LECTURE COMPLEX

Discipline: General pathology

Discipline code: GP 3214

EP name and code: 6B10115 "Medicine"

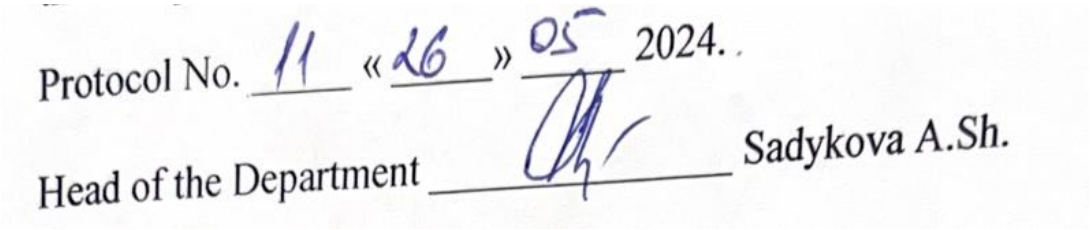
Volume of study hours/credits: 9 0 hours/ 3 credits

Course and semester of study: III year, V semester

Lecture volume: 6 hours

Shymkent, 2024 year

The lecture complex was developed in accordance with the working study program of the discipline (syllabus) 6B10115 "Medicine" and discussed at a department meeting



Lecture No. 1

1. Topic: Subject, tasks and methods of pathological physiology. General nosology. General etiology and pathogenesis.

2. Purpose: - to characterize the purpose, objectives and methods of pathophysiology; define the basic concepts of general nosology.

- explain the role of causes and conditions in the occurrence of diseases; determine cause-and-effect relationships in pathogenesis.

3. Lecture abstracts

Pathological physiology is the main fundamental medical and biological science that studies the general features of the onset, development and outcome of the disease. Pathological physiology studies the causes and mechanisms of functional and biochemical disorders that form the basis of the disease, as well as adaptive mechanisms and restoration of functions impaired during the disease.

The course of pathological physiology consists of 3 sections.

1. Nosology, or the general study of a disease, provides answers to 2 questions that a doctor faces when analyzing a disease: why the disease occurred and what is the mechanism of its development (etiology and pathogenesis).
2. Typical pathological processes - studies the processes underlying many diseases (inflammation, fever, tumors, hypoxia).
3. Systemic pathological physiology – examines disorders of individual organs and systems.

The object of study of pathophysiology is a disease, the main method of research is a pathophysiological experiment conducted on animals.

The experiment is used by many sciences (normal physiology, pharmacology, etc.). The significance of the experiment in pathological physiology is the experimental reproduction of the disease in animals, its study and the use of the obtained data in the clinic.

There are 4 stages of the pathophysiological experiment:

- experiment planning;
- reproduction of the model of the pathological process in the experiment and its study;
- development of experimental methods of therapy;
- static processing of the obtained data and analysis of the study.

To study pathological processes in living objects, the following experimental methods are used:

- method of irritation;
- shutdown method;
- switching method;
- parabiosis method;
- cell culture method.

The doctrine of disease, or general nosology, is one of the ancient problems of medicine. Health and disease are the 2 main forms of life. Health and illness can change each other many times during the life of a person and an animal.

Health is, first of all, the state of the body, in which there is a correspondence between structure and function, as well as the ability of regulatory systems to maintain homeostasis. Health is expressed in the fact that in response to the action of everyday stimuli, adequate reactions arise, which in strength, time and duration are characteristic of the majority of people in a given population. A conclusion about health is made on the basis of anthropometric, physiological and biochemical studies.

Disease is a qualitatively new condition that arises under the influence of external and internal pathogenic factors, manifested in the limitation of protective and adaptive capabilities to the action

of environmental factors and a decrease in the biological and social capabilities of the whole organism.

In illness there are always 2 opposing processes, 2 beginnings. During fever, along with high body temperature, headache and other phenomena that reduce a person's ability to work, more active production of antibodies, more energetic phagocytosis and other "measures against the disease " are observed. "Breakdown" and "measure against disease" are inextricably linked; if there is no unity between them, there will be no disease. The complete absence of a defense mechanism leads to death. The complete absence of "break" means health.

The concept of disease is close to the concepts of pathological reaction, pathological process, pathological condition.

