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Учреждение образования
«ГРОДНЕНСКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ»

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ПАТОЛОГИЧЕСКАЯ ФИЗИОЛОГИЯ

Пособие
для студентов факультета иностранных учащихся
с английским языком обучения

С приложением компакт-диска

2-е издание, дополненное и переработанное

PATHOPHYSIOLOGY

Manual
for the faculty of foreign students (in English)

With the compact disk appendix

Revised and updated 2nd edition

Гродно
ГрГМУ
2014

PROGRAMME ON PATHOPHYSIOLOGY

I. GENERAL PATHOPHYSIOLOGY

SUBJECT OF PATHOPHYSIOLOGY

Objectives of pathophysiology. Main parts: General Pathophysiology (study of disease (nosology), study of typical (standard) pathological processes, Systemic Pathophysiology (pathophysiology of organs and systems). Purpose of Pathophysiology. Methods of pathological physiology. Acute and chronic experiments. Etiology. Description of main etiological factors. Pathogenesis. Pathological reactions. Pathological process. Pathological condition.

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

DISORDERS OF MICROCIRCULATION

Causes, mechanisms and manifestations of microcirculation disturbances. Classification of microcirculation disturbances. Arterial hyperemia: causes, symptoms, and consequences. Venous hyperemia: causes, symptoms, and consequences. Ischemia: causes, symptoms, and consequences. Stasis: causes, symptoms, and consequences. Blood rheological and plasma composition disturbances.

Embolism. Exogenous and endogenous embolism. Consequences. Embolism by a blood clot. Causes of pulmonary, brain, cardiac embolism. Causes of fat, gas and air embolism development.

INFLAMMATION

Inflammation (flogosis). Causes and mechanisms of main inflammation signs development. Exogenous and endogenous causes of inflammation. Inflammation stages (alteration, exudation and emigration, proliferation). Alteration. Primary and secondary alteration. Particularities of metabolism at alteration. Physicochemical changes at alteration. Description of microcirculation breaking in area of inflammation (short ischemia, arterial hyperemia, venous hyperemia, stasis) and mechanisms of their development. Exudation. Its biological value. Difference exudates from transudations fluid.

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.

Categorization. Etiology of fevers. Pyrogens (exogenous and endogenous). Pyrogen's action mechanism. Stages of fevers, their features. Changing of metabolism in the organism in the fever. Changing the functions of organism in the fever. Types of warm-up crooked. Biological role of fever.

Hyperthermia. Causes. Disturbanses of organism in hyperthermia. Differentiation of hyperthermia from fever.

Hypothermia. Causes. Disorders in organism in hypothermia. Hypothermia using in medicine.

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). Immunol-

ogycal 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. STARVATION

Types of starvation. Metabolic and functional disturbances in starvation. Principals of starvation therapy. Therapeutic starvation.

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

PATHOLOGY OF CARBOHYDRATE METHABOLISM

Causes and consequences of carbohydrate digestion disturbances. Symptoms. Lactase deficiency.

Hyperglycemia. Types. Consequences. Hypoglycemia. Types. Consequences.

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

Diabetes mellitus. Etiology. Types and there particularities. Pathogenesis of diabetes mellitus. Main metabolic disturbances. Clinical symptoms of diabetes mellitus and mechanisms of their development. Complications of diabetes mellitus. Types of comas and their pathogenesis. Diabetic vascular complications. Pathogenesis of atherosclerosis, diabetic nephropathy, diabetic retinopathy, peripheral neuropathy.

PATHOLOGY OF LIPID METHABOLISM

Role of lipids in the organism. Causes and consequences of lipid digestion disturbances. Symptoms. Steatorrhea. Hepatic role in lipid metabolism. Plasma lipoprotein’s composition and functions. Apoproteins. Hyperlipidemias. Types (classification) (by World Health Organization). Causes and consequences.

Obesity. Types. Causes and consequences. Hepatic lipoid infiltration and dystrophy. Causes and consequences.

Atherosclerosis. Pathogenesis. Atherogenic and antiatherogenic lipoproteins. Risk factors of atherosclerosis development.

PATHOLOGY OF PROTEIN METHABOLISM.

Biological role of proteins, peptides and amino acids. Consequences of amino acid insufficiency. Causes and consequences of protein digestion disturbances. Symptoms. Celiac-sprue. Causes of protein insufficiency. Consequences. Causes and consequences of intermediary amino acids metabolism.

Pathology of plasma protein composition. Dysproteinemias types and features. Types of residual plasma nitrogen level increasing. Mechanisms of their development.

Gout. Pathogenesis. Symptoms and therapy.

PATHOLOGY OF WATER-ELECTROLITE AND MINERAL BALANCE

Role of hormones in regulation of water-mineral balance. Classification of water-electrolite disturbances. Negative water-electrolite balance. Hypo-, iso- and hyperosmolaric types of dehydration. Causes, symptoms and consequences. Therapy. Positive water-electrolite balance. Types of hyperhydration. Water poisoning. Causes, symptoms and consequences. Therapy.

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

Disturbances of mineral metabolism (Na, K, Ca, P) and microelements (F, J, Cu, Se, Mn).

CLASSIFICATION OF ACID-BASE DISTURBANCES

Acidosis and alkalosis. Features. Parameters of acid-base balance under respiratory and metabolic acidosis. Parameters of acid-base balance under respiratory and metabolic alkalosis.

PATHOLOGY OF VITAMINES METHABOLISM

Deficiency of water-soluble vitamins (B_1, B_2, B_6, B_{12}, C, PP). Deficiency of fate-soluble vitamins (A, D, E, K).

CELL PATHOLOGY

Types of cell injure. Mechanisms of cell damage. Causes and consequences of cell energy production disturbance. Membrane damage. Oxidative stress as general mechanism of cell injury. Mechanisms of antioxidative defense. Ion distribution in extra- and intracellular space. Role of ions in the cell function during pathological conditions. The mechanisms of transcellular communication (eicosanoids, hormones and cellular growth factors). Role of calcium in cell function and injury. Consequences of organelles injury (membrane, nuclear, mitochondria, ribosome, etc). Necrosis as general mechanism of accidental cell death. Role of calcium, lysosomal ferments and reactive oxygen spaces in necrosis development. Apoptosis. The mechanisms of initiation of apoptosis. Stages. Consequences. Comprising characteristic of apoptotic and necrotic cell death. Mechanisms of cell defense against damaging factors. Reparative processes in injured cells.

HYPOXIA. HYPEROXIA

Normal air, alveoli and blood gas parameters. Nervous and humoral regulation of respiration and blood gas parameters. Definition of hypoxia. Classification. Causes and changes of blood gas parameters at hypoxic hypoxia. Causes and changes of blood gas parameters at respiratory, blood-depended circulative tissue hypoxia. Hyperoxic hypoxia, hypoxia of owerload. Acute and chronic compensatory mechanisms to hypoxia.

Hyperoxia. Consequences.

ROLE OF HEREDITY IN PATHOLOGY

Base of congenital function. Cariotype, genotype, phenotype. Congenital diseases.

Etiology and pathogenesis of congenital diseases.Mutations and mutates (alcohol, nicotine, radiation e.g.). Categorization of congenital diseases. Genome diseases. Sex-chromosomes and autosomes related diseases. Gene diseases: metabolic diseases (glycogenosis, phenylketonuria, galactosemia e.g.), blood diseases (hemoglobinosis S, elliptocytosis, hemophilia e.g.). Investigative methods for congenital diseases: genealogical, population-statistical, cytogenetical, Cytochemical, dermatoglyphical. Diseases of congenital supports. Embryopathic and fetopathic disorders. The critical peri-

ods of pregnancy. Medico-genetic consulting. Constitution. Types of constitution.

EXTREME CONDITIONS. STRESS. SHOCK. COMA. COLLAPSE

Stress. Definition. Theory of stress (H. Selye, 1938). Pathophysiology of stress. The role of sympatoadrenal and hypothalamo-hypophys-adrenal systems in stress. Stress stages. Distress syndrome.

Shock. Definition. Pathophysiology of shocks. Stages of shock and their mechanisms.Types of shock. Mechanisms of decompensation at shock.

Collapse, its causes and development. Difference between collapse and shock. Coma. Definition. Causes and types.

TUMOR GROWING

The tumor growth. Malignant and nonmalignant tumors. Description.

Kinds of tumor atypism: morphological, biochemical, physicochemical, antigenic, functional. Etiology of tumors. Theories: radioactive, chemical, viral, genetical.

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

Tumor influence on the organism. Tumor disease. Pathogenesis of cancer cachexia.

Antitumor activity of human organism.

RADIATION SICKNESS

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

Mechanisms of radiation inflow on the organism. Water radiolysis, lipid peroxidation and DNA mutations. Clinical types of radiation sickness. Dependence by dose. Pathophysiology of bone marrow form of radiation sickness. Its stages. Remote consequences of radiation influence on the organism.

II. PATHOPHYSIOLOGY OF ORGANS AND SYSTEMS

PATHOPHYSIOLOGY OF BLOOD

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

Blood, its composition and functions. Hematocrit. Stages of erythropoiesis. Categorization of disorders of circulative blood volume (hypervolemia, hypovolemia).

Hypervolemia. Types (simple, polycythemic, oligocythemic). Causes and outcomes. Hypovolemia. Types (simple, polycythemic, oligocythemic). Causes and outcomes.

Erythrocytosis. Polycythemia vera or Wakes's disease.

Bleeding. Types and causes. Pathogenesis and main clinical symptoms of acute bleeding. Compensatory-adaptative reactions in acute bleeding. Stages of compensation (reflex, hydremic, bone-marrow). Parameters of severity of bleeding. Factors which effect bleeding outcome. Causes and outcomes. Therapy of bleeding.

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.

Iron deficiency anemia. Etiology, pathogenesis. Sideropenic syndrome. The picture of the blood.

Vitamin B_{12} 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).

Leukocytosis. Categorization. Causes of leukocytosis (neutrophilic, eosinophilic, basophilic, monocytic, lymphocytic).

Leucopenia. Categorization. Causes and outcomes of neutropenia, lymphopenia. Agranulocytosis.

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

LEUKEMIA

Leukemia. Definition. Etiology. Pathogenesis of leukemia. The particularities of leukemic cells. Categorization of leukemia (acute and chronic).

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 hemostasitopathias).

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

Hemorrhagic disorders of hemostasis: disorders of blood vessels, disorders of platelets, coagulation disorders. Disorders of blood vessels. Reasons and mechanisms of their development, clinical manifestations: scurvy, Henoch-Schonlein purpura, Rendu-Osler-Weber syndrome.

Thrombocytopenia and qualitative disorders of platellets. Reasons and features. Autoimmune thrombocytopenic purpura. Congenital disorders of platelet function. Glansman thrombasthenia. Von Willibrand's disease.

Coagulation disorders. Coagulopathies, classification. Causes. Hemophilia (type A, B). Features.

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

PATHOPHYSIOLOGY OF NERVOUS SYSTEM

Etiology of nervous system disorders. Neurone pathology Caus-

es. Disorders of synaptic transmission. Effects of different poisons. Myastenia gravis. Denervation syndrome in somatic and internal organs. Disturbances of locomotion. Central and peripheral paralysis. The causes. Hyperkinesias. Kinds. The causes. Disturbances of sensitivity. Character of sensitivity infringements depending on the level of damages of various departments of the sensitivity analyzer. Broun-Sequards syndrome. Pain, its role in organism ability to live. The causes and occurrence mechanisms. Kinds of pain (visceral and somatic), their characteristic. Cauzalgia. Nociceptive and antinococeptive systems.

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.

Neurosis. Classification of neuroses. The characteristic. Manifestations. Means of modelling of experimental neuroses. An information triad, its role in occurrence of neuroses. Neurasthenia. Hysteria. Phobic disorders.

Narcomanias. The causes. Effects of opiods on body systems. Abuse of narcotics. Pathogenesis. Abstinent syndrome.

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

Syndrome alcohol dependence (alcoholism). Stages, the characteristic of infringements. An abstinent syndrom. Occurrence mechanisms.

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

Alzheimer's disease. Schizophrenia.

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

PATHOPHYSIOLOGY OF ENDOCRINE SYSTEM

Nature of hormones. Mechanism of hormone action. Hormonal regulatory systems. Pathologic mechanisms of endocrine disease. Causes of hormone excess and deficiency.

Hypopituitarism. Etiology and pathogenesis. Nanism. Panhypopituitarism. Acromegaly and gigantism. Etiology and pathogenesis.

Syndrome of inappropriate antidiuretic hormone (ADH) secretion. Diabetes insipidus. Parhon syndrome.

Hyperaldosteronism (Conn´s disease), Cushings disease and syndrome, congenital adrenal hyperplasia. Phaeochromocytoma. Itsenko-Cushing's disease and syndrome, congenital adrenal hyperplasia. Adrenogenital syndroms. Acute and chronic adrenal gland insufficiency. Addisson disease. Phaeochromocytoma.

Hyperthyroidism. Etiology. Pathogenesis of main symptoms. Hypothyroidism. Etiology. Pathogenesis of main symptoms.

Hyperparathyroidism. Hypoparathyroidism. Etiology. Pathogenesis of main symptoms.

Pathophysiology of sex glands. Hypogonadism. Hypergonadism.

PATHOPHYSIOLOGY OF CARDIOVASCULAR SYSTEM

Insufficiency of blood circulation. General characteristic. Classification of blood circulation pathology. Kinds of blood circulation insufficiency (acute and chronic, it is left - and rigthheart). Etiology and pathogenesis and main symptoms of acute heart failure.

Chronic heart failure. Etiology. Stages. Kinds and characteristic of reloading forms of heart insufficiency. The basic haemodynamic indices characterising acute and chronic insufficiency of blood circulation. Mechanisms of compensation during chronic heart failure (the characteristic of cardiac and noncardiac mechanisms). Concept about heterometrical (isotonic) and homeometrical (isometric) indemnification mechanisms. Manifestations of decompensation.

Myocardium hypertrophy. Hypertrophy stages on F.Meerson. Myocardial infarction. Etiology and pathogenesis. Consequences. The causes of noncoronarogenic heart damages.

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.

HYPERTENSION. HYPOTENSION

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

Primary and secondary hypertension. Classification. Risk factors. A role of endothelium dysfunction in a pathogeny of an arterial hypertension. Clinical manifestations of hypertensia. Consequences for the organism (complication).

Hypertension pathogenesis. Outcomes of hypertension. Experimental hypertension. Primary and secondary hypertension. Classification. Hypertension of a small circle of blood circulation.

Hypotension. Causes.

ARRHYTHMIAS

Common etiology of heart rhythm disturbances. Classification of heart rhythm infringements. Mechanisms of arrhythmias. Automatism infringement. Kinds. Sinus rhythm pacemaker abnormalities (sinus tachycardia, bradycardia, arrhythmia).

Excitability infringement. Occurrence mechanisms. Kinds. Extrasystoles. Atrial ectopic beats, atrial flutter, ventricular ectopic beats, ventricular fibrillation.

Conductivity infringements. Blockages. Kinds. The causes and development mechanisms. Blocks (intraatrial, sino-atrial, atrioventricular, intraventricular). Consequences of infringements of a warm rhythm for an organism.

PATHOLOGY OF RESPIRATORY SYSTEM

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

Asthma. Emphysema. Etiology. Pathogenesis.

Diffusion infringements. Ethiopathogenesis. Infringement of perfusion. The characteristic. The causes.

Pulmonary hypertension. Pulmonary edema. Pneumothorax. Pathogenesis.

Pathogenesis of periodic breathing (Cheyne-Stokes, Biote, Cussmaul).

PATHOPHYSIOLOGY OF KIDNEY

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

Pathogeny of infringements of urine formation (infringement of filtration, reabsorption, secretion and excretion). The causes and mechanisms of diuresis infringements. Quantitative and qualitative

infringements of urine formation (oliguria, anuria, polyuria). Hypostenuria, isostenuria, hyperstenuria. Causes.

Infringements of urine composition. Pathological components of urine.

Pathogenesis of acute glomerulonephritis. Aetiology, pathogeny and basic manifestations. Mechanisms of hypertensia and hypostases development in nephrites.

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.

Hepatitises. Cirrhoses. A role of alcohol and other factors in occurrence of liver diseases.

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

Portal hypertension. Causes. The characteristic of portal hypertension. Its kinds.

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

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

Gallstone disease. Etiology. Risk factors.

PATHOPHYSIOLOGY OF GASTROINTESTINAL TRACT

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

Disorders of appetite (anorexia, hyperrexia, bulimia, polyphagia). Causes of infringements. Disorders of food intake.

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

Infringements of esophagus function. Dysphagia. Achalasia. Gastro-oesophageal reflux.

Pathology of digestion in stomach. Quantitative and qualitative disorders of stomach secretory function. Pathologic types of gastric secretion. Achlorhydria. Achylia.

Etiology and pathogenesis. Symptoms of gastritis. Nausea, vomiting.

Peptic ulcer. Etiology. Pathogenesis. A role of stress factors, Helicobacter pylori.

Pathology of digestion in the intestine. Syndromes of maldigestion, malabsorbtion. Causes and mechanisms of disturbanses. Causes of diarrhoea. Consequences.

Disbacteriosis. Etiology and pathogenesis. Consequences.

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

GENERAL PATHOPHYSIOLOGY

LESSON № 1

Topic: SUBJECT AND PURPOSE OF PATHOPHYSIOLOGY

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

QUESTIONS:

1. Subject of Pathophysiology. Main parts:
   a) General pathophysiology:
      a. study of disease (nosology);
      b. study of typical (standard) pathological processes;
   b) Systems pathophysiology (pathophysiology of organs and systems).
2. Purpose of pathophysiology.
3. Methods of patholophysiology. Modeling of pathological processes and diseases. Acute and chronic experiments.
4. Etiology. Description of main etiological factors.
5. Pathogenesis. Vicious circles. Importance of etiology and pathogenesis study.
6. Pathological reactions. Pathological process. Pathological condition.
7. Typical pathological processes.
8. Health and disease. Periods of disease (latent, beginning of disease, manifestations of disease, outcomes of diseases). Remission, recurrence, complication.
9. Principles used in classification of diseases.
10. Factors of diseases: environmental, genetic, social.
11. Adaptive reactions and their role in resuscitation and pathology.

LITERATURE:

1. Lecture material.

2. General and clinical pathophysiology / ed. by A.V. Kubyshkin. – Vinnytsa: Nova Knyha Publishers. – 2011. – P. 12-28.

3. General and systematic pathology / ed. by J. C.E. Underwood. 2nd ed. – 1996. – P. 3-13.

4. Litvitsky, P.F. Pathophysiology: concise lectures, test, clinic-pathophysiological situations and clinic-laboratory problems / P.F. Litvitsky, S.V. Pirozhkov, E.B.Tezikov // Students manual. – Moscow «Geotar-Media», 2012. – P. 12-18.

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. Microcirculation. Vessels of microcirculation.
2. Disturbances of microcirculation. Types and causes. Classification of microcirculation disturbances.
3. Arterial hyperemia. Causes. Types (physiological and pathologic), mechanisms, consequences. Manifestations of arterial hyperemia and its mechanisms.
4. Venous hyperemia. Causes, mechanisms of the development, symptoms, and consequences.
5. Ischemia. Causes, and their mechanisms, symptoms, consequences. Factors that influence on the consequences of ischemia.
6. Stasis. Types, causes, symptoms, and consequences. Sludge syndrome. Mechanisms.
7. Postischemic reperfusion syndrome. Causes, pathogenesis.
8. Types of transmural disturbances of microcirculation. An increased vascular permeability, extra and intravascular disturbances, blood rheological and plasma composition disturbances.
9. Embolism. Consequences.
10. Exogenous and endogenous embolism. Causes of fat, gas and air embolism development. Embolism by a blood clot.
11. Causes of pulmonary, brain, cardiac embolism.

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. 1 – 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.

Laboratory work 4. ***White thrombus formation in peritoneum vessels of a frog***

Work description: after fixation of the frog and exposure of mesentery students watch the normal blood flow in mesentery vessels under microscope small magnification. The sodium chloride crystal should be put on vein confluence under microscope magnification control. Students investigate the process of white thrombus formation in frog peritoneum during 20-40 min using microscope.

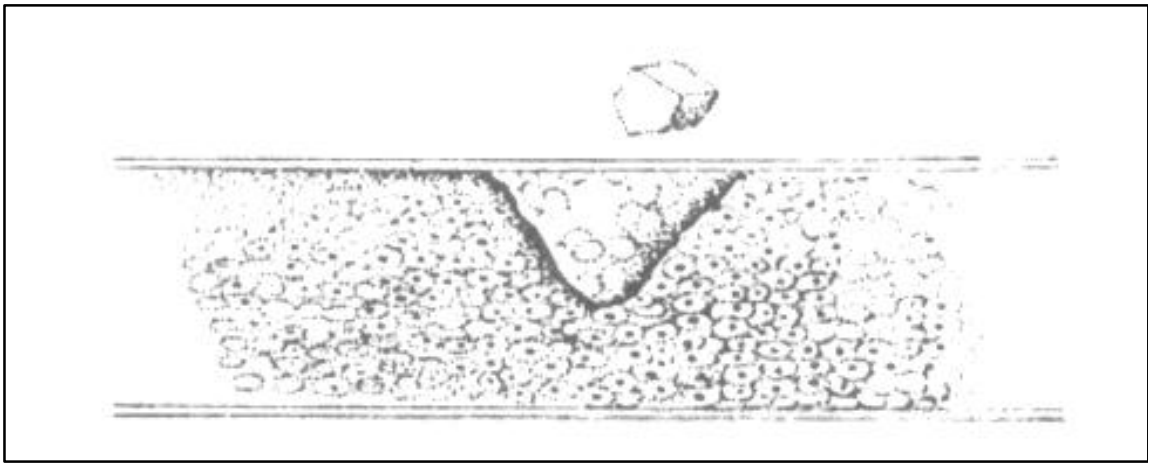

Fig. 2 – White thrombus

Students analyze, draw and make conclusions.

Laboratory work 5. ***Red thrombus development in peritoneum vessels of a frog***

Work description: using the same frog students damage the vein by sharp needle under microscope small magnification. Then students watch the process of red thrombus formation, analyze, draw and make conclusions.

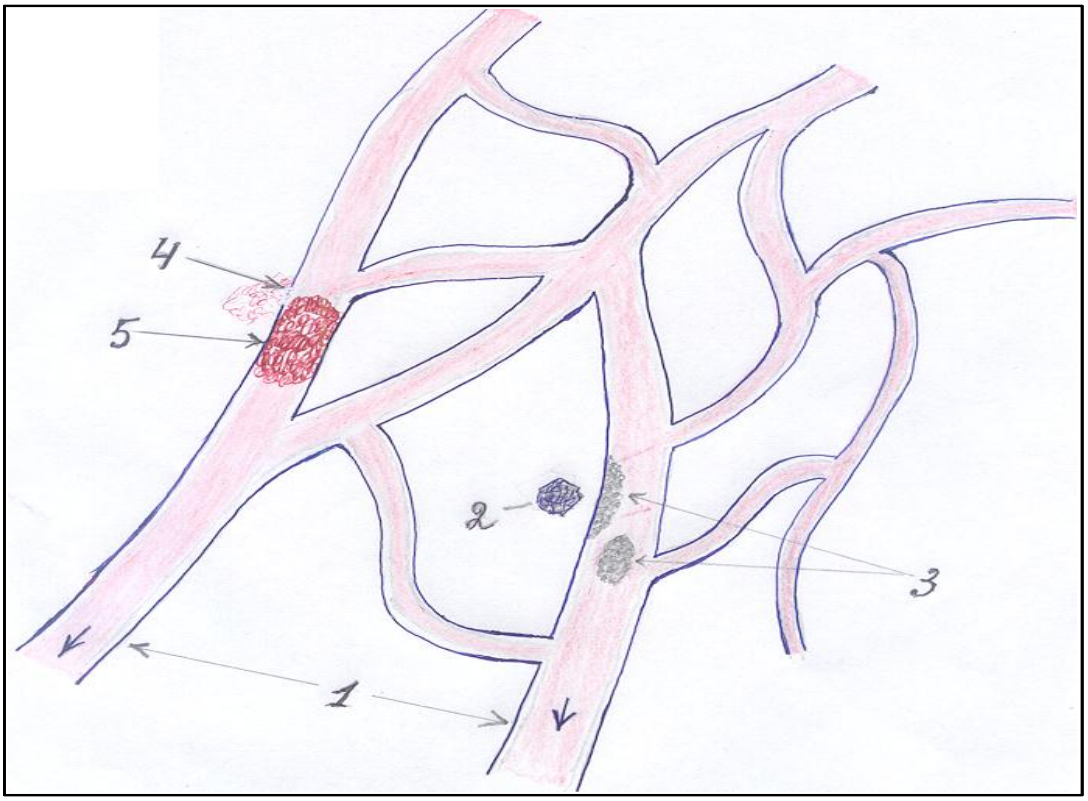

Fig. 3 – Thrombus in the veins of peritoneum of frogs
1) veins; 2) ctystall NaCl; 3) white thrombus; 4) place of the vessel lesion; 5) red (obstructive) thrombus

Students analyze, draw and make conclusions.

Laboratory work 6. ***Fat embolism of peritoneum vessels in a frog***

Work description: after fixation of the frog and exposure of mesenteric students watch the normal blood flow in mesenteric vessels under microscope small magnification. After middle thoracotomia fat solution should be administrated intracardially and students watch the emboli circulation and embolism of small peritoneum arteries.

Fill in a table:

What thrombus localization leads to?				
Pulmonary artery emboli	Stroke	Infarctum myocardium	Gangrene of low extremities	Infarctum of intestine

Fill in a table:

Consequences of a thrombus in:				
Left parts of the heart	Aorta	Venous of low extremities	Pelvic veins	Venous sinuses of the brain

Tasks:

1

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?

2

A 68-year-old male patient D. presented with chronic hepatitis and liver cirrhosis. The doctor introduced a needle into the abdominal cavity to perform aspiration of ascitic fluid. By the fifteenth minute, after 5 1 of fluid had been removed, the patient felt bad, complaining of weakness, dizziness, and nausea. But the procedure was continued. After 1,5 1 more fluid had been evacuated the patient developed syncope and lost consciousness. Several minutes later, after the emergency treatment, the patient regained total consciousness but still complained of weakness, dizziness, and nausea.

- What was the doctor’s mistake during the performance of ascitic fluid aspiration?
- What are the mechanisms of syncope after the removal of ascitic fluid?
- What are the possible mechanisms of adaptation of the brain circulation in this case?
- Why did the adaptive mechanisms turn out to be insufficient in this patient?

3

A 56-year-old male patient presented with complaints of fatigability and pains in the gastrocnemius muscles when walking. The symptoms were relieved by rest. This is called the symptom of «intermittent claudication». In addition, he complained of increased sensitivity to cold, numbness, pins- and-needles, tingling sensation in his legs at rest. The patient had a long history of heavy smoking since his teens. His occupation required working outdoors even in cold seasons when he sometimes suffered from cold. The patient's examination showed that skin on both soles was pale and felt cool and dry; the nails crumbled; no pulse was felt on posterior tibial arteries on both legs. The preliminary diagnosis was thrombangitis obliterans.

• What form of organ circulation disorders is observed in the presented patient? Name its characteristic features.

• What are the possible causes and mechanisms of this form of the circulation disorders?

• What is the possible outcome of the circulation disorder in this case?

• What are the likely mechanisms of the development of each symptoms seen in the patient?

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LESSON № 3

Topic: INFLAMMATION. ALTERATION and EXUDATION

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

QUESTIONS:

1. Inflammation. Causes of inflammation. Exogenous and endogenous causes.
2. Inflammation stages (alteration, exudation and emigration, proliferation).
3. Alteration. Primary and secondary alteration.
4. Inflammation mediators. Role of mediators.
5. Particularities of metabolism at alteration.
6. Physico-chemical changes at alteration.
7. Description of microcirculation disorders in the area of inflammation, sequence and mechanisms of their development.
8. Exudation. Its biological value. Mechanisms of vascular leakage. Difference between exudates and transudates.
9. Leukocyte recruitment. Stages of extravasation: margination, rolling, firm adhesion to vascular wall, transmigration through vascular wall.
10. Chemotaxis. Definition, mechanisms of chemotaxis, exogenous and endogenous chemoattractants.
11. Inflammatory mediators and their classification.

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 ($AgNO_3$) should be put on the center of the tongue and students fix

the changes, draw inflammatory zones and make conclusions.

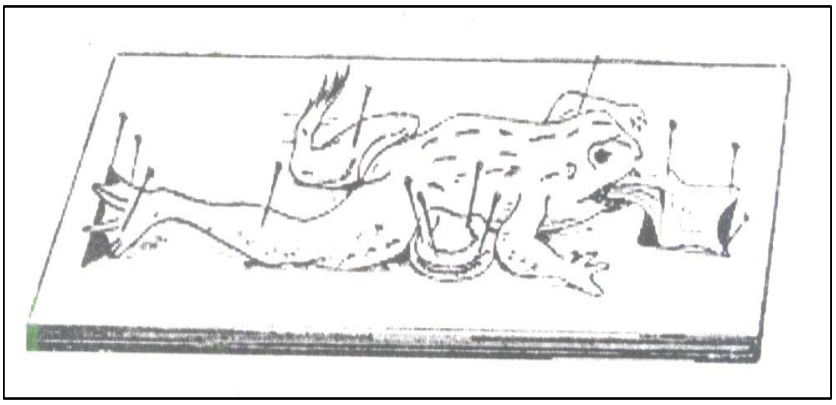

Fig. 4 – Fixation of a frog

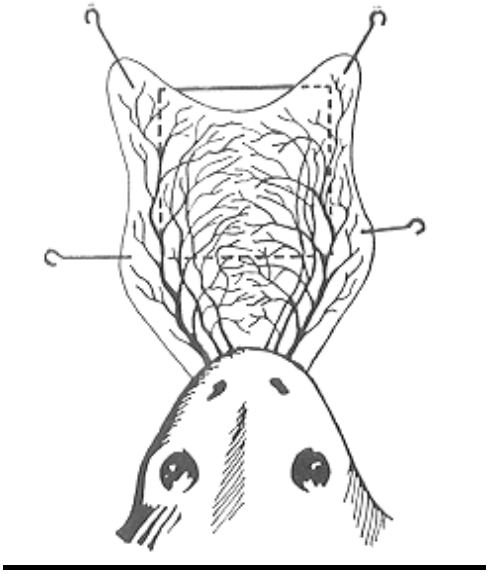

Fig. 5 – Frog's tongue preparation

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.

