MINISTRY OF HEALTH OF UKRAINE
ODESA NATIONAL MEDICAL UNIVERSITY

Faculty **Medicine**

Department **General and Clinical Pathophysiology**

CERTIFY

Vice-rector for scientific and pedagogical work
Profesor _____ Svitlana KOTYUZHYNSKA

01 September 2022

MANUAL
FOR SEMINAR CLASSES
FROM EDUCATIONAL DISCIPLINE

Faculty **Medicine**, course 3, 5

Educational selective discipline – "**Clinical neurophysiology and electrophysiological background of diagnosis"**

Certify:
At the meeting of V.V. Pidvysotsky General and Clinical Pathological Physiology Department of Odesa National Medical University

Protocol N 1 dated 31 August 2022

Head of Department _____ profesor Rooslan VASTYANOV

Developers:
_____ prof. R.S. Vastyanov
_____ prof. S.G. Kotyuzhynska
_____ Asst. Prof. I.O. Ostapenko
_____ Ass. Prof. I.A. Kuzmenko
_____ Ass. Prof. V.P. Babiy
_____ Ass. Prof. D.Ye. Lapshin
_____ Ass. Prof. O.M. Pospelov
_____ Ass. Prof. L.V. Goncharova
_____ Asst. Prof. V.V. Kirchev
_____ Asst. Prof. V.M. Sarakhan

PRACTICAL TRAINING

Seminary lesson number 1

Topic 1. Mechanisms of pathogenic action of environmental factors : physical, chemical, biological. Local and general changes in cells and organs in pathogenesis , manifestations

Seminary lesson number 2

Topic 2. Disturbance of the structure, function and metabolism of body cells during oxygen starvation. Pathological changes in muscle cells (skeletal and smooth muscles of organs and systems).

Seminary lesson number 3

Topic 3. Causes and pathogenesis of stress. General adaptive syndrome. Pathogenic effect on organs and systems.

Seminary lesson number 4

Topic 4. Electrocardiography. Its basics, graphic display. Methods of diagnosing disorders of automaticity and conduction of the heart.

Seminar session No. 5

Topic 5. Clinical pathophysiology and ECG diagnosis of heart failure arrhythmias.

Seminar lesson No. 6

Topic 6. Analysis of the ECG recording in hypertrophy of different parts of the heart, myocardial infarction

Seminar lesson No. 7

Topic 7. Electromyography. Pathogenesis and functional diagnosis of peripheral nervous systems and neuromuscular disorders .

Seminary lesson number 8

Topic 8. Pathogenesis and functional diagnosis of disorders of cerebral circulation: rheoencephalography .

Purpose: Acquisition by the student of higher education of knowledge and the formation of elements of professional competences in the field of medicine from the Clinical neurophysiology and electrophysiological foundations of diagnosis division:

Topic 1. Mechanisms of pathogenic action of environmental factors: physical, chemical, biological. Local and general changes in cells and organs in pathogenesis, manifestations

Topic 2. Disturbance of the structure, function and metabolism of body cells during oxygen starvation. Pathological changes in muscle cells (skeletal and smooth muscles of organs and systems).
Topic 3. Causes and pathogenesis of stress. General adaptive syndrome. Pathogenic effect on organs and systems.
Topic 4. Electrocardiography. Its basics, graphic display. Methods of diagnosing disorders of automaticity and conduction of the heart.
Topic 5. Clinical pathophysiology and ECG diagnosis of heart failure arrhythmias.
Topic 6. Analysis of the ECG recording in hypertrophy of different parts of the heart, myocardial infarction
Topic 7. Electromyography. Pathogenesis and functional diagnosis of peripheral nervous systems and neuromuscular disorders .
Topic 8. Pathogenesis and functional diagnosis of disorders of cerebral circulation: rheoencephalography .

Improvement skills and competencies acquired during study previous ones discipline.

The main ones concept:

Topic 1. Mechanisms of pathogenic action of environmental factors: physical, chemical, biological. Local and general changes in cells and organs in pathogenesis, manifestations
Topic 2. Disturbance of the structure, function and metabolism of body cells during oxygen starvation. Pathological changes in muscle cells (skeletal and smooth muscles of organs and systems).
Topic 3. Causes and pathogenesis of stress. General adaptive syndrome. Pathogenic effect on organs and systems.
Topic 4. Electrocardiography. Its basics, graphic display. Methods of diagnosing disorders of automaticity and conduction of the heart.
Topic 5. Clinical pathophysiology and ECG diagnosis of heart failure arrhythmias.
Topic 6. Analysis of the ECG recording in hypertrophy of different parts of the heart, myocardial infarction
Topic 7. Electromyography. Pathogenesis and functional diagnosis of peripheral nervous systems and neuromuscular disorders .
Topic 8. Pathogenesis and functional diagnosis of disorders of cerebral circulation: rheoencephalography .

Equipment : Multimedia presentations , tables .

Plan:

1. **<u>Organizational measures (greetings , inspection those present , message of</u>**

the topic, purpose of the lesson , motivation acquirers higher education of studying the topic).

2. **Control of the reference level of knowledge:**

Topic 1. Mechanisms of pathogenic action of environmental factors: physical, chemical, biological. Local and general changes in cells and organs in pathogenesis, manifestations

1. The pathogenic effect of environmental factors is mainly associated with the influence of physical factors: 1) mechanical trauma, 2) high and low temperature, 3) ionizing radiation, 4) high and low atmospheric pressure, 5) electric current, 6) of infrared and ultraviolet radiation.

Mechanical trauma is tissue damage by a solid body or the propagation of an explosive wave. Locally, the injury manifests as lacerations, punctures, fractures, crushing, or a combination thereof. Often, mechanical trauma is combined with blood loss, damage to nerve trunks and skin. The most severe general manifestations of trauma are traumatic shock and prolonged crushing syndrome (crash syndrome).

• Crush syndrome is a pathological process that develops in victims as a result of long-term (4-8 g or more) crushing of soft tissues of the limbs by fragments of destroyed buildings, structures, blocks of soil during collapses in mines, etc.

In the course of the crash syndrome, 3 periods are distinguished: 1) early (up to 3 days) with a predominance of shock phenomena; 2) intermediate (from the 3rd to the 12th day) with a preference for acute renal failure; 3) late (from the 8th-12th day to 1-2 months) - the recovery period, or the period characterized by the predominance of local symptoms. 3 pathogenetic factors are of great importance in the development of crash syndrome: a) painful irritation; b) traumatic toxemia caused by the absorption of toxic products of tissue autolysis from the site of injury; c) plasma and blood loss associated with edema and hemorrhages in the area of crushed or long-term ischemic tissues. Hyperthermia (overheating) develops when the body is exposed to high temperatures, and burns occur when local. Prolonged stay of the body in conditions of low temperature leads to the development of hypothermia, and the local effect of low temperature is the cause of frostbite.

In the development of hyper - and hypothermia, 2 stages are distinguished: 1) compensation and 2) decompensation. In the 1st stage, due to the protective and compensatory reactions of the body, the temperature of the core of the body does not change, despite the effect of thermal factors. If the specified reactions are insufficient, then the stage of decompensation occurs, the main feature of which is an increase in body temperature outside the normal range.

□ With hypothermia, protective and compensatory reactions develop in 2

directions:

1) Reactions aimed at limiting heat transfer: a) spasm of peripheral vessels; b) decrease in sweating; c) change in posture and other behavioral reactions that reduce the area of open body surfaces; d) increasing the heat-insulating properties of wool in animals due to the contraction of smooth muscles that raise hair (a rudimentary reaction - "goosebumps" - has been preserved in humans).

2) Reactions aimed at increasing heat production: a) increase in contractile thermogenesis (increased skeletal muscle tone, muscle tremors, voluntary movements); b) increase in non-contractile thermogenesis (strengthening of oxidative processes, dissociation of oxidation and phosphorylation).

The main feature of the decompensation stage is a decrease in the temperature of the body's core, which naturally leads to: a) a decrease in the speed of all biochemical reactions in the body, including the processes of biological oxidation; b) at the same time, the consumption of oxygen and the formation of ATP in cells sharply decreases; c) lack of energy leads to suppression of vital functions of the body: activity of the central nervous system, breathing, blood circulation, as a result of which oxygen starvation develops; d) hypoxia, in turn, causes an increase in ATP deficiency - a "vicious circle" closes, which can ultimately lead to death.

□ With hyperthermia, protective and compensatory reactions are aimed at increasing heat transfer. These include: 1) expansion of peripheral vessels; 2) increased sweating; 3) reactions aimed at increasing the area of the open surface of the body (change of posture, behavioral reactions); 4) thermal dyspnea in animals.

The main symptom of the 2nd period of overheating - the stage of decompensation is an increase in body temperature. It is accompanied by: a) a sharp disturbance of the CNS function, b) increased breathing, c) increased blood circulation and metabolism. Further increase in body temperature and overexcitation of the nervous centers ends in their exhaustion, which is accompanied by: a) breathing disorder, b) heart function and c) lowering of blood pressure; d) hypoxia develops.

As a result of profuse sweating: a) dehydration of the body occurs, b) electrolyte exchange is disturbed, c) thickening of blood develops and its viscosity increases, which creates an additional load on the circulatory system and contributes to the development of heart failure. Against the background of increasing phenomena of oxygen starvation, convulsions appear, death occurs.

