LECTURE COMPLEX

Discipline: Pathological physiology of organs and systems

Discipline code: POS 3202-2

Name of EP: 6B130100-"General Medicine"

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

Course and semester of study: III course, VI semester

Lecture volume: 15 hours

Shymkent, 2022

The lecture complex was developed in accordance with the working program of the discipline (syllabus) EP 6B130100 - "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: Pathophysiology of the nervous system. General etiopathogenesis of disorders of the nervous system

2. Purpose: Mastering the issues of general etiology and pathogenesis of dysfunction of the nervous system.

3. Abstracts of the lecture:

Etiology of dysfunction of the nervous system

Exogenous factors:	Endogenous factors :
• Physical - oxygen deficiency - ionizing radiation - electricity - vibration - mechanical injury - high/low temperature -noise • Chemical - neurotropic poisons (lead, vanadium, curare, drugs, ethyl and methyl alcohols, drugs) • Biological microbes (causative agents of tetanus, botulism, meningococci), - viruses (rabies, polio, influenza) • Social - deficiency of protein, vitamins - psychogenic	• Congenital : - hereditary metabolic disorders (type II glycogenosis, lipidosis, phenylketonuria) - hereditary diseases (Down's disease) - malformations, birth injuries • Acquired: - cerebral ischemia - hypoglycemia - acid-base balance disorders • electrolyte disturbances - damage to neurons in renal, hepatic insufficiency. tumors, etc. - pathological processes

Defense mechanisms of the nervous system

- membranes of the brain surrounding the neuron glial and Schwann cells
- microglia (system of mononuclear phagocytes)
- blood-brain barrier
- antisystems (antinociceptive, antiepileptic)
- a high degree of reliability of the functioning of nerve formations.

Features of the action of etiological factors

- the stronger and longer the exposure, the greater its effect
- Weak, but long-term and permanent factors can have a damaging effect at a lower total dose than with a single exposure (Behring effect)
- the degree of dysfunction depends not only on the number of damaged neurons, but also on the formation of an inhibition zone, which increases the functional defect

Routes of entry of pathogenic agents into the nervous system

- across the blood-brain barrier
- along the nerve trunks.

Neuronal damage

- damage to the neuron body
- damage to axons.

- damage to dendrites.
- dysfunction of the synaptic apparatus

In the pathogenesis of neuronal damage are important:

- damage to membranes and enzyme systems,
- disruption of power supply
- ion imbalance
- cell dysregulation

SYNAPTIC APPARATUS

mediator synthesis→ mediator transport to the synaptic cleft →deposition in the presynaptic region→ release into the synaptic cleft→ interaction with receptors→ breakdown of neurotransmitters, reuptake.

Damage to the synaptic apparatus

1. violation of mediator synthesis
2. violation of mediator transport
3. violation of mediator deposition in nerve endings.
4. violation of the secretion of the neurotransmitter into the synaptic cleft
5. violation of the interaction of the mediator with the receptor
6. violation of the reuptake of the neurotransmitter from the synaptic cleft.

In childhood, the pathology of the nervous system has its own characteristics. This is due to the immaturity of the structures of the nervous system at birth and is especially pronounced in children of the first year of life.

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 of diseases of the nervous system?
2. What is the pathogenesis of diseases of the nervous system (neurons, axons, dendrites and damage to the synaptic apparatus)?
3. What are the routes of entry of pathogenic agents into the nervous system?
4. What are the defense mechanisms of the nervous system?

Lecture #2

1. Topic: Pathophysiology of external respiration.

2. Purpose: Mastering the issues of etiology and pathogenesis of external respiration, restrictive form of hypoventilation, impaired blood flow in the lungs. Understanding the pathogenesis of respiratory failure.

3. Lecture abstracts :

External respiration is a set of processes that take place in the lungs and ensure the normal gas composition of arterial blood. The normal gas composition of arterial blood is maintained by the following interrelated processes: 1) ventilation of the lungs;
2) diffusion of gases through the alveolar-capillary membranes;
3) blood flow in the lungs;
4) regulatory mechanisms.

Impaired lung ventilation

Indicators characterizing the state of lung ventilation can be divided into:

1) for static lung volumes and capacities - vital capacity (VC), respiratory volume (DO), residual lung volume (ROL), total lung capacity (TLC), functional residual capacity (FRC), inspiratory reserve volume (RO), expiratory reserve volume (RO $_{vyd}$) (Fig. 16-1);
2) dynamic volumes, reflecting the change in lung volume per unit time - forced vital capacity (FVC), Tiffno index, maximum lung ventilation (MVL), etc.

Alveolar hypoventilation is a decrease in alveolar ventilation per unit of time below that required by the body under given conditions.

There are the following types of alveolar hypoventilation:
1) obstructive;
2) restrictive, which includes two variants of the causes of its development - intrapulmonary and extrapulmonary;
3) hypoventilation due to dysregulation of breathing.

Obstructive (from Latin obstructio - barrier, hindrance) type of alveolar hypoventilation. This type of alveolar hypoventilation is associated with a decrease in the patency (obstruction) of the airways. In this case, the obstruction to the movement of air can be both in the upper and lower respiratory tract.

Lack of external respiration

Insufficiency of external respiration is such a state of external respiration in which the normal gas composition of arterial blood is not provided or this is achieved by the voltage of the apparatus external respiration, which is accompanied by a limitation of the reserve capacity of the body.

Lecture #3

1. Topic: Pathophysiology of external respiration.

2. Purpose: . Understanding the pathogenesis of respiratory failure.

3. Lecture abstracts :

Classifications of insufficiency of external respiration are divided :

one. According to the localization of the pathological process , respiratory failure with a predominance of pulmonary disorders and respiratory failure with a predominance of extrapulmonary disorders are distinguished.

- Respiratory failure with a predominance of pulmonary disorders can lead to:
• airway obstruction;
• impaired extensibility of lung tissue;
• decrease in the volume of lung tissue;
• thickening of the alveolar-capillary membrane;
• violation of pulmonary perfusion.
- Respiratory failure with a predominance of extrapulmonary disorders lead to:
• violation of neuromuscular impulse transmission;
• thoracophrenic disorders;
• disorders of the circulatory system;
• anemia, etc.

2. According to the etiology of respiratory disorders, the following types of respiratory failure are distinguished:
• centrogenous (in violation of the function of the respiratory center);
• neuromuscular (in violation of the function of the neuromuscular respiratory apparatus);
• thoracodiaphragmatic (in violation of the mobility of the musculoskeletal frame of the chest);
• bronchopulmonary (with damage to the bronchi and respiratory structures of the lungs).

3. According to the type of violation of the mechanics of breathing , there are:
• obstructive respiratory failure;
• restrictive respiratory failure;
• mixed respiratory failure.

4. According to the pathogenesis , the following forms of respiratory failure are distinguished:
• hypoxemic (parenchymal) ;
• hypercapnic (ventilation) ;
• mixed form ;

5. **Insufficiency of external respiration according to the pace of development :**

1. Acute insufficiency of external respiration develops within minutes, hours. It requires urgent diagnosis and emergency care. Its main symptoms are progressive dyspnea and cyanosis.

2. Subacute insufficiency of external respiration develops within a day, a week and can be considered on the example of hydrothorax - accumulation of fluid of various nature in the pleural cavity.

3. Chronic insufficiency of external respiration develops over months and years. It is a consequence of long-term pathological processes in the lungs, leading to dysfunction of the apparatus of external respiration and circulation in the pulmonary circulation (for example, in chronic obstructive pulmonary emphysema, disseminated pulmonary fibrosis).

Clinically, there are **three degrees of chronic respiratory failure:**

1st degree - the inclusion of compensatory mechanisms and the occurrence of shortness of breath only under conditions of increased load. The patient performs the full volume of only everyday activities.

