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Practical work for common and private Pathophysiology. Questions, experiments, films, tests

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This manual includes all practical lessons on the subject of Pathophysiology. Every topic includes: scientific basing of theme, aim of lesson, experiments, educational film, questions and scientific literature.

Рецензенты:

Заведующий кафедрой патологической физиологии ФГБОУ ВО КГМУ Минздрава России И.И. Бобынцев

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PART I

GENERAL PATHOPHYSIOLOGY

THEME OF LESSON: SUBJECT OF PATHOPHYSIOLOGY. AIMS AND TASKS OF SUBJECT. PATHOPHYSIOLOGICAL EXPERIMENT AND ITS CHARACTERISTIC

Plan of practical work:

1. Tests and discussion of theoretical material - 60 min.
2. Fulfillment of practical work - 60 min.
3. Discussion of results - 15 min.

1. Scientific basing of topic. Pathophysiology is fundamental (paraclinical) theoretical discipline studying common causes (etiology), common pathogenesis and outcome of pathological processes and diseases. Subject of Pathophysiology consists of two parts: common and private pathophysiology. Students introduce with history of subject, nosology (etiology, pathogenesis, pathological process, pathological state and disease), typical pathological processes (hypoxia, shock, fever, inflammation, tumors, allergy, disturbance of peripheral blood circulation, pain) by studying of general pathophysiology. In second part of pathophysiology (pathophysiology of organs and systems) students introduce with common laws of disturbance of blood, cardiovascular, nervous, endocrine, respiratory systems, kidneys, liver, gastro-intestinal tract.

Pathophysiology is an experimental discipline and basic method of it's pathophysiological experiment.

Aim of pathophysiological experiment is knowledge of causes of disorder functions of the organism, dynamic and mechanism formation of these disorders and principles of treatment.

Pathophysiological experiment has three phases (periods):

First phase - studying physiological parameters (for example ECG, heart rate, rate of respiration, quantity of glucose, hormones in the blood or urine, arterial pressure and so on) in starting point. Rather rare animal is situated without anesthesia or fixed. Such situation is not physiological and for this reason we cannot say physiological indexes but indexes in starting (initial) point.

Experiment allows studying possible etiologic factors (microbes, viruses, trauma, ionization, toxins and so on).

Second phase. Reproduction and studying of pathological process (for example, hypoxia, inflammation, fever and so on) or disease. Definite pathophysiological process or disease is reproduced and the same indexes as in first phase are recorded in dynamic process and disease. It allows to study and understand pathogenesis of pathophysiological process or disease.

Third phase - experimental treatment. Pathologist tries to work out principles of prophylaxis and etiotropic and pathogenetic treatment on the bases of studied etiology and pathogenesis.

2. Aim of topic. Introduction with three phases of pathophysiological experiment.

EXPERIMENT 1. Experiment of Ber.

Aim. Study three phases of pathophysiological experiment on the bases of development hypobaric hypoxia.

Method. Rat is put into an opened germetic camera and its behavior is studied: rate of breathing, color of visible part of skin and mucosa in starting point. Then germetic camera is closed and with a special pump, air is withdrawn from the germetic camera. Air pressure in germetic camera decreases up to 170-180 mm Hg. Partial pressure of oxygen is inhaled air decreases and hypoxic hypoxia develops. The same indexes are again studied as in initial point (behavior, rate of breathing, color of skin and mucosa, possible appearance of convulsions). After 10-15 minutes oxygen is pumped (0,5-1 1). Students register changes of the above mentioned indexes.

Results. Students draw table and diagrams in dynamic of hypoxic hypoxia.

Discussion. Students discuss results and make conclusions.

Conclusions.

EXPERIMENT 2. Insulin shock by rat.

Aim. It is necessary to study three phases of pathophysiological experiment on example of insulin shock.

Method. Experiment is fulfilled on two rats. Both rats are starved 24 hours before experiment to exhaust glucose in liver.

First phase. Study behavior and reactions of the rats on irritators, color of skin and mucosa.

Second phase. Insulin (10 unites per 100 g body's weight) is injected intraperitoneally to both rats. Study during 30 minutes above, mentioned indexes and appearance of convulsions due to development of insulin shock.

Third phase. Solution of glucose (3-4 ml 20%) is injected to one animal to restore quantity of glucose in the blood and notice change behavior, color of skin and mucosa, reactions on irritators.

Results. Students describe results of experiment.

Discussion. Students discuss manifestations by insulin shock, its pathogenesis, treatment and make conclusions.

Conclusions.

QUESTIONS

1. Subject of pathological physiology.
2. Etiology. Definition. Role knowledge of etiology organization of prophylaxis and treatment (etiotropic therapy).
3. Pathogenesis. Definition.
4. Main chain of pathogenesis.
5. Cause-effect relations as a base of pathogenesis.
6. Circus virtuous in teaching about pathogenesis.
7. Idea about pathological process.
8. Teaching about health and disease.
9. Common characteristic of pathophysiological experiment.
10. Phases of pathophysiological experiment.
11. Connections between pathological physiology and paraclinical and clinical disciplines.

Tests

1. What does pathological physiology study?

A) Manifestations of diseases.

B) Functions of systems and organs.

C) Common laws appearance, development, current and outcomes pathological processes and diseases.

2. Mark main method of pathological physiology

A) Acute experiment.
B) Chronic experiment.
C) Experiment with treatment.
D) Pathophysiological experiment.

3. Mark correct understanding term of «Etiology».

A) Teaching about definite cause (reason).
B) Many reasons can produce disease.
C) Teaching about causes and conditions appearance of pathologic process or disease.

4. Mark correct understanding term of «Pathogenesis»

A) Sum of changes by development of disease.
B) Pathogenesis is origin of disease.
C) Pathogenesis is teaching about appearance, development and outcome of pathological process or disease

5. What is «Nosology»?

A) Teaching about typical pathology.
B) **Teaching about disease.**
C) Teaching about changes in organs

6. What is vicious cycle?

A) Consequence of events, by which chain of pathogenesis produces effect and interrupts development pathological process.
B) Process, by which chain of pathogenesis does not produce
C) Formation of disease.
D) It is such cause-effect correlations, which can form cycle. Such phenomenon makes current of pathological process or disease more heavy and dangerous.

7. What is «Etiology»?

A) Sum of conditions, producing disease.
B) Teaching about consequences of disease.
C) **Teaching about causes and conditions of disease**.

8. What is main chain of pathogenesis?

A) The final chain of pathogenesis.
B) The leading pathogenetic chain. Removal of its prevents next development pathological process or disease.

9. What is the base of pathogenesis?

A) The cause-effect correlations, where the cause produces effect and next effect is a cause next effect, supplying development pathologic process or disease.

B) The final pathogenetic chain.

10. Mark definition of «Nosology».

A) Teaching about causes of disease.

B) **Teaching about disease.**

C) Teaching about pathologic process.

11. Classification of secondary (acquired) reactivity.

A) Specific (immunological) and nonspecific.

B) Adaptive and constitutional.

C) Basic and allergic.

12. Choice correct definition reactivity of the organism.

A) Capacity of the organism to react on different etiological factors.

B) Capacity of the organism to react on external and internal factors with complex defense-adaptive reactions.

13. Mark the most important resistance by children.

A) Mechanisms of passive resistance.

B) Mechanisms of active resistance

14. Mark tendency to diseases in hypersthenic.

A) Hypotension.

B) Hypertension.

C) Gastro-intestinal tract.

15. Mark tendency to diseases in hyposthenics.

A) Diabetes mellitus.

B) **Pathology of lungs**.

C) Hypertension.

16. Mark big risk development of disease by Men.

A) Myxedema.

B) Hypotension.

C) Infarct of myocardium.

D) Hyperthyroidism.

17. Mark big risk development disease by women.

A) Ulcer of stomach.
B) Fever.
C) Pathology of liver.
D) Cancer of lungs.

LITERATURE

1. Ovsyannikov V.G. "Pathophysiology (questions and answers)", 2005, p. 4-10.

2. Nowak Th. J., Handford A.G. "Essentials of pathophysiology. Concepts and Applications for Health Care Professionals". London, Singapore, 1999, p. 4-5.

3. Robins, Cotran, Kumar, Collings Pathologic bases of disease. Sixth Edition, 1974.

THEME OF LESSON: DAMAGING INFLUENCE OF HIGH AND LOW TEMPERATURE ON THE ORGANISM

Plan of practical work:

1.Tests and discussion of theoretical material. - 60 min

2.Fulfillment of practical work. - 60 min.

3. Discussion of results. - 15 min.

1. Scientific basing of topic. Balance between heat production and heat loss allow to hold body temperature in rather narrow diapason (range) from 36°C to 37°C. Comfortable environmental temperature is equal to +17-18°C. Increase or decrease external environmental temperature can influence on the organism unfavorably and produce different pathological processes. Frost bite, freezing, hypothermia, cold shock and catarrhal diseases can develop in organism of the man by action of low temperature. Burn, hyperthermia and burning disease can form by influence of high temperature. Behavioral, biochemical, physiological reactions can compensate unfavorable common action of changeable temperature,

By influence of low temperature next compensatory reactions can form by man: behavioral-reactions by man: buttoning up of clothes, go into warm space, physical exercises, use dressing warm clothes.

Biochemical - intensification of metabolism and release additional quantity of "primary heat" due to activation of sympatho-adrenal and hypothalamus-hypophysis-thyroid gland system.

Splitting ATP and formation of "secondary heat".

Physiological - angiospasm of skin, decrease blood circulation in skin, due to excessive release epinephrine, norepinephrine, vasopressin.

All compensatory reactions allow to keep heat in the body and increase heat production.

By influence of high temperature next compensatory reactions can form by man: behavioral - unbuttoning of clothes, going in the shade, increase blow of wind.

Physiological vasodilatation of skin, sweating and evaporation of sweat, increase breathings rate.

All compensatory reactions directed on increase intensity of heat loss.

By development of such pathological processes as hypo-and hyperthermia two stages are distinguished: stage of compensation and stage of decompensation. Body temperature is not changed while compensatory reactions will be able to compensate and body temperature is not changed. The all above mentioned compensatory reactions cannot supply compensation; body temperature is changes (increase or decrease).

By decrease body temperature up to 24-25°C all biological processes are stopped. Such temperature is named “biological nil or zero”. By increase of body temperature up to 39-40°C can form "Heat stroke". By 44-45°C body temperature organism dies due to coagulation of protein in the cells and disorder of all vital functions.

2. Aim of topic. Study unfavorable action of high and low temperature on the organism.

EXPERIMENT 1. Influence of cold and warmth on a "frog's" heart.

***Aim*.** Study direct action of cold and warmth on the heart.

Method. Spinal cord of frog is destroyed by special needle.

Motionless frog is fixed on the back on the preparation board. Frog's thorax is opened, pericardium is removed and heart rate is counted in starting point. Then a tube of water (50-60°C) is applied to the heart in region of sino-atrial node and is counted heart rate. The same procedure with cold tube is done.

Results. Students prepare table, make diagrams,

Discussion. Students discuss results, compare with theoretical material and make conclusions.

Conclusions.

EXPERIMENT 2. Common action of cold on the organism.

Aim. Study change of heart rate, ECG and body temperature in dynamic of hypothermia.

Method. Experiment is carried out on a rat. Rat is anesthetized intramuscular. (0,4 ml of 2% nembutalum per 100 g body's weight).

ECG is recorded in 10-15 min, heart rate and rectal temperature in initial point. Then ice is put on the rat body, and the same indexes are recorded in dynamic of hypothermia (after 10, 20, 40 min; 1, 2, 3, 4, S, hours). Then animal is warmed up and the same indexes are recorded too.

Results. Students prepare tables and diagrams.

Discussion. Students discuss results, compare data with theoretical stuff and make conclusions.

Conclusions.

EXPERIMENT 3. Common action of heat on the organism.

Aim. Study change of ECG, heart rate and body temperature in dynamic of hyperthermia.

Method. Experiment is carried out on the rat. Animal is anaesthetized intramuscular with nembutalum (0,4 ml 2 % per 100 g of body's weight). Animal is fixed on experimental board. After 10-15 minutes ECG, hearts rate and rectal temperature (with help of electrothermometer) are recorded in starting point.

Then put electrical hot water bottle (temperature 37-50°C) out on the animal's body and the same indexes are recorded in dynamic of hyperthermia (10', 20°, 40', 1, 2, 3 hours).

Results. Students prepare tables and graphs.

Discussion. Students discuss results and make conclusions.

Conclusions.

3. Demonstration of educational film. "Disturbance of heart rate,
ECG and body temperature in dynamic of hypothermia"

QUESTIONS

1. Main phenomena of damaging action of low temperature on the organism.
2. Pathogenesis and stages of frost-bite.
3. Pathogenesis of hypothermia.
4. Compensatory reaction by hypothermia (behavioral, biochemical, physiological).
5. Stages of hypothermia.
6. Disturbances in the organism in stage of compensation by hypothermia.
7. Disturbances in the organism in decompensatory stage of hypothermia.
8. Use of hypothermia in medicine.
9. Main phenomena of damaging influence of high temperature on the organism.
10. Pathogenesis and stages of burn.
11. Pathogenesis of hyperthermia.
12. Types of hyperthermia.
13. Disturbances in the organism in compensatory stage of hyperthermia.
14. Disurtances in the organism in decompensatory stags of hyperthermia.
15. Heat stroke. Clinical manifestations and changes in the brain

Tests

1. Mark localization center of thermoregulation.

A) Thalamus.
B) Cortex of the brain.
C) Preoptic zone of Hypothalamus.
D) Cerebellum.

2. What reason (cause) of endogenic hyperthermia?

A) Decrease heat production. Increase heat loss.

B) Increase sweating (perspiration).

C) Disorder coupling between oxidation and phosphorylation in mitochondria.

3. By damage of what structures of the brain can be desorbed thermoregulatory reactions in the organism?

A) Cortex of the brain.

B) Thalamus.

C) Extrapyramidal centers.

D) Hypothalamus.

4. What types of hypoxia can form by hypothermia?

A) Respiratory.

B) Hemic (anemic).

C) Circulatory.

D) Tissue.

E) All above mentioned.

5. How can change quantity of primary heat by disorder coupling between oxidation and phosphorylation.

A) Increase.

B) Decrease.

C) It is not changed.

6. Why can form tachycardia in first stage of hyperthermia?

A) Increase tonus parasympathic nervosa system.

B) **Increase activity of sympatho-adrenal system**.

C) Decrease arterial blood pressure.

7. How can change diameter of vessels by influence of cold?

A) Dilatation.

B) Angiospasm.

C) Reaction will be absent.

8. How can change heat production in first stage of hypothermia?

A) **Increase**.

B) Decrease.

C) It is not changed.

9. What changes in the blood can form in first stage of hypothermia?

A) Increase quantity of glucose.
B) Decrease quantity of cholesterol.
C) Increase quantity of lipid acids.

10. How can change rhythm of the heart by hypothermia in stage of decompensation?

A) Tachycardia.
B) **Bradycardia**.
C) Rhythm is not changed.

11. What body temperature of man is lethal?

A) 30 C.
B) 28 C.
C) 24-25 C.

12. Mark clinical symptoms first stage of frost bite.

A) Cyanosis.
B) Formation of bubbles.
C) Necrosis.
D) Redness.

13. How can change rate contraction of myocardium by increase body temperature on one degree centigrade?

A) It is not change.
B) Increase on 8-10 contractions.
C) Increase on 20-30 contractions.

14. What substance is not endogenic pyrogen?

A) Insulin.
B) Interleukin 1.
C) Interleukin 2.
D) Tumor necrotic factor – alfa.

15. Mark effect surplus of perspiration (sweating).

A) **Hypohydratation**.
B) Hyperhydratation.

16. What can allow to form hyperthermia?

A) **High temperature of external environment.**
B) Low temperature of external environment.
C) Intensive cold wind.

17. What changes in the brain can appear by heat stroke?
A) Decrease intracranial pressure.
B) **Hyperemia and hemorrhages.**

LITERATURE

1. Ovsyannikov V.G. "Pathophysiology" (questions and answers), 2005, p. 11-15.

2. Nowak Th.J., Handford A.G. "Essentials of pathophysiology. Concepts, Applications for Health Care Professionals", London, Singapore, 1999. p.47-49.

3. Robins, Cotran, Kumar, Collings Pathologic bases of disease. Sixth Edition, 1974.

THEME OF THE LESSON: DAMAGING INFLUENCE OF IONIZING ENERGY ON THE ORGANISM

Plan of practical work:

1.Tests and discussion of theoretical material. - 60 min.

2.Fulfillment of practical work. - 60 min.

3. Discussion of results. - 15 min.

1. Scientific basing of the topic. Ionizing energy (a- and B-particles, neutrons, x-ray, γ-rays) can produce damage in the organism on different level (atom, molecule, cell, organ, system and organism). Excitation and ionization can form on atom's level.

Rupture (splitting) of molecules can develop on the molecule level.

Death of cell, inhibition of mitosis, mutation can develop on the level of the cell by influence of ionizing energy.

Cells are most radiosensitive in state of division (mitosis) and that is why the most radiosensitive organs are bone marrow, lymph nodes and gastrointestinal tract.

Radiant energy can produce disorder of all system of the organism (nervous, endocrine, blood, coagulate, immunological).

Ionizing disease can develop on the level of organism itself. There are three forms of ionizing sickness. They are: bone-marrow form, intestinal form, cerebral form. Distant manifestations of ionization are: tumor, regulatory disturbances, disorder of sexual function and defect of offspring.

EXPERIMENT 1. Change of the blood by radiation disease

Aim. It is necessary to show that by influence of big doses of ionization can develop ionizing disease and blood system is disturbed.

Method. Quantity of erythrocytes, hemoglobin and leukocytes are counted in the blood from rat's tail vena in starting point and are ionization.

Results. Students prepare tables and diagrams change quantity of erythrocytes, leukocytes and hemoglobin.

Discussion. Students discuss results, compare with theoretical material and make conclusions.

Conclusions.

2. Demonstration of educational film: «Radiation disease»

QUESTIONS

1. Types of ionizing energy.
2. Damaging influence of ionization on atom and molecular level.
3. Damaging influence of ionization on cellular level.
4. Damaging influence of ionization on the level of organs and systems.
5. Idea about radiosensitivity and radioresistance.
6. "The law of Bergonie and Tribandon".
7. Direct and indirect effects of ionization.
8. Dependence biological effect of ionization on dose, type of ionization, surface of ionization, reactivity of the organism.
9. Etiology, pathogenesis acute manifestations bone-marrow form of radiation disease.
10. Etiology, pathogenesis, manifestations of acute intestinal form of radiation disease.

11. Etiology, pathogenesis, manifestations of acute cerebral form of radiation disease.

12. Distant manifestations of ionization.

Tests

1. What ionizing factor can have the biggest penetration in object?

A) Alfa-particles.
B) Beta-particles.
C) Neutrons.

2. Which ionizing factor can produce the most big ionization?

A) X-rays.
B) Alfa-particles.
C) Gamma-rays.

3. Mark the most radiosensitive cells.

A) Muscle cells.
B) Cell of Thymus and lymph nodes.
C) Epithelium of skin.

4. Mark the most radio resistant cells.

A) Epithelium of skin.
B) Nerves cells.
C) Endothelium.

5. What remote (distant) consequences by damaging influence of ionizing radiation?

A) Shortening of life span.
B) Development of malignant tumors.
C) Sexual disorders.
D) Disorders in offspring's.
E) All above mentioned.

LITERATURE

1. Ovsyannikov V.G. "Pathophysiology" (questions and answers), 2005. p. 16-20.

2. Nowak T.J., Handford A.G. "Essentials of pathophysiology. Concepts and applications for health care professionals" London, Singapore, 1999.