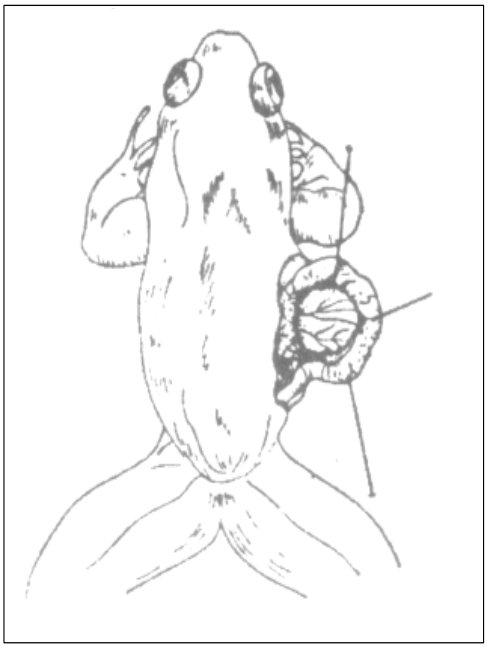

Fig. 6 – Fixation of frog's mesentery

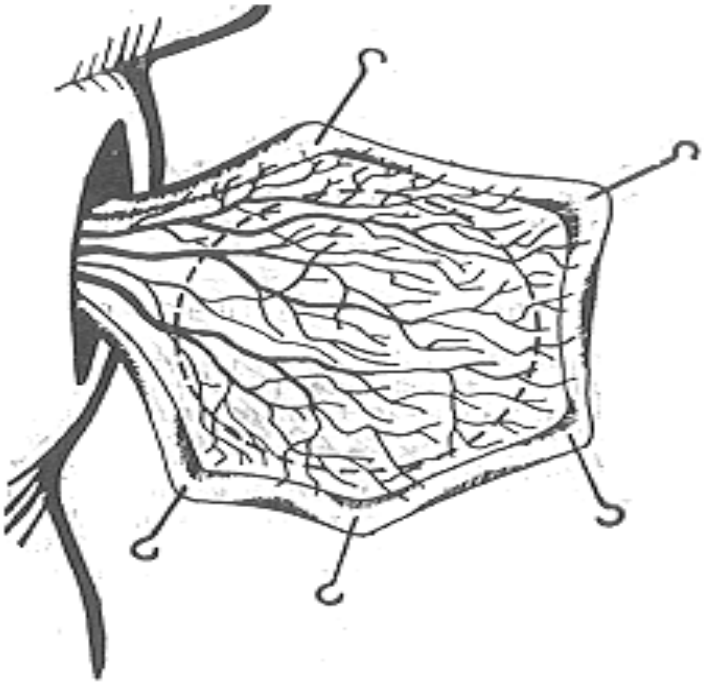

Fig. 7 – Frog's mesentery

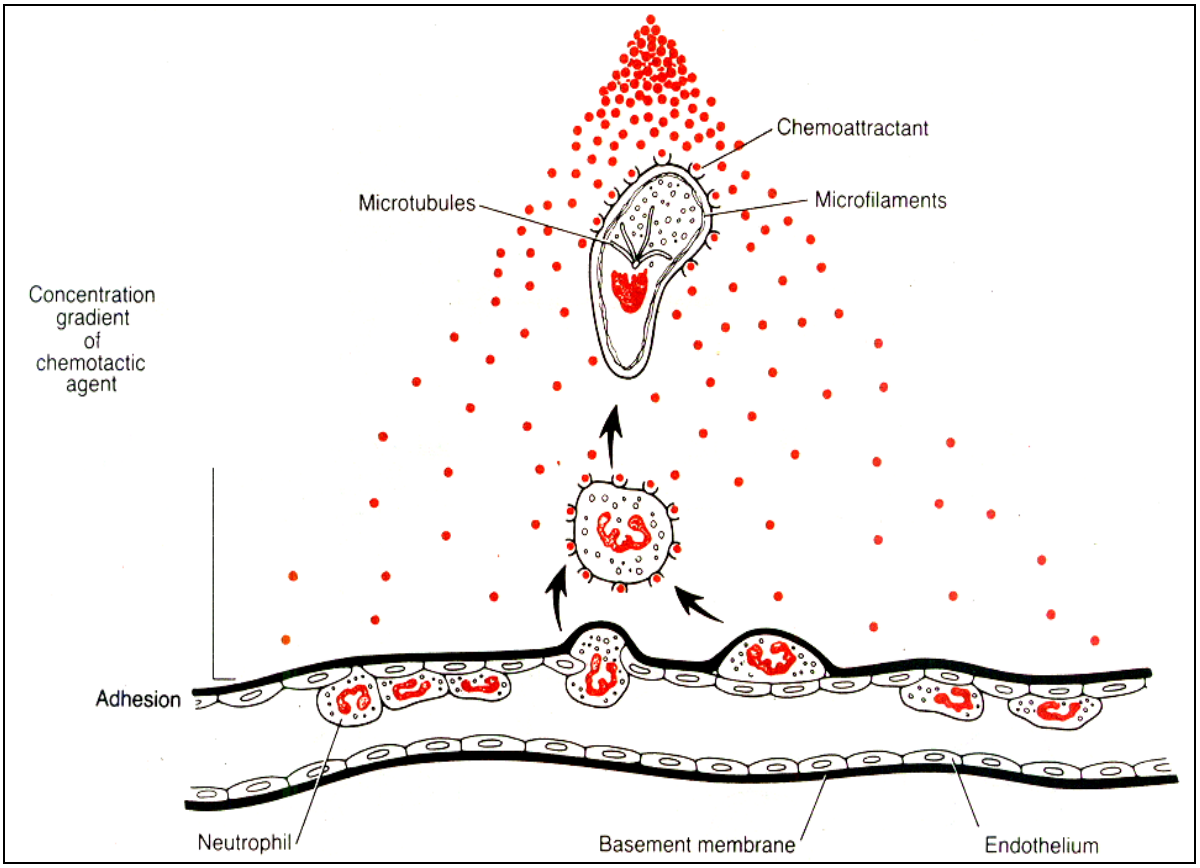

Fig. 8 – Adhesion and emigration of neutrophils in response to chemotactic agents

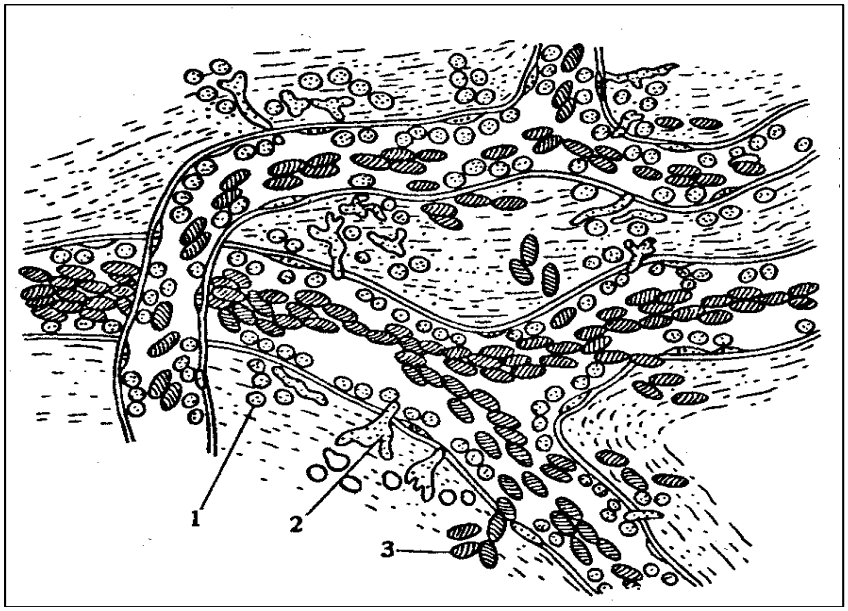

Fig. 9 – Leukocyte recruitment from the vessels

1 – leukocytes margination, rolling, firm adhesion to vascular wall;
2 – transition of leukocytes through the vascular wall; 3 – erythrocytes

LITERATURE:

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LESSON № 4

Topic: INFLAMMATION. PHAGOCYTOSIS. 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:
   a) chemotaxis;
   b) adherence to bacteria, opsonins;
   c) absorption (phagosome formation);
   d) digestion (phagolysosome formation);
2. Mechanisms of bacterial killing. Frustrated phagocytosis.
3. Pus. Composition.
4. Proliferation and regeneration.
5. Types to regenerations.
6. 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.).
7. Local signs of inflammation and mechanisms of development.
8. General features of acute and chronic inflammation (fever, leukocytosis, increase growing of settling of red blood cells and others).
9. Outcomes of inflammation. Factors, influencing the inflammation outcome. Chronic inflammation.
10. Significance of inflammation.

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 ksylol 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 intraperitoneusly to rat of peritonitis induction. After 24-48 hr bird's red blood cells should be administrated intraperitoneusly 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.

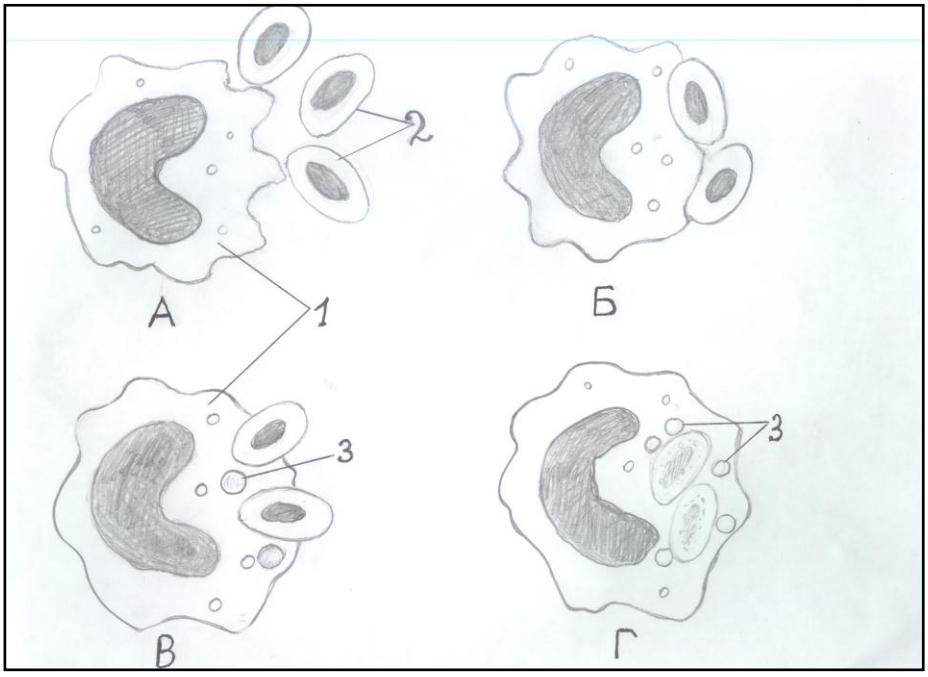

Fig. 10 – The stages of phagocytosis (approach (А), adhesion (Б), absorption (В), digestion (Г))
1– macrophages of guinea pig;
2 – red blood cells of pegion;
3 – macrophage lysosomes

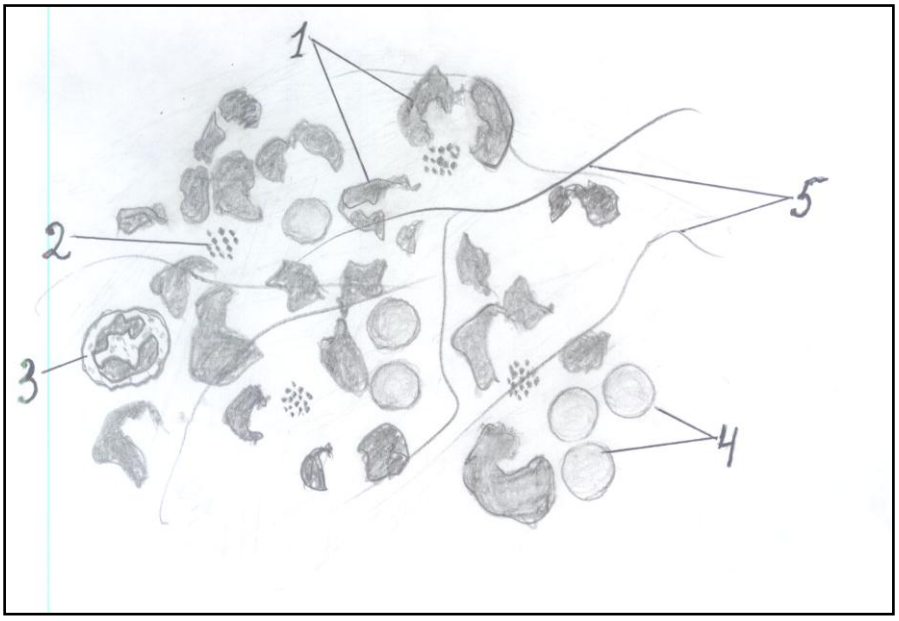

Fig. 11 – Components of purulent exudate:
1 – pyocytes;
2 – staphylococcus;
3 – neutrophil;
4 – red blood cells;
5 – collagen fibers

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:

$$X = \frac{A \times 20 \times 4000}{1600} \times 10^6, \textit{where}$$

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.

Tasks

1

A 50-year-old patient B. suffers from gastric ulcer. He has been admitted an emergency into the surgery department with a preliminary diagnosis of gastric perforation. On admission: acute pain in the epigastrial region, rigid abdomen, tenderness, signs of peritoneal irritation; body temperature 38,5°C; the absence of bowel sounds; marked leukocytosis; an increased erythrocyte sedimentation rate. Analysis of the aspirated fluid (400 ml of opalescent fluid was evacuated during the needle drainage of the abdomen) showed the presence of a large amount of leukocytes, protein – 4%, various types of bacteria including anaerobic. A plain radiograph of the abdomen demonstrated the presence of air under the diaphragm. The patient was transported to the operation room.

• Define the type of pathological process in the patient on admission.

• What are the causes and mechanisms of of the symptoms presented by the patient?

• Define the type of the fluid evacuated from the patient's abdominal cavity and explain the mechanisms of its formation.

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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:

1. Notion of temperature regulation. Heat production and heat return mechanisms.
2. Definition of fever. Fever categorization.
3. Etiology of fever. Types of pyrogens (exo- and endogenous). Pyrogen's action mechanism.
4. Pathogenesis of fever. Stage of fevers, their features. Thermoregulation at different stages of fever.
5. Metabolism changes in the organism in fever.
6. Functional changes in the organism in fever.
7. Types of fever based on the extent of temperature rise.
8. Types of temperature curves.
8. The biological significance of fever.
9. Hyperthermia. Causes. Disturbances in the organism in hyperthermia.
10. Differences between fever and hyperthermia.
11. Hypothermia. Causes. Disorders in the organism in hypothermia. Hypothermia in medicine.

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.

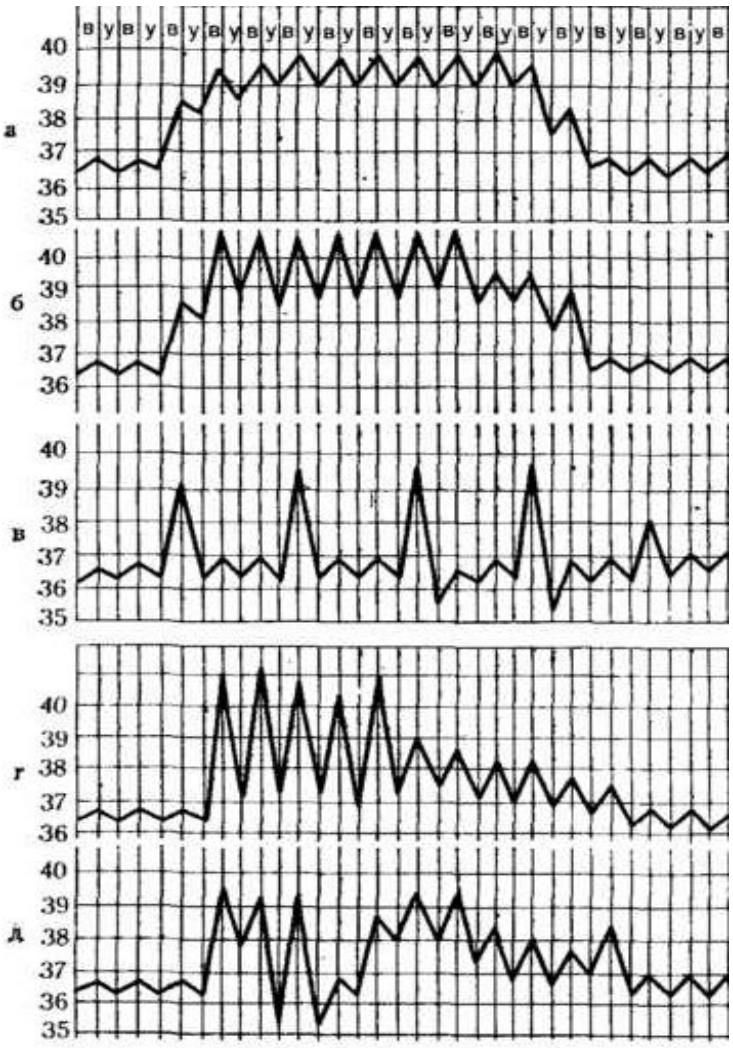
Temperature curves	The name fever	Daily fluctuation t ºC
	f. continua	day variation <1 ºC
	f. remit-tens	day variation 1,5-2 ºC
	f. inter-mittens	fever after 1-2 days
	f. hectica	day variation >3 ºC
	f. athypica	unstable changing

Fig. 12 – Types of temperature curves

Tasks

1

A 25-year-old HIV-positive female patient was admitted to hospital with the body temperature 38.9°C, cough with rales. She complained of pain in her right chest during breathing. Laboratory tests showed leukopenia due to a decrease of lymphocytes and monocytes; bronchial secretions contained large amounts of desquamating epithelium, leukocytes, various strains of bacteria; blood was positive for treponema antigens.

- What were the possible sources of pyrogens in this case?
- How can you explain the development of fever in the patient suffering from leukopenia?
- Can fever in this case be associated with AIDS?

2

A patient was admitted to the hematologic department of the hospital two weeks after he had started treatment with cytostatic drugs for chronic myelogenous leukemia. The reason of admission was worsening of condition and increase in the body temperature up to 39°C. Examination of the patient revealed a moderate hypochromatic anemia and marked leukopenia. Bacteriological analysis of his biological fluids showed the absence of pathogenic strains of microorganisms.

If you believe that the patient suffers from fever, or if you believe that he has hypothermia, explain its possible causes and mechanisms?

3

An 18-year-old patient M. felt weakness, dizziness, throbbing headache, hills and nausea when he returned home from the beach where he had spent 6 hours. Thirty minutes later he developed vomiting, and his body temperature increased up to 39 °C. He ingested aspirin but it had no much effect. Despite a moderate decrease of body temperature down to 37°C his condition continued to worsen, and he called in an ambulance. On the way to hospital he lost his consciousness and was brought to the intensive care.

- Define the type of the pathological state developed in the patient.
- What are the most likely causes, stages, and mechanisms of this pathological state?

• Why did the patient's state continue to worsen despite a decrease in the body temperature? What was the cause of loss of consciousness in this patient?

4

A 79-year old male is found apparently dead in the snow following a winter storm, where all traffic was arrested by snow. His muscles are stiff, and the heart rate is not palpable. The tendon reflexes are depressed, and the pupillary and other brainstem reflexes are lost.

The body is placed in a chapel at the hospital until the funeral. The next day the personnel are disturbed by noises from the chapel. Obviously, the man is alive.

1. What has awakened the man?
2. Suggest a likely core temperature, at the time where the man was admitted to the hospital.

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LESSON № 6

Topic: IMMUNOLOGICAL DISORDERS. 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 immunologycal 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 aquired.
6. Factors, which determine the reactivity and resistance (heredity, constitution, sex, environmental and social factors).
7. Immunologycal disorders. Categorization.
8. Primary immunodeficiencies: classification and clinical manifestations, forms of primary immunodeficiency (causes, mechanisms, clinical manifestations):
   – B-system-dependent;
   – T-system-dependent;
   – defects in leukocyte function. Defects in leukocyte adhesion and intracellular digestion.
   – complement-dependent;
   – combined.
9. Secondary (acquired) immunodeficiency states. AIDS. Etiology. Clinical symptoms and mechanisms of their development.
10. Graft-versus-host disease.

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.

Tasks

1

Patient M., male, 21 years old, suffers from hereditary form of hypogammaglobulinemia. His father also suffers from this disease; mother is healthy. An examination showed a significant decrease of IgM and IgG levels in the patient's blood.

• What are the etiology, basic mechanisms and clinical manifestations of hypogammaglobuliemia?

• Does the pattern of immune and allergic reactions change in hypogammaglobulinemia? Substantiate your opinion.

2

A patient who had undergone a surgical intervention for gallstones presented with a slow healing of the postoperative wound, its infection, and fever. The use of antibiotics (after sensitivity tests) had positive effect. Investigation of the patient's blood showed leukope-

nia due to a decreased neutrophils and monocytes, a decreased mobility of leukocytes and a low activity of leukocytic myeloperoxidase. Similar abnormalities were found in the patient's sister and brother.

Taking into account the observed changes in the leukocyte counts, name the syndrome (s) which the patient suffers from. Substantiate your answer.

What are the possible causes, manifestations, and mechanisms of this syndrome? Are there grounds for its hereditary or congenital origin?

• What are the mechanisms of the decreased neutrophil mobility and low activity of the neutrophil myeloperoxidase?

3

A 20-year-old patient who suffered from diabetes mellitus presented with slow healing of a surgical wound, purulence, and an increase in the body temperature up to 37.2°C after he had undergone the appendectomy. Treatment with antiobiotics for 6 days (after the appropriate sensitivity tests) had no effect. With an aim to find the reason for treatment failure blood glucose determination and special neutrophil tests were performed. The results of the tests are the following: hyperglycemia (320 mg/dl) is accompanied by a decrease in the number, mobility, and microbicidal activity of neutrophils.

What parameters, in your opinion, should be investigated to elucidate mechanisms of the decreased mobility and microbicidal activity of neutrophils?

What are the most likely causes and mechanisms of impairment of the neutrophils' function in this patient?

Is there any association between the abnormal phagocytosis, the infection of wound and its slow healing? If you think there is, name and characterize possible mechanisms of such an association.

Why is the body temperature in the patient with a purulent wound increased only moderately?

4

A 30-year-old patient P. visited his physician with complaints of recurrent stomatitis, tonsillitis, tracheitis, otitis, and repeated pneumonia even in the summer time. Procedures aimed at enhancing the nonspecific body resistance to infection, such as an increase of cold endurance, had no effect. The results of the laboratory tests are the fol-

lowing: lymphocytes reactivity toward phytohemagglutinine (PHA) and tuberculin is normal; activity of the complement factors and levels of IgM, IgG, and IgA in the blood serum are within the normal range. The complete blood count test with differential showed no changes in erythrocytes count or Hb concentration; leukocytes count is low due to a decrease in monocytes, but the content of granulocytes is normal. The phagocytic activity of macrophages is decreased by 45%.

• Define the type of pathology found in the patient.

• What part of the immune surveillance system is impaired in the given patient: the specific immune response or the nonspecific host defence?

• If the specific immunity is abnormal, what part of it is defective: A, B, or T? Explain the origin of symptoms presented by the patient.

• If the hostdefence system is deficient, what subsystem is responsible for the defect? Explain the mechanisms of symptoms seen in the patient.

5

An 10-year-old patient was admitted to the pediatrics department. His parents were concerned about the frequent occurrence of otitis, quinsy, rhinitis, conjunctivitis, bronchitis, pneumonia, and enterocolitis in their child. The current admission was associated with a high risk of bacterial endocarditis and sepsis in the young patient.

A test performed in the hospital showed the presence of leukopenia due to a decrease in lymphocytes, especially in T-cells, and, to a lesser degree, in B-cells, a decrease in the blood levels of IgA and IgE (by 40 and 50% respectively, compared to normal values); concentration of IgG is at the lowest normal value; proliferation of lymphocytes in the presence of phytohemagglutinine is decreased.

• Define the type of pathology developed in the patient. Substantiate your answer.

• What are the possible causes of this pathological state?

• Taking into account the results of the laboratory tests, explain the mechanism of the development and consequences of this type of pathology.

• How can you explain a decrease in the lymphocytes' reaction to phytohemagglutinine and a considerable reduction in the blood content of IgA and IgE when the levels of IgG are in the normal range?

• What symptoms in this child could be a direct result of the IgA and IgE depletion?

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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.

QUESTIONS:

1. Allergy.
2. Allergens, their categorization and description. Pathways of introduction.
3. Sensibilization. Experimental model of anaphylactic shock in guinea pigs.
4. Types of allergic reactions.
5. Pathogenesis of allergic reactions of different types. Stages:
   - immunologycal stage;
   - pathochemical stage. Allergic mediators;
   - pathophysiological stage clinical manifestations.
6. Anaphylactic (I) type of allergy. Clinical examples of the IgE-medited diseases. Systemic and local anaphylaxis.
7. Type II hypersensitivity. Complement-dependent reactions and antibody-dependent cell-mediated cytotoxicity.
8. Type III hypersensitivity. Immune complex disease.
9. Type IV hypersensitivity.
10. Description of allergic disorders (allergic rhinitis (pollinosis), bronchial asthma, e.g.).
11. Desensibilization (hyposensibilization). Types.
12. Autoimmune diseases.
13. Principles of diagnostics and therapy of allergic diseases.

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.

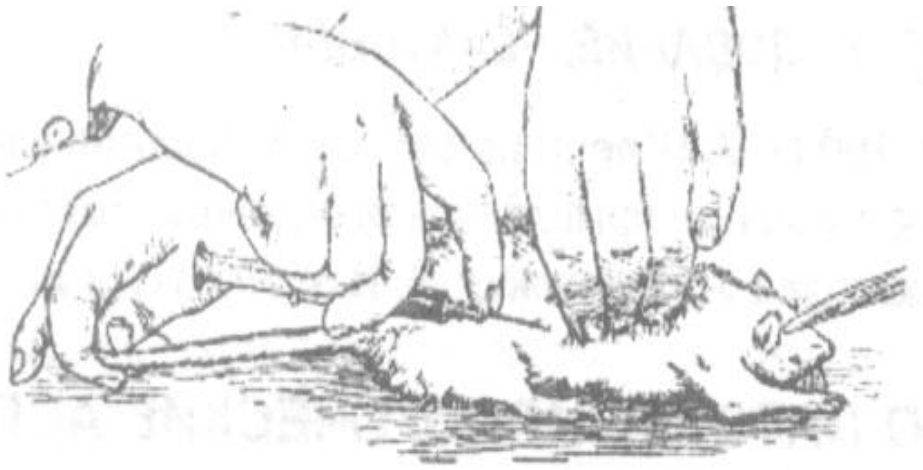

Fig. 13 – Technique of subcutaneous injections

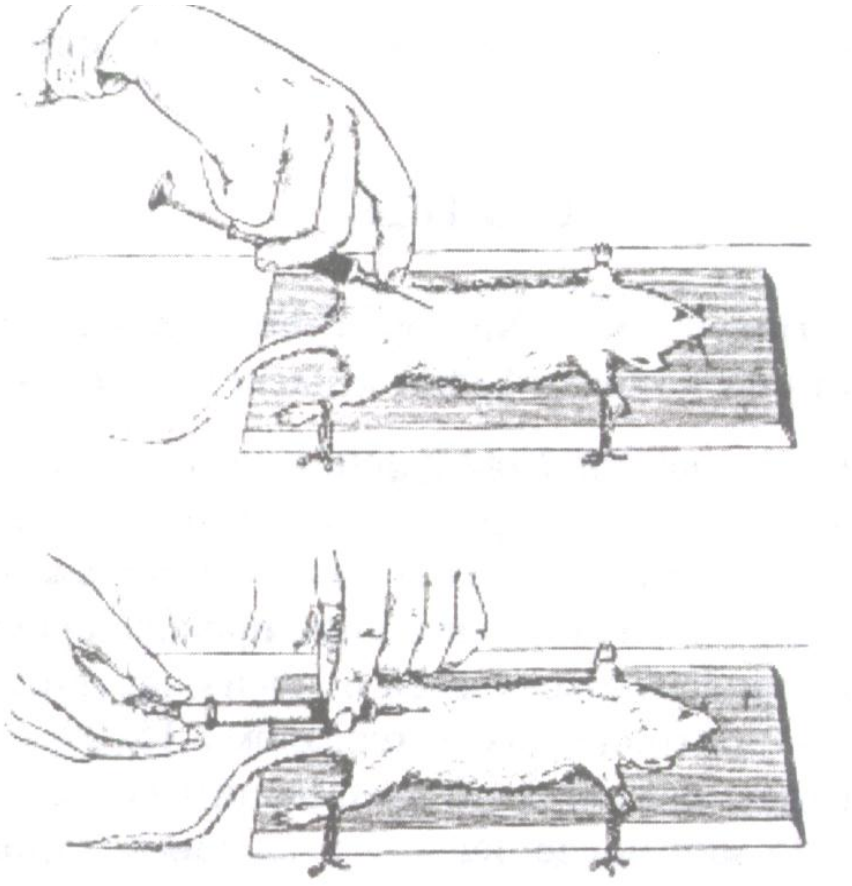

Fig. 14 – Technique of intraperitoneal injection

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

Tasks

1

After spending an hour and a half in the countryside a 30-year-old man noticed that his eyelids became red and swollen, lacrimation and nasal secretions appeared, the voice became hoarse. He had a feeling of tightness in his chest. When he returned home, the intensity of these symptoms decreased but they were still present.

- Define the type of pathological process developed in the patient. Substantiate your opinion.
- What methods can be used to determine the cause of this pathological process?
- What are the main components of the mechanism of the given process?
- What treatment approaches and preventive procedures can be used in?

2

A 33-year-old patient L. who had ingested gold-containing drugs according to the doctor's prescription noticed, first singular and then multiple, petechiae on the skin of his forearms, chest, back, and also in the oral cavity mucosa. Minor contusions were accompanied by extensive subcutaneous hemorrhages.

Blood tests showed normal count of erythrocytes, leukocytes, and Hb concentration, but a significant decrease in platelets count, and an increase in the IgG and IgM content. Therapeutic procedures used by the doctor improved the patient's condition, and he continued to take gold-containing drugs since other forms of medication had poor effect.

- Define the type of pathological reaction developed in the patient.
- What was the cause and mechanisms of this reaction?
- What procedures could be used to inhibit such pathological reaction to the gold-containing drugs?

3

A 30-year-old female patient S. visited her dermatologist several times with complaints of the red itching spots on the face, neck and hands appearing in the cold environment, such as windy cold weather. The physician prescribed her the ointment which alleviated these symptoms but did not eliminate them. One day, when she was in a hurry to get to her work, she washed her face with cold water. Thirty minutes after she had gone outdoors the sites of the skin exposed to cold water turned red, swelled and itching. The patient had to visit her doctor and later was admitted to hospital.

• What pathological process(es) developed in the patient after she had washed her face with cold water?

• What is the mechanism of this process?

• What pathological process should it be distinguished from and what is the key difference between these two processes?

• What groups of medicines can be used to prevent or inhibit this pathological process?

4

A patient with an open lower extremity trauma was repeatedly injected tetanus antitoxin in combination with antihystamines. On the ninth day after the last antiserum injection he presented with a high body temperature (up to 38 °C), severe weakness, swelling and tenderness of the shoulder and knee joints; intensely pruritic disseminated urticarial eruptions, and the enlarged tender popliteal and inguinal lymph nodes.

• What form(s) of pathology may be suspected in this patient?

• What additional data are necessary for a final conclusion about the form of pathology which the patient suffers from?

• Taking into account the given data, outline the possible cause and mechanisms of development of this pathology.

• What approaches can be used to prevent the development of this pathological process?

5

On the sixth week of his stay in hospital after extensive myocardial infarction a successfully recovering patient started to suffer from a dull pain in the chest which was aggravated by deep breathing movements, swallow, and changes in the body position. He also pre-

sented with fever (the body temperature 39°C) and a pericardial friction rub. Blood tests showed the presence of eosinophilic leukocytosis and an increased titer of the «antimyocardial» antibodies. The physician made a diagnosis of postmyocardial infarction syndrome (the Dressler's syndrome).

• Taking into account the immunogenic nature of the Dressler's syndrome, explain the type and origin of antigens causing this disease.

• Define the type of a pathological reaction developed in the patient using the classification system of Gell and Coombs.

• Describe the mechanism of this pathological reaction. To what type of immunoglobulin do the «antimyocardial» antibodies belong?

• Prove (or disprove) the fact that the Dressler's syndrome represents the allergic reaction of the delayed type.

6

A patient with an extensive full-thickness burn of the thigh got i.v. infusion of blood plasma. Soon after this, he presented with hyperemia of the face and neck, psychomotor agitation, restlessness, fear of death, an intense throbbing headache, buzzing in the ears, and nausea. The physician suspected the development of allergic reaction and gave the patient an injection of antihistamine drug. However, the patient's state continued to worsen. He developed choking sensation, acute systemic hypotension (blood pressure 65/45 mm Hg), confusion, pallor, cold sweat, and finally lost his consciousness. Later, seizures and spontaneous urination occurred.

• What pathological process (or reaction) and what type of it developed in the patient after the infusion of blood plasma? Substantiate your answer.

• Name and characterize the main steps of pathogenesis of this pathological process.

• Why did the parenteral injection of the antihistamine drug not improve the patient's condition? What procedures should be performed to prevent this pathological process (reaction)?

• What factors caused the development of respiratory, hemodynamic and psychoneurological disorders? Name these factors and

characterize mechanisms of their action.

• What emergency treatment should be employed to help the patient with this pathological process (reaction)?

7

A female, 39 years of age, works as a secretary. For 6 months she has been working with **a** new copy machine. Over the last three months she has developed a blistering rash on both hands and the rash is now turned into deep scaling of the skin. There is a mild scaling at the site of both her earrings. The patient has used a bland cream without effect. The dermatologist examines her with patch tests, where solutions of common allergens, chemicals, metals including nickel are placed on her back and occluded with dressings. The next day (24 hours later) the dressings are removed and the exposed areas examined. The examination is repeated 48 hours after the beginning of the exposure. There is a severe rash at the site of the colour powder used in the copy machine, and a mild rash at the nickel spot.

What is the diagnosis? Which therapeutic strategy is recommendable?

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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.

QUESTIONS:

1. Typical forms of derangement of carbohydrate metabolism.
2. Causes and consequences of carbohydrate digestion disturbances. Symptoms. Lactase deficiency.
3. Hyperglycemia. Types. Causes and mechanisms. Consequences.
4. Hypoglycemia. Types. Consequences. Hypoglycemic coma.
5. Causes and consequences of carbohydrate intermediary metabolism disorders.
6. Glycogenoses: classification, manifestations, mechanisms of pathology.

Aglycogenoses.

7. Diabetes mellitus. Etiology. Types and particularities.
8. Pathogenesis of type I and type II forms diabetes mellitus. Main metabolic (carbohydrate, protein, lipid) disturbances in diabetes mellitus.
9. Clinical symptoms of diabetes mellitus and mechanisms of there development.
10. Complications of diabetes mellitus. Types of comas and their pathogenesis.
11. Diabetic vascular complications. Pathogenesis of atherosclerosis, diabetic nephropathy, diabetic retinopathy, peripheral neuropathy.
12. *Hexosemias. Galactosemia: manifestation and mechanisms of pathology.
13. *Pentosemias. Fructose intolerance: manifestations and mechanisms of pathology.

Tasks

1

A 50-year-old patient F. was admitted to hospital in an unconscious state. On examination: the skin appears dry, turgor of the skin and eye ball is reduced: shallow breathing; heart rate 96 beats/min; the tongue is dry; recurrent cramps of the extremities and face muscles. Blood tests show hyperglycemia (20mM glucose), hyperazotemia, hypernatremia, pH 7,32.

The relative who accompanied the patient to hospital told the physician that the patient had been suffering from diabetes mellitus and had ingested small doses of hypoglycemic medicines. During the last month he experienced exacerbation of chronic cholecystitis and colitis, suffered from vomiting and diarrhea which occurred quite often. The patient also felt constantly thirsty and frequently urinated.

- Define the pathological state of the patient on admission.
- What was the cause of this pathological state? Describe the main stages of its pathogenesis.
- Why do patients lose consciousness during the development of this pathological state and the similar ones?
- What methods are used to treat patients in this pathological state?