2nd degree - the occurrence of shortness of breath with little physical exertion. The patient performs daily activities with difficulty. Hypoxemia may not be (due to compensatory hyperventilation). Lung volumes have deviations from proper values.

3rd degree - shortness of breath is expressed even at rest. The ability to perform even minor loads is sharply reduced. The patient has severe hypoxemia and tissue hypoxia.

In children, respiratory failure develops more rapidly. This is due to the underdevelopment of the respiratory, cardiovascular and immune 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 are the types of respiratory failure according to pathogenesis?
2. What are the types of respiratory failure based on changes in blood gas composition?
3. What are the causes of type I respiratory failure?
4. What are the causes of type II respiratory failure?

Lecture #4

1. Topic: Violations of vascular tone.

2. Purpose: Understanding the issues of pathophysiological mechanisms of occurrence and development of arterial hypertension .

3. Abstracts of the lecture :

Arterial hypertension is a persistent increase in blood pressure over 140/90 mm Hg.

Arterial hypertension (AH)

Primary Secondary,
or essential symptomatic (renal, neurogenic,
(90-95%) endocrine, etc.) - (5-10%)

AD \u003d IOC x OPSS

An increase in cardiac output (MOC) occurs:

- With an increase in the tone of the sympathetic nervous system, the neurotransmitters of which have positive chrono- and inotropic effects on the heart.
- With an increase in the volume of circulating blood (BCC)

- violation of the excretory function of the kidneys;
- hypernatremia;
- true hypervolemia;
- excessive secretion of mineralocorticoids.

To increase the total peripheral vascular resistance (OPSS) lead to:

- excitation of alpha-adrenergic receptors of arterial vessels by catecholamines;
- excessive formation of vasoconstrictor angiotensin II under the influence of renin and angiotensin-converting enzyme (ACE);
- hyperplasia and hypertrophy of myocytes of the vascular wall;
- violations of endothelium-dependent mechanisms of autoregulation of vascular tone. An increase in the production of vasoconstrictive endothelins and a decrease in the secretion of the vasodilator factor, nitroxide (NO).

Experimental models of arterial hypertension
(symptomatic arterial hypertension)

1. Central ischemic hypertension

ligation of the arteries feeding the brain (vertebral, carotid) or the introduction of kaolin into the large tank of the brain of animals and the violation of the outflow of cerebrospinal fluid →cerebral ischemia →dysregulation of vascular tone→ HELL.

2. Model of neurosis.

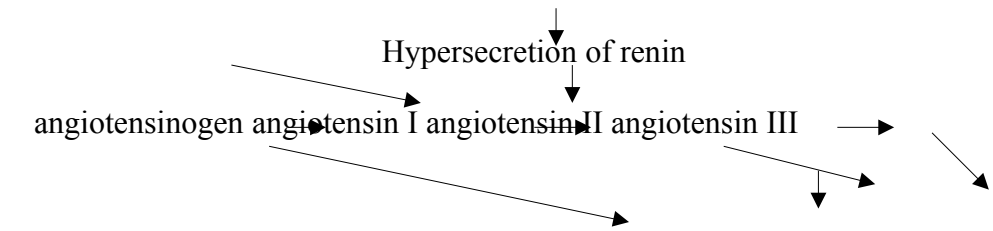
Neurosis is caused in highly organized animals by "collision", i.e. collision of excitatory and inhibitory processes in the cerebral cortex.

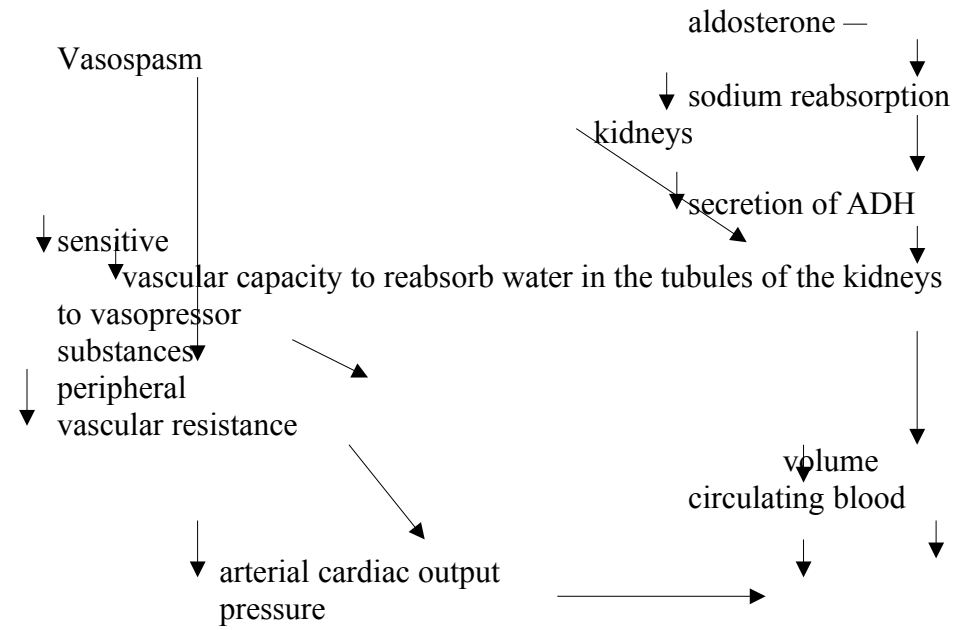
1. Reflexogenic hypertension "hypertension of disinhibition".

Bilateral transection of Zion and Ludwig depressor nerves in rabbits and dogs
and the carotid sinus nerve of Goering (Heimans model, 1931-1937; N.N. Goreva, 1939) → absence of afferent impulses along the depressor nerves to the cardiovasomotor pressor center →; a persistent increase in the tone of the cardiovasomotor center →; a persistent increase in blood pressure.

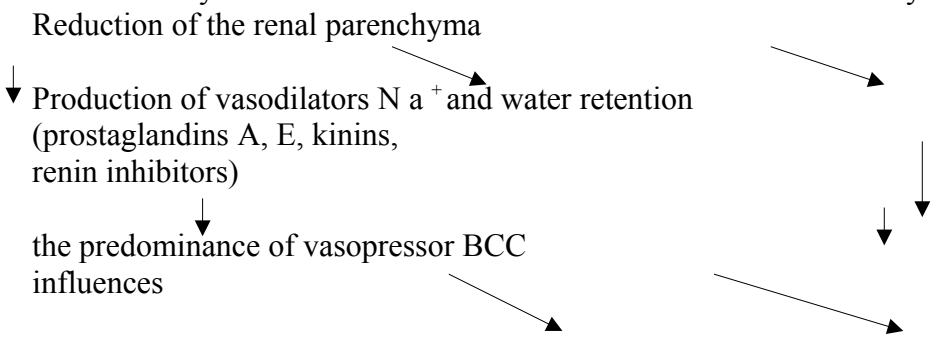
2. Renal arterial hypertension.
   - ✓ Vasorenal (renovascular) Goldblatt's experimental model, 1934: application of narrowing rings on both renal arteries

Renal ischemia





✓ Renoprival hypertension (lat. ren - kidney, privo - reduce) develops as a result of destructive lesions of the kidney parenchyma. Experimental model of Grolman, 1949 (removal of both kidneys with connection of the animal to the "artificial kidney" apparatus)



3. Endocrine hypertension

Experimental model: administration of mineralocorticoids + sodium chloride solution to animals.

Endocrine symptomatic hypertension occurs with an increase in the secretion of glucocorticoids (Itsenko-Cushing's disease and syndrome), aldosterone (Conn's syndrome, secondary aldosteronism), growth hormone (acromegaly, gigantism), adrenaline (pheochromocytoma), thyroxin (Based's disease, thyrotoxicosis), vasopressin (inadequate products of ADH).