A pathological reaction is an inadequate short-term response of the body to any stimulus. For example, a short-term increase in blood pressure under the influence of negative emotions.

A pathological process is a complex set of pathological reactions. Typical pathological processes include inflammation, fever, etc.

A pathological condition is a slowly developing pathological process or its outcome. For example, cicatricial narrowing of the esophagus that develops after a burn, injury, etc.

The course and outcomes of diseases in children differ from those in adults. With the development of the nervous system and the reactivity of the body, the picture of the disease becomes more complex and the mechanisms of protection, compensatory and adaptive reactions, barrier systems, phagocytosis, and the ability to produce antibodies are improved.

Etiology is the study of the causes and conditions of disease.

The cause of the disease is the main etiological factor that causes specific symptoms of the disease. Most often, the occurrence of the disease is associated with the influence of not one, but several factors. For example, the occurrence of lobar pneumonia can be influenced by negative emotions, poor nutrition, hypothermia, and overwork. However, without the penetration of pneumococcus into the body, these factors will not cause pneumonia.

In the history of the development of etiology, different directions were known. According to the direction of monocasualism, any disease arises due to one single cause, so the action of this cause will necessarily lead to the disease. According to a school of thought called conditionalism, disease is caused by many different conditions, but none of them can be the cause. Supporters of this direction believed that for the occurrence of a disease, all conditions of the disease are equally necessary; if at least one is not present, then the disease will not arise. They, overestimating the significance of conditions, completely excluded causal factors. At the same time, there was also a movement in etiology called constitutionalism. According to this trend, it is believed that the occurrence of a disease is determined only by constitutional features of the organism. Since constitutional features are associated with heredity, the occurrence of the disease is directly dependent not on environmental factors, but on the genotype.

The following causes of the disease are distinguished:

1. Mechanical factors (wounds, compression).
2. Physical factors (sound, change in barometric pressure, influence of high or low temperature).
3. Chemical factors (alcohol, acids and alkalis).
4. Biological factors (bacteria, viruses, fungi).
5. Social factors (medical provision, sanitary and hygienic measures).

The interaction of the cause of the disease with the body always occurs under certain conditions. The difference between conditions and cause is that there is one cause, and there are many conditions, and that the latter are not necessary for the occurrence of the disease and do not give it specificity. Pathogenesis is a branch of pathological physiology that studies the mechanisms of development and outcome of the disease. It is very closely related to the etiology of the disease. The

main and most general pattern of pathogenesis is the pattern of self-development and self-maintenance.

The change of causes and effects leads to a vicious circle. Among the links of pathogenesis there are major and minor ones. The main link is the link necessary for all the others.

Due to the peculiarities of the body's reactivity in childhood, the following pattern is characteristic of pathology during this period: the younger the child, the less pronounced the specific signs of the disease.

4. Illustrative material:

- presentation of lecture material;
- posters on the topic of the lesson;
- tables, diagrams.

5. Literature: see Appendix No. 1.

6. Test questions (feedback)

1. What does pathophysiology study?
2. What is the essence of a pathophysiological experiment?
3. What is a disease?
4. What is the difference between a pathological reaction, a pathological process and a pathological condition?
5. What are the characteristics of the course and outcomes of diseases in children?
6. What is etiology?
7. What causes the disease?
8. What is pathogenesis?
9. How does a vicious circle of pathogenesis arise?
10. What are the main causes of illness in childhood ?

Lecture No. 2

1.Topic: Cell pathology.

2.Goal: explain local and general mechanisms of cell damage.

3.Lecture abstracts

Cell damage **is** a typical pathological process. The causes of cell damage may be the following factors:

1) Hypoxia is an extremely important and common cause of cell damage. Reduced blood circulation, which occurs with atherosclerosis, thrombosis, and compression of the arteries, is the main cause of hypoxia.
2) Physical agents - mechanical trauma, temperature effects, barometric pressure fluctuations, - ionizing and ultraviolet radiation, electric current.
3) Chemical agents and drugs.
4) Immunological reactions.
5) Genetic damage (for example, hereditary membranopathies, enzymopathies, etc.).
6) Nutritional imbalance.

Cell death is the end result of its damage. There are two main types of cell death: necrosis and apoptosis. Today, there is also a third type of cell death - terminal differentiation, which, according to most modern scientists, is a form of apoptosis.

