LECTURE COMPLEX

Discipline: General pathological physiology

Discipline code: GP 3201-2

EP: 6B130100 - "General Medicine"

Volume of teaching hours/credits: 150 hours/5 credits

Course and semester of study: III course, V semester

Lecture volume: 15 hours

Shymkent, 2022

Lecture complex was developed in accordance with the working program of the discipline (syllabus) 6 B130100-"General Medicine" and discussed at a meeting of the department

Protocol No. 10 from " 6 " 06 2022

Head Department _____ Zhakipbekova G.S.

Lecture # 1

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

2. Purpose: To characterize the purpose, tasks and methods of pathophysiology; define the basic concepts of general nosology.

3. Abstracts of the lecture

Pathological physiology is the main fundamental medical and biological science that studies the general features of the onset, development and outcomes 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 disturbed during the disease.

The course of pathological physiology consists of 3 sections .

1. Nosology , or the general doctrine of the disease, - gives answers to 2 questions that the doctor faces when analyzing the disease: why the disease arose 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. Private pathological physiology - considers violations of individual organs and systems .

The object of study of pathophysiology is a disease, the main research method 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 lies in the experimental reproduction of the disease in animals, its study and the use of the data obtained in the clinic.

There are 4 stages of the pathophysiological experiment:

- planning of the experiment;
- 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:

- stimulation method;
- shutdown method;
- switching method;
- parabiosis method;
- cell culture method.

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

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 occur, which, in terms of strength, time and duration, are characteristic of most people in this population. The conclusion about health is made on the basis of anthropometric, physiological and biochemical studies.

A disease is a qualitatively new condition that occurs 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 opposite processes, 2 beginnings. During a fever, along with high body temperature, headache and other phenomena that reduce a person's ability to work, more active production of antibodies, more vigorous phagocytosis, and other "measures against the disease " are observed. " Sex " and " measure against the disease " are inextricably linked if there will be no unity, there will be no disease. The complete absence of a defense mechanism leads to death. The complete absence of " polom " 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 .

The pathological process is a complex set of pathological reactions. Type pathological processes include inflammation, fever , etc.

A pathological condition is a slowly developing pathological process or its outcome . For example, developing after a burn, injury, cicatricial narrowing of the esophagus, 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 organism, the picture of the disease becomes more complicated and the defense mechanisms, compensatory-adaptive reactions, barrier systems, phagocytosis, and the ability to produce antibodies are improved.

4. Illustrative material:

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

5. Literature: see Appendix No. 1.

6. Control questions (feedback)

1. What does pathophysiology study?
2. What is the essence of the 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 features of the course and outcomes of diseases in children?

Lecture #2

1. Topic: General etiology and pathogenesis.

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

3. Abstracts of the lecture

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

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

In the history of the development of etiology, different directions were known. According to the direction of monocausalism, any disease occurs due to a single cause, so the action of this cause will necessarily lead to illness. According to the direction called conditionalism, the disease is caused by many different conditions, but none of them can be the cause. Supporters of this trend believed that all the conditions of the disease are equally necessary for the occurrence of the disease, if there is not at least one, then the disease will not arise. They, overestimating the importance of conditions, completely excluded causal factors. Along with this, in etiology there was

also a trend called constitutionalism. According to this current, it is believed that the occurrence of the disease is determined only by
constitutional features of the organism. Since constitutional features are associated with heredity, the onset of the disease is directly
subject not to environmental factors, but to the genotype.

There are the following causes of the disease:

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 security, sanitary and hygienic measures).

The interaction of the cause of the disease with the body always occurs under certain conditions. The difference between the conditions and the cause is that the cause is one, but there are many conditions, and that the latter are not necessary for the onset of the disease and do not give it specificity. Pathogenesis is a section of pathological physiology that studies the mechanisms of development and outcome of a disease. It is 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 cause and effect leads to a vicious circle. Among the links of pathogenesis there are main and secondary ones. The main one is the link necessary for all the others.

In connection with the peculiarities of the reactivity of the organism in childhood, the pathology during this period is characterized by the following pattern: 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. Control questions (feedback)

1 . What is etiology ?
2 . What causes disease?
3 . What is pathogenesis?
4 . How does the vicious circle of pathogenesis arise?
5. What are the main causes of childhood illnesses ?

Lecture #3

1. Topic : The role of reactivity in pathology.

2. Purpose: to explain the role of the body's reactivity in the occurrence of diseases; to establish the relationship between the reactivity of the organism and the severity of the manifestations of pathological changes.

3. Abstracts of the lecture

Reactivity is the property of an organism to react in a certain way to the influence of environmental factors.

Resistance is the body's resistance to pathogenic environmental factors.

Reactivity is divided into species, group and individual. Species reactivity is the reactivity of a particular biological species.

People are divided into blood groups, gender groups, age groups, etc. In different groups, reactivity is different.

Individual reactivity shows the reactivity of a particular organism.

Each person is a unique individual. Individual reactivity is divided into primary congenital and secondary acquired.

It is noted that the basis of primary reactivity is heredity, constitution, age and gender.

The first and main element of reactivity are hereditary characteristics, they are transmitted to offspring with the help of genes. The next significant element of reactivity is gender. In the formation of the reactivity of the body there are certain gender differences. Sex differences, which determine the different reactivity of living organisms, are manifested in early postnatal ontogenesis. Newborn boys die more often than girls. Women have high resistance to strong extreme factors. They remain stable in conditions of injury, ionizing radiation, starvation, blood loss. Such phenomena were also observed under experimental conditions. We can assume that the features of sexual reactivity are associated with the properties of sex hormones.