THEME OF LESSON: DAMAGING INFLUENCE OF ELECTRICITY ON THE ORGANISM

Plan of practical work:

1.Tests and discussion of theoretical material. - 60 min.

2. Fulfillment of practical work. - 60 min.

Discussion of results. - 15 min.

1. Scientific basing on the topic. There are two main types of electricity: alternating and constant current. Alternating current can produce damaging effect the most often due to its use in industry, agriculture and private life.

Damaging influence of electricity depends on: physics parameters (power, voltage and frequency), time of action, path of conduction and reactivity of the organism. Electricity can produce electrothermial, electromechanical and biological effects.

Pass of electric current through the tissue or organ can produce release of heat, due to presence of resistance. Such heat produces electrical burn. Electric current high tension power can produce detach parts of the body (fingers, extremities), due to formation a lot of heat (effect of explosion). Electric current can produce and such biological effects as change in systemic arterial blood pressure (SABP), heart rate, respiration, metabolism, nervous and endocrine system.

There are two most dangerous paths of electricity: through the heart and through the brain. Disturbance of vital centers of the brain can lead to the death. Death can develop and by pass of electricity through the heart due to cardiac arrest or fibrillation.

Damaging influence of electric current depends on species and individual peculiarities of the organism. For example, narcosis, aggression, sleep decreases damaging influence of electricity. On the other hand insufficiency of adrenal gland, pathological process increases pathogenic effect of electricity.

2. Aim of topic. It is necessary to show, that electricity is very dangerous for the organism.

Attention. Demonstrative experiment is fulfilled only by teacher.

EXPERIMENT 1. Action of electric current on reflectory function of frog's spinal cord.

Aim. It is necessary to show that reflectory function of spinal cord is disturbed by influence of electric current.

Method. The frog's upper jaw is cut with help of scissors. Frog hangs for the lower jaw support (tripod) and after 5-7 minutes spinal reflexes determine when symptoms of spinal shock will disappear.

For this aim is used irritators of different power (0,25%, 0,5%, 1% of sulfuric acid solution). One paw is put into the glass with solution of different concentration of sulfuric acid and determines time in seconds, when frog removes paw from the solution of sulfuric acid.

Electrodes put on spinal cord and electric current (220 V) switches on during 1 second. Spinal reflexes determine 1, 5, 10 minutes after action of electricity.

Results. Students record time of spinal reflex, make table and diagrams.

Discussion. Students discuss the results and make conclusions about dangerous influence of electricity on the organism.

Conclusions.

EXPERIMENT 2. Influence of electric current on the heart.

Aim. It's necessary to show, that electric current can produce change of ECG and development of fibrillation and cardiac arrest.

Method. Rat is anesthetized and fixes on special table. In starting point is recorded ECG and then (only teacher!) electric current (220 V during 2-3 seconds) switches on animal through the electrodes. ECG recorders again.

Results. Students analyze ECG, determine heart rate, presence of fibrillation or cardiac arrest.

Discussion. (Students discuss results and make conclusions).

Conclusions.

EXPERIMENT 3. Local action of alternative electric current.

Aim. It is necessary to show, that electric current can produce local effect in form of electrical burn.

Method. A frog is anesthetized 0,5 ml solution of Hexanalum is injected intraperitoneally. A frog is fixed on the preparation board. Electrodes on distance are fixed in the frog's skin 0,4-0,5 cm and put into the electrical socket (voltage 220V, during 1 second).

Results. Students describe size and character of electrical necrosis, Discussion. Students discuss mechanism of electrical burn and make
conclusions.

Conclusions.

3. Demonstration of educational film: "Electric current. Systemic and local action". 15 min.

QUESTIONS

1. Electric current types. Its types. Physical parameters of alternative electric current, damaging influence of electric current in dependence on its physical characteristics.
2. Mechanism of local action of electricity.
3. Mechanism, of common action of electricity.
4. Mechanism of electrical burn by influence of alternative and constant electric current.
5. Mechanism of electromechanical effect of alternative current.
6. Mechanism damaging influence of high tension current lines.
7. Main causes of the death by influence of electric current.
8. What the most dangerous pathways of electricity in the organism and why?
9. Why persons who are situated under the influence of electrical current can not call for the help?
10. When damaging influence of electricity is minimal?
11. Stages of electrotrauma in dependence on its intensity.
12. Principles of help to a man, who is situated under the influence of electric current.

Tests

1. Mark main factors that can define damaging influence of electricity on the organism.

A) Physical description.
B) Path of electricity.
C) Reactivity of the organism.
D) **All above mentioned.**

2. What path of electricity in the organism of man is the most dangerous?

A) Through the legs.
B) Through the heart and brain.

3. Mark main reasons (causes) of death by effect of electricity.

A) Damage of the lungs.
B) Damage of the muscles.
C) **Fibrillation, cardiac arrest and stop of breathing**.

4. Mark states of the organism, when decreases damaging Influences of electricity.

A) By narcosis.
B) On time of sleeping.
C) In state of extreme excitation.
D) All above mentioned.

LITERATURE

1. Ovsyannikov V.G. "Pathophysiology" (questions and answers), 2005. p.20-22.

THEME OF LESSON: REACTIVITY OF THE ORGANISM AND ITS ROLE IN PATHOLOGY

Plan of practical work:

1. Tests and discussion of theoretical material. - 60 min.
2. Fulfillment of practical work. - 60 min.
3. Discussion of results. - 15 min.

1. Scientific basing of topic. Reactivity of the organism is possibility of the organism to react on action of environmental (external or internal) factors with

complex of compensatory-adaptive reactions. Resistance of the organism is possibility of the organism to resist to action of pathogenic factors.

Species, group and individual reactivity are distinguished.

Biologists are interested in species reactivity. Study of group and individual reactivity is main task of medicine. Vaccination, vitamins, physical exercises are very important in change reactivity human or animal populations such way can form resistance against many pathogenic factors.

Individual reactivity is classified on primary (basic) and secondary or acquired. Heredity, constitution, sex and age definite basic reactivity. Secondary reactivity changes during the life and classified into specific and nonspecific. Specific reactivity can be formed by vaccination, allergy, and immunodeficiency state.

Nonspecific reactivity can form under the action such nonspecific factors as temperature, weather, stress, cosmic, transferred diseases, change pH of the blood and so on.

"Trace reactions" and dominance is the base of nonspecific reactivity of the organism.

"Law of starting point" has very important role in reactivity of the organism. It means, that organism can react differently depending on state of functions of his organs and systems. For example, reactions of the organism on irritators some another than in the evening, when function of organs and systems under the influence of stress.

Students have to understand that possibility of the organism to react on environmental factors depends on state of nervous, endocrine, immune system, intensity and character of metabolism.

They are mechanisms of reactivity. By studying of pathophysiology students will introduce with multiple examples, when by change mechanisms of reactivity will change and reactivity itself.

Practical classes and theoretical material allow understanding better such very important section as reactivity of the organism and its role in pathology.

2. Aim of topic. Study reactivity and its role in pathology on examples of some experiments.

EXPERIMENT *1. Dependence reactivity on functional state of nervous system.*

Ain. It is necessary to Show that change functional state of nervous system, change reactivity of the organism to deficit of oxygen.

Method. Take three mice. One intact, second injected with 0.2-03 n 1% solution of nembutalum, the third with 0,1 ml 1% solution of Koffein. All animals are put into three hermetic glass vessels with the same volume (100-150 ml). Metabolic processes in organisms of experimental animals will be different and consequently, consumption of oxygen too. Behavior and time of death of mice will depend on rate development of hypoxia.

Results. Students study change of behavior of mice and rate development of death.

Discussion. Students discuss results, explain mechanisms change of behavior and death of experimental animals and make conclusions.

Conclusions.

EXPERIMENT 2. Change reactivity of the organism depending on age.

Aim. Study dependence of sensitivity of the organism to hypoxia depending on age.

Method. Take two rats (adult and newborn), put them into two hermetic glass vessels. Study behavior and time of death.

Results. Students register change in behavior of animals in dynamic of hypoxia and time of death.

Discussion. Students discuss mechanism change in behavior animals and death too. Make conclusions.

Conclusions.

EXPERIMENT 3. *Influence of stress on reactivity of the organism.*

Aim. It is necessary to show, that stress change reactivity of the organism (increase resistance Selje).

Method. Take two mice. One mouse swims 3-5 minutes in swimming pool. Second is control. Both mice are put into vacuum pump of Komovsky and the air

is withdrawn, imitating lift on the height. Students register height on which animal dies.

Results. Students describe results.

Discussion. Students discuss role stress in reactivity of the organism and mechanisms of death and make conclusions.

Conclusions.

QUESTIONS

1. Idea about reactivity of the organism. Definition. Role reactivity of the organism in pathology.
2. Classification of reactivity.
3. Group reactivity and its role in pathology.
4. Classification of individual reactivity.
5. Role of heredity in reactivity of the organism.
6. Role of constitution in reactivity of the organism.
7. Role of sex in reactivity of the organism.
8. Role of age in reactivity of the organism.
9. Acquired reactivity. Classification.
10. Nonspecific reactivity and its mechanisms.
11. Role of starting functional state in reactivity of the organism.
12. Role of acid-base balance in reactivity of the organism.
13. Role of metabolic mechanisms in reactivity of the organism.
14. Role of endocrine mechanisms in reactivity of the organism.
15. Role of nervous mechanisms in reactivity of the organism.

LITERATURE

1.Ovsyannikov V.G. “Pathophysiology“ (questions and answers), 2005. p.23-30.

THEME OF LESSON: SPECIFIC AND NONSPECIFIC PROTECTIVE REACTIONS AND ITS DISTURBANCES BY PATHOLOGY

Plan of practical work:

1. Tests and discussion of theoretical material. - 60 min.
2. Fulfillment of practical work. - 60 min.
3. Discussion of results. - 15 min.

1. Scientific basing of topic. Nonspecific and specific protective mechanisms are formed by warm-blooded organisms during biological evolution. Such nonspecific and specific protective mechanisms are formed first of all against foreign biological factors (microbes, viruses, rickettsia, helmints, protozoa). One can distinguish the following defense mechanisms by warm-blooded organisms:

1. Skin and mucous membranes.
2. Cellular mechanisms (phagocytosis).
3. Humoral nonspecific mechanisms.
4. Immunologic mechanisms.

Skin as a barrier can protect penetration of biological factors mechanically, due to withdraw with sweat, desquamative epithelium, acidic medium and formation of inflammation.

Mucous membranes have different defense reactions: cilia, normal microflora, immunoglobulins A, enzymes (pepsin) acidic medium (hydrochloric acid), inflammation.

Cellular mechanism is phagocytosis. Phagocytes can engulf and digest foreign biological factor. Such functions have especially polymorphonuclear leukocytes.

Humoral mechanisms are humoral substances of the blood (complement, properdin, lysozyme, B-lyzines, C-reactive protein, interferons), which can take part in inactivation and destruction of biological damaging factors.

Immunologic mechanisms (immunity, allergy), antibodies and nonspecific mechanisms can protect or damage and withdraw foreign biological factor from the

organism, and by its insufficiency can form tendency to development of local or general pathological processes.

2. Aim of topic. Study change of phagocytosis by pathology.

EXPERIMENT 1. Change phagocytic activity of leukocytes by pain stress.

Aim. Study possible change of phagocytic activity of leukocytes by acute somatic pain of 5th grade.

Method. Experiment is fulfilled on a rat. In starting point the blood 0,5 ml is taken from sublingual vein, add 1 drop suspension of staphylococcus aureus (1 million in I mm^3) and put in thermostat for 1 hour. Then smear of the blood is made and stained by Romanovsky-Giemsa. 100 leukocytes count in smear of the blood and phagocytic number.

Phagocyte index = quantity of active phagocytes/100%

Phagocytic number = quantity of engulfed microbes/100%

Then the rat is put into chamber with metallic floor, which is connected with electrical plug. Electrical plug is switched on for one second into electric rosette (tension 220 Vollts). Pain of 5^{th} grade develops (generalized motion, run with cry aggression). 0,5 ml of blood taken from sublingual vena, add into the blood 1 drop suspension of Staphylococcus aureus (1 million in 1 mm^3) puts for I hour in thermostat.

Smear of the blood prepares, stains by Romanovsky-Giemsa and under immersion of microscope counts 100 leukocytes, definite phagocytic index and phagocytic number.

Results. Students make table and graph of change phagocytosis by acute somatic pain.

Discussion. Students discuss mechanism change of phagocytosis by acute pain and make conclusions.

Conclusions.

EXPERIMENT 2. Change of phagocytosis by hypercortizolism.

Aim. Study possible change phagocytic activity by increase quantity of glucocorticoides in the organism.

Method. Experiment is fulfilled on 2 rats. Fist rat is intact. 10 ml of 3 % solution of peptone is injected intraperitonealy to intact animal. 24-48 hours later 1 ml of Exudate from abdominal cavity is taken with help of syringe. Add one drop suspension of staphylococcus 15-20 min later make smear, stained by Romanovsky-Giemsa and study phagocytosis under the microscope with the immersion. Phagocytic index and phagocytic number counts.

Prednisolon (1 ml/100 g mass of animal) is injected intramuscular to second (experimental) rat for one week (7 days).

The same 10 ml of 3% solution of peptine is injected intraperitoneally. 24-38 hours later 1 ml of blood is injected.

Exudates from abdominal cavity are taken with help of syringe, by Romanovsky-Giemsa and study smear under the microscope.

Results. Students draw table and graph change of phagocytosis by hypercortisolism.

Discussion. Students discuss change mechanisms of disturbances of phagocytosis by hypercortisolism and make conclusions.

Conclusions.

3. Demonstration of educational film "Disturbances of phagocytosis by chronic pain. (10 min)

QUESTIONS

1. Modern conceptions concerning the notion "factors of natural nonspecific resistance".
2. Functions of barrier structures of the organism.
3. Effects, which can formed by complement's activation.

3 Biological effect of lysozyme and is disorder by pathology.

5. Properdin and its effect.
6. Effects of -interferons.
7. Main consequences of deficiency or functional disorder of humoral nonspecific protective factors.
8. Hereditary pathology of phagocytosis.
9. Etiology of increase phagocytosis.

10. Etiology of decrease phagocytosis.

11. Main function of immunity.

12. Etiology of innate immunodeficiency state.

13. Etiology of acquired immunodeficiency state.

14. Pathogenetic classification of acquired immunodeficiency state.

15. Main consequences of immunodeficiency state.

LITERATURE

1.Ovsyannikov V.G. "Pathophysiology" (questions and answers), 2005, p. 36-46.

2. Nowak Th.J., Handford A.G. "Essentials of pathophysiology. Concepts and Applications for health Care, Professionals". London, Singapore, 1999, p.9-10, 34-38, 100-117.

THEME OF LESSON: ALLERGY

Plan of practical work:

1. Tests and discussion of theoretical material. - 60 min.

2. Fulfillment of practical work. - 60 min.

Discussion of results. - 15 min.

1. Scientific basing of topic. "Allergy is a typical pathological process which can appear after the influence on the organism a allergens (foreign proteins and haptens) and is characterized increase sensitivity of the organism. In the base of which lays immunological mechanisms"

Two conditions are necessary for the development of allergic reactions: presence of sensibilization and repeated influence the same allergen, which produced hypersensitivity.

By development of any type of allergic reaction are distinguished three stages

(A.D. Ado): immunological, pathochemical and pathophysiological.

Short characteristic of these stages:

1) Immunological stage. Humoral or cellular immunity is stimulated by influence of allergens. Allergic antibodies (IgE, IgG, IgM) or sensitized lymphocytes are stored and produce hypersensitivity of the organism. Maximum sensibilization forms after 2-3 weeks.

Immunologic conflict between allergen and allergic antibodies or sensitized lymphocytes develops after repeated influence of allergen on hypersensitized organism.

Immunologic conflict between allergens and allergic antibodies is the base of immediate type of allergic reactions.

Immunologic conflict between allergen and sensitized lymphocytes is a base of delayed type of allergic reactions. Four types allergic reactions are distinguished in dependence on mechanisms of its development. They are: anaphylactic type, cytotoxic type, immunocomplex type, cellular (delayed) type).

2) Pathochemical stage is characterized by release of different allergic mediators by immediate and delayed types.

3) Pathophysiological stage is characterized by disorder in structure and function of organs and systems of the organism and manifested by local (inflammation) or general reactions (anaphylactic shock, serum sickness).

Apart from allergic are distinguished paraallergic (false) reactions. Absence of immunologic stage in comparison with allergic is registered by paraallergic reactions, but pathophysiological (clinical) manifestations are the same, due to release in pathochemical stage practically the same mediators as by immediate types allergic reactions.

2. Aim of topic. Study etiology, pathogenesis and general principles diagnosis of hypersensitivity and treatment of allergic reactions.

EXPERIMENT 1. Reproduction of anaphylactic shock by dog.

Aim. Study change of systemic arterial blood pressure and rate of breathing in dynamics of anaphylactic shock.

Method. Dog is sensitized by horse serum (0,3 ml/kg) three times (intramuscular, intramuscular, intravenously) with interval 24 hours.

After 2-3 weeks, when is formed maximum of sensibilization animal is fixed. In starting point are registered systemic arterial blood pressure and respiration. To get anaphylactic shock 0,3 ml/kg of body weight is injected intravenously.

Systemic arterial blood pressure is studied in dynamics of anaphylactic shock.

Results. Students make table and graphs on change of systemic arterial blood pressure and respiration.

Discussion. Students discuss about mentioned indexes in dynamic of shock and make conclusions.

Conclusions.

EXPERIMENT 2. Reproduction of Artus phenomenon.

Aim. It's necessary to show that 5-6 injections of foreign protein with intervals 5-6 days produces hemorrhagic-necrotic allergic inflammation.

Method. Horse serum (0,5 ml) is injected to rabbit under skin 5-6 times with interval 5-6 days.

Results. First and second injection of horse serum will not produce any clinical manifestations in focus of injection. After third, fourth injection forms redness swelling. 5^{th} and 6^{th} injection produce hemorrhagic-necrotic allergic inflammation.

Discussion.

Conclusions.

PARAALLERGIC REACTIONS.

EXPERIMENT 3. Anaphylactoid reaction of rats on injection of egg albumin.

Aim. Study development of some anaphylactoid manifestations by injection on rat of egg albumin.

Method. 0,2-0,3 ml of egg albumin is injected intra-abdominal.

Study external appearance, color of mucus, size of paws during 2 hours.

Results. Due to degranulation of mast cells, basophiles release the same mediator as by allergy. Main effect of mediator is increase vascular permeability and development odema of paws, snout, and scrotum.

Discussion. Students discuss why by anaphylactoid reaction appears the same manifestation as by allergy and make conclusion.

Conclusion.

EXPERIMENT 4. <u>Reproduction of Shwarzman phenomenon.</u>

Aim. Its necessary to show that endotoxin can produce activation of complement by alternative path and produces formation of hemorrhagic-necrotic inflammation.

Method. Endotoxin is injected intradermaly on rabbit. After 24 hours endotoxin (0,3ml/kg) is injected in marginal vein of the rabbit ear. After 24 hours study development of hemorrhagic-necrotic inflammation in focus of primary intradermal injection of endotoxin.

Results. Students study development of hemorrhagic-necrotic inflammation in skin 24 hours later after intravenous injection of endotoxin.

Discussion. Students discuss why by injection of endotoxin can develop inflammation and make conclusions.

Conclusions.

3. Demonstration of educational film: "Pollinosis" - 20 min.

4. Demonstration of educational film: "Allergy under the microscope".

5. Demonstration of educational film: "Disorder of microcirculation by anaphylactic shock".

QUESTIONS

1. The definition of allergy.
2. Conditions which are necessary for the development of allergic reactions.
3. Stages of allergic reactions.
4. Classification of allergic reactions depending on the rate and mechanism of its development.
5. Sensibilization and its mechanism.
6. Pathogenesis of immediate type of allergy.
7. Pathogenesis of delayed type of allergy.
8. Mediators of immediate type of allergy and their common biological effects.
9. Mediators of delayed type of allergy and their common biological effects.
10. Pathophysiological manifestations hypersensitivity of immediate type.
11. Pathophysiological manifestations hypersensitivity of delayed type.
12. Principles diagnosis of hypersensitivity.
13. Principles treatment of allergy.
14. Idea about etiology and pathogenesis of paraallergic reactions.

