PATHOLOGICAL PHYSIOLOGY

PRACTICAL PART

Student _____

Group number _____

Teacher _____

Minsk BSMU 2015

МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ
БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ
КАФЕДРА ПАТОЛОГИЧЕСКОЙ ФИЗИОЛОГИИ

ПАТОЛОГИЧЕСКАЯ ФИЗИОЛОГИЯ

PATHOLOGICAL PHYSIOLOGY

Практикум

4-е издание

Минск БГМУ 2015

УДК 616-092.18(811.111)-054.6(076.5) (075.8)
ББК 52.52 (81.2 Англ-923)
П20

Рекомендовано Научно-методическим советом университета в качестве практикума 16.09.2015 г., протокол № 1

А в т о р ы: Ф. И. Висмонт, В. А. Касап, С. А. Жадан, А. А. Кривчик, Е. В. Леонова, Т. В. Короткевич, Л. С. Лемешонок, А. В. Чантурия, Т. А. Афанасьева, В. Ю. Перетятько, О. Г. Шуст, Н. А. Степанова, К. Н. Грищенко, Э. Н. Кучук, Д. М. Попутников, Е. В. Меленчук

Р е ц е н з е н т ы: член-корр. НАН Беларуси, д-р мед. наук, проф. каф. нормальной физиологии Л. М. Лобанок; д-р мед. наук, проф. каф. патологической анатомии
М. К. Недзьведзь

Патологическая физиология = Pathological physiology : практикум / Ф. И. Висмонт [и др.]. – 4-е изд. – Минск : БГМУ, 2015. – 191 с.

П20

ISBN 978-985-567-304-1.

Содержит описания и протоколы оформления лабораторных работ по основным разделам курса патофизиологии. Представлена информация по следующим разделам: патофизиология системы крови, нарушение сердечного ритма, кислотно-основного состояния, наследственности и изменчивости организма. Первое издание вышло в 2010 году.

Предназначен для студентов 2–3-го курсов медицинского факультета иностранных учащихся для самостоятельной подготовки к занятиям, выполнения и оформления лабораторных работ по предмету.

УДК 616-092.18(811.111)-054.6(076.5) (075.8)
ББК 52.52 (81.2 Англ-923)

ISBN 978-985-567-304-1

SECTION I
GENERAL NOSOLOGY

LESSON 1. INTRODUCTORY LESSON. THE SUBJECT, TASKS, METHODS OF PATHOLOGICAL PHYSIOLOGY

Date: «____» _____ 20___

The purpose of the Lesson: to consider the subject of studying, the essence and tasks of Pathological Physiology as a science and discipline, its place in the system of medical training; legitimacy and validity of experimental research, its significance for understanding the disease and developing the principles of treatment and prophylaxis; modeling of diseases, requirements to the experiment and researcher, ethical aspects of experimenting on animals.

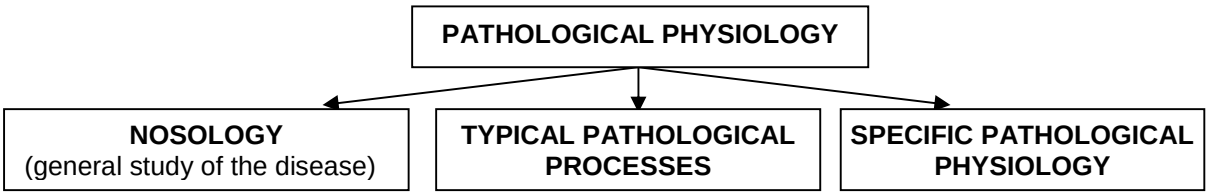
The subject of studying includes the most common basic laws and the mechanisms underlying the basis of resistance of the organism, origin, development and outcomes of pathological processes — diseases.

The object of studying is a diseased person, disease as a whole.

The method of studying is the pathophysiological experiment.

The tasks of pathological physiology include studying etiology and pathogenesis of diseases, mechanisms of their manifestations as well as the formulation of principles of diagnosis, treatment and prophylaxis.

The course of the subject is divided into 3 parts:



«Pathological physiology is a science about vital activity of a diseased human or animal organism, i. e. physiology of a diseased organism».

A. D. Ado, academician of the Russian Academy of Medical Science

«… Pathological physiology studies the essence, the natural origin of diseases: the reasons of their occurrence, laws of their development and outcomes. The term «Pathological physiology» follows from: pathos — suffering, illness; physis — the nature, essence; logos — the study, science)».

P. F. Litvitsky, Prof., Head of the dept. of Pathological physiology MMA. (I. M. Setchenov)

«The pathophysiologist distracts from particulars, trying to find those common, which characterize large groups of diseases and the disease in general. An ultimate goal of pathological physiology is revealing the laws of the disease development».

N. N. Zajko, Professor, corresponding member of the USSR AMS

Pathological physiology is «a basis of medical professional thinking».

From a preamble of the CART charter

Tasks:

- To study the significance of pathological physiology as a science, its relationship with other medical biological and clinical disciplines the significance for theoretical and clinical medicine.
- To get acquainted with the staff of the department, its history, the direction of research work, the activity of SCC and forms of academic scientific work of students.
- To find out the significance of the experiment for etiology and pathogenesis of human diseases, in developing methods of their treatment and prophylaxis; to characterize peculiarities of pathophysiologycal experiment.
- To study the principles of modeling pathological processes, basic requirements to the experiment and the researcher as well as requirements to recording protocols; moral-ethical problems associated with performing experiments on animals.
- To get acquainted with peculiarities of keeping experimental animals, methods of treating them, techniques of carrying out a series of manipulations with materials presented in educational videos, and also with some experimental models of cardiovascular pathology developed at the department of pathological physiology of BSMU.
- To undergo a safety precautions and instructions for doing practical works at laboratories of the department.

Work 1. DEMONSTRATION OF TEACHING VIDEOS

1. Practical recommendations for dealing with laboratory animals.
2. Modeling of cardiovascular pathology.
3. Alternatives in medical and biologic education.

Control questions:

1. The subject and tasks of pathological physiology. Its place in the system of the higher medical education. Pathological physiology as a theoretical basis of modern clinical medicine.
2. General characteristic of three basic parts of Pathological physiology.
3. Modeling of diseases. Sharp and chronic experiment (Claude Bernard, I. P. Paulov).
4. The requirements to the experiment and the researcher.
5. The basic conditions of performing a biological experiment.
6. Moral-ethical aspects of experimenting on animals.

The teacher's signature:

LESSON 2. ETIOLOGY AND PATHOGENESIS. PATHOGENIC EFFECTS OF ENVIRONMENTAL FACTORS. ELECTROTRAUMA

Date: «____» _____ 20___

The purpose of the Lesson:

– To understand the bases of etiology and pathogenesis. To study the peculiarities of the electric current damaging effects on the organism.

Tasks:

– To get acquainted with methods of performing experiments and their results; to analyze data of experimental protocols, to formulate conclusions.

– To get acquainted with characteristic consequences of electrotrauma in humans (demonstration of slides).

– Solution of situational tasks.

– Test control over the topic of the Lesson.

Answer the questions:

1. Give the definition of electric current

2. List the features of electric current:
   –
   –
   –
   –
   –
   –
   –

3. List the features which determine severity of electric current damage:
   –
   –
   –

4. What are the nonspecific effects of an electric current?
   –
   –
   –

5. What are the specific effects of an electric current?
   –
   –
   –
   –

6. List the biological effects of electric current:
   –
   –
   –
   –

7. List the electrochemical effects of electric current:
   –
   –
   –

8. List the electro thermal effects of electric current:
   –
   –

9. List the electromechanical effects of electric current
   –
   –

10. What are the causes of death from electric current?
   –
   –
   –
   –

11. Give the definition of electric shock

12. Give the characteristic of electric shock phases. Fill the table:

Table 1

The stage of excitation	The stage of inhibition
–	–
–	–
–	–
–	–
–	–
–	–

Work 1. DEPENDENCE STUDY OF THE SEVERITY OF ELECTRIC CURRENT INJURY AND ITS EXPOSURE DURATION

Experimental technique

To carry out the experiment 8–10 frogs are connected to each other with their forelegs. «The live chain» of frogs is suspended to a wooden stand. Needle electrodes are stuck into the forelegs of last frogs. The reflex time is taken for every frog by Turki. Then electric current from the city network (a voltage 220 V) is being passed for 2 sec., and the reflex time is taken again. In 3–5 min. the electric current from the city network is being passed repeatedly through the chain of frogs for 60 sec, and the reflex time is again recorded.

Results of the experiment

№	The reflex time by Turki (in seconds)			Note
	Initial data	Electric current exposure		
		2 seconds	60 seconds	
1	1	5	15	Short-term convulsive muscular contractions of extremities and the trunk, squeak
2	2	3	10	
3	2	3	20	
4	1	2	10	
5	1	2	9	
6	1	2	10	
7	1	2	15	
8	1	2	17	
9	1	3	12	
10	1	4	16	

Conclusions:

1. In what way and why does the reflex time change after electric current exposure?

2. In what way does the reflex time depend on duration of electric current exposure? Why?

Work 2. DEPENDENCE STUDY OF THE SEVERITY OF ELECTRIC CURRENT INJURY AND THE WAY OF ITS PASSAGE THROUGH THE ORGANISM

Experimental technique

Three mice of the same sex and weight are fixed separately by ligatures on special little tables. General condition of mice is estimated, respiration rate is counted. Electrodes are fixed:

– In the **1-st** mouse — to hind paws (switched on electric current will pass through hind extremities of the animal);

– In the **2-nd** — to auricles, thus providing the passage of the current through the head of the animal;

– In the **3-rd** mouse — to the fore left and hind right paws (switched on electric current will pass through the heart).

When mice calm down after the electrodes have been fixed, electric current from a city network is being consequently passed through the organism of the experimental animal for 1–2 seconds (the duration of exposure is strictly dosed, which is provided by a special push-button breaker).

Results of the experiment

№ mice	Current passage way	The general condition after electric current exposure	Respiration rate and breath character	Defecation, urination	Survival rate	Notes
1	Hind extremities	Excitation, short-term (1–2 sec.) convulsive muscular contractions of hind extremities	Acceleration	+	Survives	In 2–3 min the general condition returns to the initial state
2	The brain	General tonic spasms, «a pose of the bull», then clonic spasms. In 1–2 min convulsive muscular contractions have stopped. General inhibition	Short-term arrest, then acceleration	+	Survives (up to 20 %)	In 5–8 min the general condition returns to the initial state
3	The heart	General tonic spasms	Arrest	+	Dies	On autopsy of the thorax fibrillation of the heart is observed

Conclusions:

1. Which way of the electric current passage through the organism is most dangerous and why?

Control questions:

1. The notion of etiology and pathogenesis. The significance of reasons and conditions of disease development. The essence of monocausealism, conditionalism and constitutionalism.
2. Peculiarities of electric current as a damaging factor.
3. Factors affecting injury severity for the organism exposed to electric current.
4. Kinds of electric current injuries (local and general, specific and nonspecific) and their characteristic.
5. The reasons of lethal electrocutions and their mechanisms. «Imaginary death».
6. Rules of giving first-aid to an electrocuted person.

The teacher's signature:

LESSON 3. REACTIVITY OF THE ORGANISM AND ITS ROLE FOR PATHOLOGY. TYPICAL IMPAIRMENTS OF IMMUNOLOGIC REACTIVITY

Date: «____» _____ 20___

The purpose of the Lesson:

– To study factors and the mechanisms determining reactivity and resistance of the organism, their role in pathology; to discuss possible ways of directed effect on reactivity and resistance. To study typical impairments of immunologic reactivity.

Tasks:

– To get acquainted with conditions and results of Konstantinov's and Maystrah's experiments while studying the effect of the central nervous system functional state on reactivity of the organism.

– To draw graphs and diagrams on the basis of experimental protocols data (tab. 1–2) and illustrative material for the topic presented in tables showing basic experimental results.

– To answer questions and formulate conclusions on the basis of experimental results presented as graphs and diagrams.