Necrosis is a pathological form of cell death due to its irreversible chemical or physical damage (high and low temperature, organic solvents, hypoxia, poisoning, hypotonic shock, ionizing radiation, etc.). Necrosis is a spectrum of morphological changes resulting from the destructive action of enzymes on the damaged cell. Two competing processes develop: enzymatic digestion of the cell (collective, liquefying necrosis) and denaturation of proteins (coagulative necrosis). Both of these processes require several hours to manifest, so in the event of sudden death, for example, with myocardial infarction,

the corresponding morphological changes simply do not have time to develop. This type of cell death is not genetically controlled.

Necrosis may be preceded by periods of paranecrosis and necrobiosis.

Paranecrosis is noticeable but reversible changes in the cell: turbidity of the cytoplasm, vacuolization, appearance of coarse sediments, increased penetration of various dyes into the cell.

Necrobiosis is a state "between life and death", changes in a cell that precede its death. With necrobiosis, unlike necrosis, it is possible for the cell to return to its original state after eliminating the cause that caused the necrobiosis.

The peculiarity of cell damage in a child's body is associated with increased lability of the nervous system, especially its sympathetic part, which leads to excessive formation of adrenaline, and adrenaline, in turn, causes the rapid breakdown of glycogen in the liver and muscles.

4.Illustrative material:

-presentation of lecture material;

-posters on the topic of the lesson;

- tables, diagrams.

5.Literature: see Appendix No. 1.

6.Test questions (feedback)

1.What is cell damage?

2. What are the types of cell damage?

3. What are the causes of cell damage?

4. What are the general mechanisms of cell damage?

5. What are the features of cell damage in a child's body?

Lecture No. 3

1. Topic: Hypoxia. Peripheral circulation disorders.

2. Purpose: - explain the etiology and pathogenesis of hypoxic conditions, give an idea of the significance of hypoxia in the pathogenesis of structural and functional changes in cells and tissues during pathology.

- explain the main causes and mechanisms of development of peripheral circulatory disorders.

3. Lecture abstracts

Hypoxia is a typical pathological process that occurs as a result of insufficiency of biological oxidation and the resulting energy deficiency of life processes.

Classification of hypoxic conditions

1. Exogenous:

A) hypobaric;

B) normal pressure.

2. Respiratory (breathing).

3. Circulatory (cardiovascular).

4. Hemic (blood).

5. Tissue (primary tissue).

6. Mixed.

According to the criteria for the prevalence of hypoxic conditions, they are distinguished: a) local; b) general hypoxia.

According to the speed of development and duration: a) fulminant; b) acute; c) subacute; d) chronic.

By severity: a) mild; b) moderate; c) severe; d) critical.

Hypobaric hypoxia develops when atmospheric pressure decreases. It is most often observed during high mountain ascents. The leading pathogenetic factor in its occurrence is also hypoxemia, but in contrast to normobaric hypoxia, hypocapnia is an additional negative factor. Normobaric hypoxia occurs in cases when, at normal atmospheric pressure, the oxygen content in the inhaled air

decreases. A similar situation can arise during a long stay in unventilated spaces of small volume, when working in wells and mines.

Respiratory hypoxia occurs as a result of insufficient gas exchange in the lungs due to alveolar hyperventilation, ventilation-perfusion disorders relationships, excessive outside-and intracellular shunting of venous blood or when oxygen diffusion in the lungs is difficult. The pathogenetic basis of respiratory hypoxia, as well as exogenous one, is also arterial hypoxemia, in most cases combined with hypercapnia.

Circulatory disease develops when there are circulatory disorders, leading to insufficient blood supply to organs and tissues. The main reason for the development of this type of hypoxia is circulatory disorders: general and local.

During hemic hypoxia, due to quantitative and qualitative changes in hemoglobin, the function of oxygen transport in the blood is disrupted. Quantitative changes in hemoglobin are associated with a decrease in the number of red blood cells. A decrease in the number of red blood cells can occur with anemia (anemia) and with acute or chronic blood loss.

Tissue hypoxia develops due to a disruption in the ability of cells to absorb oxygen.

Mixed hypoxia is a combination of 2 or more basic types.

Urgent compensatory reactions occur reflexively and are manifested in deepening and quickening of breathing, an increase in the minute volume of breathing, and the inclusion of reserve alveoli.