Tests

1. Show right definition of «Allergy».

A) Allergy is state of increased sensitivity organism to repeated contact with allergens. The base of allergy is immunological mechanism.

B) Allergy is reaction on enter in the organism foreign macromolecule.

2. Mark peculiarities of specific hyposensibilization.

A) Produces with antihistamine drugs.

B) **Use allergen, which produced allergy.**

C) Produces with glucocorticoid hormones.

3. Anaphylactic type of allergy can form by immunological conflict between allergen and:

A) Ig M.

B) Ig G.

C) Ig A.

D) Ig E.

4. Mark immunological conflict by delayed (cellular) type of allergy.

A) Sensitized T-lymphocytes (T-killers) + allergen.

B) Allergic antibodies + allergen.

C) T-lymphocyte + T – suppressors.

5. Immunocomplex types of allergy can form by interaction between allergen and:

A) Ig M and G.

B) Ig A.

C) Ig E.

6. Mark substances, which can be liberated in pathochemic stage cellular (delayed) allergy.

A) Histamine and serotonin.

B) Lymphokines.

C) Prostaglandins.

7. Schwartsman phenomenon can form due to:

A) Activation of lymphocytes.

B) **Activation of complement**.

C) Increase sensitivity of cholinoreceptors.

8. Mark main mediator of immediate type allergy.

A) Interferon.

B) Transfer factor.
C) Histamine.

9. Mark role of T-helpers in antibodygenesis.

A) Recognition of antigen.
B) Formation of antibodies
C) Release of lymphokines.

10. Show local delayed allergic reactions.

A) Anaphylactic shock.
B) Tuberculin test.
C) Pollinosis.

11. Mark conditions which are necessary for the development of allergic reaction.

A) Contact with allergen.
B) Hanger and contact with allergen.
C) Sensibilization of the organism and repeated contact with allergen, which produced sensibilization.

12. Stimulation of what type of immunity are necessary for the development of immediate type allergy.

A) Cellular.
B) **Humoral**.
C) Active.
D) Passive.

13. Mark main chain pathogenesis of immediate type allergy.

A) Increase potassium.
B) Increase of histamine.
C) Increase of interferon.

LITERATURE

1. Ovsyannikov V.G. "Pathophysiology" (questions and answers), 2005, p.47-56.

2. Nowak Th.J., Handford A.G. "Essentials of pathophysiology. Conceptions and Applications for Health Care Professionals", London, Singapore, 1999, p.89-94.

THEME OF LESSON: DISORDER OF PERIPHERAL BLOOD CIRCULATION AND MICROCIRCULATION. HYPEREMIA, ISCHEMIA

Plan of practical work:

1. Tests and discussion of theoretical material. - 60 min.
2. Fulfillment of practical work. - 60 min.
3. Discussion of results. - 15 min.

1. Scientific basing of topic. Peripheral vessels average and small arteries and veins, microcirculatory net, supply peripheral organs and tissues with blood and consequently with nutrient, oxygen, hormones and other regulatory factors and allow to fulfill functions of organs and tissues. Main phenomena of disorder of peripheral blood circulation are the following: hyperemia, ischemia, thrombosis, embolism.

2. Aim of topic. Study etiology, pathogenesis, consequences of arterial hyperemia and ischemia.

Hyperemia is a local plethora of organ or its part.

Hyperemia is classified depending on origin: arterial (active) and venous (passive, stagnant). Arterial hyperemia is a local plethora due to increase in blood inflow through arteries to organ or its part. Neurotonic, neuroparalytic, myoparalytic and metabolic arterial hyperemia can occur.

By arterial hyperemia in microcirculatory net is registered increase in diameter of arterioles, quantity of functional capillaries, hydrostatic pressure. Clinically, arterial hyperemia is characterized by the following common clinical symptoms: redness, increase in size and temperature of hyperemized region.

Ischemia - is such disorder of peripheral blood circulation which is characterized by decrease or complete restriction of blood inflow through the arteries to organ or its part. Ischemia is classified depending on origin: angiospastic, obturative, compressive.

Decrease in diameter of arterioles, hydrostatic pressure, quantity of functioning capillaries, centralization of blood circulation are registered in microcirculatory net by ischemia.

Ischemia is characterized by the following common clinical symptoms: paleness, decrease in size and temperature (only in skin) of ischemized focus, pain.

EXPERIMENT 1. Reproduction of arterial huperemia.

Aim. Reproduction of arterial hyperemia and study of changes in vascular net and temperature of skin in hyperemized region.

Method. Vascular net of ear and temperature of skin is noted in starting point. Ear is smeared carefully by turpentine and vascular net, temperature of the skin of the ear (with help of electrothermometer) again is noted.

Discussion. Students discuss results and make conclusions.

Conclusions.

EXPERIMENT 2*. Change in microcirculation by arterial hyperemia.*

Aim. Study peculiarities of change in microcirculation by arterial hyperemia.

Method. Spinal cord of the frog is damaged. Heard of animal is fixed on the preparation board near the aperture. Take out the tongue of the frog, stretch it and fix it with pins on the angle to aperture. Study microcirculatory net under the microscope (ocular 8, objective- 8). Note the diameter of arterioles, the number of functional capillaries, and character of blood flow and general picture of blood circulation.

Then the surface of the tongue is carefully smeared by turpentine.

Discussion. Students discuss results and make conclusions.

Conclusions.

EXPERIMENT 3. *Venous hyperemia on the rabbit's ear.*

Aim. Study clinical symptoms of venous hyperemia.

Method. Skin temperature and vascular net of both ears of the rabbit are noted in starting point.

In started point Study the color of skin, vascular net and skin temperature of both ears in dynamics of venous hyperemia (during 1 hour).

Results. Students describe change in color of skin, vascular net and skin temperature.

Discussion. Students discuss results and make conclusions.

Conclusions.

EXPERIMENT 4. ***Disorder of microcirculation by venous hyperemia.***

Aim. Study peculiarities of disorder of microcirculation by venous hyperemia.

Method. Spinal cord of the frog is damaged bloodlessly. The frog is fixed on the preparation board in position of its abdomen down.

Take out the tongue; stretch it over the side of aperture fixing it with pins. Study the set up preparation under the microscope (ocular 8, objective 8). Ligature carefully vein of the frog's tongue. Study the peculiarities of disorder of microcirculation.

Results. Students describe delay of blood flow rate, decrease in linear and volumetric blood inflow, push and balance - like blood flow, stasis and make conclusions.

Conclusions.

3. Demonstration of educational film "Microcirculation" - 20 min.

4. Demonstration of educational film "Experimental reproduction of hyperemia, ischemia. Disorder of microcirculation by arterial, venous hyperemia and ischemia" - 10 min.

EXPERIMENT 5. ***Experimental reproduction of ischemia on the rabbit's ear.***

Aim. It's necessary to show that severe pain produces angiospastic ischemia.

Method. Experiment is fulfilled on rabbit. Study color of rabbit ear and its vascular net in starting point. Pain is produced due to severe mechanical pressure on rabbit's tail. Study the change in color of rabbit's skin of the ear and vascular net.

Results. Students describe change in color of skin and vascular net by angiospastic ischemia.

Discussion. Students discuss results and make conclusions.

Conclusions.

QUESTIONS

1. Definition of hyperemia.
2. Classification of hyperemia.
3. Change of microcirculation by arterial and venous hyperemia.
4. Etiology of arterial hyperemia.
5. Etiology of venous hyperemia.

6. Common clinical symptoms of arterial hyperemia and its origin.

7. Common clinical symptoms of venous hyperemia and its origin.

8. Outcomes of arterial and venous hyperemia.

9. Common phenomena of disorder of microcirculation.

10. Notion of ischemia.

11. Etiology of ischemia.

12. Pathogenesis of ischemia.

13. Disorder of microcirculation by ischemia.

14. Common clinical symptoms of ischemia and its origin.

15. Outcomes of ischemia.

Tests

1. What does mean «centralization of blood circulation»?

A) Circulation of blood in big vessels.
B) Circulation of blood in vital organs (brain, heart, liver).
C) **Circulation of blood through arteriolo-venule anastomosis (shunts). Blood can't pass through the capillaries.**

2. In what section of vascular system can begin formation aggregation of blood cells?

A) Big vessels.
B) Arterioles.
C) Venules.

3. Neurotonic hyperemia can form due to:

A) **Increase tonus of dilatators**.
B) Decrease tonus of vasoconstrictors.
C) Increase contractility of myocardium.

4. Neuroparalytic hyperemia can form:

A) By decrease tonus of parasympathetic nervous system.
B) By desympatization.
C) By paralysis of extremities.

5. Mark vessels, where thrombus can form the most frequent.

A) Big arteries.
B) Arterioles.
C) Venus.

6. **What embolus is endogenic origin?**

A) **Drops of fat by fractures of bones.**

B) Store of aerial bubbles.

C) Hemorrhage

D) Increase os potassium in cells.

7. **Show possible localization of thrombus by its detouch from vein of lower extremities**

A) Femoral vein

B) Branches of lings artery

C) Intestinal vein

8. **Why in focus of ischemia can form paleness?**

A) Increase diameter of artery and quantity of functional capillaries.

B) Increase quantity of oxyhemoglobin in functional capillaries.

C) Decrease quantity of oxyhemoglobin in focus of ischemia.

LITERATURE

1. Ovsyannikov V.G. "Pathophysiology" (questions and answers) 2005. p.57-63.

2. Nowak TI. Gordon A.G. "Essentials of pathophysiology concepts and applications for Health Care Professionals", London, Singapore, 1999, p.21-22, 29-31, 222, 247-249, 392-393.

THEME OF LESSON: DISORDER OF PERIPHERAL BLOOD CIRCULATION. THROMBOSIS, EMBOLISM

Plan of practical work:

1. Tests and discussion of theoretical material. - 60 min.

2. Fulfillment of practical work. - 60 min.

3. Discussion of results. - 15 min.

1. Scientific basing of topic. "Thrombosis means formation of blood clot inside the vessel near the wall during life" Four conditions are necessary to form thrombus: damage to blood vessel wall, delay in blood flow rate, activation of coagulative system and weakness of anticoagulative system. Initial moment in coagulation of the blood is aggregation and lysis of thrombocytes. There are a lot of aggregant factors: collagen, thrombin, tromboxane A2, adrenaline,

noradrenaline, serotonin. At the same time are activated XII, XI, IX, VIII plasmin factors and forms blood thromboplastine (prothrombinase). It is first stage of blood coagulation.

Second stage of blood coagulation is the formation of thrombin from prothrombin under the influence of prothrombinase.

Fibrinogen is converted into fibrin under the influence of thrombin in third stage of blood coagulation. There are three types of thrombus: white, red and mixed. Outcomes of thrombi are the following: Thromboembolism, lysis, canalization, organization, and calcification. Consequences of embolism depend on which part of vascular system can form thrombus. Formation of thrombus in arteries leads to the development of ischemia, in venas - venous hyperemia. "Embolism is the obturation of vessels with embolus." Embolus is foreign object which can circulate in the blood and obturate the vessels. Embolus can be in the form of thromboembolus, air gas, fat, cells.

Embolism can be classified depending on the section of vascular system where it occurs: embolism of vessels in small circle of blood circulation, in big circle of blood circulation and embolism of vena porta.

Heaviness and outcome of embolism depend on localization of emboli, state of collateral blood circulation, size of emboli and their total weight, speed of emboli beginning.

2. Aim of topic. Study etiology, pathogenesis, outcome and consequences of thrombosis and embolism.

EXPERIMENT 1. *Experimental reproduction of white thrombus.*

Aim. It is necessary to show that damage of blood vessels wall can lead to formation of thrombus.

Method. A frog is made motionless due to of spinal cord destroy by needle. The frog must be put and fixed on preparation board with aperture for the frog tongue. Take out the tongue, stretch it and fix by pins on the angles of aperture. Put the tongue under the microscope (ocular 8, objective 8). Study common picture of microcirculation, find venule, take crystal of sodium chloride and put it near the wall of venule. Study formation of white thrombus.

Results. Students describe consequences of events by formation of white thrombus and make conclusions.

Conclusions.

EXPERIMENT 2. ***Experimental reproduction of oil embolism.***

Aim. Study obturation of frog tongue vessels with vaseline oil.

Method. Experiment is carried out on a frog.

The frog is made motionless due to a needle of destroy spinal cord. The frog is fixed on the preparation board with its back down aperture for the frog tongue. The thorax of the frog is opened, pericardium is cut and the frog's heart is opened too. Take out the tongue, stretch it and fixed by pins on the angles of aperture. Study under the microscope common picture of microcirculation. Inject 0,2 ml of vaseline oil into the left ventricle and study obturation of microvessels with oil.

Results. Students describe embolism of vessels and make conclusions.

Conclusions.

3. Demonstration of educational film "Thrombosis" - 7 min.

4. Demonstration of educational film «Embolism»

QUESTIONS

1. Definition of thrombosis
2. Conditions which are necessary for the formation of thrombus.
3. Outcome of thrombus
4. Consequences of thrombosis
5. Definition of embolism
6. Classification of embolism
7. Consequences of embolism
8. General principles of prophylaxis and treatment of thrombosis and embolism.

LITERATURE

1. Ovsyannikov V.G "Pathophysiology" (questions and answers). 2005, p.63-67.
2. Novak T.J. Gordon A.G "Essentials of pathophysiology Concepts and applications for Health Care Professionals", London, Singapore, 1999, p.21-22, 29-31, 222, 247-249, 392-393.

3.

THEME OF LESSON: HYPOXIA

Plan of practical work:

1. Tests and discussion of theoretical material. - 60 min.
2. Fulfillment of practical work. - 60 min.
3. Discussion of results. - 15 min.

1. **Scientific basing of the topic.** Hypoxia is a typical process which is characterized by decrease of oxygen in the cells (tissues) or disorder of its biological utilization. Man can use oxygen which is situated in external environment. Partial pressure of oxygen on the level of the sea is equal.159 Hg mm. To come to the cells, oxygen has to pass several very important pathways. Apparatus of external respiration is the first, which can take part in gaseous exchange (oxygen and carbon dioxide between external air and the blood. Oxygen enters with air through the trachea, bronchus, bronchioles up to alveoli, where partial pressure decreases to 90-100 Hg. Oxygen diffuses up to alveolar-capillary membrane into the lungs capillary and where is combines with hemoglobin of erythrocytes and forms oxyhemoglobin (only minimal quantity of oxygen is dissolved in plasma). Cardio-vascular system transfers the blood and oxygen in form of oxyhemoglobin to organs, tissues, cells where in blood capillaries oxygen detach from hemoglobin and passes into the cells, here inside of mitochondria. Oxygen and energetic substrate are used for tissue breathing. Four final products are formed by tissue breathing. They are ATP, primary heat, water, carbon dioxide. Main products of tissue breathing are ATP and primary heat. Any biological process inside cells is not possible without ATP and organ and organism itself can die. Normal temperature, homeostasis and biochemical and physiological processes are not possible without of primary heat. Water and carbon dioxide are the waste products of the organism. To utilize oxygen and energetic substrates are necessary so called tissue enzymes. Tissue breathing is not possible by damage of enzymes of cell of mitochondria.

Deficit of oxygen can be formed in organism of healthy person. For example, after physical exercises, by pregnancy, by living on high mountains. Such hypoxia is physiological.

Deficit of oxygen and its biological utilization can appear and by pathology. Hypoxia by pathology can be classified depending on the level of inhibition of oxygen entering to the cells. It is pathogenetic classification of hypoxia by pathology:

1. Respiratory hypoxia
2. Hemic (anemic) hypoxia
3. Circulatory stagnant hypoxia
4. Tissue (hystotoxic) hypoxia

Respiratory hypoxia can develop due to the impossibility of apparatus of external breathing to supply gaseous exchange between external air and the blood. It can appear as a result of damage of the lungs itself, regulatory influences of brain, spinal cord, nerve damage, damage of the thorax and diaphragm or disorder of blood circulation in the lungs.

Hemic hypoxia can develop due to qualitative formation of carboxyhaemoglobin or methaemoglobin or by quantitative (anemia) change of hemoglobin in the blood.

Circulatory (stagnant) hypoxia can appear due to the damage of the heart (cardiac insufficiency) or disorder of peripheral blood circulation (ischemia, venous hyperemia). Stagnation of the blood can be formed by cardiac insufficiency, venous hyperemia and ischemia. That is why such hypoxia is named stagnant or circulatory.

Respiratory, hemic and circulatory hypoxia can lead to decrease in quantity of oxygen in the cells. Quantity of oxygen can be normal inside the cells but by damage of tissue enzymes, oxygen cannot be used in tissue breathing (its biological utilization is not possible).

Such hypoxia is named tissue or histotoxic.

Main result of deficit of oxygen or disorder of its biological utilization inside cells is impossibility of tissue breathing and consequently ATP, primary heat,

water and carbon dioxide are not formed. Deficit of energetic substrate can lead to the same consequences: deficit of ATP, primary heat, water and carbon dioxide. Such hypoxia is named substrate hypoxia, because oxygen cannot be used in biological oxidation without energetic substrate.

2. Aim of topic: study common etiology, pathogenesis, compensatory reactions disorder in the organism and outcomes of hypoxia.

EXPERIMENT 1. ***Experimental reproduction of hypoxic hypoxia.***

Aim. It is necessary to show that decrease in partial pressure of oxygen can lead to development of hypoxic hypoxia and it is characterized by change of electrocardiogram, rate of heart contraction, change in behavior of experimental animal.

Method. Experiment is fulfilled on an intact rat. Animal is put into a hermetic chamber. Behavior and electrocardiogram are registered in starting point. Heart rate is calculated on the basis of electrocardiogram. Chamber closes hermetically and with help of an air-pump, air is removed from the hermetic chamber. Such way we imitate lift on the mountain on the height of 5-6 km. Partial pressure of oxygen decreases and hypoxic hypoxia is formed. ECG, heart rate and change of behavior of animal is registered in dynamic of hypoxic hypoxia.

Results. Students record change in behavior of animal, heart rate and ECG in dynamic of hypoxic hypoxia, draw tables and diagrams.

Discussion. Students discuss pathogenesis of hypoxic hypoxia, mechanisms change in behavior of animal, change in ECG and heart rate and make conclusions.

Conclusions.

EXPERIMENT 2. ***Experimental reproduction of hemic (anemic) hypoxia.***

Aim. It is necessary to show that when intoxication by sodium nitrate, methemoglobin occurs, hemic hypoxia develops.

Method. Breathing rate and content of methemoglobin are determined by 5 rats in starting point. Content of methemoglobin is determined with help of photo calorimeter. Then sodium nitrate is injected under the skin by 4 rats in different doses (from 1 to 12 mg/100 g of weight).

Results.

Indexes	***Starting point***	***Sodium nitrate***			
		1 mg	***2 mg***	***5 mg***	***12 mg***
Rate of respiration (per min)	40	50	60	70	80
Content of methemoglobin (%)	0,4%	10%	20%	40%	50%

Students draw graphs of change of respiration and quantity of methemoglobin after injection of sodium nitrate.

Discussion. Students discuss change of respiration depending on intensity of hemic hypoxia and make conclusions.

Conclusions.