The primary factors determining reactivity include
person's age. The reactivity of a newborn child is different from
adult reactivity. In children, the function of the immune system
is at a low level, so their body does not form antibodies on its own. In an adult organism, due to the sufficient development of the nervous system, the normal state of the immune system, and the formation of antibodies, reactivity is high compared to children. In old people, reactivity decreases, sensitivity to the influence of environmental factors increases. The reason for this is that in old people, metabolic processes in the nervous system, the function of the immune system, and the function of antibody production decrease. In this regard, old people are susceptible to infectious diseases.

Reactivity can be specific and nonspecific. The protective mechanisms of specific reactivity are characterized by allergy and immunity. Nonspecific acquired reactivity is closely related to numerous environmental factors. The human and animal organism is in a strong dynamic balance with the environment, and the violation of this balance leads to a change in reactivity.

The reactivity of the child's body is different from the reactivity of adults. It changes in the process of development and differentiation of organs and tissues.

4. Illustrative material:

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

5. Literature: see Appendix No. 1.

6. Control questions (feedback)

1. What is reactivity ?
2. What are the types of reactivity ?
3. What is resistance ?
4. What factors determine individual reactivity ?
5. What are about the features of the reactivity of the child's body ?

Lecture #4

1. Topic : The role of heredity in pathology.

2. Purpose: to determine the role of the organism 's heredity in the occurrence of diseases; explain the general etiology and pathogenesis of hereditary diseases.

3. Abstracts of the lecture

Mutations that cause changes in the genotype of an organism lead to hereditary diseases. Distinguish gene, chromosomal and genomic mutations.

Gene mutations occur when there are changes in individual genes. They develop in the absence of certain nucleotides in the DNA molecule or their replacement with other nucleotides.

Chromosomal mutations are observed as a result of changes in chromosomes (deletion, duplication, inversion, translocation).

A change in the number of chromosomes with their structure unchanged is called a genomic mutation.

Depending on the type of cells in which the mutation occurred, gametic and somatic mutations are distinguished. A gametic mutation occurs in germ cells and is passed on from generation to generation. Somatic mutation is not passed on to offspring.

Hereditary diseases are divided into 3 large groups:

- actually hereditary diseases - the main etiological factor of these diseases is considered to be altered hereditary information;
- diseases that develop with a change in the genetic apparatus, but for their clinical manifestation, the action of specific factors is necessary - for example, for the manifestation of sickle cell anemia in a heterozygous organism, a decrease in oxygen in the inhaled air is necessary;
- multifactorial diseases - for example, they include hypertension, coronary heart disease, peptic ulcer of the stomach and duodenum, malignant tumors, diabetes mellitus , which are common especially among adults and the elderly.

All gene diseases by type of inheritance are autosomal dominant, autosomal recessive, codominant, mitochondrial and sex-related.

Chromosomal diseases are diseases associated with changes in the structure or number of chromosomes. Changes in the structure of chromosomes are called aberrations.

Methods for studying hereditary diseases include:

- genealogical;
- twin;
- population;
- cytological;
- biochemical;

Congenital disorders can be hereditary or non-hereditary, with non-hereditary congenital disorders being more common. Most of the hereditary diseases manifest themselves immediately after birth and are congenital pathologies.

4. Illustrative material:

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

5. Literature: see Appendix No. 1.

6. Control questions (feedback)

1 . What is the general etiology and pathogenesis of hereditary diseases ?
2 . What are the types of inheritance of diseases?
3. Types of hereditary diseases?
4. What are the principles of prevention and treatment of hereditary diseases?
5. What are What is the difference between hereditary and congenital diseases ?

Lecture #5

1. Topic: General cell pathology.

2. Purpose: to explain the local and general mechanisms of cell damage.

3. Abstracts of the lecture

Cell damage **is a** typical pathological process . Causes of cell damage can be the following factors:

1) Hypoxia is an extremely important and common cause of cell damage. The decrease in blood circulation that occurs with atherosclerosis, thrombosis, arterial compression is the main cause of hypoxia.
2) Physical agents - mechanical trauma, temperature effects, fluctuations in barometric pressure, - 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 cell damage. There are two main types of cell death , necrosis and apoptosis. To date, a third type of cell death is also distinguished - terminal differentiation, which, according to most modern scientists, is one of the forms of apoptosis.

Necrosis is a pathological form of cell death due to its irreversible chemical or physical damage (high and low temperatures, organic solvents, hypoxia, poisoning, hypotonic shock, ionizing radiation, etc.). Necrosis is a spectrum of morphological changes resulting from the destructive action of enzymes on a damaged cell. 2 competing processes develop: enzymatic digestion of the cell (coagulative necrosis) and protein denaturation (coagulative necrosis). Both of these processes require several hours to manifest, so in case of sudden death, for example, in 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 a noticeable but reversible changes in the cell: turbidity of the cytoplasm, vacuolization, the appearance of coarse sediments, an increase in the penetration of various dyes into the cell .

Necrobiosis is a state “between life and death”, changes in the cell that precede its death. In necrobiosis, in contrast to necrosis, it is possible for the cell to return to its original state after the elimination of the cause that caused necrobiosis.

