffected

8. How does circulation time change in microvessels during shock?
   a) it is reduced
   b) it is increased
   c) it is unaffected

9. How is quantity of functional arteriovenous shunts changed in shock?
   a) it is decreased
   b) it is increased
   c) it is unaffected

10. The permeability of vessels in shock is:
    a) decreased
    b) increased
    c) unaffected

11. The blood supply of kidneys, intestines, muscles due to centralization of blood is:
    a) reduced
    b) increased
    c) unaffected
12. Centralization of circulation during shock helps to:
   a) reduce brain damage
   b) prevent from ischemia skin and muscles
   c) reduce liver damage
   d) reduce heart damage

13. The negative consequences of centralization of blood circulation are:
   a) cerebral edema
   b) increased overloading of heart
   c) oliguria

14. What type of acid-base disturbances occur in torpid phase of shock?
   a) metabolic acidosis
   b) respiratory acidosis
   c) metabolic alkalosis
   d) respiratory alkalosis

15. What terminal condition is characterized by temporary cessation of breathing?
   a) preagony
   b) terminal pause
   c) agony

16. Clinical death is characterized by:
   a) absence of breathing
   b) reversible changing in neurons of brain
   c) irreversible changing in neurons of brain
   d) shallow breathing

17. Clinical death is characterized by:
   a) irreversible changing of neurons of brain
b) shallow breathing
c) fibrillation of heart
d) cessation of heartbeat

18. **Biological death is characterized by:**
a) presence of breathing
b) presence of heart rate
c) irreversible changing of neurons of brain

19. **Biological death is characterized by:**
a) absence of heart beats
b) reversible changing of neurons of brain
c) absence of breathing
d) irreversible changing of neurons of brain

20. **The succession of terminal condition are:**
a) predagony, terminal pause, agony
b) terminal pause, predagony, agony
c) predagony, agony, terminal pause

21. **Phases of shock are:**
a) erectile
b) latent
c) prodromal
d) torpid

22. **Erectile phase of shock is characterized by:**
a) centralization of blood circulation
b) decreased vascular tone
c) blood storage in depo
d) activation of central nervous system

23. **Torpid phase of shoke is characterized by:**
a) centralization of blood circulation  
b) decreased vascular tone  
c) loss of consciousness  
d) activation of central nervous system

24. **Volume of blood circulation in torpid phase in traumatic shock is:**
   a) reduced  
   b) increased

25. **Blood pressure in erectile phase in traumatic shock is:**
   a) reduced  
   b) increased

26. **Collapse is characterized by:**
   a) decreased vascular tone  
   b) centralization of blood circulation  
   c) Reduced blood pressure

27. **Coma is characterized by:**
   a) loss of consciousness  
   b) presence of reflex to external irritants  
   c) normal vital functions of organism

28. **Coma is characterized by:**
   a) normal consciousness  
   b) absence of reflex to external irritants  
   c) disturbances of vital functions of organism

29. **Ammonia has an important role in the development of:**
   a) ketoacidic coma  
   b) hypoglycemic coma  
   c) hepatic coma
d) hyperosmolar coma

30. High glucose level has pathogenic role in the development of:
   a) hyperosmolar coma
   b) hypoglycemic coma
   c) hepatic coma

   **Answers:**
   1abd, 2bcd, 3abc, 4acd, 5abc, 6ab, 7b, 8b, 9b, 10b, 11a, 12ad, 13c, 14a, 15b, 16ab, 17d, 18c, 19acd, 20a, 21ad, 22ad, 23bc, 24a, 25a, 26ac, 27a, 28bc, 29c, 30a.

   **LITERATURE:**
   1. Lecture material.
LESSON № 17

Topic: TUMOR GROWING

Aim of lesson: to study the pathogenesis and main causes of malignant growth.

STUDY QUESTIONS:

Fig. 92. Cancer registration in different age groups (incidence increases with age)

Fig. 93. Benign and malignant tumor growing
Fig. 94. Types of cancerogens
Fig. 95. Simplified mechanisms of integration of oncogenic viral genes into the host cell DNA

Fig. 96. Simplified mechanisms of integration of DNA transcripts into the host cell DNA
Fig. 97. Simplified mechanisms of integration of oncogenic viral genes, or DNA transcripts, into the host DNA cell
Fig. 98. Stages of tumour pathogenesis: Initiation. Promotion. Persistance.
Fig. 99. Experimental tumors
Fig. 100. Cellular effectors of antitumor immunity and some cytokines that modulate antitumor activities