3. Demonstration of educational film "Hemic hypoxia". - 20 min.

QUESTIONS

1. Definition of hypoxia. Hypoxia as pathogenetic factor of different diseases.
2. Classification of hypoxia according to its origin and by pathology.
3. Oxygenation of the blood by different types of hypoxia
4. Disturbance of metabolism and physiological functions by hypoxia.
5. Etiology and pathogenesis of respiratory hypoxia.
6. Etiology and pathogenesis of circulatory hypoxia.
7. Idea about substrate hypoxia.
8. Etiology and pathogenesis of hemic hypoxia.
9. Etiology and pathogenesis of tissue hypoxia.
10. Urgent mechanisms of compensation on different levels and its origin.
11. Prolonged mechanisms of compensation.
12. Mechanism death of the cells by hypoxia.
13. Pathophysiological bases of prevention of hypoxia and treatment.

Tests

1. What is base of respiratory hypoxia?

A) Disorder gas exchange in the lungs.

B) Increase use of oxygen.

C) Decrease use of oxygen.

2. Mark correct definition of hypoxia.

A) Decrease oxygen in the blood.

B) Decrease oxygen or its utilization into the cells.

3. What is base hemic (anemic) type of hypoxia?

A) Intensive use of oxygen in the cells.

B) Decrease oxygen capacity and oxygen transport function of the blood.

C) Decrease pumping function heart.

4. What is base of circulatory hypoxia?

A) Acceleration of blood inflow to the cells.

B) Decrease of blood inflow to the cells (ischemia, venous hyperemia, cardiac insufficiency).

5. What is base of tissue hypoxia.?

A) Decrease functional capillaries.

B) Centralization of blood circulation.

C) **Disorder use of oxygen or energetic substances by tissue breathing.**

6. What type of metabolism is discorded by hypoxia first of all?

A) Water-electrolyte.

B) Carbohydrate

C) Energetic.

D) Protein.

E) Lipid.

7. Effects of what hormones can have privilege by hypoxia?

A) Insulin.

B) Somatotropin.

C) Corticotropin.

D) Contrinsular hormones.

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THEME OF LESSON: TYPICAL DISORDER OF METABOLISM.

Plan of practical work:

1. Tests and discussion of theoretical material - 60 min.
2. Fulfillment of practical work - 60 min.
3. Discussion of results - 15 min.

1. Scientific basing of topic. There are several typical disorders of metabolic processes. They are:

1. Typical disorders of energetic exchange.
2. Typical disorders of protein exchange.
3. Typical disorders of carbohydrate exchange.
4. Typical disorders of lipid exchange.
5. Typical disorders of water-electrolyte exchange.

Metabolism can be disturbed on different stages: on stage of entrance of nutrients into the organism, on stage of its utilization by the cells, on stage of its neuro-endocrine regulation, on stage of metabolite release from the organism. Deficit of nutrients can appear by starvation, which can be complete or incomplete, endogenic or exogenic. Disorder in utilization appears by damage of organs and systems and pathology of nervous and endocrine system, deficit of vitamins. Disturbance of withdraw of metabolites. Common index of disturbed metabolism can be due to change internal environment, especially pH of the blood. Shift pH of the blood, extracellular liquid and cells in acidic or basic direction can stimulate formation of the following groups of compensatory reactions:

1. Dilution.
2. Reaction of chemical buffers (bicarbonate, phosphate, protein, haemoglobin).
3. Physiological compensatory reactions
(respiration, kidneys, stomach, intestine).
4. Cellular mechanisms.

Decompensatory acidosis and alkalosis produce multiple and complex disturbances of organs and systems and can lead to death.

Very important and such homeostatic index is water-electrolyte exchange. Main manifestations by disorder of water-electrolyte exchange are dehydration and formation of edemas hemoconcentration, decrease systemic arterial blood pressure, disorder of microcirculation and haemostasis can be formed by dehydration.

Development of edemas depend on change of membrane permeability, change of hydrostatic and oncotic pressure in blood vessels, delay of lymph out flow and change of electrolyte balance.

Very important role in regulation of water-electrolyte exchange have such hormones as ADH, aldosterone, cortisol, corticosterone, parathyrin, calcitonin.

2. Aim of topic. Study possible manifestations in the organism by different disorders of metabolism.

EXPERIMENT *1. Experimental reproduction odema of the lungs.*

Aim. It's necessary to show that by very severe increase systemic hydrostatic pressure of the blood can develop odema of the lungs.

Method. Experiment is fulfilled on rat. Breathing rate is counted per minute in starting point. Then 0,2-0,3 ml adrenaline/100 g of body weight is injected subcutaneously. Breathing rate counts every 10 minutes for one hour in dynamic of lungs odema.

A rat is anaesthetized by solution (1%) of hexenalum (0,4-0,5 ml) and after decapitation is definited weight of the lungs. Ratio of lung's weight to body weight in healthy animal is equal 0,7-0,8 g to100 g.

Results. Students make table and diagram (graph) of breathing rate in dynamic of lung's odema development and compare weight of lungs of healthy animal and animal with odema.

Discussion. Students discuss results of experiment and mechanism of development of odema by increase of hydrostatic pressure of the blood and make conclusions.

Conclusions.

EXPERIMENT 2. Disturbances of functions of the organism by shift of acid-base balance.

Aim. It's necessary to show that compensatory and decompensatory acidosis can lead to different change of ECG and heart's rate.

Method. Experiment is fulfilled on anaesthetized rat (0,4-0,5 hexenalum subcutaneously). A rat is fixed on preparation board.

ECG and heart's rat on the basis of ECG is registered in starting point. Then 0,5 ml 0,5 % of lactic acid is injected into v. jugularis. ECG and heart's rate is registered.

To produce decompensatory acidosis 2 ml, 0,5 ml of lactic acid is injected into vena jugulars and the same indexes are recorded in dynamic of acidosis (2°. 5°. 10 and 20 minutes after injection of lactic acid.

Results. Students make table and graph on change of ECG and heart's rate by compensatory and decompensatory acidosis.

Discussion. Students discuss results of experiment, compensatory reactions and manifestations by compensatory and decompensatory acidosis and make conclusions.

Conclusions.

3. Demonstration of educational film "Change reactivity of the organism by shift of acid base balance" - 15 min.

QUESTIONS

1. Etiology of energetic metabolism disorders.
2. Compensatory reaction on cellular level by deficit of ATP.
3. Possible compensatory reaction by deficit of ATP on the level of systems and organism itself.
4. Name four main final products which are formed by tissue breathing.
5. Main consequences of energy metabolism disorder.
6. Etiology of disorders of carbohydrate metabolism.
7. Disorder of what processes in gastro-intestinal can lead to derangement of carbohydrate metabolism.
8. Possible consequences of carbohydrate starvation.
9. Compensatory reactions by carbohydrate deficit.
10. Main contrinsular hormones and its effects.
11. Etiology of hypoglycemia.
12. Main mechanisms of hypoglycemia.

13. Main consequences of hypoglycemia.

14. Etiology of hyperglycemia.

15. Consequences of hyperglycemia.

16. Etiology of disorder of protein hydrolysis in gastrointestinal tract.

17. Consequences of derangement of digestion and absorption of proteins.

18. Consequences of excessive protein synthesis.

19. Main biochemical reactions of amino acids conversion.

20. Types of hyperazotemia.

21. Alternative paths of nitrogen release from the organism.

22. Etiology and mechanisms of disorder of lipolysis and absorption of lipids in intestine.

23. Main consequences of disorder lypolysis and absorption of lipids in intestine.

24. Etiology of hyperlipidemia.

25. Etiology of obesity.

26. Etiology and mechanism of decrease body weight (emaciation).

27. Etiology of ketonemia.

28. Mechanism of ketonemia.

29. Etiology of metabolic acidosis.

30. Etiology of gaseous acidosis.

31. Etiology of metabolic acidosis.

32. Etiology of gaseous alkalosis.

33. Compensatory reactions by shift of acid-base balance.

34. Manifestations by decompensatory metabolic acidosis.

35. Manifestations by decompensatory gaseous acidosis.

36. Manifestations by decompensatory metabolic alkalosis.

37. Manifestations by decompensatory gaseous alkalosis.

38. Indexes of acid-base balance.

Tests

Topic: Typical disorders of metabolism.

18. Main mechanism disorder energetic exchange.

A) Stabilization membranes of lysosomes.

B) Activation of RNA.

C) Disorder coupling between oxidation and phosphorylation.

19. Show mechanism disorder splitting carbohydrates.

A) Increase proteolytic enzymes.

B) **Deficit of hydrolysis.**

C) Increase activity of lipases.

20. Main etiological factor of hyperglycemia**.**

A) Excess of insulin.

B) Deficit of of contrinsular hormones.

C) Deficit of insulin, prevailing effect of contrinsular hormones.

21. Show final **substances of** protein metabolism.

A) Acetone.

B) Ammonia, urea, glutamine.

C) Aminoacids.

22. Main path inactivation of ammonia in Kidneys.

A) Connection with proteins.

B) Ammoniogenesis.

C) Interaction with lipids.

23. By insufficiency of what organs can form hyperazotemia?

A) Insufficiency of the lungs.

B) Insufficiency of the Heart.

C) **Insufficiency of kidneys and liver.**

24. When can form disorder digestion and absorption of proteins in gastrointestinal tract.

A) Increase secretion of hydrochloric acid.

B) Secretion of gastromucopronein.

C) Decrease secretion of pepsin, gastricsin, trypsin, HCL.

25. What mechanism is base disorders of splitting and absorption of lipids?

A) Deficit of amilasa.

B) Insufficiency emulsification of lipids.

C) Excess of peptidasis.

26. What mechanism does have important role in development of atherosclerosis.

A) Hyperpoteinemia.
B) **Hyperlipoproteinemia**.
C) Reflectory.

27. What vessels are damaged by atherosclerosis?

A) Venas.
B) **Musculo-elastic vessels**.
C) Capillaries.

28. When can form mobilization of fat from fat depots?

A) By hyperlipidemia.
B) By decrease quantity of glucose in the blood.
C) By hypoproteinemia.

29. What components does ketone bodies consist of?

A) Lipids.
B) Lipid acids.
C) Aceton, aceto-acetic acid, Beta-oxibuthiric acid.

Topic: Disorder of acid-base balance.

30. Mark physiological level pH of the blood.

A) 7,5-8,0
B) 7,36-7,44
C) 6.0-7,0

31. By what process can form metabolic acidosis?

A) Hypoxia.
B) Renal insufficiency.
C) Hepato-cellular insufficiency
D) Cardiac insufficiency.
E) All above mentioned.

32. Show endocrine disorders, by which can form alkalosis.

A) Hyperthyroidism.
B) Hypothyroidism.
C) Primary aldosteronism

33. What function in buffer system of the blood can fulfill hemoglobin?

A) Base.
B) Acid**.**

34. How can change reaction of urine by respiratory alkalosis?
A) It is not change.
B) Urine can be alкaline.
C) It can be acidic.

35. What is acidosis?
A) **Excess of hydrogen ions**.
B) Excess of bases.

36. What is alkalosis?
A) Excess of acids.
B) Deficit of bases.
C) Deficit of hydrogen ions.

37. Patient has pH of the blood 7,2. Mark type disorder of acid – base balance.
A) Compensatory alkalosis.
B) Decompensatory alkalosis.
C) Compensatory acidosis.
D) Decompensatory acidosis.

38. Patient has pH of the blood 7,7. Mark type disorder of acid – base balance.
A) Compensatory alkalosis.
B) Compensatory acidosis.
C) Decompensatory alkalosis.

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1. Ovsyannikov V.G. "Pathophysiology" (questions and answers), 2005, p. 73-89.

2. Nowak Th., J., Handford A.G. "Essentials of pathophysiology. Concepts and applications for Health Care Professionals", London, Singapore, 1999, p.281, 353-355, 419-425, 227.

THEME OF LESSON:

PATHOPHYSIOLOGY OF PAIN.

Plan of practical work:

1. Tests and discussion of theoretical material - 60 min.

2. Fulfillment of practical work - 60 min.

3. Discussion of results - 15 min.

1. Scientific basing of topic. "Pain is a typical evolutionary formed pathological process, which appears by influence on the organism damaging (nociceptive) factors or weakness of antinociceptive system and characterized by perception, vegetative, emotional, behavioral, motor reactions and compulsory activation of antinociceptive system, directed on disappearance of pain and restoration of damaged focus of organ or tissue".

All factors which can produce pain are named algogenic or nociceptive. They are classified on exo- and endoalgogens.

Mechanical physical, chemical, stress, hypoxia, deficit of information through thick myelin fibers are exogenic algogens. Substance P, histamine, serotonin, kinins, prostaglandins are endogenic nociceptive factors.

Trauma, inflammation, damage different sections of central nervous system, ischemic heart disease, myositis, radiculitis, metastasis of tumors are the most often causes of pain in patients. Localized and nonlocalized pain in patients. Localized and nonlocalizad pain is distinguished by formation of acute pain. They have not only different paths for conducting pain impulses, involvement different sections of central system, but and manifestations. Antinociceptive system is excited first of all by conducting pain impulses through paleospinothalamic tract.
Antinociceptive structures of the brain (back horn, giant nucleus, raphe nucleus, aquaductus gray, hypothalamus) and humoral antinociceptive mechanisms (opiate adren-, serotonin, - cholinergic systems) are excited.

By pain excited all organs and systems and as a result can form different manifestations:

1. Main reactions (jerk-back, jump back, as a spinal reflex;
escape of damaging factors, as conditional reflex);

2. Activation of antinociceptive system structures of the brain and humoral

(antinociceptive antinociceptive mechanisms);

3. Vegetative reaction (tachycardia, angiospasm, increase systemic arterial blood pressure change quantity of leukocytes and its phagocytic activity, coagulation of the blood, metabolism);

4. Change of respiration (delay, tachypnoe);

5. Emotional reactions (crying, terror, aggression).

Origin of such manifestations students can understand on the basis of pathogenesis knowledge.

2. Aim of topic. Study etiology, pathogenesis, manifestations and common principles of pain treatment.

EXPERIMENT *1. Experimental reproduction of pain.*

Aim. Study of motor, emotional and behavioral reactions by pain.

Method. Rat puts into special chamber the floor of which is metallic and connected with electrostimulator. Students study behavior of rat in starting point. Then electrostimulator is switched on (tension is equal 220 Volts) during one second and study motor, behavior and emotional reactions of rat by pain.

Results. Students describe change motor, behavior and emotional reactions by pain.

Discussion. Students discuss origin of motor, behavioral and emotional reactions by pain and make conclusions.

Conclusions.

EXPERIMENT 2. *Change reactions on pain depending on pain thresh hold.*

Aim. Study reactions on pain depending on strength of irritator by intact animal and anesthesia with nembutalum.

Method. Rat puts into chamber floor of which metallic and connected with electrostimulator. Electroctimulator is switches (power 0,50 A, tension 5 Volts, duration of impulses - 1 sec) and

reactions of animal study in starting point. Then only tension increases up to 10 V, 30V and notice change behavioral reactions of animal. Animal is anesthetized of 2 ml 2 % solution of nembutalum and after 10 minutes the same researches are repeated.

Results. Students make table and analysis results.

Discussion. Students discuss results, try to explain and make conclusions.

Conclusions.

3. Demonstration of educational film "Reproduction of neuropathic syndromes" **-** 20 min.

QUESTIONS

1. Definition of pain.
2. Etiology of pain.
3. Classification of pain.
4. Pathogenesis of acute pain.
5. Pathogenesis of chronic pain.
6. Differences between acute and chronic pain (etiology, pathogenesis, duration and manifestation).
7. Motor reactions by pain and it mechanisms.
8. Vegetative reactions by pain and its mechanisms.
9. Emotional reactions by pain and its mechanisms.
10. Structure of pain and antinociceptive system.
11. Mechanisms activation of antinociceptive system by pain.
12. Biological meaning of pain.
13. Common principles of pain's treatment.

Tests

1. Mark term, which means increased Pain sensitivity.

A) Hypersensitivity.
B) Hyperalgesia.
C) Placebo.

2. Mark changes by Pain, leading to increase arterial blood pressure.

A) Decrease common peripheral resistance.
B) Bradycardia.
C) Tachycardia and increase general peripheral resistance.

3. Localized somatic Pain occur by pass Impulses along:

A) C- fibers.
B) **A-delta fibers**.

C) A-beta fibers.

4. Increase systemic arterial pressure by somatic Pain connect with:

A) Activation parasympatho-nervous system.

B) Activation sympatho-adrenal system.

C) Stimulation betta-cells of Pancreas.

5. Increase quantity of leucocytes in circulated blood by somatic Pain occurs due to:

A) Hypothyroidism.

B) Hyperglycemia.

C) Activation of sympatho-adrenal system.

6. What term is used for etiology of stress?

A) Adaptogen.

B) Algogen.

C) Stressor.

7. What hormone can stimulate cortex of adrenal glands?

A) Corticoliberin.

B) **Corticotrophin (corticotrophin)**.

C) Vasopressin.

8. What system does fulfill leading role in development of Stress?

A) Hypothalamus-hypophysal-ovarial .

B) **Sympatho-adrenal.**

C) Hypothalamo-neurohypophysal

9. What effect can produce glucocorticoids by stress.

A) Stimulation gluconeogenesis.

B) Stimulation of glycogenolysis (permissive effect).

C) Disorder coupling between oxidation and Phosphorylation.

10. What effect can produce Stress on immunity?

A) Stimulation.

B) **Inhibition (immunodeficiency state).**

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2. Nowak Th.J., Handford A.G. "Essentials of pathophysiology. Concepts and applications for Health Care Professionals" London, Singapore, 1999, p.631-649.

THEME OF LESSON: SHOCK.

Plan of practical work:

1.Tests and discussion of theoretical material. - 60 min.

2.Fulfillment of practical work. - 60 min.

3. Discussion of results. - 15 min.

1**. Scientific basing of topic**. "Shock is a typical pathological process, which can form by influence on the organism by extreme severe damaging factors and is characterized by disorder of systemic arterial blood pressure, microcirculation and systems".

Etiologic factors which can produce shock - mechanical trauma, loss of much quantity of blood, influence of high and low temperature, and release of a lot of endotoxins by infection, neurosis of myocardium, immunologic conflict. Three main pathogenetic stages can be distinguished: disorder of neuro-endocrine system, disorder of cardio-vascular system, disorder of metabolism.

Stage of excitation (erectile stage), stage of inhibition (torpid stage) and terminal stage are distinguished clinically.

Stage of excitation can form due to very intensive pain formation. That is why this stage develops by traumatic, burning, cardiogenic, cold shock and practically absent by septic, anaphylactic, posthemorrhagic shock. Main pathogenic chain of Shock is excessive pain and not pain information in central nervous system. This can lead to involvement of all endocrine glands (adrenal system, hypothalamus - hypophysis, ad System and so on), change of systemic arterial blood pegs (increase in stage of excitation and decrease in stage of inhibit due to hypotension decreases perfusional pressure in all organs ay generalized hypoxia is formed. Main result of such changes is disorder of all types of metabolism (energetic, carbohydrate, protein, lipid, water-electrolyte). All phenomena of disorder of peripheral blood circulation (ischemia, hyperemia, thrombosis and embolism) and microcirculation (change in diameter of blood vessels, increase in permeability, aggregation of blood cells, sludges, centralization

of blood circulation, plasmatic vessels) and development of thrombo-hemorrhagic syndrome can occur as a result of shock.

2. Aim of lesson. Experimental reproductions of shock and discussion of its etiology and pathogenesis.

EXPERIMENT. *Change of systemic arterial blood pressure and respiration by anaphylactic shock*

Aim. Study change of arterial blood pressure and rate of respiration by shock

Method. Experiment is fulfilled on a dog. First animal is sensitized with horse serum three times 0,3 ml/kg of weight at intervals 24 hours (intramuscular, intravenously, intramuscular). After 2-3 weeks animal is fixed on table. Systemic arterial blood pressure and rate of breathing (make records of the pneumogram) are measured. Then horse serum is injected intravenously in dose 0,3 ml/kg of weight.