Acute overheating with a rapid rise in body temperature is called heat stroke.

□ The complex of general changes in the body that occur with large and deep burns is called a burn disease.

The following stages of burn disease are distinguished: 1) burn shock, 2) burn toxemia, 3) burn infection, 3) burn exhaustion, 4) completion.

□ In the development of burn shock, the main role belongs to the pain factor. This, in particular, explains the very long erectile phase of shock (excitement phase).

□ Burn toxins appear in the body as a result of metabolic disorders, but most of them will be formed at the site of injury. Denatured protein and toxic products of its enzymatic hydrolysis enter the general bloodstream from damaged tissues.

□ Intoxication of the body is enhanced by infection. Its source is damaged tissues and intestinal contents. This complication is explained by a decrease in the barrier properties of the body, in particular, skin damage, a malfunction of the mononuclear phagocyte system, and a change in the protective properties of the intestinal mucosa.

Dehydration and disorders of electrolyte metabolism are of great importance in the pathogenesis of burn disease. Loss of proteins and fluid occurs mainly at the site of injury as a result of increased permeability of the vascular wall. Thickening of the blood and increase in its viscosity make it difficult for the blood circulation and the work of the heart.

□ Burn exhaustion is characterized by: a) progressive cachexia, b) edema, c) anemia, d) dystrophic changes in internal organs, e) complications (pneumonia, glomerulonephritis), f) insufficiency of the cortical substance of the adrenal glands.

In the process of recovery, the following occurs: a) complete rejection of necrotic tissues, b) filling of the defect with granulations, c) scarring, and d) epithelization .
• All types of ionizing radiation have a pathogenic effect: 1) corpuscular (α- and β-particles, neutrons, protons) and 2) electromagnetic waves (X-rays, γ-radiation).

There are: a) direct and b) indirect effects of ionizing radiation.

□ Direct action means the direct influence of the ionizing agent
radiation on macromolecules and supramolecular structures of cells. Energy
of ionizing radiation exceeding the energy of intramolecular and
of intraatomic bonds , causes: a) ionization of molecules, b) rupture the least
stable connections , c) formation of free radicals , etc.
Structural manifestations of the direct effect of radiation are damage to the genome of cells: a) breakage of chromosomes, b) cleavage of DNA, RNA molecules, c) swelling of organoids cells.
It was established that the direct effect of radiation causes about 45% of its total
biological effect.
□ The indirect effect of ionizing radiation is determined by formation in cells a large number of free radicals (hydrogen and hydroxyl radicals), the main the source of which is water molecules. Therefore, the process of formation of free radicals from water under under the influence of radiation received the name radiolysis of water.
Radiolysis of water:
$H_2O + e^- \rightarrow OH^- + H^{\bullet}$;
H_2O is $-\rightarrow OH^{\bullet} + H^+$
Formation of secondary free radicals:
$OH^{\bullet} + OH^{\bullet} \rightarrow H_2O_2$;
$H_2O_2 + OH^{\bullet} \rightarrow H_2O + HO^{\bullet}$
$H_2O_2 + HO^{\bullet}$

$2 \rightarrow H_2O + O_2 + OH\bullet$;
$H_2O_2 + H\bullet \rightarrow H_2O + OH\bullet$.

Free radicals formed interact with each other, as a result so-called secondary free radicals and hydroperoxides are formed . Theirs accumulation leads to rapid activation of free radical oxidation processes: a) nitrogenous bases of DNA and RNA, b) protein-enzymes (especially sulfhydryl groups that are part of the active centers), c) lipids, d) amino acids.

Indirect action accounts for 55% of the total biological effect ionizing radiation.

• Radiotoxins are products of free radical oxidation, which are formed in tissues under the action of radioactive irradiation. The greatest value in lipid radiotoxins (lipid hydroperoxides , epoxides , aldehydes, ketones), which are intermediate and final products of lipid peroxidation. They accumulate in membranes cells and disrupt their barrier properties.

In addition, in irradiated cells from some amino acids (tyrosine, tryptophan) quinone radiotoxins are formed, which suppress the activity many enzymes.

Radiotoxins, entering the blood from damaged cells, carry out pathogenic effect on organs and tissues distant from the place of their formation. Hereby it is possible to explain general disorders in the body with local radiation injuries

• The main factor that determines tissue sensitivity to ionizing radiation, are: a) the ability of cells to divide and b) the intensity of division. Thus, the highest radiosensitivity is possessed by tissues in which the processes of cell division are most pronounced intensively : 1) it is primarily hematopoietic and lymphoid tissue, 2) it is next epithelial tissue, especially the epithelium of the digestive tract and gonads, as well as covering epithelium of the skin, then - endothelium of vessels; 3) the last in this series - cartilage, muscle and nervous tissue.

The development of radiation damage is facilitated by: a) an increase in temperature, b) increase in oxygen tension, c) increase in water content in tissues. Under these conditions, it grows the rate of free radical reactions, and therefore the indirect effect of ionizing radiation.

On the contrary: a) a decrease in temperature, b) oxygen starvation and b) dehydration are present factors that slow down the development of radiation damage.

• In the human body, under normal conditions, there are protective and compensatory mechanisms aimed at prevention and elimination of radiation damage.

To protect cells from radiation damage, the most important are:

a) antioxidant systems and b) DNA repair mechanisms, which will be mentioned in in the following lectures.

In order to prevent or reduce the degree of development of radiation damage radio protectors are used. The most studied today are: a) antioxidants and b) drugs - donors of sulfhydryl groups. When using the latter have the effect of protecting their own SH-groups, which are part of the active ones centers of many enzymes.

• Acute radiation sickness.

Depending on the radiation dose, there are 3 forms of acute radiation diseases: 1) bone marrow (dose 0.5 - 10 Gy), 2) intestinal (10 - 50 Gy) and 3) brain (50 - 200 Gy).

□ In the bone marrow clinic, 4 periods are distinguished: 1) period primary reactions (1-2 days); 2) a period of imagined (imaginary) well-being (several days); 3) the period of pronounced clinical signs; 4) completion.

The period of pronounced clinical signs of acute radiation sickness is characterized by:

1) Hematological syndrome - manifested by pancytopenia (a decrease in the content in the blood of all formed elements). First of all, lymphocytes disappear from the blood (lymphopenia manifests itself already in the period of imaginary well-being). Then it decreases the content of granulocytes (neutropenia), then platelets (thrombocytopenia) and, finally, erythrocytes (anemia).

The development of pancytopenia is caused by red bone damage brain and the natural death of mature formed elements contained in the blood.

Since the lifespan of different blood cells is not the same, it initially decreases the content of short-lived formed elements (lymphocytes and neutrophils, and significantly later - erythrocytes.

2) Hemorrhagic syndrome - caused by: a) thrombocytopenia ; b) radiation damage to the endothelium of vessels; c) increasing vascular permeability walls under the action of biogenic amines (histamine , serotonin), which are released tissue basophils under irradiation conditions; d) Disturbance of blood coagulation in as a result of release of large amounts of heparin by tissue basophils.

3) Infectious complications - associated with: a) Disturbance of external barriers organism (damage to the covering epithelium of the skin, epithelium of the mucous membrane oral cavity, pharynx, intestines) and b) leukopenia, the result of which is a violation immune reactions of the body (immunological deficiency) and phagocytosis.

4) Autoimmune reactions - caused by the appearance of autoantigens in the irradiated tissues, which are their own tissue proteins, changed under the action of an ionizing agent radiation.

5) Asthenic syndrome - a complex set of clinical signs that occur in as a result of functional disorders of the central nervous system: a) general weakness, b) dizziness, c) fainting , d) sleepiness during the day, e) insomnia at night etc.

6) Intestinal syndrome - manifested by intestinal function disorders (diarrhea, spastic pains), as a result of damage to the epithelium of the mucous membrane.

□ The most important long-term effects of ionizing radiation on the body radiations are: a) mutations in sex cells and b) mutations in somatic cells.

The first many years after exposure may manifest themselves in the following ones generations by the development of hereditary diseases , the second by the emergence of malignant tumors (leukemia, cancer).

The effect of reduced and increased atmospheric pressure on the human body.

• A person will experience the influence of reduced atmospheric pressure on himself in moderation climbing to a height: in an airplane, in the mountains. Under these conditions, the following affect the human body pathogenic factors:

1) Inherent decrease in atmospheric pressure. This factor causes development decompression syndrome, manifested by pain in the ears and frontal sinuses as a result expansion of the air that fills their cavities; flatulence; bleeding from the nose due to rupture of small vessels.

2) A decrease in the partial pressure of oxygen in inhaled air is the cause development of oxygen starvation (hypoxia).

• A person will experience the effect of increased atmospheric pressure on himself immersion under water during diving and caisson work. At the same time, on the body the following pathogenic factors act on a person.

1) Inherent increase in atmospheric pressure (compression). This factor causes compression of the eardrums, resulting in pain in the ears. With a sharp and very rapid increase in atmospheric pressure, pulmonary rupture is possible alveoli In the conditions of compression in the blood and tissues of the body, additional dissolves amount of gases (saturation).

2) When breathing compressed air, nitrogen exerts its pathogenic effect, which manifests itself in a disturbance of the activity of the central nervous system: a) initially slight excitement, which resembles euphoria ("deep delight"), b) later - phenomena of narcosis and intoxication.