Work 1. DYNAMICS STUDY OF RESPIRATION AND EXCHANGE PROCESSES CHANGES IN THE DEVELOPMENT OF HYPOXIA IN MICE WITH VARIOUS FUNCTIONAL STATE OF THE CENTRAL NERVOUS SYSTEM

(experiments № 1, 2)

Experiment № 1

Research is performed on white mongrel mices of identical weight. Hexenal (i/p, 100 mg/kg) is injected to one of them, then the mouse falls asleep after 7–10 min. The occurrence of narcosis is determined by disappearance of a corneal reflex. The sleep lasts for 1.5–2.0 h.

Both mice — an intact, unnarcotized one (control) and narcotized (tested) — are placed in two large-mouthed flasks of identical capacity (100 ml). The flasks are simultaneously closed by rubber corks with subsequent hermetic sealing by paraffin. We observe the behavior of mice, count respiration rate every 3–5 min, and also register their life expectancy in hermetically closed space. Later on, immediately after death of animals we determine the contents of O_2 and CO_2 in flasks.

Table 1

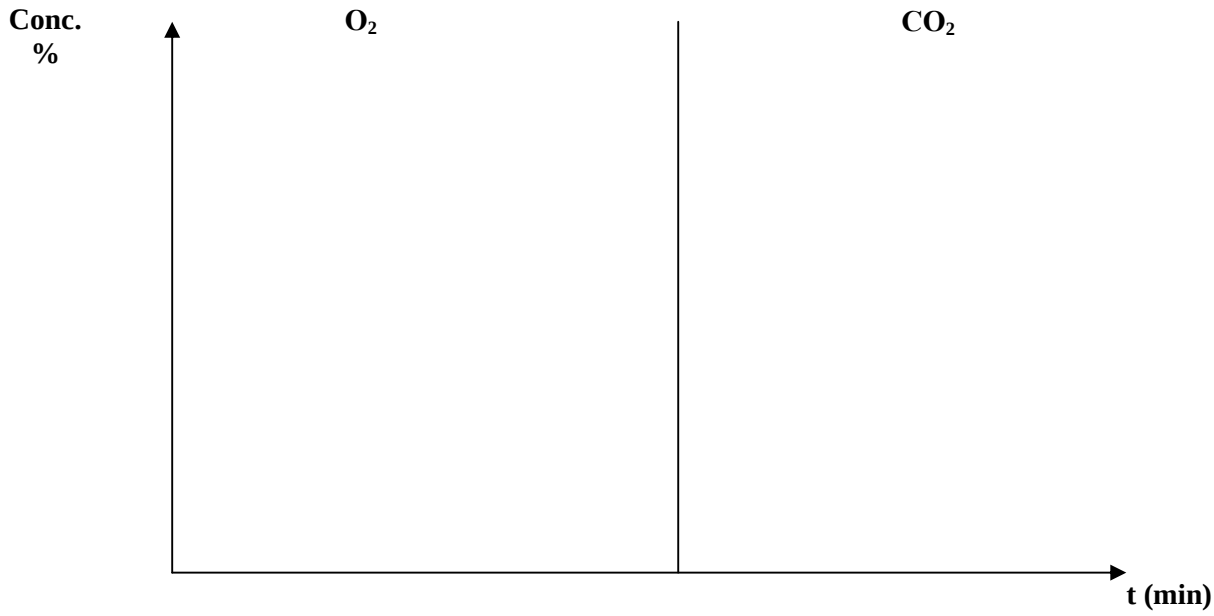
Respiration rate (RR), the general state and life expectancy of control and tested mice

Min	The control		The Experiment	
	RR/min	General state	RR/min	General state
0	118	The mouse is quiet. Regular respiration	108	The mouse is asleep. Regular respiration

Min	The control		The Experiment	
	RR/min	**General state**	**RR/min**	**General state**
1	132	Oriented motor reaction: the mouse stands up on hind paws, sniffs at a flask	108	The mouse is asleep lying on one side
3	120	The mouse has calmed down	108	No changes
6	122	Periodically stands up on hind paws, rubs its muzzle	100	No changes
9	140	The mouse is anxious. It stands up on hind paws more often. The breath has accelerated and is deeper	84	Is asleep. Respiration is calm
12	162	The anxiety of the mouse increases. It makes sharp movements. It pulls at the cork with paws. Cyanosis of ears, the nose tip, paws	72	The mouse sleeps. The respiration is regular
15	180	Sharp anxiety. Cyanosis. Breathlessness	68	No changes.
18	176	Motor activity is weakened. Sharp cyanosis	62	Cyanosis signs of ears, the nose tip, paws have appeared
22	22	The mouse is lying on one side. Breath is intermittent	50	Cyanosis
23	22	Spasms, tail reaction, defecation, urination	50	Marked cyanosis
24	–	Respiratory arrest	48	–
35	–		12	–
35	–		12	–
45	–		6	–
48	–			Respiratory arrest
	Gas mixture content in the flask: O_2 = 7,1 %; CO_2 = 11,8 %		**Gas mixture content in the flask: O_2 = 3,4 %; CO_2 = 14,6 %**	

1. Construct a graph of respiratory rate (RR) changing in the control and teste mice in dynamics of the experiment.

2. Construct a graph of changing [c] O_2 and [c] CO_2 in the control and tested mice on the basis of the initial and final concentration in the flasks.



Answer the questions:

1. Explain the mechanisms of tachypnea development in the control mouse on the 1–20 min of the experiment.

2. Explain the reasons of tachypnea absence in the tested mouse in the same terms of the experiment.

3. Give pathogenic and prognostic estimation of tachypnea in the animal under hypoxia-hypercapnia.

4. Explain the reason of bradypnea and subsequent apnea in the control and tested mouse on the last minutes of the experiment.

5. Calculate and compare an average speed (V) concentration changing of oxygen and carbon dioxide in the flasks with a control (V_1) and tested (V_2) mice, having assumed the initial concentration of O_2 and CO_2 equals to 21 % and 0,03 %, accordingly:

$V_1 = d_{O_2/t_1} =$

$V_1 = d_{CO_2/t_1} =$

$V_2 = d_{O_2/t_2} =$

$V_2 = d_{CO_2/t_2} =$

6. Explain possible mechanisms of decreasing the consumption of oxygen (and, accordingly, power expenditures) under the effect of narcosis in the tested mouse.

7. Explain possible mechanisms of narcosis effect on increasing life expectancy of the tested mouse under hypoxia-hypercapnia.

Experiment № 2

In the second experiment both mice — a narcotized and unnarcotized ones — are placed into one flask with the capacity of 20__ ml. The flask is hermetically closed. In this experiment both mice are in the same gas environment. After death of the control mouse we take some air from the flask for analysis of gas content. The results of the experiment are presented in the following table.

Table 2

Respiration rate (RR), general state and life expectancy of the control and tested mice

Min	Control		Tested	
	RR/min	General state	RR/min	General state
0	120	The mouse is quiet	102	The mouse is asleep. The respiration is regular
1	136	Oriented reaction of the mouse	102	The mouse is asleep
3	110	The mouse has calmed down	102	No changes
10	120	Periodically the mouse stands up on hind paws, sniffs at the cork. It pulls at it	98	–

Min	Control		Tested	
	RR/min	**General state**	**RR/min**	**General state**
15	148	The same behavior. Cyanosis signs have appeared	98	–
20	160	Cyanosis increases. Signs of motor activity have increased. Respiration is deeper and more accelerated	76	Weak cyanosis signs
25	168	The same condition	70	No changes
28	150	The mouse has fallen down. Periodically it jumps up. Sharp cyanosis	58	No changes
31	–	The mouse is lying on one side. Sharp cyanosis. Intermittent respiration. Spasms. Agonal breathing. Respiratory arrest	50	The mouse is asleep. Sharp cyanosis
32	–	Spasms. Agonal breathing	50	No changes
33		Respiratory arrest	44	–
38			36	–
43			20	–
46			2	–
47				Respiratory arrest
	Gas mixture content in the flask: O_2 = 7,1 %; CO_2 = 11,8 %		**Gas mixture content in the flask: O_2 = 5,6 %; CO_2 = 12,5 %**	

1. Construct a graph of changing the respiration rate (RR) in the control and tested mice in dynamics of experiment.

Answer the questions:

1. Draw a conclusion on the speed significance of developing hypoxia-hypercapnia for reactivity of the organism and life expectancy of animals.

2. What is the interrelation between reactivity and resistance of the organism?

3. Specify two basic strategies of increasing nonspecific resistance of the organism.

4. Which of these two strategies of a survival in extreme conditions is used in experiments of Konstantinov and Maistrah?

5. What is a possible practical application of these experimental results?

Control questions:

1. Definition of the notions «reactivity» and «resistance». Their relationship.
2. Forms of reactivity (normergy, hypoergy, hyperergy, dysergy).
3. Basic parameters of reactivity, their characteristic, mechanisms, master factors.
4. Classification of reactivity.
5. Phylo-and ontogenesis of reactivity and resistance. Peculiarities of reactivity depending on sex and age.
6. Factors lowering nonspecific resistance of the organism.
7. Ways and methods of increasing nonspecific resistance:
   a) in preserving or increasing the level of vital activity;
   b) in decreasing vital activity, losing the ability of independent existence.
8. Immunologic reactivity. Understanding of immunopathological processes.
9. Immunodeficient conditions. Classification, etiology, pathogenesis, manifestations.
10. Allergy, definition of this notion. Forms of allergic reactions, their characteristic. Stages of allergic reactions. Mediators of allergies, their main effects.

The teacher's signature:

LESSON 4. THE ROLE OF HEREDITY IN PATHOLOGY

Date: «____» _____ 20___

The purpose of the Lesson:

– To study general issues of etiology and pathogenesis of hereditary pathologic forms, types of their inheritance, principles of their prevention and treatment. To get acquainted with the most common hereditary diseases and development abnormalities.

Tasks:

– To solve situational tasks in medical genetics.

– To study the genotype and clinical manifestations of hereditary pathology with tables and slides. The description of slides is applied.

– Computer control over the topic of the Lesson.

SITUATIONAL TASKS

№ 1

What is the probability of a child being born **syndactylism (fused fingers)** in the family, where the father has this developmental defect, while the mother and the first child have a normal structure of fingers?

Character	Gene	Genotype

№ 2

Determine the birth probability of **short-fingered children** in the family where parents have a developmental defect and are heterozygotes.

Character	Gene	Genotype

№ 3

In a family, where both spouses suffer from **achondroplasia**, a normal child was born. What is the birth probability of healthy children?

Character	Gene	Genotype

№ 4

Determine the birth probability of children with **otosclerosis** in the family, in which parents are heterozygous by the analyzed character (penetrance of 30 %).

Character	Gene	Genotype

№ 5

Determine the birth probability of children with **astigmatism** in the family, where father is heterozygous and mother does not suffer from astigmatism.

Character	Gene	Genotype

№ 6

Homozygous individuals by a gene of **sickle-cell disease** usually die before puberty, heterozygotes are viable, anemia is revealed during hypoxia. What is the birth probability of phenotypically and genotypically healthy children, if both parents are heterozygous by the analyzed character?

Character	Gene	Genotype

№ 7

In genetic consultation a woman informed the doctor, that her sister was ill with a severe form of a **sickle-cell anemia**, she herself had never been ill with anything, her husband is healthy. The woman wonders, whether the probability of this disease being passed down for her children is great on? To answer this question a biochemical test of hemoglobin types was carried out; it revealed that the woman's blood contains: HbA — 70 % and HbS — 28 %; and her spouse's blood: HbA — 98 % and HbS — 0 %.

Character	Gene	Genotype

№ 8

What is the birth probability of sick children in the family where one of the parents is heterozygous by a gene of **phenylketonuria**, and another is healthy (his parents, brothers and sisters are healthy)?