2

A 23-year-old male patient visited his physician with complaints of intermittent muscle weakness, dizziness, headache, episodes of poor vision, tremor of hands, irritability, and, occasionally, confusion. The paroxysms of this condition occurred more frequently during the last four months. The patient attributed his malaise to the psychological stress that accompanied his professional activity, and also associated it with an acute feeling of hunger. After evaluation of the patient the physician made a diagnosis of neurasthenia and recommended an appropriate treatment. However, the disease went on progressing, and 1,5 months later the patient was brought by ambulance to the emergency room with a diagnosis of coma of unclear etiology.

On admission: consciousness is lost; there is evidence of midriasis, muscle cramps; tachycardia, arterial hypotension; irregular breathing; blood glucose level 30 mM.

• What form of pathology caused the clinical manifestations presented by the patient during his first visit to the physician?

• Define the pathological state of the patient in the emergency room.

• What are the main steps of pathogenesis of this pathological state?

• What other forms of pathology should be kept in mind when you are making a differential diagnosis of this pathological state?

3

A 43-year-old patient C. with an excessive body mass suffers from diabetes mellitus. To control his blood glucose he uses hypoglycemic drugs. Two weeks before he was admitted to hospital he had had an episode of excessive alcohol drinking, and soon after that he noticed an enhanced feeling of dry mouth. He drank a lot of water (up to 8-10 1 daily) and urinated frequently. He felt general weakness and pain in the legs. On the night before the admission the patient was delirious. When he woke up in the morning he was agitated, restless, and confused. The patient's relatives called in the ambulance. On admission: consciousness is absent; the skin is dry and pale. The results of the blood tests: glucose 45mM, lactic acid 2,9 mM, pH 7,29. Urine glucose level 2 mM.

• What other signs, in addition to the observed in this patient, may be found in a) diabetes mellitus and b) diabetic coma? Describe these signs and explain their pathogenesis.

• What are the main stages of pathogenesis of this coma?

4

A female patient, approximately 40 years of age, had been brought to the emergency room in an unconscious state. Witnesses of the incident told the physician that she had suddenly lost her consciousness in the bus on her way back from her summer house in the countryside. A card that was found among her papers indicated that she suffered from diabetes mellitus and took a slow-release form of insulin.

On examination: consciousness is absent, corneal and deep tendon reflexes are not observed, the pattern of breathing is unremarkable; blood pressure 80/60 mm Hg; tachycardia is present; the skin appears moist: turgor of the eye balls is increased; general trembling is alternating with episodes of clonic and tonic seizures.

The patient was treated with insulin, but no improvement was observed: the patient's condition even worsened: breathing became irregular, blood pressure decreased to 70/50 mm Hg, tachycardia progressed, and the duration of seizures increased.

What pathological state developed in the patient before and after administration of insulin? Describe causes and mechanisms of this state. What therapeutic procedure would have been appropriate for pathient before and after the administration of insulin?

5

In two monozygous sibs (8 months of age) an examination revealed a significant increase in the liver size (hepatomegaly), decreased fasting level of blood glucose (hypoglycemia), and lack of fasting blood glucose changes after the adrenaline administration. The liver biopsy investigation showed an increased content of glycogen and a significantly diminished activity of glycogen phosphorylase in hepatocytes.

- Give the definition of the pathological process developed in the infants.
- What are the possible causes of this process?
- Explain the sequence of events leading to glycogen accumulation in the liver cells in this form of pathology.

Explain the association, if any, between the low activity of glycogen phosphorylase in the patients' liver and a) an increased content of glycogen in the hepatocytes; b) hepatomegaly; c) a decreased fasting blood glucose levels; d) lack of hyperglycemic effect of adrenaline.

6

Two series of experiments were performed with an aim to determine the effect of a new antibiotic drug on the skin epithelium. In the *in vivo* studies using rats, the solution of the tested drug was applied to the skin surface. The employed dose of the antibiotic was much higher than the therapeutic one. The results of this experiment were assessed by in vivo microscopy studies which were performed

during the first 24 hours after the end of drug application with 8-hour intervals. In the in vitro experiments the tested drug was added to the suspension of epithelial cells isolated from the same stock of rats. Six hours later the cells were washed out by repetitive centrifugation and suspending in the same media containing no tested drug. The effects of the antiobiotic were studied at the same time points using regular light microscopy and electron microscopy.

The results of the experiments.

• In vivo: 8 hours after the completion of the drug application the morphological analysis of the epithelial cells revealed the signs of dystrophy and focal necrosis; the extent of pathological alterations increased by the end of the 24-hour observation period.

• In vitro: no signs of the cellular or subsellular damage were seen in the isolated epithelial cells after the drug treatment. The only changes observed included a reversible cell aggregation found in the first test (8 hours after the drug withdrawal), but not in the tests that followed.

• How can you explain the difference in results of the in vivo and in vitro experiments?

• Was damage to the epithelial cells in the in vivo test direct or indirect? Explain your point of view.

• What are the possible mechanisms of the injurious action of the drug on the epithelium?

• Taking into account the results of the in vitro experiments, which of these mechanisms are most likely?

• What processes or cell functions should be studied in the further in vivo experiments to confirm your presumptions about the mechanisms of the pathogenic effect of the tested drug?

7

A 61-year-old female patient had been suffering from arterial hypertension many years. During the last 2 years she began to feel cold intolerance in his legs, numbness and pain in the gastrocnemius muscles on walking and then at rest. These symptoms were more intense at night and caused sleep disturbances. Six months ago she developed a skin lesion on the front surface of her right shin; later it

transformed into ulcer. The ulcer was painless but resistant to therapy. During the visit to the doctor the patient complained of dryness in the mouth, constant feeling thirsty, frequent urination. On examination: the skin of the right shin was dry, pale, and cold; no pulsation of the sole artery was detected. Repeated blood tests showed increased levels of cholesterol, fibrinogen; high platelet counts; glucose 20-25 mM. The urine is positive for ketone bodies and glucose.

- What forms of pathology can be observed in the patient?
- Name the likely type of lesion affecting the patient's arteries? Name and characterize the main mechanisms of pathology underlying changes in the patient's arterial wall.
- What factors caused the development of erosion and ulcer on the patient 's shin?
- Can you assume the presence of the microcirculatory disorders in the vascular system of the patient's right leg? What are the likely causes of these disorders?

8

A 34-year-old female patient who had been suffering from diabetes mellitus for more than 13 years presented with visual losses manifested is blurring of vision, «sand in the eyes» when reading small prints. Dr. examination: a significant decrease in vision acuity, narrowing of several visual fields in both eyes; irregular thickening of the retinal – microvessels wall; capillary microaneurisms and mural thrombi in the retinal microvessels; focal serous exudates and hemorrhages in the retina, ir.d its neovascularization.

The doctor informed the patient that her vision impairment was the result the diabetic microangiopathy. The latter includes pathological alteration of the orbital microvessel wall. The patient received appropriate advice and treatment.

- What types of microcirculatory disorders can you distinguish in the patient's retina?
- Which form of microcirculation disorders may give rise to focal retinal edema? Can the microcirculatory changes described produce vision impairment in the patient?

9

A 19 year old male, body weight 80 kg, was suddenly com-

plaining of fever during his work and ordered home to bed. The patient was living alone. Fortunately a colleague visited him the next morning. He had to break the door down and found the patient unconscious. The patient arrived at the hospital in deep coma. The urine contained glucose and ketone bodies.Explain the condition of the patient concerning thermo-balance, carbohydrate metabolism.

10

A 23-year old male was saved after 30 days in the ruins of a house following earth quake. There was no food but sufficient water. At the arrival to the hospital the patient was in syncope with frequent, deep respiration, and the expired air smelled of acetone. The skin was dirty with brown pigmentation. The cardiac rate was 85 bpm, and the arterial blood pressure was 11.3/7.3 kPa (85/55 mmHg).

The blood [glucose] was 2.2 mM, and the plasma [FFA] was increased. The serum concentrations of proteins and essential amino acids were reduced. The blood [haemoglobin] was 95 g l^{-1}. There was moderate antidiuresis with ketonuria with signs of water retention and a high nitrogen loss in the urine.

The patient was treated with parenteral administration of glucose, amino acids and electrolytes. Following the glucose intake, the blood [glucose] was increased to 10 mM, and glucosuria occurred. A glucose tolerance test was performed and resulted in a high blood [glucose] level that had not reached the normal level within 2 hours.

- Describe the energetic events leading to survival.
- Why did the patient smell of acetone?
- What happened to the carbohydrate metabolism of the patient?
- Explain the high nitrogen loss in the urine.

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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 it complications.

QUESTIONS:

1. Biological role of lipids in the human body.
2. Typical forms of disorders of lipid metabolism.
3. Causes and consequences of lipid digestion disturbances. Symptoms. Steatorrhea.
4. Causes and consequences of lipid intermediary metabolism.
5. Pulmonary and hepatic role in lipid metabolism.
6. Plasma lipoprotein's composition and f their unctions. Apoproteins.
7. Characteristics and mechanisms of hypolipidemia.
8. Hyperlipidemias. Types (classification) according to Fredrickson. Causes, mechanisms and consequences of hyperlipidemias.
9. Atherosclerosis: description and pathological features.
10. Pathogenesis of atherosclerosis. Atherogenic and antiatherogenic lipoproteins. Stages of atherogenesis:initiation; formation and evolution of atheroma;complications.
11. Risk factors of atherosclerosis development.
12. Obesity. Types and pathogenesis of obesity. Causes and consequences.
13. Hepatic lipoid infiltration and dystrophy. Causes and consequences.

Tasks

1

A 13-year-old boy visited a doctor with complaints of recurrent pain in tire region of the heart. The pain was exacerbated at exertion. Examination of rhe patient showed the presence of small, firm, yellow nodules (xanthomas) over the course of the hand muscles tendons, the presence of corneal lipid ircus. Angiographic examination revealed the coronary artery stenosis. Blood tests results demonstrate: total plasma cholesterol – 22 mM, triglycerides – 1,7 mM, HDL-cholesterol – 0,7 mM (normal values: >0,9 mM), LDL-cholesterol – 9 mM (normal values: 3.0-4.5 mM). The patient's parents also have increased plasma levels of cholesterol – more than 8mM. Immunocytochemical analysis of the patient's leukocytes revealed abnormality of the LDL-receptor.

- What type of hyperlipidemia is observed in the patient?
- Is heredity important in the development of this pathology? If it is, what is its type of inheritance? What is the prevalence of this pathology in general population?
- What mechanisms underlie hyperlipidemia in this patient?
- Assess the atherogeneicity of the patient's plasma by calculating the index of atherogeneicity.
- What are the pathological consequences of hyperlipidemia of this type? What therapeutic approaches may be used to treat this pathology?

2

During examination of a 5-year-old boy a physician found an enlargement of the liver and spleen (hepatosplenomegaly), swelling of the tonsils which had red-orange color, and an enlargement of inguinal, axillary, and other superficial lymph nodes. Also clouding of the cornea was observed.

Biochemical tests revealed: total cholesterol of the blood plasma 0,1 mM, triglycerides – 2,3 mM, HDL fraction almost absent. During electrophoretic separation of the patient's plasma apoproteins A-I and A-II bands were not observed.

- What type of disorders of lipid metabolism is observed in the

patient?

• What mechanisms underlie the development of this disorder?

• Why is HDL fraction considered antiatherogenic? What factors determine HDL levels in the blood plasma?

• What role do apoproteins of the A class play in the cholesterol metabolism?

3

During experimental studies of atherosclerosis one group of rabbits was fed cholesterol added to a standard pelleted food in a dose of 5 g per day. Cholesterol was previously purified to remove contaminating oxides. The sec end group was treated similarly, but cholesterol was not purified, and the third group received the same diet with cholesterol that was previously subjected to peroxidation by hydrogen peroxide in the presence of ferrous chloride ($FeCl_2$).

Six months after the start of the experiment the rabbits were killed by a lethal dose of a narcotic drug; the abdominal portion of the aorta and coronary arteries were isolated and their internal surface were examined for the presence of lesions. Simultaneously blood samples were obtained from all rabbits of the experimental groups; plasma was separated from formal particles and used to isolate LDL fraction. In the following in vitro studies samples of the isolated LDL fractions from various groups were added to a media containing cultures of murine peritoneal macrophages, and the extent of intracellular accumulation of cholesterol esters was assessed following incubation for three lays. LDL fraction of the intact rabbits was used as a control.

• Which group of the rabbits is supposed to incur the maximum damage to their arteries due to the excess of cholesterol in the food?

• Describe the pathways by which LDL can enter peripheral cells, including macrophages.

• What differences in accumulation of lipids in murine macrophages can be expected with LDL fraction isolated from various experimental animals? Compare LDLs of the control and experimental groups of rabbits.

• What role do monocytes/macrophages play in atherogenesis?

4

During his visit to the doctor a 55-year-old male patient complains of anginal pain which recently has increased in frequency and intensity. The raiient is the president of a big trading company. During the last months he ra > experienced great psychological stress, spent 12-14 hours at his office daily and slept little; he began smoking more than one pack of cigarettes per day. The patient is a hearty eater, preferring high-calorie fat food. His father died of myocardial infarction. On examination: his height is 173 cm, weight 89 kg, blood pressure 175/100 mm Hg. Blood tests show the following: total plasma cholesterol – 6,8 mM, triglycerides – 1,9 mM. HDL cholesterol 0.8 mM, LDL cholesterol – 5,3 mM; glucose tolerance is decreased.

- What form of pathology is the cause of angina pectoris in this patient?
- What risk factors of this pathology are observed in the patient?
- Does the patient have signs of lipid metabolism disorder? If he does, what forms of it?
- What therapeutic approaches can be used to treat lipid metabolism disorders in this case?

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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:

1. Biological role of proteins, peptides and amino acids. Consequences of amino acid insufficiency.
2. Starvation. Types of starvation. Metabolic and functional disturbances in starvation.
3. Causes of protein insufficiency. Protein-calorie malnutrition. Particularities in children (Kwashiorkor).
4. Causes and consequences of protein digestion disturbances. Symptoms. Celiac-sprue.
5. Causes and consequences of intermediary amino acid metabolism.
6. Pathology of plasma protein composition. Disproteinemia types and their features.
7. Types of residual plasma nitrogen level increasing. Mechanisms of their development.
8. Gout. Pathogenesis of symptoms.

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LESSON № 11

Topic: DISORDERS OF WATER-ELECTROLITE METABOLISM

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

QUESTIONS:

1. Water-electrolite balance. Its regulation.
2. Classification of water-electolite disturbances.
3. Negative water-mineral balance. Hypo-, iso- and hyperosmolaric types of dehydration. Causes, mechanisms of development, manifestations and consequences.
4. Positive water-electrolite balance (overerhydration). Types, causes, mechanisms of development, manifestations, consequences, and principles of treatment.
5. Water poisoning. Causes, symptoms and consequences. Therapy.
6. Edema. Classification. Factors of edema development.
7. Pathogenesis of cardiac, renal, hepatic, inflammatory, allergic, toxic edemas.
8. Disorders of sodium, potassium, calcium, manganese, and phosphate metabolism: causes, manifestations, and mechanisms of development.
9. Disturbances of microelements metabolism (J, F, Mn, Se, Cu etc).

Tasks

1

Examination of a 32-year-old patient revealed various signs of pathology, .rciuuing excessive body mass: his height is 168 cm and weight 84.5 kg. The patient also has a pasty face, periorbital puffiness, pale skin; he had slow rebound of tissue to its original contour after pressing the feet or shin with the fingertip. The patient told the physician about tightness of a ring and shoes m me evening. The in-

vestigation of the cardiovascular system revealed the following: minor arterial hypotension, areas of cardiac dullness are slightly increased: other parameters are unremarkable. The daily urine volume is withing the normal range.

- What is the possible cause of the patient's excess of body mass?
- C an we assume that water-ions balance is deranged in this patient?
- What type of edema is observed in the patient?
- What additional data are required to specify the type of edema in this case?

2

A 42-year-old patient has been admitted to hospital with a diagnosis of uncompensated chronic heart failure due to valvular disease. The patient has normal constitution with paucity of subcutaneous tissue. His height is 165 cm, body weight 81 kg. On examination: the patient needs to sit in bed; he has dyspnea, acrocyanosis, marked lower extremities edema, rales and wheezes during auscultation of the chest. An X-ray investigation of the abdominal area shows an accumulation of fluid: the liver is enlarged; stroke volume and cardiac output are decreased: hematocrit 38%; the daily urine volume is decreased. Biochemical tests reveal an increased plasma activity of renin and an increased sodium concentration.

- Are there any signs of derangement of water balance in this patient?
- What type of dyshydria is observed in this case?
- Is there any association between the accumulation of fluid in the subcutaneous tissue, the abdomen, and the lungs?
- Explain pathogenesis of increased blood levels of renin and Na~ in this patient.
- Explain pathogenesis of edema in this patient.
- Explain the role of edema in deterioration of the patient's condition.
- What therapeutic approaches can be used to treat edema in this case?

3

A 22-year-old patient who recovered from severe scarlet fever two weeks ago complains of headache, pain in the back, dyspnea, and palpitations. During the last week she has increased her body weight

by 11,5 kg. On examination: her face is pale; she has periorbital puffiness and edema of the shins and feet; the boundaries of the heart dullness are increased: blood pressure is 180/100 mm Hg; the daily urine volume is reduced. Urine tests show the presence of erythrocytes and protein. An increased titer of antistreptolysin O antibodies is found in the blood.

- Is there evidence of the kidney damage in this patient? What is the possible mechanism of this pathology?
- What is the cause of hyperhydration in this case: a decrease in water excretion or an increase in water retention?
- Explain the mechanisms of edema in this patient.

4

A 7-year-old boy developed a progressive swelling of the soft palate with a swallowing difficulty, and then asphyxia after he had drunk mango juice. The mucosal membrane in the swelled area is hyperemic without tenderness; a moderate increase in eosinophils is seen in the blood. The patient's body temperature is normal. His senior sister suffers from attacks of bronchial asthma.

- Is edema in this case the result of ordinary inflammation?
- What is the cause of edema in this patient?
- Explain the pathogenesis of the given pathology.
- Does this type of edema lead to life-threatening condition?

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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:

1. Mechanisms of acid-base balance maintaining. Role of buffer systems, lungs and kidney in the acid-base balance regulation.
2. Parameters of acid-base balance and their importance. Siggart-Andersen nomograms.
3. Classification of acid-base disturbances. Compensatory and decompensatory, absolute and relative, respiratory and metabolic disorders.
4. Causes and parameters of acid-base balance in:
   a. respiratory acidosis;
   b. metabolic acidosis;
   c. respiratory alkalosis;
   d. metabolic alkalosis.
5. Consequences of acid-base disturbances. Clinical manifestations of alkalosis and acidosis.

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:

pH = 7,47

pCO_2 = 33,4 mm Hg

HCO_3^- = 20 mM

BB = 41,4 mM

BE = 2,0 mM

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:

pH = 7,48

pCO_2 = 53,4 mm Hg

HCO_3^- = 31 mM

BB = 55 mM

BE = 5 mM

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.

pH = 7,28

pCO_2 = 31,4 mm Hg

HCO_3^- = 21 mM

BB = 36,4 mM

BE = -8,5 mM

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

4

The patient with bronchial athma 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^- = 31 mM

BB = 44,4 mM

BE = -2,5 mM

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

5

pH – 7,18

$pC0_2$ – 75 mm Hg

SB – 24 mM

BE – +2,5 mM

The patient is undergoing surgical operation with cardiopulmonary bypass.

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

6

pH– 7.25
$pC0_2$ – 52 mm Hg
SB – 22 mM
BB – 45 mM
BE – +2 mM

The patient has a bronchial asthma attack. Make a conclusion about the type of acid-base disorder.

7

pH –7.20
$pC0_2$ – 32 mm Hg
SB – 14 mM
BB – 29 mM
BE – -12 mM

The patient is undergoing surgical operation with cardiopulmonary bypass. Make a conclusion about the type of acid-base disorder.

8

pH –7.36
$pC0_2$ – 31 mm Hg
SB – 19.5 mM
BB – 39 mM
BE – -5 mM
Ketone bodies in blood –10 mM
Titratable acidity in urine – 37 mmol/d (N=10-30 mmol/d)

The patient suffers from diabetes mellitus. Make a conclusion about the type of acid-base disorder.

9

pH –7.30
$pC0_2$ – 32 mm Hg
SB – 18 mM
BB – 36 mM
BE – -6 mM
Lactic acid in blood – 10 mM

The patient is admitted to hospital with a preliminary diagnosis of an acute myocardial infarction. Make a conclusion about the type of acid-base disorder.

10

pH – 7.30
$pC0_2$ – 35 mm Hg
SB – 16.5 mM
BB – 35 mM
BE – -9 mM
Titratable acidity – 8 mmol/d
NH_4^+ – 17 mmol/d (N = 30-50 mmol/d)

The patient suffers from an acute glomerulonephritis. Make a conclusion about the type of acid-base disorder.

11

pH – 7,22
$pC0_2$ – 32 mm Hg
SB – 14 mM
BB – 24 mM
BE – -8 mM

The patient has intestinal fistula; he has been losing intestinal juice for a long period of time. Make a conclusion about the type of acid-base disorder.

12

pH – 7.22
$pC0_2$ – 27 mm Hg
SB – 18.5 mM
BB – 40.5 mM
BE – -7 mM
Lactic acid – 28 mg/dL
Titratable acidity in urine – 8 mmol/d
NH_4^+ in urine – 15 mmol/d

The patient suffers from severe toxic injury of the liver and kidneys with oligouria. Make a conclusion about the type of acid-base disorder.

13

pH –7.11
$pC0_2$ – 24 mm Hg
SB – 15.5 mM
BE – -13 mol/L
BB – 38 mM
Ketone bodies of the blood – 33 mM

Titratable acidity urine – 70 mmol/d

The patient has hepatic coma. Make a conclusion about the type of acid-base disorder.

14

pH – 7.17

$pC0_2$ – 51 mm Hg

SB – 24 mM

BB – 45 mM

BE – 2,5 mM

The patient suffers from an acute left ventricular insufficiency with lungs edema. Make a conclusion about the type of acid-base disorder.

15

pH – 7.52

$pC0_2$ – 25 mm Hg

SB – 21 mM

BE – -2 mM

BB – - 44 mM

The patient is undergoing surgery with mechanical ventilation. Make a conclusion about the type of acid-base disorder.

16

pH – 7.51

$pC0_2$ – 56 mm Hg

SB – 29 mM

BB – 57 mM

BE – +5.5 mM

The patient suffers from uncontrolable vomiting. Make a conclusion about the type of acid-base disorder.

17

pH – 7.59

$pC0_2$ – 50 mm Hg

SB – 30 mM

BE – +5 mM

BB – 56 mM

The patient with brain contusion has cyclic vomiting. Make a conclusion about the type of acid-base disorder.

18

pH – 7.27
$pC0_2$ – 25 mm Hg
SB – 11 mM
BB – 27 mM
BE – -17 mM
Hematocrit – 0.36

The patient had an acute blood loss. Make a conclusion about the type of acid-base disorder.

19

pH – 7.33
$pC0_2$ – 35 mm Hg
SB – 18 mM
BB – 42 mM
BE – -5.5 mM
Titratable acidity urine – 12 mM
Hematocrit – 0.3

The patient has been admitted to hospital with a diagnosis of hemolytic anemia of unclear etiology. Make a conclusion about the type of acid-base disorder.

20

pH – 7.57
$pC0_2$ – 28 mm Hg
SB – 24 mM
BE – +2,5 mM
BB – 48 mM
Titratable acidity of urine – 20 mmol/d

The patient has had the hysteria hyperventilation episode just before the analysis. Make a conclusion about the type of acid-base disorder.

21

pH – 7,09
$pC0_2$ – 51 mm Hg
SB – 15 mM
BE – -12.5 mM
BB – 38 mM
Lactic acid (blood) – 15 mM

Titratable acidity of urine – 18 mmol/d

The patient with diabetes mellitus in coma. Make a conclusion about the type of acid-base disorder.

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2. Litvitsky P.F., Pirozhkov S.V., Tezikov E.B. Pathophysiology: Concise Lectures, test, clinic-pathophysiological situations and clinic-laboratory problems. Students manual / Moscow «Geotar-Media», 2012. – P. 56-61.

3. General and systematic pathology / ed. by J. C.E. Underwood. 2nd ed. – 1996. – P. 150-152.

PATHOLOGY OF VITAMINES METHABOLISM

Deficiency of water-soluble vitamins (B_1, B_2, B_6, B_{12}, C, PP).
Deficiency of fat-soluble vitamins (A, D, E, K).

LITERATURE:

1. Lecture material.
2. General and systematic pathology / ed. by J. C.E. Underwood. 2^{nd} ed. – 1996. – P. 153-155.

LESSON № 13

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:

1. Types of cell injuries. Causes and main mechanisms of cell damage.
2. The main etiopathogenic variants of cell injury. Ischemic and hypoxic injury. Free radical-induced injury.Toxic injury.
3. Reversible and irreversible ischemic/hypoxic cell injury.
4. Causes and consequences of cell energy production disturbance.
5. Changes in the cellular genome or disorders of its realization.
6. Membrane damage. Oxidative stress as general mechanism of cell injury. Mechanisms of antioxidative defense.
7. Ionic distribution in extra- and intracellular space. Role of ions in the cell function during pathological conditions.
8. The mechanisms of transcellular communication (eicosanoids, hormones and cellular growth factors).
9. Disorders of intracellular regulatory mechanisms. Role of calcium in cell function and injury.
10. Derangement of the subcellular structures and components. Consequences of organelles injury (membrane, nuclear, mitochondria, ribosome, ect.).
11. Dystrophy.
12. Dysplasia.
13. Necrosis as general mechanism of accidental cell death. Role of calcium, lysosomal ferments and reactive oxygen spaces in necrosis development.
14. Apoptosis. The mechanisms of initiation of apoptosis. Stages of apoptosis. Consequences of apoptosis disturbance.
15. Comprising characteristic of apoptotic and necrotic cell death.
16. Mechanisms of cell defense against damaging factors. Reparative processes in injured cells.

Tasks

1

A technician of the chemical laboratory was not careful working with a toxic volatile chemical. He dropped a flask containing the toxic substance and smashed it. Before he left the room, he had inhaled noxious vapours of the chemical. Two days later he was admitted to hospital with the following complaints: malaise, somnolence, headache, nausea, back pains, blood in the urine. Blood analysis: erythrocytes $2.7x10^{12}$/l, Hb 80 g/l, platelets $120xl0^{9}$/l, leukocytes $3,1xl0^{9}$/l; compensated acidosis (metabolic and renal). The resuls of the special blood biochemistry analysis: an increased concentration of free fatty acid, lipid hydroperoxides, and adenosinephosphate; an elevated total creatinephosphate kinase (CPK) activity and potassium content.

- One of the consequences of the patient's poisoning is a significant depletion of cells in the peripheral blood. What are the possible mechanisms of this effect?
- According to the results of the special blood tests what mechanisms of the cell injury can you propose? Which of them caused damage to the membrane of the formal particles? Explain your point of view.
- What are the origin and consequences of acidosis in this case?
- Why did the signs of intoxication develop in the patient not immediately but two days after the incident?

LITERATURE:

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2. General and clinical pathophysiology / ed. by A.V. Kubyshkin – Vinnytsa: Nova Knyha Publishers. – 2011. – P. 134-164.
3. Litvitsky P.F., Pirozhkov S.V., Tezikov E.B. Pathophysiology: Concise Lectures, test, clinic-pathophysiological situations and clinic-laboratory problems. Students manual / Moscow «Geotar-Media», 2012. – P. 19-22.
4. Pathology / ed. by E. Rubin and J.L. Farber, 2nd ed. – 1994. – P. 1-32.

5. General and systematic pathology / ed. by J. C.E. Underwood. 2nd ed. – 1996. – P. 111-137.

CONTROL QUESTIONS TO PATHOPHYSIOLOGY OF METABOLISM AND CELL PATHOLOGY

1. Starvation. Types of starvation. Metabolic and functional disturbances in starvation. Principals of starvation therapy. Therapeutic starvation.

2. Consequences of total and partial starvation (carbohydrate's, lipid's and protein deficit). Protein-calorie malnutrition. Particularities in children (Kwashiorkor).

3. Causes and consequences of carbohydrate digestion disturbances. Symptoms. Lactase deficiency.

4. Causes and consequences of lipid digestion disturbances. Symptoms. Steatorrhea.

5. Causes and consequences of protein digestion disturbances. Symptoms. Celiac-sprue.

6. Hyperglycemia. Types. Consequences.

7. Hypoglycemia. Types. Consequences.

8. Causes and consequences of carbohydrate intermediary metabolism.

9. Causes and consequences of lipid intermediary metabolism.

10. Causes and consequences of intermediary amino acids metabolism.

11. Diabetes mellitus. Etiology. Types and their particularities.

12. Pathogenesis of diabetes mellitus. Main metabolic disturbances.

13. Clinical symptoms of diabetes mellitus and mechanisms of their development.

14. Complications of diabetes mellitus. Types of comas and there pathogenesis.

15. Diabetic vascular complications. Pathogenesis of atherosclerosis, diabetic nephropathy, diabetic retinopathy, peripheral neuropathy.

16. Pulmonary and hepatic role in lipid metabolism.

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

18. Hyperlipidemias. Types (classification). Causes and consequences.

19. Obesity. Types. Causes and consequences.

20. Hepatic lipoid infiltration and dystrophy. Causes and consequences.

21. Pathogenesis of atherosclerosis. Atherogenic and antiatherogenic lipoproteins. Risk factors of atherosclerosis development.

22. Biological role of proteins, peptides and amino acids. Consequences of amino acid insufficiency.

23. Causes of protein insufficiency. Consequences.

24. Pathology of plasma protein composition. Disproteinemia types and features.

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

26. Gout. Pathogenesis. Symptoms and therapy.

27. Water-mineral balance. Its regulation. Classification of water-mineral disturbances.

28. Negative water-mineral balance. Hypo-, iso- and hyperosmolaric types of dehydration. Causes, symptoms and consequences. Therapy.

29. Positive water-mineral balance. Types of hyperhydration. Water poisoning. Causes, symptoms and consequences. Therapy.

30. Edema, classification. Factors leading to edema development.

31. Pathogenesis of cardiac, renal, hepatic, inflammatory, allergic, toxic edema.

32. Disturbances of mineral metabolism.

33. Classification of acid-base disturbances. Acidosis and alkalosis. Features.

34. Parameters of acid-base balance at respiratory and metabolic acidosis.

35. Parameters of acid-base balance at respiratory and metabolic alkalosis.

36. Deficiency of water-soluble vitamins (B_1, B_2, B_6, B_{12}, C, PP).

37. Deficiency of fate-soluble vitamins (A, D, E, K).

38. Cell pathology. Causes and consequences of nuclear, mitochondria, reticulum, cell membrane and lysosomal injury.

39. Reactive oxygen species and their role in the cell membrane damages. Mechanisms of lipid peroxidation.

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.
2. Definition of hypoxia. Classification. Acute and chronic hypoxia. Exogenous and endogenous hypoxia.
3. Causes and changes of blood gas parameters (P_a0_2, P_v0_2, P_{50}, oxygen blood capacity, C_a0_2, C_v0_2, $C_{a-v}0_2$, S_a0_2, S_v0_2, , P_aC0_2 , P_vC0_2,) in:
   a. hypoxic hypoxia;
   b. hyperoxic hypoxia;
   c. respiratory hypoxia;
   d. blood-depended hypoxia;
   e. circulatory hypoxia;
   f. histotoxic (tissue) hypoxia;
   g. hypoxia of owerload (overutilization);
4. Conditions that determine the susceptibility of cells to hypoxia.
5. Signs of acute hypoxia.
6. Acute and long-term adaptation compensatory mechanisms of hypoxia.
8. Conditions that determine the susceptibility of cells to hypoxia.
9. Hyperoxia.

Tasks

Make a conclusion about blood gas values

№ 1	№ 2
$P_{atm}O_2$ – 160 mm Hg	$P_{atm}O_2$ – 158 mm Hg
P_{alv} O_2 – 105 mm Hg	P_{alv} O_2 – 105 mm Hg
PaO_2 – 96 mm Hg	PaO_2 – 95 mm Hg
PvO_2 – 60 mm Hg	PvO_2 – 38 mm Hg
$PaCO_2$ – 30 mm Hg	$PaCO_2$ – 40 mm Hg
SaO_2 – 98 %	SaO_2 – 70%
SvO_2 – 91%	SvO_2 – 20%
Pulmonary min. volume – 7.3 l/min	MetHb – 40%

Circulation min. volume – 6.9 l/min
pH – 7.31
Lactic acid – 26.5 mg/dl

№ 3

$P_{atm}O_2$ – 150 mm Hg
P_{alv} O_2 – 94 mm Hg
PaO_2 – 76 mm Hg
PvO_2 – 21 mm Hg
$PaCO_2$ – 48 mm Hg
SaO_2 – 90%
SvO_2 – 32%
Pulmonary min. volume – 4,6 l/min
Circulation min. volume – 6,4 l/min
pH – 7.31
Lactic acid – 25 mg/dl

№ 5

$P_{atm}O_2$ – 105 mm Hg
P_{alv} O_2 – 55 mm Hg
PaO_2 – 40 mm Hg
PvO_2 – 12 mm Hg
$PaCO_2$ – 58 mm Hg
SaO_2 – 67%
SvO_2 – 11%
Pulmonary min. volume – 4,5 l/min
Circulation min. volume – 3,4 l/min
pH – 7,28

Pulmonary min. volume – 8.8 l/min
Circulation min. volume – 7.0 l/min
pH – 7.3
Lactic acid – 20.5 mg/dl.