These violations are explained by the fact that as a result of saturation, the number nitrogen in the body increases several times, and most of all it accumulates in organs rich in adipose tissue, in particular, in the tissues of the brain, which contain a large amount of lipids. Nitrogen in high concentrations causes narcotic effects an action that resembles the action of nitrous oxide ("laughing gas").

To avoid the adverse effect of nitrogen in the breathing mixture, it is replaced helium Heliox mixture is obtained.

3) When atmospheric pressure increases, oxygen has a toxic effect. It due to the fact that in conditions of hyperoxia , the processes of free radical oxidation are activated, which cause cell damage.

In addition, with hyperoxia , the removal of carbon dioxide from tissues is disturbed, which causes their kind of "suffocation".

• Decompression sickness occurs when a person quickly returns to conditions of normal atmospheric pressure after diving work, work in caissons (caisson disease). At the same time, gases (nitrogen, oxygen) dissolved in the blood and tissues in large quantities pass into a gaseous state, forming many bubbles, - desaturation occurs . Gas bubbles can cause a gas embolism. The clinical picture of this disease is determined by the localization of gas bubbles. The most common symptoms are: a) pain in the joints, b) itching of the skin, c) impaired vision, d) paralysis, e) loss of consciousness.

To avoid such violations, decompression must be carried out slowly, so that the rate of formation of gases does not exceed the capacity of the lungs to remove them.

• Explosive decompression occurs in cases of a rapid drop in atmospheric pressure pressure from normal to reduced, which happens during high-altitude depressurization aircraft (planes, spaceships). In the development of this syndrome has meaning: a) barotrauma of the lungs, b) of the heart and large vessels as a result of a acute increased intrapulmonary pressure.

Rupture of alveoli and blood vessels contributes to the penetration of gas bubbles into the bloodstream system (gas embolism). In the most extreme cases, instant death occurs as a result of boiling blood and other body fluids, as well as as a result of lightning forms of hypoxia.

• Effect of pathogenic factors on the body during space flight

1) On the dynamic part of the flight (at the launch and landing of the spacecraft) a person is exposed to: a) overloads, b) vibrations, c) noise.

Overload is a force that acts on the body during movement with acceleration.

Depending on the nature of the movement, the following are distinguished: a) rectilinear and b) radial acceleration relative to the longitudinal axis of the body: a) transverse and b) longitudinal overload.

The main mechanism of action of overloads is the displacement of organs and liquid media direction, reverse movement. If the action of overloads coincides with the longitudinal axis body, then a sharp and dangerous redistribution of blood occurs in the brain system blood circulation, which is accompanied by: a) in some cases - overflow of blood vessels brain and hemorrhages, b) in others - brain ischemia.

Therefore, in space flights, the cosmonaut's body is oriented in relation to movement in such a way so that the action of overloads did not coincide with the longitudinal axis of the body, but was directed transversely.

2) In orbital flight, a person is subject to weightlessness and hypokinesia.

The period of adaptation to weightlessness is preceded by a period of acute reaction: a) sensitivity is disturbed (disorientation, illusory sensations, dizziness); b) accuracy, strength and coordination of movements are disturbed; c) appear vegetatively disturbances (nausea, vomiting, salivation, pulse and blood pressure instability).

The cause of these disorders: a Disturbance of the function of the analyzers as a result of a twisted afferentation from receptor zones: a) vestibular apparatus, b) skin, c) organ of vision, d) proprioceptors .

□ Adaptive changes are most pronounced in the circulatory and locomotor systems device.

As a result of the drop in the hydrostatic component of blood pressure there is a redistribution of blood with an increase in the blood filling of the upper vessels half of the body Increased excretion of sodium and water through the kidneys leads to a decrease volume of circulating blood, the load on the heart decreases.

Weightlessness and hypokinesia are the cause of excretion of calcium and phosphorus from the body, as a result of which: a) bone structure changes, b) osteoporosis develops, c) there is a decrease in the mass of skeletal muscles, d) the strength of their contractions decreases, e) signs of atrophy appear.

The effect of electric current is determined by:

1) Physical parameters of electric current: a) alternating or constant current (at a voltage of more than 500 V, direct current is more dangerous), b) frequency alternating current (the most dangerous is the current of the city network - 50 Hz), c) voltage and electric current strength (there is a direct relationship between voltage and strength current, on the one hand, and its damaging effect - on the other).
2) Through the passage of current in the body. The most dangerous is passage of electric current through the heart and brain. In these cases death may occur as a result of cardiac fibrillation or central arrest breath.
3) Physiological state of the body. The following are important: a) the condition of the skin (when moistening or damaging the skin, the degree of electric shock increases); b) general condition (the damaging effect of the current increases with overheating, cooling, blood loss, etc.); c) adaptation to electric current (people who work with current, are less sensitive to its action).
The local effect of electric current is manifested by: a) electrothermal action – burns; b) electrochemical action - electrolysis; c) electromechanical action - breaks tissues, bone fractures.
• The direct cause of death when an electric current affects the body may be:
1) Heart fibrillation. When the current passes through the heart, there are frequent beats asynchronous contractions of individual muscle fibers of the ventricular myocardium, which leads to to asystole and cardiac arrest.
2) Central respiratory arrest. Occurs when current passes through brain structures that regulate external breathing. Death comes in as a result of paralysis of the respiratory center.
3) Peripheral respiratory arrest. Occurs as a result of: a) respiratory spasm muscles, b) spasm of the vocal cords.
Effect of infrared and ultraviolet radiation on the body.
• Infrared radiation has a thermal effect, so with intense affecting tissues, it can cause thermal burns.
• Ultraviolet radiation has: a) thermal, b) photochemical and c) weak ionizing effect.
□ Erythema (redness) may develop with local exposure.
At first, it is short-lived, appears after a few minutes and passes quickly. It occurs reflexively and is associated with the thermal effect of ultraviolet radiation (primary erythema). b) After a few hours, a steady state appears redness with swelling, pain, general manifestations (weakness, headache pain, intoxication). This is secondary erythema, which is caused by the formation of and release of biologically active substances into the tissue (histamine , serotonin, kinins , prostaglandins), as well as the formation of toxic products that are formed during breakdown of tissue proteins caused by ultraviolet radiation.
In addition, prolonged exposure to ultraviolet radiation can have genetic consequences: a) mutations, b) development of malignant skin and superficial tumors fabrics
• Increased sensitivity of the body to the action of ultraviolet radiation is called photosensitization .

Substances that cause the effect of photosensitization have received a name photosensitizers.

There are: a) exogenous photosensitizers: eosin, rivanol , acridine, quinine, sulfonamides) and b) endogenous photosensitizers: bile acids, hematoporphyrins , cholesterol, bilirubin.

It is believed that ultraviolet rays are under the influence of photosensitizers begin to interact with the molecules of those tissues with which in the absence photosensitizers do not interact.

In addition, there are reasons to think that under such conditions the passage is accelerated energy of ultraviolet rays on the carbon skeletons of biological molecules.

Topic 2. Disturbance of the structure, function and metabolism of body cells during oxygen starvation. Pathological changes in muscle cells (skeletal and smooth muscles of organs and systems).

Hypoxia is a typical pathological process that develops as a result of insufficient supply of tissues with oxygen or a Disturbance of its use by tissues in redox processes.

Classification of the main types of hypoxia by pathogenesis:

1. Hypoxic ;
2. Respiratory;
3. Circulatory ;
4. Chemical ;
5. Fabric;
6. Mixed - a combination of different types of hypoxia.

Classification according to the rate of development and duration of the course:

a) lightning-fast - within several tens of seconds,

b) acute - several minutes or tens of minutes (acute heart failure),

c) subacute - several hours,

d) chronic - weeks, months, years.

Hypoxic hypoxia is an exogenous type of hypoxia that develops when the barometric pressure of O_2 decreases (altitude and mountain sickness) or when the partial pressure of O_2 in inhaled air decreases. At the same time, *hypoxemia* develops (pO_2 in arterial blood decreases , saturation of hemoglobin (Hb) with oxygen (O_2) and its total content in the blood. *Hypocapnia* (pCO_2 in arterial blood decreases), which develops in connection with compensatory hyperventilation of the lungs, also has a negative effect. Hypocapnia leads to deterioration of blood supply to the brain and heart, *gas alkalosis* , electrolyte imbalance in the body's internal environment, and increased consumption of O_2 by tissues .

The respiratory (respiratory) **type of hypoxia** occurs as a result of insufficient gas exchange in the lungs due to alveolar hypoventilation , violations of ventilation-perfusion relations, or due to difficulty in diffusion of O_2, Disturbance of the patency of the respiratory tract, or disorders of the central regulation of breathing.

The minute volume of ventilation decreases, the partial pressure of O_2 in the alveolar air and the tension of O_2 in the blood decrease (*hypoxemia*), and hypoxia is joined by *hypercapnia (*pCO_2 in the arterial blood increases), *gas acidosis develops* .