Make records of systemic arterial blood pressure and respiration in dynamics of shock.

Results. Students draw table and diagram of change in arterial blood pressure and rate of respiration.

Discussion. Students discuss mechanism of change in arterial blood pressure and rate of breathing by shock).

Conclusions. Students have to make conclusion on the bases of results).

3. Demonstration of educational film "Disorder of microcirculation by anaphylactic shock" (20 min).

QUESTIONS

1. Definition of shock
2. Etiology of shock
3. Pathogenesis of shock
4. Pathogenetic stages of shock
5. Clinical stages of shock
6. Clinico-laboratory manifestations of shock in stage of excitation.
7. Clinico-laboratory manifestations of shock in stage of inhibition.

8. Mechanism of increase and decrease of systemic arterial blood pressure by shock.

9. Classification of shock depending on change in systolic and diastolic arterial blood pressure.

10. Common principles of shock treatment.

Tests

1. What is main chain of shock:

A) Circulatory hypoxia.
B) **Excessive afferentation**.
C) Increase formation of enkephalines and endorphins.

2. What type of hypoxia can develop by shock?

A) Respiratory.
B) Anemic.
C) Cardio-vascular.
D) Mixed.

3. What pathological types of breathing can develop by shock.

A) Chain-Stoks.
B) Biot.
C) Kussmaul.
D) All above mentioned.

4. What type of metabolism can be disordered first of all by shock?

A) Protein.
B) Lipid.
C) **Energetic**.
D) Carbohydrate.

5. What type of shock can develop the most often?

A) Anaphylactic.
B) Septic.
C) Traumatic.

6. **Show first clinical phase of shock:**

A) Torpid phase
B) Terminal phase
C) Erectile phase

7. **Show first pathogenetic stage of shock:**

A) Metabolic stage

B) Stage of cardio-vascular disorders

C) Stage of neuro-endocrine disorders

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THEME OF LESSON: INFLAMMATION

Plan of practical work:

1. Tests and discussion of theoretical material. - 60 min.
2. Fulfillment of practical work. - 60 min.
3. Discussion of results. - 15 min.

1. Scientific basing of the topic. "Inflammation is a typical pathological process formed during evolution. It is a local reaction of organism as a whole on damaging factors and is characterized by damage of the cells, disorder of its metabolism, microcirculation, exudation, emigration of leukocytes and proliferation of cells"

Inflammation occurs only in vascularized tissues and organs. Two conditions are necessary: damage of cells and reaction of organism on such damage.

Local damage can be produced by exogenic flogogens (mechanical, physical, chemical, infections, immunological conflict) and endogenic (thrombosis, embolism, store of salts). By inflammation, three groups of symptoms can be distinguished: common clinical, morphological and physiochemical.

Pathogenesis of inflammation can be presented schematically as following:

By damage of cell, metabolism of cells is disordered and some physio-chemical symptoms develop (hyperkalemia, local acidosis, increase in osmotic and oncotic pressure and the most important is the store of biological active substances

mediators of inflammation). Mediators have different origin and nature (cell and plasma derived).

Cell derived: mast cells and basophiles (histamine, prostaglandins, leukotrienes, nitric oxide, cytokines, chemakines, and platelet activation factor).
Plasma derived: fibrinolytic system (products of splitting fibrin - fibrinogen), kinin system (kinin, bradykinin), complement system (proteolytic enzymes, opsonins, ation, anaphylotoxins C3a C5a).

All above mentioned mediators have different biological effects: change in diameter and permeability of microvessels, pain, chemotaxis and coagulation of blood, degranulation of mast cells and basophiles, stimulation of phagocytosis, damage of cells, lysis, and fever.

Such effects allow to understand secondary alteration, disorder of blood circulation and microcirculation in focus of inflammation. They are the following.
First stage: angiospasm during short period and development of arterial hyperemia (in microcirculatory net, increase in diameter of arterioles, linear and volumetric blood inflow, increase in quantity of functioning capillaries and hydrostatic pressure are noticed).

Second stage- development of venous hyperemia (in microcirculation net are noticed delay of lineal and volumetric blood flow rate, push-like and balance like movement of blood, increase in hydrostatic pressure; third stage - stasis.

Increase in permeability allows escape of the liquid part of blood into inflamed region (exudation). There are four mechanisms of exudation: increase in permeability, hydrostatic, osmotic and oncotic pressure in focus of inflammation. Due to chemotaxis can occur emigration of leukocytes. Main functions of leukocytes are: phagocytosis of damaged cells, clearing focus of inflammation, stimulation of proliferation of the cells, stimulation of immunity and release of cytokines.

There are three possible outcome of acute inflammation: complete restoration of damaged focus, formation of scar, death.

2. Aim of lesson. Study common etiology, pathogenesis and outcome of acute and chronic inflammation.

EXPERIMENT 1. Disorder of blood circulation in focus of inflammation. (Experiment of Kongeim)

Aim. Study consequence of events in microvessels and microcirculation in focus of inflammation.

Method. Frog is anesthetized with nembulatum (0,1-0,2 ml 2% solution) After 7-10 min. frog is fixed backup on special table with a hole for mesenterium near of abdomen. Skin from axillary line is out and mesentery stretches over the hole and fixed. Inflammation develops due to damage of cells of mesentery.

Study under the microscope (Ocular 8, objective 10 and 40) change of blood circulation, microcirculation and emigration of leukocytes in dynamic of inflammation.

Results. Students describe change of blood circulation, microcirculation and emigration of leukocytes by inflammation.

Discussion. Students discuss results of experiment and make conclusion.

Conclusions.

EXPERIMENT *2. Microscopic study of pus.*

Aim. Study components of pus.

Method. Make smear of pus, fix it and stain by Romanovsky-Giemsa. Study pus under the microscope under the immersion.

Results. Students describe components of pus.

Discussion.

Conclusions. Students make conclusions about components of pus.

EXPERIMENT 3. *Study proteolytic activity of pus.*

Aim. Study possible increase in proteolytic activity of pus.

Method. Take 8 tests tube with 1 ml of egg protein diluted 150-200 fimes. Then in 7 test - tubes add drops of pus except of 8^{th} test-tube (1, 3, 5, 7, 9, 11, 13). In all test tubes add drops of physiological solution except 7^{th} (12, 10, 8, 6, 4, 2, - 13) (tabl.). All test-lubes are put in thermostat for 30 minutes.

After 30 min add in all test-tubes 2 drops 20% of sulfosalicylic acid and according to intensity of turbidity we can say about proteolytic activity of pus.

Reagent	Test-tubes

	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**
Egg protein (ml)	1	1	1	1	1	1	1	1
Pus (drops)	1	3	5	7	9	11	13	-
Physiological solution (drops)	12	10	8	6	4	2	-	13

3. Demonstration of educational film "Inflammation" 20 min.

QUESTIONS

1. Definition of inflammation.
2. Conditions necessary for development of inflammation.
3. Etiology of inflammation.
4. Classification of inflammation.
5. Common clinical symptoms of inflammation and its Latin names.
6. Morphologic symptoms of inflammation.
7. Physio-chemical symptoms of inflammation.
8. Pathogenesis of acute inflammation.
9. Mediators of inflammation.
10. Common biological effects mediators of inflammation.
11. Disorder of blood circulation and microcirculation in focus of inflammation.
12. Exudation and its mechanisms
13. Emigration of leukocytes and its mechanisms.
14. Proliferation of cells and its mechanisms in focus of inflammation.
15. Healing by primary intention.
16. Healing by secondary intention.
17. Outcomes of acute inflammation.
18. Role of reactivity in the development of inflammation.
19. Common effects of inflammation.
20. Etiology and pathogenesis of chronic inflammation "Types of chronic inflammation".
21. Biological significance of inflammation.

39.Mark process, which increase oncotic pressure in focus of Inflammation.

A) Increase synthesis of carbohydrates.

B) Decries synthesis of proteins.

C) Damage cells and increase quantity of proteins in focus of Inflammation.

Tests.

1. How can form emigration of leucocytes in focus of inflammation?

A) **Active**.
B) Passive.

2. Why can form redness in focus of inflammation?

A) **Due to development of arterial hyperemia**.
B) Due to venous hyperemia.
C) Due to depot of venous blood.

3. Mark process, which produce heat in focus of inflammation.

A) Increase cellular breathing.
B) Decrease metabolism.
C) Depot venous blood.
D) Centralization of blood circulation.

4. Mark influence of sympato-adrenal system on inflammation.

A) Don't influence.
B) Acceleration.
C) **Inhibition**.

5. Mark influence of parasympathic nervous system on inflammation.

A) Inhibition.
B) **Acceleration**.
C) Doesn't influence.

6. By what types of inflammation is the most intensive emigration of leucocytes?

A) Proliferative.
B) Serous.
C) Fibrinous.
D) **Purulent.**

7. What processes are the most intensive by chronic inflammation?

A) Exudative.
B) Alterative.

C) Proliferative.

8. What leucocytes can produce proteolytic enzymes?

A) Eosinophils.
B) Basophils.
C) Lymphocytes.
D) Neutrophils.

9. What process can be used for treatment of inflammation?

A) Ischemia.
B) **Arterial hyperemia**.
C) Venous hyperemia.

80. Development of Fever by inflammation can form due to release:

A) Exogenic pyrogens.
B) **Endogenic pyrogens**.
C) Serous exudate.

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THEME OF LESSON: FEVER.

Plan of practical work:

1.Tests and discussion of theoretical material. - 60 min.

2.Fulfillment of practical work. - 60 min.

3.Discussion of results. - 15 min.

1. Scientific basing of topic. "Fever is a typical pathological process which has formed during evolution. It appears after the influence of high molecular substances (pyrogens) and is characterized by increase in body temperature due to active change of thermoregulation on higher level' Fever forms as defense reaction on influence first of all infectious agents, which have endotoxines, main component of it are lipopolysaccharides.

Endogenic pyrogens are formed and by infectious and noninfectious inflammation.

Two cells (polymorphonuclear cells and macrophages) can synthesize endogenic pyrogens (IL-1,6, TNF, macrophage inflammatory protein). Endogenic pyrogens go with flood to the center of thermoregulation (preoptic zone of hypothalamus) and stimulate synthesis of prostaglandins El E2. The last change sensitivity center of thermoregulation and normal body temperature is accepted as decreased. Cells function of set-point center of thermoregulation is changed and sympathoadrenal and hypothalamus-hypophysis-adrenal system are activated. Main result of such activation is increase in heat production and decrease in heat loss. Body temperature is elevated. Such way can form first stage of fever - increase body temperature. When increase body temperature reaches those which has programmed under the influence of prostaglandins it accepted as normal. Heat initially increases due to vasodilatation of skin vessels. The second stage of fever - keeping body temperature on increased level - forms balance heat production and heat loss. Later, due to activation of specific and non-specific defense reactions of the organism decreases quantity of endogenic pyrogens and prostaglandins in center of thermoregulation and the organism turns to mechanisms directed on decrease heat production and increases heat loss and body temperature decreases to normal.

Such way can form third stage of fever. Knowledge of fever allows to understand etiology mechanisms of its development and treatment. That is very important for understanding infections and non-infectious pathology.

2. Aim of lesson. Experimental reproduction of fever and study of its etiology and pathogenesis.

EXPERIMENT. *Experimental reproduction of fever and study of some pathogenetic chain.*

Aim. Study of rectal, skin temperature, rate of breathing, heart contraction and quantity of leukocytes in dynamic of fever.

Method. In starting point rectal and skin temperature are measured with an electrothermometer. Count the cardiac (on the basis of electrocardiogram) and respiratory rate per min. Quantity of leukocytes is counted in the blood.

Pyrogenalum is injected into marginal auricule ear vein of the rabbit (10mg/kg of weight)

All above mentioned indexes are measured in dynamics of fever (20; 40; 1h; 2h; 3h)

Results. Students have to draw a table and make diagrams of the changes in body and skin temperature, heart and respiration rate, change in the quantity of leukocytes.

Discussion. Students have to discuss results in comparison with theoretical knowledge of fever pathogenesis).

Conclusions. Students have to make conclusions on the basis of results)

QUESTIONS

1. Definition of fever
2. Etiology of fever
3. Stages of fever depending on the change of body temperature and the most impressible clinical manifestations.
4. Change of heat production and heat loss in different stages o fever
5. Mechanism of increase body temperature.
6. Mechanism of keeping body temperature on increased level.
7. Mechanism of decrease body temperature.
8. Disturbance of vital body functions of the organism (nervous, endocrine, respiratory, cardio-vascular, gastro-intestinal tract, systems metabolism).
9. Biological significance of fever (positive, negative.
10. Common principles of fever treatment (etiotropic, pathogenetic).
11. Main differences between fever and hyperthermia.

Tests

1. Can organism to regulate heat balance by fever?

A) Yes.
B) Not.

2. Decrease heat loss can form by:

A) Dilatation of skin vessels.
B) **Angiospasm of skin vessels**.

C) Decrease of perspiration.

3. Mark the source of endogenic pyrogens.

A) Viruses.
B) Rickettsia.
C) Neutrophils and macrophages.

4. In what stage of fever thermoregulatory center accept temperature of inflow blood as normal?

A) First stage.
B) **Second stage**.
C) Third stage.

5. How can change heat loss in third stage of fever?

A) It is not change.
B) Increases.
C) Decreases.

6. What complication in third stage of fever can develop?

A) Dispnoe.
B) Intensive perspiration (sweating).
C) Collapse (decrease arterial blood pressure).

7. How can change blood flow in vessels of skin in first stage of fever?

A) It is not change.
B) Increases.
C) Decreases.

8. How can change diuresis in second stage of fever?

A) **Decrease**.
B) Increases.
C) Absence of changes.

9. How can change thermoregulation by fever?

A) It is disordered.
B) Decreased.
C) Reconstruction on new more high level.

10. How can change heat loss in first stage of fever?

A) **Decreases**.

B) Increases.

11. When by fever can form trembling (shivering).

A) By aseptic inflammation.
B) **By infectious inflammation**.
C) By hypoxia.

12. How can change frequency of pulse in second stage of Fever?

A) It is not change.
B) Increase.
C) Decrease.

13. Perspiration can form by fever in:

A) First stage.
B) Second stage.
C) Third stage.

14. What functions of lymphnodes are barriers?

A) Fixation of antigens in tissue of regional lymph-nodes and development of inflammation.
B) Contractile activity.

15. Mark barrier structures.

A) Bones.
B) Thymus.
C) Muscles.
D) **Lymphnodes.**

16. Mark the most powerful humoral factor of nonspecific resistance.

A) Lysozyme.
B) Complement.
C) Properdin.
D) Betta-lysine's.

17. Show reasons (causes) disorders of phagocytosis due to Its suppression.

A) Excess of blocking antibodies.
B) Deficit of transfer factor.
C) Deficit and excess thyrotropin.

18. Mark bactericidal effect mucosa of gastro – intestinal and respiratory tracts.

A) Glucuronic acid.
B) T-lymphocytes.
C) Secretory immunoglobulins A.
D) Must cells.

19. Mark physiological and pathological acts, directed on removal toxic and infectious substances.

A) Vomiting.
B) Cough.
C) Perspiration.
D) All factors.

20. Show cooperation of cells, which are necessary for antibody genesis.

A) Macrophage + T-helper.
B) Macrophage + T- helper + B –lymphocyte.
C) Macrophage + B-cell + mast cell.

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PART II

PATHOPHYSIOLOGY OF ORGANS AND SYSTEMS

THEME OF LESSON: POSTHEMORRHAGIC SYNDROM

Plan of practical work

1. Tests and discussion of theoretical material - 60 min.
2. Fulfillment of practical work - 60 min.
3. Discussion of results - 15 min.

1. Scientific basing of topic. Blood has a very great role in the vital activities of the organism. It takes part in transport nourishment, respiration, metabolism, defense functions. Normal volume of the blood is a very important condition for vital activity of the organism.

Quantity and volume of the blood is rather constant for any individuum and depends on mass of the body. Quantity of the blood changes from 6 to 9 % of body's weight or in average 80-85 ml/kg.

Normal quantity of the blood is named normovolemia. Decrease is hypovolemia. Increase in quantity of the blood is hypervolemia. The blood consists of two main components: plasma and blood cells (erythrocytes, leukocytes, thrombocytes). Ratio of its 52-59 % to 48-41 %. Normal quantity of blood cells is named normocythemia.

Decrease in quantity of the blood cells is named oligocythemia, and increase is polycythemia.

Change volume of the blood can occur most often by acute hemorrhage. Loss of 200-400 ml of the blood doesn't lead to change in systemic arterial blood pressure. Loss of 0,7 l and more leads to decrease in systemic arterial blood pressure. It was found that loss of 30% of the blood is lethal without treatment. Loss of 50-60 % of the blood is absolutely lethal. Acute bleeding produces disturbance of organs and systems, but compensatory reactions are activated. There are four main stages of compensation: reflex, hydremic, protein and bone marrow. All of these reactions are directed on restoration of quantity and quality of the blood, that is to say volume of the blood, proteins, and blood cells. Volume of the circulating blood is restored due to reflex and the hydremic stage of compensation. Extracellular liquid enters into the blood and produces dilution and as a result forms acute posthemorrhagic anemia.

Anemia is characterized by decrease of hemoglobin and erythrocytes in the blood.

2. Aim of topic. Reproduction of acute bleeding and study changes of the blood caused by acute posthemorrhagic anemia.

EXPERIMENT *1. Acute posthemorrhagic anemia.*

Aim. Study changes of the blood by acute posthemorrhagic anemia Study quantity change of erythrocytes, hemoglobin, reticulocytes on third-fifth day of posthemorrhagic anemia.

Method. Experiment is fulfilled on a rat. The rat is anesthetized (0,4 ml 2% solution of nembutalum) and fixed on preparation board.

Then femoral artery are found operatively and produces bleeding (30%). 3-5 days later the blood takes from the rat's tail to count quantity of erythrocytes, hemoglobin, changes of color coefficient, form and size of erythrocytes. One smear stains by Romanovsky-Giemsa for studying under the microscope size, form, color of erythrocytes. Second smear stains supervitally brilliantkresilbau to count under the microscope reticulocytes.

There are special automatic apparatus (Avdia) which allow to count automatically quantity of erythrocytes, hemoglobin, leukocytes (granulocytes, monocytes, lymphocytes), thrombocytes. (See below)

WBC - quantity of leukocytes ($4\text{-}9\times10^9$/l)

RBC - quantity of erythrocytes ($3.7\text{-}5.0\times10^{12}$ /l)

HGB - hemoglobin (11-16.1 g/dl)

HCT - hematocrit (35.0-50.0%)

PLT - thrombocytes (150-390×103 /l)

PCT - volume of thrombocytes (throbocytocrit) (0.15-0.40%)

MCV - medium volume of erythrocytes (Hb/quantity of erythrocytes)

MCH - medium quantity of hemoglobin in erythrocytes (color coefficient)

MCHC - medium concentration of hemoglobin in erythrocyte

RDW - index of anisocytosis (increase tendency to macrocytosis, decrease-tendency to microcytosis)

MPV - medium platelet's volume

PDW- index of anisocytosis of thrombocytes

%LYM - % lymphocytes and absolute quantity/ in the blood (17- 48%)

%MON - % monocytes and absolute quantity/ in the blood (4-10%)

% GRA - % granulocytes and absolute4 quantity/l in the blood (43-76%)

Method of erythrocytes count. Erythrocytes are counted in Goriaev's chamber. The blood is collected in a red blood blender up to sign of 0.5 and is diluted by physiologic solution to the sight of 101 and is shaken 2 min. 1-2 drops of contents is moved away and the rest content places in Goriaev's chamber. Erythrocytes count in 5 large squares. Special formula is used for the count quantity of erythrocytes.