Long-term compensatory reactions occur during chronic hypoxia. This is manifested in the respiratory system by an increase in the diffusion volumes of the lungs, in the cardiovascular and blood systems by myocardial hypertrophy, due to the activation of erythropoiesis processes in the bone marrow, an increase in the number of red blood cells and hemoglobin concentration.

The peculiarity of the course of hypoxic conditions in young children is associated with the immaturity of the respiratory and cardiovascular systems.

The main forms of peripheral circulatory disorders include: 1) arterial hyperemia; 2) ischemia; 3) venous hyperemia; 4) disturbance of the rheological properties of blood, causing stasis in microvessels.

Arterial hyperemia is blood filling that develops as a result of an increase in blood flow through the arteries to organs and tissues.

External manifestations of arterial hyperemia include:
- expansion of small arteries and arterioles;
- increase in the number of functioning capillaries;
- redness of organs and tissues;
- increase in the volume of organs and tissues;
- increase in local temperature.

Arterial hyperemia can be physiological and pathological. Physiological arterial hyperemia is observed with increased organ functioning, overheating, massage, and emotional stress. Pathological arterial hyperemia develops under various conditions (inflammation, allergies, burns, fever, trauma, neuralgia, etc.).

Ischemia is a disorder of peripheral circulation that develops as a result of a decrease or complete cessation of blood flow to organs and tissues through arterial vessels.

Causes of ischemia: compression of the artery from the outside (compressive ischemia); blockage of the artery lumen by a thrombus, embolus, or foreign body (obstructive ischemia); artery spasm (angiospastic ischemia).

External manifestations of ischemia include:
- pallor of organs and tissues;
- reduction in the volume of organs and tissues;
- decrease in local temperature;

- dysfunction of organs.

Venous hyperemia is a blood filling that develops as a result of a violation of the outflow of blood through the veins from organs and tissues.

External manifestations of venous hyperemia include:
- cyanosis of organs and tissues (cyanosis);
- increase in the volume of organs and tissues;
- decrease in local temperature;
- development of edema.

In the fetus, newborn and child of the first 3 years of life, general and local plethora, anemia, stasis occur more easily and more often than in adults, which depends on the immaturity of the regulatory mechanisms of blood circulation.

4. Illustrative material:
- presentation of lecture material;
- posters on the topic of the lesson;
- tables, diagrams.

5. Literature: see Appendix No. 1.

6. Test questions (feedback)
1. What is hypoxia?
2. What types of hypoxia are there ?
3. What is the body's emergency adaptation to hypoxia?
4. What is the body's long-term adaptation to hypoxia?
5. What are the features of the development of hypoxic conditions in children ?
6. What types of peripheral circulatory disorders are there?
7. What causes arterial hyperemia?
8. What are the mechanisms of ischemia ?
9. What are the symptoms of venous hyperemia?
10. What are the features of peripheral circulatory disorders in children?

Lecture No. 4

1. Topic: Inflammation.

2. Purpose: to explain the main causes and mechanisms of inflammation.

3. Lecture abstracts

Inflammation is a typical process, which is based on the influence of a damaging (phlogogenic) factor. With inflammation in damaged tissue or organ, disturbances in cell structure, changes in blood circulation, increased vascular permeability and tissue proliferation are observed. Phlogogenic factors are divided into 2 groups – exogenous and endogenous.

Exogenous microorganisms include bacteria (bacteria, viruses, fungi); animal organisms (protozoa, worms, insects); chemicals (acids, alkalis); mechanical influences (foreign body, pressure); thermal effects (cold, heat); radiation energy (X-rays, radioactive, ultraviolet rays).

Endogenous factors include: accumulation of salts in joints, thrombosis, embolism. For example, at the site of a heart attack associated with impaired microcirculation, an inflammatory process develops.

The inflammatory process consists of 3 stages:
Stage 1 – alteration;
Stage 2 – exudation with emigration of leukocytes;
Stage 3 – proliferation.

Vascular changes occur in 4 phases:
Phase 1 – vasospasm;
Phase 2 – arterial hyperemia;
Phase 3 – venous hyperemia;

Phase 4 – stasis.