Character	Gene	Genotype

№ 9

In genetic consultation pregnant woman C. informed, that her sister was ill with **phenylketonuria**, but she herself didn't suffer from this pathology. C.'s husband was healthy. There were kindred marriages between close relatives in her husband's family, but none of them was ill with phenylketonuria. Is there any danger for her child to get this disease? What is the probability of it? What is the role of sex? Is it possible to treat such a disease? Make up possible genealogical trees and answer the above questions.

Character	Gene	Genotype

№ 10

Successes of modern medicine allow to prevent the development of **galactosemia** and to avoid consequences of metabolic impairments. What is the birth probability of sick children in the family where one of the spouses is homozygous by a gene of galactosemia, but the development of his disease is prevented by diet, and the other is heterozygous with a galactosemia gene?

Character	Gene	Genotype

№ 11

What descendants can be expected from heterozygous parents on a gene of **alcaptonuria**?

Character	Gene	Genotype

№ 12

Determine the birth probability of sick children with hepatocerebral dystrophy (**Wilson's desease**) in the family where the father is sick, and the mother is healthy (her parents, brothers and sisters are healthy).

Character	Gene	Genotype

№ 13

In the family, where one of the spouses is an albino and the other is normal, binovular twins were born, one of which is normal concerning the analyzed character, and the other is an albino. What is the birth probability for the following child to be an albino?

Character	Gene	Genotype

№ 14

Healthy woman H., whose father was ill with **colour blidness**, and mother was healthy, referred to genetic consultation with a question, whether is there any danger for her children to get this disease. The spouse of this woman is healthy. What could you answer to this woman? Draw a possible family trees.

Character	Gene	Genotype

№ 15

A man, ill with **hemophilia A**, married a healthy woman whose father suffered from hemophilia A. Determine the birth probability of healthy children in this family?

Character	Gene	Genotype

№ 16

Healthy woman H., whose father is ill with **hemophilia A** and mother is healthy, referred to genetic consultation with a question: whether probability for her grandsons to get this disease is great? Spouse H. and their three children — a son and two daughters — are healthy. What is the type of inheritance and what is the development of hemophilia A caused by? Is the development of a lethal form of the given pathology possible? What is the probability for her grandsons to get this disease in her son's lineage?

Character	Gene	Genotype

№ 17

A man, sick with **hemophilia B**, married to a healthy woman (in whose family nobody was ill with hemophilia), referred to the doctor with a question: what is the probability of their children to get this disease?

Character	Gene	Genotype

№ 18

In the family where the parents have **hypoplasia of dental enamel**, a son was born with normal teeth. What is the birth probability of sons with normal teeth?

Character	Gene	Genotype

№ 19

What is the birth probability of children with the absence of lateral incisors if the parents have this dental abnormality and they are heterozygous by the analyzed character?

Character	Gene	Genotype

№ 20

How many bodies of sex chromatin are there in people with genotype OX? XXУ? XXX? XXXУ? What is the sex of these people and what are they ill with?

№ 21

The karyotype of the given patient is characterized by the presence of 3 sex chromosomes. It is associated with a large stature, eunuch-like constitution, spermatogenesis impairment, microorchia, psychical impairment. What is the name of the mentioned syndrome? What is the karyotype of the mentioned syndrome?

№ 22

In patient M., height of 153 cm, there skin fold on the neck, «sphinx» neck, primary amenorrhea, sterility. There are congenital defects of the heart and kidneys. What is the name of the mentioned syndrome? What is the karyotype of the mentioned syndrome?

ADDITIONAL INFORMATION

Inheriotance type	Pathology form
1. Autosomal-dominant (A-D)	Polydactylism Brachydactylism Dactylion Curvature of fingers, nails Anonychia (underdevelopment of nails) Absence of lateral incisors Short-sightedness Far-sightedness Astigmatism Otosclerosis Achondroplasia Family hypercholesteremia Chorea of Huntington Polyposes of the large intestine Neurofibromatosis
2. Autosomal-recessive (A-R)	Crescent — cellular anemia (by incomplete domination) Galactosemia Phenylketonuria Alcaptonuria Albinism Glycogenoses Mucoviscidosis Wilson-Konovalov disease (hepato-cerebral dystrophy) Adrenogenital syndrome Congenital deaf-muteness Microcephaly

Inheriotance type	Pathology form
3. Dominant X-linked (D-X)	Frontal-nasal dysplasia Hypoplasia of dental enamel Cataract Rickets, resistant tovitamin D
4. Recessive X-linked (R-X)	Hemophilia A and B Daltonism Hypogammaglobulinemia Duchenne's muscular dystrophy Hemeralopia
5. Hollandric Y-linked (H-Y)	Escessive hairiness of auricles Azospermia
6. Mitochondrial (M)	Leber's optic atrophy Mitochondrial encephalopathy Myoclonal epilepsy Cardiomyopathy

PRINCIPLES OF FIGHTING AGAINST MUTATIONS

– **Technological** — Creation of wasteless productions.

– **Componental** — Excluding the production of substances which can be mutagens (pesticides, medicines, etc.).

– **Compensatory** — Increasing the resistance of the genetic system to environmental factors (using anti-mutagens).

Control questions:

1. Medical genetics, its tasks.
2. Classification of diseases taking into account the specificity of heredity and environment in their development.
3. Hereditary and congenital forms of pathology.
4. Classification principles of hereditary forms of pathology.
5. Phenocopies. The definition, causes of development. Examples.
6. Methods of studying hereditary forms of pathology: clinical-genealogical, cytogenetic, twin, biochemical, dermatoglyphics, demographic-statistical, experimental.
7. Mutation, the definition of the notion. Kinds of mutation. Mutagen and antimutagen factors. Ways of preventing mutations.
8. Etiology of hereditary forms of pathology.
9. General development mechanisms of hereditary diseases and abnormalities of development.
10. Mono- and polygenic hereditary diseases. Hereditarily determinant metabolic diseases: alcaptonuria, phenylketonuria, hepatocerebral dystrophy, family hypercholesterinemia, galactosemia, etc. Pathological heredity linked with sex (daltonism, hemophilia A and B, hypoplasia of dental enamel, etc.). Type of

inheritance, causes, mechanisms of development, semiology, principles of prophylaxis.

11. Chromosomal diseases: Down's syndrome, Patau syndrome, Edwards' syndrome, Klinefelter syndrome, a trisomy syndrome of X-chromosomes, Shereshevsky-Turner's syndrome, a syndrome of «the cat's cry». Causes of development, karyotype, semiology.

12. Hereditarily determined diseases and abnormalities of dentition (for the faculty of dentistry).

13. Pathology of intra-uterine development. Gametopathies, blastopathies, embriopathies, phetopathies, still-birth rate.

14. Relationship of fetal pathology with harmful effects on the maternal organism. A pathogenetic role of hypoxia, hormonal and metabolic impairments, infection, industrial and household intoxications; harmfulness of alcoholism and smoking (for pediatric faculty).

15. Principles of prophylaxis and treatment of hereditary diseases and developmental defects, diseases with hereditary predisposition.

16. Topical tasks of environmental protection and protection of labour for prophylaxis of hereditary congenital diseases for the faculty of medical prevention).

The teacher's signature:

LESSON 5. TOPICAL PROBLEMS OF GENERAL NOSOLOGY

Date: «____» _____ 20___

The purpose of the Lesson:

– To learn basic points of the study about diseases, to consolidate and check knowledge received by the students at the lessons while studying the section «General nosology» by manuals.

Tasks:

– To get acquainted with the contents of the album «Topical problems of general nosology».

– To make a conclusion (comment) on a number of thematic series of slides, illustrating various aspects of general nosology.

– Test control over the topic «General nosology».

– Solution of situational tasks.

Answer the questions:

1. Put down a definition of the notion «disease», given at the lesson.

Disease is …

2. Beginnings of the disease is caused by:

1 –

2 –

3 –

3. Stages of the disease:

1 –

2 –

3 –

4 –

4. Outcomes of the disease:

1 –

2 –

3 –

4 –

5. Give the definition of *pathological reaction*:

6. Give the definition of *pathological process*:

7. Give the definition of *pathological condition*:

8. Name the factors that determine *specificity of pathological process*:

–

–

–

9. Name the factors that *determine selectivity of localization of basic structural-functional impairments*:

–

–

–

–

–

10. Give the characteristic of pathological reactions:

1 –

2 –

3 –

4 –

11. Give the characteristic of compensatory reactions:

1 –

2 –

3 –

4 –

5 –

12. Fill in table 1:

Table 1

Periods of a terminal condition

Periods	**Consciousness (+/-)**	**Corneal and pupillary reflexes (+/-)**	**Blood circulation state: BP, pulse**	**Character of respiration**	**Metabolic state**	**Duration**
I Preagonal						

Periods	Consciousness (+/-)	Corneal and pupillary reflexes (+/-)	Blood circulation state: BP, pulse	Character of respiration	Metabolic state	Duration
II Agonal						
III Clinical death						

13. Give the definition of adaptation:

14. Give the definition of compensation:

15. List of the structural basis and mechanisms of compensation:
    –
    –
    –
    –
    –

16. Fill in table 2:

Table 2

The comparative characteristic of physiological and pathological systems

Criterion of comparison	System	
	Physiological	Pathological
Biological Expediency		
The basic mechanism of system formation		
The role of feedback in functioning of the system		
The basic mechanism that stops the activity of the system		
The result of the system activity		

Control questions:

1. The definition of the notion «disease». Evolution of the idea about the disease essence on different development stages of medicine.

2. The notion of a pathological process, pathological reaction, a pathological condition. Interrelation between «pathological process» and «disease».

3. Interrelation between local and the general, specific and nonspecific in development of the disease.

4. Factors that determine specificity of pathological processes and selectivity of localization of basic structural-functional impairments.

5. Development stages of the disease, outcomes of the disease.

6. Terminal condition, its stages, characteristic. Laws of fading of vital functions. Main principles of reanimating the organism. Social-deontological aspects of reanimation. General laws of restoring vital functions. Post-reanimation disease (Postresuscitation disease).

7. The study about pathogenesis. The definition of «pathogenesis». Interrelation between etiology and pathogenesis. The notion of the main (initial) link in development of the disease. The role of vicious circles in the disease pathogenesis.

8. Integrity of a complex organism:

   a) interrelation of both the mental and somatic in norm and in pathology;

   b) verbal irritant as a pathogenic and therapeutic factor. Iatrogenias.

9. The notion of a pathological system (G. N. Kryzhanovsky). Its distinction from a physiological system. Biological significance.

10. The notion of a dual internally inconsistent nature of the disease.

11. The definition of «adaptation» and «compensation».

12. Pathological and compensatory reactions of the organism:

– Their general characteristic;

– Levels of formation, examples;

– Structural bases and functional mechanisms of compensation

– The role of the genetic system in developing compensatory reactions and phenomena of decompensation;

– The notion of cross adaptation and compensation;

– The «price» of adaptation and compensation.

13. The staging character of the disease. Dynamics and expressiveness of pathological and compensatory reactions of the organism in the process of the disease.

14. Stage dependence of therapeutic actions effect.

The teacher's signature:

LESSON 6. PATHOGENIC ACTION OF ENVIRONMENTAL FACTORS. DAMAGING EFFECT OF IONIZING RADIATION ON THE ORGANISM

Date: «____» _____ 20___

The purpose of the Lesson:

– To study pathophysiological aspects of radiation injuries, their nature, development mechanism, outcomes. To give a pathogenetic characteristic of various kinds of radiation injuries.

Tasks:

– To get acquainted with local and general manifestations of acute radiation sickness.

– Solving situational tasks.

– Test control over the topic of the Lesson.

Work 1. ACUTE RADIATION SICKNESS

Answer the questions:

Give a brief characteristic of basic syndromes developing in the given form of acute radiation sickness, and those manifestations that took place in this patient.