The peculiarity of damage to the cells of the child's body is associated with an increased lability of the nervous system, especially its sympathetic part, which leads to excessive production of adrenaline, and adrenaline, in turn, is the cause of 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. Control 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 damage to the cells of the child's body?

Lecture #6

1. Topic: General body reactions to damage.

2. Purpose: to explain the causes and mechanisms of development of the body's general reactions to damage.

3. Abstracts of the lecture

Common body reactions to injury include shock, stress, and coma.

Stress is the body's response, manifested by non-specific reactions to any adverse environmental effects and is accompanied by a restructuring of protective and adaptive capabilities. These adverse factors include:
- physical influences;
- chemical influences;
- biological effects;
- psychogenic factors;
- social factors;

Such unfavorable factors are called stressors.

The term “stress” was first introduced into medicine by the Canadian scientist Hans Selye in the 1960s.

Selye called stress the general adaptation syndrome. This syndrome occurs in 3 stages: 1 - stage of anxiety; 2 - stage of resistance; 3 - stage of exhaustion.

Shock is a severe pathological process characterized by a significant decrease in the vital functions of the body. During this period, due to a deep violation of the mechanisms of regulation and microcirculation in organs and tissues, metabolic processes, the body is between life and death.

Types of shock by etiology:
- traumatic;
- burn;
- operational;
- blood transfusion;
- anaphylactic, etc.

Types of shock according to pathogenesis:
- painful;
- humoral;
- psychogenic.

Shock proceeds in 3 stages: 1 - erectile; 2 - torpid; 3 - terminal.

Coma is a condition characterized by a deep loss of consciousness due to a pronounced degree of pathological inhibition of the central nervous system, the absence of reflexes to external stimuli and a disorder in the regulation of vital body functions.

There are the following types of com : 1 - neurological; 2- endocrinological; 3 - toxic; 4 - hypoxic.

In children , shock is the most severe, which is associated with the transitional type of blood circulation in the early period of adaptation and the immaturity of the circulatory system, which is characteristic of the neonatal period.

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

5. Literature: see Appendix No. 1.

6. Control questions (feedback)

1 . What are the stages of stress?
2. What is the protective-adaptive and pathogenic significance of stress?
3 . What are the types of shock?
4 . What is k oma?
5. What are about the features of general reactions to damage in children ?

Lecture No. 7

1. Topic: Violations of water and electrolyte metabolism.

2. Purpose: to explain the main mechanisms of water and electrolyte metabolism disorders.

3. Abstracts of the lecture

Dehydration of the body can develop with the removal of large volumes of water, insufficiency of electrolytes. At this time, intracellular and extracellular water decreases.

There are 2 types of dehydration: 1) with a lack of water in the body; 2) with a lack of mineral salts (electrolytes) in the body.

Dehydration from loss of water occurs in various pathological conditions: with difficulty swallowing; in the weakened and seriously ill; in premature or seriously ill children; hyperventilation of the lungs; when passing too much urine.

Dehydration can develop when electrolytes are deficient, because, even if you do not take into account the other beneficial properties of electrolytes, they have the ability to bind and retain water. Such properties are possessed by sodium, potassium, chlorine ions.

Hypoosmotic dehydration develops with a greater excretion of salts than water from the body. Increased excretion of electrolytes from the body can occur through the gastrointestinal tract, kidneys and skin.

Isoosmotic dehydration develops with an equal loss of water and salts. This condition is possible with polyuria, dyspepsia and blood loss. In this case, the extracellular fluid mainly decreases.

Hyperosmotic dehydration develops when more water is removed from the body than salts. This condition can develop with a large secretion of saliva, increased deep breathing and diabetes insipidus.

With dehydration, after the deterioration of blood circulation in the renal parenchyma, the ability of the kidneys to urinate decreases. This leads to azotemia, then to uremia.

Dehydration from lack of electrolytes cannot be restored with water alone. Water must contain electrolytes. Electrolytes are lost along with water through the digestive system: with vomiting, diarrhea, etc. In addition, the loss of electrolytes and water is observed in certain types of nephritis, Addison's disease. Water and electrolytes are excreted in large quantities even with abundant
sweating. Dehydration affects many body systems. On the part of the cardiovascular system, a decrease in blood pressure, a decrease in the volume of circulating blood, and a thickening of the blood are observed.

Water retention in the body occurs when a large amount of water is consumed or
decrease in excretion processes. Hypoosmotic hyperhydration develops when a large amount of water is introduced into the body. Isoosmotic hyperhydration can be observed for a short time with excessive administration of isotonic fluids for therapeutic purposes. Hyperosmotic hyperhydration is observed with the forced use of sea water, the introduction of a large amount of hypertonic solutions. Since the osmotic pressure is greater outside the cell, more fluid leaves the cell. Because of this, dehydration of the cell develops.

Delay and accumulation of fluid in tissues due to impaired water exchange between blood and tissues is called edema.

The pathological accumulation of fluid in the serous cavities of the body is called dropsy. The accumulation of fluid in the abdominal cavity is called ascites, in the pleural cavity - hydrothorax. There are cardiac, renal, hepatic edema.

With the development of edema, the tissues are mechanically compressed, and blood circulation in them is disturbed. A large amount of fluid in the tissues makes it difficult for the exchange of substances between the cell and the blood. On the other hand, edema has a protective and adaptive property. Edema reduces the concentration of toxic substances entering the body, their absorption, distribution throughout the body.

Instability of water and electrolyte balance in young children is due to the intensive growth of the child and the tension of water-electrolyte metabolism.