№ 4

$P_{atm}O_2$ –158 mm Hg
P_{alv} O_2 – 105 mm Hg
PaO_2 – 96 mm Hg
PvO_2 – 18 mm Hg
$PaCO_2$ – 28 mm Hg
Hb – 40g/l
SaO_2 – 95%
SvO_2 – 27%
Pulmonary min. volume – 8,8 l/min
Circulation min. volume –2,9 l/min
pH – 7,31
Lactic acid – 26,5 mg/dl

№ 6

$P_{atm}O_2$ –158 mm Hg
P_{alv} O_2 – 88mm Hg
PaO_2 – 61 mm Hg
PvO_2 –16 mm Hg
$PaCO_2$ – 59 mm Hg
SaO_2 – 88%
SvO_2 – 25%
Pulmonary min. volume – 2,85 l/min
Circulation min. volume – 8,5 l/min
pH–7,25
Titratable acidity of the daily urine – 60 mmol/d
Hb –140 g/l

LITERATURE:

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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.

QUESTIONS:

1. Base of congenital function. Cariotype, genotype, phenotype. Penetrance and expressivity. Factors determining penetrance and expressivity.
2. Congenital diseases.
3. Etiology and pathogenesis of congenital diseases.
4. Mutations and mutates (alcohol, nicotine, radiation e.g.).
5. Categorization of congenital diseases.
6. Genome diseases. The concept of chromosome disorders, their mechanism. Mosaicism. Types of chromosome aberrations: deletion, balanced and Robertsonian translocation, ring chromosome, inversion.
7. Types of genome diseases trait. Sex-chromosomes and autosomes related diseases.
8. Examples of the most common chromosome disorders. Monosomies and trisomies.
9. Biochemical and molecular basis of single-gene (Mendelian) disorders.
10. Features of disorders and examples of disorders transmitted by
    - autosome-dominant trait;
    - autosome-recessive trait;
    - sex chromosome-linked (X-linked) trait
11. Gene diseases:
    - metabolic diseases (glycogenosis, phenylketonuria, galactosemia e.g.);
    - blood diseases (hemoglobinosis S, elliptocytosis, hemophilia e.g.).

12. Investigative methods for congenital diseases:
    - genealogical;
    - population;
    - statistical;
    - cytogenetical;
    - biochemical;
    - dermatoglyphical.

13. Features of multifactorial (polygenic) disorders. The most common multifactorial disorders.

14. Diseases of congenital supports.

15. Embryopathic and fetopathic disorders. The critical periods of pregnancy. Phenocopies.

16. Medical genetic consulting.

17. Constitution. Types of constitution.

Tasks

1

N., a healthy woman, visited a genetic counseling unit for consultation. She told her doctor that her father suffered from color blindness, but her mother had no vision problems. N. was anxious about the risk of color blindness to her future children.

What are the manifestations of color blindness and the pattern of its inheritance?

Can N. carry the gene of color blindness in her genotype?

What is the probability of disease and the probability that her children (separately boys or girls) will be the carriers of the abnormal gene?

2

N., a healthy woman, whose father suffered from hemophilia A and mother was healthy, went to a genetic counseling unit. She was anxious about the risk of hemophilia to her grandsons. Her husband, as well as her son and two daughters were healthy.

What is the pattern of inheritance of hemophilia A? What are the main features of this type of inheritance?

What is the risk of the disease descending from her son to her grandson and from her daughter to the grandchildren?

What are the etiology and pathogenesis of hemophilia A?

Can this disease have lethal and sublethal forms?

3

Patient S., a pregnant woman, went to a genetic counseling unit for consultation. She told her doctor that her sister suffered from phenylketonuria. In her husband's pedigree there are marriages between close relatives, but none of the children have had phenylketonuria. A thorough examination of the patient and her husband revealed no signs of pathology.

How great is the risk of phenylketonuria to patient S.'s sons?

What are the etiology and basic mechanisms of phenylketonuria? Is gender significant for its inheritance?

What are the main manifestations of the disease and their pathogenesis?

What is the approach to early diagnostics of phenylketinuria in the newborns?

Is it possible to prevent phenylpyruvate oligophrenia in children?

4

A male patient, 3 years old, was admitted to the pediatric department. On examination: signs of growth retardation, flat facial profile, half-open mouth, oblique palpebral fissures and epicanthic folds are evident; transverse skin folds on the palms. A study of the patient's karyotype revealed the following: 46, XY, + t (+14, 21).

What disease does the patient suffer from?

Describe and characterize the patient's karyotype? By what features does it differ from the normal one?

What are the possible causes and pathogenesis of this disease?

Are other variants of karyotype modification possible in this disease? Which of the variants is the most common?

5

Patient M., male, 21 years old, suffers from hereditary form of hypogammaglobulinemia. His father also suffers from this disease; mother is healthy. An examination showed a significant decrease of IgM and IgG levels in the patient's blood.

What is the pattern of its inheritance?

Characterize the genotype of the patient's mother with respect

to this pathology, and also the possible genotypes of the patient's siblings. How much is the risk for the patient's sibs to develop hypogammaglobuliemia?

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5. Pathophysiology/ ed. by C. Paradiso (Lippincott's review series). – 1995. – P. 383-394.
6. Pathophysiology of disease: an introduction to clinical medicine / ed. by S. J. McPhee, W.F. Ganong. – 2006. – P. 2-32.

LESSON № 16

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

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

QUESTIONS:

1. Pathogenic action of environmental factors. Pathogenic physical factors. Mechanical Factors. Local sequelae of mechanical injury.
2. The general effects of mechanical injury.
3. Effects of electric energy.
4. Stress. General adaptation syndrome. Theory of stress (H. Selye, 1938).
5. Pathophysiology of stress. The role of sympatoadrenal and hypothalamohypophysadrenal systems in stress.
6. Stress stages.
7. Distress syndrome.
8. Shock. Definition.
9. Shock. Types, etiology, stages of shock and their mechanisms, manifestations.
10. Major types of shocks. Hypovolemic, cardiogenic, distributive (septic, neurogenic, anaphylactic) shock.
11. Mechanisms of compensation and decompensation during shock.
12. Collapse: characteristics, types, etiology, pathogenesis, manifestations, principles of treatment.
13. Differences between collapse and shock.
14. Coma. Definition. Causes and types, general pathogenesis, manifestations.
15. Terminal states (preagony, agony, clinical and biological mors).
16. Pathophysiological fundamentalses of reanimation
17. Postreanimation distress.

LITERATURE:

1. Lecture material.
2. General and clinical pathophysiology / ed. by A.V. Kubyshkin. – Vinnytsa: Nova Knyha Publishers. – 2011. – P. 641-651.
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4. Pathophysiology / ed. by C. Paradiso (Lippincott's review series). – 1995. – P. 77-90.
5. Selye H., Experimental evidence supporting the conception of «adaptation energy», Am. J. Physiol. – 1938. – V. 123. – P. 758-765.

LESSON № 17

Topic: TUMOR GROWING

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

QUESTIONS:

1. The tumor growth. Malignant and benign tumors. Description.
2. Kinds of tumor atypism: morphological, biochemical, physicochemical, antigenic, functional.
3. Etiology of tumors. Carcinogenes and its types. Theories: radioactive, chemical, viral, genetic.
4. The concept of oncogenes. Mechanisms of transformation of protooncogene to oncogene.
5. Pathogenesis of tumor growth. Stages: initiation (transformation), promotion, progression.
6. Tumor influence on the organism. The concept of tumor progression. Tumor disease. Pathogenesis of cancer cachexia.
7. Antineoplastic mechanisms. Principles of prevention and treatment of neoplastic diseases.

Tasks

1

A 48-year-old patient N. who has been smoking for 25 years works at the chemical plant where he deals with dyes and organic solvents. He visited his primary care physician with complaints of

malaise, weakness, decreased appetite, and persistent cough without secretions. During the previous three weeks he noted the presence of blood in the urine, burning sensation in the lower abdomen aggravated by urination. The results of computer tomography scanning. X-ray, and ultrasonography attest against renal or ureter pathology in the patient. Cystoscopy shows proliferation of the bladder's mucosa with erosion of its surface. Biopsy of the lesion in the bladder shows the presence of malignant cells.

What factors could be a potential cause of cancer of the bladder mucosa in this case? Substantiate your answer.

Characterize stages of carcinogenesis after the first contact of the bladder mucosa with carcinogen and up to the emergence of the tumor cells.

Can you exclude the possibility of metastases of the lung cancer in the bladder?

2

Seven months after a patient had undergone a surgical removal of the srrmach carcinoma and completed a course of chemotherapy he presented an enlargement of a supraclavial lymph node. Biopsy of the swelled r-mph node showed the presence of malignant cells. Some of these cells were surilar to the removed tumor cells by their morphology.

Can you attribute the appearance of the tumor cells in the lymph node to the tumor progression phenomenon? Substantiate your answer and characterize the phenomenon of tumor progression (its mechanisms and biological significance).

Do you think that the presence of the tumor cells in the lymph node is the result of the primary tumor metastases?

A multicentered tumor growth?

Recidivation of the stomach carcinoma?

A new tumor growth?

Substantiate your opinion.

What factors of the anticancer defence system were ineffective in this case? What are the possible mechanisms of their action?

3

A 56-year-old patient M., who has been suffering from gastric

atrophy ire hypoacidity for more than 20 years, complains of fatigability, weakness, pain in the epigastrium, a decreased appetite, rapid satiety during meal, nausea, a great loss of body mass during the last four months, and a persistent fever.

Laboratory tests showed anemia, leukocytosis, hypochlorhydria. and a re:reased activity of gastric juice enzymes. Gastroscopy revealed flattening of the mucosal folds in the pyloric area, and the presence of a saucer-like tumor with ulcerative alterations in the center.

Why does chronic gastric atrophy promote the development of a gastric tumor?

Describe these the insufficiency of the antineoplastic mechanisms.

What are the possible causes and mechanisms of fever and anemia in this patient?

What are the possible causes of cachexia in this patient?

4

A 40-year-old patient B. visited his physician with complaints of considerable weakness, dizziness, persistent cough with minor secretions. He had participated in the liquidation of the nuclear power station accident 1,5 years before. B. told the physician that he had been a heavy smoker for 20 years, but 2 years before had quitted smoking. During the last 6 months he repeatedly suffered infectious diseases, such as quinsy, bronchitis, pneumonia. During bronchoscopy a tumor of the main right bronchus was found. Histology of the tumor showed the presence of the squamous epithelium malignant cells.

What factor was the most likely cause of bronchial cancer in this patient?

What factors could potentiate the effect of carcinogens in this patient? What are their possible mechanisms of action?

What antineoplastic mechanisms should have become activated in this patient:

− under the effect of the carcinogens;

− in the course of neoplastic transformation of the bronchial epithelial cell;

− during the formation of the primary tumor nodule?

Why did the antineoplastic mechanisms fail in this case?

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6. General and systematic pathology / ed. by J. C.E. Underwood. 2nd ed. – 1996. – P. 31-61.

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.
3. Mechanisms of radiation inflow on the organism. Water radiolysis, lipid peroxidation and DNA mutations.
4. Clinical types of radiation sickness. Dependence from dose.
5. Pathophysiology of bone marrow form of radiation sickness. It stages.
6. Mechanisms of remote consequences of radiation inflow on the organism.

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SYSTEMIC PATHOPHYSIOLOGY

LESSON № 19

Topic: PATHOLOGY OF CIRCULATING BLOOD VOLUME. BLEEDING

Aim of the lesson: to study the types of circulating blood volume disorders, their causes and mechanisms of development, to study pathogenesis and compensatory mechanisms after acute bleeding.

QUESTIONS:

1. Blood, its composition and functions. Hematocrit.
2. Categorization of disorders of circulating blood volume (hypervolemia, hypovolemia).
3. Hypervolemia. Types (simple, polycythemic, oligocythemic). Causes and outcomes.
4. Hypovolemia. Types (simple, polycythemic, oligocythemic). Causes and outcomes.
5. Erythrocytosis: causes and mechanisms. Polycythemia or Wakes's disease.
6. Bleeding. Types and causes. Pathogenesis and main clinical symptoms of acute bleeding.
7. Compensatory-adaptative reactions of organism at acute bleeding. Stages of compensation (reflectoric, hydremic, bone-marrow initiation).
8. Parameters of severity of bleeding.
9. Factors which affect bleeding outcome.
10. Blood rheological and plasma composition disturbances. Causes and outcomes.

Laboratory work 1. ***Measurment of reticulocyte count***

in the blood using microscope

Description of the work 1. To measure reticulocyte count students use light microscope.

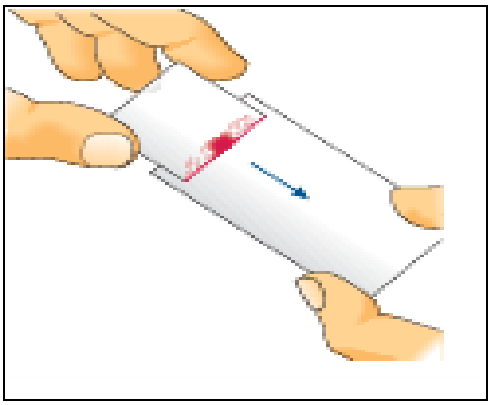

Fig. 15 – Technology of preparation of blood smears

The blood samples with *brilliant creasil blue* paint should be used for investigation. Rt count estimation is perfomed simultaneously with erythrocytes count (1000) in separated area. Rt count reports the number of reticulocytes as a percentage (%) of the number of red blood cells or promille (1‰ = 0,1%). Reticulocytes are newly-produced red blood cells. They are slightly larger than totally mature red blood cells, and have some residual ribosomal RNA. The presence of RNA is visualized as a blue web-like structure.

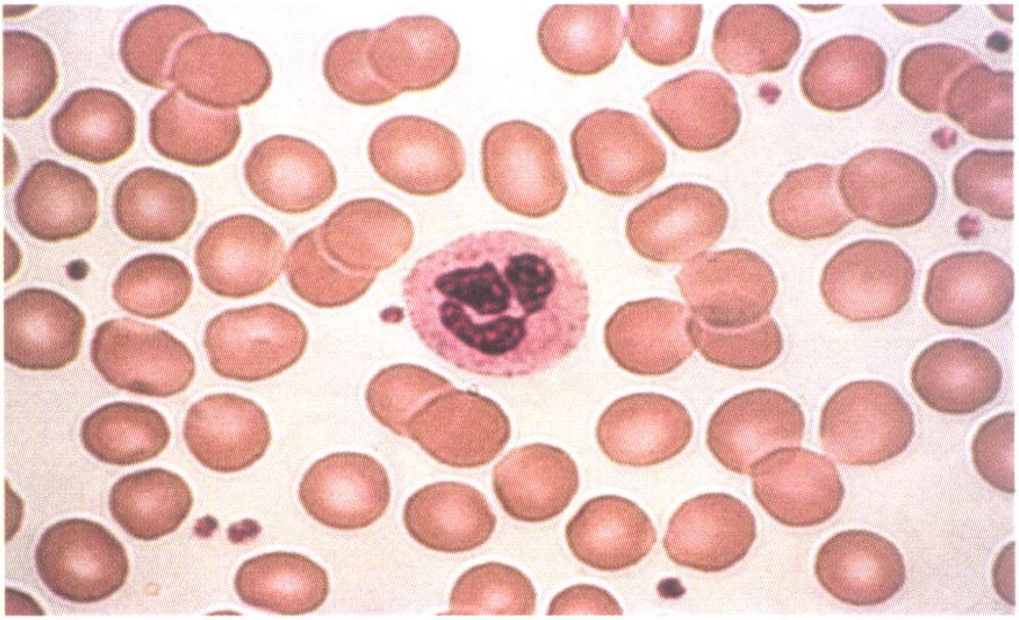

Fig. 16 – The peripheral blood smears

Tasks

1

Patient, 35 years old, male, arrives to the hospital after chest trauma.

Clinical findings: paleness, blood pressure 90/60 mm Hg, weak puls, frequent breath, X-ray chest examination reveals severe darkness in thoracic cavity.

Blood analysis, after 4 days of hemostatic operation: Нв – 71 g/l, RBC – 3 x 10^{12}/l, Rt – 12 %.

Blood sample: a lot of polychromatophills, 2 oxyphilic normocytes.

Make conclusion.

2

The patient, 32 years old man, was arrived to hospital after traffic accident. The bleeding was about 750 ml. Patient body weight is 75 kg, normal constitution. Estimate the severity of bleeding for him. Will you perform some treatment actions?

3

The patient, 28 years old woman, was arrived to hospital after traffic accident. The bleeding was about 750 ml. Her body weight is 55 kg, normal constitution. Estimate the severity of bleeding for him. Will you perform some treatment actions?

4

The patient, 5 years old boy, was arrived to hospital after traffic accident. The bleeding was about 400 ml. His body weight is 17 kg, normal constitution. Estimate the severity of bleeding for him. What treatment actions will you perform?

LITERATURE:

1. General and clinical pathophysiology / ed. by A.V. Kubyshkin. – Vinnytsa: Nova Knyha Publishers. – 2011. – P. 363-371.

2. Pathology / ed. by E. Rubin, J.L. Farber. – 2nd ed. – 1994. – P. 994. – 1010.

3. Internal medicine / ed. by Harrisons. – 17th edition. – N. Y. – 2008. – P. 363-369.

LESSON № 20

Topic: ANEMIAS

Aim of lesson: to study causes and mechanisms of different types of anemias. Analyse changes in blood in patients with different types of anemias.

QUESTIONS:

1. Anemias. Clinical symptoms of anemia and mechanisms of their development.
2. Classification of anemia by:
   - etiopathogenesis:
     - posthemorrhagic (of blood loss),
     - due to the erythropoietic disorders,
     - hemolytic,
   - color parameter (index),
   - severity of anemia,
   - regenerative possibility,
   - mechanism of erythropoiesis,
   - erythrocyte's size.
3. Posthemorrhagic anemia. Description, types, causes, hematological signs of blood in acute and chronic posthemorrhagic anemias.
4. Iron deficiency anemias. Etiology, pathogenesis, manifestations, Hematological signs. Sideropenic syndrome.
5. Megaloblastic anemias (vitamin B_{12}, folic acid deficiency). Etiology, pathogenesis. Manifestations. Hematological signs. Addison-Biermer disease.
6. Aplastic anemia: causes and pathogenesis, hematological signs.
7. Hemolytic anemias. Types (congenital, autoimmune e.g.). Common causes, manifestations, hematological signs. Clinical symptoms.
8. Hereditary spherocytosis: pathogenesis, manifestations, hematological signs.
9. Sickle cell disease: pathogenesis, manifestations, hematological signs.

10. Thalassemia syndromes: types, pathogenesis, manifestations, hematological signs.

11. Newborn hemolytic anemia.

Laboratory work 1. ***Measurment of hemoglobin concentration in the blood by Sali method***

Description of the work. Few drops of 0,1N HCl solution should be added to cylinder of the Sali hemometer untill low border.

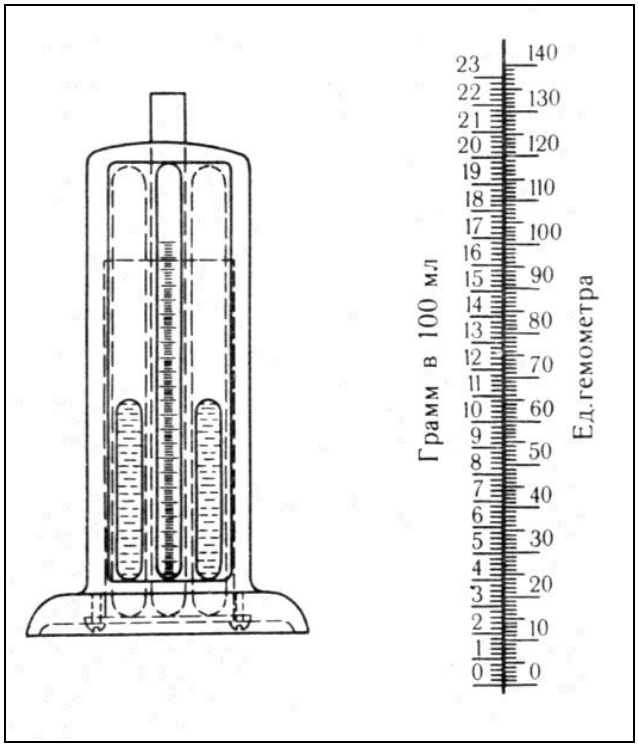

Fig. 17 – Sali hemometr

Then 0,02 ml of investigated blood is added to HCl solution and is mixed for 5 min in hemometer at room temperature. The brown color of hematin chloride will develop. This color should be changed to standard color of hematin chloride, which is presented in hemometer, by water addition. Hemoglobin level is estimated by solution level in cylinder (1g% = 10g/l).

Laboratory work 2. ***Measurment of hemoglobin concentration in the blood by spectrophotometer***

Description of the work. Then 0,02 ml of investigated blood

should be added to 5 ml of transforming solution. It should stay for 10 min at room temperature. After that the investigated sample is insered in photometer KFK-3, and is compared to transforming solution as control at optical distance (λ) of 520 nm. Students estimate hemoglobine concentration using calibration curve.

Laboratory work 3. ***Calculation of erythrocyte count in Goryaev camera***

Description of the work. A rabbit blood should be dissolved 200 times by 3% solution of sodium chloride. The volume 0,02 ml of investigated blood is added to 4 ml of 3% sodium chloride solution. Mix and flood Goryaev camera by this solution.

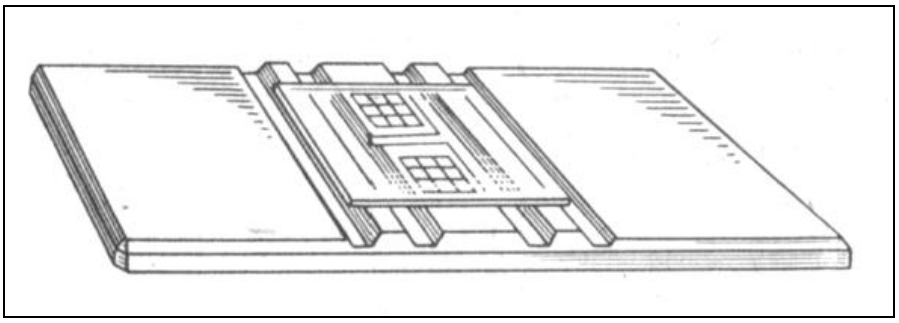

Fig. 18 – Goryaev camera for calculation of the blood cells

Under microscopat small magnification students calculate erythrocyte count in 5 big shade quadrants in diagonal direction.

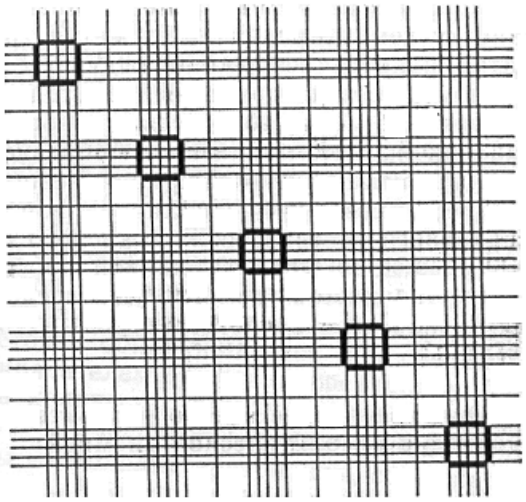

Fig. 19 – Scheeme of quadrants at Goryaev camera

for calculation of the blood cells

The sum of calculated RBCs should be put to the formula:

$$X = \frac{A \times 200 \times 4000}{80} \times 10^6, \textit{where}$$

X – RBC count per 1 liter of the blood,
A – sum of RBC in 5 big shade quadrants,
200 – dissolving degree,
4000 – total level of small quadrants in Goryaev camera.
80 – sum of small quadrants in 5 big shade quadrants.

Laboratory work 4. ***Calculation of color idex***

Description of the work 4.

Color index (CI) = (Hb(g/l) × 3) /first three figures of RBC count

Normal color index is 0,85 – 1,05

For example: Hb – 56 g/l
RBC – $3{,}5 \times 10^{12}$/l

$$\text{CI} = \frac{56 \times 3}{350} = 0{,}48$$

Conclusion: hypochromic anemia

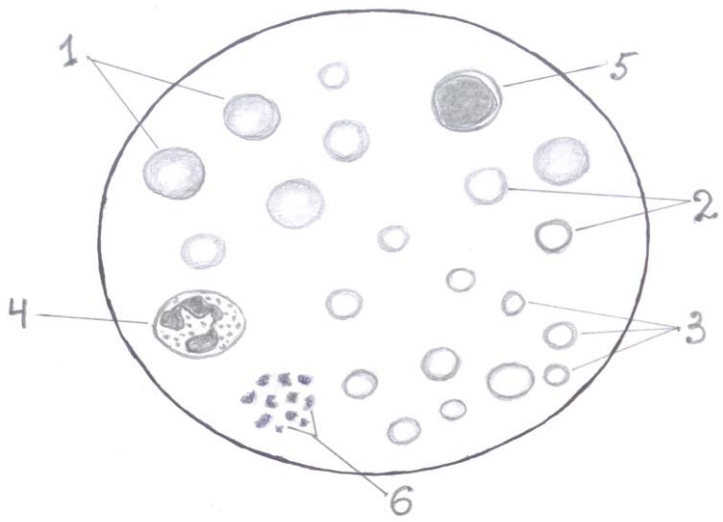

Fig. 20 – Blood film in iron deficiency anemia

1 – normochromic erythrocyte
2, 3 – erythrocyte hypochromic, microcyte
4 – polymorphnucleic leucocyte
5 – lymphocyte
6 – plattelets

Tasks:

1

Patient, 42 years old woman, arrives to the genicological hospital after prolonged uteric bleeding (about 2-3 weeks).

Clinical findings: paleness, rapid puls, uteric myoma (benign tumor).

Blood analysis: Нв – 68g/l, RBC – $2,8x10^{12}$/l, Rt – 0,5%, erythrocyte sedimentation rate (ESR) – 8 mm/hr.

Blood sample: hypochromia of erythrocyties, anyzocytosis, microcytosis, poikilocytosis, and single polychromatophills.

Biochemical analysis: serum Fe concentration 6 μM.

What pathology has got the patient? Is it Fe-deficite anemia?

Make hematological conclusion.

2

A grey-haired male with blue eyes, 52 years old, is complaining of precordial pain, Dyspnoea upon stair climbing, and nausea. He is depressed and suffers from frequent coughs.The doctor observes icteric skin and eyes, ataxic walking, dysdiadochokinesis, and positive Babinski. Massive subcutaneous bleeding was found at the left hip.Laboratory tests revealed the following abnormal results: Lack of HCl in the gastric fluid during fasting and following a pentagastrin test. Haematology tests revealed large erythrocytes - many with nuclei. The red cell count was $1.4*10^{12}$ per l. The haematocrit was 0.21, and the blood [haemoglobin] was 4 mM. The bleeding time was 90 min and the platelet count was $50*10^{9}$ per l. The concentration of vitamin B_{12} in serum was 90 ng per l. The total [bilirubin] in serum was 18 mg per l, and the rise mainly due to non-conjugated bilirubin. A test with radioactive B_{12} was specific for lack of intrinsic factor production from the patient's parietal cells.

1. What was the cause of this severe pancytopenia (lack of all blood cell types)?

2. Calculate the oxygen capacity for haemoglobin.

3. Why did the patient develop leucopenia and thrombocytopenia? Was the lack of leucocytes and platelets of any consequences to the patient?

4. Does a severe, chronic anaemia trigger physiologic adaptations?

Hemogramm № 1		**Hemogramm № 2**	
Hemoglobin (Hgb)	95 g/l	Hemoglobin (Hgb)	88 g/l
Erythrocytes (RBC)	$3,6x10^{12}$/l	Erythrocytes (RBC)	$3,1x10^{12}$/l
Reticulocytes –	38 ‰	Reticulocytes	0,4%
Hct	0,28	Hct	0,48
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC-?		MCHC - ?	
Hemogramm № 3		**Hemogramm № 4**	
Hemoglobin (Hgb)	68 g/l	Hemoglobin (Hgb)	120g/l
Erythrocytes (RBC)	$3,8x10^{12}$/l	Erythrocytes (RBC)	$3,9x10^{12}$/l
Reticulocytes	1,2%	Reticulocytes	0,6%
Hct	0,38	Hct	0,32
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	
Hemogramm № 5		**Hemogramm № 6**	
Hemoglobin (Hgb)	120 g/l	Hemoglobin (Hgb)	128 g/l
Erythrocytes (RBC)	$3,9x10^{12}$/l	Erythrocytes (RBC)	$4,4x10^{12}$/l
Reticulocytes –	8‰	Reticulocytes	0,7%
Hct	0,38	Hct	0,48
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC -?		MCHC - ?	