Circulatory hypoxia occurs when blood circulation is impaired, which leads to insufficient blood supply to organs and tissues with massive blood loss, dehydration of the body, and impaired cardiovascular activity. Circulatory hypoxia of vascular origin develops with an excessive increase in the capacity of the vascular bed as a result of reflex and centrogenic disturbances of vasomotor regulation, insufficient glucocorticoids , with an increase in blood viscosity and the presence of other factors that prevent the normal movement of blood through the capillary network. The gas composition of blood is characterized by a normal voltage and content of O_2 in arterial blood, a decrease in venous blood and *a high arterio -venous difference in* O_2.

Hemic hypoxia occurs as a result of a decrease in *the oxygen capacity of the blood* in anemia, hydremia and impaired ability of Hb to bind, transport and give O_2 to tissues, in CO poisoning, in the formation of methemoglobin (MetHb) and in some Hb abnormalities. Hemic hypoxia is characterized by a combination of normal O_2 tension in the arterial blood with its reduced content in severe cases up to 4-5% .

Tissue hypoxia occurs as a result of a Disturbance of the ability of tissues to absorb O_2 from the blood or in connection with a decrease in the efficiency of biological oxidation due to a sharp decrease in the combination of oxidation and phosphorylation, due to inhibition of biological oxidation by various inhibitors, a Disturbance of enzyme synthesis or damage to cell membrane structures, for example, cyanide poisoning , heavy metals, barbiturates. At the same time, tension, saturation, and O_2 content in arterial blood can be normal up to a certain point, and in venous blood significantly exceed normal values. *A decrease in the arterio - venous difference in* O_2 is characteristic of a disturbance of tissue respiration.

Changes in metabolism occur first of all from the side of carbohydrate and energy metabolism. In all cases of hypoxia, the primary shift is a deficiency of macroergs. Glycolysis increases, this leads to a drop in glycogen content, an increase in pyruvate and lactate. An excess of lactic, pyruvic and other organic acids contributes to the development of metabolic acidosis. A negative nitrogen balance occurs. Hyperketonemia develops as a result of disorders of lipid metabolism.

The exchange of electrolytes and, first of all, the processes of active movement and distribution of ions on biological membranes are disrupted, the amount of extracellular potassium increases.

The sequence of changes in the cell: increased permeability of the cell membrane → disruption of the ion balance → swelling of mitochondria → stimulation of glycolysis → reduction of glycogen → inhibition of synthesis and increased breakdown of proteins → destruction of mitochondria → fat decomposition of the cell → destruction of lysosomal membranes → release of hydrolytic enzymes - autolysis and complete cell breakdown .

Adaptive and compensatory reactions:

Reactions of the respiratory system to hypoxia are an increase in alveolar ventilation due to deepening and increasing the frequency of respiratory excursions and mobilization of reserve alveoli. Increased ventilation is accompanied by increased pulmonary blood flow. Compensatory hyperventilation can cause hypocapnia , which in turn is compensated by ion exchange between plasma and erythrocytes, increased excretion of bicarbonates and basic phosphates with urine.

The reactions of the circulatory system are expressed by increased heart rate, an increase in the mass of circulating blood due to the emptying of blood depots, an increase in venous inflow, stroke and minute OS, blood flow rate and redistribution of blood in favor of the brain and heart. When adapting to long-term hypoxia, the formation of new capillaries can occur. In connection with hyperfunction of the heart and changes in neuro -endocrine regulation, hypertrophy of the myocardium can occur, which has a compensatory -adaptive nature.

Reactions of the blood system are manifested by an increase in the oxygen capacity of the blood due to increased washing of erythrocytes from the bone marrow and activation of erythropoiesis due to increased formation of erythropoietins . Acidosis promotes HbO_2 dissociation .

Tissue adaptive mechanisms - limiting the functional activity of organs and tissues that are not directly involved in ensuring O_2 transport, increasing the conjugation of oxidation and phosphorylation, increasing the anaerobic synthesis of ATP due to the activation of glycolysis. The synthesis of glucocorticoids , which stabilize lysosome membranes , activate enzyme systems of the respiratory chain, increases . The number of mitochondria per unit of cell mass increases.

Therapy and prevention. In all cases of hypoxia - respiratory, hemic , circulatory , *hyperbaric oxygenation is a universal technique* . It is necessary to break the vicious circle in case of ischemia, heart failure. Thus, at a pressure of 3 atmospheres, a sufficient amount of O_2 (6% by volume) dissolves in the plasma even without the participation of erythrocytes, in some cases it is necessary to add 3-7% CO_2 to stimulate the respiratory center, expand the blood vessels of the brain and heart, and prevent hypocapnia.

Cardiac and hypertensive drugs, blood transfusions are prescribed for circulatory hypoxia. With chemical type:

- transfusion of blood or erythrocyte mass, stimulation of hematopoiesis , use of artificial O_2 carriers - substrates of perfocarbohydrates (perftoran – “blue blood”)
- removal of metabolic products - hemosorption, plasmaphoresis ,
- fight against osmotic edema - solutions with osmotic substances,
- with ischemia - antioxidants, membrane stabilizers, steroid hormones,
- increasing the energy supply of tissues - glucose.

Topic 3. Causes and pathogenesis of stress. General adaptive syndrome. Pathogenic effect on organs and systems.

Stress. One of the achievements of modern medicine is the discovery of the important role of endocrine glands, in particular the hypothalamus-pituitary-adrenal

system, in the body's adaptation to the action of pathogenic factors. Canadian scientist Hans Selye 's theory about stress is widely known.

The term "stress" (from the English stress — tension) denotes a non-specific reaction of the body, which occurs under the influence of any strong irritants (stressors) and is accompanied by a restructuring of the body's protective forces. Selye drew attention to the fact that, despite the variety of stressors (trauma, infection, hypothermia, intoxication, anesthesia, muscle strain, strong emotions, etc.), they all cause the same type of changes in the thymus, adrenal glands, lymph nodes, blood composition and metabolism . In experiments on rats, he observed a typical triad: hypertrophy of the cortical substance of the adrenal glands,

involution of the thymus -lymphatic apparatus and hemorrhagic ulcers on the mucous membrane of the stomach and duodenum.

Stress manifests itself in the form of a general adaptation syndrome, which goes through three successive stages: the anxiety reaction, the resistance stage, and the exhaustion stage. The reaction of anxiety means the immediate mobilization of the body's defense forces. It consists of a shock and anti-shock phase. In the phase of shock, muscle hypotonia and arterial hypotension , hypothermia, hypoglycemia, and blood clotting are observed. Changes in the body indicate the predominance of catabolism processes. The anti-shock phase is characterized by changes in the opposite direction (increased blood pressure, muscle tone, blood glucose), which lead to the development of the next stage — resistance. The main pathogenetic link of the anti-shock phase is a persistent increase in the secretion of corticotropin and corticosteroids.

In the stage of resistance, the cortical substance of the adrenal glands hypertrophies and secretes a significant amount of hormones, anabolic processes are activated.

In the case of prolonged action of the harmful the agent's adaptation is disrupted. A sharp decrease in functional reserves and atrophy of the cortical substance of the adrenal glands, a decrease in blood pressure, and the breakdown of protein substances characterize the transition from the stage of resistance to the stage of exhaustion.

The consequences of stress depend on the strength and duration of the stressor and the potential capabilities of the body's defenses.

The biological significance of the adaptation syndrome lies not only in the fact that in the second, longest stage, the body's resistance to the factor that caused the state of stress increases, but also in the fact that not very strong and prolonged stress is able to create or increase non-specific resistance of the body to other unfavorable factors.

Corticotropin and corticosteroids play a leading role in the formation of non-specific resistance, therefore they are called adaptive hormones. Systematic impact on the body of weak and moderate stimuli (for example, a cold shower, physical exercises) supports the readiness of the endocrine system for adaptive reactions.

Loss of consciousness (LS) is a state when a person lacks any reactions, is motionless, does not answer questions.

The reasons may be different, but all of them are related to damage to the center of consciousness - the brain (in case of injuries, lack of oxygen, freezing, etc.). Signs of VS are manifested in a wide range of symptoms, starting from shock, unconsciousness and ending with a state of clinical death. In case of acute respiratory distress syndrome, the greatest danger to the life of the victim is the sinking of the tongue and the entry of vomitus into the respiratory tract, which leads to their blockage and asphyxiation.

Shock (from the English shock — shock) is a typical pathological process that has a phase course and arises as a result of a disorder of neurohumoral regulation. Its development is due to the influence of extreme factors and a sharp decrease in blood supply to tissues. General signs of shock are acute progressive insufficiency of microcirculation, which leads to the development of hypoxia, metabolic disorders and, finally, to morphological changes. A critical decrease in capillary blood flow in the affected organs is life-threatening.

In the Middle Ages, the term "shock" was used to describe the state of stupor into which knights fell when they clashed in tournaments. As a medical name for a pathological process and a diagnosis, this term was introduced by the French military surgeon Landran , who published a treatise on this topic in Amsterdam in 1741.

The reasons are severe pain, blood loss, the formation of harmful products in damaged tissues, which lead to the cessation of the body's protective capabilities, as a result of which there are disorders of blood circulation, breathing, and metabolism.

According to modern ideas about the main etiological factors and mechanisms of development, hypovolemic , hemorrhagic, traumatic (and, in particular, burn), dehydration , anaphylactic and other (more than 100 names in total) types of shock are distinguished.