Etiology and pathogenesis of essential hypertension (EG)

Essential hypertension is a disease in which an increase in blood pressure is not associated with primary organic damage to organs and systems. The basis of the disease is a violation of the neurohumoral regulation of vascular tone.

Essential hypertension is a polygenic disease, which is based on mutations in various genes involved in the regulation of vascular tone. Hereditary predisposition is realized under the influence of environmental factors.

Risk factors:

- Genetic factors (hereditary predisposition)

✓ High concordance in identical twins
✓ Increased risk of developing hypertension (6 times) in families where one of the parents suffers from hypertension
✓ Possibility of developing hypertension at a young age
✓ Creation of strained rats predisposed to AH (SHR – spontaneously hypertensive rats)
✓ Transmembrane ion transport defects (Ca^{++})
↑Ca^{++} in myocytes of resistive vessels → persistent muscle contraction
✓ Defects in transmembrane transport of ions in the renal tubules → sodium and water retention
✓ Violations of endothelium-dependent mechanisms of regulation of resistance vascular tone, leading to a decrease in the formation of endothelial vasodilators (nitroxide, prostacyclin, etc.) by endotheliocytes
✓ Structural features of the angiotensinogen gene
✓ Defects in aldosterone metabolism enzymes
• Prolonged exposure to stressors
✓ Hypertension is more likely to develop in people after emotional upheavals
✓ Hypertension occurs more often in people whose professional activities are associated with psycho-emotional overload
• Excess consumption (over 5 g per day) of table salt
• Smoking, alcohol, hypokinesia, noise, vibration, night work
• Obesity, vascular atherosclerosis, endocrine diseases

Pathogenesis

1. Neurogenic mechanism

Risk factors → Disorders of blood pressure regulation centers → Disturbances in the dynamics of cortical processes (excitation and inhibition) → Inhibition deficit in the cerebral cortex → Formation of a generator of pathologically enhanced excitation (GPUV) in the neurons of the SDC and the formation of a pathological system → Stable activation of the sympathetic division of the autonomic nervous system → Increase frequency and strength of heart contractions, spasm of resistive vessels → ↑ IOC and OPSS

2. Hormonal mechanism

Activation of the hypothalamic-pituitary-adrenal system → Increased secretion of glucocorticoids and other stress hormones.

4. renal mechanism.

✓ Hereditary defects of the renin-angiotensin-aldosterone system (RAAS) and tubular epithelium
✓ RAAS activation due to renal ischemia

Arterial hypertension in childhood develops extremely rarely and in most cases has a secondary symptomatic character. Its development is based on a combination of factors of hereditary predisposition and unfavorable environmental factors.

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 arterial hypertension, what is the concept, types?

2. What are the experimental studies of the pathogenesis of arterial hypertension? What is secondary (symptomatic) arterial hypertension, concept?
3. What are the risk factors for primary arterial hypertension?
4. What is the pathogenesis of primary arterial hypertension?

Lecture #5

1. Topic : Coronary insufficiency. Heart rhythm disturbances.

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. Lecture abstracts :

Coronary insufficiency is a mismatch between myocardial oxygen demand and metabolic substrates and their delivery through the coronary arteries.

Types by pathogenesis:
- Absolute (reduction of the lumen or complete closure of the coronary arteries)
- Relative (increased myocardial demand for oxygen and metabolic substrates)

Causes of absolute coronary insufficiency :
- atherosclerosis of the walls of the coronary arteries
- aggregation of blood cells and thrombosis of coronary arteries
- prolonged spasm of the coronary arteries (catecholamines, prostaglandins F 2 α, thromboxane A2)

Causes of relative coronary insufficiency
- Increase in blood and myocardial catecholamines

- consumption of oxygen and metabolic substrates due to positive chrono- and inotropic effects ↓of the efficiency of energy production (LPO, uncoupling of oxidation and phosphorylation)
- A significant increase in the work of the heart :

- excessive exercise
- prolonged tachycardia
- hypervolemia

Types of coronary insufficiency depending on the degree and reversibility of myocardial damage:
✓ Reversible (transient disorders of coronary blood flow, manifested by various forms of angina pectoris, condition after myocardial reperfusion, including the state of myocardial stunned)
✓ Irreversible (myocardial infarction)

Heart dysfunction

Hibernated ("sleeping") myocardium is a prolonged inhibition of myocardial function in chronic coronary hypoperfusion, which does not lead to loss of cardiomyocyte viability. In this case, the function of the heart is normalized after the resumption of blood flow by angioplasty or coronary artery bypass grafting.

Acute myocardial infarction

Myocardial infarction is the death of a section of the myocardium due to the cessation of coronary blood flow for more than 20 minutes.

The reasons:
- Atherosclerosis complicated by thrombosis (90%);
- Prolonged coronary spasm;
- Embolism of the coronary arteries.

In the sarcolemma, mitochondria and the nucleus of cardiomyocytes, in muscle fibers, destruction and myolysis occur. Damaged cardiomyocytes are surrounded by mononuclear

leukocytes, which release various cytokines, including fibroblast growth factors, interleukins, tumor necrosis factor, etc.

Necrotic focus of the myocardium disrupts the rhythm of the heart (extrasystole, paroxysmal tachycardia, fibrillation) and reduces the contractile function of the myocardium.

Violations of the contractile function of the heart:

- Necrotized muscle cells are not involved in the contraction and relaxation of the myocardium;
- Energy deficiency does not provide the functioning of the heart with the necessary amount of energy;
- ↓activity of Ca^{2+}-ATPase SPR and overload of Ca^{2+} ions of the sarcoplasm disrupts the relaxation of the myocardium and leads to contracture contraction of the heart.

Ventricular remodeling is a change in its size, shape and wall thickness in the affected and healthy segments of the myocardium. At the same time, dilatation of the cavity of the left ventricle and hypertrophy of healthy areas of the myocardium are observed.

Cardiac arrhythmias in childhood are more often of extracardiac origin. At the same time, perinatal pathology plays an important role (unfavorable course of pregnancy and childbirth, prematurity, intrauterine malnutrition, infection), leading to impaired morphogenesis and functional immaturity of the cardiac conduction system.

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 etiopathogenesis of coronary insufficiency?
2. What are the characteristics of reversible and irreversible coronary blood flow disorders, causes, pathogenesis?
3. What are the changes in the main indicators of heart function in acute and chronic coronary insufficiency?

Lecture #6

1. Topic : Pathophysiology of the heart. Congenital and acquired heart defects

2. Purpose: To form an understanding of the pathophysiological mechanisms of disorders in the body with heart defects . Assimilation of the etiology and pathogenesis of heart failure .

3. Lecture abstracts :

Heart defects - these are congenital or acquired defects in the standard architectonics of the heart and (and) violations of the structure, location, as well as the relationship of its main vessels, with increasing probability leading, as a rule, to disorders of intracardiac and (as a result) systemic hemodynamics. Heart defects are divided into congenital and acquired (valvular).

congenital heart defects

Congenital heart defects are a consequence of its abnormal embryonic development or a consequence of the inability of the progressive development of heart structures in the perinatal or early postnatal period.

Congenital heart defects are more common in male children.

Congenital heart defects can also occur as a result of a violation of the normal changes in the circulatory system that occur at the time of birth.

The fetus actually has only one circle of blood circulation, since its lungs are not expanded, the pulmonary vessels are surrounded by fluid, have relatively thick walls and a small lumen. As a result, the blood that enters the right atrium from the vena cava is largely discharged into the left atrium through the foramen ovale (a hole in the atrial septum).