Hemogramm № 7		**Hemogramm № 8**	
Hemoglobin (Hgb)	66 g/l	Hemoglobin (Hgb)	129 g/l
Erythrocytes (RBC)	$1,44x10^{12}$/l	Erythrocytes (RBC)	$4,1x10^{12}$/l
Reticulocytes	0,4%	Reticulocytes	0,9%
Hct	0,25	Hct	0,34
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	
Hemogramm № 9		**Hemogramm № 10**	
Hemoglobin (Hgb)	119 g/l	Hemoglobin (Hgb)	54 g/l
Erythrocytes (RBC)	$4,1x10^{12}$/l	Erythrocytes (RBC)	$1,8x10^{12}$/l
Reticulocytes –	10‰	Reticulocytes	0,2%
Hct	0,42	Hct	0,28
MCV - ?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	
Hemogramm № 11		**Hemogramm № 12**	
Hemoglobin (Hgb)	180 g/l	Hemoglobin (Hgb)	120 g/l
Erythrocytes (RBC)	$6,3x10^{12}$/l	Erythrocytes (RBC)	$4,25x10^{12}$/l
Reticulocytes	5%	Reticulocytes	4‰
Hct	0,52	Hct	0,38
MCV - ?		MCV - ?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	
Hemogramm № 13		**Hemogramm № 14**	
Hemoglobin (Hgb)	120 g/l	Hemoglobin (Hgb)	110 g/l
Erythrocytes (RBC)	$4,36x10^{12}$/l	Erythrocytes (RBC)	$3,8x10^{12}$/l
Reticulocytes	12‰	Reticulocytes	18‰
Hct	0,48	Hct	0,33
MCV -?		MCV - ?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	

Hemogramm № 15		**Hemogramm № 16**	
Hemoglobin (Hgb)	102 g/l	Hemoglobin (Hgb)	82 g/l
Erythrocytes (RBC)	3,9 x 10^{12} / l	Erythrocytes (RBC)	3,2x10^{12}/l
Reticulocytes	25 ‰	Reticulocytes	0%
Hct	0,35	Hct	0,32
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	
Hemogramm № 17		**Hemogramm № 18**	
Hemoglobin (Hgb)	40 g/l	Hemoglobin (Hgb)	47 g/l
Erythrocytes (RBC)	0,797x10^{12}/l	Erythrocytes (RBC)	0,99x10^{12}/l
Reticulocytes	3‰	Reticulocytes	2‰
Hct	0,20	Hct	0,22
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	
Hemogramm № 19		**Hemogramm № 20**	
Hemoglobin (Hgb)	80 g/l	Hemoglobin (Hgb)	99 g/l
Erythrocytes (RBC)	2,8x10^{12}/l	Erythrocytes (RBC)	3,8x10^{12}/l
Reticulocytes	18%	Reticulocytes	28‰
Hct	0,26	Hct	0,34
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	
Hemogramm № 21		**Hemogramm № 22**	
Hemoglobin (Hgb)	130 g/l	Hemoglobin (Hgb)	140 g/l
Erythrocytes (RBC)	4,2x10^{12}/l	Erythrocytes (RBC)	4,3x10^{12}/l
Reticulocytes	1,2%	Reticulocytes	38‰
Hct	0,24	Hct	0,46
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	

Hemogramm № 23		**Hemogramm № 24**	
Hemoglobin (Hgb)	95 g/l	Hemoglobin (Hgb)	88 g/l
Erythrocytes (RBC)	$3,6x10^{12}$/l	Erythrocytes (RBC)	$3,1x10^{12}$/l
Reticulocytes	1,8%	Reticulocytes	0,4%
Hct	0,35	Hct	0,30
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	
Hemogramm № 25		**Hemogramm № 26**	
Hemoglobin (Hgb)	68 g/l	Hemoglobin (Hgb)	120 g/l
Erythrocytes (RBC)	$3,8x10^{12}$/l	Erythrocytes (RBC)	$3,9x10^{12}$/l
Reticulocytes	1,2%	Reticulocytes	0,1%
Hct	0,28	Hct	0,38
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	
Hemogramm № 27		**Hemogramm № 28**	
Hemoglobin (Hgb)	185 g/l	Hemoglobin (Hgb)	180 g/l
Erythrocytes (RBC)	$7,2x10^{12}$/l	Erythrocytes (RBC)	$5,8x10^{12}$/l
Reticulocytes	2,6%	Reticulocytes	0,5%
ESR	10 mm/h	ESR	1 mm/h
Hct	0,48	Hct	0,68
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC-?		MCHC - ?	
Hemogramm № 29		**Hemogramm № 30**	
Hemoglobin (Hgb)	80 g/l	Hemoglobin (Hgb)	60 g/l
Erythrocytes (RBC)	$2,9x10^{12}$/l	Erythrocytes (RBC)	$2,0xl0^{12}$/l
Reticulocytes	0,3%	Reticulocytes	0%
Hct	0,38	Hct	0,22
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	

Hemogramm № 31		**Hemogramm № 32**	
Hemoglobin (Hgb)	150 g/l	Hemoglobin (Hgb)	60 g/l
Erythrocytes (RBC)	$1,5x10^{12}$/l	Erythrocytes (RBC)	$0,8x10^{12}$/l
Reticulocytes –	40‰	ESR	80 mm/h
ESR	-	Reticulocytes	0,1%
Hct	0,28	Hct	0,28
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	
Hemogramm № 33		**Hemogramm № 34**	
Hemoglobin (Hgb)	135 g/L	Hemoglobin (Hgb)	110 g/L
Erythrocytes (RBC)	$3,8x10^{l2}$/l	Erythrocytes (RBC)	$3,0xl0^{12}$/l
Reticulocytes	0,4%	Reticulocytes	0,3%
Hct	0,28	Hct	0,32
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	
Hemogramm № 35		**Hemogramm № 36**	
Hemoglobin (Hgb)	70 g/l	Hemoglobin (Hgb)	80 g/l
Erythrocytes (RBC)	$2.5x10^{12}$/l	Erythrocytes (RBC)	$3,4x10^{12}$/l
Reticulocytes	0,1%	Reticulocytes	0%
Hct	0,28	Hct	0,28
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC-?		MCHC - ?	
Hemogramm № 37		**Hemogramm № 38**	
Hemoglobin (Hgb)	58 g/l	Hemoglobin (Hgb)	62 g/l
Erythrocytes (RBC)	$3,0 x10^{12}$/l	Erythrocytes (RBC)	$3,9x10^{12}$/l
Reticulocytes –	18‰	Reticulocytes –	30‰
Hct	0,28	Hct	0,28
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	

Hemogramm № 39		**Hemogramm № 40**	
Hemoglobin (Hgb)	90 g/l	Hemoglobin (Hgb)	100 g/l
Erythrocytes (RBC)	$3,6x10^{12}$/l	Erythrocytes (RBC)	$3,6x10^{12}$/l
Reticulocytes	34‰	Reticulocytes	55‰
Hct	0,28	Hct	0,28
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	
Hemogramm № 41		**Hemogramm № 42**	
Hemoglobin (Hgb)	110 g/l	Hemoglobin (Hgb)	70 g/l
Erythrocytes (RBC)	$3.2x10^{12}$/l	Erythrocytes (RBC)	$2,1x10^{12}$/l
Reticulocytes	0,4%	Reticulocytes	0,1%
Hct	0,28	Hct	0,28
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	
Hemogramm № 43		**Hemogramm № 44**	
Hemoglobin (Hgb)	90 g/l	Hemoglobin (Hgb)	80 g/l
Erythrocytes (RBC)	$3,4x10^{12}$/l	Erythrocytes (RBC)	$2,7x10^{12}$/l
Reticulocytes	3,6%	Reticulocytes	0,2%
Hct	0,28	Hct	0,28
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	
Hemogramm № 45		**Hemogramm № 46**	
Hemoglobin (Hgb)	62 g/l	Hemoglobin (Hgb)	100 g/l
Erythrocytes (RBC)	$3,5x10^{12}$/l	Erythrocytes (RBC)	$3,4x10^{12}$/l
Reticulocytes	25%	Reticulocytes	0,2%
Hct	0,28		
MCV -?			
MCH - ?			
MCHC - ?			
Hemogramm № 47		**Hemogramm № 48**	

Hemoglobin (Hgb) 125 g/l Erythrocytes (RBC) $7,3x10^{12}$/l Reticulocytes 48‰ Hct 0,28 MCV -? MCH - ? MCHC - ?	Hemoglobin (Hgb) 45 g/l Erythrocytes (RBC) $1,4x10^{12}$/l Reticulocytes 10‰ Hct 0,28 MCV -? MCH - ? MCHC - ?
Hemogramm № 49 Hemoglobin (Hgb) 125 g/l Erythrocytes (RBC) $4,2x10^{12}$/l Reticulocytes 25% Hct 0,28 MCV -? MCH - ? MCHC - ?	**Hemogramm № 50** Hemoglobin (Hgb) 120 g/l Erythrocytes (RBC) $4,2x10^{12}$/l Reticulocytes 5% Hct 0,28 MCV -? MCH - ? MCHC - ?
Hemogramm № 51 Hemoglobin (Hgb) 60 g/l Erythrocytes (RBC) $1,3x10^{12}$/l Reticulocytes 1,8% Hct 0,28 MCV -? MCH - ? MCHC - ?	**Hemogramm № 52** Hemoglobin (Hgb) 102 g/l Erythrocytes (RBC) $3,6x10^{12}$/l Reticulocytes 1,1% Hct 0,28 MCV -? MCH - ? MCHC - ?
Hemogramm № 53 Hemoglobin (Hgb) 119 g/l Erythrocytes (RBC) $4,1x10^{12}$/l Reticulocytes 18‰ Hct 0,28 MCV -? MCH - ? MCHC - ?	**Hemogramm № 54** Hemoglobin (Hgb) 50g/l Erythrocytes (RBC) $1,0x10^{12}$/l Reticulocytes 0,2% Hct 0,28 MCV -? MCH - ? MCHC - ?
Hemogramm № 55	**Hemogramm № 56**

Hemoglobin (Hgb) 170 g/l Erythrocytes (RBC) $6,5x10^{12}$/l Reticulocytes 4,5% Hct 0,28 MCV -? MCH - ? MCHC - ?	Hemoglobin (Hgb) 110 g/l Erythrocytes (RBC) $3,6x10^{12}$/l Reticulocytes – 20‰
Hemogramm № 57 Hemoglobin (Hgb) 120 g/l Erythrocytes (RBC) $4,36x10^{12}$/l Reticulocytes 8‰ Hct 0,28 MCV -? MCH - ? MCHC -?	**Hemogramm № 58** Hemoglobin (Hgb) 100 g/l Erythrocytes (RBC) $3,1x10^{12}$/l Reticulocytes 25‰ Hct 0,28 MCV -? MCH - ? MCHC - ?
Hemogramm № 59 Hemoglobin (Hgb) 45 g/l Erythrocytes (RBC) $0,84x10^{12}$/l Reticulocytes 0‰ Hct 0,28 MCV -? MCH - ? MCHC - ?	**Hemogramm № 60** Hemoglobin (Hgb) 44 g/l Erythrocytes (RBC) $1,2x10^{12}$/l Reticulocytes 1‰ Hct 0,28 MCV -? MCH - ? MCHC-?
Hemogramm № 61 Hemoglobin (Hgb) 65 g/l Erythrocytes (RBC) $2,4x\ 10^{12}$/l Reticulocytes 15% Hct 0,28 MCV -? MCH - ? MCHC - ?	**Hemogramm № 62** Hemoglobin (Hgb) 90 g/l Erythrocytes (RBC) $3,4x10^{12}$/l Reticulocytes – 8‰ Hct 0,28 MCV -? MCH - ? MCHC-?

Hemogramm № 63		**Hemogramm № 64**	
Hemoglobin (Hgb)	85 g/l	Hemoglobin (Hgb)	80 g/l
Erythrocytes (RBC)	3,0x 10^{12}/l	Erythrocytes (RBC)	2,7x10^{12}/l
Reticulocytes	1,0%	Reticulocytes	0,3%
Hct	0,28	Hct	0,28
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	
Hemogramm № 65		**Hemogramm № 66**	
Hemoglobin (Hgb)	68 g/l	Hemoglobin (Hgb)	100 g/l
Erythrocytes (RBC)	3,8 x 10^{12}/l	Erythrocytes (RBC)	3,4x10^{12}/l
Reticulocytes	1,2%	Reticulocytes	0,2%
Hct	0,28	Hct	0,28
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	
Hemogramm № 67		**Hemogramm № 68**	
Hemoglobin (Hgb)	110 g/l	Hemoglobin (Hgb)	90 g/l
Erythrocytes (RBC)	1,2 x10^{12}/l	Erythrocytes (RBC)	3,8 x 10^{12}/l
Reticulocytes	3,8%	Reticulocytes	0,3%
Hct	0,28	Hct	0,28
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC-?	
Hemogramm № 69		**Hemogramm № 70**	
Hemoglobin (Hgb)	56 g/l	Hemoglobin (Hgb)	80 g/l
Erythrocytes (RBC)	1,3x10^{12}/l	Erythrocytes (RBC)	2,5xl0^{12}/l
Reticulocytes	0%	Reticulocytes	2%
ESR	52 mm/h	Hct	0,28
Hct	0,28	MCV -?	
MCV -?		MCH - ?	
MCH - ?		HC - ?	
MCHC - ?			

Hemogramm № 71		**Hemogramm № 72**	
Hemoglobin (Hgb)	48 g/l	Hemoglobin (Hgb)	170 g/l
Erythrocytes (RBC)	1,2xl0^{12}/l	Erythrocytes (RBC)	4,0x 10^{12}/l
Reticulocytes	0,3%	Reticulocytes	1,0%
Hct	0,28	Hct	0,28
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC-?		MCHC - ?	
Hemogramm № 73		**Hemogramm № 74**	
Erythrocytes (RBC)	3,0xl0^{12}/l	Erythrocytes (RBC)	3,7x10^{12}/l
Hemoglobin (Hgb)	100 g/l	Hemoglobin (Hgb)	95 g/l
Reticulocytes	0,6%	Reticulocytes	3%
Hct	0,28	Hct	0,28
MCV -?		MCV -?	
MCH - ?		MCH - ?	
MCHC - ?		MCHC - ?	

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LESSON № 21

Topic: QUANTITATIVE DISORDERS OF NEUTROPHILS. LEUKOCYTOSIS AND LEUKOPENIA

Aim of the lesson: to study main causes and mechanisms of leukocytosis and leucopenia development.

QUESTIONS:

1. Leukocytes. Stages of leukopoiesis. Leukocyte's description at different stages of maturation.
2. Functions of leukocytes (neutrophils, eosinophils, basophils, monocytes, lymphocytes).
3. Leukocyte formula. Leukocyte formula in children.
4. Clinical importance of calculation of leukocyte formula.
5. Classification of disorders of white blood cells.
6. Leukocytosis. Categorization. Biological significance of leukocytosis.
7. Causes and mechanisms of different leukocytosis: neutrophilic, eosinophilic, basophilic, monocytic, lymphocytic.
8. Patterns shift in leukocyte formula. Types (to-left, to-right).
9. Leukopenia. Categorization. Causes, General mechanisms and outcomes of neutropenia, lymphopenia.
10. Agranulocytosis. Outcomes.

HAEMOGRAMMS

Hemogramm № 1

Hemoglobin (Hgb)	95 g/l
Erythrocytes (RBC)	$3,6x10^{12}$/l
Leucocytes (WBC)	$16x10^{9}$/l
Basophils	1%
Eosinophils	3%
Neutrophiles:	
myelocytes	1%
methamyelocytes	2%
band forms	8%
neutrophils	64%
Lymphocytes	18%
Monocytes	3%
Thrombocytes (Plt)	$300x10^{9}$/l
Reticulocytes	3,8%

Hemogramm № 2

Hemoglobin (Hgb)	54 g/l
Erythrocytes (RBC)	$1,8x10^{12}$/l
Leucocytes (WBC)	$3,2x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	0%
neutrophils	26%
Lymphocytes	63%
Monocytes	11%
Thrombocytes (Plt)	$94x10^{9}$/l
Reticulocytes	0,2%

Hemogramm № 3

Hemoglobin (Hgb)	68 g/l
Erythrocytes (RBC)	$3,8x10^{12}$/l
Leucocytes (WBC)	$5,4x10^{9}$/l
Basophils	1%
Eosinophils	2%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	6%
neutrophils	54%
Lymphocytes	30%
Monocytes	7%
Thrombocytes (Plt)	$280x10^{9}$/l
Reticulocytes	1,2%

Hemogramm № 4

Hemoglobin (Hgb)	120 g/l
Erythrocytes (RBC)	$3,9x10^{12}$/l
Leucocytes (WBC)	$3,4x10^{9}$/l
Basophils	0%
Eosinophils	2%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	2%
neutrophils	29%
Lymphocytes	55%
Monocytes	2%
Thrombocytes (Plt)	$210x10^{9}$/l
Reticulocytes	0,6%

Hemogramm № 5

Hemoglobin (Hgb)	120 g/l
Erythrocytes (RBC)	$3,9x10^{12}$/l
Leucocytes (WBC)	$25x10^{9}$/l

Hemogramm № 6

Hemoglobin (Hgb)	128 g/l
Erythrocytes (RBC)	$4,4x10^{12}$/l
Leucocytes (WBC)	$13,6x10^{9}$/l

Basophils	0%
Eosinophils	3%
Neutrophiles:	
myelocytes	0%
methamyelocytes	4%
band forms	16%
neutrophils	58%
Lymphocytes	15%
Monocytes	4%
Thrombocytes (Plt)	$230x10^9$/l

Basophils	1%
Eosinophils	18%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	4%
neutrophils	46%
Lymphocytes	23%
Monocytes	8%
Thrombocytes (Plt)	$210x10^9$/l
Reticulocytes	0,7%

Hemogramm № 7

Hemoglobin (Hgb)	66 g/l
Erythrocytes (RBC)	$1,44x10^{12}$/l
Leucocytes (WBC)	$2,8x10^9$/l
Basophils	0%
Eosinophils	5%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	1%
neutrophils	43%
Lymphocytes	48%
Monocytes	3%
Thrombocytes (Plt)	$100x10^9$/l
Reticulocytes	0,4%

Hemogramm № 8

Hemoglobin (Hgb)	129 g/l
Erythrocytes (RBC)	$4,1x10^{12}$/l
Leucocytes (WBC)	$36x10^9$/l
Basophils	0,5%
Eosinophils	2,5%
Neutrophiles:	
promyelocytes	2%
myelocytes	2%
methamyelocytes	7%
band forms	9%
neutrophils	52%
Lymphocytes	20%
Monocytes	5%
Thrombocytes (Plt)	$280x10^9$/l
Reticulocytes	0,9%

Hemogramm № 9

Hemoglobin (Hgb)	180 g/l
Erythrocytes (RBC)	$6,3x10^{12}$/l
Leucocytes (WBC)	$6,25x10^{9}$/l
Basophils	0%
Eosinophils	1%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	1%
neutrophils	70%
Lymphocytes	21%
Monocytes	7%
Thrombocytes (Plt)	$380x10^{9}$/l
Reticulocytes	5%

Hemogramm № 10

Hemoglobin (Hgb)	120 g/l
Erythrocytes (RBC)	$4,36x10^{12}$/l
Leucocytes (WBC)	$16,2x10^{9}$/l
Basophils	0%
Eosinophils	1%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	5%
neutrophils	37%
Lymphocytes	50%
Monocytes	7%
Thrombocytes (Plt)	$270x10^{9}$/l

Hemogramm № 11

Hemoglobin (Hgb)	80 g/l
Erythrocytes (RBC)	$2,8x10^{12}$/l
Leucocytes (WBC)	$14x10^{9}$/l
Basophils	1%
Eosinophils	8%
Neutrophiles:	
myelocytes	0%
methamyelocytes	6%
band forms	16%
neutrophils	53%
Lymphocytes	10%
Monocytes	6%
Reticulocytes	18%
Thrombocytes (Plt)	$400x10^{9}$/l

Hemogramm № 12

Hemoglobin (Hgb)	80 g/l
Erythrocytes (RBC)	$2,9x10^{12}$/l
Reticulocytes	0,3%
Platelets	$0,15x10^{9}$/l
Leucocytes (WBC)	$0,5x10^{9}$/l
Basophils	0%
Eosinophils	10%
Neutrophiles:	22
Lymphocytes	58%
Monocytes	10%

Hemogramm № 13

Hemoglobin (Hgb)	102 g/l
Erythrocytes (RBC)	$3,9x10^{12}$/l
Leucocytes (WBC)	$17,5x10^{9}$/l
Basophils	0%
Eosinophils	0,5%
Neutrophiles:	
myelocytes	4,5%
methamyelocytes	16%
band forms	39%
neutrophils	32%
Lymphocytes	4,5%
Monocytes	3,5%
Thrombocytes (Plt)	$310x10^{9}$/l

Hemogramm № 14

Hemoglobin (Hgb)	82 g/l
Erythrocytes (RBC)	$3,2x10^{12}$/l
Leucocytes (WBC)	$0,325x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	0%
Lymphocytes	83%
Monocytes	17%
Thrombocytes (Plt)	$10x10^{9}$/l
Reticulocytes	0%

Hemogramm № 15

Hemoglobin (Hgb)	40 g/l
Erythrocytes (RBC)	$0,797x10^{12}$/l
Leucocytes (WBC)	$3,4x10^{9}$/l
Basophils	0%
Eosinophils	0,5%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	4%
neutrophils	38%
Lymphocytes	50,5%
Monocytes	7%
Thrombocytes (Plt)	$120x10^{9}$/l

Hemogramm № 16

Hemoglobin (Hgb)	47 g/l
Erythrocytes (RBC)	$0,99x10^{12}$/l
Leucocytes (WBC)	$9,3x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	2%
neutrophils	10%
Lymphoblastes	62%
Lymphocytes	20%
Monocytes	6%
Thrombocytes (Plt)	$12,1x10^{9}$/l

Hemogramm № 17

Hemoglobin (Hgb)	130 g/l
Erythrocytes (RBC)	$4,2x10^{12}$/l
Leucocytes (WBC)	$12x10^{9}$/l
Basophils	1%
Eosinophils	3%
Neutrophiles:	
myelocytes	0%
methamyelocytes	4,5%
band forms	12%
neutrophils	48,5%
Lymphocytes	20%
Monocytes	11%
Thrombocytes (Plt)	$340x10^{9}$/l
Reticulocytes	1,2%

Hemogramm № 18

Hemoglobin (Hgb)	140 g/l
Erythrocytes (RBC)	$4,3x10^{12}$/l
Leucocytes (WBC)	$2,9x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	33%
neutrophils	14%
Lymphocytes	46%
Monocytes	7%
Thrombocytes (Plt)	$260x10^{9}$/l

Hemogramm № 19

Hemoglobin (Hgb)	85 g/l
Erythrocytes (RBC	$2,2x10^{l2}$/l
Leucocytes (WBC)	$12,0xl0^{9}$/l
Basophils	0,5%
Eosinophils	1%
Neutrophiles:	
band forms	1%
segmented	2%
Lymphoblasts	30%
Lymphocytes	65%
Monocytes	0,5%

Hemogramm № 20

Hemoglobin (Hgb)	170 g/l
Erythrocytes (RBC)	$6,5x10^{12}$/l
Reticulocytes	4,5%
Leucocytes (WBC)	$6,8x10^{9}$/l
Basophils	0%
Eosinophils	1%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	1%
neutrophils	70%
Lymphocytes	21%
Monocytes	7%
Thrombocytes (Plt)	$420x10^{9}$/l

Hemogramm № 21

Hemoglobin (Hgb)	185 g/l
Erythrocytes (RBC)	$7,2x10^{12}$/l
Reticulocytes	2,6%
ESR	10 mm/h
Leucocytes (WBC)	$14,0x10^{9}$/l
Basophils	1,5%
Eosinophils	8%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0,5%
band forms	13%
segmented	59%
Lymphocytes	15%
Monocytes	3%
Platelets	$580x10^{9}$/l

Hemogramm № 22

Hemoglobin (Hgb)	90 g/l
Erythrocytes (RBC)	$3,4x10^{12}$/l
Reticulocytes	3,6%
Leucocytes (WBC)	$15,5x10^{9}$/l
Basophils	1%
Eosinophils	2%
Neutrophiles:	
myelocytes	1%
methamyelocytes	2%
band forms	8%
neutrophils	64%
Lymphocytes	19%
Monocytes	2%
Thrombocytes (Plt)	$320x10^{9}$/l

Hemogramm № 23

Hemoglobin (Hgb)	50 g/l
Erythrocytes (RBC)	$1,0x10^{12}$/l
Reticulocytes	0,2%
Leucocytes (WBC)	$2,5x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	2%
neutrophils	28%
Lymphocytes	60%
Monocytes	10%
Thrombocytes (Plt)	$94x10^{9}$/l

Hemogramm № 24

Hemoglobin (Hgb)	110 g/l
Erythrocytes (RBC)	$1,2x10^{12}$/l
Reticulocytes	0,4%
Leucocytes (WBC)	$18,0x10^{9}$/l
Basophils	0%
Eosinophils	2%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	15%
segmented	55%
Lymphocytes	20%
Monocytes	4%

Hemogramm № 25

Hemoglobin (Hgb)	110 g/l
Erythrocytes (RBC)	$4,0x10^{12}$/l
Leucocytes (WBC)	$10x10^{9}$/l
Basophils	0%
Eosinophils	2%
Neutrophiles:	
myelocytes	2%
methamyelocytes	4%
band forms	11%
neutrophils	49%
Lymphocytes	21%
Monocytes	11%
Thrombocytes (Plt)	$300x10^{9}$/l
Reticulocytes	0,8%

Hemogramm № 26

Hemoglobin (Hgb)	110 g/l
Erythrocytes (RBC)	$3,6x10^{12}$/l
Leucocytes (WBC)	$2,0x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	30%
neutrophils	17%
Lymphocytes	40%
Monocytes	13%
Thrombocytes (Plt)	$260x10^{9}$/l

Hemogramm № 27

Hemoglobin (Hgb)	90 g/l
Erythrocytes (RBC)	$3,6x10^{12}$/l
Leucocytes (WBC)	$34,0x10^{9}$/l
Platelets	$180x10^{9}$/l
Basophils	0%
Eosinophils	8%
Neutrophiles:	
Promyelocytes	0%
myelocytes	0%
methamyelocytes	3%
band forms	14%
segmented	60%
Lymphocytes	12%
Monocytes	3%

Hemogramm № 28

Hemoglobin (Hgb)	100 g/l
Erythrocytes (RBC)	$3,6x10^{12}$/l
MCH	9
Leucocytes (WBC)	$23,9x10^{9}$/l
Platelets	$490x10^{9}$/l
Eosinophils	5%
Basophils	3%
Neutrophiles:	
Promyelocytes	2%
myelocytes	3%
methamyelocytes	6%
band forms	14%
segmented	44%
Lymphocytes	18%
Monocytes	5%

Hemogramm № 29

Hemoglobin (Hgb)	62 g/l
Erythrocytes (RBC)	$3,5x10^{12}$/l
Leucocytes (WBC)	$5,1x10^{9}$/l
Basophils	1%
Eosinophils	2%
Neutrophiles:	
myelocytes	0%
methamyelocytes	2%
band forms	4%
neutrophils	54%
Lymphocytes	28%
Monocytes	9%
Thrombocytes (Plt)	$200x10^{9}$/l
Reticulocytes	25%

Hemogramm № 30

Hemoglobin (Hgb)	100 g/l
Erythrocytes (RBC)	$3,4x10^{12}$/l
Leucocytes (WBC)	$30x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	
myelocytes	85%
methamyelocytes	0%
band forms	0%
neutrophils	9%
Lymphocytes	6%
Monocytes	0%
Thrombocytes (Plt)	$100x10^{9}$/l
Reticulocytes	0,2%

Hemogramm № 31

Hemoglobin (Hgb)	125 g/l
Erythrocytes (RBC)	$7,3x10^{12}$/l
Leucocytes (WBC)	$14,6x10^{9}$/l
Basophils	1%
Eosinophils	11%
Neutrophiles:	
myelocytes	2%
methamyelocytes	4%
band forms	10%
neutrophils	57%
Lymphocytes	18%
Monocytes	5%
Thrombocytes (Plt)	$520x10^{9}$/l

Hemogramm № 32

Hemoglobin (Hgb)	45 g/l
Erythrocytes (RBC)	$1,4x10^{12}$/l
Leucocytes (WBC)	$2,2x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	0%
neutrophils	20%
Lymphocytes	14%
Monocytes	8%
Monoblastes	56%
Thrombocytes (Plt)	$70x10^{9}$/l

Hemogramm № 33

Hemoglobin (Hgb)	125 g/l
Erythrocytes (RBC)	$4,2x10^{12}$/l
Leucocytes (WBC)	$23x10^{9}$/l
Basophils	0%
Eosinophils	2%
Neutrophiles:	
myelocytes	0%
methamyelocytes	5%
band forms	13%
neutrophils	61%
Lymphocytes	12%
Monocytes	4%
Thrombocytes (Plt)	$210x10^{9}$/l
Reticulocytes	25%

Hemogramm № 34

Hemoglobin (Hgb)	120 g/l
Erythrocytes (RBC)	$4,2x10^{12}$/l
Leucocytes (WBC)	$13,1x10^{9}$/l
Basophils	0%
Eosinophils	15%
Neutrophiles:	
myelocytes	0%
methamyelocytes	2%
band forms	6%
neutrophils	46%
Lymphocytes	23%
Monocytes	8%
Thrombocytes (Plt)	$200x10^{9}$/l
Reticulocytes	5%

Hemogramm № 33

Hemoglobin (Hgb)	125 g/l
Erythrocytes (RBC)	$4,2x10^{12}$/l
Leucocytes (WBC)	$23x10^{9}$/l
Basophils	0%
Eosinophils	2%
Neutrophiles:	
myelocytes	0%
methamyelocytes	5%
band forms	13%
neutrophils	61%
Lymphocytes	12%
Monocytes	4%
Thrombocytes (Plt)	$210x10^{9}$/l
Reticulocytes	25%

Hemogramm № 34

Hemoglobin (Hgb)	120 g/l
Erythrocytes (RBC)	$4,2x10^{12}$/l
Leucocytes (WBC)	$13,1x10^{9}$/l
Basophils	0%
Eosinophils	15%
Neutrophiles:	
myelocytes	0%
methamyelocytes	2%
band forms	6%
neutrophils	46%
Lymphocytes	23%
Monocytes	8%
Thrombocytes (Plt)	$200x10^{9}$/l
Reticulocytes	5%

Hemogramm № 35		**Hemogramm № 36**	
Hemoglobin (Hgb)	60 g/l	Hemoglobin (Hgb)	65 g/l
Erythrocytes (RBC)	$1,3x10^{12}$/l	Erythrocytes (RBC)	$2,4x10^{12}$/l
Leucocytes (WBC)	$2,0x10^{9}$/l	Leucocytes (WBC)	$11x10^{9}$/l
Basophils	0%	Basophils	1%
Eosinophils	2%	Eosinophils	9%
Neutrophiles:		Neutrophiles:	
myelocytes	0%	myelocytes	0%
methamyelocytes	0%	methamyelocytes	5%
band forms	1%	band forms	13%
neutrophils	40%	neutrophils	56%
Lymphocytes	51%	Lymphocytes	18%
Monocytes	6%	Monocytes	8%
Thrombocytes (Plt)	$110x10^{9}$/l	Reticulocytes	15%
Reticulocytes	1,04%	Thrombocytes (Plt)	$390x10^{9}$/l

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LESSON № 22

Topic: LEUKEMIA

Aim of the lesson: to study the picture of perefericial blood of more frequent forms of human leukemia.

QUESTIONS:

1. Leukemia. Definition. Etiology.
2. Pathogenesis of leukemia: hyperplasia, anaplasia, dysplasia, metaplasia.
3. The particularities of leukemic cells.
4. Categorization of leukemia (acute and chronic).
5. Morphological picture of blood in patients with acute and chronic myelogenous leukemia.
6. Morphological picture of perefericial blood in patients with acute and chronic lymphocytic leukemia.
7. Clinical syndromes of leukemia: anemic, hemorrhagic, infective, metastatic, and intoxication.
8. The difference between leukemia and leukocytosis.
9. Leukemoid reactions: typical features, causes, mechanisms of development, physiological significance.

Laboratory work 1. ***Microscopia of blood samples in patients with acute myeloid leukemia***

Description of the work. Blood sample painted by Romanovski-Gymza is used for diagnostic procedure of acute myeloid leukemia. Under microscope large magnification students estimate the type of leukocytes.

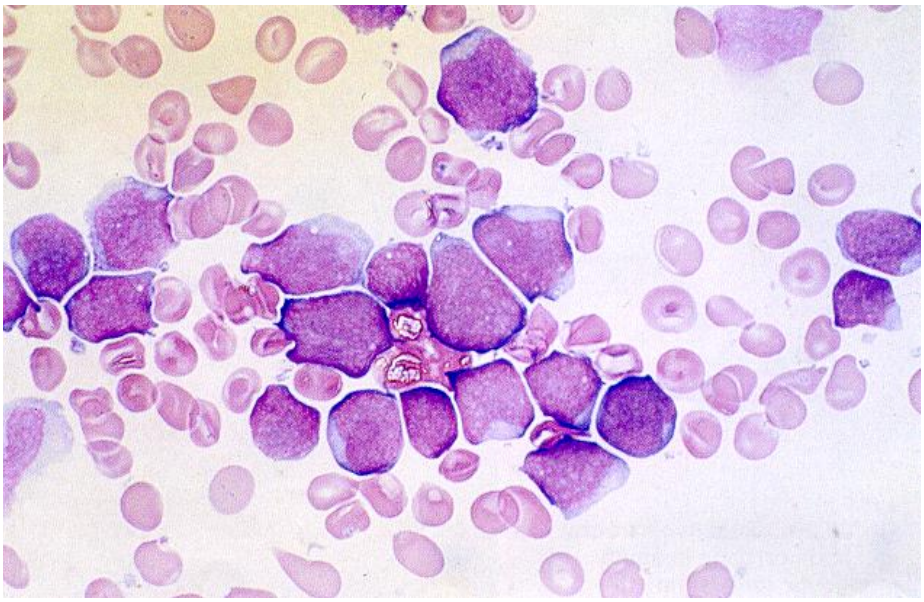

Fig. 21 – Acute myeloid leukemia

The myeloblasts are marker of myeloid leukemia in peripheral blood. The characteristic sign of acute myeloid leukemia is the big amount of myeloblasts, small level of mature segment neutrophills and absence of metamyelocytes and myelocytes (hiatus leukemicus). Such signs occur during acute myeloid leukemia only.

Students make a drawing of acute myeloid leukemia in copybooks.

Laboratory work 2. ***Microscopia of blood samples in patients with chronic myeloid leukemia***

Description of the work. Blood sample painted by Romanovski-Gymza is used for diagnostic procedure of chronic myeloid leukemia. Under microscope large magnification students estimate the leukocytes according to their nuclea form and size, granul's color. The myeloblasts are marker of myeloid leukemia in peripheral blood.

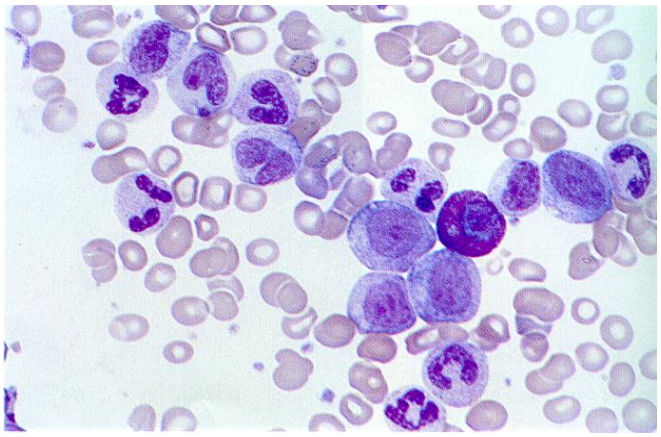

Fig. 22– Blood film with chronic myeloid leukemia

The characteristic sign of chronic myeloid leukemia is the presense of metamyelocytes, myelocytes and eosinophilic-basophilic accociation. Eosinophilic-basophilic accociation is the increasing percent of basophills and eosinophills in leukocytemic formula semultaniosly. Such increase generally occurs during chronic myeloid leukemia.

Students make a drawing of chronic myeloid leukemia in copybooks.

Laboratory work 3. ***Microscopia of blood samples in patients with chronic lymphoid leukemia***

Description of the work. Blood sample painted by Romanovski-Gymza is used for diagnostic procedure of chronic lymphoid leukemia. At microscope large magnification students estimate the type of leukocytes. The lymphoblasts are markers of lymphoid leukemia in peripheral blood.

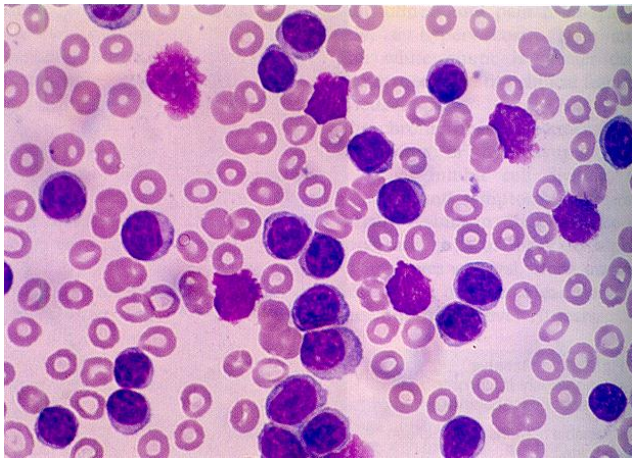

Fig. 23 – Blood picture in chronic lymphoid leukemia

The characteristic sign of chronic myeloid leukemia is increased persent of lymphocytes (lymphocytosis) and Botkin-Humpreht bodies. Such signs occur during chronic lymphoid leukemia only.

Students make a drawing of chronic lymphoid leukemia in copybooks.