(See below)

$$\mathbf{Er = a \times 4000 \times 200/80,}$$

a - sum of erythrocytes in five of large squares;

1/4000 - volume of little square in mm;

1/200 - a degree of blood dilution;

80 - the quantity of the counted little square.

Method of staining smear of the blood for the count of reticulocytes.

Primary prepares smear of brilliant-cresil-blue smear. Then on this smear makes blood's smear and puts on 10 min in humid chamber. Smear dries and studies under the microscope under the immersion. 100 erythrocytes is counted and is defined percentage of reticulocytes. Reticulocytes differ from erythrocytes by presence in cytoplasm granules and filaments (substance of granulofilamentosa)

Method of hemoglobin's determine. (colometric method). Hemometer of Sally is used for determination of hemoglobin by colometric method. 0.1 solution of hydrochloric acid is put in a graduated test-tube up to circular mark. Then 20 mm of the blood put in the same test-tube and during 5 min there is formed hydrochloride hematin. Add drops of distillated water into test-tube and mix it, allow getting color in test-tube with control-tube. Test-tube will show quantity of hemoglobin in mm.

Results. Students record change quantity of erythrocytes, hemoglobin, reticulocytes, size, form and color of erythrocytes. (See below in table.)

Table

Change in the blood by acute posthemorrhagic anemia

Indexes	Just after acute hemorrhage	2 days after acute hemorrhage	3 days after acute hemorrhage
Quantity of erythrocytes	Normal	Decreased	Decreased
Quantity of hemoglobin	Normal	Decreased	Decreased
Color coefficient	0.9	0.7	0.7-0.8
Volume of the blood	Hypovolemia	Hypovolemia	Normovolemia
Volume index	non changed	Decreased	Decreased
Size and form of erythrocytes	Isocytosis	Anisopoikilocytosis	Aniso-, poikilocytosis
Cells of regeneration (reticulocytes)	0.5-1.0%	2%	8% (reticulocyticrisis)
Bilirubin	non changed	non changed	non changed

Discussion. Students discuss results and make conclusions.

Conclusions.

3. Demonstration of educative film: "Acute hemorrhage"

QUESTIONS

1. Change volume of the blood: hypo-, hypervolemia, types, causes, mechanism of development.
2. Acute bleeding and posthemorrhagic syndrome.
3. Leading pathogenetic chain of posthemorrhagic shock.
4. Mechanism disorder of respiration by bleeding.
5. Mechanism disorder of nervous and endocrine system by hemorrhage.
6. Mechanism disorder of liver, kidneys by hemorrhage.
7. Compensatory reactions by posthemorrhagic syndrome.

8. Characteristic of reflex stage of compensation by posthemorrhagic syndrome.

9. Characteristic of hydremic stage of compensation by posthemorrhagic syndrome.

10. Characteristic of protein stage of compensation by posthemorrhagic syndrome.

11. Characteristic of bone-marrow stage of compensation by posthemorrhagic syndrome.

12. Principles of treatment for acute hemorrhage.

Tests

1. Mark factors, which can influence on outcome of hemorrhage.

A) Volume hemorrhage.
B) Speed (rate) hemorrhage.
C) Reactivity of the organism.
D) All above mentioned.

2. Mark phase of compensation by acute hemorrhage.

A) Cardiac.
B) Reflectory.
C) Cerebral.

3. When after hemorrhage extra cellular liquid can enter in the capillaries the most intensive?

A) **First 30 minutes**.
B) After 1-2 Hours
C) After one day.

4. What volume of hemorrhage is lethal?

A) 10%.
B) 30%
C) 60%

5. When after small hemorrhage can be reestablished proteins in the blood?

A) After one day.
B) After 3 days.
C) After 6 days.

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THEME OF LESSON: PATHOLOGY OF ERYTHRONE. ERYTHROCYTOSIS, ERYTHREMIA, ANEMIAS

Plan of practical work:

1. Tests and discussion of theoretical material- 60 min.
2. Fulfillment of practical work- 60 min.
3. Discussion of results- 15 min.

1. Scientific basing of topic. Erythrone is special system of the organism which includes erythrocytes circulating in the blood, organs where they can be formed and destroyed, nervous and humeral regulatory mechanisms.

Main chain of erythrone is erythrocytes. They can be characterized according to size, form, color, grade of maturity.

All these characteristics are changed by pathology and can be found in smear of the blood, stained supervitally and by Romanovsky - Giemsa.

Microcytes, macrocytes, megalocytes and megaloblasts can appear by pathology.

Appearance of erythrocytes of different size is named as anisocytosis. Appearance in smear of the blood erythrocytes of different forms (oval, irregular, sickle - form) is named poikilocytosis.

Color of erythrocytes supplies hemoglobin.

Hypochromic erythrocytes appear by decrease of hemoglobin (for example by iron deficiency anemia) Hyperchromic erythrocytes appear due to increase quantity of hemoglobin (for example by vitamin B12- folio deficiency anemia). Immature cells (reticulocytes, polichromatophils) increase in the blood due to activation of erythropoiesis in red bone-marrow (for example by acute posthemorrhagic and hemolytic anemias).

Pathology of erythrone can be manifested in form of erythrocytosis, erythremia, anemia.

Erythrocytosis is a temporary increase quantity of erythrocytes in the blood.

Erythremia (special type of leucosis).

Anemia is such state of the organism which is characterized by decreased quantity of hemoglobin and erythrocytes in the blood.

There are several principles of anemia's classification:

According to change of color coefficient:

1. Hyperchromic.
2. Normochromic.
3. Hypochromic.

According to intensity of erythropoiesis:

1. Regenerative.
2. Hyporegenerative.
3. Hyperregenerative.
4. Aregenerative.

According to size of erythrocytes:

1. Normocytic.
2. Microcytic:
3. Macrocytic.

According to pathogenesis:

1. Posthemorrhagic anemias.
2. Anemias due to disturbance of erythropoiesis.
3. Hemolytic anemia.

2. Aim of topic. Study morphology of the blood of patients by hemolytic and iron-deficiency anemia.

3.Demonstration of educational film: "Anemias by children",

QUESTIONS

1. Erythrocytosis classification.
2. Etiology and pathogenesis of absolute erythrocytosis.
3. Etiology and pathogenesis of relative erythrocytosis.

4. Etiology and pathogenesis of erythremia.

5. Anemia. Definition. Principles of classification.

6. Common symptoms of anemia.

7. Etiology and pathogenesis of acute and chronic posthermorthagic anemia. Changes of the blood.

8. Etiology and pathogenesis of iron deficiency anemia. Changes of the blood.

9. Etiology and pathogenesis vitamin B12-folic acid deficiency anemia. Changes of the blood.

10. Etiology and pathogenesis of hypo-aplastic anemia. Changes of the blood.

11. Etiology and pathogenesis of hemolytic anemias. Changes of the blood.

12. Mechanism formation of anemia by pathology of hypophysis, adrenal and thyroid gland, kidneys.

Tests

1. In what direction can form curve of Price-Jones by iron deficiency anemia?

A) On the right.
B) On the left.
C) Changes are absent.

2. Hypochromia of erythrocytes can develop by anemia:

A) Hemolytic.
B) Aplastic.
C) **Iron deficiency**.

3. Mark anemia, by which practically are absent reticulocytes.

A) Aplastic.
B) Hemolytic.
C) Acute posthemorrhagic.

4. Mark anemia, by which it is necessary to wait (expect) hyper regenerative erythropoiesis.

A) Vitamin B12 deficiency.
B) Aplastic.
C) Hemolytic.

5. Absolute erythrocytosis can develop by:

A) **Cardial insufficiency**.
B) Dehydration.
C) Diarrhea.

6. Show cause relative erythrocytosis.

A) Increase synthesis of erythropoietins in kidneys

B) Increase metabolism in red bone marrow.

C) Dehydration.

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THEME OF LESSON: PATHOLOGY OF LEUKON AND HAEMOSTASIS

Plan of practical work:

1. Tests and discussion of theoretical material - 60 min.

2. Fulfillment of practical work - 60 min.

3. Discussion of results - 15 min.

1. Scientific basing of topic. Leukon consists of leukocytes of peripheral blood, bone-marrow, lymph nodes, tissues, where leukocytes can fulfill their function and regulatory systems.

Basophiles, eosinophils, stick forms, segment forms, lymphocytes and monocytes of the blood are found in healthy person. Percentage ratio of leukocytes in the blood is leukocytic formula. Absolute quantity of different leukocytes in the blood is called profile of Mashkovsky.

Leukocytes can fulfill many functions: they can fulfill phagocytosis, synthesis cytokines, chemokines, release a lot of mediators, inactivation of biologic active substances, formation of immunity.

In pathology, quantity of leukocytes can increase or decrease.

Increase in quantity of leukocytes in the blood is named leukocytosis, decrease - leukopenia.

In malignancy of hemopoietic tissue can develop a special disease - leucosis. Very great role for a doctor has change of leukocyte formula by different diseases

of man. Regenerative and degenerative shift of leukocyte formula can occur. Increase percentage of stick forms, metamyelocytes and sometimes myelocytes means regenerative shift of leukocyte formula. Change in leukocyte like by leucosis and such change is named leukemoid reaction.

There are next leukemoid reactions: neutrophilic, eosinophilic, monocytic, lymphocytic, plasmacytic. Lymphocytic and eosinocytic leukemoid reactions can occur the most often in children.

By development different type of leucosis one can distinguish so called common pathophysiological reactions. They are: hyperplastic syndrome, fever, anemia, hemorrhagic syndrome and cachexia.

2. Aim of topic. Study smears of the blood by inflammation, acute myeloid and chronic myeloid and lymphoid leucosis. Count of leukocytes in leukocytosis

EXPERIMENT *1. Experimental reproduction of leukocytosis.*

Aim. Study in the blood change of quantity of leukocytes and leukocyte formula by inflammation.

Method. Experiment is fulfilled on rabbit with inflammation. The blood from an auricular vein of a rabbit is used for the preparation of a smear. Smear stains by Romanovsky-Giemsa and studies under the microscope to count leukocyte formula. To calculate the number of

leukocytes in 1 mm3 of the blood a special mixer for leukocytes is used. The blood is collected in mixer for leukocytes till to sign 0,5 and diluted by 3% solution of acetic acid, stained blue till to sign 11.

The mixer shakes 3-4 min, 2-3 drops lets out and the rest mixture fulfills Goryaev's chamber. Number of leukocytes calculates under small magnification in 100 large squares, which are not divided into small squares. The next formula allows to determine number of leukocytes in 1 mm3 of the blood.

L = a×4000×20/1600, where:

a - quantity of leukocytes 1 in 100 large squares.

4000 - is number of small squares, their volume in 1 mm.

20 - is degree of blood's dilution.

1600 - is the number of small squares.

Results. Students design leukocyte formula by inflammation, acute myeloid, chronic myeloid and lymphoid leucosis.

Discussion. Students discuss results and make conclusions

Conclusion.

QUESTIONS

1. Leukocyte formula and its disturbance by pathology.
2. Leukocytosis, types, causes. Phases of leukocytic reaction by infectious processes.
3. Leukemoid reactions. Causes. Change of the blood.
4. Leucosis. Etiology, pathogenesis, classification.
5. Peculiarities of leukocytic formula by different types of leucosis.
6. Common pathophysilogic manifestations by leucosis.
7. Mechanism of hemostasis.
8. Main phenomena of hemostasis disturbance.
9. Stages of blood coagulation.
10. Anticoagulative system.
11. Hemorrhagic diathesis
    a) Angiopathy
    b) Thrombocytopathy
    c) Coagulopathy
12. Thrombophylic diathesis.
13. Thrombohemorrhagic syndrome. Etiology, pathogenesis.

Tests

1. Mark process, by which can form leukemoid reaction.

A) Infarct of the myocardium.
B) Renal insufficiency.
C) Sepsis.

2. Mark hormone, effect of which can produce leukocytosis.

A) Aldosterone.
B) Cortisol.
C) Antidiuretic. (ADH).

3. Mark changes leucocytes in the blood by infection in stage neutrophilic struggle.

A) Neutropenia.
B) Neutrophilia with regenerative shift of leucocyte formula on the left.
C) Eosinophilia.

4. Mark natural antiaggregants.

A) cAMP, prostacyclin.
B) Serotonin.
C) Adrenalin.

5. Show aggregants.

A) Collagen.
B) Thrombin.
C) Adrenalin.
D) Noradrenalin.
E) cADP.
F) Thromboxane A2
G) **All above mentioned**.

6. What leucopenia can form by influence ionization and cytostatic?

A) Leucopenia due to redistribution of leucocytes.
B) **Inhibition of leucopoiesis in red bone marrow**.
C) Loss of leucocytes.

7. What stage coagulation of the blood is impaired by decrease quantity of thrombocytes?

A) First.
B) Second.
C) Third.

8. By leukemic form of leucosis quantity of leucocytes in 1 mm3:

A) Hundred thousand.
B) Tenth thousands.
C) Normal.

9. Mark peculiarity of leucocyte formula by acute myeloid leucosis.

A) 95-98 lymphocytes.
B) Leukemic gap (trap).
C) Shadow of Botkin-Gumprecht.

10. Mark factor, which is necessary for the development third phase of blood coagulation.

A) **Fibrinogen.**
B) Prothrombin.
C) Thromboplastin.

11. Mark factor, which is necessary for the development second phase of blood coagulation.

A) Proconvertin (YII).
B) **Prothrombin (II**).
C) Fibrinogen.

12. Deficit of what plasmatic factor is base pathogenesis of hemophilia A?

A) Proconvertin (YII).
B) Antihemophylic A (YIII).
C) IX.
D) X.

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THEME OF LESSON: DISTURBANCES OF SYSTEMIC ARTERIAL BLOOD PRESSURE

Plan of practical work:

1.Tests and discussion of theoretical material - 60 min

2. Fulfillment of practical work - 60 min

3. Discussion of results - 15 min

1.Scientific basing of topic. Systemic arterial blood pressure SABP depends on two labor mechanisms: minute volume of blood circulation (MVC) and common peripheral resistance (CPR. MVC supplies systolic volume and rate of contraction of the heart per minute GPR depends on resistance of vessels, volume of circulating blood, rheologic properties of blood.

Regulation of systemic arterial pressure is very complex. Brain (hypothalamus, hypophysis, medulla oblongata), endocrine glands (adrenal, thyroid glands), baro- and chemoreceptor, endothelium, such organs as kidneys, lungs, liver, heart, regulate arterial pressure.

SABP can increase (hypertension) and decrease (hypotension). All hyper- and hypotensions are classified on essential and symptomatic.

The main cause of essential hyper- and hypotension is disorder of the highest centers of CNS. Symptomatic hyper- and hypotensions is a symptom of pathologic processes or diseases. For example, hyper-bypofunctions hypophysis, adrenal, thyroid gland, kidneys.

2. Aim of topic. Introduction with mechanisms of hypertension Experimental reproduction of hypertension of different origin.

EXPERIMENT *1. Experimental reproduction of reflexogenic hypertension.*

Aim. Study the role of baroreceptors in regulation of systemic arterial blood pressure.

Method. Experiment is fulfilled on a rat. Animal is anesthetized (I ml 1% Nembutal / 150g of body's weight). Operatively are found common carotid arteries. Systemic arterial blood pressure is measured. Then both carotid arteries are clamped. Reflexogenic arterial hypertension develops.

Results. Students show increase in systemic arterial blood pressure.

Discussion. Students discuss mechanism of formation of reflexogenic hypertension and make conclusion.

Conclusion.

3. Demonstration of educational film "Malignant hypertension" - 20 min

QUESTIONS

1. Systemic arterial blood pressure.

Labor and regulatory mechanisms.

2. Consequences of hypertension and hypotension.

3. Classification of hypertension:

a) According to character

b) According to pathogenesis

c) According to the change of systemic arterial pressure

4. Role of morphologic and functional change of vessels in development of hypertension.

5. Role of morphologic and functional change in the heart in development of hypertension.

6. Etiology, pathogenesis of reflexogenic hypertension.

7. Hypertension by aldosteronism. Etiology, pathogenesis.

8. Hypertension by hypercortisolism. Etiology, pathogenesis.

9. Renal hypertension. Etiology, pathogenesis.

10. Common principles of treatment of hypertensions.

11. Classification of hypotensions.

12. Essential and symptomatic hypertensions.

Tests

1. In what artery can form atherosclerosis the most often?

A) **Coronary**.
B) Subclavicular.
C) Axillary.

2. Name theories, which can explain development of atherosclerosis.

A) Vascular.
B) Plasmatic.
C) **All above mentioned.**

3. Mark hemodynamic mechanisms, which can define systemic arterial blood pressure.

A) Minute volume of blood circulation.
B) Common(general) peripheral resistance.
C) All above mentioned.

4. Mark diseases of arterial vessels depending on damage its wall.

A) Intimal.
B) Inflammatory.
C) Degenerative.

5. What presser mechanism takes part in formation of reflexogenc hypertension?

A) Baroreceptor.
B) Endothelial.
C) Hepatic.

6. Name depressor mechanism regulation of vascular tonus.

A) Cellular.
B) Renin-angiotensin- aldosterone.
C) **Kallicrein-kinin system**.

7. Mark refectory presser mechanism regulation of arterial pressure.

A) Chemoreceptory.
B) Endothelial.
C) Volume.

8. Mark factor, which allow development atherosclerosis.

A) Growth.
B) **Obesity.**
C) Hyperthyroidism.

9. Mark antiatherogenic factor.

A) Chylomicrons.
B) Lipoproteins of high density.
C) Lipoproteins of very low density

10. Atherosclerosis is:

A) Local inflammatory process.
B) Local degenerative process.
C) Systemic disease, the base of which is degenerative change intima elastic and muscular-elastic vessels with formation of atheroms.

11. Disorder of what type metabolism by hypercortisolism can lead to development of hypertension?

A) Carbohydrate.
B) Protein.
C) Lipid
D) Water-electrolyte.

12. Is it possible involution of atherosclerosis?

A) Yes.
B) No.

13. Mark renal depressor mechanism.

A) Renin-angiotensin-aldosterone.
B) Serotonin.

C) Adrenalin (epinephrine).

D) **Volume mechanism.**

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THEME OF LESSONS: ARRHYTHMIAS OF THE HEART

Plan of practical work:

1. Tests and discussion of theoretical material - 60 min
2. Fulfillment of practical work - 60 min
3. Discussion of results - 15 min

1. Scientific basing of topic. Arrhythmia of the heart is such disorder which is characterized by change of rhythm and periodicity of excitation and contraction of myocardium. Arrhythmia of the heart can occur due to pathologic changes in the heart itself or due to extracardiac influences. It can lead to change of rhythm contraction of myocardium. Arrhythmia can develop due to disorder of all the functions of myocardium (automatism, conductivity, excitability, contractility)

2. Aim of topic. Reproduction of arrhythmias of different origin.

EXPERIMENT *1. Bradycardia of reflex origin.*

Aim. Study the role of reflex mechanisms in development of bradycardia.

Method. Experiment is performed on an anesthetized rat. ECG is registered in starting point and heart's rate is counted. Then adrenalin is injected intraperitoniely (0.5 - 0.7 ml) and ECG is recoded again. Heart's rate is counted too.

Results. Students show change of heart's rate and ECG.

Discussion. Students discuss results and make conclusions.

Conclusions.

EXPERIMENT *2. Tachocardia of reflex origin*.

Aim. Study the role of reflex mechanisms in development of tachycardia.

Method. Experiment is performed on an anesthetized rat. ECG is registered in starting point and heart's rate is counted. Then acetylcholine is injected intravenously (0.5 ml) ECG and heart's rate are registered.

Results. Students show change of heart's rate and ECG.

Discussion. Students discuss results and make conclusions.

Conclusions.