Congenital pulmonary hypertension, which develops due to thickening of the walls of the pulmonary vessels, as well as due to changes in the content of various biologically active substances in the lungs, their vessels and in the blood as a whole, can lead to a violation of the above pressure ratios in the cavities of the heart and its main vessels , as a result of which the oval window or the ductus ductus, or both together, will not close, which will lead to the development of heart defects.

In total, congenital heart defects occur in approximately 1-2% of live births, that is, about one million children with congenital heart defects are born annually in the world.

Congenital heart defects are divided into ***"white",*** that is, not accompanied by cyanosis *and* ***"blue",*** in which cyanosis is usually pronounced.

Acquired (valvular) heart defects

Acquired heart defects most often occur as a result of a rheumatic process, but syphilis and atherosclerosis can also be their causes. Valvular heart disease can also be congenital. These defects are called valvular, because. *they are based on a violation of the structure and function of either the atrioventricular valves or the valves of the aorta and pulmonary artery.* For each of the valves, there can be two types of damage: either insufficiency, that is, incomplete closure of its valves, or stenosis, that is, narrowing of the corresponding valve opening.

metabolic needs of organs and tissues with the necessary amount of blood.

Heart failure develops when:

- Overload of cardiac activity;
- Primary myocardial disorder;
- Primary damage to the pericardium;
- Severe cardiac arrhythmias;
- Combined lesions of the heart.

According to the predominant lesion of the heart, insufficiency is distinguished:

1. Left ventricular
2. Right ventricular
3. Total

Downstream: acute and chronic

According to the predominant insufficiency of the phase of the cardiac cycle

1. Systolic (violation of the pumping function of the heart → decrease in cardiac output)
2. Diastolic (impaired relaxation of the walls and filling of the left ventricle due to its hypertrophy or fibrosis → increase in end diastolic pressure)

Overload form of heart failure

(develops as a result of increased load on the myocardium and leads to chronic congestive heart failure).

Types of overload:

1. Blood volume overload (“preload”)
- Hypervolemia
- Heart valve insufficiency → ↑filling of the heart cavities with blood during diastole
- Prolonged compensatory hyperfunction of the heart

2. Overload by resistance or pressure (“afterload”)
- Valvular stenosis

- Coarctation of the aorta
- Arterial hypertension of small and large circulation → ↑ resistance to ejection of blood from the heart into systole

Compensation mechanisms for increased heart load

Short term:

1. Increase the strength of heart contractions
- Heterometric mechanism - Frank-Starling law (↑ myofibril length during diastole enhances systole → tonogenic dilation)
- Homeometric mechanism (↑ myocardial voltage power without increasing the length of myofibrils)

2. Increased heart rate
(tachycardia)
- long-term
- myocardial hypertrophy

3. Stages of progressive cardiosclerosis and depletion of myocardial function

Loss of vital activity of a part of cardiomyocytes → proliferation of fibroblasts → proliferation of connective tissue → gradual decrease in the strength and speed of contraction and relaxation of the heart → myogenic dilation → chronic congestive heart failure.

Myocardial form of heart failure :
- Coronary origin (coronary insufficiency)
- Non-coronary origin (myocardial damage by chemical, physical, biological factors)

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 acquired heart defects, what are the concept, types, etiology, pathogenesis of hemodynamic changes?
2. What are congenital heart defects in children (atrial septal defects, interventricular septal defects, tetralogy of Fallot, non-closure of the Botallian duct)?
3. What is heart failure, what is the definition and classification?
4. What is the overload form of heart failure, from the stage of pathological hyperfunction of the heart according to F. Z. Meyerson, characteristic?
5. What is myocardial form of heart failure, what are the causes and pathogenesis?
6. What are the main circulatory changes in acute (cardiac asthma, pulmonary edema) and chronic (edema, bruising, shortness of breath, tachycardia) forms of heart failure?

Lecture No. 7

1. Topic: Pathophysiology of digestion. Digestive disorders in the stomach and intestines.

2. Purpose: Mastering the issues of etiology and pathogenesis of gastric and intestinal dyspepsia.

3. Lecture abstracts :

The causes that cause pathology of the gastrointestinal tract are divided into damaging digestive organs directly and indirectly.

Factors that directly damage the digestive organs:
- *Chemical (tobacco combustion products, toxins, drugs, food additives, alcohol)*
- Physical (rough food, excessively cold or hot food, foreign bodies, radiation)

- Biological (microbes, viruses, helminths, etc.)

Factors indirectly damaging the digestive organs:

- Damage to other organs and physiological systems: blood circulation, kidneys, endocrine glands, liver
- Disorders of the mechanisms of regulation of the gastrointestinal tract (deficiency or excess of BAS-hormones, prostaglandins, biogenic amines, peptides; deficiency or excess of sympathetic or parasympathetic influences).

Risk factors

The conditions conducive to the implementation of the action of causative agents (risk factors) include many factors.

• Violation of the body's reactivity (disorders of the digestive system are often observed in hyperreactive states, for example, against the background of sensitization or prolonged emotional overstrain).

• Gender (for example, peptic ulcer of the stomach and duodenum is more often detected in men). • Age (digestive disorders are much more common in adulthood and old age).

• Hereditary predisposition (for example, peptic ulcers of the stomach often found a family predisposition).

Absorption disorders in the stomach

Normally, water, alcohol, and electrolytes are absorbed in the stomach. With accidental or deliberate intake, toxic agents can be absorbed. With destructive changes in the stomach wall (including violations of the barrier function), protein may enter the internal environment of the body, which is fraught with the development of immunopathological processes: allergic reactions and conditions of immune autoaggression.

Violation of the barrier and protective function of the stomach

The muco-bicarbonate barrier protects the mucosa from acid, pepsin, and other potential damaging agents.

Intestinal malabsorption is a hereditary autosomal recessive enzyme disorder. A feature of carbohydrate digestion in infants is the different rate of hydrolysis of α-lactose and β-lactose.

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 abdominal digestion, causes, consequences?
2. What are the disorders of parietal digestion, causes, consequences?
3. What is malabsorption, what are the concept, types, causes, pathogenesis of the main manifestations?

Lecture No. 8

1. Topic: Pathophysiology of the liver. Violations of the external secretion of the pancreas.

2. Purpose : to study the issues of etiology and pathogenesis of liver dysfunctions.

3. Abstracts of the lecture

Causes of primary and secondary liver damage

1. Biological factors:

- viruses (hepatitis A, B, C, D, F, G; infectious mononucleosis)
- bacteria (causative agents of tuberculosis, syphilis)

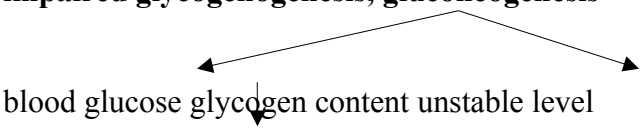
- protozoa (giardia, amoeba)
- fungi, actinomycetes
- helminths

2. Chemical factors (hepatotropic poisons): - alcohol, industrial poisons (carbon tetrachloride, heavy metals, chloroform, arsenic, organophosphate insecticides);
- plant poisons (aflatoxin, muscarine); - tissue breakdown products, products of impaired metabolism; - drugs (PASK, sulfonamides, biomycin, tetracycline, etc.).
3. Physical factors: ionizing radiation, mechanical trauma
4. Nutritional factors: protein, vitamin starvation, fatty foods
5. Dysfunction of other organs and systems:
- circulatory failure
- endocrine and metabolic diseases
- tumors, allergies
- kidney failure

Metabolic disorders in liver failure

1. Violation of carbohydrate metabolism:

impaired glycogenogenesis, gluconeogenesis



blood glucose glycogen content unstable level

neutralizing function tendency to hypoglycemia and liver (violation of the formation **glucuronic acid)**

2. Violation of fat metabolism
- free cholesterol); decrease in the formation of cholesterol esters (
- decrease in the formation of phospholipids;
- increased synthesis of ketone bodies;
- fatty infiltration of the liver.