HEMOGRAMMS

Hemogramm № 1

Hemoglobin (Hgb)	129 g/l
Erythrocytes (RBC)	$4,1x10^{12}$/l
Leucocytes (WBC)	$36x10^{9}$/l
Basophils	0,5%
Eosinophils	2,5%
Neutrophiles:	
promyelocytes	2%
myelocytes	2%
methamyelocytes	7%
band forms	9%
neutrophils	52%
Lymphocytes	20%
Monocytes	5%
Thrombocytes (Plt)	$280x10^{9}$/l
Reticulocytes	0,9%

Hemogramm № 2

Hemoglobin (Hgb)	88 g/l
Erythrocytes (RBC)	$3,1x10^{12}$/l
Leucocytes (WBC)	$93x10^{9}$/l
Basophils	4%
Eosinophils	9%
Neutrophiles:	
myeloblastes	1%
promyelocytes	6%
myelocytes	20%
methamyelocytes	20%
band forms	13%
neutrophils	12%
Lymphocytes	10%
Monocytes	5%
Thrombocytes (Plt)	$390x10^{9}$/l
Reticulocytes	0,4%

Hemogramm № 3

Hemoglobin (Hgb)	119 g/l
Erythrocytes (RBC)	$4,1x10^{12}$/l
Leucocytes (WBC)	$57x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	0%
eutrophils	9%
Lymphoblastes	7%
Lymphocytes	81%
Monocytes	3%
Thrombocytes (Plt)	$160x10^{9}$/l
The Botkin-Humprecht particles are found	

Hemogramm № 4

Hemoglobin (Hgb)	120 g/l
Erythrocytes (RBC)	$4,25x10^{12}$/l
Leucocytes (WBC)	$2,7x10^{9}$/l
Basophils	0%
Eosinophils	0,5%
Neutrophiles:	
myeloblastes	78%
myelocytes	0%
methamyelocytes	0%
band forms	1,5%
neutrophils	8,5%
Lymphocytes	7%
Monocytes	4,5%
Thrombocytes (Plt)	$410x10^{9}$/l

Hemogramm № 5

Hemoglobin (Hgb)	110 g/l
Erythrocytes (RBC)	$3,8x10^{12}$/l
Leucocytes (WBC)	$84x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	
myeloblastes	95%
myelocytes	0%
methamyelocytes	0%
band forms	0%
neutrophils	2%
Lymphocytes	3%
Monocytes	0%
Thrombocytes (Plt)	$160x10^{9}$/l

Hemogramm № 6

Hemoglobin (Hgb)	47 g/l
Erythrocytes (RBC)	$0,99x10^{12}$/l
Leucocytes (WBC)	$9,3x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	2%
neutrophils	10%
Lymphoblastes	62%
Lymphocytes	20%
Monocytes	6%
Thrombocytes (Plt)	$12,1x10^{9}$/l

Hemogramm № 7

Hemoglobin (Hgb)	95 g/l
Erythrocytes (RBC)	$3,6x10^{12}$/l
Leucocytes (WBC)	$36x10^{9}$/l
Basophils	2%
Eosinophils	7%
Neutrophiles:	
myeloblastes	3%
promyelocytes	6%
myelocytes	11%
methamyelocytes	12%
band forms	18%
neutrophils	21%
Lymphocytes	18%
Monocytes	2%
Thrombocytes (Plt)	$300x10^{9}$/l
Reticulocytes	1,8%

Hemogramm № 8

Hemoglobin (Hgb)	88 g/l
Erythrocytes (RBC)	$3,1x10^{12}$/l
Leucocytes (WBC)	$93x10^{9}$/l
Basophils	4%
Eosinophils	9%
Neutrophiles:	
myeloblastes	1%
promyelocytes	6%
myelocytes	20%
methamyelocytes	20%
band forms	13%
neutrophils	12%
Lymphocytes	10%
Monocytes	5%
Thrombocytes (Plt)	$390x10^{9}$/l
Reticulocytes	0,4%

Hemogramm № 9

Hemoglobin (Hgb)	180 g/l
Erythrocytes (RBC)	
$5,8x10^{12}$/l	
Leucocytes (WBC)	$13,5x10^{9}$/l
Platelets	$520xl0^{9}$/l
Myeloblasts	30%
Segmented	59%
Basophils	0%
Eosinophils	1%
Neutrophiles:	
Lymphocytes	3%
Monocytes	7%
Reticulocytes	0,5%
ESR	1 mm/h

Hemogramm № 10

Hemoglobin (Hgb)	120 g/l
Erythrocytes (RBC)	$3,9x10^{12}$/l
Leucocytes (WBC)	$34x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	
myeloblastes	91%
myelocytes	0%
methamyelocytes	0%
band forms	0%
neutrophils	6%
Lymphocytes	3%
Monocytes	0%
Thrombocytes (Plt)	$110x10^{9}$/l
Reticulocytes	0,1%

Hemogramm № 11

Leucocytes (WBC)	$35x10^{9}$/l
Basophils	3%
Eosinophils	11%
Neutrophiles:	
myeloblastes	2%
myelocytes	5%
methamyelocytes	6%
band forms	12%
neutrophils	35%
Lymphocytes	20%
Monocytes	6%

Hemogramm № 12

Hemoglobin (Hgb)	40 g/l
Erythrocytes (RBC)	$1,2x10^{12}$/l
Leucocytes (WBC)	$2,4x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	0%
neutrophils	21%
Lymphocytes	16%
Monoblastes	57%
Monocytes	6%

Hemogramm № 13

Hemoglobin (Hgb)	58 g/l
Erythrocytes (RBC)	$3x10^{12}$/l
Leucocytes (WBC)	$182xl0^{9}$/l
Myeloblasts	4%
Basophils	9%
Eosinophils	5%
Neutrophiles:	
myelocytes	4,5%
methamyelocytes	10%
band forms	8%
segmented	37,5%
Promyelocytes	12%
Lymphocytes	0%
Monocytes	0%

Hemogramm № 14

Leucocytes (WBC)	$2,4x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	
band forms	19%
segmented	21%
Lymphoblasts	21%
Lymphocytes	27%
Monocytes	12%

Hemogramm № 15

Hemoglobin (Hgb)	150 g/l
Erythrocytes (RBC)	$1,5x10^{12}$/l
Leucocytes (WBC)	$36,0xl0^{9}$/l
Basophils	2%
Eosinophils	3%
Neutrophiles:	
myelocytes	8%
methamyelocytes	20%
band forms	24%
segmented	20%
Lymphoblasts	0%
Lymphocytes	10%
Monocytes	13%
Platelets	$280x10^{9}$/l

Hemogramm № 16

Hemoglobin (Hgb)	60 g/l
Erythrocytes (RBC)	$0,8x10^{12}$/l
Reticulocytes	0,1%
ESR	80 mm/h
Leucocytes (WBC)	$0,9xl0^{9}$/l
Basophils	0%
Eosinophils	2%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	0%
segmented	8%
Lymphocytes	60%
Monocytes	%
Lymphoblasts	28%

Hemogramm № 17

Hemoglobin (Hgb)	119 g/l
Erythrocytes (RBC)	$4,1x10^{12}$/l
Leucocytes (WBC)	$57x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	0%
Neutrophils	9%
Lymphoblastes	7%
Lymphocytes	81%
Monocytes	3%
Thrombocytes (Plt)	$160x10^{9}$/l

The Botkin-Humprecht bodes are found

Hemogramm № 18

Hemoglobin (Hgb)	110 g/l
Erythrocytes (RBC)	$3,6x10^{12}$/l
Leucocytes (WBC)	$2,4x10^{9}$/l
Basophils	0%
Eosinophils	0,5%
Neutrophiles:	
myeloblastes	75%
myelocytes	0%
methamyelocytes	0%
band forms	2,5%
neutrophils	10,5%
Lymphocytes	7%
Monocytes	4,5%
Thrombocytes (Plt)	$140x10^{9}$/l

Hemogramm № 19

Hemoglobin (Hgb)	90 g/l
Erythrocytes (RBC)	$3,6x10^{12}$/l
Leucocytes (WBC)	$34,0x10^{9}$/l
Platelets	$180x10^{9}$/l
Basophils	0%
Eosinophils	8%
Neutrophiles:	
Promyelocytes	0%
myelocytes	0%
methamyelocytes	3%
band forms	14%
segmented	60%
Lymphocytes	12%
Monocytes	3%

Hemogramm № 20

Hemoglobin (Hgb)	100 g/l
Erythrocytes (RBC)	$3,6x10^{12}$/l
MCH	9
Leucocytes (WBC)	$23,9x10^{9}$/l
Platelets	$490x10^{9}$/l
Eosinophils	5%
Basophils	3%
Neutrophiles:	
Promyelocytes	2%
myelocytes	3%
methamyelocytes	6%
band forms	14%
segmented	44%
Lymphocytes	18%
Monocytes	5%

Hemogramm № 21

Hemoglobin (Hgb)	85 g/l
Erythrocytes (RBC)	$3,0x10^{12}$/l
Leucocytes (WBC)	$30x10^{9}$/l
Basophils	2%
Eosinophils	2%
Neutrophiles:	
myeloblastes	4%
promyelocytes	7%
myelocytes	9%
methamyelocytes	10%
band forms	20%
neutrophils	20%
Lymphocytes	19%
Monocytes	2%
Thrombocytes (Plt)	$270x10^{9}$/l
Reticulocytes	1,0%

Hemogramm № 22

Hemoglobin (Hgb)	80 g/l
Erythrocytes (RBC)	$2,7x10^{12}$/l
Leucocytes (WBC)	$85x10^{9}$/l
Basophils	3%
Eosinophils	10%
Neutrophiles:	
myeloblastes	2%
promyelocytes	5%
myelocytes	16%
methamyelocytes	24%
band forms	10%
neutrophils	15%
Lymphocytes	12%
Monocytes	3%
Thrombocytes (Plt)	$330x10^{9}$/l
Reticulocytes	0,3%

Hemogramm № 23

Hemoglobin (Hgb)	56 g/l
Erythrocytes (RBC)	$1,3xl0^{12}$/l
Leucocytes (WBC)	$14,1xl0^{9}$/l
Platelets	few
Neutrophiles:	
myeloblastes	0
promyelocytes	4%
myelocytes	0
methamyelocytes	0
band forms	0%
neutrophils	
Lymphocytes	74%
Band	2%
Segmented	8%
Monocytes	5%
Reticulocytes	0
Lymphoblasts	5%
ESR	52 mm/h

Hemogramm № 24

Hemoglobin (Hgb)	80 g/l
Erythrocytes (RBC)	$2,5x10^{12}$/l
MCH	9
Platelets	$80x10^{9}$/l
Leucocytes (WBC)	$0,85xl0^{9}$/l
Basophils	0
Eosinophils	0
Neutrophiles:	20%
myeloblastes	8
promyelocytes	0%
myelocytes	0
methamyelocytes	0
band forms	
neutrophils	
Lymphocytes	53%
Monocytes	12%
Reticulocytes	2

Hemogramm № 25

Erythrocytes (RBC)	$3,4x10^{12}$/l
Hemoglobin (Hgb)	100 g/l
Reticulocytes	0,6%
Leucocytes (WBC)	$30,0x10^{9}$/l
Basophils	2%
Eosinophils	6%
Neutrophiles:	
myeloblastes	0%
methamyelocytes	0%
band forms	5%
neutrophils	27%
Lymphocytes	11%
Monocytes	6%
Lymphoblasts	43%
Platelets	$200x10^{9}$/l

Hemogramm № 26

Erythrocytes (RBC)	$3,7x10^{12}$/l
Hemoglobin (Hgb)	95g/l
Reticulocytes	3%
Leucocytes (WBC)	$2,9x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	
Myeloblastes	0%
Promyelocytes	0%
methamyelocytes	0%
band forms	2%
neutrophils	21%
Lymphocytes	12%
Monocytes	8%
Monoblastes	57%
Platelets	$110x10^{9}$/l

Hemogramm № 27

Hemoglobin (Hgb)	80 g/l
Erythrocytes (RBC)	$3,4x10^{12}$/l
Reticulocytes	0%
Leucocytes (WBC)	$2,2x10^{9}$/l
Basophils	0%
Eosinophils	2%
Neutrophiles:	
myeloblasts	35%
promyelocytes	1%
myelocytes	0%
methamyelocytes	0%
band forms	4%
segmented	10%
Lymphocytes	40%
Monocytes	8%
Platelets	$5,0x10^{9}$/l

Hemogramm № 28

Hemoglobin (Hgb)	100 g/l
Erythrocytes (RBC)	$3,1x10^{12}$/l
Leucocytes (WBC)	$76x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	
myeloblastes	92%
myelocytes	0%
methamyelocytes	0%
band forms	0%
neutrophils	5%
Lymphocytes	2%
Monocytes	1%
Thrombocytes (Plt)	$80x10^{9}$/l

Hemogramm № 29

Hemoglobin (Hgb)	68 g/l
Erythrocytes (RBC)	$3,8x10^{12}$/l
Leucocytes (WBC)	$5,4x10^{9}$/l
Basophils	1%
Eosinophils	2%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	1%
neutrophils	34%
Lymphoblastes	5%
Lymphocytes	50%
Monocytes	7%
Thrombocytes (Plt)	$280x10^{9}$/l
Reticulocytes	1,2%

Hemogramm № 30

Hemoglobin (Hgb)	100 g/l
Erythrocytes (RBC)	$3,4x10^{12}$/l
Leucocytes (WBC)	$30x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	
myeloblastes	85%
myelocytes	0%
methamyelocytes	0%
band forms	0%
neutrophils	9%
Lymphocytes	6%
Monocytes	0%
Thrombocytes (Plt)	$100x10^{9}$/l
Reticulocytes	0,2%

Hemogramm № 31

Leucocytes (WBC)	$30x10^{9}$/l
Basophils	2%
Eosinophils	12%
Neutrophiles:	
myeloblastes	4%
myelocytes	4%
methamyelocytes	4%
band forms	10%
neutrophils	37%
Lymphocytes	18%
Monocytes	8%

Hemogramm № 32

Hemoglobin (Hgb)	60 g/l
Erythrocytes (RBC)	$0,8x10^{12}$/l
Leucocytes (WBC)	$2,0x10^{9}$/l
Basophils	0%
Eosinophils	0%
Neutrophiles:	
myelocytes	0%
methamyelocytes	0%
band forms	0%
neutrophils	28%
Lymphocytes	14%
Monocytes	8%
Monoblastes	50%

Hemogramm № 33

Hemoglobin (Hgb)	90 g/l
Erythrocytes (RBC)	$3,8x10^{l2}$/l
Leucocytes (WBC)	$18,5xl0^{9}$/l
MCH	7
Basophils	0
Eosinophils	0
Neutrophiles:	
myeloblastes	0
myelocytes	0
methamyelocytes	0
band forms	0
segmented	4%
Lymphocytes	88%
Monocytes	8%
Reticulocytes	0,3%

Few lymphoblasts are present

LITERATURE:

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LESSON № 23

Topic: DISORDERS OF HEMOSTASIS. THROMBOPHILIC DISORDERS OF HEMOSTASIS

Aim of the lesson: to study causes and mechanisms of thrombosis and embolism development.

QUESTIONS:

1. Hemostasis, its components (blood vessels, platelets, plasma coagulation factors).
2. Description of vessel-platelet hemostasis.
3. Description of hemocoagulate hemostasis.
4. Mechanisms of white red thrombus development
5. Disorders of hemostasis.
6. Causes and conditions of thrombosis development. Virchow's triad:
   - vascular injury,
   - blood coagulant system activation,
   - blood flow decrease.
7. Thrombophylic syndrome: its main causes, mechanisms of development, manifestations, outcomes and consequences.

Laboratory work. Thrombelastography (TEG)

Work description: Thrombelastography (**TEG**) is a method of testing the efficiency of coagulation in the blood. The method is performed on the Thromboelastometer at constant temperature of 37°C.

A small blood sample (typically 0,36 ml) is placed into a cuvette (cup) which is rotated gently through 4º 45´ (cycle time 6/min) to imitate sluggish venous flow and activate coagulation. When a sensor shaft is inserted into the sample a clot forms between the cup and the sensor. The speed and strength of clot formation is measured in various ways (now usually by computer), and depends on the activity of the plasmatic coagulation system, platelet function, fibrinolysis and other factors which can be affected by illness, environment

and medications.

The patterns of changes in strength and elasticity in the clot provide information about how well the blood can perform hemostasis (the halting of blood flow), and how well or poorly different factors are contributing to clot formation.

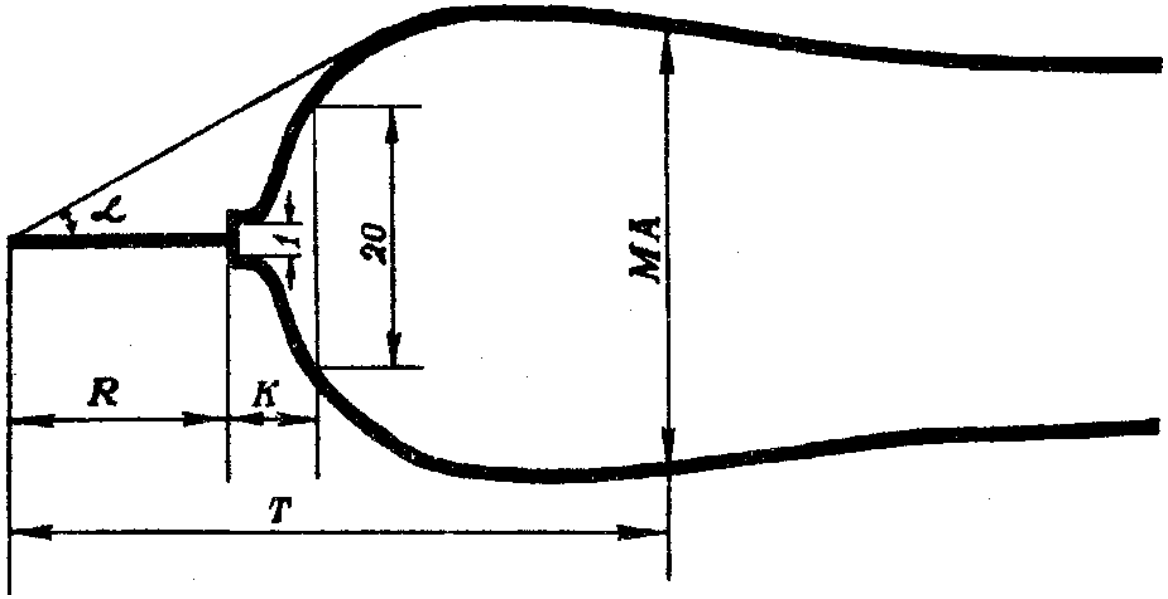

Fig. 24 – Outline of thrombelastogamm and its indices
(the indices are described lower in the text)

Four values that represent clot formation are determined by this test:

R – value (or reaction time). The R value represents the time until the first evidence of a clot is detected.

K – value (thrombin time). The K value is the time from the end of R until the clot reaches 20 mm and this represents the speed of clot formation. The angle is the tangent of the curve made as the K is reached and offers similar information to K.

MA – maximum amplitude. The MA is a reflection of clot strength.

T – total time of blood coagulation (5-10 min normally).

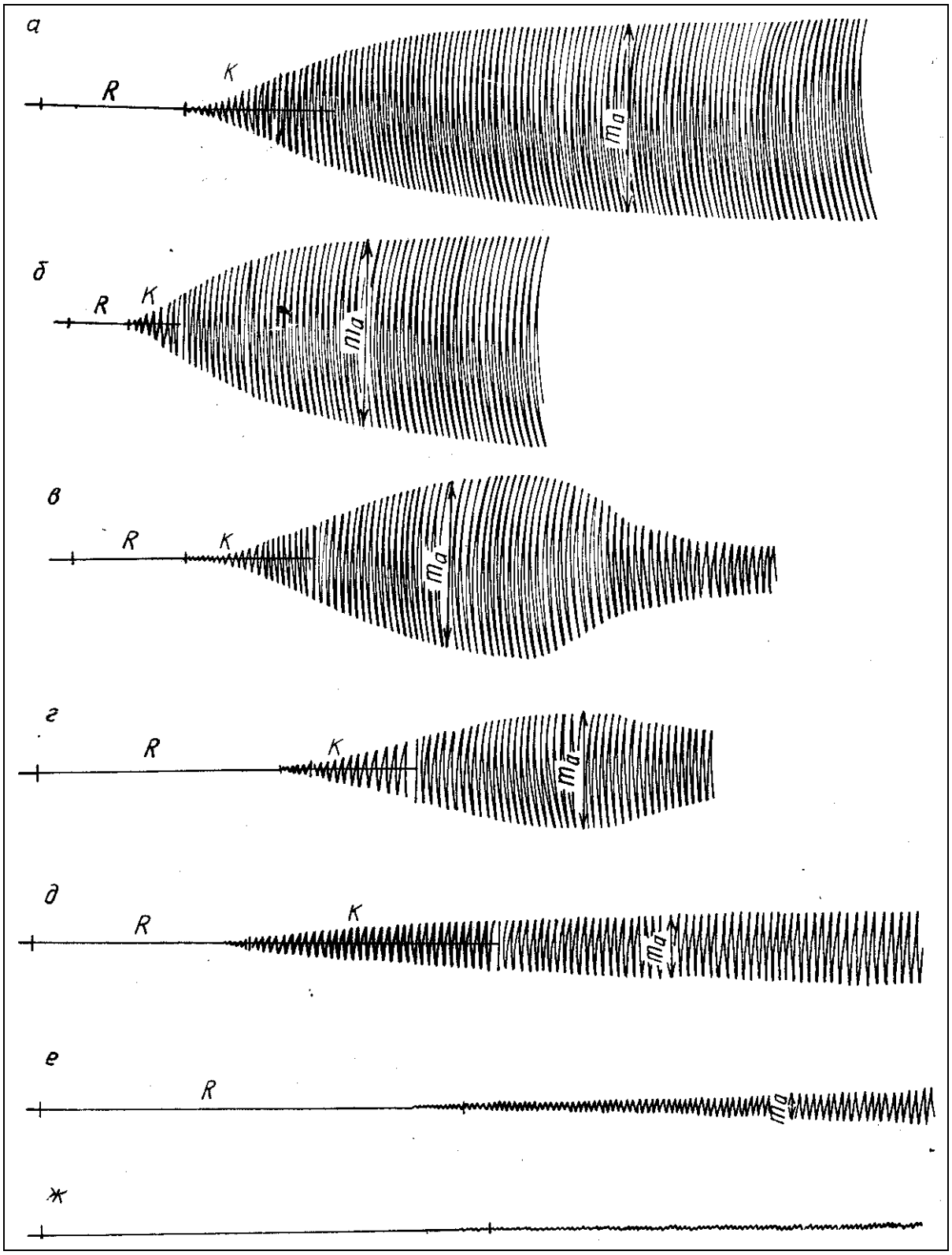

Fig. 25 – Thromboelastogramms
a – healthy
b – hypercoagulation phase of the DIC-syndrom
c – connecting phase of DIC-syndrom;
d–e – hypocoagulation phase of the DIC-syndrom

LITERATURE:

1. Lecture material.

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LESSON № 24

Topic: DISORDERS OF HEMOSTASIS. HEMORRHAGIC DISORDERS OF HEMOSTASIS

Aim of the lesson: to study hemostatic disorders with hemorrhagia, to meet the methods of hemostatic investigation.

QUESTIONS:

1. Hemorrhagic disorders of hemostasis. Causes, mechanisms of development, manifestations and consequences.
2. Disorders of blood vessels. Reasons and mechanisms of their development, clinical manifestations:
   - scurvy,
   - Henoch-Schonlein purpura,
   - Rendu-Osler-Weber syndrome.
3. Thrombocytopenia and qualitative disorders of platelets. Von Willebrand's disease. Reasons and features.
4. Coagulation disorders. Categorization. Features.
   - coagulation disorders of the 1-st phases of blood coagulation. Hemophilia.
   - coagulation disorders of the 2nd phases of blood coagulation.
   - coagulation disorders of the 3d phases of blood coagulation.
5. Thrombo-hemorrhagic conditions. Disseminated intravascular coagulation (DIC-syndrome). Reasons and mechanisms of development. Stages of DIC-syndrome. Manifestations and consequences.
6. Laboratory parameters used to characterize hemostatic disorders: bleeding time, prothrombin time, partial thromboplastin time.

Laboraroty work 1. ***Calculation of thrombocytes amount in blood***

Work description. The students investigate blood samples painted by Romanovsky-Gymza in seporated area. Thrombocytes look like small violet points between erythrocytes. These points should be calculated per 1000 RBC, the level of which has already been detected. The sum of platlets (A) is used for thrombocytes amount calculation according to the formula:

$$\text{Thrombocytes} = \frac{A \times RBC}{1000}$$, where

A – sum of platlets, estimated per 1000 RBCs,
RBC – erythrocytes level per liter.

Tasks

1

A 65-year-old patient was admitted to hospital with a diagnosis of myocardial infarction of the left ventricle and ischemic stroke associated with systemic atherosclerosis. On the next day his state was aggravated by the development of thromboembolism of the left popleteal artery and acute renal insufficiency with signs of uremia. On the third day of his stay in the hospital the patient's condition was complicated by the emergence of multiple subcutaneous hemorrhages and gastrointestinal bleeding. Blood tests showed marked thrombocytopenia, a considerable decrease in fibrinogen and prothrombin levels, a increased concentration of heparin and fibrin split products, and the enhancement of the blood fibrinolytic activity.

• What forms of pathology are obseved in this patient? Substantiate your answer.

2

Parents of a 3-year-old boy noticed that he often had inflammation of his knees and elbows. They told the doctor that their boy often has extensive nasal bleedings and develops large bruises after he falls down or hurts himself. During teething he had transient gums bleeding. On examination: physical and mental development of the boy is commensurate with his chronological age; knees and elbows have signs of inflammation (swelled, hyperemic, and tender). The results of the complete blood count test are unremarkable; coagulogram shows normal capillary bleeding time and prothrombin time, but a considerably increased partial thromboplastin time.

What pathology can ‘be suspected in the patient?

3

A 55-year-old female patient F. with decompensated insufficiency of :he aortic valve underwent an implantation of the prosthetic valve. During :he surgery extracorporeal circulation was employed. Three weeks later :he patient's condition deteriorated. She developed marked dyspnea, high fever; complained of pain in the heart. For this reason one more operation was performed to replace the current prosthesis and implant a new one Examination of the removed valve prosthesis showed thrombotic vegetations with microbial colonies on its

surface. A day later the patient died. A: autopsy: multiple focal hemorrhages in the brain and other organs, signs o: systemic vasculitis, mural thrombi; intravascular conglomerates of formal particles and fibrin sheath were observed.

• What disturbances of the blood clotting system are present in this case?

4

A 40-year-old patient A., an alcohol addict, was admitted to hospital with gastrointestinal bleeding and hematuria. On examination: the patient had a tint of jaundice in the skin and mucosal membranes, the hard and nodular liver which extended 2 cm below the right costal margin. The results of the complete blood count test were unremarkable. Blood biochemistry tests revealed an increased concentration of conjugated and unconjugated bilirubine, decreased levels of clotting factors II, VII, IX, X, an increased plasma aspartate transaminase (AST) activity, increased prothrombin time and partial thromboplastin time. Suspecting the vitamin K-dependent hypocoagulative state, the physician prescribed the patient the vitamin K containing medicine, but this treatment had no positive effect.

• What forms of pathology developed in the patient.

5

A 65-year-old patient H. with unresectable gastric carcinoma was admitted to hospital with signs of significant worsening of his condition. He complained of severe dyspnea after a minor physical exertion, pain on the rtght lateral side of the chest, nonproductive cough, and fever. Six hours after the admission he presented with signs of angina pectoris that were resistant to nitroglycerine, and also cerebral vascular insufficiency manifested by right sided hemiparesis. The patient was delivered to the intensive care room. When he was being transferred from the gurney to the bed he developed emesis with riood. Considering the risk of myocardial infarction and aggravation of the terebral vascular problems, the physician planned to treat the patient with mticoagulative and fibrinolytic drugs. Before the start of the antithrombotic :herapy immediate blood tests were performed, and the following results ^ere obtained: Hb 105 g/L, erythrocytes $3{,}5x10^{12}$/L, leukocytes $12x10^{9}$/L, piatelets $40x10^{9}$/L;

a decreased concentration of fibrinogen; an increased prothrombin time and partial thromboplastin time; a 50% decrease in innthrombin III blood level. Taking into account the blood test data the physician changed his mind about treatment schedule.

• What forms of pathology are observed in this patient? What is the cause – effect relationship between them?

6

Patient S. has been admitted to hospital with complaints of intense retrosternal pain. The use of nitroglycerin orally did not relieve it. Moreover, the patient's condition worsened, and he was transported to the intensive care _nit. Taking into account the risk of myocardial infarction, the physician .tedded to administer anticoagulant and fibrinolytic drugs to the patient. Before the start of anticoagulant therapy an immediate blood test was performed. The blood test data: Hb 105 g/L, erythrocytes $3{,}5x10^{12}$/L, ieukocytes $12x10^{9}$/L, platelets $80x10^{9}$/L, hypofibrinogenemia, an increase :n prothrombin time and partial thromboplastin time, antithrombin III concentration is decreased by 50% below normal values. Having considered these data the physician withdrew from administration of anticoagulants and fibrinolytics.

• What type of hemostasis disorder developed in the patinet?

7

A 22-year-old patient A. and a 25-year-old patient B. have come to see their physician with similar complaints of an easy formation of bruises and a prolonged bleeding after laceration. Patient A. noted often gums bleeding during cleaning the teeth, and both of them mentioned that after tooth extraction the wound was oozing more than a day. Patient B. also complained of swelling and inflammation of his knee joints, and pain in the knees during walking. Patient A. informed the physician that his mother has the same symptoms as he does, while the patient B.'s parents have no signs of hemostatic disorders. On examination: A. shows an increased capillary bleeding time; in B. this parameter is within the normal range. Patient B. has a significantly increased partial thromboplastin time (PTT), but in A. this parameter is normal. In both patients prothrombin time,

platelet count, prothrombin and fibrinogen content in the blood are within normal limits.

• What components of the hemostatic system are abnormal in these patients? What disorders can be suspected in patients A. and B.?

• What role does heredity play in manifestation of these diseases?

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LESSON № 25

CONTROL QUESTIONS ON PATHOPHYSIOLOGY OF BLOOD:

1. Blood, its composition and functions. Hematocrit.
2. Categorization of disorders of circulative blood volume (hypervolemia, hypovolemia).
3. Hypervolemia. Types (simple, polycythemic, oligocythemic). Causes and outcomes.
4. Hypovolemia. Types (simple, polycythemic, oligocythemic). Causes and outcomes.
5. Bleeding. Types and causes. Pathogenesis and main clinical symptoms of acute bleeding.
6. Compensatory-adaptative reactions after acute bleeding. Stages of compensation (reflect, hydremic, bone-marrow).
7. Parameters of severity of bleeding. Therapy of bleeding.
8. Anemias. Clinical symptoms of anemia and mechanisms of their development.
9. Categorization of anemias by: etiopathogenesis, color parameter, severity of anemia, regenerative possibility, type of hematopoiesis, erythrocyte's size.
10. Post-hemorrhagic anemia. Description, the picture of the blood under acute and chronic post-hemorrhagic anemia.
11. Iron deficiency anemia. Etiology, pathogenesis. Sideropenic syndrome. The picture of the blood.
12. Vitamin B_{12}, folic acid deficiency anemia. Etiology, pathogenesis. The picture of the blood. Addison-Biermer disease.
13. Hemolytic anemias. Types (congenital, autoimmune e.g.). The picture of the blood. Clinical symptoms.
14. Newborn hemolytic anemia.
15. Leukocytes. Stages of leucopoiesis. Leukocyte's description on different stages of maturation.
16. Leukocyte's functions (neutrophils, eosinophils, basophils, monocytes, lymphocytes).
17. Leukocytosis. Categorization. Causes of leukocytosis (neutrophilic, eosinophilic, basophilic, monocytic, lymphocytic).

18. Nuclear shift of leukocyte's formula to the left, to the right.

19. Leucopenia. Categorization. Causes and outcomes of neutropenia, lymphopenia. Agranulocytosis.

20. Leukocyte's formula. Leukocyte's formula in children.

21. Leukemia. The particularities of leukemic cells.

22. Characteristic of morphological picture of blood at acute and chronic myeloid and lymphoid leukemia.

23. Leukemia clinical syndromes.

24. Hemostasis, its components (blood vessel, platelets, plasma coagulation factors) and disorders of hemostasis (thrombophulic, hemorrhagic, thrombohemorrhagic hemostasiopathias).

25. Thrombosis. Outcomes and consequences. Mechanism of white and red thrombosis development.

26. Causes and conditions of thrombosis development. Virchow's triad (vascular injury, blood coagulant system activation, blood flow decrease).

27. Hemorrhagic disorders of primary hemostasis.

28. Disorders of blood vessels. Vasopathy. Hereditary hemorrhagic telangiectasia (Rendu-Osler-Weber Syndrome). Scurvy. Henoch-Schonlein disease or anaphylactoid purpura.

29. Thrombocytopenia. Causes. Autoimmune thrombocytopenic purpura.

30. Congenital disorders of platelet function. Clanzmann thrombasthenia.

31. Von Willibrand's disease.

32. Disorders of blood coagulation. Coagulopathies, classification. Causes.Hemophilias (A, B).

33. Disseminated introvascular coagulation (DIC). Causes. Stages.

34. Thromboelastogram at hypo- and hypercoagulation.

LESSON № 26

Topic: PATHOPHYSIOLOGY OF NERVOUS SYSTEM

Aim of the lesson: to study the causes and mechanisms of nervous cell damaging, consequence of organ denervation and pathogenesis of central and peripheral paralysis.