Tests

1. "Anaplasia" – is:
   a) increase of tumor mass
   b) high speed of proliferation of tumor cells
   c) disorder in maturation of tumor cells

2. Glycolysis in tumor cells is:
   a) increased
   b) decreased
   c) unchanged
3. **Biosynthesis of nucleoacids in tumor cells is:**
   a) increased
   b) decreased
   c) unchanged

4. **The type of regulation of tumor cell proliferation is:**
   a) endocrine
   b) autocrine
   c) paracrine

5. **Proliferation of tumor cells is caused by:**
   a) activation of onco-genes
   b) activation of apoptosis
   c) inactivation of suppressor-genes
   d) disturbances in apoptosis

6. **The carbohydrate metabolism in tumor cells is characterized by:**
   a) activated glycogen synthesis
   b) decreased glycolysis
   c) increased glycolysis
   d) increased glyconeogenesis

7. **Lipid metabolism in tumor cells is characterized by:**
   a) increased lipolysis
   b) increased phospholipid synthesis
   c) decreased synthesis of fatty acids

8. **During kidney sarcoma the metastasis first of all occurs in:**
   a) liver
   b) uterus
   c) intestine
d) lungs

9. Antigens of tumor cells are characterized by:
   a) normal structure
   b) embryonic structure
   c) specific structure

10. Malignant tumors are characterized by:
    a) slow growth
    b) absence of metastasis
    c) autonomous growth
    d) atypical cells

11. Benign tumors are characterized by:
    a) slow growth
    b) absence of metastases
    c) presence of metastases
    d) atypical cells

12. The stages of tumor growth are:
    a) promotion
    b) progression
    c) translocation
    d) inversion

13. The local effects of tumors are:
    a) bleeding
    b) ileus
    c) anemia
    d) anorexia
    e) endocrine changes

14. The common effects of tumors are:
a) bleeding
b) ileus
c) anemia
d) anorexia
e) endocrine changes

15. Anticancerogenic factors are:
   a) antioxidants
   b) lymphocytes
   c) glucose
   d) ammonia

16. The mechanisms of anticancerogenic defense are:
   a) anti-cellular
   b) anti-mutagenic
   c) antibacterial

Answers:

1c, 2a, 3a, 4c, 5acd, 6c, 7b, 8d, 9b, 10ab, 11cd, 12ab, 13ab, 14cde, 15ab, 16ab.

LITERATURE:
1. Lecture material.
LESSON № 18

Topic: RADIATION SICKNESS

Aim of the lesson: to study the pathogenesis radiation sickness.

QUESTIONS:

1. Radiation. Issues and power of different radiation rays.
2. Units of radiation assay.
5. Pathophysiology of bone marrow form of radiation sickness. It stages.

Fig. 101. Effects of ionizing radiation on DNA
Fig. 102. An overview of the major morphologic consequence of radiation injury
Tests

1. **What radiance possesses the greatest ionising ability?**
   a) $\alpha$ – irradiation
   b) $\beta$- irradiation
   c) $\gamma$-irradiation

2. **Which of radiances possesses the greatest making through ability?**
   a) $\alpha$ – irradiation
   b) $\beta$- irradiation
   c) $\gamma$-irradiation

3. **Which of radiances possesses the least ionising ability?**
   a) $\alpha$ – irradiation
   b) $\beta$- irradiation
   c) $\gamma$-irradiation

4. **Which of radiances possesses the least making through ability?**
   a) $\alpha$ – irradiation
   b) $\beta$- irradiation
   c) $\gamma$-irradiation

5. **What blood cells are most sensitive to ionising radiation action?:**
   a) erythrocytes
   b) neutrophils
   c) basophils
   d) lymphocytes
6. **What form of acute radiation sickness arises at irradiating of a person in doses of 1-10 Gr?**
   a) the marrowy  
   b) the intestinal  
   c) toxemic  
   d) the cerebral

7. **What form of acute radiation sickness arises at irradiating of a person in doses 10-20 Gr?**
   a) the marrowy  
   b) the intestinal  
   c) toxemic  
   d) the cerebral

8. **What form of acute radiation sickness arises at irradiating of a person in doses 20-50 Gr**
   a) the marrowy  
   b) the intestinal  
   c) toxemic  
   d) the cerebral

9. **What form of acute radiation sickness arises at irradiating of a person in doses more than 80 Gr?**
   a) the marrowy  
   b) the intestinal  
   c) toxemic  
   d) the cerebral