Mediators of inflammation:

a) mediators of humoral origin (kinins, complement system);

b) mediators of cellular origin, ready or pre-existing (mast cell mediators, serotonin, heparin, lysosomal enzymes);

c) mediators of cellular origin, newly formed (eicosanoids, lymphokines, monokines, free radicals).

Alteration can be primary and secondary. Primary alteration occurs due to direct exposure to a damaging agent. Secondary alteration is the body's response to primary alteration.

Exudation is the exudation of the protein-containing liquid part of the blood through the vascular wall into the inflamed tissue. The fluid released from the vessels into the tissue during inflammation is called exudate. Depending on the qualitative composition, the following types of exudates are distinguished: serous, fibrinous, purulent, putrefactive, hemorrhagic, mixed.

According to the development mechanism, the exudation process is associated with the influence of inflammatory mediators. The leading factor in exudation is considered to be an increase in vascular permeability.

Emigration is the movement of leukocytes outside the vessels. Polymorphonuclear leukocytes are the first to be found at the site of inflammation. The main function of leukocytes at the site of inflammation is the absorption of foreign bodies (phagocytosis).

Proliferation – 3rd stage of inflammation. After several hours of performing the phagocytic function, leukocytes die. Macrophages cleanse the site of inflammation from microorganisms. Dead cells release substances that stimulate proliferation.

of the inflammatory process in children is the tendency to generate it. Children usually develop necrosis, widespread in most organs and mucous membranes of the skin.

4. Illustrative material:

- presentation of lecture material;
- posters on the topic of the lesson;
- tables, diagrams.

5. Literature: see Appendix No. 1.

6. Test questions (feedback)

1. What is inflammation ?
2. What factors cause inflammation?
3. What are the stages of inflammation?
4. What vascular changes occur at the site of inflammation?
5. What are the features of the course of the inflammatory process in children?

Lecture No. 5

1. Topic: Immunopathological processes.

2. Purpose: to explain the main causes, mechanisms of development and manifestations of allergies; explain the mechanisms of development of the main types of allergic reactions.

3. Lecture abstracts

Allergy is an altered immune response to foreign substances, characterized by damage to one's own tissues.

Many substances have antigenic properties to cause allergic reactions. They are called allergens.

Classification of allergens

Allergens are exogenous and endogenous. Exogenous allergens enter the body from the environment, while endogenous allergens are formed in the body itself. Exoallergens are divided into two types: infectious and non-infectious. Infectious allergens include bacteria, viruses, fungi and helminths. Among non-infectious allergens one can name household (house dust, cosmetics), epidermal (wool, down and animal hair), plant (pollen, fruits), food (fish, chocolate, nuts, eggs),

medicinal (antibiotics, sulfonamides, chloramine). These allergens enter the body from the outside through the respiratory, digestive tract, and skin.

Classification of allergic reactions

There are several views regarding the classification of allergic reactions. R. Cook (1930) divided all allergic reactions into 2 types: allergic reactions of the immediate type and allergic reactions of the delayed type. Immediate allergic reactions are observed a few minutes after the allergen re-enters the body.

Delayed allergic reactions are observed after 24-48 hours after the allergen re-enters the body.

In 1969, Jell and Coombs divided allergic reactions into 4 types:

- allergic reactions of reagin or anaphylactic type – type I (atopic bronchial asthma, hay fever);
- allergic reactions of the cytotoxic type – type II (hemolytic anemia, agranulocytosis);
- allergic reactions of the immune complex type – type III (serum sickness);
- allergic reactions of the cytotoxic type – type IV (contact dermatitis);

The mechanism of development of allergic reactions consists of 3 stages:

I. Immune stage. The body produces antibodies or sensitized T-lymphocytes to a specific allergen. This stage is called sensitization. Sensitization is a gradual increase in sensitivity after an allergen enters the body.

II. Pathochemical stage. At this stage, as a result of the interaction of the allergen and a specific antibody or sensitized T-lymphocyte, allergy mediators are released.

III. Pathophysiological stage. Under the influence of mediators, violations of specific functions of organs and systems occur: increased blood pressure, increased permeability of the vascular wall, edema, bronchospasm.