4. Illustrative material:

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

5. Literature: see Appendix No. 1.

6. Control questions (feedback)

1 . What are the types of dehydration ?
2 . What is dehydration ?
3 . What is hyperhydration ?
4 . What causes swelling ?
5. What are the features of water-electrolyte metabolism disorders in children?

Lecture No. 8

1. Topic: Disorders of carbohydrate metabolism.

2. Purpose: to explain the etiology and pathogenesis of the main disorders of carbohydrate metabolism .

3. Abstracts of the lecture

Digestion of carbohydrates and their absorption are disturbed in case of insufficiency of amylolytic enzymes of the gastrointestinal tract. One of the main signs of carbohydrate metabolism is a change in carbohydrates in the blood. With the excitation of the central nervous system and the sympathetic nervous system, the breakdown of glycogen in the body increases. On the contrary, its formation is increased in hereditary diseases called glycogenoses. These diseases develop when there is a lack of enzymes that break down glycogen, resulting from gene mutations.

Intermediate metabolism disorders are observed during: 1) hypoxia; 2) liver diseases; $_3$) vitamin B1 deficiency .

An increase in blood glucose is called hyperglycemia. The following types of hyperglycemia are distinguished .

1. Alimentary hyperglycemia develops after taking a large amount of easily digestible carbohydrates. The content of glucose in the blood rises.

2. Emotional hyperglycemia (neurogenic) occurs with strong excitation of the central and sympathetic nervous system. At the same time, the breakdown of glycogen in the liver increases and the transition of carbohydrates into fat decreases.

3. Hyperglycemia in convulsive states - muscle glycogen breaks down , glucose is formed.

4. Hormonal hyperglycemia is observed in violation of the function of the endocrine glands . Hormone α-cells of the pancreas glucagon and
adrenal medulla hormone adrenaline, affecting liver phosphorylase, enhance glycogenolysis. Glucocorticoids increase
gluconeogenesis and inhibit the activity of hexokinase. Growth hormone of the pituitary gland reduces the formation of glycogen, inhibits the activity of hexokinase. These hormones are referred to as contrainsular hormones.

5. Hyperglycemia is observed with some types of anesthesia .

6. Hyperglycemia in insulin deficiency is the most pronounced and constant. Insulin deficiency can be pancreatic (absolute) or extrapancreatic (relative). Insulin deficiency underlies the disease of diabetes mellitus.

Hypoglycemia is a decrease in blood glucose levels . It develops with an increase in insulin and impairs the function of the heart as well as muscles .

The causes of hypoglycemia include: 1) an overdose of insulin in the treatment of diabetes mellitus; 2) a tumor from pancreatic β-cells - insulinoma ; 3) insufficiency of contrainsular hormones ; 4) glycogenoses ; 5) damage to liver cells; 6) alimentary hypoglycemia; 7) impaired absorption of carbohydrates ; 8) renal diabetes .

A feature of the digestion of carbohydrates in children is the different rate of hydrolysis of α-lactose and β-lactose. The incompletely hydrolyzed β-lactose in breast milk reaches the lower small intestine.

4. Illustrative material:

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

5. Literature: see Appendix No. 1.

6. Control questions (feedback)

1. What are the disorders of digestion and absorption of carbohydrates ?
2. What are the causes of hyperglycemia and ?
3 . What are the types of diabetes?
4 . What are the causes of hypoglycemia and ?
5. What are the features of carbohydrate metabolism disorders in children?

Lecture No. 9

1. Topic: Violations of fat metabolism.

2. Purpose: to explain the etiology and pathogenesis of the main disorders of fat metabolism .

3. Abstracts of the lecture

Violations of fat metabolism in the body are observed in the following cases:

- with violations of the breakdown and absorption of fats in the intestine;
- with violations of the transport of fats and their transition from the blood to the tissue;
- in violation of the intermediate metabolism of fats;
- with violations of the metabolism of fats in adipose tissue.

Hyperlipemia is considered one of the indicators of impaired fat transport. In this case, the concentration of fats in the blood exceeds 2 mmol / l.

Hyperlipemia is: alimentary (food), transport, retention.

If the fats coming from the blood do not break down and are not oxidized in the cells, they accumulate in the cells after being stored for a long time. This condition is called fatty infiltration. If at the same time there is a violation of the structure of the cytoplasm , then this is called fatty degeneration .

Obesity is a pathological condition manifested by a significant accumulation of triglycerides in the body. It is primary, symptomatic (secondary), hypertrophic, hyperplastic, android, gynoid and mixed.

Primary obesity is a pathological condition manifested by an increase in the point of lipostasis due to a violation of hormonal connections between adipose tissue and the hypothalamus.

Symptomatic (secondary) obesity is a syndrome that develops as a result of pathological disorders that lead to a decrease in energy expenditure and the accumulation of fats in the body.

Hypertrophic obesity is manifested by an increase in the volume of fat droplets in fat cells with an unchanged total number of fat cells.

Hyperplastic obesity is manifested by an increase in the total number of fat cells.

Android obesity is the accumulation of fat in the upper body, on the abdomen. This obesity is typical for men.

Gynoid obesity is the accumulation of fat in the thighs, buttocks, and lower torso. This obesity is typical for women.

Mixed obesity - occurs when a combination of android and gynoid types.