Pathogenesis of fatty infiltration of the liver:
- increased intake of fat in the liver;
- synthesis of phospholipids and synthesis of TG (triacylglycerols)
- lipolysis and oxidation of fatty acids;
- violation of the release of fats from the liver due to a violation of the synthesis of lipoproteins.

3. Violation of protein metabolism:
4. Violation of vitamin metabolism
5. Disruption of hormone metabolism
6. Violation of the "barrier", neutralizing function of the liver and

Clinical manifestations of liver failure and their pathogenesis

Manifestations	Pathogenesis
- Syndrome of malnutrition - loss of appetite, nausea, abdominal pain, unstable stool, weight loss - Jaundice syndrome	Metabolic disease Violation of the biliary function of the liver, an increase in blood bilirubin and bile acids
- Syndrome of endocrine disorders -decreased libido, testicular atrophy, infertility	an increase in estrogens, as a result of a violation of their inactivation, a violation of the function of the pituitary gland

-gynecomastia - violation of the menstrual cycle - secondary aldosteronism - Syndrome of disturbed hemodynamics -diffuse vasodilation - edema, ascites	a violation of the inactivation of steroid hormones Accumulation of histamine and other vasoactive substances Hemodynamic, ocotic, neuroendocrine factors
- Hemorrhagic diathesis frequent bleeding, development of DIC - Ascites, "head of a jellyfish", dilatation of the veins of the esophagus and hemorrhoidal veins, splenomegaly	Violation of the synthesis of blood coagulation factors Portal hypertension

Understanding the etiology and pathogenesis of pancreatic dysfunction

Causes of violation of the exocrine function of the pancreas:

- Reducing the mass of the pancreas (for example, with necrosis, resection of part of it, tumor damage, sclerosis, pancreatitis, abdominal trauma, exposure to chemicals - alcohol, phosphorus, lead, cobalt).
- Violation of the outflow of the secretion of the gland through its ducts into the duodenum as a result of obstruction of the ducts (stone, tumor, etc.) or compression of the ducts (for example, a neoplasm or scar), with duodenitis.
- Dyskinesia of the ducts of the gland (due to a decrease in tone or, on the contrary, spasm of the SMC ducts).
- Violation of the activity of the gland as a result of nervous and humoral regulatory disorders
- Binge eating
- Increased acidity of gastric juice (pH in the duodenum decreases → activity of pancreatic enzymes decreases

The most common cause of pancreatic insufficiency is pancreatitis. Acute pancreatitis is an acute inflammation and self-digestion of the pancreas and peripancreatic tissues. The causes of acute pancreatitis in 70% of cases are cholelithiasis and alcohol abuse.

The pathogenesis of acute pancreatitis

The leading link in pathogenesis is the activation of trypsin, which, in turn, activates chymotrypsin, elastase, phospholipase A2, etc.

Clinical manifestations	Pathogenesis
Pain is acute "girdle" in the epigastric region with irradiation to the back and iliac region	Stretching of the pancreatic capsule due to edema, decomposition products can enter the retroperitoneal space, cause irritation of the peritoneum
Nausea, vomiting and intestinal obstruction	Capsule distension, peritoneal irritation, electrolyte disturbances (hyperkalemia)
Fever	Inflammatory mediators IL-1, IL-6, TNF. A fever that lasts more than 5 days, or a temperature rise above 40 ^{0}C indicates infectious complications
Jaundice (in 20% of cases)	Compression of the bile duct with swelling of the head of the pancreas
hypovolemia	Exudation and bleeding into the retroperitoneum, or accumulation of fluid in the intestine with intestinal obstruction

hypotension	Hypovolemia, activation of the kallikrein-kinin system, proteolytic enzymes → vasodilation
Shock	Hypovolemia, cytokines, proteolytic enzymes
Increased amylase and lipase in the blood	Amylase level rises 10-20 times immediately after pancreatic injury and lasts 48-72 hours Lipase level rises 72 hours after the onset of symptoms and takes longer to normalize (better for diagnosis)
Increased blood clotting	Massive tissue damage, activation of proteolytic enzymes → DIC
Hypocalcemia, increased neuromuscular excitability, tetany	Lipolysis of adipose tissue →↑ blood FFA → binding to Ca^{2+}
Hyperglycemia (in 25% of cases)	Damage to insulocytes during inflammation, increased levels of glucocorticoids and adrenaline

Chronic pancreatitis

Main reasons : chronic alcoholism and difficulty in the outflow of pancreatic juice.

Pathogenetic mechanisms for the development of chronic pancreatitis:

- Reflux of bile into the pancreatic duct
- Sphincter of Oddi obstruction
- Viscosity increase and protein hypersecretion
- Decrease in lithostatins (peptides that are secreted into pancreatic juice and inhibit the formation of protein conglomerates and the aggregation of calcium carbonate crystals. A decrease in lithostatin occurs under the influence of alcohol, and can also be hereditary)
- hypersecretion of calcium
- Decreased secretion of trypsin inhibitors
- Increased lactoferrin, which promotes the formation of protein aggregates

The bile of children differs in composition from the bile of adults. It is poor in bile acids, cholesterol and salts, but rich in water, mucin, pigments, and in the neonatal period, in addition, urea.

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 liver failure, what types, etiology?
2. What are the changes in metabolism and functions of physiological systems in liver failure?
3. What is the neutralizing function of the liver, the study in the experiment?
4. What are the causes of violations of the secretory function of the pancreas?
5. What is the pathogenesis of digestive disorders in pancreatic achylia?
6. What are the main causes of pancreatitis?

Lecture No. 9

1. Topic: Pathophysiology of the kidneys. Dysfunction of the glomeruli and tubules.

2. Purpose: Mastering the issues of etiology and pathogenesis of renal dysfunction. Understanding the etiology and pathogenesis of renal failure

3. Lecture abstracts :

The kidneys are vital organs. The main purpose of the kidneys is to maintain the constancy of the internal environment (homeostasis) of the body .

The kidneys perform their homeostatic functions and form urine through the processes of filtering the constituent components of plasma, reabsorption and secretion, as well as the synthesis of a number of substances.

Violation of filtration can be expressed in its decrease or increase, regardless of the amount of fluid that has entered the body.

Both renal and extrarenal factors can lead to a decrease in filtration volume . Renal reasons for decreased filtration include:

• decrease in the number of functioning glomeruli due to their replacement with fibrous tissue, destructive processes in the kidneys;

• decrease in the permeability of the filtering membrane due to the germination of the connective tissue, the deposition of immune complexes, autoantibodies on it;

• sclerotic changes in the afferent arterioles and interlobar vessels;

• an increase in pressure in the cavity of the Bowman's capsule due to an increase in intrarenal pressure with edema of the interstitium or impaired patency of the tubules and urinary tract.

Extrarenal reasons for reduced filtration may be:

1) decrease in systemic blood pressure due to heart or vascular insufficiency, blood loss, dehydration; when systolic blood pressure falls below 50 mm Hg. filtration stops completely;

2) an increase in the oncotic pressure of the blood plasma as a result of an increase in the concentration of proteins, which can occur with their increased synthesis (for example, with multiple myeloma), the administration of protein preparations, or blood clotting.

Tubulopathies are diseases caused by a violation of the transport functions of the epithelium of the renal tubules due to the absence or qualitative changes in carrier proteins, certain enzymes, receptors for hormones, or dystrophic processes in the wall of the tubules.

According to etiology, primary (hereditary) and secondary (acquired) tubulopathies are distinguished.