Allergic reactions type I (reagin)

In the immune stage, T cells, under the influence of allergens and macrophages, produce interleukin-4 and stimulate B cells. After this, they turn into plasma cells and produce IgE. IgE attaches to mast cells or basophils in the blood. When the allergen is re-entered, it binds to IgE. After this, intracellular granules are released (degranulation). In the 2nd stage of allergic reactions, the interaction of allergens with antibodies occurs. This will lead to the release of mediators. In immediate allergic reactions, histamine, serotonin, and bradykinin are released. The 3rd stage of allergic reactions is considered a combination of functional, biochemical and structural changes. At this stage, disorders of the cardiovascular, respiratory, digestive, endocrine and nervous systems may develop. These include microcirculation disorders (dilation of capillaries, increased permeability, changes in the rheological properties of blood), bronchospasm, increased glucocorticoids, changes in the processes of excitation and inhibition at different levels of the nervous system.

Allergic reactions type II (cytotoxic)

At the stage of immune reactions, autoallergen recognition occurs with the participation of macrophages, T- and B-lymphocytes, B-lymphocytes turning into plasma cells produce IgG_1 and IgM. These antibodies attach to cells with autoallergens. Then the stage of pathochemical changes develops, allergy mediators are formed. These mediators include complement components, lysosomal enzymes, and oxygen free radicals. At the stage of pathophysiological disorders, destruction of cells containing allergens is observed. According to cytotoxic type II allergic reactions develop hemolytic anemia, thrombocytopenia, autoimmune thyroiditis, myocarditis, hepatitis and other autoimmune diseases.

Allergic reactions type III (immune complex)

Allergens are drugs, medicinal serums, foods, mushrooms, etc. Plasma cells form IgG_1, IgG_4 and IgM. These antibodies in biological fluids bind to allergens and form allergen-antibody immune complexes. If this complex contains a slight excess of antigen, it sticks to the capillary wall. Due to the formation of the allergen-antibody complex, a certain amount of allergy mediators (complement, lysosomal enzymes, oxygen free radicals, histamine, serotonin) are released. Complement

components increase the permeability of blood vessel walls. Immune complexes attach to platelets and destroy them.

Allergic reactions type IV (cell-mediated)

Allergens are proteins, glycoproteins and chemicals that bind to proteins. These reactions develop to proteins of low molecular weight and weak ability to form antibodies. The cellular immune response is carried out by T cells. Allergens that enter from outside or form in the body bind to macrophages and develop. After the re-entry of the allergen into the body, T-cells are very important in the formation of a rapid immune response. They bind to allergens. Attached to cells that have allergens on their surface, sensitized T cells produce cytokine mediators. From their exposure, after a few hours, inflammation develops at the location of the allergen.

Due to the differentiation of antibodies in young children, a certain resistance to allergies is observed. Therefore, allergic manifestations in children have a number of features.

4. Illustrative material:

- presentation of lecture material;
- posters on the topic of the lesson;
- tables, diagrams.

5. Literature: see Appendix No. 1.

6. Test questions (feedback)

1. What are allergies ?
2. What factors cause allergies?
3. How can allergic reactions be classified?
4. What is the general pathogenesis of allergic reactions?

Lecture No. 6

1. Topic: Tumors.

2. Purpose: to explain the etiology, mechanisms of development, biological features of tumor growth and mechanisms of antiblastoma resistance.

3. Lecture abstracts

A tumor is a typical pathological process of unlimited tissue growth, not related to the general structure of the damaged organ.

There are 2 clinical types of tumors: benign and malignant.

Benign tumors grow by pushing them away, squeezing surrounding tissues. This type of growth is called expansive. Benign tumors, depending on the tissue located, are named by adding the ending "oma" to the name of this tissue. For example, fibroma, osteoma, adenoma, melanoma, neuroma, angioma, etc.

Malignant tumors grow, damaging surrounding tissues, spreading through the vessels. This type of growth is called infiltrative. In the mechanisms of development of infiltrative growth and spread of metastases, the characteristics of tumor cell membranes are of great importance.

Causes of carcinogenesis

1. Viral carcinogenesis.
2. Chemical carcinogenesis.
3. Radiation carcinogenesis.

Pathogenesis of tumors

There are the following stages of carcinogenesis: Stage 1 - the transformation of a healthy cell into a tumor cell, called initiation. Stage 2 – rapid development and proliferation of tumor cells and the formation of primary tumor nodes, called promotion. Together, initiation and promotion are transformation. Stage 3 – increased persistent qualitative changes and malignant properties of tumor cells, or increased tumor growth, called progression.