QUESTIONS:

1. General etiology, pathogenesis, and typical forms of nervous system disorders.
2. Neurone pathology causes. Parabiosis.
3. Disorders of synaptic transmission. Effects of different poisons. Myastenia gravis.
4. Denervation syndrome in somatic and internal organs. Disorders of trophism.
5. Disturbances of locomotion. Hypokinetic movement disorders: central and peripheral paralysis. The causes.
6. Disorders of the extrapyramidal system.
7. Hyperkinesias. Kinds. The causes.
8. Ataxia. Types and mechanisms.
9. Disturbances of sensation: general etiology and classification. Hypo- and hypersthesia, dysesthesia.
10. Localization of sensory abnormalities. Character of sensitivity infringements depending on the level of damages of various departments of the evaluator of sensitivity. Broun-Sequard syndrome.
11. Pain, its role in organism's ability to live. The causes and occurrence mechanisms. Kinds of pain (visceral and somatic), their characteristic. Cauzalgia. Nociceptive and antinococeptive systems.

Tasks:

1

Transection of dorsal white columnin of the spinal cord in lumbar area in a rat has caused back extremities deafferentation. Whether the muscle tone of the extremities will change? Whether proper muscle reflexes and autokinesias of extremities will be damaged?

2

A lethal dose of tetanic toxine was injected to an animal. What movement disorder will be observed in this case?

3

The sciatic nerve of the left back extremity in the middle third of the hip was cut in a dog. Ten days after operation the wound has completely healed with primary intenion. Achilles reflex is not provoked, reaction to pricks is absent. What paralysis has developed in the dog? Is restoration of movement function possible in extremity after denervation?

4

In experimental rats extremity paralysis was induced by injection of curare toxine into the gastrocnemius muscle. But the conduction of impulces through the nerve fibers retained as well as extremity muscles ability to respond to direct irritation. Explain, what mechanism of paralysis development is observed in this case. Which paralysis has developed?

5

In a dog the right half of spinal cord was transected at the level of the 2^{nd} -3^{rd} chest segments. Extremities of which side will be paralyzed? What sensitivity disorders have developed in this case?

6

In an experimental animal the cerebral cortex was destroyed in the area of posterior central gyrus on the right. How sensitivity in the experimental animal will be damaged?

7

Patient T, 32 years, complains of weekness in the right hand, difficulties in using it. Considers himself/herself a patient for 6 years when for the first time felt awkwardness of the hand, especially when writing. In the neurologic status: active movements of the right hand are limited, moderate atrophy of shoulder girdle and hand muscles. Strength of the hand is reduced, muscle tone is increased. Tendon and periostal reflexes of the right upper extremity are strong, their areas are extended. Sensitivity is not disturbed. What form motion activity disorder has the patient? What is approximate localisation of pathological process and possible mechanism of motion function disorder in this case? What caused the development of muscle atrophy of the right hand?

8

Patient L, 22 years, has arrived in clinic two years after the bachbone and spinal cord trauma. Recently the patient is troubled by leg muscles spastisity, so he can lie only on the back. Moderate muscles hypotrophy. Babinsky bilateral symptom. Which paralysis has developed in the patient and what is its possible mechanism? What are the mechanisms of muscles spasticity in this case?

9

A professor in linguistics, 59 years old, consults his doctor because of speech and movement problems. The patient is intellectually well functioning, but his speech has changed from motivating to a slow monotonous sequence of words. His gait is slow with small steps, and the standing position is difficult for this previous long distance runner. His facial expression is motionless, and he seems to have difficulties in initiating normal movements. There is tremor of the hands and fingers of the pill-rolling type. When the doctor examines the patient for rigidity, he finds high tonus (plastic rigidity) and cogwheel-movements.

1. What is the main pathological mechanism of this disease?
2. What is it called?
3. Why is the muscle tone so high?

10

A male of 65 years suddenly falls and is found in deep coma by the doctor. There is a left-sided hemiplegia with short arm-long leg as a flexion reflex. The paralysis and areflexia turns into spastic hemiparesis with a positive sign of Babinski. The deep stretch reflexes (patellar- and Achilles-tendon reflexes) are enhanced. There is loss of superficial reflexes (the abdominal and cremasteric reflexes). When the Achilles-tendon reflex is triggered it releases foot clonus. When the patient is awake from coma his facial nerve paresis is examined. He can knit his brows and turn his eyes upwards.

What is the pathophysiologic basis for this condition?

What are spasticity and foot clonus?

Is the facial nerve paresis central or peripheral?

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LESSON № 27

Topic: PATHOPHYSIOLOGY OF HIGHER NERVOUS ACTIVITY

Aim of the lesson: to study the causes and mechanisms of higher nervous activity, and pathogenesis of neuroses, narcomania and alcoholism.

QUESTIONS:

1. Classification of types of higher nervous activity. Basics of conditioning: conditioned and uncoditioned responses. A role of the generator of pathologically enhanced excitation, pathological system in excitatory system pathology.
2. Common etiology of higher nervous activity disorders.
3. Neurosis. Pathological basis of neurosis. Classification of neuroses. Characteristics. Manifestations.
4. Methods of experimental neuroses. Types of experimental neurosis: neurosis with domination excitation, neurosis with domination of inhibition, neurosis due to overexertion of the lability function.
5. Neurosis in humans: definition of the concept, characteristic features, relation to experimental neuroses. The main causes, prevalence, and predisposing factors of neurosis in humans. Information triad, its role in neuroses development.
6. The main types of neuroses: neurasthenia, hysteric neurosis, compulsory neurosis; their characteristic features and pathological basis.
7. Narcomanias. The causes. Effects of opiods on body systems. Narcotic abuse. Pathogenesis. Abstinent syndrome. Stages.
8. Influence of alcohol on human organism. Characteristics of manifestations of acute intoxication. Severity levels.
9. Alcoholism. Stages, characteristics of infringements. Abstinence syndrome. Mechanisms of occurence.
10. Infringements of intelligence, memory, emotions, behaviour, etc.
11. Alzheimer's disease.
12. Schizophrenia.
13. Characteristic of manifestations and causes of sleep-wakefulness cycle disorders.

Tasks

1

An outstanding Russian composer, 63 years of age, recovered from a cerebral insult. However, he could no longer understand spoken or written language, although his speech was fluent. The composer also maintained his ability to compose excellent music.

1. What is the name of this deficit in language function?

2. Where in the brain is the lesion localized and in what side of the brain?

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LESSON № 28

Topic: PATHOPHYSIOLOGY OF ENDOCRINE SYSTEM. PITUITARY GLAND AND ADRENAL GLAND FUNCTIONS DISORDERS

Aim of the lesson: to study the causes and mechanisms of hyper- and hypofunction of pituitary and adrenal glands.

QUESTIONS:

1. Nature of hormones. Mechanism of hormone action. Hormonal regulatory systems. A role of releasing-factors.
2. General etiology and pathogenesis of endocrine diseases. Causes of hormone excess and deficiency. Role of infringement of the central, gland and peripheric mechanisms in endocrine diseases.
3. Classification of endocrinopathias. Concept about central (secondary and tertiary) and peripheric (primary) endocrinopathias.
4. Pathology of hypothalamo-pituitary system.
5. Hyperpituitarism: types, causes, mechanisms of development and manifestations.
6. Hypopituitarism. Etiology and pathogenesis and manifestations. Partial and total insufficiency of an adenopituitary. Panhypopituitarism. The basic infringements and symptoms. The Sheehan's syndrome.
7. The diseases caused by infringement of formation of a somatotropic hormone. Acromegaly and gigantism hyperprolactinemia. Dwarfism. A role of somatomedins.
8. Typical forms of disorders of neurohypophysis. Syndrome of inappropriate antidiuretic hormone (SIADH) secretion pathology. Diabetes insipidus.Parchon (Swartz-Bartter) syndrome.
9. Pathology of adrenal gland. Acute and chronic insufficiency (Addisson disease). The causes, a pathogenenesis of infringements. Hypoaldosteronism.
10. Hyperfunction of adrenals' gland. Primary and secondary hyperaldosteronism. Conn´s disease. Pathogenesis and manifestations.
11. Itsenko-Cushing's disease and syndrome. Pathogenesis and manifestations.

12. Adrenogenital syndroms. Mechanisms of development and its basic manifestations.

13. Pheochromocytoma.

Tasks:

1

Patient A., 26 years woman, complained of general weekness, headaches, change of appearance, extension of hands and feet. For two years the size of footwear has increased from 39 to 42.

Objectively: integration of features (massive superciliary and zygomas, big nose, lips, ears) is marked.

The thorax is of the barrel-shaped form, thickened clavicles. Hands and feet are increased. Internal organs (liver and spleen) are increased. Pulse – 78 bpm, the AP – 150/90 mm Hg.

What endocrine pathology can be suspected?

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LESSON № 29

Topic: PATHOPHYSIOLOGY OF ENDOCRINE SYSTEM. PATOLOGY OF THYROID, PARATHYROID AND SEX GLANDS

Aim of the lesson: to study the causes and mechanisms of hyper- and hypofunction of thyroid, parathyroid and sexual glands.

QUESTIONS:

1. Thyroid gland. Synthesis and control of secretion of thyroid hormones. Physiological effects of thyroid hormones.
2. Thyroid gland pathology types, causes, mechanisms of development and clinical manifestations.
   a. hyperthyroidism. Graves' disease. Etiology. Pathogenesis of main symptoms.
   b. hypothyroidism. Etiology. Pathogenesis of main symptoms. Myxedema. Cretinism. Endemic goiter.
3. Parathyroid gland pathology types, causes, mechanisms of development and clinical manifestations.
   a. hyperparathyroidism. Etiology. Pathogenesis of main symptoms.
   b. hypoparathyroidism. Etiology. Pathogenesis of main symptoms.
4. Pathophysiology of sex glands. Causes, mechanisms of development and clinical manifestations.
   a. hypogonadism. Etiology. Pathogenesis of main symptoms.
   b. hypergonadism. Etiology. Pathogenesis of main symptoms.

Tasks:

1

An experimental dog from the moment of a birth got food with-

out of iodine. A year and a half later the thyroid gland mass of the dog has reached 100 g while the gland mass of the control dog receiving usual water was 1 g.

- How can you explain increasing of thyroid gland in the experimental dog?
- What is the pathology in which the thyroid gland increase occurs due to iodine insufficiency?

2

The parathyroid glands have been removed in the experimental dog.

- How will the calcium level variate in blood?
- How is the pathologic state, caused by this calcium level in blood called? How is the syndrome called?

3

Patient A, 16 years girl, complained on thyroid gland enlargement. Other complains are absent. For the first time she noticed the gland enlargement 4 years ago, a year later after moving to the present district. Examination detected goiter.

What is the struma? What are the main mechanisms of the struma development? Which goiter is involved in this case?

4

In a dog both adrenal glands have been removed. The day after adrenalectomy pathological changes (slackness, muscular delicacy, an anorexia, vomiting, anuria) have developed. Three days after the operation the animal died. Why the dog died?

5

A man, 49 years of age (height 1.86 m; weight 62 kg) is in hospital due to the following symptoms and signs. He is nervous and has a diffuse struma. A characteristic, blowing sound is heard from the thyroid gland with a stethoscope. The blood pressure is 145/70 mm Hg. An attack of cardiac arrhythmia is recorded with an ECG. A P-wave frequency above 400 per min is present during the attack. The concentration of thyroxine in blood serum is 180 nM. The distribution volume for radioactive thyroxine is 8 1. The elimination rate of this thyroxine is 14% of the total content per 24 hours.

Explain the condition of this patient**.**

6

A 22-year-old medical student is treated with an intravenous dose of PTH. This changes his renal excretion flux of two substances and their plasma concentrations.

1. Describe the alterations and explain the mechanisms.
2. What is the diagnosis?
3. Describe the most likely symptoms and signs of this patient before treatment.

7

A female (52 years of age; height 1.68 m; weight 62 kg) is in hospital due to her third attack of kidney stone pains. The first routine examination with arterial blood analysis reveals the following. Her blood pH is 7.21 and her plasma [Ca^{2+}] is 2 mmol per l (mM) in ionised form. Her ionised [Ca^{2+}] constitute 62% of the total calcium concentration in plasma. The inorganic phosphate concentration (total) is 0.84 mmol per l of plasma. The patient excretes 2-3 l of urine per day. $pK_2 = 6.8$ for H_3PO_4.

1. Could this condition be the result of a classical endocrine disease?
2. Why did this patient develop kidney stones? Was her diuresis normal? If not explain why.

8

A female, 62 years of age, suffers from pernicious anaemia for which she has received 1 (one) mg cyanocobalamine intramuscularly every 3.month for the last 10 years. At a routine visit the patient is found with a puffy swollen face due to a non-pitting oedema. Her skin is dry and cold, the heart rate is 55 beats per min, her hair is sparse, and she complains of constipation and fatigue. A series of blood tests reveals the following: High levels of microsomal autoantibodies against the thyroid gland and autoantibodies against her parietal cells. The TSH concentration in the plasma is high, whereas the T_4 is low. The haematological variables are satisfying.

1. What is the probable diagnosis?
2. Is there any connection between pernicious anaemia and the other condition?

9

A 24-year-old female is going through her last menstrual cycle before pregnancy.

Summarise schematically the most important hormonal events in her menstrual cycle. Summarise schematically the most important hormonal events during continued pregnancy and delivery.

10

A pregnant woman delivers oxygen to her foetus. Her A - haemoglobin (A = adult) is functionally different from that of her foetus (F - haemoglobin).

1. Why is this difference important? How are the two dissociation curves related?

2. FSH and LH are important for this woman. Describe why. Describe the function of the two hormones in her husband.

3. Following birth the mother breastfeed her baby and experience a feeling of sexual pleasure including uterine contractions. Describe the mechanism.

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LESSON № 30

Topic: INSUFFICIENCY OF BLOOD CIRCULATION. MYOCARDIAL INFARCTION

Aim of the lesson: to study the basic mechanisms of systemic blood flow disturbances, heart failure and compensative and decompensative reactions during myocardial infarction.

QUESTIONS:

1. Insufficiency of blood circulation. General characteristics.
2. Classification of blood circulation failure. Types of blood circulation insufficiency (acute and chronic, left- and rigthheart).
3. Hemodynamic abnormalities in heart failure. The basic haemodynamic indices characterising acute and chronic insufficiency of blood circulation.
4. Clinical symptoms of blood circulation insufficiency and substantiation of their development mechanisms.
5. Etiology, pathogenesis and main symptoms of acute heart failure.
6. Chronic heart failure. Etiology. Stages.
7. Adaptive reactions during acute and chronic heart failure: Frank-Starling mechanism, myocardial hypertrophy, redistribution of cardiac output, salt and water retention, activation of the sympathetic system.
8. Myocardial and nonmyocardial cardiac insufficiency.
9. Types and characteristics of overloading forms of heart insufficiency.
10. Mechanisms of compensation during chronic heart failure (the characteristic of cardiac and noncardiac mechanisms). Concept about heterometrical (isotonic) and homeometrical (isometric) indemnification mechanisms. Decompensation manifestations.
11. Myocardium hypertrophy. Hypertrophy stages by F.Meerson. Pathogenesis of the myocardium decompensation during hypertrophy.
12. The syndromes of coronary insufficiency: angina pectoris, myocardial infarction, chronic ischemic heart disease, sudden coronary death.

13. Myocardial infarction.

- etiology and pathogenesis. consequences.
- causes of death during myocardium heart attack.
- mechanisms of injury to the myocardial cells during ischemia.
- biochemical, mechanical and electrical changes elicited in the myocardium during ischemia. signs of reversible and irreversible ischemic injury.
- the causes of noncoronarogenic myocardial infarction.
- adaptive mechanisms developing during acute and chronic coronary insufficiency.
- complicatioms of myocardial infarction.

14. Cardiogenic shock and acute pulmonary edema. Etiology and pathogenesis.

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

Tasks:

1

Guinea pigs with experimental atherosclerosis have subjected to a long exercise stress – run in running track that has caused development of a heart attack of the myocardium. Describe the mechanism of the cardiac muscle lesion?

Themes of abstracts:

- Pathogeny of myocardium heart attack.
- Mechanisms of cardiogenic shock.

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LESSON № 31

Topic: PATHOLOGY OF ARTERIAL PRESSURE

Aim of the lesson: to study disorders of arterial pressure and their consequences.

QUESTIONS:

1. Factors involved in regulation of blood pressure.
2. Typical forms of disorder of systemic blood pressure.
3. Definition and classification of arterial hypertension. Severity degree of arterial hypertensia. Stages.
4. Classification of arterial hypertension. Primary and secondary hypertension. Essential hypertension: etiology and pathogenesis.
5. Risk factors. A role of endothelium dysfunction in pathogenesis of arterial hypertension.
6. Clinical manifestations of hypertension.
7. Consequences for organism (complication). Outcomes of hypertension.
8. Experimental hypertension. Classification.
9. Renal arterial hypertension: pathogenesis of renovascular, renoprival, renal parenchymal hypertension.
10. Pathogenesis of endocrinogenic and neurogenic arterial hypertension.
11. Pulmonary hypertension.
12. Essential arterial hypotension. Causes.
13. Types and mechanisms of development of symptomatic arterial hypotension.

Laboratory work 1. ***Ortostatic test***

It is used to examine functional state of vegetative nervous system, its sympathetic department. A patient lies for 5 minutes, his/her pulse is taken by 10-seconds intervals, the arterial preassure is measured too. Then the patient stands up and his/her pulse is taken for (10 seconds), the arterial preassure is measured. In normal excitability of sympathetic department is such, when the heart rate increases to 20-25% from the initial level. Higher figures indicate (unfavora-

ble) hyperexcitability of sympathetic department of the vegetative nervous system. The AP normally isn't significantly changed after standing up, in comparison to horizontal position. Systolic pressure is ±10 mm hg, diastolic ±5 mm Hg.

Laboratory work 2. ***Klinostatic test***

It is used to examine parasympathetic department of vegetative nervous system. After 5 minutes of adaptation in standing position the AP and pulse are measured, then a patient lies down. Pulse and the AP are measured again. Normally bradycardia during position changing is no more than 6-12 bpm while more significant bradycardia indicates the prevalence of parasympathetic influences. Systolic AP will be ±10 mm Hg pressure, diastolic will be ±5 mm Hg.

Tasks:

1

Hypertensia model was produced in the rabbit. Silver rings were put on renal arteries and caused their constriction and renal ischemia. Describe the mechanism of arterial pressure increase during renal bloodcirculation disturbance.

2

A male, age 50 years, visits an ophthalmologist in order to have measured new lenses for myopia and astigmatism. Ophtalmoscopy reveals irregular vessel diameter, bleeding, yellow-white spots, and papillary stasis. The patient is advised to see his general practitioner, which finds a constant arterial blood pressure of 200/110 mmHg (26.66/14.66 kPa). The heart frequency is 85 beats per min and the cardiac output at rest is normal. The patient is an office clerk, and also has a sedentary off- duty life. The patient is a heavy smoker using 40 cigarettes per day. His father had high blood pressure and died from cerebral infarction at the age of 62 years.An X-ray of thorax reveals clear lung fields and left ventricular hypertrophy.

1. What is the diagnosis?
2. What is the treatment of choice?
3. What is the main risk for this patient?
4. What happens in the lungs and the left ventricle of this patient?

3

A 59-year old office worker is known to have systemic hypertension. From the initial arterial pressure of 195/115 mmHg, he was brought down to a stable level of 160/95 mmHg by antihypertensive drugs. During work the patient suddenly collapses, and he is brought to hospital in an unconscious state with an arterial blood pressure of 75/45 mmHg. There are no signs of hemiplegia. Assume that the brain is hypoxic, and that the brain is producing lactic acid out of 30% of all glucose molecules combusted here. Among other values the blood glucose concentration is determined to 5 mM, and the arteriovenous glucose concentration difference increases to 300% of normal (0,5 mM). The cerebral bloodflow (CBF) is reduced to 50% of the normal value (650 ml min^{-1}). The total production by oxidative phosphorylation is 36 ATP per glucose molecule, and by anaerobic metabolism 2 ATP per glucose molecule.

What is the most likely diagnosis?

4

A female, 66 years of age, complains of frontal headache. She has been treated for migraine for the last 40 years. The new headache is different from migraine. The doctor measures her arterial blood pressure to 195/115 mmHg (25,9/15,3 kPa). By ultrasound screening the length of her left kidney is measured to be half the length of the right. Renal arteriography reveals a stenosis of the left renal artery. The stenosis is relieved by balloon dilatation, where a catheter with a balloon at its tip is inflated at the right site. The success of the treatment is confirmed over the following weeks, where her blood pressure reach a level of 145/95 mmHg (19,3/12,6 kPa).

1. What is the cause of her hypertension?
2. Explain the pathophysiological mechanism.
3. What is the most likely cause of her renal artery stenosis?

Themes of abstracts:

Etiology and pathogenesis of pulmonary hypertension.

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LESSON № 32

Topic: PATHOLOGY OF HEART RATE

Aim of the lesson: to study the types and mechanisms of arrhythmias and their consequences.

QUESTIONS:

1. Cardiac arrhythmias: common etiology, mechanisms of development. Reentry.
2. Classification of infringements of heart rhythm.
3. Automatism infringement. Types. Sinus rhythm pacemaker abnormalities (sinus tachycardia, bradycardia, arrhythmia).
4. Excitability infringement. Occurrence mechanisms. Types.
   a) extrasystoles
   b) atrial ectopic beats,
   c) atrial flutter,
   d) ventricular ectopic beats,
   e) ventricular fibrillation
5. Conductivity infringements.
6. Blockages. Types. The causes and pathophysiology mechanisms.
7. Blocks (intraatrial, sino-atrial, atrioventricular, intraventricular).
8. Hemodynamic consequences of heart rhythm disturbances.

Tasks:

1

A 17-year-old girl periodically feels palpitation attacks which last for several minutes. Pulse rate reaches 200 per 1 minute. Pulse is rhythmical. What heart disorder has developed? Describe its mechanism.

2

A 52-year-old patient S. had been admitted to the hospital's cardiology department. He had a history of syncope preceded by a

prolonged period: recurrent episodes of palpitations. They were usually accompanied by weakness, dizziness, and choking sensation. The day before the admission the patient incurred a severe psychological stress related to death of his close relative, who was heavy smoker, and suffered from ischemic heart disease.

On examination: blood tests the unremarkable. Holter ECG monitoring showed 11 episodes of arrhythmia lasting from 20 to 60 sec. During these episodes P wave was poorly exhibited and occasionally superimposes on QRS complex. The frequency of P wave was usually about 70 per minute: QRS complexes were regular and occurred at rates of 190 per minute; they were often distorted and look similar to ventricular extrasystoles. Episodes of arrhythmia where accompanied by a decrease in the systemic blood pressure.

• Define the form of heart pathology developed in the patient. Substantiate your answer.

• What are the possible causes of this pathology?

• What electrophysiological mechanisms underlie the ECG changes observed in the patient? What metabolic disorders in the myocardium could promote the observed ECG changes?

• Is there a risk of a sudden death during the described episode of heart dysfunction? If there is, what could be the immediate cause of death?

3

A 62-year-old patient K. experienced myocardial infarction in the posterosuperior area of the left ventricle and interventricular septum five days ago. Suddenly he felt weakness, dizziness, nausea, turned pale and lost consciousness. ECG shows regular atrial waves at rates of 109 per minute, and regular ventricular rhythm with a frequency of 42 per minute; there is no association between P wave and QRS complexes; systemic blood pressure is 65/50 mm Hg.

• Define the form of cardiac pathology developed in the patient. Substantiate your answer using the clinical data and ECG.

• What electrophysiological mechanism underlies this form of pathology?

• What metabolic changes and in what area of the heart can produce the electrophysiological disturbancies developed in the patient? Substantiate your answer.

• Describe the principles of adequate treatment in this case.

4

A 58-year-old patient M. was hospitalized three days ago with a diagnosis of myocardial infarction in the middle one third of the anterior wall of the left ventricle. Suddenly he felt weakness, dizziness, discomfort in the heart. ECG registered during the attack revealed regular atrial «sawtooth» waves at rates of 350 per minute; each two atrial waves were accompanied by the QRS complex of a normal shape. The systemic blood pressure was 90/50 mm Hg. The physician gave the patient an intravenous infusion of beta-adrenoblockers and calcium antagonists. Twenty minutes later the patient's condition improved. His blood pressure increased up to 110/75 mm Hg, but changes in ECG were still present, though the rate of atrial waves and QRS complexes decreased. The next day after meal a similar episode of cardiac disorder occurred. In this case pharmacological treatment had no effect. The patient was urgently transported to the intensive care unit.

• Define the type of cardiac disorder developed in the patient. Substantiate your answer using the clinical data and ECG.

• What electrophysiological disturbances and in what area of the heart underlie changes in ECG in this case? Substantiate your answer.

• What metabolic disorders in the myocardium could result in the indicated electrophysiological changes?

• Is the described episode of cardiac disorder lifethreatening? Explain why if it is.

Themes of abstracts:

Modern conception on mechanisms of rhythm adoption disorders and associated automatism and conductivity disorders.

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LESSON № 33

Topic: PATHOLOGY OF RESPIRATORY SYSTEM

Aim of the lesson: to study the mechanisms of obstructive and restrictive respiratory failure, pulmonary edema, periodic breathing.

QUESTIONS:

1. Pulmonary volumes and capacities used to characterize ventilation.
2. Types of disturbances of alveolar ventilation. Alveolar hyper- and hypoventilation: causes, mechanisms of development, consequences, and typical changes in breathing volumes. Obstructive and restrictive disturbances.
3. Diffusion, diffusion- and perfusion-limited gas exchange. Factors of importance for the lung diffusion capacity and pulmonary perfusion, and its measurements; the ventilation-perfusion ratio ($V°_A$ / Q° -ratio).
4. Diffusion infringements. Etiopathogenesis.
5. Perfusion disorders. Characteristic. Causes. Disorders of ventilation-perfusion matching.
6. Respiratory failure (etiology, causes, symptoms).
7. Asthma. Pneumothorax. Emphysema. Etiology. Pathogenesis.
8. Pulmonary hypertension.
9. Pulmonary water balance. Pulmonary edema.
10. Pneumothorax. Causes and consequences. Types of pneumothorax.
11. Pathological forms of breathing.
12. Pathogenesis of periodic breathing (Cheyne-Stokes, Biote, Kussmaul).
13. Respiratory distress syndrome.

Tasks:

1

Patient T., 19 years old. On the 3-rd day of the disease has arrived to the hospital with "acute pneumonia" diagnosis, and was hos-

pitalized. Respiratory rate was 32 per minute, superficial. Intercostal muscles participate in respiratory locomotions. Small bubbling and dry rales are heard during auscultation. Lungs radioscopy shows bilateral croupous pneumonia. Examination of external respiration efficiency detects decreased blood oxygenation – arterial blood saturation was 86 %.

- What form of external respiration disturbance is observed in the patient and what are the mechanisms of it development?
- What can cause decreasing of blood oxygenation in this case?

2

Patient K, 8 years old, complains on frequent asthma attacks without obvious causes. During the attack breath becomes heavy, accompanied by cough, secretion of some viscous mucoid sputum. Sibilant rales are heard during breathing. Bronchial asthma is in the anamnesis from the age of 5 years.

- What type of dyspnea is presented in this pathology?
- What type of a lung ventilation disorder takes place in this case during asthma attacks?

3

Patient V., 56 years old, arrived in neurology department due to cerebral stroke. The state is severe. The Chejn-Stoke type of periodic breathing is observed. What factor has major importance in the periodic breathing pathogenesis?

4

Pneumothorax types. Which pneumothorax is considered to be most (least) dangerous? Why? Which type (obstructive, restrictive) of ventilation disorder is observed in pneumothorax?

5

What is normal ventilation-perfusion factor? How should it calculated if: Minute Volume of blood flow = 5 liter; Tidal volume (BV) = 500 ml; Respiratory rate (RR) = 12 per minute; volume of dead space = 150 ml?

6

In a child with diphtheria pharyngeal edema is observed. What kind of respiratory insufficiency developes and why? What type of breath is detected in the child? Explain the mechanism of dyspnea in this case.

7

In one patient respiratory rate – 30, tidal volume – 300 ml; in another – respiratory rate – 15, tidal volume – 600 ml. Is the breath efficiency present in any patient? Prove RR.

8

A 35-year-old patient F. has a history of heavy smoking more than 1.5 packs of cigarettes per day for the last 12 years. He complains of recurrent bronchitis and tracheitis, persistent cough with secretions, and dyspnea during physical exertion. On examination: the patient has asthenic body habitus, the barrel chest; he uses accessory muscles during breathing. X-ray of the chest shows diminished lungs vascular markings, diaphragm depression and the bronchioles' walls thickening.

Arterial blood gases:

$Pa0_2$ – 85 mm Hg.

$PaC0_2$ – 45 mm Hg.

Oxygen carrying capacity – 18.0 vol%.

$Sa0_2$ – 94.1%.

The results of spirometry (% to normal values):

Total lung capacity – 114%.

Vital capacity – 78%.

Inspiratory reserve volume – 84%.

Expiratory reserve volume – 88%.

Functional residual volume – 110%.

Residual volume – 110%.

Tiffeneau ratio (FEV_1/FVC) – 85%.

The patient has signs of a diminished diffusive capacity of the alveolo-capillary membrane; his breathing rate is 20 per minute. After injection of bronchodilator drug aminophylline (euphylline) the value of Tiffeneau ratio increases by 7%.

Make a general conclusion about the condition of the respirato-

ry function in this patient.

9

A 34-year-old patient K., a mine worker, has been admitted to hospital with a preliminary diagnosis of silicosis. He complains of dyspnea, more severe on walking and in physical exercise; sustained cough (occasionally with secretions), chest pain.

Arterial blood gases:

$Pa0_2$ – 94 mm Hg.

$PaC0_2$ – 40 mm Hg.

Oxygen carrying capacity – 19.2 vol%.

$Sa0_2$ – 94.0%.

Spirometry:

Forced vital capacity (FVC) – 4.2 l.

FVC in % to normal – 94%.

Forced expiratory volume in 1 s (FEV,) – 2.6 l.

Tiffeneau ratio (FEV_1/FVC).

Pulmonary minute volume (% of normal) – 126%.

Additional data:

Breathing rate – 17 per minute.

The voluntary hyperventilation test yields the $Pa0_2$ value of 94 mm Hg. Make a conclusion about the type of respiratory system disorder in patient K.

10

A 59-year-old patient R. experienced myocardial infarction four weeks before. He complains of progressive dyspnea, accompanied by a cough with scanty blood-stained sputum. ECG demonstrates signs of the former myocardial infarction. Evaluation of the respiratory function shows the following:

Breathing rate – 24/min.

Forced vital capacity (% of the normal value) – 70.

Total lung capacity (% of the normal value) – 72.

Pulmonary minute volume (% of the normal value) – 145.

Tiffeneau ratio – 76%.

Make a conclusion about the state of the respiratory system in this patient.

11

A 65-year-old patient A. visited his physician with complaints

of choking sensation that emerges when he falls to sleep. Choking attacks are accompanied by fear of death and severe palpitations, which are sometimes followed by retrosternal pain radiating to the left scapula. The use of nitroglycerin during choking attacks and pain in the heart had no effect. However, if the patient wakes up and makes several deep voluntary inspirations, the unpleasant sensations subside. The blood test data and

ECG are unremarkable. The patient has a history of viral infection complicated by signs of polyneuropathy; in his childhood he had polymyelytis.

What form of pathology of the respiratory system does the patient suffer from?

12

A 36-year-old patient K. has been admitted to hospital with complaints of dyspnea, exacerbating on walking and in physical exercise and persistent cough. Examination shows cyanosis of the mucous membranes, rhonchi during auscultation of the lungs, hyperresonance on percussion of the chest. Testing of pulmonary function and arterial blood gases reveals the following:

Arterial blood gases:

$Pa0_2$ – 86 mm Hg.

$PaC0_2$ – 52 mm Hg.

Oxygen-carrying capacity – 19.6 vol%.

$Sa0_2$ – 94.2%.

Spirometry:

Total lung capacity – increased.

Forced vital capacity – decreased.

Inspiratory reserve volume – decreased.

Expiratory reserve volume – decreased.

Functional residual capacity – increased.

Residual volume – increased.

Tiffeneau ratio (FEV_1/FVC) – Decreased.

The voluntary hyperventilation test yields the $Pa0_2$ value of 86 mm Hg. Administration of bronchodilator drug aminophylline (euphylline) results in a significant increase in Tiffeneau ratio in this patient.

Make a conclusion about the state of respiratory function in this patient.

13

A 56-year-old patient M. has been repeatedly admitted to the therapeutic department of the hospital with complaints of dyspnea during moderate physical exertion, a nonproductive cough, episodes of choking, associated with cough and wheezing which is more marked on expiration. Testing of respiratory function and arterial blood gases reveals the following:

$Pa0_2$ – 94 mm Hg.

$PaC0_2$ – 32 mm Hg.

Pulmonary minute volume (% of the normal value) – 119%.

Forced vital capacity (FVC) – 3.6 l.

FVC (% of the normal value) – 86%.

Forced expiratory volume in 1 s (FEV_1) – 2,1 l.

Tiffeneau ratio – ?

Residual volume/total lung capacity (% of the normal value) – 110% Maximal expiratory flow rate (% of the normal value) – 98%.