Primary hypovolemic shock . The occurrence of hypovolemic shock is associated with external or internal blood loss (trauma, including postoperative, damage to blood vessels in pathologically changed organs, Disturbance of the blood coagulation process); loss of plasma (burn, fragmentation of tissues); loss of fluid and electrolytes (intestinal obstruction, pancreatitis, peritonitis, enterocolitis, overheating); redistribution of blood (thrombosis and embolism of main veins).

At the same time, the deficit of blood volume causes a decrease in the amount of venous return to the heart and the volume of cardiac output, a decrease in blood pressure. Due to the stimulation of adrenergic receptors of peripheral vessels, the frequency of heart contractions increases, the peripheral resistance of vessels increases, which contributes to the normalization of blood pressure and blood supply, primarily to the heart and brain.

The insufficiency of these mechanisms, as well as the negative consequences of vasoconstriction, is accompanied by a sharp decrease in blood supply to organs and tissues and characteristic signs of shock.

Traumatic shock occurs with significant injuries to bones, muscles, and internal organs, and is always accompanied by damage to nerve endings, trunks, and plexuses. Traumatic shock is complicated bleeding and wound infection. In its

course, two stages are observed: excitement (or erectile stage) and inhibition (or torpid stage).

The first stage is short-lived , it is characterized by a state of excitation of the central nervous system, the result of which is an increase in the function of the circulatory system, respiration, and some endocrine glands with the release of an excess amount of so-called stress hormones into the blood, which increase metabolism and stimulate the activity of organs.

The second stage is longer (from several hours to a day). It is characterized by the development of inhibition processes in the central nervous system and a decrease in the functions of vital organs and systems.

The clinical picture of traumatic shock is as follows: at first, the patient is pale, shouts, gesticulates, fusses, pupils are dilated, heart rate is increased, blood pressure is elevated. Reactions to any influences increase as a result of increased excitability of the central nervous system. In the future, suppression of speech and motor activity increases, an indifferent attitude to reality arises. The patient does not react to any stimuli, including pain. The activity of the cardiovascular system weakens — cardiac output decreases, blood pressure falls. Microcirculation is disrupted and blood properties change, blood clots may appear in blood vessels and intravascular coagulation may develop. When shock phenomena increase, "shock" parenchymatous organs appear. So, the severity of the shock is indicated by the amount of urine excreted: if in 1 hour secreted less than 30 ml. urine, kidney function incompatible with life ("shock kidney").

In the mechanism of occurrence and development of traumatic shock, a certain role is played by toxemia caused by the entry into the blood of products of decay of non-viable tissues. The value of this factor was proved by V. Cannon using the example of "tourniquet" shock, which occurs 4 hours or later after the removal of the tourniquet or after the cessation of long-term compression of body parts during collapses of mines, mines , as a result of earthquakes, bombings, etc.

Cardiogenic shock is observed in the event of a decrease in the pumping function of the heart (myocardial infarction, myocarditis), severe heart rhythm disturbances, cardiac tamponade (effusion or bleeding into the pericardial cavity).

The leading mechanism of the development of cardiogenic shock is a decrease in the performance of the heart due to a Disturbance of the pumping function or the presence of obstacles to filling the ventricles. As in the case of hypovolemic shock, as a result of stimulation of adrenergic receptors, tachycardia and an increase in peripheral vascular resistance are observed, which only complicate hemodynamic disturbances.

Vascular forms of shock. These include septic and anaphylactic shock. Common in the development of vascular forms of shock is a primary Disturbance of vascular tone, which leads to microcirculation disorders.

Anaphylactic shock develops as a result of increased sensitivity of the body to substances of an antigenic nature. Antigens can most often be various protein preparations, serums and vaccines containing proteins, toxoid, extracts from organs, insect and animal poisons. Of the drugs, anaphylactic shock is mainly caused by

penicillin, streptomycin, sulfonamides, iodine preparations, local anesthetics (novocaine) and aspirin. The pathogenesis of shock is due to the movement of huge masses of blood to the periphery as a result of a decrease in vascular tone; loss of part of the plasma due to increased permeability of the vascular wall; breathing disorder due to spasm of bronchioles. The clinical picture has an acute, sometimes lightning-fast course: the shock can last literally minutes and end in death.

Therefore, the term "shock" is a collective concept that unites extreme states of the body's vital functions that occur as a result of an impact or disorder that is extraordinary in strength or duration, external or internal, and is manifested by a whole complex of disorders of physiological systems, mainly blood circulation , metabolism and the central nervous system. Sometimes it combines states that are far apart in terms of etiological, pathogenetic and clinical signs. However, this term

correctly orients us to the emergency and danger of the situation in which the patient is. Previous illnesses (radiation sickness, anemia, starvation, etc.) reduce the body's resistance to shock. Especially sensitive to

the child's body is susceptible to shock due to its physiological features, namely: a high level of fluid exchange, a high frequency of heart contractions, a small stroke volume of the heart, insufficient regulation of vascular tone and thermoregulation.

Help. The prevention of the development of shock is timely and effective help, which is provided in case of any injury. In case of traumatic shock, it is necessary to act in the following sequence:

- stop bleeding by applying a tourniquet, tight bandage, tamponade, compression of blood vessels;
- if possible, provide analgesia in the form of local anesthesia (inject 0.25-0.5% novocaine solution in places close to the fractures, 150...200 ml each);
- ensure free breathing of the victim, if necessary perform artificial respiration;
- perform indirect heart massage in case of cardiac arrest;
- immobilize fractures by applying transport tires or tires made of improvised materials.

Then the victim should be wrapped in a blanket, put in a horizontal position. Measures that prevent the occurrence and development of shock are silence, warmth (but not overheating), reduction of pain, drinking fluids.

Topic 4. Electrocardiography. Its basics, graphic display. Methods of diagnosing disorders of automaticity and conduction of the heart.

Acquire knowledge of a normal ECG, master the skills of recording an ECG in the generally accepted 12 leads , its analysis, writing an ECG protocol, conclusion and clinical interpretation of the obtained data. To master the knowledge of ECG changes in disorders of automatism, excitability and conduction. Study ECG signs hypertrophy of the heart, ECG changes in various clinical forms of coronary artery disease, electrolyte and metabolic changes and ventricular preexcitation syndromes.

- I): with visual aids, including with tables and set
ECG on the topic of the lesson, with the organization of the work of the office of functional diagnostics
of the department and the ECG office of the basic medical institution.
Know (□-II):
- the principle of the ECG method and its anatomical and physiological basis (basic concepts of
electrophysiology of the heart, structure of the conducting system of the heart);
- basics of ECG analysis (estimation of voltage, sources of ventricular rhythm, definition
electrical axis of the heart, measurement and assessment of waves and intervals P, PQ, QRS, QT,
RRaverage , with heart rate determination);
- mechanisms of occurrence of arrhythmias, ECG - signs of violations of automatism functions and
excitability, principles of emergency care for paroxysms tachyarrhythmia ;
- ECG signs of blockages;
- ECG signs of ventricular preexcitation syndromes ;
- classification of heart rhythm and conduction disorders;
- ECG changes in hypertrophies of the heart.
- ECG changes in various clinical forms of coronary artery disease;
- ECG for metabolic and electrolyte disorders.
Be able to (□-III):
- take an ECG in the generally accepted 12 leads ;
- conduct an ECG analysis (estimate voltage, source of ventricular rhythm, determine
the electrical axis of the heart, measure and evaluate ECG waves and intervals);
- to diagnose rhythm and conduction disorders of the patient based on clinical manifestations and
electrocardiogram, provide emergency assistance for rhythm disturbances;
- to identify ECG signs of hypertrophy of the heart and evaluate primary disorders repolarization processes ("systolic overload") in left hypertrophy
ventricle;
- identify signs of focal (post-infarction cardiosclerosis) and diffuse changes
of the myocardium (violations of repolarization processes associated with ischemia, metabolic and electrolyte changes) and give them an assessment;
- write a report of the ECG conclusion.

What is electrocardiography

Electrocardiography is used for instrumental research of heart muscle activity. The study can be carried out at rest, during physical exertion and when using some special drugs - during the ECG, the condition of the heart muscle, heart rhythm, and blood flow in the myocardium are determined.

Electrocardiography is a method of graphic registration of electrical phenomena that occur in the heart muscle during its activity, from the surface of the body. A curve that reflects the electrical activity of the heart is called an electrocardiogram (ECG). Thus, the ECG is a recording of the potential difference oscillations that occur in the heart during its excitation.

Electrocardiography is one of the main methods of examining the heart and diagnosing diseases of the cardiovascular system. ECG is indispensable in the diagnosis of rhythm and conduction disorders, hypertrophy , coronary heart disease. This method makes it possible to speak with great accuracy about the localization of focal changes in the myocardium, their distribution, depth and time of appearance. ECG allows to detect dystrophic and sclerotic processes in the myocardium, disturbances of electrolyte metabolism that occur under the influence of various toxic substances. ECG is widely used for functional research of the cardiovascular system. The combination of an electrocardiographic study with functional tests helps to detect hidden coronary insufficiency, transient rhythm disturbances, to carry out a differential diagnosis between functional and organic disorders of the heart.

Electrocardiography is absolutely safe, which allows you to conduct research repeatedly and evaluate the dynamics of changes, as well as during patient treatment.