Transformation is the transformation of a normal cell into a tumor cell under the influence of carcinogenic substances. The primary mechanisms of transformation of a normal cell into a tumor cell are still unknown.

Cells of malignant tumors can remain in a latent (hidden) period for a long time after transformation, without showing active growth processes.

Transformed cells, under the influence of one additional factor, can enter the 2nd stage of carcinogenesis - promotion. In organs with transformed cells, mechanical influences and the influence of inflammatory processes increase their activity. In the 3rd stage of cellular carcinogenesis - progression in any population of young malignant tumors, an increase in tumor growth is observed.

Tumor progression is an increase in the malignant properties of a tumor in tumor cells due to their proliferation.

Relationship between tumors and the body

During the development of a tumor, the functioning of the entire body is disrupted. This is due to the peculiarities of metabolic processes in tumor cells. Due to decreased immunity, resistance against infectious diseases decreases. So, if death does not occur as a result of significant damage, it may occur as a result of sepsis. During swelling, a lot of energy is consumed. And under these conditions, under-oxidized products accumulate, and metabolic acidosis occurs. The permeability of cell membranes increases, and disruption of cell structure is observed.

Tumors in children are a consequence of a violation of embryogenesis - dysembryoplasia, sometimes they are combined with congenital malformations.

4. Illustrative material:

- presentation of lecture material;
- posters on the topic of the lesson;
- tables, diagrams.

5. Literature: see Appendix No. 1.

6. Test questions (feedback)

1. What is a tumor ?
2. What types of tumors are there ?
3. What are the causes of carcinogenesis ?
4. What is the pathogenesis of tumors ?
5. What are the features of the development of tumors in children?

Appendix No. 1

Literature: basic and additional

In Russian

main:

1. Ado. Pathophysiology: Textbook, T. 1-2. - Evero, 2015.
2. Pathological physiology (General and Particular): textbook / V.A. Frolov [and others]. - 4th ed., transl. and add. - M.: Publishing House «Higher Education and Science», 2019. - 730 pp.: with ill.
3. Pathophysiology. In 2 volumes. T. 1-2: textbook / ed. V. V. Novitsky, E. D. Goldberg, O. I. Urazova; Ministry of Education and Science of the Russian Federation. - 4th ed., revised. and additional; Rec. State Educational Institution of Higher Professional Education «MMA named after I.M. Sechenov». - M.: GEOTAR - Media, 2012. - 1488 p.

additional:

1. Pathological physiology. The same thing: pathophysiology. Guide to practical exercises: textbook. allowance / V. V. Novitsky [f. b.]; ed. Bask. V. V. Novitsky, O. I. Urazova; Kaz. tiline aud. S. B. Zhutikova. - M.: GEOTAR - Media, 2014. - 768 bet p.
2. Pathophysiology. Tasks and test tasks: textbook / ed. P. F. Litvitsky; Ministry of Education and Science of the Russian Federation. - Rec. State Educational Institution of Higher Professional Education «MMA named after I.M. Sechenov». - M.: GEOTAR - Media, 2013. - 384 p.: ill.
3. Pathophysiology: hands. for practical exercises: textbook. allowance / edited by V. V. Novitsky, O. I. Urazova. - M.: GEOTAR - Media, 2011. - 336 p.

electronic resources:

1. UMKD is posted on the educational portal https :// skma . kz
2. Pathological physiology. Tazhiribelik sabaktarga nuskau [Electronic resource]: oku kuraly = Pathophysiology. Guide to practical exercises: textbook/Kaz. tiline aud. S. B. Zhutikova; ed. V. V. Novitsky. - Electron. text data (63.4MB). - M.: GEOTAR - Media, 2014. - 768 bet, email. wholesale disk.
3. Pathophysiology. In 2 vols. T. 1-2 [Electronic resource]: textbook / ed. V. V. Novitsky. - 4th ed., revised. and additional - Electron. text data (59.9 MB). - M.: Publishing group «GEOTAR - Media», 2010. - 1488 p. email wholesale disk (CD-ROM).