Fat metabolism in children also differs in some age characteristics. Fats are certainly an essential component of the children's diet. When fat breaks down, 2 times more energy is released than when an equal amount of proteins and carbohydrates break down.

4. Illustrative material:

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

5. Literature: see Appendix No. 1.

6. Control questions (feedback)

1. In what cases are lipid metabolism disorders observed?
2. What are the types of hyperlipemia?
3. How does fatty infiltration develop ?
4. What is obesity?
5. What are the features of fat metabolism disorders in children?

Lecture No. 10

1. Topic: Hypoxia.

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

3. Abstracts of the lecture

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

Classification of hypoxic conditions

1. Exogenous:
A) hypobaric;
B) normobaric.
2. Respiratory (respiratory).
3. Circulatory (cardiovascular).
4. Hemic (blood).
5. Tissue (primary tissue).
6. Mixed.

According to the criteria for the prevalence of a hypoxic state, there are: a) local; b) general hypoxia.

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

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

Hypobaric hypoxia develops with a decrease in atmospheric pressure. Most often it is observed during high-mountain ascents. The leading pathogenetic factor of its occurrence is also hypoxemia, but in contrast to normobaric hypoxia, hypocapnia serves as an additional negative factor. Normobaric hypoxia occurs when, at normal atmospheric pressure, the oxygen content in the inhaled air falls. A similar situation can arise during a long stay in unventilated spaces of small volume, when working in wells, mines.

Respiratory hypoxia occurs as a result of insufficient gas exchange in the lungs due to alveolar hypoventilation, impaired ventilation-perfusion relations, excessive extra- and intracellular shunting of venous blood or with difficulty in the diffusion of oxygen in the lungs. The pathogenetic basis of respiratory hypoxia, as well as exogenous, is also arterial hypoxemia, in most cases combined with hypercapnia.

Circulatory develops with 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 by 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 as a result of a violation of the ability of cells to absorb oxygen.

Mixed hypoxia is a combination of 2 or more of its main 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 .

4. Illustrative material:

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

5. Literature: see Appendix No. 1.

6. Control questions (feedback)

1. What is hypoxia?
2. What are the types of hypoxia ?
3 . What is the emergency adaptation of the body to hypoxia?
4 . What is the long-term adaptation of the body to hypoxia?
5. What are the features of the development of hypoxic conditions in children ?

Lecture No. 11

1. Topic: Peripheral circulatory disorders.

2. Purpose: to explain the main causes and mechanisms of development of peripheral circulatory disorders.

3. Abstracts of the lecture

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

Arterial hyperemia is a blood supply 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;
- an increase in the volume of organs and tissues;
- an increase in local temperature.

Arterial hyperemia is physiological and pathological. Physiological arterial hyperemia is observed with an increase in the functioning of organs, overheating, massage, emotional overstrain .

Pathological arterial hyperemia develops under various conditions (inflammation, allergies, burns, fever, trauma, neuralgia, etc.).

Ischemia is a violation of peripheral circulation, which 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 (compression ischemia); blockage of the lumen of the artery by a thrombus, embolus, foreign body (obstructive ischemia); spasm of the artery (angiospastic ischemia).

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

Venous hyperemia is a blood supply 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);
- an 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. Control questions (feedback)
1. What disorders of the forms of peripheral circulation are there?
2. What causes arterial hyperemia ?
3. What are the mechanisms of ischemia ?
4. What are the symptoms of venous congestion?
5. What are the features of peripheral circulation disorders in children?

Lecture No. 12

1. Topic: Inflammation.

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

3. Abstracts of the lecture

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

Microorganisms (bacteria, viruses, fungi) belong to ex o genes ; animal organisms (protozoa, worms, insects); chemicals (acids, alkalis); mechanical influences (foreign body, pressure) ; thermal effects (cold, heat); radiation energy (X-ray, radioactive, ultraviolet rays).

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

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:
1 phase - vasospasm;
2 phase - arterial hyperemia;
3 phase - venous hyperemia;
4 phase - stasis.

Mediators of inflammation:
a) mediators of humoral origin (kinins, complement system);
b) mediators of cellular origin, ready-made or pre-existing (mediators of mast cells, serotonin, heparin, lysosomal enzymes);
c) mediators of cellular origin, newly formed (eicosanoids, lymphokines, monokines, free radicals).

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

Exudation - exudation of the protein-containing liquid part of the blood through the vascular wall into the inflamed tissue. The fluid that comes out of 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 mechanism of development, the process of exudation is associated with the influence of inflammatory mediators. The leading factor in exudation is an increase in vascular permeability.

Emigration - the release of leukocytes outside the vessels . Polymorphonuclear leukocytes are the first to be found in the focus of inflammation. The main function of leukocytes in the focus of inflammation is the absorption of foreign bodies (phagocytosis).

Proliferation is the 3rd stage of inflammation. Leukocytes die after several hours of phagocytic function. Macrophages clear the focus of inflammation from microorganisms. Dead cells secrete substances that stimulate proliferation.

A feature of the inflammatory process in children is the tendency to generate it. Children usually develop necrosis, common 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. Control questions (feedback)
1. What is inflammation ?
2. What factors cause inflammation?
3. What are the stages of inflammation?
4. What vascular changes occur in the focus of inflammation?
5. What are the features of the course of the inflammatory process in children?

Lecture No. 13

1. Topic: Fever.

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

3. Abstracts of the lecture

Fever is a typical pathological process characterized by both damaging and protective-adaptive reactions of the body. At the same time, a change in the activity of the thermoregulation

center under the influence of pyrogens leads to an increase in body temperature. With fever, the mechanisms of thermoregulation are not violated, but rise to a higher level.