1. What is the typical form of acute radiation sickness?

2. List basic syndromes, characteristic of this form of acute radiation sickness?

3. What are the development mechanisms of these syndromes?

4. Pathogenic principles of correcting a marrowy form of ARS (acute radiation sickness).

5. Explain the development mechanism of agranulocytosis under the action of ionizing radiation.

6. In what way does the agranulocytosis approach term depend on the absorbed doze of irradiation?

7. Why does the abortive rise of leukocyte count in blood develop in small irradiation dozes?

8. Explain the restoration mechanism of normal leukocyte count in blood after the agranulocytosis period.

Fill in table

Table

Severity estimate of a marrowy form of ARS by the earliest prognostic criterion (vomiting)

ARS severity	Occurrence time of vomiting (minutes-hours) from the moment of irradiation	Frequency rate of vomiting
Mild		
Average		
Severe		
The most severe		

Control questions:

1. Ionizing radiation. The definition, general characteristic.
2. Peculiarities of ionizing radiation effect as a damaging factor.
3. Dose characteristics of ionizing radiations.
4. Radiosensitivity of cells and tissues. Main factors. The notion of critical organs.
5. Reversible and irreversible radiation-induced injuries of cells; destruction of cells, its kinds.
6. Radiation injuries. Etiology. Classification. General characteristic.
7. Pathogenesis of radiation injuries.
8. Acute radiation sickness. Its forms, course, outcome.
9. The characteristic of the formation period of a typical marrowy form of acute radiation sickness, basic clinical syndromes, therapeutic principles.
10. General characteristic of chronic radiation sickness; peculiarities of etiology and pathogenesis, clinical manifestations, basic clinical syndromes.
11. Radiation sickness due to internal irradiation, its peculiarities.
12. Local effect of ionizing radiations.
13. Remote consequences of small doses of ionizing radiation on the organism.

The teacher's signature:

SECTION II
TYPICAL PATHOLOGICAL PROCESSES

LESSON 1. TYPICAL IMPAIRMENTS OF PERIPHERAL BLOOD CIRCULATION. ARTERIAL AND VENOUS HYPEREMIA. ISCHEMIA

Date: «____» _____ 20___

The purpose of the Lesson:

– To study the incidence causes, development mechanisms, basic manifestations, outcomes and the significance for the organism of both arterial and venous hyperemia and ischemia.

Tasks:

– To get acquainted with conditions of experimental work, to take part in accomplishment of experiments.

– To analyze experimental data, to present them in figures, to formulate conclusions, to give them a written substantiation.

– Solving situational tasks.

Work 1. STUDYING ARTERIAL HYPEREMIA ON A RABBIT'S EAR

We investigate manifestations of arterial hyperemia on a white rabbit, that appears on mechanical and chemical irritation of the ear skin. For this purpose we rub its ear with dry or slightly moistened cotton wool with xylol and compare both ears of the rabbit under the passing light. We notice characteristic changes of blood circulation. We sketch the initial condition of vessels and the revealed changes.

Fig. 1. Arterial hyperemia of a rabbit's ear:
1 — the control (an intact ear); 2 — heat exposure (arterial hyperemia)

Conclusion:

Specify the development mechanism of arterial hyperemia in this experiment.

Work 2. STUDYING VENOUS HYPEREMIA ON A RABBIT'S EAR

A cork is inserted into an auricle of the rabbit so that the groove has fallen on the central artery of the ear. Then, using a ligature, the rabbit's ear is tightly fixed to the cork resulting in the impairment of blood circulation, i. e. the outflow of blood by veins is impeded. In 30–40 min we notice the signs of venous hyperemia to appear. We describe it and sketch them.

Fig. 2. Venous hyperemia a rabbit's ear:
1 — the control (an intact ear); 2 — impairment of venous outflow (venous hyperemia)

Conclusion:

Specify the development mechanism of venous hyperemia in the given experiment.

Work 3. STUDYING ISCHEMIA ON A RABBIT'S EAR

Local anemia is caused by squeezing the central artery of a rabbit's ear. In passing light we observe blood-filling changes in vessels of an ischemic ear. We notice a temperature difference between an ischemic and intact ear. We draw schematic changes of vascular pattern of the rabbit's ear.

Fig. 3. Ischemia of a rabbit's ear:

1 — the control (an intact ear); 2 — squeezing of the central ear artery (ischemia)

Conclusion:

Specify the development cause of ischemia in the given experiment.

Describe basic visible manifestations of peripheral blood circulation impairments in this experiment, having filled in the table:

Peripheral blood circulation impairment	**Color of integuments**	**Vascular pattern**	**Pulsation of vessels**	**T °C of the ear skin**	**Organ volume (edema +/-)**	**Tissue turgor**	**Characteristic sensations (a pain +/-)**
Arterial hyperemia							
Venous hyperemia							
Ischemia							

Answer the questions:

1. List the basic biologically active substances affecting the vascular lumen and the amount of peripheral blood flow.

Vasodilatators —

Vasocontrictors —

2. List main factors for an outcome of acute ischemia:

3. Fill in the table:

№	**The type of collaterals between arteries**	**Organs with prevalence of the given type of collaterals**	**An ischemia outcome in these organs at full occlusion of arteries**
1	Functionally absolutely sufficient		
2	Functionally relatively insufficient		
3	Functionally absolutely insufficient		

Control questions:

1. Typical forms of impairments of peripheral blood circulation. General characteristic.

2. The definition of the notion of arterial and venous hyperemias, ischemia; external manifestations, the reasons and development mechanisms, outcomes.

3. Changes in tissues in the area of arterial and venous hyperemias and ischemias, their significance and possible consequences.

4. The state of microcirculation in peripheral blood circulation impairments: ischemia, arterial and venous hyperemia.

5. Compensatory reactions in the impairments of local blood circulation. Post-ischemic reperfusion. Mechanisms of triggering and developing collateral blood circulation . Types collaterals. Cerebral and cardiac steal syndromes.

6. General changes in the organism during impairments of peripheral blood circulation (arterial and venous hyperemias, ischemia) in vital organs (the heart, the brain).

7. Comparative characteristic of impairment manifestations of peripheral blood circulation: both arterial and venous hyperemias and ischemia.

The teacher's signature:

LESSON 2. TYPICAL IMPAIRMENTS OF PERIPHERAL BLOOD CIRCULATION. THROMBOSIS. EMBOLISM. HEMOSTASIS

Date: «____» _____ 20___

The purpose of the Lesson:

– To study the reasons, incidence conditions, development mechanisms, basic manifestations and consequences for the organism of thrombosis, embolism, hemostasis.

Tasks:

– To study the reasons and development mechanisms of typical impairments of microcirculatory channels on the basis of a teaching video «Microcirculation. Norm and Pathology».

– To get acquainted with modeling thrombosis and embolism in vessels of the tongue and intestinal mesentery of a frog.

– Solving situational tasks.

Work 1. STUDYING MATERIALS OF A TEACHING VIDEO «MICROCIRCULATION. NORM AND PATHOLOGY»

While viewing the teaching video pay special attention to the parts showing the role of various damaging factors and biologically active substances:

– in changing the character of a blood-stream in microcirculatory channels (slowing down, acceleration, arrest; retrograde and pendulum-like blood-stream);

– formation of reversible and irreversible hemostasis;

– aggregations of corpuscular elements of the blood;

– in formation of a sludge-phenomenon and thrombosis.

According to the experiments presented in the video, fill in the table:

The factor affecting microcirculatory channels	**Vascular tone of microcirculatory channels (↓↑)**	**Bloodstream character of microcirculatory channels (See above)**	**Components Virchow's triads****			**Outcome**		
			1 (+/-)	**2 (+/-)**	**3 (+/-)**	**sludge (+/-)**	**hemostasis (kind) (+/-)**	**thrombosis (like a blood clot) (+/-)**
Cooling								
Overheating								
Trauma								
Histamines								
Catecholamine								

The factor affecting microcircula-tory channels	Vascular tone of microcir-culatory channels (↓↑)	Bloodstream character of microcircula-tory channels (See above)	Components Virchow's triads**			Outcome		
			1 (+/-)	2 (+/-)	3 (+/-)	sludge (+/-)	hemo-stasis (kind) (+/-)	thrombo-sis (like a blood clot) (+/-)
Irritation n. sympathicus								
Ergotamine								
Nicotinic acid								

** **The note:** components of Virchow's triad:

1 — damage of endothelium;

2 — imbalance between coagulation and anticoagulation systems, the activity of the coagulating system prevailing;

3 — slowing down of blood stream.

Conclusions:

1. List the principal causes of hemostasis:

– reversible:

– irreversible:

2. Specify the basic development mechanisms of hemostasis:

– reversible:

– irreversible:

3. Specify the consequences of irreversible hemostasis:

Work 2. THE FORMATION PROCESS OF A WHITE MURAL BLOOD CLOT IN MESENTERIC VESSELS OF A FROG

An immobilized frog is placed on a plate with its back upwards so that its right side is adjoined to a round aperture of the board. Cut the skin with eye scissors in layers, muscles and peritoneum on the right lateral surface of the abdomen. Carefully, so as not to injure the interiors, take a loop of small intestines, mesentery of which should be straightened above a lateral aperture of the plate. The intestines should be placed over the edge of the aperture and fixed

to the plate by pins, pricked in an inclined outward position not to interfere with the movement of the microscope objective.

Use the obtained preparation for examining the picture of normal blood circulation in mesentery vessels of the frog's intestines under the microscope with small magnification. Then we find a place of fusing of two veins with uniform, not too fast blood-stream, and then a small crystal of **sodium chloride is placed near to the site of a vessel chosen earlier**. Observe the changes in the blood flow and the process of thrombosis for 10–40 min. Mark as the blood flow is gradually slowing down its course, a leukocyte-thrombocyte aggregate is being formed at the wall of a venous microvessel and subsequent loss of blood stream lamination occurs.

Fig. 1. A mural blood clot in a mesentery vessel of the frog's intestines

Answer the questions:

1. List (in the order of their importance) the factors promoting the development of thrombosis:

– in microcirculatory channels:

– in arteries:

– in veins:

2. Specify the consequences of thrombosis:

– of microcirculatory channels

– arteries:

– veins:

3. List the outcomes of thrombosis:

Work 3. MODELING FATTY EMBOLISM OF THE FROG'S TONGUE VESSELS

An immobilized frog it is placed on a plate with its abdomen upwards. Open the thorax and expose the heart. A thin layer of cotton wool moistened with 0.65 % solution of sodium chloride is applied on the exposed heart. Turn over the frog on the plate and prepare a section of the tongue observing the blood circulation in its vessels. Then inject **0.1 ml of slightly warmed up liquid paraffin into the cavity of cardiac ventricle with a syringe.** Quickly place the preparation of the tongue under the microscope. Observe the movement of emboli in the vascular lumen and the impairment of microcirculation. Similar changes can be observed in mesenteric vessels of the intestines and a swimming membrane of the frog.

Fig. 2. Fatty embolism of the frog's tongue vessels

Fill in the table:

Basic localization and signs of thromboembolism

Vascular region-source of thromboembolism	**Veins of lower extremities, small pelvis organs, right heart chambers ↓**	**Pulmonary veins, left heart chambers ↓**	**Veins of unpaired abdominal organs ↓**
The vascular region exposed to embolization			
Embolism result			
The basic embolism signs in vessels of the given localization			

Control questions:

1. The definition of the notions: «thrombosis», «embolism», «hemostasis». General characteristic.
2. The reasons and incidence conditions of thrombosis. Main factors of thrombosis.
3. Stages and mechanisms of thrombosis. Types of blood clots and thrombosis outcomes. Thrombosis consequences for the organism. Prophylaxis of thrombosis.
4. The reasons and mechanisms of embolus formation.
5. Types of embolism. The significance, outcomes and consequences of embolism for the organism. Prophylaxis of embolism.
6. The reasons, types and development mechanisms of hemostasis. Changes in tissues and possible consequences of hemostasis.

The teacher's signature:

LESSON 3. TYPICAL IMPAIRMENTS OF PERIPHERAL BLOOD CIRCULATION. IMPAIRMENTS OF MICROCIRCULATION

Date: «____» _____ 20___

The purpose of the Lesson:

– To study the reasons, incidence conditions, developmental mechanisms, basic manifestations and consequences of typical impairments of microcirculation for the organism.