After administration of bronchodilator drug aminophylline (euphylline) Tiffeneau ratio increases by 15%. The cough at the end of a wheezing episode produces thick, stringy mucus taking the form of casts of the distal airways (Curschmann's spirals). Blood test reveals the following: Hb 136 g/l, erythrocyte count $5.5xl0^{12}$/l, leukocyte count $9xl0^9$/l, eosinophilia. The chest roentgenogram shows increased retrosternal translucency.

Make a conclusion about the state of respiratory function in this patient.

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LESSON № 34

Topic: KIDNEY PATHOLOGY

Aim of the lesson: to study the causes and mechanisms of kidney failure and its consequences.

QUESTIONS:

1. Role of kidneys in human organism. Etiology and pathogenesis of kidney diseases.
2. Disorders of infringements of urine formation (filtration, reabsorption, secretion and excretion).
3. The causes and mechanisms of diuresis infringements. Quantitative and qualitative infringements of urine formation (oliguria, anuria, polyuria). Hypostenuria, isostenuria, hyperstenuria. The causes.
4. Infringements of urine composition. Pathological components of urine.
5. Pathogenesis of acute glomerulonephritis. Etiology, pathogeny and basic manifestations. Mechanisms of development of hypertension and hypostases in nephrites.
6. Consequences of chronic glomerulonephritis.
7. Pyelonephritis. Ethiopathogenesis. Characteristics of infringements.
8. Nephrotic syndrome. Clinical manifestations. Pathogeny of hypostases in nephrotic syndrome.
9. Pathogenesis of nephritic and nephrotic oedema.
10. Renal insufficiency. Renal insufficiency: definition, general causes, mechanisms and manifestations. Uraemia. Concept of hemodialysis (an artificial kidney).
11. Nephrolitiasis: causes, mechanisms of development, consequences. Factors and mechanisms promoting formation of stones.

Tasks:

1

Patient M., 16 years, arriveded to hospital with extremely severe shock. He was knocked down by a car. There are multiple fractures of both legs. The arterial preassure – 80/40 mm Hg, daily dieresis – 60-80 ml of urine per day. In urine: protein – 0,66 g/l, relative density of urine – 1,029. Residual nitrogen of blood – 120 mmol/l; blood urea – 35 mmol/l.

- What mechanism of anuria is observed in the patient?

2

Patient N., 36 years, was hospitalized in nephrology department. Renal disease was detected 2 years ago when after recent acute respiratory disease face edemas were developed and protein was detected in the urine.

The urine analysis: straw-yellow colour, transparent, acid reaction, protein – 0,8 g/l, glucose is absent.

Precipitate: small amount of epithelium, leucocytes – 1-5, erythrocytes – 2-3 in sight, fresh and variated; hyaline cylinders – individual in sight.

Zimnitsky test: comparative density of urine 1,004-1,015 at a diurnal diuresis of 2600 ml. In blood: urea – 5,1 mmol/l, kreatinine – 70 μmol/l.

- What pathological changes of urine composition are revealed in the patient?
- Are these signs indicate disturbance of renal filtration ability?
- What signs indicate disturbance of renal concentration ability?
- What is the possible mechanism of renal concentration ability disturbance in this case?

3

Patient K, 24 years old, fall ill acutely after catching a cold. He complains to general weekness face edemas, headache, dyspnea in insignificant exercise stress.

The urine analysis: red-brown colour, cloudy, sour reaction, protein – 1,2 g/l, glucose is absent.

Precipitate: moderate quantity of epithelium, leucocytes – 3-8, erythrocytes – 20-40, hyaline cylinders – 0-2 in sight, urates, uric acid.

Zimnitsky test: comparative density of urine 1,012-1,031 at a diurnal diuresis of 780 ml. Endogenic kreatinine clearance –

56 ml/minutes.

- What pathological components of urine are revealed in the patient?
- What signs indicate disturbance of renal concentration ability?
- What is the possible mechanism of renal concentration ability disturbance in this case?
- Are these signs indicate disturbance of renal concentration ability?

4

Patient N, 45 years old. Eight years ago he felt pain in lumbar area, moderate face and extremities edemas appeared. Five years later the exacerbation of the same symptoms occured. Later no symptoms were observed. Now the patient is hospitalised due to worsening of the general state. The urine analysis: yellow colour, acid reaction, protein – 0,6 g/l, glucose is absent, in deposit: moderate quantity of epithelium, leucocytes – 10-15 in sight, erythrocytes are single in a preparation, cylinders, waxy, acinose – 2-3 in sight.

Zimnitsky test: comarative density of urine 1,010-1,016 at diurnal diuresis of 860 ml.

In blood: urea – 9 mmol/l, kreatinine – 115 μmol/l.

- What pathological changes are indicated in the urine analysis?
- Are these data indicate glomerular filtration disorder?
- Are these data indicate renal concentration ability disturbance?

5

A 48-year-old patient K. has been suffering from chronic diffuse glomerulonephritis for five years. During the last several weeks he notes the emergence of the following symptoms: dull aching in the region of the heart, palpitations, generalized edema, especially prominent in the lower extremities on walking.

Urine test data: 24-h urine volume 1.100 ml, specific gravity 1.042, protein content 3.3%. The urinary sediment microscopic examination reveals the presence of a large amount of granular and waxy casts. Blood pressure 170/95 mm Hg. Blood biochemistry data: blood urea nitrogen 6.9 mmol/l (normal values: 3.3-6.6 mmol/l), total protein content 48 g/L (N: 70-80 g/l), albumin 15 g/L (N: 40-50 g/l), globulins 28 g/L (N: 20-30 g/L), hyperlipidemia, hypernatremia.

pH=7.34

What forms of pathology are observed in this patient? Are there signs of uremia in this patient?

6

A 35-year-old patient M. complains of generalized edema, more evident after sleep, back pains, fever, and body weight gain of 6 kg. These symptoms developed two weeks after he had had an acute sinusitis. His urine test results are as follows: 24-h urine volume 650 ml, specific gravity 1.028, protein content 0.1%, glucose and acetone are not detected. Microscopy of the urinary sediment reveals 40 erythrocytes per high-power microscopic field, a great amount of leukocytes, hyaline and erythrocyte casts in moderate quantities. Blood pressure 150/110 mm Hg. Blood test data: total protein content 73 g/l (N: 70-80 g/l), hypernatremia, hypokalemia. Endogenous creatinine clearance 50 ml/min (N: 120 ml/min). Acid-base status: pH=7.3. What forms of pathology can be distinguished in this patient? What is their cause-effect and chronological association? Does this patient have renal insufficiency?

7

A victim of the vehicle accident was transported to hospital five hours after the accident. He was examined by a physician of the emergency service who found multiple ribs fractures, contusions of soft tissues of the pelvis and lower extremities with extensive hemorrhages. On admission: the patient is confused, markedly pale, he has a thready pulse; blood pressure 60/20 mm Hg, periodic pattern of breathing. After a day of intense treatment by volume expanders (he received 3 l of polyglukin) and infusion of 0.5 L of blood his blood pressure increased to 110/60 mm Hg. On the next day after infusion therapy diuresis was still absent, and during the following three days the patient’s condition remained guarded. He complained of severe headache, dizziness, cyclic vomiting, inhibited state. The patient had short-termed episodes of seizures, edema of the skin, bradycardia and extrasystole on the ECG records; his diuresis was at the level of 150-250 ml/day, but blood pressure increased to 160/90 mm Hg. Blood test shows: BUN (blood urea nitrogen) 90 mg/dl (N: 20-40 mg/dl), hyperkalemia, hypermagnesimea, hyponatremia, hypo-

chloremia, pH – 7.30. Urine test: specific gravity 1.040, mild proteinuria, myoglobinuria; sediment shows the presence of casts and few leukocytes in the h.p.f.

On the 5-7th day in the hospital the patient developed great elevation of diuresis (up to 2.500 ml/day), and his condition markedly improved. Vomiting, seizures and headache ceased, and the extent of edema reduced. Repeated urinalysis shows: specific gravity 1.010-1.012, slight proteinuria, large quantity of granular casts in the urine sediment.

Define the type of renal syndrome in this patient. What are the mechanisms of symptoms observed on the 2-4th day after the trauma?

8

A 22-year-old female patient K. developed pain in the back, dyspnea, palpitations, and headache two weeks after she had had severe tonsillitis. She gained 9 kg of body weight in four days. On examination: paleness of the face, eyelids are edematous, the palpebral fissures are narrowed; trace pretibial and feet edema; the area of cardiac dullness is increased. Blood pressure 140/95 mm Hg; the 24-h urine volume significantly decreased; there is marked proteinuria. Microscopic examination of the urine sediment reveals the presence of a great amount of erythrocytes, leukocytes, granular casts. In the blood there are high titers of the antistreptolysin O and antihyaluronidase antibodies.

What forms of pathology developed in the patient?

9

A 33-year-old patient N. had acute diffuse glomerulonephritis four years ago. He is visiting his physician with complaints of recurrent headaches, malaise, dizziness, and moderate generalized edema. Urine test results: 24-h urine volume 3.600 ml, specific gravity 1.006, protein content 0.6%, glucose and acetone are not detected. Microscopic examination of the urine sediment shows the presence of occasional dysmorphic erythrocytes per high-power microscopic field, a small quantity of hyaline and waxy casts. Blood pressure 160/95 mm Hg. Blood test data: BUN 11.5 mmol/L (normal values: 3.3-6.6 mmol/L), total protein 56 g/L (N: 70-80 g/L). Endogenous creatinine clearance 50 ml/min (N: 120 ml/min). Acid-base status: pH=7.3.

What specific physiological processes in the kidneys are deranged to produce impairment of the excretory function in this patient?

• What are the possible causes and pathogenesis of each of these processes?

• Can we assume the presence of renal insufficiency in this patient?

• What are the mechanisms of polyuria, hyposthenuria, arterial hypertension, hyperazotemia, hypo- and dysproteinemia?

10

A 52-year-old patient M. who has suffered from chronic glomerulonephritis for 12 years visits his physician with complaints of recently appeared symptoms of drowsiness in the daytime and difficulty in falling asleep at night, fatigability, apathy, sensation of fullness in the retrosternal and epigastric regions, nausea, diarrhea, and itching of the skin. On examination: blood pressure is 165/95 mm Hg, the area of the cardiac dullness is increased to the left, pericardial friction rub over the whole area of the heart during auscultation. Blood test data: anemia, leukopenia, significant azotemia, hypo- and dysproteinemia. Endogenous creatinine clearance is 45 ml/min (N: 120 ml/min). Urine test data: 24-h urine volume 450 ml, specific gravity 1.029, proteinuria. Microscopic examination of the urine sediment shows the presence of 10 erythrocytes per high-power microscopic field, a great amount of leukocytes, occasional granular and waxy casts. Acid-base status: pH=7.32.

Define the condition developed in this patient.

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LESSON № 35

Topic: PATHOPHYSIOLOGY OF THE LIVER. JAUNDICE

Aim of the lesson: to study the causes and mechanisms of hepatic failure and jundance and their consequences.

QUESTIONS:

1. General causes and mechanisms of the liver dysfunction.
2. Investigation of the liver functions in the experiment. Functional trials.
3. Hepatitises. Etiology and pathogenesis.
4. Cirrhoses. Pathological regeneration. Portal hypertensia. Causes. Characteristic of portal hypertension. Its types.
5. Liver failure. Causes. Pathogenesis. Typical disorders of carbohydrate, amino acid, protein, and lipid metabolism during hepatic insufficiency; failure of detoxification function.
6. Fatty liver dystrophy. A role of alcohol and other factors in occurrence of liver diseases.
7. Hepatic coma. Types. Symptoms and mechanisms of their development. Therapy principles. Concept about haemosorption. Liver transplantation.
8. Jaundice. Types. The characteristic of bilirubine pigmental exchange infringements, clinical and laboratory manifestations in different types of jaundices (haemolytic, liver (parenchymal, hepatocellular), mechanical (obstructive)).
9. Cholemia. Basic mechanisms and manifestations of cholemia. Cholestasis. Causes of cholestasis. Disorders in human organism in cholestatic syndrome.
10. Gallstone disease. Etiology. Risk factors.
11. Primary enzymopathic jaundices: etiopathogenesis and manifestations of the Gilbert, Crigler–Najjar, and Dubin–Johnson syndromes.

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LESSON № 36

Topic: PATHOPHYSIOLOGY OF DIGESTION

Aim of the lesson: to study the causes and mechanisms of gastritis, peptic ulcer, pancreatitis, and disbacteriosis and their consequences.

QUESTIONS:

1. Role of digestive organs. Etiology of diseases of gastrointestinal tract. A role of alcohol, smoking and other factors in development of these disorders.
2. Appetite disorders (anorexia, hyperrexia, bulimia, polyphagia). Causes. Disorders of food intake.
3. Digestion disorders in oral cavity (mastications, salivations, swallowings). The causes and consequences. Caries. Ethiopathogenesis, preventive maintenance.
4. Esophageal functions disorders. Dysphagia. Achalasia. Gastro-oesophageal reflux.
5. Pathology of digestion in the stomach. Quantitative and qualitative disorders of stomach secretory function. Pathologic types of gastric secretion. Achlorhydria. Achylia. Disturbance of motor function.
6. Etiology and pathogenesis. Symptoms of gastritis. Nausea, vomiting.
7. Peptic ulcer. Etiology. Pathogenesis. Role of stress factors, Helicobacter pylori.
8. Disorders of digestion in the intestine (syndrome of maladigestion): disturbance of secretion, motor function, and absorption.
9. The syndrome of malabsorption: etiopathogenesis, manifestations and consequences.
10. Diarrhoea. Consequences.
11. Disbacteriosis. Etiology and pathogenesis. Consequences.
12. Pancreatitis. Etiology and pathogenesis.

Laboratory work 1. ***Role of pH in protein digestion in the stomach***

Work description. Two Petry cups with gastric juce are used for experiment. The 1st cup pH should be about 1,0 and in 2nd – about 7,0 (arrived by 0,1 l HCl or NaOH and controlled by lacmus paiper). The equal amount of bull fibrin is added to cups and after 15 min students fix results.

Students make conclusion about role of pH in protein digestion in thestomach.

Laboratory work 2. ***Detection of digestion ability of gastric juice of various acidity***

Work course: In Petri dishes we pour gastric juice with various acidity (increased and decreased). We put fibrin pieces in cups. We maintain constant temperature within 37^0C with the help of thermostat for an hour. We estimate a state of fibrine pieces during the experiment.

Tasks:

1

Hidrocortizonum at a dose of 0,5-1,0 mg per 100g of body weight was intramuscularly injected to rats with weight of 160-180 g. daily. After 10-15 injections in all animals developed erosions or ulcers in secretory department of the stomach. Explain the development mechanisms of «hydrocortizonum» stomach ulcers in experimental animals.

2

For modulation of experimental stomach ulcers the pylorus was ligatured with retention of its patency (Shay method). Explain the mechanism of stomach ulcer development after ligation of pyloric stomach department.

3

Patient G, 34 years old, presented to hospital for examination. Considers himself suffering for 4 months, when he felt «aching» pains in epigastrium, especially on an empty stomach. Fractional stomach contents research detected: a portion on an empty stomach – 140 ml, free muriatic acid – 40 t.u. (thousand units). A unit, total acid

it – 55 t.u. A unit of Bazal secretion: hour strain – 340 ml, free hydrochloric muriatic acid – 33-54 t.u. A unit, total acid it, – 48-72 t.u. A unit of, debit-hour of free muriatic acid – 8 milliequivalent. In reply to submaximum histamine stimulation hour strain level of secretion – 396 ml, free hydrochloric acid – 65-80 t.u, total acidity – 80-95 t.u, debit-hour of free hydrochloric acid – 12 millieqivalent. Characterise the secretory function of the stomach according received data.

4

Patient G, 53 years old, was hospitalised with complains to pains in epigastric areas, especially after food intake, air eructation, nausea, sometimes vomiting, absence of appetite. In fractional stomach contents research 10 ml of fluid with slime was extracted, free hydrochloric acid – 0, total acid – 10 t.u. are taken on an empty stomach. Bazal secretion: hour strain of 25 ml, free hydrochloric acid – 5-10 t.u. Total acidity – 15-20 t.u. Debit-hour of hydrochloric acid – 0,3 milliequivalent. In response to submaximum histamine stimulation: hour strain of secretion – 45 ml, free hydrochloric acid – 15-25 t.u. Total acidity – 30-40 t.u. Debit-hour of hydrochloric acid – 0,5 millieqiuvalent. Characterise the secretory function of the stomach according received data.

5

Patient Z., 63 years old, complains to pains in epigastric area. After food intake pain increases, feeling of heaviness is observed. Vomiting by eaten food is common. For last 3-4 months began more weaken and he loss weight considerably. Examination of stomach function by one-stage Boas-Evald method the following data is revealed. Fourty five minutes after a trial breakfast 180 ml of stomach contents was extracted. The flaking quotient, which is the ratio of dense layer (the grinded bread) to liquid one equals 1:5. Free hydrochloric acid – 10 t.u., total acidity – 35 t.u. A reaction to milk acid is positive.

What is the character of stomach secretory function disorder?

6

Patient M., 52 years old, was hospitalised in clinic for examination. About 3 months ago appetite worsened, disgust to meat appeared, started to loos weight. In the anamnesis 12 years felt unexpressed pains in epigastric area, heartburn. Fractional investigation of

gastric secretion detected: a portion of the empty stomach – 5 ml, free hydrochloric acid – 0, total acid – 35 t.u. A unit of Bazal secretion: hour strain – 25 ml, free muriatic acid – 0, the total acidity – 12-18 t.u. A unit in response to histamine stimulation: hour strain of secretion – 20 ml, free hydrochloric acid – traces, total acid – 15-21 t. unit.

What is the character of stomach secretory function disorder?

7

The rat preliminarily starving for 24 h, was immobilized and put in the cold chamber with temperature +4 C for 4 hours. Results of the rat's stomach contents analysis 24 h later: total acidity – 90 mmol/l, free HCl – 60 mmol/l. Dissection detected that the stomach mucosa was hyperemic, some erosions were detected too. Explain the mechanism of the observed changes.

8

Patient, 38 years old, complains of burning in the tongue, heaviness in the epigastric area, air eructation, diarrhoeia, fatigability, dyspnea. Objectively: skin and mucosa is pale, tongue is crimson. In gastric juice free HCl is absent. Total acid – 12 mmol/l. In excrement not digested muscular fibres are found.

Results of the blood analysis: erythrocytes – $2,1x10^{12}$/l, colour index> 0,5, anisopoikilocytosis. How can you explain dispeptic syndrome? What is pathogenesis of the observed syndrome? Calculate the colour index.

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Symbols And Units

1. Force is measured in Newton (N). *One Newton* (kgm s^{-2}) is the force required to accelerate a mass of 1 kg with an acceleration of 1 m s^{-2}. The acceleration due to gravity is generally accepted as g or G = 9.8067 or 9.807 m s^{-2}.

2. Joule established already in 1848 that *mechanical work* and *heat energy* were interchangeable. The commonly used unit of energy is the calorie (cal), which is the energy, required to raise the temperature of 1 gram (g) of water from 14.5 to 15.5^{o}C. Work is force times distance, and it is measured as Newton-meter or Joule (J). The *Joules equivalent* has been determined to be 4.187 J cal^{-1}.

3. Finally, *work-rate* or *power* is calculated as work per second (s). The power unit 1 W equals 1 J s^{-1}.

4. *Pressure* is measured as force per area unit that is in *N* m^{-2} or *Pascal*.

In the gravity field of the earth G or g equals 9.807 m s^{-2}. Blood and sea water has a relative density of 1033 kg m^{-3}. A 10 m high sea water column resting on one square m, corresponds to the following pressure: (10 m Ч 1033 kg m^{-3} Ч 9.807 m s^{-2}) =

101 306.3 (kg m s^{-2}) m^{-2}. This is *101 306.3 N* m^{-2} or *101.3 kPa* (= 1 atmosphere). The classical concept is that 1 atmosphere equals 760 mmHg. Accordingly, 1 Torr or 1 mmHg equals (101 306.3 Pa/760 =) *133.3 Pa*. In this book pressures are given in Pa (or kPa) together with mmHg.

5. *Concentration* is mass per volume unit. Squared brackets around a substance or **C** denote concentration. The international unit is mM = mmol l^{-1} = mol m^{-3}.

6. A prefix scale for different units is used as follows: milli = m = 10^{-3}; micro = μ = 10^{-6}; nano = n = 10^{-9}; pico = p = 10^{-12}; femto = f = 10^{-15}.

International Symbols

(Fed.Proc. 9: 602-605, 1950).

This is a precise short-cut for intellectual transfer used by all physiologists.

A *dash* next to any symbol ($\bar{}$) indicates a mean value. A *dot* next to any symbol ($\dot{}$) denotes a time derivative. *Small letters* in a suffix denote gas dissolved in blood, whereas *large letters* denote gas in air. The symbol is often the first letter in the English word.

A:		
α	=	Solubility: The Bunsen solubility coefficient (ml STPD per ml fluid per 760 mmHg)
A	=	Alveolar gas
AA	=	arachidonic acid
Ach	=	acetylcholine
ACTH	=	adrenocorticotropic hormone
Ad	=	adrenaline
ADH	=	antidiuretic hormone
ADP	=	adenine diphosphate
AIDS	=	acquired immunodeficiency syndrome
AMP	=	adenine monophosphate
AMPA	=	special glutamate receptors
ANF	=	atrial natriuretic factor
ANH/ANP	=	atrial natriuretic hormone/peptide
AP	=	action potential
AR	=	absolute refractory period
ASA	=	acetylsalicylic acid
ATP	=	adenine triphosphate
ATPS	=	ambient temperature, pressure, saturated with water vapour
AV node	=	atrioventricular node
B:		
BB	=	buffer base
BD	=	base deficit
BE	=	base excess
BMR	=	basal metabolic rate
BSA	=	body surface area
BTPS	=	body temperature and ambient pressure, saturated with water vapour
C:		
C	=	concentration of gas in blood. Squared brackets

		around a substance also denote concentration
Cal	=	calorie
C_{v_CO2}	=	concentration of CO_2 in mixed venous blood
CA	=	carbonanhydrase
cAMP	=	cyclic adenine monophosphate
CBF	=	cerebral bloodflow
CBG	=	corticosteroid binding globulin
CCh	=	carbacholine
CCK	=	cholecystokinin
cGMP	=	cyclic guanosine monophosphate
CNS	=	central nervous system
CSF	=	cerebrospinal fluid
COLD	=	chronic obstructive lung disease
COMT	=	catechol-O-methyl transferase
C peptide	=	connecting peptide
CRH	=	corticotropin releasing hormone
CVP	=	central venous pressure
D:		
D	=	diffusion capacity
Da	=	Daltons (MW units)
DAG	=	diacylglycerol
1, 25-D_3	=	1,25-dihydroxy-cholecalciferol
25-OH-D	=	25-hydroxy-cholecalciferol
DIT	=	di-iodine-thyronin
DM	=	Diabetes mellitus
DMNV	=	dorsal motor nucleus of the vagus
DMPP	=	dimethylphenylpiperazine
DNA	=	deoxyribonucleic acid
DOPA	=	dihydroxy-phenylalanine
2,3-DPG	=	diphosphoglycerate
DPPC	=	dipalmitoyl phosphatidylcholine
E:		
E	=	expiration
E_{net}	=	mechanical net-efficiency of external work
EAA	=	excitatory amino acids

ECG	=	electrocardiogram
ECF	=	extracellular fluid
ECV	=	extracellular fluid volume
EDIP	=	end-diastolic intraventricular pressure
EDRF	=	endothelium-derived relaxing factor
EDTA	=	ethylene-diamine-tetra-acetate
EEG	=	electroencephalogram
EF	=	excretion fraction
EGF	=	epidermal growth factor
e.p.	=	equilibrium potential
EPSP	=	excitatory postsynaptic potential
ER	=	endoplasmic reticulum
ERBF	=	effective renal blood low
ERPF	=	effective renal plasma flow
ERV	=	expiratory reserve volume
ESV	=	end systolic volume
F:		
F	=	fraction of gas in dry air or force
f	=	respiratory frequency (breath/min)
FABP	=	fatty acid binding protein
FAD	=	flavine adenine dinocleotide
$FADH_2$	=	flavine adenine dinucleotide (reduced)
FFA	=	free fatty acids
FGF	=	fibroblast growth factor
FRC	=	functional residual capacity (= RV + ERV)
FSH	=	follicle stimulating hormone
FU	=	Flow units in ml of blood (100 g tissue)$^{-1}$ min^{-1}
G:		
G	=	Gibbs energy (free, chemical energy)
GABA	=	gamma-aminobutyric acid
GFF	=	glomerular filtration fraction
GFR	=	glomerular filtration rate (normal 118-120 ml min^{-1})
GH	=	growth hormone
GHIH	=	growth hormone inhibiting hormone
GHRH	=	growth hormone releasing hormone

GIP	=	gastric inhibitory peptide or glucose-dependent insulin-releasing peptide
GLP	=	glucagon-like peptide
GnRH	=	gonadotropin releasing hormone
GLUT	=	glucose transporter
GRP	=	gastrin releasing peptide
GTP	=	guanosine triphosphate
H:		
H	=	heat content (enthalpy; all energy when the pressure-volume work is zero)
Hb	=	haemoglobin (haemoglobin F = foetal haemoglobin)
HBF	=	hepatic blood flow
hCG	=	human chorionic gonadotropin
HDL	=	high density lipoprotein
HGF	=	hepatocytic growth factor
HGH	=	human growth hormone
HIP	=	hydrostatic indifference point
HIV	=	human immunodeficiency virus
hPL	=	human placental Lactogen
HPLC	=	high pressure liquid chromatography
HSS	=	hepatocyte stimulating substance
I:		
I	=	inspired gas
ICSH	=	interstitial cell stimulating hormone
ICV	=	intracellular fluid volume
IDDM	=	insulin-dependent diabetes mellitus
IDL	=	intermediate density lipoprotein
IGF	=	insulin-like growth factor
IGF-BP	=	IGF-binding protein
IP_3	=	inositol triphosphate
IRV	=	inspiratory reserve volume
ISF	=	interstitial fluid (tissue fluid)
Iso	=	isoprenaline
ISS	=	interpreted signal strength
i.v.	=	intravenous
J:		

J	=	flux of a substance (mol min^{-1}) through an area unit
J	=	Joule
JG	=	juxtaglomerular
K:		
K	=	Kelvin degrees of temperature
L:		
LAT	=	lactic acid threshold
LBNP	=	lower-body-negative-pressure
LES	=	lower oesophageal sphincter
LH	=	luteinizing hormone
LHRH	=	luteinizing hormone releasing hormone
LPL	=	lipoprotein-lipase
LDL	=	low density lipoprotein
LTH	=	prolactin
LVET	=	left ventricular ejection time
M:		
MAO	=	monoamine oxidase
MAP	=	mean arterial pressure/mean aortic pressure
MeCH	=	metacholine
MEOS	=	microsomal ethanol oxidation system
MG	=	monoglycerides
2MG	=	2-monoglyceride
MIH	=	Muller inhibiting hormone
MIT	=	mono-iodine-thyronin
mM	=	mmol l^{-1}
MR	=	metabolic rate
MSH	=	melanocytic stimulating hormone
MW	=	molecular weight (in Daltons)
N:		
N	=	Newton
NA	=	noradrenaline
NAD	=	nicotinamide adenine dinucleotide
$NADH_2$	=	nicotinamide adenine dinucleotide (reduced)
NANC	=	non-adrenergic, non-cholinergic
NBB	=	normal buffer base/neutral brush border
NGF	=	nerve growth factor

NIDDM	=	non-insulin-dependent diabetes mellitus
NMDA	=	*N*-methyl-D aspartate
NOS	=	nitric oxide synthase
NSAID	=	non-steroid anti-inflammatory drug
P:		
P	=	partial pressure of gas in air or blood
PAH	=	para-amino hippuric acid
PCV	=	packed cell volume
PDE	=	phosphodiesterase
PDGF	=	platelet derived growth factor
PEF	=	peak expiratory flow
PG	=	prostaglandins
PG_2	=	prostacyclin
PGE_2	=	prostaglandin E_2
PIF	=	prolactin inhibiting factor
PIP_2	=	phosphatidyl-inositol diphosphate
P_B	=	barometric pressure
$P_{c'CO2}$	=	partial pressure of CO_2 in end-capillary blood
P_{IO2}	=	partial pressure of O_2 in inspired air in trachea
P_{aO2}	=	partial pressure of O_2 in arterial blood
POMC	=	pro-opiomelanocortin
PP	=	pancreatic polypeptide/ pulse pressure amplitude
PRL	=	prolactin
PRU	=	pressure resistance unit
PTH	=	parathyroid hormone
PVR	=	pulmonary vascular resistance
Q:		
$Q\circ$	=	Cardiac output (l min^{-1})
QRS	=	the ventricle complex of the ECG
R:		
R	=	ventilatory exchange ratio (pulmonic)
R	=	Gas constant
RAS	=	reticular activating system
RBF	=	Renal bloodflow
RC	=	respiratory controller/ respiratory centres
REM	=	rapid eye movements

RES	=	reticulo-endothelial system
RIA	=	radio-immuno assay
RMP	=	resting membrane potential
RNA	=	ribonucleic acid
RPF	=	renal plasma flow
RPM	=	revolutions per minute
RQ	=	respiratory quotient (metabolic)
RR	=	relative refractory period
RV	=	residual volume
S:		
S	=	entropy (the tendency to spread in a maximum space)
S	=	saturation degree
SA	=	specific activity
SAmode	=	sinoatrial node
SB	=	standard bicarbonate concentration
SBE	=	standard base excess
SDA	=	specific dynamic activity
SR	=	sarcoplasmic reticulum
SS	=	steady state/stimulus strength
STPD	=	standard temperature and pressure, dry (0^{o}C, 760 mmHg)
STN	=	solitary tract nucleus
sv	=	stroke volume
T:		
T	=	*t*ension (force)
T	=	temperature
T_3	=	Tri-iodo-thyronine
T_4	=	tetra-iodo-thyronine
TBA	=	thyroxine-binding albumin
TBG	=	thyroxine-binding globulin
TBPA	=	thyroxine-binding prealbumin
TBV	=	total blood volume
TCA	=	tri-carboxylic acid
TEV	=	total erythrocyte volume
TFGF	=	transforming growth factor
TG	=	triglycerides

TGF	=	tubuloglomerular feedback
TH	=	total haemoglobin content
TLC	=	total lung capacity (=RV+VC)
TP	=	threshold potential
TPVR	=	total peripheral vascular resistance
TRH	=	thyrotropin-releasing hormone
tRNA	=	transfer RNA
TSH	=	thyroid-stimulating hormone
TV	=	tidal volume
TxA2	=	thromboxane A2
V:		
v dash	=	linear mean velocity
V°	=	volume velocity of gas
V	=	volume
V°_{A}	=	expired alveolar ventilation (l min^{-1})
VC	=	vital capacity (=IRV+TV+ERV)
V_D	=	dead volume
W	=	Watts (J s^{-1})
W	=	external work (with pressure-volume work zero)

Essential Atomic And Molecular Weights

These are given in g mol^{-1} (or Daltons, Da) throughout the text. Calcium 40; Carbon 12; Glucose 180; Helium 4; Hydrogen 1; Nitrogen 14; Oxygen 16; PAH 194.2; Phosphorus 31; Potassium 39; Sodium 23; Xenon 131.

Physical Constants And Conversion Factors

Acceleration due to gravity (standard 1 G): 9.81 m/s^2.

Avogadro's constant: 6.02 10^{23} molecules mol^{-1} .

Diffusion coefficients for most molecules: 10^{-10} m^2 s^{-1} per molecule.

Energy (J = N m = Volts Coulomb): 1 cal = 4.187 J.

Faraday's constant: 96 487 (10^4) Coulomb/mol monovalent ion.

Molar gas constant (R): 8.31 J mol^{-1} per degree Kelvin (K).

Pressure (Pascal = Pa = N m^{-2}): 1 mmHg = 1 Torr = 133.3 Pa.

Temperature conversion between degrees of Fahrenheit (^{o}F) and degrees of Celsius (^{o}C): (^{o}F) = 9/5 (^{o}C) + 32.

Calculated Partial Pressures

The partial pressures of respiratory gasses are calculated in the alveoli and in the surrounding air of a healthy person, resting at sea level (101.3 kPa = 760 mmHg or Torr = 1 atmosphere).

The water vapour tension in a fluid (air or liquid) of the temperature 310 K (37^{o}C) is *6.27 kPa* or *47 mmHg*. At 293 K (20^{o}C) the tension is 2.4 kPa or 18 mmHg. The alveolar gas fractions are: F_{AO2} = 0.15, and F_{ACO2} = 0.056. The composition of atmospheric air is: F_{IO2} = 0.2093 and F_{ICO2} = 0.0003.

$P_{O2} = F_{O2}$ (101.3 – 6.27) kPa.

P_{AO2} = 13.3 kPa (100 mmHg); P_{aO2} = 12.7 kPa (95 mmHg); P_{vO2} = 6 kPa (45 mmHg).

P_{ACO2} = 5.3 kPa (40 mmHg) ; P_{aCO2} = 5.3 kPa (40 mmHg); P_{vCO2} = 6.1 kPa (46 mmHg).

CONTENTS

Учебное издание

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ПАТОЛОГИЧЕСКАЯ ФИЗИОЛОГИЯ

Пособие
для студентов факультета иностранных учащихся
с английским языком обучения

С приложением компакт-диска

2-е издание, дополненное и переработанное

PATHOPHYSIOLOGY

Manual
for the faculty of foreign students (in English)

Revised and updated 2nd edition

With the compact disk appendix

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