According to the causes of fever, it is divided into infectious and non-infectious.

Infectious fever develops under the action of bacteria, viruses, protozoa. Non-infectious fever occurs under the influence of external and internal factors leading to tissue damage. These include: burn, wound, heart attack, blood transfusion, internal hemorrhage, allergies, tumors, cirrhosis, etc.

Pyrogenic substances have a huge place in the mechanism of development of fever. They are divided into exogenous and endogenous (leukocyte). Exogenous pyrogens are substances formed as a result of vital activity or decay of microbes.

Fever occurs in 3 stages:

1. stage of temperature increase (stadium incrementum);
2. stage of standing temperature at a high level (stadium decrementum);
3. stage of lowering the temperature .

There are the following types of fever:

1. subfebrile fever - an increase in body temperature up to 38 ^{0}C;
2. moderate fever - an increase in body temperature up to 38-39 ^{0}C;
3. high fever - an increase in body temperature up to 39-40 ^{0}C;
4. hyperpyretic fever - an increase in body temperature above 41 ^{0}

In newborns with fever, the level of production heat, without participation trembling mechanism, can increase by 100-200% or more compared to the state of rest.

4. Illustrative material:

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

5. Literature: see Appendix No. 1.

6. Control questions (feedback)

1. What is a fever ?
2. What factors cause fever?
3. What are the stages of fever ?
4. What are the types of fever?
5. What are the features of the course of fever in children?

Lecture No. 14

1. Topic: Allergy.

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. Abstracts of the lecture

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.

Allergen classification

Allergens are exogenous and endogenous. Exogenous allergens enter the body from the environment, 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 are household (house dust , cosmetics) , epidermal (wool, fluff and animal hair) , vegetable (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

Regarding the classification of allergic reactions, there are several views. R. Cook (1930) divided all allergic reactions into 2 types: immediate-type allergic reactions and delayed-type allergic reactions. Allergic reactions of the immediate type are observed a few minutes after the re-entry of the allergen into the body.

Allergic reactions of the delayed type are observed after 24-48 hours
after re-entry of the allergen into the body.

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

- allergic reactions of reaginic 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 immunocomplex type - type III (serum sickness);
- allergic reactions of 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 the specific functions of organs and systems occur: increased blood pressure, increased permeability of the vascular wall, edema, bronchospasm.

Allergic reactions type I (reaginic)

In the immune stage, T cells under the influence of allergens and macrophages, producing interleukin-4, stimulate B cells. After that, they turn into plasma cells and produce IgE. IgE attaches to mastocytes or basophils in the blood. When exposed to the allergen again, it binds to IgE. This is followed by the release of intracellular granules (degranulation). In the 2nd stage of allergic reactions, allergens interact with antibodies. This will lead to the release of mediators. In allergic reactions of the immediate type, 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 can develop. These include microcirculation disorders (expansion of capillaries, increased permeability, changes in the rheological properties of blood), bronchospasm, an increase in glucocorticoids, changes at different levels of the nervous system in the processes of excitation and inhibition.

II allergic reactions (cytotoxic)

In the stage of immune reactions, the autoallergen is recognized 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 self-allergens. Then the stage of pathochemical changes develops, allergy mediators are formed. These mediators include complement components, lysosomal enzymes, free oxygen radicals. In the stage of pathophysiological disorders, the destruction of cells that have allergens is observed. According to the II cytotoxic type in
allergic reactions develop hemolytic anemia, thrombocytopenia, autoimmune thyroiditis, myocarditis, hepatitis and other autoimmune diseases.

Allergic reactions type III (immunocomplex)

Allergens are medicines, therapeutic serums, food products, fungi, 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 is with a slight excess of antigen, then 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 the walls of blood vessels. Immune complexes, attaching to platelets, destroy them.

IV allergic reactions (cell-mediated)

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

In connection with 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. Control questions (feedback)

1. What is an allergy ?
2. What factors cause allergies ?
3. How can allergic reactions be classified ?
4. What is the general pathogenesis of allergic reactions ?
5. What are the features of the development of allergic reactions in children?

Lecture No. 15

1. Topic: Tumors.

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

3. Abstracts of the lecture

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

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

Benign tumors grow pushing back, squeezing surrounding tissues. Such growth is called expansive. Benign tumors, depending on the location of the tissue, are called by attaching the ending "oma" to the name of this tissue. For example, fibroma, osteoma, adenoma, melanoma, neuroma, angioma, etc.

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

Causes of carcinogenesis

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

Tumor pathogenesis

There are the following stages of carcinogenesis: Stage 1 - the transformation of a healthy cell into a tumor cell, is called initiation. Stage 2 - the rapid development and reproduction of tumor

cells and the formation of primary tumor nodes, is called promotion. Together, initiation and promotion are
transformation. Stage 3 - strengthening of persistent qualitative changes and malignant properties of tumor cells, or an increase in tumor growth,
called a progression.

Transformation is the transformation of a normal cell into a tumor cell under the action of carcinogens. The primary mechanisms of transformation of a normal cell into a tumor cell are still unknown.

Cells of malignant tumors for a long time after transformation can be in a latent (hidden) period, not showing active growth processes.