Tasks:

– To study the reasons, developmental mechanisms and consequences of typical impairments of the microcirculation presented in a teaching video «Pathology of microcirculation».

– Test control of the section «Typical impairments of peripheral blood circulation and microcirculation».

Work 1. STUDYING THE MATERIALS OF A TEACHING VIDEO «PATHOLOGY OF MICROCIRCULATION»

While viewing the teaching video pay attention to the parts showing:

– the structure of an organ functional element and its microcirculatory component;

– effects of various vasoactive compounds (vasopressin in different dozes, histamine, etc.) and damaging agents on the condition of microcirculatory channels (intravascular, extravascular and transmural impairments of microcirculation);

– impairments of microcirculation in ischemia, embolism and inflammation.

Present the structure of an organ functional element and its microcirculatory component on the figure in schematic form, designate their basic components:

Fig. 1. An organ functional element:

1 – 3 –

2 – 4 –

According to experiments presented in the video, fill in the table

Factor, affecting vessels of microcirculatory channels	Vascular tone of microcirculatory channels	Permeability of vessels	Blood velocity	Hemostasis	Aggregation of corpuscular elements	Thrombosis	A kind of infringements microcirculation		
							Intravascular	Transmural	Extravascular
	(↑↓)	(↑↓)	(↑↓)	(+/-)	(+/-)	(+/-)	(1)	(2)	(3)
Vasopressin									
Histamine									
Alcohol									
Albumine									
Prostaglandins of E group									
Intravascular laser radiation									
Extravascular laser radiation									
Ischemia									

Draw a microcirculatory unit scheme, designate its basic components:

Fig. 2. Microcirculatory unit:

1 – 4 –

2 – 5 –

3 – 6 –

Conclusions:

1. Specify basic types of microcirculation impairments:

2. Specify the outcome of a complete arrest of microcirculation:

Control questions:

1. The definition of the notions «system of microcirculation», «microcirculatory unit of the organ», their components.
2. Principal causes and forms of typical impairments of microcirculation.
3. Developmental mechanisms of intravascular impairments of microcirculation.
4. The reasons, developmental mechanisms, manifestations of transmural impairments of microcirculation.
5. The reasons, developmental mechanisms, manifestations of extravascular impairments of microcirculation.
6. The definition of the notion «sludge»; the reasons, developmental mechanisms; manifestations, consequences, the significance for the organism.
7. The definition of the notion «capillarotrophic insufficiency», developmental mechanisms and consequences.
8. Typical impairments of lymphodynamics (mechanical, dynamic, resorptional insufficiency of lymphatic vessels) and their role in microcirculation impairments.

The teacher's signature:

LESSON 4. CELL INJURY

Date: «____» _____ 20___

The purpose of the Lesson:

– To study the reasons and general mechanisms of damaging a cell. To characterize damage as a typical pathological process. To discuss manifestations of cell damage, changing of the structure and function of cellular organelles, cellular compensatory mechanisms in cellular damage.

Tasks:

– To get acquainted with the reasons of cell damage, their types.

– To study general mechanisms of damaging a cell, the reaction of the organism to damage.

– To get acquainted with impairments of the structure and function of some cellular organelles, compensatory mechanisms in cellular damage on the basis of materials presented on slides «Damage of a cell», and also in the manual «Damage. Pathophysiological aspects».

– Solving situational tasks.

– Test control of the topic «Damage of a cell».

Work 1. STUDYING MORPHOFUNCTIONAL MANIFESTATIONS OF CELL INJURY BY SLIDES AND TABLES

Answer the questions:

1. Put down a definition of the notion «damage».

2. The mediated damage of a cell arises at:
   –
   –
   –
   –

3. Name the general mechanisms of cell damage:
   –
   –
   –
   –
   –

4. Derangements in the energy supply and utilization in cell can be connected with:

–

–

–

5. List basic manifestations of cell damage:

6. What morphofunctional changes of subcellular structures are usually revealed earlier than the other in case of cell damage?

7. Necrosis is —

8. Specific manifestations in cell damage

9. Nonspecific manifestations in cell damage

10. List the main intracellular mechanisms of adaptation and compensation in response to damage:

–

–

–

–

–

–

–

Control questions:

1. The definition of the notion «damage». Damage as a typical pathological process.
2. Principal causes and types of cell damage. Direct and indirect effect of a damaging agent on a cell.
3. General mechanisms of cell damage.
4. The impairment of energetic supply of processes taking place in cells, as one of master mechanisms of damage.
5. The role of damage of membranes and enzymes in the impairment of cellular vital activity, mechanisms of its development.
6. The role of genetic program impairments and its realization mechanisms in damaging a cell.
7. Perception impairments of regulatory effects on a cell. Regulation impairments of intracellular processes as a major mechanism of damaging a cell.
8. Basic manifestations of cellular damage, their mechanisms. Changes of the structure and functions of some cellular organelles in cell damage.
9. Specific and nonspecific manifestations in cell damage.
10. Intracellular mechanisms of adaptation and compensation in response to damage.
11. Integrated mechanisms of cellular damage and death (mechanisms of hypoxic necrobiosis and apoptosis).
12. General reactions of the organism to damage.

The teacher's signature:

LESSON 5. INFLAMMATION. IMPAIRMENTS OF BLOOD CIRCULATION IN THE FOCUS OF INFLAMMATION

Date: «____» _____ 20___

The purpose of the Lesson:

– To study principal causes of occurrence, developmental mechanisms, clinical manifestations, a dual nature and biological essence of inflammation as a typical pathological process. To discuss blood circulation impairment in the focus of inflammation; exudation and leukocytes emigration, the reasons and mechanisms of their development.

Tasks:

– To get acquainted with the reasons of occurrence and developmental mechanisms of the inflammatory process, impairments of peripheral blood circulation and microcirculation in inflammation on the basis of materials of the teaching video «Inflammation».

– To study the character of vascular reaction and a phenomenon of marginal state of leukocytes in inflammation of the frog's intestinal mesentery of (experiment of Kongame).

– Solving situational tasks.

Work 1. STUDYING THE MATERIALS OF A TEACHING VIDEO «INFLAMMATION»

On the basis of materials of the teaching video, the textbook and the lecture **answer the following questions**:

1. Inflammation is …

2. The basic local signs of acute inflammation according to Cells-Halen are:

3. The basic stages of inflammation are:

4. List vascular reactions arising in the focus of inflammation in consecutive order:

5. List the basic mediators of inflammations:
5.1. Cellular:
Derivatives of amino acids:
Biogenic amines:

Derivative arachidonic acids:
Metabolites of cyclooxygenic ways:

metabolites of lipooxygenic ways:

Low-molecular metabolites:

Mediators of albuminous and peptide nature:
Proinflammatory cytokines:

neuropeptides:

5.2. Plasma:
Components of the bradykinin systems:

Components of the complement system:

Fill in the table:

Basic effects of inflammation mediators
(specify with «+» or «↑↓» if mediator of this effect is present)

Inflammation mediator	**Vascular permeability**	**Tone of smoothmuscular cells of vessels (↑↓)**	**Pain**	**Thrombosis**	**Emigration, chemotaxis of leukocytes**	**Opsonization**	**Bacteriocidity, secondary alteration**	**Stimulation of leukopoesis**	**Fever**
Histamines									
Serotonin									

Inflammation mediator	Vascular permeability	Tone of smoothmuscular cells of vessels (↑↓)	Pain	Thrombosis	Emigration, chemotaxis of leukocytes	Opsonization	Bacteriocidity, secondary alteration	Stimulation of leukopoesis	Fever
Prostaglandins of group E									
Leukotrienes (LTC_4, D_1, E_4)									
Prostacyclin (PGI_2)									
Thromboxanes (TxA_2)									
NO									
Lyzosomal enzymes									
Cytokines (IL-1β, TNF-α)									
Bradykinin									
Components of the complement system (C3a, C5a, C5, C9)									

6. Specify general manifestations of inflammation:

7. List basic enzymes of an acute phase:

Work 2. STUDYING VASCULAR REACTIONS AND LEUKOCYTES EMIGRATION IN INFLAMMATION OF THE FROG'S INTESTINAL MESENTERY (KONGAME'S EXPERIMENT)

An immobilized frog is placed on a cork-tree plate with its back upwards so that its right side adjoined to a round aperture of the plate. Cut the skin, muscles and peritoneum on the right lateral surface of abdomen with eye scissors. Take a loop of small intestines, mesentery of which is straightened over a lateral aperture of the plate, from the opened abdominal cavity. The intestines should be placed at the edge of the aperture and fixed to the plate with pins stuck in an inclined outward position so as not to interfere the movement of the microscope objective.

Extraction of the intestines from the abdominal cavity and its fixation to the plate is accompanied by a mechanical trauma, drying up, that causes the development of an acute inflammatory reaction characterized by a number of vascular changes.

For studying vascular reactions, we observe blood circulation in tiny vessels on the prepared section under the microscope with small magnification for approximately 60 min with small breaks. We pay attention to changing of the lumen in various vessels, the amount of functioning capillaries, blood velocity, the ratio of the central (axial) blood-stream containing corpuscular elements of the blood, and a peripheral plasmatic layer. We notice the appearance of leukocytes in the plasmatic layer as if silvery balls were moving along vascular walls (redistribution of corpuscular elements in blood stream), and then marginal staying of leukocytes. Under large magnification we can mark, in what vessels (arterioles, venules, and capillaries) the marginal staying of leukocytes is expressed.

We sketch the observed vascular phenomena (hyperemia) and mural standing of leukocytes.

Fig. 1. Marginal standing of leukocytes in vessels of the frog's intestinal mesentry in inflammation

Conclusions:

1. What factors causes the inflammation of the frog's intestinal mesentery in the given experiment?

2. What factors provide adhesion and margination of leukocytes to a vessel wall in inflammation?

3. Specify the sequence and ways of migration through a vessel wall of different types of leukocytes in the focus of inflammation:

Control questions:

1. The definition of the notion and general characteristic of components of inflammation.
2. Inflammation as a typical pathological process. Local and systemic manifestations of inflammation.
3. Etiology of inflammation. Primary and secondary alteration in inflammation.
4. Basic mediators of inflammation, their origin, principles of classification.
5. The significance of inflammation mediators in the development of secondary alteration.
6. Metabolic changes in the focus of inflammation.
7. Physical and chemical changes in the focus of inflammation, mechanisms of their development and significance.
8. Functional element of the organ as a substrate of alteration and formation of inflammatory reaction.
9. Impairment stages of peripheral blood circulation in the focus of inflammation and mechanisms of their development.
10. The reasons and mechanisms of increasing the permeability of a vascular wall in the focus of inflammation.
11. The definition, mechanism and significance of exudation in inflammation.
12. Types of exudates, their distinctions from transudate.

The teacher's signature:

LESSON 6. INFLAMMATION. PHAGOCYTE REACTION IN INFLAMMATION

Date: «____» _____ 20___

The purpose of the Lesson:

– To study phagocytosis as a protective reaction of the organism, to discuss phagocytosis stages during inflammation. To characterize the significance of inflammation as a response of the whole organism, to study the effect of the nervous system, hormonal and humoral factors on the development of inflammation.

Tasks:

– To get acquainted with the role of granulocytes for the development of a phagocyte (protective) reaction of the organism in inflammation on the basis of materials presented in the teaching video «The role of the granulocyte colony-stimulating factor (G-CSF) in regulating phagocytosis».

– To study phagocytosis stages of bird's erythrocytes by leukocytes of a guinea-pig on micropreparations.

– To study the role of superficial tension in the process of phagocytosis in Danilevsky's modeling experiment.

– Solving situational tasks.

– Test control of the topic «Inflammation».

Work 1. STUDYING PHAGOCYTOSIS OF BIRD'S ERYTHROCYTES BY PERITONEAL MACROPHAGES OF A GUINEA-PIG ON MICROPREPARATIONS

A guinea pig with aseptic peritoneal inflammation, induced by preliminary intraperitoneal injection of sterile peptic infusion broth, is injected 3,0 ml of 3 % suspension of hen’s erythrocytes in isotonic solution of sodium chloride into the abdominal cavity, the solution being heated up to 38 ºС (erythrocytes, containing a nucleus, serve as an object of phagocytosis).