Preparation for electrocardiography

Special preparation for electrocardiography is not required. The procedure is performed on the day of the patient's visit to the medical institution. The study is conducted after 10-15 minutes of rest no earlier than 2 hours after eating.

In the diagnostic office, electrodes equipped with suction cups are fixed on the chest area at special points to ensure close contact with the skin, which will eliminate the possibility of interference. During the recording of the electrocardiogram, you should relax as much as possible and not make any movements. You may need to hold your breath. The duration of the procedure is several minutes.

Indications for electrocardiography:

- preventive examinations as a screening technique for detecting pathology of the cardiovascular system;
- pains in the area of the heart;
- dyspnea;
- arrhythmia;
- deterioration of the condition of patients with cardiac pathology;
- before any surgical interventions and complex medical procedures;
- in diseases of internal organs, glands of internal secretion (endocrine);
- in the diagnosis of tumor processes (both benign and malignant);
- diseases of various organs, which can potentially be complicated by pathology of the heart and blood vessels;
- expert assessment of persons employed in high-risk jobs.

People with heart problems often have such a situation: they have complaints, but they arise when it is not possible to see a doctor immediately - in the evening, at night, during some events. The person goes to the clinic the next day, they take an ECG, but no abnormalities are found. Why can this happen? Because a standard electrocardiogram is a record similar to a “snapshot” that captures heart activity over a short period of time. A normal ECG records only a few contractions of the myocardium: from three to twenty - depending on the cardiograph used for this purpose. Whereas the heart contracts about 100,000 times a day. Then Holterivsky's method comes to the rescue ECG monitoring .

Holterovsky's method ECG monitoring

Holter is a portable medical recorder used to record electrocardiogram and blood pressure during the day in order to collect, analyze the received information and detect possible violations. A halter is put on the patient - it is attached to the body on belts and electrodes are installed in specified places. Doctors often use the phrase "hang a halter " or "make a halter ", meaning that the device will be used for 24 hours, on an outpatient basis, in the patient's usual life conditions - at home, at work, during night sleep. This process is called Holter monitoring , named after research scientist Norman J. Holter , who first used this technique in 1961.

The Holter registers the ECG throughout the day, and no heart contraction during this period will remain unnoticed by the device, because the electrocardiogram is recorded continuously. Holter makes available for analysis that information about the state of the patient's heart, which cannot be obtained during a short visit to the doctor, and this is the most important diagnostic value of this device.

Then the holter is removed from the patient, and with the help of a special computer program that processes the received data, all heart rhythm disorders, painless and painful attacks of myocardial ischemia, etc. are detected and analyzed . The 24-hour monitoring proposed by Holter is a procedure that detects almost all possible disturbances of cardiac activity that occur during the day, which cannot be done with the help of other diagnostic methods used today.

Holter monitoring is a simple and safe procedure. In order to conduct it, two visits to the doctor will be required. During the first visit, the holter is programmed and installed on the patient, this procedure takes no more than a quarter of an hour. After a day, the holter will need to be removed and its records analyzed.

An electrocardiogram is a graphic recording of heart potentials from the surface of the body. Graphical recording of biocurrents of the heart was first carried out in 1913 by Einthoven . The connection of two points of the body that have different potentials is called a lead.

The first standard lead (И) registers the difference in the potentials of the right and left hands. The second (II) is of the right hand and left leg, and the third (III) is of the left hand and left leg.

Characteristics of the waves and intervals of the electrocardiogram:

The P wave occurs as a result of excitation (depolarization) of the right and left atria. Its duration is 0.06 - 0.10 seconds , height - 0.5 - 2.5 mm. The tooth in leads I and II is always positive.

The PQ interval reflects atrioventricular (atrioventricular) conduction. The duration of the PQ interval is 0.12 - 0.2 seconds , with a heart rate of 90 - 60 per minute.

Q wave - reflects the excitation of the interventricular membrane and the inner surface of the ventricles. The Q wave is the least constant wave, only 3% of patients have it in all leads . The most important thing to remember is that in a healthy person, the depth of the Q wave should not exceed ¼ of the R wave in the same lead, which is approximately 3 mm. The width of the wave Q is 0 – 0.03 sec .

The ventricular QT complex consists of the initial part of the QRS, the intermediate ST segment and the final T wave.

The highest wave of the ventricular complex R has a height of 6–16 mm. If the voltage R in one of the standard leads is within 10 mm, and the sum of the 3 leads is 15-25 mm, the voltage is considered normal.

The width of the QRS interval is 0.06-0.1 sec . If the width of the QRS is different in the standard leads , the conclusion is made according to the largest one.

The S wave reproduces the termination of excitation of both ventricles. The width of the S wave is 0.06 seconds , the depth is 2.5 – 6.0 mm.

The ST segment is the intermediate part of the ventricular complex, which corresponds to the period of full excitation of both ventricles. ST displacement below or above the isoline should not exceed 1 mm. A sharp displacement of the dome-shaped ST upward is observed in the first days of a large-focal heart attack and in Prinzmetal's angina , and its downward displacement is observed in chronic coronary insufficiency.

The T wave reflects the process of the final repolarization of the ventricles. The width of the T wave varies from 0.16 to 0.24 sec , the height in leads I, II, III is 2-6 mm. In lead III, the T wave can be lowered, biphasic or negative in the norm.

The U wave follows after 0.04 seconds , it is unstable, its width is 0.16-0.25 seconds , its height is up to 2 mm, it becomes high during hypokalemia .

The QT interval (QRS-T) is the electrical systole of the ventricles, the duration of which in healthy people is 0.34 - 0.44 seconds , measured from the beginning of Q to the end of the T wave.

The T-P interval characterizes the electrical diastole of the heart, there are no potentials, and a zero isoelectric line is registered.

You should pay attention to the reinforced unipolar leads from the extremities: the graph of the waves of aVL is the same as in the I standard lead, and aVF is the same as in the III standard lead. At the same time, lead aVL registers the potentials of the anterior -lateral wall of the myocardium, and lead aVF registers the potentials of the posterior -lower wall of the myocardium.

In addition, reinforced unipolar chest leads are recorded and marked as V1, V2.... Leads V1 and V2 reflect the potentials of the front wall, V3 - the membranes, V4 - the apex, V5 and V6 - the side wall of the left ventricle. In lead V3, the height of the R wave = the depth of the S wave (1/1), which corresponds to the transition zone.

Topic 5. Clinical pathophysiology and ECG diagnosis of heart failure arrhythmias.

ARRHYTHMIAS

The concept of **arrhythmias** includes heart rhythm disorders, which are based on heart function disorders: automaticity, excitability, conduction, and contractility, which are manifested by a change in the pacemaker, frequency, rhythmicity, speed, and direction of conduction of excitation pulses (biopotentials), coherence, strength of individual contraction of the heart muscle.

Etiological factors of arrhythmias:

1). Diseases of the cardiovascular system acquired and congenital (IHD, GC, myocarditis , heart defects and others).

2). Dysregulation of cardiac activity in non-cardiac pathology: (nerve- volo-reflex influence, humoral and endocrine disorders, electrolyte shifts, etc.)

- · with pathology of the gastrointestinal tract;
- · with pathology of the central nervous system;
- · with endocrine diseases.

3). Physical and chemical impact:

- · hyper and hyperthermia;
- chest injuries;
- · vibrations;
- · medicines;
- · alcohol;
- · nicotine;
- · ionizing radiation.

4). Idiopathic arrhythmias (the cause is unknown).

Pathogenesis of arrhythmias:

1). Mechanisms of impulse formation are disturbed, in particular:

and). the automatism of the sinus node is disturbed;
b). pathological automatism is formed.
2). Conduction of impulses is disturbed:
and). the refractoriness of extinction and conduction of pulses is prolonged ;
b). anatomical damage occurs in the conduction system;
in). emergence of the phenomenon of re-entry of excitation (re-entry).
3). Combined mechanisms of disturbance of formation and conduction of impulses.

Disturbance of the function of automatism.

The sinus node may remain the driver of the rhythm, but the number and sequence of the occurrence of impulses changes. These rhythms are called sinus or nomotopic . Nomotopic include : sinus tachycardia, sinus bradycardia, sinus arrhythmia.
In cases where the starting point of the pulses are other automatic centers, the rhythms are called heterotropic . Heterotropic rhythms include :
and). atrioventricular (nodal) rhythm;
b). idioventricular (intraventricular) rhythm;
in). migration of the rhythm driver;
d). pop-up contractions.
Disturbance of sinus node automatism - nomotopic sinus rhythm .

Heterotropic or ectopic heart automatism disorders:

Nodal-AV rhythm - occurs when the automatism of the sinus node is temporarily or permanently turned off.
Impulses in the atria propagate retrogradely , and go to the ventricles in the usual way. The lower the pacemaker, the slower the pace.

ECG signs:

heart rate 40-55 per 1 min.;
the QRS complex is not changed;
the placement of P before or after the QRS freezes from the place of origin of the impulse, it is negative in leads II, III and aVF .

Clinically : the patient sometimes complains of a feeling of pulsation in the neck vessels.
Idioventricular rhythm is actually a ventricular rhythm most often observed in transverse blockade.
Patients indicate a sharp but increased heartbeat, headache, dizziness, shortness of breath during stress, even emotional ones. During auscultation, against the background of weakened heart sounds, Strazhesko's gun tone is heard - a consequence of the coincidence of contractions of the atria and ventricles.