Transformed cells under the influence of one additional factor can go to the 2nd stage of carcinogenesis - promotion. In organs with transformed cells, mechanical influences, the influence of inflammation 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 - an increase in the malignant properties of a tumor in tumor cells due to their reproduction.

The relationship of tumors with the body

During the development of the tumor, the work of the whole organism is disrupted. This is due to the peculiarities of metabolic processes in tumor cells. Due to the decrease in immunity, resistance against infectious diseases decreases. So, if death does not occur as a result of significant damage, it can occur as a result of sepsis. During a tumor, a lot of energy is expended. And under these conditions, incompletely oxidized products accumulate, and metabolic acidosis occurs. The permeability of cell membranes increases, a violation of the structure of cells is observed.

Tumors in children are the result 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. Control questions (feedback)

1. What is about swelling ?
2. What are the types of tumors ?
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

In Russian

main:

1. Ado A.D. Pathophysiology: a textbook: in 2 volumes - Evero, 2015 .-- T. 1.
2. Ado A.D. Pathophysiology: a textbook: in 2 volumes - Evero, 2015 . - T. 2.
3. Pathophysiology. Guide to practical exercises: a training manual. - M.: GEOTAR-Media, 2014.
4. Fr. about fishing VA et al. General pathological physiology: a textbook. - M., 2013.
5. Pathophysiology. In 2 vol. T. 2: textbook / ed. V.V. Novitsky, E. D. Goldberg, O. I. Urazova ; M-education of the science of the Russian Federation. - 4th ed., Revised . and additional ; Rec. GOU VPO "MMA named after I. Sechenov." - M.: GEOTAR - Media, 2012 .-- 640 p. + email Optical disk (CD-ROM): ill.

additional:

1. Pathophysiology. Guide to practical exercises: a training manual / Ed. V.V. Novitsky, O.I. Urazova . - M .: GEOTAR-Media, 2011.
2. Pathophysiology: a textbook: in 2 volumes / Ed. V.V. Novitsky, E.D. Goldberg, O.V. Urazova . - 4th ed., Revised . and add. - M.: GEOTAR-Media, 2010 .-- T. 1.
3. Pathophysiology: a textbook: in 2 volumes / Ed. V.V. Novitsky, E.D. Goldberg, O.V. Urazova . - 4th ed., Revised . and add. - M.: GEOTAR-Media, 2010 .-- T. 2.
4. Litvitsky P.F. Pathophysiology: a textbook. - 4 th ed. Ispra . and add. - M.: GEOTAR-Media, 2010.
5. Pathophysiology: manual for practical training: textbook. allowance / ed. V.V. Novitsky, O. I. Urazova . - M.: GEOTAR - Media, 2011 .-- 336 s
6. Pathophysiology. Tasks and test tasks: textbook.-methodical manual / ed. PF Litvitskogo ; M-education and science of the Russian Federation. -; Rec. GOU VPO "MMA named after I. Sechenov." - M.: GEOTAR - Media, 2013 .-- 384 p. : silt

electronic resources :

1. Pathophysiology [Electronic resource] : textbook: in 2 volumes / Ed. V.V. Novitsky, E.D. Goldberg, O.V. Urazova . - 4th ed., Revised . and add. - Electronic textual data. (59.9 Mb). - M.: GEOTAR-Media, 2010. - T. 1, T. 2. - El. opt. disk (CD-ROM).
2. Pathological physiology [Electronic resource]: textbook / ed. N. N. Zaiko. - 2nd ed. - Electronic textual data. (20.5 Mb). - M.: Medpress-inform , 2004. - 1 email. Optical disk (CD-ROM).
3. Medicine. 3 course [Electronic resource]: lecture course. - Electronic textual data. (24.0 Mb). - M.: Publishing House "Equilibrium", 2005. - 1 email. Optical disk (CD-ROM).
4. Spiders, V. S. Pathological anatomy and pathological physiology [Electronic resource]: textbook. for colleges. - The electron. text data (44 , 7 Mb). - M.: Publishing House group "GEOTAR-Media", 2010. - 256 p. email optical disc (CD-ROM)

In Kazakh

main:

1. Nұrmұhambetuly Ә. Pathophysiology-1. Clinicals қ practice өte maңyzdy birtektes dertik үrdisterdің pathogenesis me mendeu zholdaryna nұsқama. Volume 1: Oқy Urals. - Almaty: Evero, 2016.
2. Nұrmұhambetuly Ә. Pathophysiology-1. Clinicals қ practice өte maңyzdy birtektes dertik үrdisterdің pathogenesis me mendeu zholdaryna nұsқama. Volume 2: Oқy құrylы. - Almaty: Evero, 2016.