Depending on the localization of the defect, proximal and distal tubulopathies are distinguished.

Glomerular filtration disorders in children are characterized by low renal blood flow and high renal vascular resistance. Phosphate diabetes (hypophosphatemic rickets) - proximal tubulopathy. The main defect in this case is a decrease in phosphate reabsorption with subsequent hypophosphatemia.

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 etiology and pathogenesis of disorders of the renal tubes and glomeruli?
2. What are the clinical manifestations of dysfunction of the renal tubes and glomeruli?
3. What is the pathogenesis of hematuria?
4. What is the pathogenesis of proteinuria?
5. What are the general provisions, types of renal failure, pathogenesis?
6. What is the classification of kidney failure?
7. What is the etiology, classification, pathogenesis of acute renal failure?

8. What is the etiology, classification, pathogenesis of chronic renal failure?

Lecture No. 10

1. Topic: General pathophysiology of the endocrine system

2. Purpose: To study the general etiology and pathogenesis of endocrinopathies

3. Lecture abstracts :

The regulation of many phenomena occurring in the life of the body is provided by the joint functions of the nervous and endocrine systems. These systems are closely interconnected. Therefore, the integration of the body into a single complex self-regulating system occurs as a result of neuro-endocrine regulation.

The endocrine glands secrete hormones (Greek, hormao - excite) into the blood and intergastric fluid. The endocrine glands include the pituitary gland, thyroid gland, parathyroid gland, pancreas, adrenal glands, gonads, pineal gland, thymus, etc.

Disorders of the endocrine system can develop in three pathogenic ways:

1. Due to violations of the central regulatory mechanisms;
2. Diseases of the glands located in them
3. Due to over-the-counter hormone activity disorders.

Violations of the central regulatory mechanisms.

The central nervous system has a regulatory effect on the functions of the endocrine glands. The outer cortex of the brain, the anterior thalamus, the limbic system, the hippocampus, the hypothalamus, and the structure of the brain are present in the regulation of the function of these glands. Various stresses and under stressful conditions from the structures of the central nervous system to the hypothalamus send information along the nerve pathways.

When exposed to prolonged stressful situations, thyrotoxicosis (Based's disease) or some types of diabetes mellitus may develop. Such disorders of the endocrine system are called psychogenic endocrinopathies. The hypothalamus is of particular importance in the regulation of the endocrine glands.

Violation of its regulation of peripheral endocrine glands can be caused by many reasons. These include infection, inflammation, hemorrhage, thrombosis, trauma, tumor development, stressful situations.

Diseases located in the glands themselves

Numerous diseases can be observed in the peripheral endocrine glands. Therefore, hormone production and its excretion into the blood are disrupted in these glands. Painful changes in the endocrine glands appear: from exposure to microbes and their toxins, lack of blood supply to blood vessels, hemorrhage, inflammation, tumor development, from autoimmune damage to the glands, from congenital disorders of hormone formation , weakening of the function of the glands after prolonged work.

Effects of infectious processes and toxins.

Many microorganisms and their toxins cause damage to the endocrine glands. For example, infectious diseases lead to dysfunction of the endocrine glands. Meningoccal infection may be accompanied by hemorrhage in the adrenal glands. Diphtheria leads to coagulative necrosis in these glands. Mumps causes orchitis in men. Tuberculosis and syphilis disrupt the functions of the adrenal glands, ovaries, and thyroid glands. Chronic insufficiency of these glands or bronze disease (Addison's disease) develops due to damage to the tubercle bacillus of the adrenal glands. Also in other glands, the development of tuberculosis is possible and there is a lack of their functions.

Disorders of the breakdown of hormones in the body.

Violation of the breakdown of hormones occurs with hepatitis and cirrhosis of the liver. When the breakdown of cortisol slows down, it increases in the body. This, according to the feedback law, inhibits the function of the adrenal glands. Such cases are observed in the absence of sex hormones, aldosterone hormones; the activity of these hormones is maintained at a high level in the body for a long time. In the pathogenesis of many endocrine disorders, great importance has insufficient (hypofunction) or high (hyperfunction) gland function. But some endocrine glands produce several hormones. So, for example, the pituitary gland produces several tropic hormones (ACTH, STH, TSH, HTG), the adrenal glands produce glucocorticoid, mineralo-corticoid, sex hormones.

The significance of endocrine disorders in the pathogenesis of non-endocrine diseases.

Violation of the hormonal balance of the body creates favorable conditions for the emergence and development of non-crine diseases. For example, insufficiency of relative or true corticosteroids can lead to the development of rheumatism, bronchial asthma. Excessive formation of glucocorticoid hormones, aldosterone, catecholamines occupies an important place in the pathogenesis of arterial hypertension.

The central regulation of the endocrine glands in children has its own characteristics. This is due to the fact that in functional terms, the hypothalamic-pituitary system is immature at the time of the birth of the child and develops as it grows.

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 three pathogenesis of endocrine disorders?
2. What are the violations of the central regulatory mechanisms?
3. What are the violations of the decomposition of hormones in the body?
4. What is the significance of endocrine disorders in the pathogenesis of non - endocrine diseases?

Lecture No. 11

1. Topic: Pathophysiology of individual endocrine glands .

2. Purpose: Understanding the etiology and pathogenesis of individual endocrine glands .

3. Lecture abstracts :

Pathophysiology of the pituitary gland . Insufficiency of the function of the pituitary gland.

Panhypopituitarism. In humans, complete insufficiency of the function of the pituitary gland is detected when 90% of its tissue is destroyed. This syndrome is called panhypopituitarism, or Simmonds-Sheehan syndrome. The following causes can lead to the development of this syndrome: vascular disorders in the pituitary and hypothalamus (most often postpartum prolonged spasm of the vessels of the brain and pituitary due to blood loss against the background of adenohypophysis hyperplasia - postpartum hypopituitarism), trauma to the base of the skull, tumors of the pituitary and hypothalamus, inflammatory damage (tuberculosis, sepsis) of the pituitary gland, congenital aplasia and hypoplasia of the pituitary gland, etc.

Isolated insufficiency of hormones of the anterior pituitary gland Insufficiency of somatotropic hormone . Insufficient formation of growth hormone leads to the development of pituitary dwarfism, or dwarfism. In more than half of the cases, the development of the disease is associated with a genetically determined decrease in GH secretion, which is manifested by two main types of disorders: congenital pituitary aplasia and familial panhypopituitarism or isolated GH

deficiency. In this case, inheritance can be both autosomal and sex-linked. In other patients, the cause of the disease is either not established (idiopathic dwarfism), or it is caused by organic disorders of the hypothalamic-pituitary region (trauma, tumors, circulatory disorders, inflammatory changes).

Deficiency of adrenocorticotropic hormone. Insufficient formation of ACTH leads to secondary partial insufficiency of the adrenal cortex. Mainly glucocorticoid function suffers. The mineralocorticoid function practically does not change, since the mechanisms of its regulation are different. The difference from the primary hypofunction of the adrenal cortex is the absence of the development of hyperpigmentation, due to the fact that the level of **ACTH is reduced and its melanophoric effect is not manifested.**

Deficiency of thyroid-stimulating hormone. A decrease in TSH production causes a secondary decrease in thyroid function, which leads to the development of symptoms of secondary hypothyroidism. In contrast to the primary hypofunction of the thyroid gland, the introduction of TSH can restore its function. The content of TSH in the blood (but may also decrease due to the inclusion of a feedback mechanism in hyperfunction of the thyroid gland. For example, in diffuse toxic goiter, due to hyperfunction of the gland and excessive formation of T3 and T4, the formation of TSH is inhibited.