In 15 min about 1,0 ml of exudate with bird's erythrocytes is taken out by a syringe from the abdominal cavity of a guinea-pig and smear cultures are prepared. Then, every 15–20 min after the first sample the second and a third samples of exudates are taken and smear cultures are prepared too. The smear cultures are stained according to Romanowsky–Giemsa and then they are investigated under the microscope.

Fig. 1. Phagocytosis stages of bird's erythrocytes by macrophages of a guinea pig

While doing the microscopy of smears **find, sketch and designate**:

1. A leukocyte of a guinea pig surrounded by oval bird's erythrocytes, containing nuclei *(phases of approaching and sticking)*.

2. A leukocyte absorbing alien erythrocytes *(phase of absorption)*.

3. A leukocyte concluding fragments of erythrocytes *(phase of digestion*).

Conclusion:

What stages of phagocytosis prevail in the first sample and what — in the subsequent samples of peritoneal exudate?

On the basis of the textbook materials and the lessons **answer the following questions**.

1. List **basic chemoattractants** causing the approach (chemotaxis) of leukocytes to the objects of phagocytosis:

2. List basic proteins functioning as **opsonins**:

3. List major factors causing bacteriocidity of phagocytes.

4. Specify principal causes of incomplete phagocytosis:

4.1 –

4.2 –

4.3 –

4.4 –

4.5 –

5. Specify peculiarities of the morphological structure and metabolism of some the bacteria allowing them to persist in phagocytes or to avoid phagocytosis:

5.1 –

5.2 –

5.3 –

6. Fill in the table.

Hereditary defects of phagocytes

Syndrome (disease) name	Type of inheritance	Character of phagocytes functional impairments	Clinical manifestations of diseases
Chediak–Higashi syndrome			
Granulomatosis			

Work 2. THE SIGNIFICANCE OF CHANGING THE SUPERFICIAL TENSION OF A LEUKOCYTE MEMBRANES IN PHAGOCYTOSIS MECHANISMS (DANILEVSKY'S MODELING EXPERIMENT)

Place in a Petri dish 10–20 ml of 10 % solution of nitric acid and apply a drop of mercury. At a distance of 1 cm from the mercury a potassium bichromate crystal is placed. Observe as the mercury drop is extending towards the crystal, surrounding it, simulating phagocytosis. This movement of a mercury drop is explained by changing of superficial tension of its various parts due to formation and adsorption of superficially active products of reaction of potassium bichromate with nitric acid on its surface. This modeling experiment resembles the process that takes place in the focus of inflammation and evidences that during inflammation one of the conditions of leukodiapedesis is the formation of substances (chemoattractants, etc.), lowering the superficial tension of leukocytes and thus causing their emigration from vessels into the focus of inflammation as well as the subsequent stages of phagocytosis.

Sketch (schematically) revealed changes:

Fig. 2. Interaction of a potassium dichromate crystal with a droplet of mercury

Conclusion:

What is the role of superficial tension forces of a granulocytes membrane in phagocytosis mechanisms?

Specify major factors regulating reparation and proliferation processes:

Control questions:

1. The definition of the notion and biological significance of phagocytosis.
2. I. I. Mechnikov's study about phagocytosis as a protective reaction of the organism.
3. Stages, ways and mechanisms of leukocytes emigration in inflammation.
4. Factors regulating activity of phagocytes in the focus of inflammation. Chemotaxis mechanisms, factors stimulating and oppressing chemotaxis.
5. Stages of phagocytosis and their mechanisms.
6. The reasons and types of phagocytosis impairments.

7. The proliferation stage, its basic signs and development mechanisms.

8. General manifestations of inflammation, mechanisms of its development and the significance for the organism.

9. Endogenic pro- and anti-inflammatory factors.

10. Relationship of local and general phenomena in inflammation. The role of the nervous, endocrine and immune systems in the development of inflammation. General biological significance of inflammation.

11. Positive and negative significance of inflammation for the organism.

12. The basic pathogenesis theories of inflammation. Modern conceptions of inflammation mechanisms.

The teacher's signature:

LESSON 7. HYPOXIA

Date: «____» _____ 20___

The purpose of the Lesson:

– To study etiology and pathogenesis of hypoxic conditions, their types, basic manifestations, urgent and long-term mechanisms of compensatory-adaptive reactions in response to hypoxia.

Tasks:

– To study pathogenic action of the lowered barometric pressure on the organism in experiment.

– To study dysbaric phenomena on modeling experiment.

– To study the reasons and developmental mechanisms of some kinds of hypoxia using the material of the teaching video «Hypoxia».

– Solving situational tasks.

– Test control of the topic «Hypoxia».

Work 1. STUDYING PATHOGENIC EFFECT OF THE LOWERED BAROMETRIC PRESSURE ON THE ORGANISM

For reproducing conditions of the lowered barometric pressure in experiment a manual rarefying pump of Komovsky with a support for a bell is used. The experiment is performed on laboratory animals. An experimental animal is placed under the bell (a guinea pig, a white mouse, a frog). We observe the animals, their behavior in normal atmospheric pressure, and then we gradually pump out the air from under the bell. The degree of rarefying the air under the bell is determined with the mercury manometer available in Komovsky's pump. We mark changes of the animals' condition while «raising the altitude».

Kind of animal	General condition while « raising the altitude », km					
	3–4	7	9	10–11	12	19
Guinea pig	Hurried respiration and palpitation	Anxiety, mild excitation	Rare respiration, falls sideways, clonic spasms	Death		
White mouse	–//–	–//–	Rare respiration	The animal is lying on one side, clonic spasms	Tonic spasms, death	
Frog	No changes	–	–	–	–	–

Answer the questions:

1. What are the distinctions in behavior, general condition and survival rate of the animals while «raising the altitude»?

2. What are the mechanisms of changing the respiratory functions, blood circulation and nervous system while «raising the altitude» in a guinea pig and a white mouse?

Work 2. STUDYING «DYSBARIC» PHENOMENA (MODELING EXPERIMENT)

Under the bell connected to the Komovsky's pump, place a tied up rubber glove and a glass with water, t 37 ºC (the temperature of water corresponds to the body temperature). At pumping out the air from under the bell there occurs stretching of the rubber glove and at the «altitude» corresponding to 19 kms — «boiling» of water in the glass — a model of decompression disease (expansion of gases in cavities, gas embolism and tissue emphysema).

Answer the questions:

1. Why on pumping out the air from under the bell the following occurs:

a) Stretching of a rubber glove?

b) «Boiling» of water in the glass at body temperature on the altitude corresponding to 19 km?

Work 3. STUDYING THE MATERIALS OF THE TEACHING VIDEO «HYPOXIA»

While getting acquainted with the video pay attention to the reasons and development mechanisms of some types of hypoxia, changes occurring in the blood and tissues.

Fill in the tables:

Pathological compounds of hemoglobin	Their formation causes in the organism	The action of pathological compounds in the organism	Bias character of the curve HbO_2 dissociation

Some parameters of the organism oxygen supply in various types of hypoxia (- or ¯ in comparison with the norm)

Type of hypoxia	P_AO_2	PaO_2	$Pv\ O_2$	$Da\text{-}v\ O_2$	HbO_2 content	$PaCO_2$	$Pv\ CO_2$
1. Hyperbaric							
2. Normobaric							
3. Respiratory							
4. Circulatory							
5. Hemic							
6. Tissue							
7. Loading							

Control questions:

1. The definition of the notion «hypoxia». Hypoxia as a typical pathological process.
2. Principles of classification of hypoxic conditions. Types of hypoxias.
3. Etiology and pathogenesis of hypoxic conditions.
4. Compensatory-adaptive reactions in hypoxia.
5. Functional impairments of organs and systems in hypoxia. Mechanisms of hypoxic necrobiosis.
6. Mechanisms of urgent and long-term adaptation to hypoxia.
7. Mountain and high-altitude diseases.
8. Dysbarism, its clinical manifestations and pathogenesis.
9. The effect of hypoxic trainings on nonspecific resistance of the organism.

The teacher's signature:

LESSON 8. TYPICAL IMPAIRMENTS OF METABOLISM. THE IMPAIRMENTS OF WATER EXCHANGE. EDEMAS

Date: «____» _____ 20___

The purpose of the Lesson:

– To study the reasons and development mechanisms of water balance impairment in the organism, pathogenesis of cardiac, renal, toxic, inflammatory, cachectic, allergic and other kinds of edema and dropsy.

Tasks:

– Studying developmental mechanisms of pulmonary edema in experimental acute cardiac insufficiency induced by injection of adrenaline.

– Studying developmental mechanisms of a toxic pulmonary edema in experiment, when the central nervous system plays a pathogenetic role.

– Solving situational tasks.

– Test control of the topic of the Lesson.

Work 1. ADRENALINE PULMONARY EDEMA IN A RAT

For the experiment take two white rats with the body mass of 200 g, and count their respiratory rate per 1 minute. One of the rats (tested) is injected intraperitoneally 0.1 % solution of chloride adrenaline with 1 ml / 100g of body mass, the second (control) — physiological solution of the same volume. Observe the animals' general condition, we count the respiratory rate ever 1–2 min to the moment of death. Euthanasia of the control rat is performed by stretching cervical vertebrea. After death of the animals open the thorax of both rats, apply a ligature at the trachea, take out the lungs, weigh them and do a pathomorphological examination.

Results of the experiment.

Table 1

Clinical and pathomorphological manifestations Adrenalin-induced pulmonary edema in a rat

Type of effect	Respiratory rate (resp/min)	General state	Pathomorphological changes in lungs
i/p injection of 0,l % Adrenaline solution			Weight of the lungs — 5,8 g, pulmonary weight factor — 0,029. Foamy liquid in the trachea. The lungs volume is enlarged, looks like marble, foamy discharge on the section
– Initial	120	Normal	
– 1 min	160	General excitation, impairment of motor coordination	
– 2 min	Rare deep respiration	Foamy discharge from the mouth	
– 3 min	Terminal respiration	– // – // – // –	

Type of effect	Respiratory rate (resp/min)	General state	Pathomorphological changes in lungs
– 4 min i/p injection of 0,9 % solution of NaCl	Respiratory arrest	Death of the animal	Weight of the lungs — 1,2 g, pulmonary weight factor — 0,006. The trachea is freely passable. Lungs are collapsed, of light pink color
– Initial	130	General state without visible changes.	
– 1 min	–		
– 2 min	–		
– 4 min	–		

Conclusion:

Explain the development mechanism of adrenaline-induced pulmonary edema.

Work 2. STUDYING THE ROLE OF THE CENTRAL NERVOUS SYSTEM IN THE DEVELOPMENT TOXIC PULMONARY EDEMA

The experiment is performed on two white rats with weight of 200 g. **One of them (tested) is narcotized by a subcutaneous injection of 0.3 ml of 10 % solution of hexenal**, the second (control) is given 0.3 ml of physiological solution subcutaneously. Sleep occurs in 10 minutes. Then both animals are injected 6 % solution of ammonium chloride i/p at a rate of 0.7 ml per 100 g of body weight. Observe the general condition and respiration rate of the animals. Record the findings of the experiment. The unnarcotized rat dies in 55 min after the injection of ammonium chloride of developed pulmonary edema. During this period no changes of general condition and respiration rate were revealed in the narcotized rat.

The narcotized rat is subjected to euthanasia by a stretching cervical vertebrea. After death, open the thorax, apply a ligature on the trachea, take out the lungs, weigh them and carry out the pathomorphologic investigation.