3. Nұrmұhambetuly Ә. Pathophysiology: Ouly. - 4 bass. - Evero, 2015 .-- T. 1.
4. Nұrmұhambetuly Ә. Pathophysiology: Ouly. - 4 bass. - Evero, 2015 .-- T. 2.
5. Nұrmұhambetuly Ә. Pathophysiology: Ouly. - 4 bass. - Evero, 2015 .-- T. 3.
6. Nұrmұhambetuly Ә. Pathophysiology: Ouly. - 4 bass. - Evero, 2015 .-- T. 4.
7. Ado A.D. Pathophysiology: Ouly. 1 volume - Evero, 2015.
8. Pathophysiology. 2 tomdyқ. 1 t .: oқulyқ / қazaқ tіl. aud. B. A. Zhetpisbaev ; ed. V.V. Novitsky. - M.: GEOTAR - Media, 2017. - 640 bet.s .
9. Pathophysiology. Eki tomdyқ. 2 t.: Oқulyқ / қazaқ tіl. aud. S. B. Zhutikova ; ed. V.V. Novitsky. - M.: GEOTAR - Media, 2018 .-- 464 b. + email Optical disk (CD-ROM).
8. Ado A.D. Pathophysiology: Ouly. 2 volume - Evero, 2015.
9. Pathology physiology. Tәzhiribelіk sabaқtarға nұsқau: oқu құralы. - M.: GEOTAR-Media, 2014.
10 . Urazalina N.M. Tapsyrmalary test. I-bөlim. Biriktes dertik үrdister. –Almaty: Evero, 2014.
11. Urazalina N.M. Tapsyrmalary test. II-бөlім. Biriktes dertik үrdister. –Almaty: Evero, 2014.
1 2. Urazalina N.M. Tapsyrmalary test. III-бөlім. Biriktes dertik үrdister. –Almaty: Evero, 2014.
13. Urazalina N.M. Tapsyrmalary test. IV-бөlім. Biriktes dertik үrdister. –Almaty: Evero, 2014.
14. Zhәutikova S.B. Mamandyrylғan pathology physiology courses: Oқu-әdіstemelіk құralы. - Karagandy: LCD "Ak Nur", 2013.
15. Zhәutikova S.B. Pathologies қ physiology ә ә і ы ы ситуация ситуация ситуация ситуация еп еп еп еп:::: лы лы лы лы лы лы лы лы лы лы: құ құ еп құ і лы лы лы құ. - Karagandy: LCD "Ak Nur", 2013.

additional:

1. Nұrmұhambetuly, Ә. Pathophysiology-1. Clinicals қ practice өte maңyzdy birtektes dertik үrdisterdің pathogenesis me mendeu zholdaryna nұsқama. Volume 2: Oқu құrylы. - Almaty: Evero, 2016 .-- 248 b.
2. Pathologies қ physiology. Tәzhiribelіk sabaғtarға nқsқau [Mәtіn]: oқu құrylы = Pathophysiology. Guide to practical exercises: textbook / V.V. Novitsky [f. b.]; ed. bass . V.V. Novitsky, O. I. Urazova; Kaz . tiline aud. S. B. Zhutikova . - M.: GEOTAR - Media, 2014. - 768 bet.s .
3. Urazalina , N. M. Test tapsyrmalary . II- Бөlім . Birtektes dertik үrdister : - Almaty: Evero , 2014. - 184 bet.
4.Zhәutikova, S. B. Mamandandyrylғan pathology physiology courses: Oқu-әdіstemelіk құralы. - Karagandy: LCD "Ak Nur", 2013
5.Zhәutikova, S. B. Pathology қ physiology of the бой boy’s situation қ ester ғ ғ ғ қ: қu-әdіstemelіk құral. - Karagandy: LCD "Ak Nur", 2013
6. Nұrmұhambetuly Ә. Ұrуқ damuy men balalық shaқ aurularynң pathophysiology: monograph - A., 2006

electronic resources:

1. Pathologiesқ physiology: Pathophysiology: tәzhiribelik sabaқtarға nұsқau: oқu құraly / V.V. Novitskydin ed. - M.: GEOTAR-Media, 2014.
2. Pathology: oқulyқ: 2 vols. [Electronic resource] / M.A. Pal'tsev, V. S. Paukov revised by son Basқarғandar: M.: GEOTAR.- Media. 2015
3. Pathologists қ physiology. Tәzhiribelіk sabaқtarға nұsқau [Electronic resource]: oқu құralы = Pathophysiology. Guide to practical exercises: a training manual / қaz.tіlіne Aud . S. B. Zhutikova ; ed. V.V. Novitsky. - Electronic textual data. (63.4 Mb). - M.: GEOTAR - Media, 2014. - 768 bet.el . opt. Disk

In English

main :

1. Pathophysiology: Concepts of Altered Health States / Porth CM, Matfin G .; Lippincott Williams & Wilkins / Wolters Kluwer, 2013.
2.Pathophysiology. Volume 1 .: the book for medical institutes / AD Ado [and others]. - Almaty: "Evero", 2017. - 216 p.
3.Pathophysiology. Volume 2 .: the book for medical institutes / AD Ado [fnd others]. - Almaty: "Evero", 2017. - 188 p.
4.Pathophysiology. Volume 3 .: the book for medical instutes / AD Ado [and others]. - Almaty: "Evero", 2017. - 328 p
5.Zhautikova, SB Review of pathophysiology: educational-methodical manual / SB Zhautikova, U. Faroog. - Karaganda: АҚҺҰР, 2017 .-- 388 p.
6.Zhautikova, SB Collection of situational problems for discipline of pathological physiology-2: educational-methodical manual / SB Zhautikova, U. Faroog. - Karaganda: АҚҺҰР, 2017 .-- 126 p

additional:

1. Pathophysiology / Copstead LC, Banasik JL; Saunders / Elseviere , 2012.

E-sources

№	Title	Link
1.	«BooksMed»	http://www.booksmed.com
2.	«Web of science» (Thomson Reuters)	http://apps.webofknowledge.com
3.	«Science Direct» (Elsevier)	https://www.sciencedirect.com
4.	«Scopus» (Elsevier)	www.scopus.com
5.	PubMed	https://www.ncbi.nlm.nih.gov/pubmed