10. **What changes of blood are characteristic of the marrowy form of sharp radiation sickness in clinically expressed signs?**
    a) reduction of quantity of leucocytes  
    b) augmentation of quantity of leucocytes
c) reduction of quantity of erythrocytes

11. What changes of blood are characteristic of the marrowy form of sharp radiation sickness in clinically expressed signs?
   a) augmentation of the maintenance of erythrocytes
   b) augmentation of the maintenance of thrombocytes
   c) reduction of quantity of thrombocytes

12. What organism is more sensitive to ionising radiation action?
   a) the young
   b) the mature

13. In what case action of a radioactive irradiating will be more pathogenic?
   a) at the single exposition equal 400 rentgens
   b) at the fractional irradiation, integrally compounding 400 rentgens

14. What cells are most affected in ionising radiation action?
   a) the mature
   b) embrionic
   c) not differentiated

15. In what case a cell radiosensitiveness of above?
   a) at intensively going metabolism processes
   b) at low intensity of a cellular metabolism

16. What part of a cell is more sensitive to radiation?
   a) a nucleus
   b) cytoplasm
17. Specify mechanisms of direct action of radiation in the irradiated medium:
   a) ionisation of molecules
   b) damage by free radicals of chemical bonds
   c) change of chemical constitution of DNA
   d) formation lipids and chinone radio toxines

18. Specify mechanisms of the indirect (mediated) action of radiation in the irradiated medium:
   a) ionisation of molecules
   b) damage of chemical bonds by active forms of oxygen
   c) change of chemical constitution of DNA
   d) formation of lipids and chinone radio toxines

19. Specify the tissues possessing high radio damage ability (3 answers):
   a) the lymphoid
   b) the osteal
   c) hemopoetic
   d) the epithelial
   e) nervous

20. Specify the tissues possessing low radiodamage ability:
    f) the lymphoid
    g) the osteal
    h) hemopoetic
    i) the epithelial
    j) nervous

21. What is characteristic of the 1st period of acute radiation sickness?
a) a headache  
b) a nausea, vomiting  
c) hemorrhages in an internal organs  
d) absence of visible clinical exhibitings of disease

22. What is characteristic of the 2nd period of acute radiation sickness?  
a) hemopoiesis oppression  
b) hemorrhages  
c) absence of visible clinical exhibitings of disease  
d) infectious diseases

23. What is characteristic of the 3rd period of acute radiation sickness?  
a) infringement of liver function  
b) hemorrhages in an internal  
c) infringement of function of the excitatory nervous tissue  
d) anaemia

24. Changes in blood in 1 period of acute radiation sickness  
a) neutrophilic leukocytosis  
b) agranulocytosis  
c) a lymphopenia  
d) a lymphocytosis

25. Changes in blood in 2 period of acute radiation sickness:  
a) leukocytosis  
b) leucopenia  
c) a lymphocytosis
26. Changes in blood in the 3 period of acute radiation sickness:
   a) leukocytosis
   b) agranulocytosis
   c) an anaemia
   d) a thrombocytopenia

27. Manifestations of the 3rd period of acute radiation sickness
   a) infectious complications
   b) CNS Excitation
   c) bleedings
   d) thrombosis

28. The Haemorrhagic syndrome in acute radiation sickness is caused:
   a) decrease in quantity of thrombocytes
   b) increase in quantity of thrombocytes
   c) plasmin system activation
   d) increase in ability of thrombocytes to aggregation
   e) increase in permeability of vascular wall

29. Non-ionising radiances concern:
   a) γ – beams
   b) an invisible heat
   c) β – particles
   d) a visible part of a spectrum

30. Ionising radiation concerns:
   a) β- particles
   b) X-rays
   c) an ultraviolet radiation
31. What biological effects of an ultraviolet radiation are used with the medical purpose?
   a) erythema
   b) thermal action
   c) bactericidal effect
   d) blastomogenic action

32. Manifestations of pathogenic action of UV radiation on organism.
   a) a thermal shock
   b) bactericidal effect
   c) cancerogenic effect
   d) mutagen action

Answers:
1a, 2c, 3c, 4a, 5d, 6a, 7b, 8c, 9d, 10ac, 11c, 12a, 13a, 14b, 15a, 16a, 17ac, 18bd, 19acd, 20be, 21ab, 22ac, 23bd, 24ac, 25b, 26bcd, 27ac, 28ace, 29bd, 30ab, 31bd, 32ad.