3. Demonstration of educational film «Ventrical tachycardias» - 20 min

QUESTIONS

1. Definition of arrhythmia of the heart.
2. Classification of arrhythmias.
3. Etiology of arrhythmias.
4. Localization and characteristic of slow depolarization.
5. Arrhythmias of the heart due to disorder of automatism.
6. Characteristic of nomotopic sinus arrhythmias.
7. Characteristic of heterotopic arrhythmias.
8. Etiology of blockers.
9. Characteristic of blockers of different level.
10. Edem - Stocks syndrome.
11. Walf - Parkinson - White syndrome.
12. Etiology of excitability and conductivity disorders.
13. Characteristic of premature beat (extrasystole).
14. Paroxysmal tachycardia. Etiology, pathogenesis.
15. Flutter of atriums. Etiology, pathogenesis, change in ECG.
16. Fibrillation. Change in ECG. Defibrillation.
17. Transformations of rhythm.
18. Alternating pulse.
19. Common principles of arrhythmia's treatment.

Tests

1. Mark reasons of reflector sinus tachycardia.

A) Hypoxia.
B) Decrease systemic arterial blood pressure.
C) Pain.
D) **All above mentioned**.

2. Mark reasons of refectory sinus bradycardia.

A) Golts reflex.
B) Ashner reflex
C) Chermak – Hering reflex.
D) **All above mentioned.**

3. Mark peculiarity of ECG by sinus tachycardia.

A) Widening ventricle complex.
B) Lengthening interval PQ.
C) Shortening of common diastole.

4. Mark reasons disorder of conductivity in myocardium.

A) Decrease quantity of potassium ions.
B) **Increase quantity of potassium ions.**
C) Increase quantity of sodium ions.

5. Show arrhythmia of myocardium, which can form due to disorder of excitability and conductivity.

A) Transformation of rhythm.
B) Alternative pulse.
C) Exstrasystole (premature beat).

6. Mark clinical manifestations syndrome of Morgany-Edem-Stock.

A) Increase systemic arterial blood pressure.
B) **Loss of consciousness**.
C) Hyperemia of face.

7. Show nomotopic arrhythmia.

A) **Sinus tachycardia**.
B) Ideoventricule rhythm.
C) Exstrasystole.

8. Mark arrhythmia, the base of which is re-enter-mechanism.

A) Sinus bradycardia.
B) Sinus tachycardia.

C) Paroxysmal tachycardia.

9. Mark normal charge membrane of cells sino-atrial node.

A) **50-60 mv.**
B) 60-70 mv.
C) 80-90 mv.

10. Mark the most often cause of paroxysmal tachycardia.

A) Vagotonia.
B) Emotional and physical stress.
C) Leukocytosis.

11. Mark possible clinical manifestations of cardial arrhythmias.

A) Accelerathon cardiac rhythm.
B) Bradycardia.
C) Decrease systemic arterial blood pressure.
D) Pain in the heart.
E) **All above mentioned**.

12. By what stage of atrioventricular block can form different contractions of atriums and ventricles?

A) First.
B) Second.
C) Third.
D) **Forth.**

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2. Nowak T., Handford A. "Essential of pathophysiology. Concepts and applications for Health Care Professionals", London, Singapore, 1999, p.249.

3. Guyton, «Textbook of medical physiology», p. 171-203.

4. Andreoli and all «Cecil essential of medicine», p. 80-105.

THEME OF LESSON: INSUFFICIENCY OF BLOOD CIRCULATION

Plan of practical work:

1. Tests and discussion of theoretical material - 60 min

2. Fulfillment of practical work - 60 min

3. Discussion of results - 15 min

1. Scientific basing of topic. Insufficiency of blood circulation means impossibility of system of blood circulation to supply organs and tissue with blood according to its metabolic requirements. 250 - 300 ml of oxygen is required for basic metabolism. Consumption of oxygen increases by emotional and physical stress, digestion, pregnancy.

Some compensatory reactions are switched by insufficiency of blood circulation. They are cardiac and extracardiac.

Activation it is directed on improvement supplying organs and tissues with blood.

Cardiac compensatory reactions are the following:

1) Heterometric mechanism (m-m Frank-Starling)

2) Homeometric mechanism (m-m rate and strength of Hill)

3) Tachycardia

4) Hypertrophy of myocardium

Extracardiac compensatory mechanisms are the following.

1) Tachypnoe

2) Increase volume of circulating blood and quantity of erythrocytes

3) Increase activity of tissue enzymes

Insufficiency of blood circulation is classified according

duration (acute, chronic), intensity,

(compensatory, subcompensatory, decompensatory) mechanism of its development. Two forms insufficiency are distinguished according

mechanisms of development:

1) Cardiac (central)

2) Vascular (peripheral)

2. Aim of topic. Reproduction of cardiac insufficiency.

EXPERIMENT *1. Experimental reproduction infarct of myocardium.*

Aim. Reproduction infarct of myocardium and study change of electrocardiogram.

Method. A frog makes motionless due to destroy of spinal cord is fixed on special table and operates to recover of the heart. In initial point ECG is recorded. Then on the surface of the heart puts crystal of AgNO3, which produces necrosis of myocardium. ECG records again in dynamic of infarct.

Results. Students mark change in color of myocardium in focus of infarct and analysis of ECG.

Discussion. Students analyze results and make conclusions.

QUESTIONS

1. Definition of "insufficiency of blood circulation"
2. Classification of insufficiency of blood circulation:

a. According to duration

b. According to intensity

c. According to mechanism of development

3. Compensatory reactions by insufficiency of blood circulation.
4. Etiology of cardiac insufficiency.
5. Heterometric mechanism of compensation.
6. Homeometric mechanism of compensation.
7. Tachycardia as urgent mechanism of compensation.
8. Cardiac hypertrophy as prolonged mechanism of compensation.
9. Cardiac hypertrophy. Etiology, mechanism.
10. Disorder of coronary blood circulation. Experimental reproduction infarct of myocardium
11. Pathogenesis of cardiac insufficiency.
12. Clinico-laboratory manifestations of cardiac insufficiency.

Tests

1. Reperfusion by ischemic heart disease is due to:

A) Inflow of nutrients.

B) Formation excess (surplus) of peroxydes.

2. Mark possible complications of ischemic heart disease.

A) Aneurism of the heart.

B) Atherosclerosis.

C) Amyloidosis.

3. Mark etiology of Ischemic heart disease.

A) Hypodynamia.

B) Thrombosis of coronary artery.

C) Obesity.

4. Mark stages infarct of myocardium in order of its development.

A) Latent. Ischemic. Necrotic.

B) **Ischemic. Necrotic. Organization.**

5. Mark unfavorable outcome infarct of myocardium.

A) Aneurism.

B) True breaking of myocardium.

C) Death.

D) **All above mentioned.**

6. Resorbtive-necrotic syndrome is characterized by:

A) Formation of endogenic pyrogens.

B) Appearance in the blood products of proteolysis and enzymes.

C) Loss of electrolytes from cardio myocytes.

D) **All above mentioned.**

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THEME OE LESSON: INSUFFICIENCY OF EXTERNAL BREATHING

Plan of practical work:

1. Tests and discussion of theoretical material - 60 min

2. Fulfillment of practical work - 60 min

3. Discussion of results - 15 min

1. Scientific basing of topic. The main functions of the apparatus of external breathing are supplying gas exchange (O_2, CO_2) between external air and the blood. Three main processes in the lungs supply gas exchange: ventilation, diffusion and perfusion.

Disturbances of these processes can lead to formation of respiratory insufficiency. Insufficiency of external breathing means the inability of the apparatus of respiration to supply gas exchange according to metabolic requirement.

According to the mechanism of development, we can distinguished the following forms of respiratory insufficiency;

1) Central
2) Peripheral
3) Thoraco-diaphragmal
4) Cardio - vascular
5) Pulmonary
6) Mixed

Acute respiratory insufficiency leads to hypoxemia, hypercapnia, increase work of respiratory apparatus, dyspnos, cyanosis.

By chronic respiratory insufficiency except above mentioned, increases volume of circulating blood, quantity of erythrocytes,

cordial hypertrophy.

2. Aim of topic. Introduction with disturbances of breathing by insufficiency of external respiration.

EXPERIMENT *1.* *Change external breathing by artificial pneumothorax.*

Aim. Study rate of breathing by pneumothorax.

Method. Rat is anesthetized and in starting point counts rate of breathing 3-5 ml of airs is injected intrapleural space and rate of breathing is counted again in dynamic pneumothorax.

Results. Students analyze change of respiration.

Discussion. Students discuss results and make conclusions.

Conclusions.

3. Demonstration of educational film "Acute respiratory insufficiency" - 20 min

QUESTIONS

1. Definition of respiratory insufficiency.
2. Classification of respiratory insufficiency.
3. Mechanism of respiratory insufficiency.
4. Etiology of respiratory insufficiency.
5/ Clinical and laboratory manifestations of respiratory insufficiency
6. Pathological types of respiration.

Tests

1. By disorder of what process in the lungs can form mechanism of «alveolar shunt»?

A) **Ventilation.**
B) Diffusion.
C) Perfusion.

2. Mark diseases in the lungs, by which can form mechanism «expiratory closing of breathings ways» (gas or air trap)?

A) Pneumonia.
B) Bronchial asthma.
C) Bronchitis.
D) All above mentioned.

3. How can changed pH of the blood by respiratory insufficiency?

A) Metabolic acidosis.
B) Metabolic alkalosis.
C) **Gas acidosis.**

4. How can change minute volume of respiration by respiratory insufficiency?

A) Increased.
B) Decreased
C) Normal.

5. How can change consumption of oxygen by disorder «coupling between oxidation and phosphorylation»?

A) Decrease.
B) **Increase.**
C) It is not changed.

6. What substances can form methemoglobin in the blood?

A) **Nitrates**.
B) Antibiotics.
C) Vitamins.

7. By what type of hypoxia utilization of oxygen and nutrients can be disturbed?

A) Anemic (hemic).
B) **Tissue (histotoxic**).
C) Respiratory.
D) Cardio-vascular (circulatory).

8. What middle indexes partial pressure of oxygen and carbon dioxide in the blood coming to the lungs?

	Oxygen	Carbon doxyde
A)	90 mm Hg	60 mm Hg
B)	70 mm Hg	50 mm Hg
C)	**40 mm Hg**	**46 mm Hg**
D)	30 mm Hg	35 mm Hg

9. Mark causes by which can be disturbed diffusion in the lungs.

A) Lungs edema.
B) Pneumonia.
C) Pneumoconiosis.
D) All above mentioned.

10. Mark etiology of cardio-vascular type respiratory Insufficiency.

A) Pneumonia.
B) **Shock and collapse**.
C) Bronchial asthma.

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THEME OF LESSON: PATHOLOGY OF KIDNEYS

Plan of practical work:

1. Tests and discussion of theoretical material - 60 min
2. Fulfillment of practical work - 60 min
3. Discussion of results - 15 min

1. Scientific basing of topic. Kidneys perform two main functions: excretory (withdrawal or delay of electrolytes water, products of metabolism, hydrogen ions) and regulatory (regulation of SABP, erythropoiesis, calcium-phosphor exchange, hemostasis, immunity). Performing of these functions is possible due to such processes in kidneys as filtration reabsortion, secretion and synthesis of new regulatory substances (renin, angiotensin, kinins, prostaglandins, erythropoietins, Vit. D, cytokines).

Pathology of kidneys lead to the following syndromes: urine syndrome, nephrotic syndrome, acute nephritic syndrome, acute and chronic renal insufficiency, renal hypertension.

2. Aim topic. Study main manifestations by acute renal insufficiency.

EXPERIMENT 1. ***Experimental uremia.***

Aim. Reproduction of uremia and studying clinical manifestations.

Method. Rat is anesthetized 2-3 day before classes. In starting point, ECG and body temperature are recorded. Then operatively ureters are clamped.

Students record ECG, body temperature, clinical symptoms 2-3 days after operation.

Results. Students show table with changes in ECG, rate contraction of myocardium, body's temperature and notice urine odor, convulsions, vomiting.

Discussion. Students discuss results, explain the origin of manifestations and make conclusions.

Conclusion.

3. Demonstration of educational film "Chronic renal insufficiency"

QUESTIONS

1. Functions of kidney.

2. Common etiology and pathology of kidneys.

3. Disturbances of filtration.

4. Disturbances of absorbtion.

5. Disturbances of secretion.

6. Urine syndrome

7. Etiology, pathogenesis, clinical and laboratory manifestations of nephrotic syndrome.

8. Etiology, pathogenesis, clinical and laboratory manifestations of acute and chronic renal insufficiency.

Tests

1. In what section of nephron reabsorption of water is maximal?

A) Proximal.
B) Descending loop of Henle.
C) Ascending loop of Henle.
D) Collective tubules.

2. By what pathology in kidneys in final urine will be released big quantity of proteins?

A) Chronic renal insufficiency.
B) Acute renal insufficiency.
C) Nephrotic syndrome.

3. What is main cause of acute renal insufficiency?

A) Inflammation.
B) Fever.
C) **Shock**.

4. What type of acidosis can form by acute renal insufficiency?

A) Metabolic acidosis.
B) Gas acidosis.

5. Mark hormone, which can stimulate reabsorption water in kidneys.

A) **ADH (antidiuretic hormone).**
B) Aldosterone.
C) Cortisol.
D) Corticosterone.

6. How can change quantity of protein in the blood by nephrotic syndrome?

A) It is not change.
B) Increase.
C) Decrease very intensively.

7. Mark main mechanism of renal hypertension.

A) Decrease synthesis of renin.
B) Decrease release of aldosterone.
C) Activation renin-angiotensin-aldosterone system.

8. What is oliguria?

A) Increase release of urine.
B) Decrease diuresis.
C) Painful urination.

9. How can influence decrease oncotic pressure of the blood on glomerular filtration?

A) Increase.
B) It is not influence.
C) Decrease.

10. What is main reason (cause) of anemia by renal pathology?

A) Hemolysis of erythrocytes.
B) Decrease synthesis of erythropoietins.
C) Loss of Fe, Ni.

11. Hypercalcemia can form by chronic renal insufficiency due to surplus effect:

A) Thyroxin.
B) Parathyrine.
C) Glucagon.

12. What is main factor allow to develop oedema by nephrotic syndrome.

A) Increase oncotic pressure in the blood.
B) **Decrease oncotic pressure in the blood**.
C) Decrease permeability of blood vessels.

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2. Nowak T., Handford A. "Essential of pathophysiology. Concepts and applications for Health Care Professionals", London, Singapore, 1999, p. 207- 210.

THEME OF LESSON: PATHOLOGY OF LIVER

Plan of practical work:

1. Tests and discussion of theoretical material - 60 min
2. Fulfillment of practical work - 60 min
3. Discussion of results - 15 min

1. Scientific basing of topic. Liver is a very important organ. It performs multiple functions: exchange of proteins, carbohydrates, lipids, electrolytes, hormones; synthesis and secretion of bile, proteins, carbohydrates, lipids, plasmatic factors of blood coagulation, heparin, intestinal hormones, deport of iron, vitamin Biz; inactivation of microbes; detoxication; hemopoiesis by fetus.

Pathology of liver can lead to the following syndromes: hepato-cellular insufficiency, jaundice and cholestasis, portal hypertension, hepato-renal syndrome, mesenchymal - inflammatory syndrome.

2. Aim of classes. Introduction with possible manifestations by pathology of liver.

EXPERIMENT *1. Experimental jaundice.*

Aim. Modeling experimental jaundice and introduction with clinical symptoms and changes in ECG.

Method. Experiment is fulfilled on the rat. Students study behavior of animal, count breathing rate and ECG is recorded. 2-3 days before classes, the animal is operated and common bile duct ligatures. Students study activity, behavior and color of conjunctiva. ECG is recorded again.

Results. Students note changes in ECG, behavior and color of conjunctiva.

Discussion. Students discuss results and make conclusions.

Conclusion.

EXPERIMENT *2. Influence of bile on the heart.*

Aim. Study the changes in ECG and the rate of contraction of the heart by increase quantity of bile in the blood.

Method. Experiment is performed on a frog. Spinal cord is destroyed. ECG is recorded in starting point. Then bile (0.3-0.4 ml 5-10%) is injected into abdominal vein and ECG is recorded again.

Results. Students count heart's rate and analyze the changes in ECG.

Discussion. Students discuss results and make conclusions.

Conclusion.

QUESTIONS

1. Etiology of hepato-cellular insufficiency.
2. Disorder of protein exchange by hepato-cellular insufficiency.
3. Disorder of carbohydrate exchange by hepato-cellular insufficiency.
4. Disorder of lipid exchange by hepato-cellular insufficiency.
5. Disorder of metabolism of hormones and biologic active substances by hepato-cellular insufficiency.
6. Syndromes of portal hypertension. Etiology, manifestations.
7. Hepatic coma. Etiology, manifestations.
8. Jaundice. Etiology, classification, clinical and laboratory manifestations.
9. Hepato-renal syndrome.
10. Mesenchymal - inflammatory syndrome.

Tests

1. Disorder of what type metabolism by hepato-cellular Insufficiency can lead to decrease arterial blood pressure?

A) Protein.
B) Carbohydrate.
C) Lipid.
D) Water-electrolyte.

214.What hormones can mobilize fat from fat depots?
A) Insulin.
B) Contrinsular hormones.
C) Sexoides.

2. Why can form steatorrhea by hepatocellular insufficiency?

A) Due to splitting fat in intestine.
B) Due to absence of bile and release fat with excrement's.

3. Why can form hypercholesterinemia by hepatocellular Insufficiency?

A) Increase absorption cholesterol in intestine.
B) Conversion cholesterol into steroid hormones.
C) Decrease release cholesterol with urine.

4. How can change quantity of albumines in the blood by Intensive hepatocellular insufficiency?

A) Increase.
B) Decrease.
C) It is not changed.

5. What plasma proteins will decrease first of all by hepato-cellular insufficiency?

A) Alfa-globulins.
B) Betta-globulins.
C) Gamma-globulins.
D) Albumins.

6. How can change quantity of glucose in the blood by hepatocellular insufficiency?

A) Increase.
B) **Decrease**.
C) It is not change.

7. How can change effects of hormones by hepatocellular insufficiency?

A) Increase.
B) Decrease.
C) It is not change.

8. What cells can form bilirubin?

A) Epithelium of kidneys.
B) Cells of reticulo-endothelial system.
C) Muscular.
D) Nervous.

9. In what organ can form direct or conjugative bilirubin?

A) Lunges.
B) Muscles.
C) **Liver.**
D) Heart.

10. Itch or pruritus can form by hepatic and obturative jaundice (icterus) by:

A) Decrease in the blood quantity of bile acids.
B) Increase in the blood quantity of bile acids.

11. Bradycardia by obturative jaundice can form due to:

A) Increase intracranial pressure.

B) Irritation of tissue receptors with bile acids.
C) Reflectory influences from dilated Gall bladder.
D) Daniny-Ashner reflex.
E) Chermak-Hering reflex.

12. What toxical substances are formed by hepatocellular Insufficiency due to disorder of carbohydrate metabolism:
A) Ammonia.
B) Valerian acid
C) **Acetoin and 2,3 butilenglicol**.

13. What acids in liver don't take part in inactivation toxic substances?
A) **Betta-oxybuthiric acid.**
B) Glucuronic acid.
C) Sulfuric acid.

14. How can change function of CNS by hepatic coma?
A) Activation.
B) **Inhibition**.
C) It is not change.

15. Mark possible change acid-base balance by hepatic coma.
A) Development acidosis.
B) Development alkalosis.

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THEME OF LESSON: INSUFFICIENCY OF DIGESTION

Plan of practical work:

1. Tests and discussion of theoretical material - 60 min

2. Fulfillment of practical work - 60 min

3. Discussion of results - 15 min

1. Scientific basing of topic. Digestion is the unity hydrolytic splitting of nutrients (food) on simple substances (monomers) and its absorption. The base of digestion consists of secretion of digestive juices, peristalsis of gastrointestinal tract and

absorption of products of digestion into the lymph and blood. Insufficiency of digestion means impossibility of gastrointestinal tract to supply of the organism with alimentary substances. It can form due to disturbance secretion of digestive juices, peristalsis and absorption.