Table 2

Effect of narcosis (hexenal) on the development of toxic Pulmonary edema in a rat

Type of effect	Respiration rate (resp/min)	General condition	Pathomorphological changes in the lungs
Innarcotized rat + injection of NH_4Cl			Lungs weight — 6,0 g, pulmonary weight factor — 0,03. Foamy liquid in the trachea. Lungs volume is enlarged, they remind marble, foamy discharge on dissection
– Initial	128	Normal	
– 15 min	150	Impairment of movements coordination	
– 30 min	20_	The rat is motionless, is lying on one side	
– 45 min	Rare deep respiration	Neck and mouth muscles take part in respiration	

Type of effect	Respiration rate (resp/min)	General condition	Pathomorphological changes in the lungs
– 55 min	Terminal respiration Respiratory arrest	Foamy discharge from the mouth Death of the animal	
Hexenal narcosis + injection of NH_4Cl – Initial – 15 min – 30 min – 55 min	 100 103 102 102 Quiet, rhythmic respiration	General condition without visible changes	Lungs weight — 1,4 g, pulmonary weight factor — 0,007. The trachea is freely passable. The lungs collapsed, are of light pink color

Conclusions:

1. Explain the protective action mechanism of hexanol narcosis on the development of toxic pulmonary edema.

2. List the basic pathogenetic factors of edema development:

1 –

2 –

3 –

4 –

3. Give the definition of the notions:

– anasarca —
– edema —
– ascites —
– hydrothorax —
– hydropericardium —
– hydrocele —
– hydrocephaly —

Control questions:

1. Regulation mechanisms of water exchange and their impairment (hypo- and hyperhydrations).
2. Edemas and dropsies (definition).
3. Kinds of edemas.
4. Pathogenetic factors of edema development.
5. Pathogenesis of cardiac,renal, toxic, cachectetic, angioneurotic and other kinds of edemas.
6. Pulmonary edema (etiology, pathogenesis, clinical and pathomorphological picture of pulmonary edema).
7. The significance of edema for the organism.

The teacher's signature:

LESSON 9. TYPICAL IMPAIRMENTS OF THERMOREGULATION. FEVER

Date: «____» _____ 20___

The purpose of the Lesson:

– To study the incidence reasons, development mechanisms and general biological significance of fever.

Tasks:

– To study the state of heat exchange processes in the development of feverish reaction in rabbits after injection of bacterial endotoxin.

– To study the character of thermoregulatory reactions of rabbits with endotoxic fever under overheating.

– To construct the most typical temperature curves in various kinds of fever.

– Solving situational tasks.

– Test control of the topic «Fever».

Work 1. STUDYING THE CHARACTER OF THERMOREGULATORY REACTIONS IN THE RABBIT WITH EXPERIMENTAL ENDOTOXIC FEVER

For the experiment take two adult rabbits of one sex with body weight of 2,0–2,5 kg, take the initial rectal body temperature, temperature of the ear skin, respiratory frequency and heart beat rate.

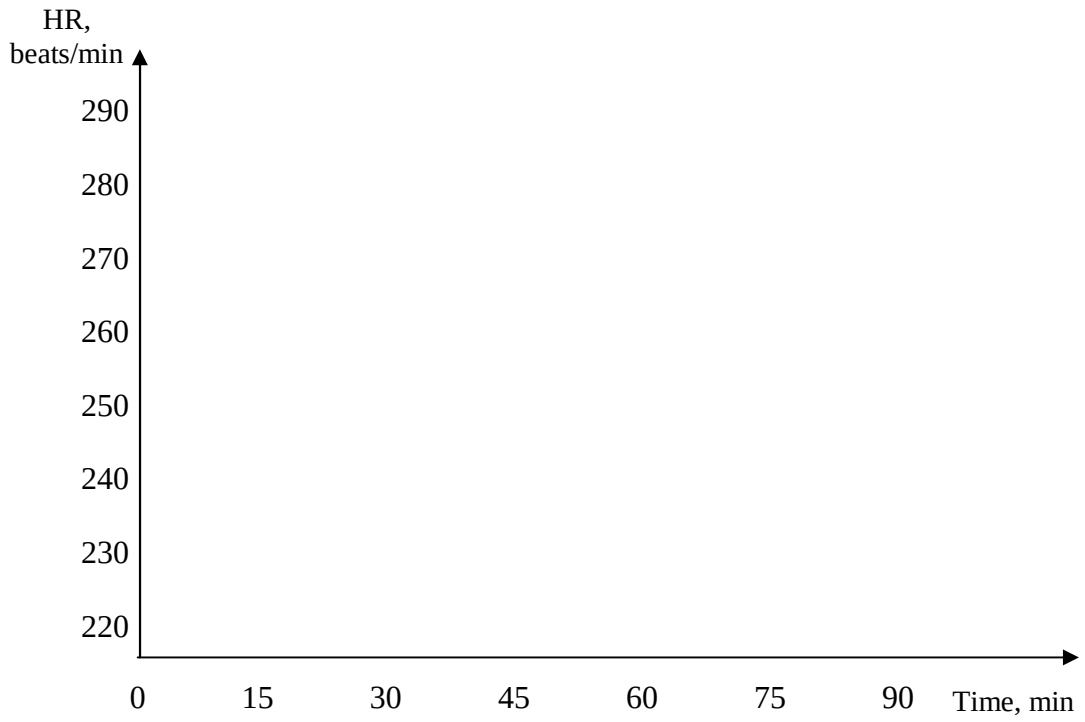
The skin temperature of an auricle external surface, and also deep body temperature (temperature in the rectum at the depth of 5 cm) is taken by electric thermometer TPEM-I. The respiratory rate is registered using a coal cuff and by an ink-writing electrocardiograph. Heart beat rate is determined by an electro-cardiogram. The initial parameters are recorded into the protocol.

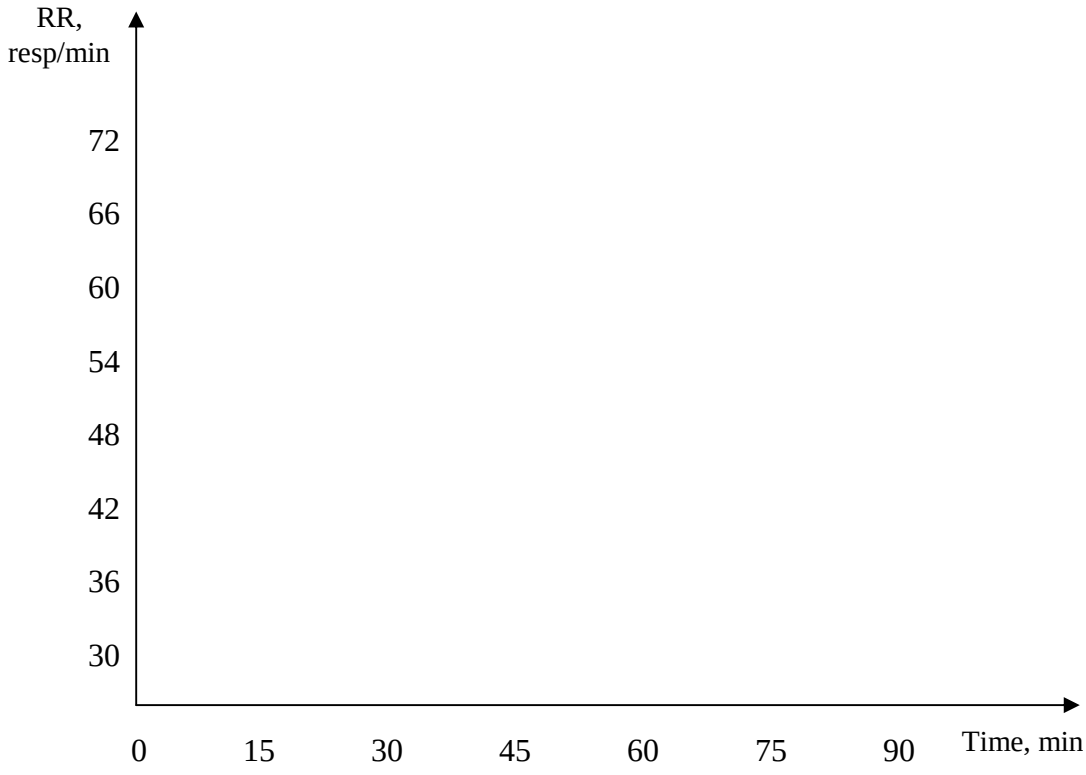
To induce experimental fever we use endotoxin — bacterial liposaccharid pyrogenal.

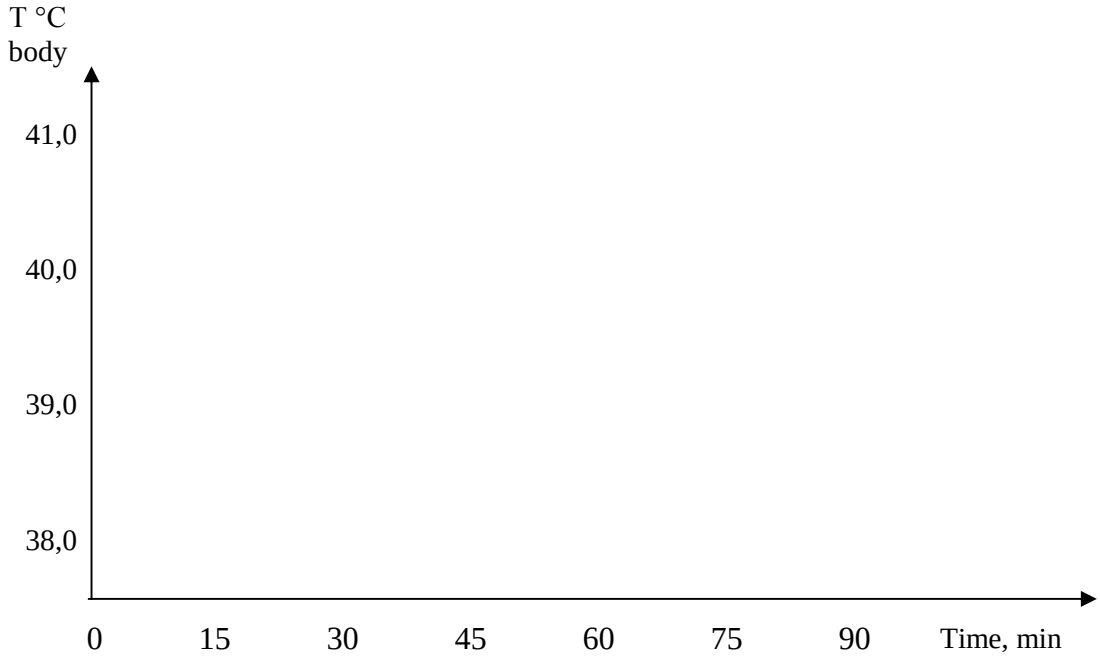
The first rabbit (tested) is injected pyrogenal (0.5 mcg/kg) in 0.5 ml of physiological solution **into the marginal vein of the ear**, and **the second (control) — 0.5 ml of physiological solution**. Then observe the condition and behavior of the animals. In every 15 min after injections take rectal temperature of the rabbits, the skin temperature of the ear, respiratory rate and heart beat rate.