In Kazakh language

main:

1. Pathophysiology. Ekitomdyk. 1-2 t.: okulyk/ kazakh til. room B. A. Zhetpisbaev, S. B. Zhutikova; ed. V. V. Novitsky. - M.: GEOTAR - Media, 2018. - 1104 b. + email optical disk (CD-ROM).
2. Ado. Pathophysiology: Okulyk. 1-2 t. - Evero, 2015.
3. Nurmukhambetuly, A. Pathophysiology. 1-4 t.: okulyk/Ә. Nurmuhambetuly. - Ond., tolykt. 4-bass - Almaty: Evero, 2015. - 766 bet p.

additional:

1. Nurmukhambetuly, A. Pathophysiology-1. Clinic practiceda ote manyzdy bertektes derttik urdisterdin pathogenesis men emdeu zholdaryna nuskama. 1-2 t.: oku kuraly. - Almaty: Evero, 2016. - 504 b.
2. Pathological physiology. The same thing: pathophysiology. Guide to practical exercises: textbook. allowance / V. V. Novitsky [f. b.]; ed. Bask. V. V. Novitsky, O. I. Urazova; Kaz. tiline aud. S. B. Zhutikova. - M.: GEOTAR - Media, 2014. - 768 bet p.
3. Urazalina, N. M. Tapsyrmalary test. I-II bolim. Nosology, etiology and pathogenesis, reactive pathology and allergy. Birtektes derttik urdister. - Almaty: Evero, 2014. - 386 bet.

4. Zhautikova, S. B. Mamandandyrylgan pathology physiology courses: oku-adistemelik kuraly. - Karaganda: Residential complex «AkNur», 2013.
5. Zhautikova, S. B. Pathological physiology of the situation in the esepter zinagy: oku-adistemelik kural. - Karaganda: Residential complex «AkNur», 2013.

electrons resourcetar:

1. Pathology. Ekitomdyk. 1-2 t.: [Electronic resource]: okulyk/ kaz. tel. room S. A. Apbasova; ed. M. A. Paltsev. - Electron. text data (1.42MB). - M.: GEOTAR - Media, 2015. - 536 bet, email. wholesale disk.
2. Pathological physiology. Tazhiribelik sabaktarga nuskau [Electronic resource]: oku kuraly = Pathophysiology. Guide to practical exercises: textbook/Kaz. tiline aud. S. B. Zhutikova; ed. V. V. Novitsky. - Electron. text data (63.4MB). - M.: GEOTAR - Media, 2014. - 768 bet, email. wholesale disk.

In English

main:

1. Norris Tommie L. Porth's Pathophysiology: Concepts of Altered Health States: Textbook/ Tommie L. Norris. - 10th ed. - [sl]: Wolters Kluwer, 2019. - 1573 p.
2. Kumar V. Robbins Basic Pathology: Textbook/ V. Kumar, AK Abbas, JC Aster. - 10th ed. - [sl]: Elsevier, 2018. - 935 p.
3. Pathophysiology. Volume 1-3: the book for medical institutes/ AD Ado [and others]. - Almaty: «Evero», 2017. - 732 p.
4. Zhautikova, SB Review of pathophysiology: educational-methodical manual/ SB Zhautikova, U. Faroog. - Karaganda: АҚҺҰР, 2017. - 388 p.
5. Zhautikova, SB Collection of situational problems for discipline of pathological physiology-2: educational-methodical manual/ SB Zhautikova, U. Faroog. - Karaganda: АҚҺҰР, 2017. - 126 p.
6. Kumar V. Robbins and Cotran. Pathologic Basis of Disease: Textbook/ V. Kumar, Abyl K. Abbas, Jon C. Aster. - 9th ed. - [sl]: Elsevier/Saunders, 2015. - 1391 p.

Electronic databases

No.	Name	Link
1	SKMA repository	http://lib.ukma.kz/repository/
2	Republican Interuniversity Electronic Library	http://rmebrk.kz/
3	Student Advisor	http://www.studmedlib.ru/
4	Open University of Kazakhstan	https://openu.kz/kz
5	Law (access in the reference and information sector)	https://zan.kz/ru
6	Paragraph	https://online.zakon.kz/Medicine/
7	Scientific electronic library	https://elibrary.ru/
8	Ashyk kitapkhana	https://kitap.kz/
9	Thomson Reuters « Web of Science «	www.webofknowledge.com
10	ScienceDirect	http://www.sciencedirect.com/
eleven	Scopus	https://www.scopus.com/