ECG signs :

a) bradycardia 30-40 in 1 minute;

b) the shape of the ventricular complex is deformed, the QRS complex is lengthened to 0.12-0.16;
c) P waves are negative, often superimposed on the T wave.

Disturbance of excitability function.

Violations of the excitability function include:
· extrasystolic arrhythmia;
· paroxysmal tachycardia;
· parasystole .

Extrasystole – this is a premature out-of-order excitation and contraction of the heart that occurs as a result of pathological heterotopic impulses that occur in the atria, AV node , and ventricles in the diastolic phase.

Classification extrasystole :

1. **According to etiology** :
1.1. functional ;
1.2. organic _
2. **By localization hearth excitement** :
2.1. sinus ;
2.2. atrial ;
2.3. atrioventricular (upper -, middle - lower nodal);
2.4. ventricular (left and right ventricles).
3. **By frequency occurrence** :
3.1. single ;
3.2. frequent _
4. **Compensatory _ pause** :
4.1. with a full compensatory pause;
4.2. with a very large compensatory pause;
4.3. incomplete compensatory pause;
4.4. without a compensatory pause.
5. **Depending from quantity sources excitement** :
5.1. monotopic ;
5.2. polytopic _
6. **Alorrhythmia :**
6.1. bigeminy ;
6.2. trigeminy ;
6.3. quadrigeminy .

With extrasystoles occurs compensatory pause, except sinus extrasystoles .
A compensatory pause is this distance from extrasystoles to the following normal P-QRST complex. **Incomplete compensatory pause** is characteristic of for atrial and AV extrasystole - it's time for now ectopic pulse will reach sinus knot and discharge _ his _ It is insignificant longer than usual RR interval . With ventricular extra systole compensatory pause very long , even doubled of the RR interval ,

because nights are coming after extrasystoles refractory to the next after sinus pulse extrasystoles .

Clinically extrasystole appear complaints about work interruptions _ heart , less often for dizziness with group ones extrasystoles . Irregular pulse (p.irregularis), deficient pulse (p. deficiens) , increased 1st tone, during emergency extrasystolic abbreviations hearts _

Paroxysmal tachycardia is **_ sudden onset and sudden the end of the attack is abrupt tachycardia with heart rate abbreviations from 150 to 220-250 in 1 minute while maintaining the correct regular rhythm.**
When there are five or more on the ECG extrasystole is a paroxysm of tachycardia . Attack may begin suddenly even at night sudden palpitation , compression in the chest , dizziness . Objectively : expressed pallor , decline systolic blood pressure, strengthening of the first tone at the apex . When the attack is prolonged , there are signs cardiac deficiencies .

Flashing arrhythmia - or shimmering arrhythmia - disturbance everyone functions heart with frequent disordered excitation and contraction atrial muscle fibers - 350-700 in 1 minute .
Causes of flashing arrhythmias :
· Atherosclerotic cardiosclerosis ≈ 45%;
· Mitral stenosis ≈ 45%;
· Myocarditis and cardiomyopathy ;
Heart attack myocardium ;
· Thyrotoxicosis ≈ 5%.
Forms shimmering heart rate arrhythmias :
Tachysystolic ; _
· Bradysystolic ;
· Nohmosystolic .
By form shimmering arrhythmia share also on :
· Permanent ;
· Constant with paroxysms, accelerations of the rhythm of the ventricles ;
· Paroxysmal or periodic _
Clinic of paroxysmal flickering arrhythmias .
Complaints:
· Heart palpitations ;
Shortness of breath ;
Pain _ in the chest on the left ;
· Weakness ;
· Dizziness , or loss consciousness _
Objectively : the pulse is full arrhythmia ; uneven filling and tension ; lack of pulse; decrease systolic pressure _
ECG signs blink atrium :

· Absence wave P;
· Availability atrial Fleming's f waves (300-700 in 1 minute). Better expressed in V_1 and V_2 leads _
· Irregularity QRS complexes , or not the same RR distances ;
· Electric alternation QRS complexes , or different in height R in one lead .

Violation functions conductivity - BLOCKADES.

Blockade is **_ slowdown conductivity some kind parts leading systems heart with conduction in the myocardium , or complete interruption carrying out excitement _**

Classification of blockades by location occurrence :

1. sinoatrial (sinoauricular) – CA blockade;
2. intraatrial ;
3. atrioventricular ;
4. intraventricular .

Reasons for blockades:

1. Inflammation ;
2. Sclerotic processes ;
3. Dystrophic processes ;
4. Intoxication ;
5. Heart attack myocardium ;
6. Violation electrolyte composition of blood

Topic 6. Analysis of the ECG recording in hypertrophy of different parts of the heart, myocardial infarction

PATHOLOGY ACCORDING TO THE DATA OF ECG diagnostics

Hypertrophy of the heart is a complex adaptive reaction of the myocardium to an increased load on it, characterized by an increase in the mass of the heart muscle. With normal heart sizes, the weight of the heart in women is 250 g , in men - 330 g , with hypertrophy of the left and right parts, it reaches a weight of 800-1000 g.

Hypertrophy of the left atrium is a characteristic P - **" mitralae "** wave - split, double-humped, lasts more than 0.1 sec . widened , enlarged, > 2.5 mm in leads I and II.

Hypertrophy of the right atrium is most often observed in chronic pulmonary embolism . ECG signs: high P waves in leads II and III and aVF > 2.5 mm, lasting no more than 0.1 sec ., it seems to be acute and is called "P - pulmonale ".

With hypertrophy of the left ventricle, we observe a leftogram and a shift of the transition zone to the right to V_2 and V_1 when the height of R to S = 1:1.

With hypertrophy of the right ventricle, there is a rightogram , displacement of the transition zone to V_5 and V_6 .

pathological Q waves: any Q waves ≥0.02 s or QS complexes in leads V2 and V3; Q waves ≥0.03 s and ≥1 mm deep or QS complexes in 2 leads from a group of adjacent leads (I, aVL , and possibly V6; V4–V6; II, III, aVF). QS complexes or Q waves can be detected in lead aVR , less often in III and V1, sometimes in V1–V2, which should be interpreted as a variant of the norm. In all other leads, their presence

belongs to pathological symptoms. If new pathological Q waves are detected on a standard ECG, which are not accompanied by subjective symptoms, a silent myocardial infarction is diagnosed. The appearance within 28 days of the first or subsequent myocardial infarction of ST elevation ≥1 mm or pathological Q waves indicates a recurrent infarction, especially if changes on the ECG are combined with anginal pain that lasts for ≥20 minutes. Causes of pathological Q waves and QS complexes:

a) change in the conditions of impulse conduction through working cardiomyocytes — focal necrosis of the myocardium of the left ventricle (myocardial infarction), muffled myocardium, cardiomyopathy (mainly hypertrophic, with subaortic stenosis), premature ventricular excitation syndrome;

b) change in the conditions of impulse conduction through the intraventricular conduction complex — blockade of the left leg (complexes QS in leads V1–V3), blockade of the anterior branch of the left leg (complexes qrS in lead V2);

c) displacement of the heart in the chest - enlargement of the right atrium (complexes qR in V1, V1–V2 or V1–V3), emphysema of the lungs (complexes QS in V1–V3), hypertrophy of the left ventricle (complexes QS in V1–V3).

ST segment: reflects the initial phase of repolarization of the ventricular myocardium, is located mainly on the isoelectric line in standard and thoracic left ventricles leads _ In the right ventricular thoracic leads, there is often an oblique ascending elevation of the ST segment, which smoothly transitions into the ascending knee of the T wave

1) ST elevation (significant elevation measured at point J — elevation in leads V2–V3 ≥1.5 mm in women and ≥2.5 mm in men under 40 years and ≥2 mm in men after 40 years, and in others leads ≥1 mm in men and women);

a) J-point elevation with "trough-like" ST elevation in the thoracic, less often in the thoracic leads and leads from the limbs, in exceptional cases only in the leads from the limbs — the syndrome of early repolarization of the ventricles (variant of normal ECG recording, Fig. 25.1-5). According to the modern definition, this syndrome includes cases with elevation of the J point regardless of the position of the ST segment. It is considered that the elevation of the point J ≥1 mm in the form of inflections (Fig. 25.1-5) or the rounded departure of the final phase of the R wave in 2 from the leads from the extremities II, III and aVF and/or atrial (or extracardiac) V4–V6, especially with a concomitant horizontal location or slanted depression of the ST segment, can be a sign of electrical instability of the myocardium of the ventricles, which in turn can be a harbinger of life-threatening ventricular arrhythmia and sudden cardiac death.

b) elevation of the J point ≥2 mm in V1–V2 (in ≥1 of the indicated leads) with oblique-ascending elevation of the ST segment and a smooth transition to a negative T wave — Brugada syndrome (after excluding other causes);
c) horizontal or convex upward (Pardy wave) with a depression in reciprocals leads — acute transmural ischemia or acute myocardial infarction Persistent elevation of ST segments in leads with pathological Q waves or QS complexes is a manifestation of impaired myocardial contractility in the infarct zone.

d) horizontal, in the vast majority of leads , with discordant depression only in leads aVR and V1 — suspicion of the acute phase of pericarditis (damage due to the inflammatory process in the subepicardial layers of the myocardium). This diagnosis is supported by concomitant depression of the PQ segments.

e) oblique-ascending — hypervagotonia , ventricular depolarization disorders (leg blocks, premature ventricular excitation syndrome , ventricular extrasystoles and rhythms);
ST depression (significant ST depression , measured at point J — depression in the chest leads V 1– V 3 ≥0.5 mm, and in other leads ≥1 mm in men and women);

Topic 7. Electromyography . Pathogenesis and functional diagnosis peripheral nervous systems and neuromuscular violations _

Electromyography

EMG is a diagnostic procedure for assessing the condition of muscles , nerves and nerves cells that _ their control _ The method helps identify the cause of such frequent problems as muscular weakness in the limbs , disorders sensitivities (numbness , etc.) Basic research methods

The study of nerve conduction with the help of stimulation EMG is more often used in the pathology of peripheral nerves (traumatic and compressive neuropathies (tunnel syndromes), polyneuropathies of various genesis), pathology of neuromuscular transmission (myasthenia). When performing this study, peripheral nerves are stimulated with short-term electrical impulses. The technique is non-invasive , as skin stimulating and lead electrodes are used.