2. Aim of topic. Introduction to some pathologies of gastrointestinal tract, which can lead to digestion's insufficiency.

EXPERIMENT *1. Experimental intestinal obstruction.*

Aim. Production of intestinal obstruction and study change of the blood (quantity of erythrocytes, leucocytes, hematocrit).

Method. The blood is taken in starting point for analysis from rat's tip of tail or sublingual vena. Quantity of erythrocytes, leucocytes, hematocrit are determined. Animal is operated under ethers anesthesia and mesenterium intestine is ligated and wound is sutured. Students study symptoms of intestinal obstruction and above mention indexes of the blood two - three days later.

Results. Students describe clinical manifestations and change of the blood by intestinal obstruction.

Discussion. Students discuss origin of clinical manifestations and change of the blood by intestinal obstruction and make conclusions.

Conclusions.

EXPERIMENT 2. *Experimental reproduction*

of erosions and ulcer in stomach.

Aim. Study role of stress in formation of stomach's erosions and ulcers.

Method. Rat is immobilized during 24 hours. Immobilization is repeated during 5 days with complex of low temperature (+4) during two hours. After 5 days such complex stress (immobilization and low temperature), animal makes autopsy and studies mucosa of stomach.

Results. Students describe change of stomach's mucosa and discuss results.

Discussion.

Conclusion.

QUESTIONS

1.Intestinal hormones and its disturbance by pathology.

2. Etiology, pathogenesis and possible manifestations by insufficiency of digestion.

3. Disturbance of digestion in mouth.

4. Disturbance of digestion in stomach.

5. Disturbance of digestion in intestine.

6. Disturbance of digestion by pathology of pancreas

7. Protective mechanisms of gastro-intestinal tract and its disorder by pathology.

8. Specialty of digestion in infantile age.

Tests

1. Deficit of what type vitamin is it necessary to wait by atrophy stomach mucous?

A) Vitamin C.
B) Vitamin A.
C) Vitamin D.
D) Vitamin B 12

2. How can change intensity fermentation and putrefaction by delay evacuation of food from stomach?

A) **Increases**.
B) Decreases.
C) It is not changed

3. How stress can influence on development stomach ulcer and duodenum?

A) It does not influence.
B) Accelerate.
C) Inhibit.

4. What is main cause (reason) of stomach ulcer?

A) Deficit effects of acidic-peptic factor.
B) Superiority effect of acidic-peptic factor.

5. What effect of disorder during prolonged time absorption of nutrients from intestine?

A) Decries body weight.
B) Deficit of vitamins.
C) Immunodeficit.
D) All above mentioned.

6. When can form hypersalivation?

A) By fever.
B) By activation sympatho- adrenal system.
C) By action of parasympathomimetics.

7. How can change evacuation of food by acidic gastritis from stomach in duodenum**?**

A) Accelerated**.**
B) Inhibited.
C) It is not changed.

8. How can influence stress on secretion stomach juice and its acidity?

A) Increased secretion and acidity.
B) Inhibited secretion and acidity.
C) Stimulated secretion and inhibited acidity**.**

9. Mark phenomenon achylia**.**

A) Absence of hydrochloric acid.
B) Absence proteolytic enzymes.
C) Absence hydrochloric acid and proteolytic enzymes.

10. What is main cause(reason) of stomach ulcer?

A) Smoking.
B) Alcohol.
C) Helicobacter pillory.

11. What reason does not influence on development of disbacteriosis?

A) Disorder of peristalsis.
B) Immunodeficiency state.
C) Intensive treatment with antibiotics.
D) **Hemolytic jaundice**.

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THEME OF LESSON: PATHOLOGY OF ENDOCRINE SYSTEM.

Practical work 1. Plan of practical work:

1. Tests and discussion of theoretical material - 60 min.
2. Fulfillment of practical work - 60 min.
3. Discussion of results - 15 min.

1. Scientific basing of topic. Endocrine system fulfills control of different metabolic functions of the body: rates of chemical reactions in the cells or transport of substances through the cells membrane or other aspects of cellular metabolism like growth and secretion, development of CNS. Some hormonal effects occur in seconds, others require days, weeks, years. Endocrine system has several chains: chain of regulation, synthesis, secretion, transport, effect, metabolism.

Pathology of endocrine system can occur on the level of any of the above-mentioned chains. For example: by pathology of hypothalamus or hypophysis can be disturbed function of adrenal, thyroid, sex glands; by deficit of iodine can be disordered synthesis of thyroxin and triiodothyronine; by congenital disorder of synthesis of cortisol due to feedback will increase secretion of corticotrophin in hypophysis and sex hormones and aldosterone in adrenal gland; hormones are transported in the blood or in free state or in the form of complex with proteins. Effect of hormones can change by deficit of proteins; hormones can connect with receptor on membrane of the cells or with receptors on membrane of cell's nucleus (by quality and quantity change of receptors effects of hormones change too, for example, by diabetes mellitus type II); all hormones are metabolized in liver. Metabolism of hormones is delayed by hepato-cellular insufficiency and influence of hormones can be more prolonged.

2. Aim of topic. Introduction with pathology of hypothalamus-hypophysis-adrenal, thyroid and parathyroid system.

EXPERIMENT 1. Role of pathology of hypothalamus in disturbance of adrenal, thyroid glands.

Aim. Show that destruction of hypothalamus can produce pathology of adrenal and thyroid glands.

Method. Corticosterone, T3, T4 are found in the blood of rat, in starting point. Hypothalamus is destroyed with help of stereotaxical apparatus in anesthetized rat. The same hormones are recorded in the blood one week after operation.

Results. Results are shown in the table 1.

Table 1.

Groups of animal	Corticosterone, mmolle	T3, mmolle	T4, mmole
1. Intact rat	533.3	2.24	124
2. Rat with destroyed hypothalamus	176	1.17	56.2

Discussion. Students discuss result and make conclusions.

Conclusion.

QUESTIONS

1. Hormones of hypothalamus and its effects.
2. Hormones of adrenal glands and its effects.
3. Hormones of thyroid and parathyroid glands and its effects.
4. Etiology, pathogenesis and manifestations of Simmond's disease.
5. Origin of manifestations by increase and decrease in quantity of somatotropins.
6. Effects of increase and decrease of prolactin.
7. Etiology and manifestations by increase and decrease of prolactin.
8. Etiology and manifestations by increase and decrease in quantity of gonadotropins.
9. Etiology and manifestations by increase and decrease in quantity of vasopressin (ADH).
10. Biologic effects of aldosterone. Etiology, pathogenesis and possible clinical and laboratory manifestations by aldosteronism.
11. Biologic effects of glucocorticoids (on carbohydrate, protein, lipid, mineral exchange).
12. Etiology and possible manifestations by hypercortisolism.

13. Etiology and possible manifestations by isosexual and heterosexual adreno-genital syndrome.

14. Biologic effects of triiodothyronine and thyroxin.

15. Etiology and manifestations of hyperthyroidism.

16. Etiology and manifestations of hypothyroidism.

17. Endemic goiter. Etiology, pathogenesis.

18. Biologic effects of parathyrin.

19. Etiology and manifestations of hyperparathyroidism.

20. Etiology and manifestations of hypoparathyroidism.

21. Influence of androgens on foetus.

22. Influence of androgens in period of maturation.

23. Etiology of primary and secondary hypergonadism.

24. Clinical manifestations of hypogonadism.

25. Clinical manifestations of hypergonadism before and after sex maturation in males and females.

Tests

a). Pathology of hypothalamus – hypophysis – adrenal system.

1. What is Simmonds disease?

A) Hyperfunction of hypophysis.
B) **Total insufficiency of hypophysis.**
C) Total hyperfunction of adrenal cortex.

2. What can develop by deficit of corticotrophin?

A) Primary insufficiency of adrenal cortex.
B) Secondary insufficiency of adrenal cortex.

3. What syndrome can form(appear) by deficit of gonadotrophines.

A) Adrenogenital syndrome.
B) Adiposogenital syndrome.

4. What effect is it necessary to wait by deficit of somatotrophines?

A) Gigantic(ism).
B) Hypothyroidism.
C) Insufficiency of adrenal glands.
D) Nanism (dwarfism).

5. What hormones are released from cortex of adrenal glands?

A) Liberines.
B) Statines.
C) Corticosteroides.

6. What hormones are released from medulla substance of adrenal glands?

A) Androgens.
B) Catecholamines.
C) Estrogens.

7. How can change level of glucose in the blood by insufficiency of adrenal glands?

A) Increase.
B) Decrease.
C) It is not change.

8. Mark reason of primary aldosteronism.

A) Tumor of glomerular zone cortex of adrenal glands.
B) Cardiac insufficiency.
C) Hepatocellular insufficiency.

9. Convulsions by primary aldosteronism can form due to:

A) Increase potassium in the blood.
B) Increase potassium in the muscles.
C) Decrease hydrogen ions.

10. How can change muscle tonus by adrenal glands insufficiency?

A) Increase.
B) It is not change.
C) Decrease.

11. Why can form strias and osteoporosis by hypercortisolism?

A) Due to mobilization of glycogen.
B) Due to mobilization of fat.
C) Due to mobilization of endogenic proteins.

12. What manifestations can appear by chronic insufficiency of adrenal glands (Addison disease)?

A) Arterial hypertension.
B) Black color of skin.
C) Increase sodium ions.
D) Decrease potassium ions.

13. Mark symptoms Itsenco-Cushing disease.

A) Hyperplasion of one adrenal gland.
B) **Hyperplasion of both (two) adrenal glands**.
C) Low level of corticotrophin in the blood.

14. By what pathology can develop secondary aldosteronism?

A) Aldosteroma of glomerular zone of adrenal glands.
B) Quincke's edema.
C) Hepatocellular insufficiency, cirrhosis.

b) Topic: Pathophysiology of thyroid and parathyroid glands.

15. Is water-mineral metabolism by hyper functions of the parathyroid glands changed?

A) Yes.
B) Not.

16. Mark possible manifestations by hyperthyroidism.

A) Decrease body temperature.
B) Increase tissue breathing and body temperature.
C) Hypoglycemia.
D) Obesity.

17. Mark manifestation by hypothyroid coma.

A) Decrease tissue breathing.
B) Tachycardia.
C) Increase body temperature.

18. How can change protein metabolism by increase function of thyroid gland?

A) It is activated.
B) It is disorded.
C) It is not change.

19. Intolerance to cold can appear by:

A) Increase function of thyroid gland.
B) Decrease function of thyroid gland.
C) Hypoparathyroidism.

20. When can form erythrocytosis?

A) By decrease function of thyroid gland.
B) By increase function of parathyroid glands.
C) By increase function of thyroid gland.

21. Mark endocrine gland by pathology of which can develop anemia.

A) Parathyroid glands.
B) Thyroid gland (hyperfunction).
C) Thyroid gland (hypofunction).
D) Pancreas.

22. By what pathology of endocrine glands can form endogenic hyperthermia?

A) Increase function of parathyroid glands.
B) Increase function of thyroid gland.
C) Decrease function of thyroid gland.

23. What hormone can form in parathyroid glands?
A) Calcitonin.
B) **Parathyrin**.
C) Thyroxin.

24. Why by hyperparathyroidism can form osteoporosis and tendency to fracture (break) of bones.
A) Decrease calcium in the blood.
B) Mobilization calcium from bones.
C) Disorder of lipid metabolism.

25. Mark manifestations by hyperparathyroidism.
A) Increase calcium ions and decrease phosphates in the blood.
B) Calcinosis.
C) Pain in muscles.
D) Osteoporosis.
E) Chronic renal insufficiency.
F) All above mentioned.

c). Topic: Diabetes mellitus.

26. How insulin can influence on protein exchange?
A) Inhibit synthesis.
B) Stimulate synthesis.
C) Mobilize endogenic proteins.

27. How insulin can influence on lipid exchange?
A) Stimulate lipolysis.
B) Stimulate lipogenesis.
C) Stimulate gluconeogenesis.

28. Type 2 diabetes mellitus (insulin nondependent) can develop due to:
A) Decrease sensitivity insulin receptors.
B) Increase sensitivity and quantity insulin receptors.

29. Decrease body weight by diabetes mellitus can form due to mobilization:
A) Carbohydrates.
B) Proteins.
C) Lipids

30. Disorder of what type metabolism by diabetes mellitus can stimulate formation microangiopathy?

A) Carbohydrate and protein.
B) Protein.
C) Lipid.

31. Why by treatment patients with diabetes mellitus is recommended moderate physical loading?
A) Stimulates reabsorption of glucose from intestine.
B) Glucose is used for synthesis of glycogen.
C) Glucose intensive used in muscles.

LITERATURE

1. Ovsyannikov V.G. "Pathophysiology" (questions and answers), 2005, p.242-265.

2. Nowak Th, J., Handford A.G. "Essentials of pathophysiology. Concepts and applications for Health Care Professionals". London, Singapore, 1999, p. 429-445, 453-463.

THEME OF LESSON: PATHOLOGY OF ENDOCRINE SYSTEM. DIABETES MELLITUS.

Practical work 2. Plan of practical work:

1. Tests and discussion of theoretical material - 60 min.

2. Fulfillment of practical work - 60 min.

3. Discussion of results - 15 min.

1. Scientific basing of topic. Pancreas produces two groups of hormones: hormones (insulin, glucagon), which regulate level of glucose in the blood and hormones (gastrin, somatostatin, pancreatic peptide), which regulate function of gastro-intestinal tract. Diabetes mellitus is the most often disease, which can develop by pathology of endocrine function of pancreas. 4% of world's population suffers from diabetes mellitus. Two types of diabetes mellitus are distinguished: type I and type II. They have different etiology. All types of metabolism are disturbed by diabetes mellitus (carbohydrate, protein, lipid). Complications of diabetes mellitus are the most dangerous for the life of patients. They are ischemic

heart, infarct of myocardium, ischemia of the brain, legs and hemorrhage in the brain, cataract, blindness hypertension, diabetic coma.

2. Aim of topic. Introduction to possible manifestations of diabetes mellitus, pathology of thyroid and adrenal glands.

3. Demonstration of educational film: «Endocrine diseases by children» (20 min).

QUESTIONS

1. Hormones of pancreas and its biologic effects.
2. Classification of diabetes mellitus.

Etiology of diabetes mellitus type I.

4. Etiology of diabetes mellitus type II.
5. Clinical and laboratory manifestations of diabetes mellitus.
6. Clinical and laboratory manifestations by diabetes mellitus due to disorder of carbohydrate exchange.
7. Clinical and laboratory manifestations by diabetes mellitus due to disorder of protein exchange.
8. Clinical and laboratory manifestations by diabetes mellitus due to impairment of lipid exchange.
9. Origin of macro- and microangiopathy.
10. Pathogenesis of diabetic coma.
11. Common principles of diabetes mellitus treatment.

LITERATURE

1. Ovsyannikov V.G. "Pathophysiology" (questions and answers), 2005, p.266-272.
2. Nowak T., Handford A. «Essential of pathophysiology. Concepts and applications for Health Care Professionals", London, Singapore, 1999, p. 446-453.

THEME OF LESSON: PATHOLOGY OF NERVOUS SYSTEM.

Plan of practical work:

1. Tests and discussion of theoretical material - 60 min.

2.Fulfillment of practical work - 60 min.

3. Discussion of results.

1. Scientific basing of topic. Nervous system supplies connection of the organism with external environment, adaptation of the organism to its change. Afferent nerves, analyzers and efferent nerves supply information, its analysis, synthesis and effects. Disturbance in function of nervous system is possible on different levels: sensitivity, analyzers, efferent nerves and can lead to disorder of sensitivity, locomotion, trophic vegetative functions, memory and so on. Such disturbances can be formed due to direct damage on different sections of nervous system or by reflex.

2. Aim of topic. Introduction to disorder of locomotion by pathology of nervous system.

3. Demonstration of educational film «Extrapyramidal disturbances» (18 min).

4. Demonstration of educational film «Epilepsy» (36 min).

QUESTIONS

1. Common etiology and common pathogenesis of disturbances of nervous system functions.

2. Disturbance of sensitivity on different levels.

3. Pathophysiology of reticular formation.

4. Pathology of hypothalamus.

5. Disorder of vegetative function of nervous system. Idea about vegetative crisis.

6. Disorder of locomotion. Pyramidal and extrapyramidal disturbances.

7. Etiology, pathogenesis and manifestations of central and peripheral paralysis.

8. Neurodystrophy, mechanism of its development.

9. Etiology, pathogenesis of spinal shock.

10. Etiology and manifestations of Brown-Sekar syndrome.

11. Possible manifestations of vagoinsular crisis.

12. Possible manifestations of sympatho-adrenal syndrome.

13. Theories of nervous dystrophy.

14. Morphological manifestations of nervous dystrophy on the level of cell and organ.

15. Etiology, pathogenesis of neurosis.

Tests

1. Mark process, which is not disorder of sensitivity.

A) Pain.
B) **Paresis**.
C) Hyperesthesia.
D) Anesthesia.

2. Why nervous dystrophy is considered as typical (standard) process?

A) It has different etiology.
B) **It has different etiology and united(common) pathogenesis.**
C) It can produce the same(similar) etiological factor.

3. Mark constitutional type of people, which can be predisposed to development neurosis.

A) Sanguinnic.
B) Melancholic.
C) Phlegmatic.

4. What process is not neurosis?

A) Neurasthenia.
B) Encephalitis.
C) Hysteria.

5. What does mean «anesthesia»?

A) Decrease sensitivity.
B) Absence temperature sensitivity.
C) Absence all types sensitivity.

6. When can form syndrome of «differentiation»?

A) By cut locomotors nerve.
B) By cut sensitive nerve.

7. When can form syndrome of Braun-Secar?

A) Complete cut of spinal cord.
B) Damage medulla oblongata.
C) Cut a half of spinal cord from left or right side.

8. By damage of what section CNS can form bulbar paralysis?

A) Cortex of the brain.
B) Spinal cord.
C) Hypothalamus.

D) Medulla oblongata.

9. What types of sensitivity are disordered by damage of thalamus?

A) Tactile.
B) All type of sensitivity on opposite side.
C) Only Pain.

10. How is named paralysis by damage motor neurons of spinal cord and locomotive nerves?

A) Spastic.
B) Cerebral.
C) Atrophic (weak).

11. Can appear hyper- or hypo kinesis by damage of extrapyramidal system?

A) Yes.
B) Not.

12. What is base of spinal shock pathogenesis?

A) Superiority processes of excitation in spinal cord.
B) Superiority processes of inhibition in spinal cord.

13. Is the depending manifestations of spinal shock on level of damage spinal cord?

A) Yes.
B) Not.

14. Trophic function of nervous system supplies structure and function of cells and tissues due to:

A) Regulation of metabolism.
B) Regulation of blood circulation.
C) Regulation of endocrine system.
D) All above mentioned.

15. How is changed quantity of catecholamines (adrenaline and noradrenaline) in the blood by extreme excitation and aggression?

A) Decrease.
B) Increase.
C) It is not change.

16. How can change quantity of catecholamines in the blood by depression?

A) Increase.
B) Decrease.
C) It is not change.

17. How psychic trauma does influence on secretion of stomach juice and its acidity?

A) Increase secretion and decrease acidity.
B) Inhibit secretion and decrease acidity.
C) Inhibit secretion and increase acidity.

LITERATURE

1. Ovsyannikov V.G. "Pathophysiology" (questions and answers),

2005, p.273-284.

2. Nowak T., Handford A. «Essential of pathophysiology». Concepts and applications for Health Care Professionals, Second Edition, London, Singapore, 1999, p.515-629.