Insufficiency of gonadotropic hormones. With insufficient formation of HTH, various disorders occur, the picture of which depends on which HTH is not formed and how much their deficiency is combined with the loss of secretion of other hormones of the adenohypophysis

Excess secretion of adrenocorticotropic hormone. Increased secretion of ACTH by the pituitary gland leads to the development of Itsenko-Cushing's disease, which is manifested by bilateral adrenal hyperplasia and increased secretion of adrenal hormones. Itsenko-Cushing's syndrome should be distinguished from Itsenko-Cushing's syndrome, which has a similar clinical picture, but is caused by a hormonally active adenoma or adenocarcinoma of the adrenal cortex, as well as malignant tumors of extra-adrenal localization that produce ACTH-like peptides (for example, bronchogenic lung cancer).

corticosteroid deficiency. Corticosteroid insufficiency can be total, when the action of all hormones falls out, and partial - when the activity of one of the hormones of the adrenal cortex drops out. Total corticosteroid deficiency in the experiment is caused by adrenalectomy. After adrenalectomy, the animal inevitably dies with symptoms of severe adynamia and hypotension. Life expectancy ranges from several hours to several days

Hypercorticosteroidism. Hypercorticosteroidism (hypercorticism) refers to such changes in the body that correspond to an increase in the function of the adrenal cortex. Hypercorticosteroidism can develop due to excessive formation (or increased activity) of one or several hormones at once. The most common types of hypercorticosteroidism are: hypercortisolism, aldosteronism, and adrenogenital syndromes.

Hypercortisolism is a complex of such changes in the body that are caused either by excessive formation of cortisol in the fascicular zone of the adrenal cortex, or by an increase in cortisol activity due to a decrease in its binding by transcortin.

Excess production of estrogen. Less commonly, a tumor of the reticular zone produces estrogen (corticoestroma). In girls, it causes premature sexual and physical development. In men, feminization develops, during which male secondary sexual characteristics disappear and female ones appear. Changes in physique, voice, deposits of adipose tissue according to the female type are noted.

Hyperthyroidism is a syndrome caused by an overactive thyroid gland. Severe hyperthyroidism is called thyrotoxicosis. Hyperthyroidism, depending on the place where the primary violation occurred, can be divided into primary, secondary and tertiary. The causes of primary hyperthyroidism may be a dysfunction of the thyroid gland, which develops in diseases such as diffuse toxic goiter (Basedow's disease, Graves' disease, Parry's disease), thyrotoxic adenoma of the thyroid gland. The cause of secondary hyperthyroidism may be the development of a TSH-secreting tumor of the adenohypophysis, and the cause of tertiary hyperthyroidism may be a disorder in the hypothalamus.

Hypothyroidism is a condition that occurs when there is a lack of thyroid hormones in the body. Like hyperthyroidism, it can be primary, secondary, or tertiary. Primary hypothyroidism occurs with Hashimoto's thyroiditis, defects in the biosynthesis of thyroid hormones, thyroidectomy, radioactive iodine treatment, insufficient intake of iodine in the body, and other pathological processes in the gland. Secondary and tertiary hypothyroidism are the result of loss of regulatory influences (pituitary damage, thyroliberin deficiency).

Hypothyroidism in older children and adolescents is most often the result of autoimmune thyroiditis, iodine deficiency, removal of the thyroid gland, pituitary adenoma, CNS tumor, and traumatic brain injury.

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 pathophysiology of the pituitary gland , pituitary insufficiency?
2. What is the isolated insufficiency of anterior pituitary hormones?
3. What is insufficiency of adrenocorticotropic and gonadotropic hormone a ?
4. What is the pathophysiology of the gonads ?

Lecture No. 12

1. Topic: .Pathophysiology of the blood system. Pathology of erythrocytes.

2. Purpose: Mastering the issues of etiology and pathogenesis of anemic syndrome.

3. Lecture abstracts :

Anemia is a pathological condition characterized by a decrease in the concentration of hemoglobin and, in the vast majority of cases, the number of red blood cells per unit volume of blood.

Etiology of anemia

- includes acute and chronic bleeding.
- infections, inflammation.
- intoxication (salts of heavy metals).
- helminthic invasions, malignant neoplasms, vitamin deficiencies.
- diseases of the endocrine system, kidneys, liver, stomach, pancreas.

Classification

1. According to the mechanism of development, there are three main types of anemia ;
- Anemia due to blood loss (posthemorrhagic) .
- Anemia due to impaired blood formation.
- Anemia due to increased blood destruction (hemolytic) .

2. Severity _

-Light
-Medium
-Heavy

3. According to the color index, anemia is divided into
- hypochromic
- normochromic
- hyperchromic

With anemia in the peripheral blood, fixed or supravitally stained smears may contain erythrocytes and erythroid forms of the bone marrow, which are not detected in healthy people .

Variant of pathological changes
- Change in the size of red blood cells (anisocytosis)
- Discoloration of red blood cells (anisochromia)
-Change in the shape of red blood cells (poikilocytosis)
- Inclusions in erythrocytes

The group of regenerative forms of erythrocytes includes immature forms of erythropoiesis - nucleated erythrocytes (normoblasts, megaloblasts), erythrocytes with remnants of a nuclear substance (Jolly bodies, Kabo rings).

Hemolytic disease of the newborn (HDN) is a disease based on hemolysis of fetal and newborn erythrocytes, associated with incompatibility of maternal and fetal blood for erythrocyte antigens.

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 congenital and acquired forms of anemia and pathogenesis?
2. What is the classification of acquired hemolytic anemia ?
3. What are the causes of iron deficiency anemia?
4. What is the pathogenesis of iron deficiency anemia?

Lecture No. 13

1. Topic: Pathology of leukocytes. Hemostasis disorders

2. Purpose: Mastering the issues of etiology and pathogenesis of leukemia

3. Lecture abstracts :

Leukemia is a tumor originating from ancestral (stem) hematopoietic cells with a primary lesion of the red bone marrow. Leukemia is based on uncontrolled proliferation of cells with a violation of their ability to differentiate and mature. According to the degree of differentiation of leukemia cells, leukemias are divided into acute and chronic. In acute leukemia, the substrate of the tumor is blast cells of II, III and IV classes of hematopoiesis, which have lost the ability to mature. In chronic leukemia, the tumor substrate consists of maturing and mature cells. There is a partial delay in maturation.

Etiology leukemias .

In the etiology of leukemia, the role of oncogenic viruses, ionizing radiation, chemical carcinogens, and genetic abnormalities has been established.

The role of viruses :

More often, leukemia is caused by RNA-containing oncoviruses, less often by DNA-containing oncoviruses related to herpes viruses. RNA-containing oncoviruses cause spontaneous leukemia in birds, mice, cattle, monkeys and other animals. Viruses can be transmitted through urine, feces, nasal secretions, pharynx, and from mother to offspring. In the experiment, leukemia is reproduced by introducing cell-free filtrates of leukemia cells from sick animals to healthy ones. The viral origin of human leukemia has been proven in relation to T-cell leukemia (RNA-containing virus HTLV-I type). It is considered possible transmission of the virus through blood transfusion, sexual contact.

The role of ionizing radiation

The role of ionizing radiation in the occurrence of leukemia has been experimentally proven. Both acute and chronic exposure to X-rays induce leukemia in rats and mice. An increase in the incidence of acute and chronic myeloid leukemia was observed among residents of Hiroshima and Nagasaki, among radiologists and radiologists. There is evidence of an increase in the frequency of leukemia in patients treated with high doses of X-rays, yttrium, radium for malignant neoplasms.