Needle EMG is more often used when there is suspicion of motoneuron diseases (spinal amyotrophy , lateral amyotrophic sclerosis, etc.), muscles themselves (myopathy, inflammatory diseases, etc.). At the same time, a thin needle electrode is inserted into the muscle, which registers the activity of the muscle at rest and during contraction.

As a rule, in the diagnosis of the pathology of the neuromuscular system, a complex of electromyography is used methods (electroneuromyography), which complement each other and allow different diseases to be differentiated, to determine the nature of the pathological process and its course.

EMG makes it possible to confirm the clinical diagnosis, monitor the course and treatment of the disease.

Topic 8. Pathogenesis and functional diagnosis of disorders of cerebral circulation: rheoencephalography .

Pathogenesis. There is chronic insufficiency of cerebral circulation
heterogeneous condition , which is reflected in the etiology, clinical,
neuroimaging and morphological features of its individual forms.
in cardiohemodynamics play a significant role in the pathogenesis of PPNKM ,
heart failure. The presence of close ones is proven
of cerebrocardial relationships in the early stages of vascular formation
brain pathologies. With the development of persistent arterial hypertension and
functional cardiac and cerebral disorders of atherosclerosis
transform into coronary heart disease (CHD) and brain.
A significant place in the pathogenesis of PPNKM belongs to dystonic changes in blood vessels
cerebral angioparesis with slowing of blood flow and disturbance

venous outflow that occurs under such conditions. Pathology is also important
main arteries of the head (hemodynamically significant stenosis of internal
carotid and vertebral arteries, as well as vessels of the circle of Willis), which is confirmed by the data of ultrasound dopplerography . Development of clinical symptoms in
in such cases, it occurs against the background of permanent insufficiency of blood supply
brain One of the mechanisms of the initial manifestations of insufficient blood supply
of the brain, there may be an increase in the viscosity of whole blood, rheological disorders
properties of blood and microcirculation . In the development of PPNKM have
the value is also a Disturbance of neuronal metabolism and
functional state of the brain.
Clinical variants of the course of PPNKM:
- preclinical ,
- clinical
- paroxysmal.
Preclinical (asymptomatic) course.
Diagnostic algorithm: unstable blood pressure,
• signs of vegetative-vascular dystonia: (acrocyanosis , acrohyperhidrosis ,
persistent red dermographism, paleness or redness of the skin,
trembling of fingers, eyelids, invigoration of tendon reflexes).
• absence of complaints from patients.
• functional tests reveal signs of insufficient blood supply
brain (changes in bioelectrical activity are detected
of the brain, hypersynchronous and desynchronous types of EEG; on REG
there is an increase or decrease in the tone of the vascular wall,
lability of pulse waves, interhemispheric asymmetries, difficulty
venous outflow).
6
• Sinus arrhythmia and disturbances are common on ECT
depolarization phase , ST segment shift and T wave.
• changes in biochemical parameters: an increase in cholesterol content,
triglycerides , lipoproteins.
Clinical (permanent) variant of the course of PPNKM.
Diagnostic algorithm:
• subjective signs of insufficient blood supply to the brain are revealed.
The most typical complaints of patients:
• headache,
• dizziness,
• noise in the head,
• memory disorders,
• decrease in mental capacity, etc.

Diagnosis algorithm: a combination of two or more of the above
subjective symptoms that are observed for a relatively long time and often
are repeated (at least once a week during the last 3 months).
Complaints are aggravated during mental stress, especially in conditions
hypoxia, fatigue, poor sleep.
After rest, the patient's condition improves or completely normalizes.
Such patients have no focal neurological symptoms
is observed, but may be subcortical
reflexes, asymmetry of abdominals, revitalization of tendons and
periosteal reflexes.
Signs of general vascular disease are also found in patients
(coronary cardiosclerosis , hypertrophy of the left ventricle of the heart, vascular changes
fundus (angiopathy), symptoms of atherosclerotic lesions of others
plots).
The diagnosis is also confirmed by changes in brain biopotentials , rheoencephalographic , biochemical indicators.
Algorithm for diagnosis of paroxysmal course of PPNKM:
• mainly in patients with vegetative-vascular dystonia, arterial
hypertension and much less often - in the case of atherosclerosis of vessels;
• dominant in the clinical picture are vegetative-vascular paroxysms
such as cephalic , vestibular, syncopal , sympathetic-adrenal ,
vagoinsular and mixed.
• paroxysmal disorders in most cases are a manifestation of dysfunction
suprasegmental level of the autonomic nervous system, in particular formations
limbic -reticular complex.
Additional examination data. Psychological research. At
NPNKM against the background of vegetative-vascular dystonia in the vast majority of patients
increased irritability, instability of attention, weakening of memory and
narrowing of the scope of perception, and in some patients - a decrease in the pace
activity Violations of mental activity are less pronounced than in patients
on atherosclerosis.
7
The diagnosis of PPNKM is made on the basis of complaints and clinical picture
diseases, as well as based on data of electrophysiological and
laboratory research methods. Identifying symptoms is important
general vascular disease: atherosclerosis, arterial
hypertension, vegetative-vascular dystonia.
Treatment of patients with PPNKM is of particular importance because
neurological disorders at this stage of the disease are reversible, and therefore timely therapy is at the same time a prevention of acute disorders of cerebral blood circulation.

A system of staged treatment of patients has been developed: polyclinic — inpatient —
the resort is a polyclinic that includes dispensary supervision. At each of the stages modern schemes of examination and treatment of patients are used taking into account
a variant of the clinical course, as well as a general vascular disease.
Algorithm of treatment in the preclinical stage:
• used when signs of vegetative-vascular lability are detected
a set of preventive measures aimed at eliminating factors
risk, stabilization of blood pressure in the presence of arterial
hypertension, compensation of the initial manifestations of atherosclerosis;
• measures aimed at improving the working and living conditions of patients.
• compliance with the diet: food rich in animal fats should be avoided,
cholesterol, salt; it is necessary to consume dairy-vegetable food, fish, boiled food meat;
• recommended sanatorium-resort treatment.
Algorithm of treatment in the stage of clinical manifestations of the disease
outpatient treatment is carried out taking into account vascular pathology, on the background
which PPNKM developed:
- in the case of vegetative dystonia syndrome, sedatives are recommended
(bromine, valerian), intermittent courses of treatment with tranquilizers in children doses, as well as plant-stabilizing drugs;
- in case of arterial hypertension - correction of blood pressure;
- apply various methods of reflexology , electrosleep;
- the treatment of vascular atherosclerosis requires the appointment of lipotropic drugs
means;
- correction of hemorheological changes requires an appointment
antiplatelet agents ;
- correction of neuronal metabolism is carried out by appointment
nootropics ;
- physiotherapeutic measures, sanatorium-resort facilities are also recommended
treatment, improvement of working and living conditions.

Rheoencephalography (REG) of the cerebral vessels is the research method that allows you to determine the tone of the vessels in the area of interest and assess the blood supply. The special device takes into account the difference in electrical conductivity of tissues caused by the oscillations of the vascular wall during heartbeats and the corresponding change in blood flow.
Deciphering the REG of the head often causes difficulties for patients. But this is not necessary, because the decryption of the result and drawing up the conclusion is done by a certified specialist. Questions that can be answered with REG: How fast does the pulse wave travel? What is the intensity of blood flow? How viscous is

blood? Are the vessels elastic? Is the outflow through the veins enough? Etc. Regional vascular reactions are also determined during the research .
Shows:
neurocirculatory dystonia (NCD);
initial manifestations of cerebral blood circulation insufficiency;
dyscirculatory encephalopathy;
consequences of an acute disturbance of cerebral blood flow;
frequent headaches.

Rheovasography (RVG) is a method of diagnosing diseases of peripheral vessels (mainly - extremities). According to the results of this study, it is possible to judge about a Disturbance of blood circulation, which, as a rule, is caused by atherosclerotic or inflammatory lesions of vessels.

Manifestations of diseases of the vessels of the limbs, which indicate a possible Disturbance of blood flow, are pain that increases after physical exertion (walking, monotonous work with hands), periodic local spastic muscle spasms, numbness of the skin with a tingling sensation in it (paresthesia).