LITERATURE:
1. Lecture material.
# ABBREVIATIONS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
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<tbody>
<tr>
<td>AIDS</td>
<td>Acquired immunodeficiency syndrome</td>
</tr>
<tr>
<td>ACTH</td>
<td>Adrenocorticotropic hormone</td>
</tr>
<tr>
<td>ABB</td>
<td>Actual buffer base</td>
</tr>
<tr>
<td>BB</td>
<td>Buffer base</td>
</tr>
<tr>
<td>BD</td>
<td>Base deficit</td>
</tr>
<tr>
<td>BE</td>
<td>Base excess</td>
</tr>
<tr>
<td>BWI</td>
<td>Body weight index</td>
</tr>
<tr>
<td>BW</td>
<td>Body weight</td>
</tr>
<tr>
<td>DIG</td>
<td>Disseminated intravascular coagulation</td>
</tr>
<tr>
<td>DNA</td>
<td>Desoxy ribonucleic acid</td>
</tr>
<tr>
<td>Fig</td>
<td>Figure</td>
</tr>
<tr>
<td>G</td>
<td>Growth</td>
</tr>
<tr>
<td>GABA</td>
<td>Gamma-aminobutyric acid</td>
</tr>
<tr>
<td>Gr</td>
<td>Gray</td>
</tr>
<tr>
<td>IL</td>
<td>Interleukine</td>
</tr>
<tr>
<td>HDL</td>
<td>High density lipoprotein</td>
</tr>
<tr>
<td>LDL</td>
<td>Low density lipoprotein</td>
</tr>
<tr>
<td>PAF</td>
<td>Platelet-activating factor</td>
</tr>
<tr>
<td>pH</td>
<td>Active reaction</td>
</tr>
<tr>
<td>NO</td>
<td>Nitric oxide</td>
</tr>
<tr>
<td>TNF</td>
<td>Tumor necrotic factor</td>
</tr>
<tr>
<td>RBC</td>
<td>Red blood cells</td>
</tr>
<tr>
<td>WBC</td>
<td>White blood cell</td>
</tr>
<tr>
<td>VLDL</td>
<td>Very low density lipoprotein</td>
</tr>
</tbody>
</table>
CONTENTS

PROGRAMME ON PATHOPHYSIOLOGY ..................... 3

LIST OF PRACTICAL SKILLS .................................. 21

LESSON № 1
SUBJECT AND PURPOSE OF PATHOPHYSIOLOGY ........ 22

LESSON № 2
DISORDERS OF PERIPHERAL CIRCULATION ............ 25

LESSON № 3
INFLAMMATION. ALTERATION AND EXUDATION 35

LESSON № 4
INFLAMMATION. PROLIFERATION AND REGENERATION ........................................ 42

LESSON № 5
THERMAL REGULATORY DYSFUNCTION. FEVER. HYPERTERMIA. HYPOTHERMIA .................. 59

LESSON № 6
IMMUNOLOGICAL DISORDERS. CATEGORIZATION. AIDS .......................................................... 69

LESSON № 7
ALLERGY ............................................................... 79

LESSON № 8
DISORDERS OF CARBOHYDRATE METABOLISM .. 91

LESSON № 9
DISORDERS OF LIPID METABOLISM ..................... 107

LESSON № 10
DISORDERS OF PROTEIN METABOLISM. GOUT ... 120
LESSON № 11
DISORDERS OF WATER-MINERAL METABOLISM 137

LESSON № 12
DISORDERS OF ACID-BASE BALANCE ................... 152

LESSON № 13
CONTROL QUESTIONS TO PATHOPHYSIOLOGY OF
METABOLISM .......................................................... 182

LESSON № 14
HYPOXIA. HYPEROXIA ........................................... 185

LESSON № 15
ROLE OF HEREDITY IN PATHOLOGY 196

LESSON № 16
EXTREME CONDITIONS. STRESS. SHOCK. COMA.
COLLAPSE ................................................................. 216

LESSON № 17
TUMOR GROWING .................................................. 224

LESSON № 18
RADIATION SICKNESS ........................................... 235

ABBREVIATIONS ......................................................... 244
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в 2-х частях
Часть 1
ОБЩАЯ ПАТОФИЗИОЛОГИЯ
Пособие
для студентов факультета иностранных учащихся
с английским языком обучения

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in 2 parts
Part 1
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Manual for the faculty of foreign students

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