The role of chemical carcinogens

Chemical carcinogens can cause acute leukemia in humans through occupational contact with benzene, organic solvents. An increase in cases of leukemia was noted in patients with malignant neoplasms treated with cytostatics such as cyclophosphamide, chlorbutine, methotrixate, myalosan. Butadion, chloramphenicol are also referred to drugs that can induce leukemia. In the experiment, leukemia is caused by the introduction of chemical carcinogens (dimethylanthracene, methylcholanthrene), as well as derivatives of tryptophan, tyrosine, and indole.

Features of leukemia in children

Leukemia in children is a significant part of malignant neoplasms.

In children, leukemia is almost always acute lymphoblastic. The highest incidence of leukemia in children occurs at the age of 2 to 4 years, then some increase is observed in adolescents aged 12-14 years. Quite often children of the first months, the first weeks and even the first days of a life get sick. Boys suffer from acute lymphocytic leukemia 5 times more often than myeloid leukemia, and girls have lymphocytic leukemia 2 times more often than myeloid leukemia. In young children, as well as in adults, acute leukemia is most often aleukemic or subleukemic. While chronic myeloid leukemia is almost always leukemic.

The onset of the disease in children is usually sudden, the earliest symptom of the disease is bone pain, swollen lymph nodes, liver, and spleen. Frequent manifestations of acute leukemia include necrotic lesions of the skin and oral mucosa, with the development of gingivitis of stomatitis, as well as the intestinal wall with corresponding manifestations of indigestion.

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 concept of hemoblastoses ?
2. What is the etiology and pathogenesis of leukemia?
3. What is the pathogenesis of clinical manifestations of myelo- and lymphoproliferative syndrome?
4. What are the features of leukemia in children?

Lecture No. 14

1. Topic: Pathophysiology of the musculoskeletal system and skin

2. Purpose: To study the etiopathogenesis of inflammatory and degenerative lesions of the musculoskeletal system.

3. Abstracts of the lecture:

The musculoskeletal system is formed by the bones of the skeleton, joints, cartilage, muscles and ligaments.

Functions of the musculoskeletal system:

∨ locomotor, which is carried out in cooperation with all body systems;

∨ protective, thanks to which the brain and back, the brain, chest cavity and pelvic organs, as well as the bone marrow are protected from external influences;

∨ metabolic: 99% calcium, 87% phosphorus, 50% magnesium and 46% sodium are deposited in the bone tissue, which determines its participation in various types of metabolism;

∨ hematopoietic: carried out by the red bone marrow located in the spongy substance of flat bones, vertebral bodies and metaphyses of tubular bones.

Structure and physiology of bones

Spongy and compact substance, periosteum and endosteum, as well as the medullary canal in tubular bones and medullary lacunae of flat bones are isolated in the bone. Mature bone tissue has a mineralized and non-mineralized matrix (osteoid). There are two cell lines in the bone - synthesizing (osteoblasts) and destroying (osteoclasts), which provides a balance between the synthesis and resorption of bone tissue.

• A compact layer is formed by osteons, consisting of collagenous bone plates, circularly located around the osteon canal, or the haversian system of tubules. Nerves, blood and lymphatic vessels pass through them. Osteons are delimited from each other and externally by a cementing substance. The compact layer is covered on the outside by a connective tissue periosteum (periosteum).

• The spongy layer is located inside the bone and consists of intertwining bone trabeculae surrounding cavities filled with bone marrow. The spongy layer is shrouded in endosteum, which consists of connective tissue.

• The bone matrix makes up 50% of the dry weight of the bone and consists of inorganic (50%), organic (25%) components and water (25%).

◊ The mineral component contains predominantly two chemical elements - calcium (35%) and phosphorus (50%), forming hydroxyapatite crystals, which are connected to collagen molecules through osteonectin. The composition of the inorganic part of the bone also includes bicarbonates, citrates, fluorides, Mg2+, K+, Na+ salts.

◊ The organic component of the bone is built from predominantly type I (90-95%) and type V collagens, non-collagen proteins (fibronectin, osteonectin, etc.), as well as glycosaminoglycans. 25% is water.

• Osteoid - non-mineralized organic bone matrix around osteoblasts that synthesize and secrete matrix components. In the future, the osteoid is mineralized, which requires 1α,25-dihydroxycholecalciferol, the active form of vitamin D.

• Osteogenic cells are located in the periosteum and endosteum and are represented by the following forms.

◊ Osteoblasts are process cells that synthesize procollagen and other bone matrix proteins. They provide the formation and mineralization of bone, and also secrete alkaline phosphatase, which is involved in the exchange and deposition of calcium and in the metabolism of other electrolytes. Osteoblasts have receptors for sex hormones, which largely regulate their function.

◊ Osteocytes are flat mononuclear cells located in bone cavities, or lacunae. Their function is to preserve the bone matrix and its mineral composition.

• Osteoclasts - multinucleated cells belonging to the system of mononuclear phagocytes. Their function is bone resorption. The activity of osteocytes is regulated mainly by osteoblasts.

Diseases of the musculoskeletal system are divided into the following groups: ∨ diseases caused by genetic disorders (dysplasia), ∨ metabolic diseases of the bone tissue, ∨ infectious diseases of the bones, ∨ bone tumors, ∨ skeletal bone injuries, ∨ diseases of the joints, ∨ pathology of the ligamentous-muscular apparatus.

The pathology of the musculoskeletal system in children has its own characteristics. They are associated with the continuation of the formation of the child's skeleton after birth. In children, diseases caused by genetic disorders (dysplasia) and injuries of the bones of the skeleton are more common.

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 functions of the musculoskeletal system?
2. What is the structure and physiology of bones?
3. What is osteopetrosis, achondroplasia, osteogenesis imperfecta?
4. What is Paget's disease, what are the etiology and complications?

Lecture No. 15

1. Topic: Features of leading syndromes in gerontology and geriatrics

2. Purpose: to study the causes and mechanisms of aging development. .

3. Abstracts of the lecture:

Modern gerontology, which studies the patterns of aging of living beings (including humans), suggests that genetically the life expectancy of people is 100-130 years. According to the regulatory-adaptive theory of aging by V.V. Frolkis, life expectancy (vitality) of a person is determined by two multidirectional processes: aging and vitality. Aging is a destructive universal process of withering, leading to a limitation of the adaptive capabilities of the body, contributing to the development of diseases that limit life expectancy, and leading to death.

Under premature (pathological) aging understand any acceleration of the rate of aging, characteristic of the middle age group of people. Unfortunately, in the last two decades in Russia, there has been an acceleration of the aging process not only among old and elderly people, but even among people 45-55 years old. Aging (both physiological and pathological) is influenced by both internal, especially genetic, and external factors. Long-livers have a biological age less than a calendar one. In contrast, in pathological aging, the biological age is ahead of the calendar age. The aging process is characterized by heterochronism, heterotopicity, heterokineticity, heterocategory. Heterochronism is the difference in the time of onset of aging of individual tissues, organs and systems. So, hypotrophic changes in the thymus begin after 13-15 years, the sex glands - in the menopause, and the pituitary gland - shortly before death. 93 Heterotropy - unequal expression of aging in different structures of the same organ or in different organs. Heterokineticity is the unequal rate of development of age-related changes. Heterocatephism is the multidirectionality of age-related changes. For example, with aging, there is a decrease in the production of sex hormones by the peripheral glands and an increase in the production of gonadotropic hormones by the adenohypophysis. Pathological aging, which limits life expectancy and increases mortality, is caused by a variety of pathological processes and conditions, intoxication, diseases (mostly

chronic), overwork, physical inactivity, distress, malnutrition, ionizing radiation, bad habits, aggravating heredity, disorders of the nervous, endocrine, immune systems, etc.

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 theories of aging ?
2 . What are the mechanisms of aging ?
3 . What changes occur in organs during aging ?
4 . What are the principles of prevention of premature aging?

Appendix No. 1

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