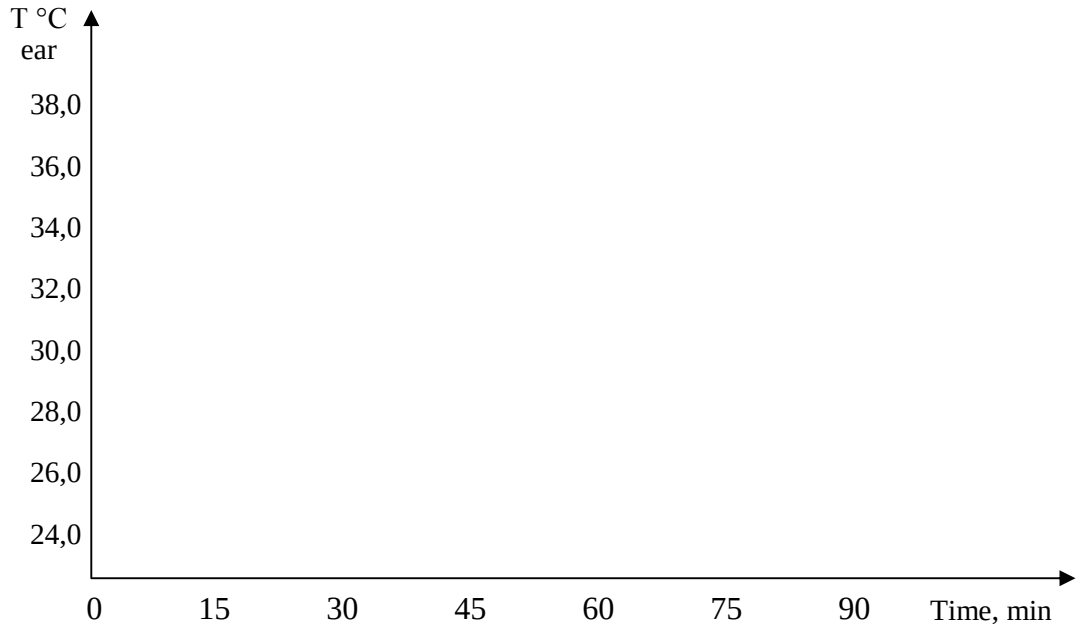
Results of the experiment

№	Group of animals. Time since the beginning of the experiment	Temperature, °C		Respiratory rate (resp/min)	Heart rate (beats/min)	Notes
		rectal	ear skin			
1.	***Tested:*** **IV injection of pyrogenal (0.5 mcg/kg)**					
	The ref. data	38,8	33	60	220	Ears are pale, cold, vessels are narrowed
	– 15 min	39,2	24,0	72	260	
	– 30 min	39,6	24,0	30	270	
	– 45 min	39,9	27,0	46	280	
	– 60 min	40,2	28,0	58	280	
	– 75 min	40,4	28,0	60	290	
	– 90 min	40,4	30,0	70	280	
2.	***Control:*** **IV injection of 0.9 % NaCl**					
	The ref. data	39,2	31,0	68	220	Ears are pink, warm, vessels are moderately dilated
	– 15 min	39,2	30,0	70	242	
	– 30 min	39,0	30,0	72	236	
	– 45 min	39,0	32,0	72	230	
	– 60 min	39,2	32,0	72	230	
	– 75 min	39,3	31,0	70	220	
	– 90 min	39,2	31,0	70	220	




RR,
resp/min
72
66
60
54
48
42
36
30
0
15
30
45
60
75
90
Time, min


T °C
body
41,0
40,0
39,0
38,0
0
15
30
45
60
75
90
Time, min



Construct the graphs, allowing to compare changing the body temperature, auricle temperature, respiratory rate and heart rate of an intact and tested rabbits in dynamics of experiment.

Draw conclusions, answer the following questions:

1. What causes the temperature decrease in the auricle, reduction of respiratory rate and acceleration of HB in the tested rabbit?

2. What are the possible mechanisms of increasing heat production and reduction of heat emission on the first stage of pyrogenal-induced fever?

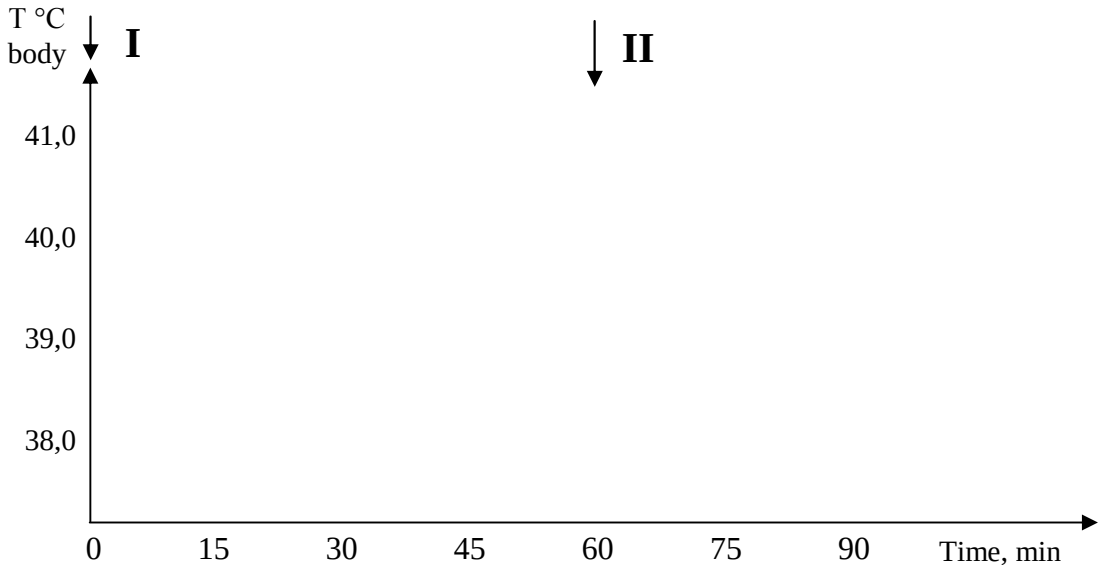
Work 2. STUDYING THE PECULIARITIES OF THERMOREGULATORY REACTIONS IN RABBITS WITH ENDOTOXIC FEVER UNDER OVERHEATING

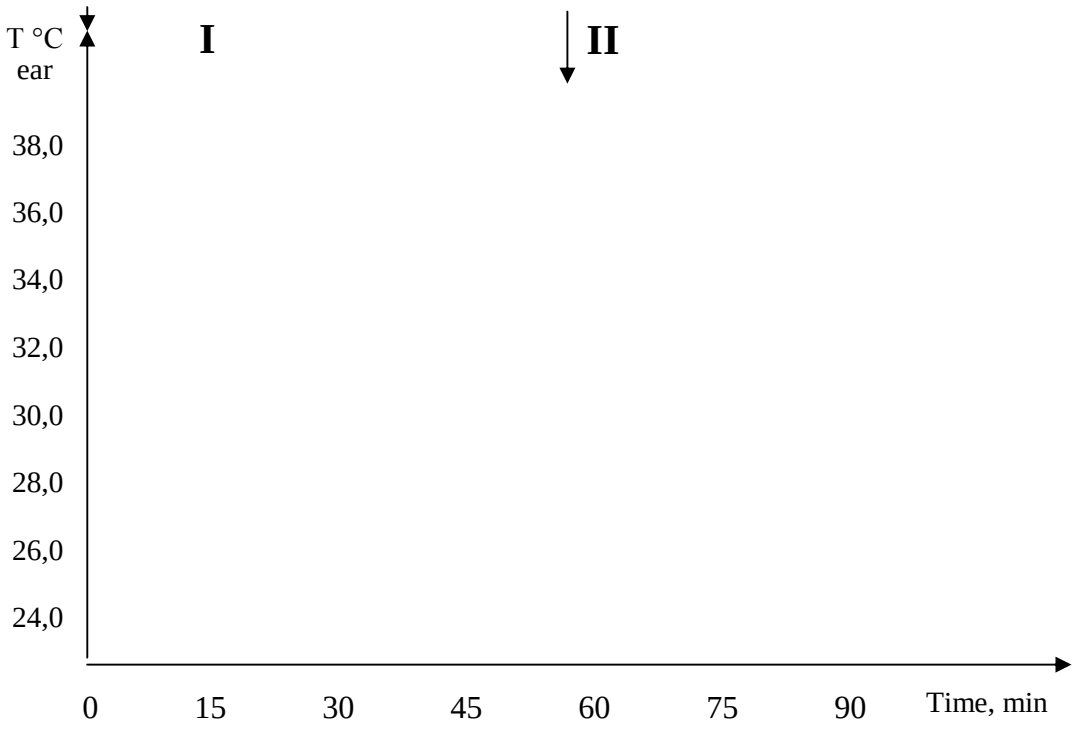
The experiment is performed on two adult rabbits of one sex with body weight of 2,0–2,5 kg. **One (tested) is injected pyrogenal (0,5 mcg/kg) in 0,5 ml of physiological solution into the marginal vein of the eat, to the other (control) — 0,5 ml of physiological solution**. Immediately after injections the animals are placed in **the thermochamber with dry air** and overheating at temperature of air **40–42 °C** is performed. Thermometry, as well as registration of respiratory rate and heart beat are performed every 15 min within one hour, according to a technique described in work 1. Then the animals are taken from thermochambers and measurement of body temperature, respiratory rate and heart rate are continued everyone 15 min during their stay in **thermo-neutral conditions (20–21 °C)**.

The data received are recorded into the table.

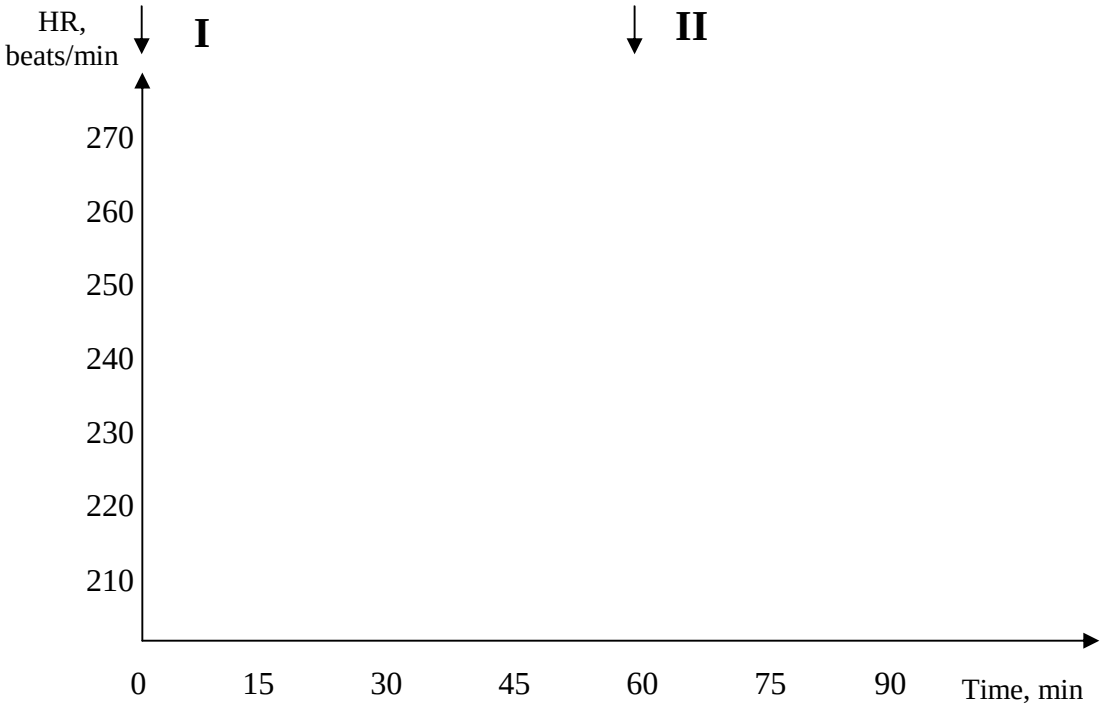
Group of animals. Time since the beginning of the experiment	Temperature, °C		Respiratory rate (resp/min)	Heart rate (bpm)
	rectal	ear skin		
***Tested*:** **Intravenous introduction injection of pyrogenal (0,5 mcg/kg) + overheating**				
Initial data	38,8	33,0	62	220
– 15 min	39,0	31,0	68	220
– 30 min	39,2	26,0	78	242
– 45 min	39,6	28,0	48	260
– 60 min	40,0	32,0	92	272
Placing the feverish rabbit in thermo-neutral conditions				
– 75 min	40,4	31,0	90	270
– 90 min	40,4	31,0	92	258
***Control*:** **Intravenous introduction injection of 0,9 % Solution of NaCl + overheating**				
Initial data	38,8	32,0	63	225
– 15 min	39,0	30,0	68	236
– 30 min	39,0	29,0	72	218
– 45 min	39,3	30,0	90	205
– 60 min	40,8	35,6	128	252
Placing the feverish rabbit in thermo-neutral conditions:				
– 75 min	40,6	34,4	116	248
– 90 min	40,2	33,0	102	240

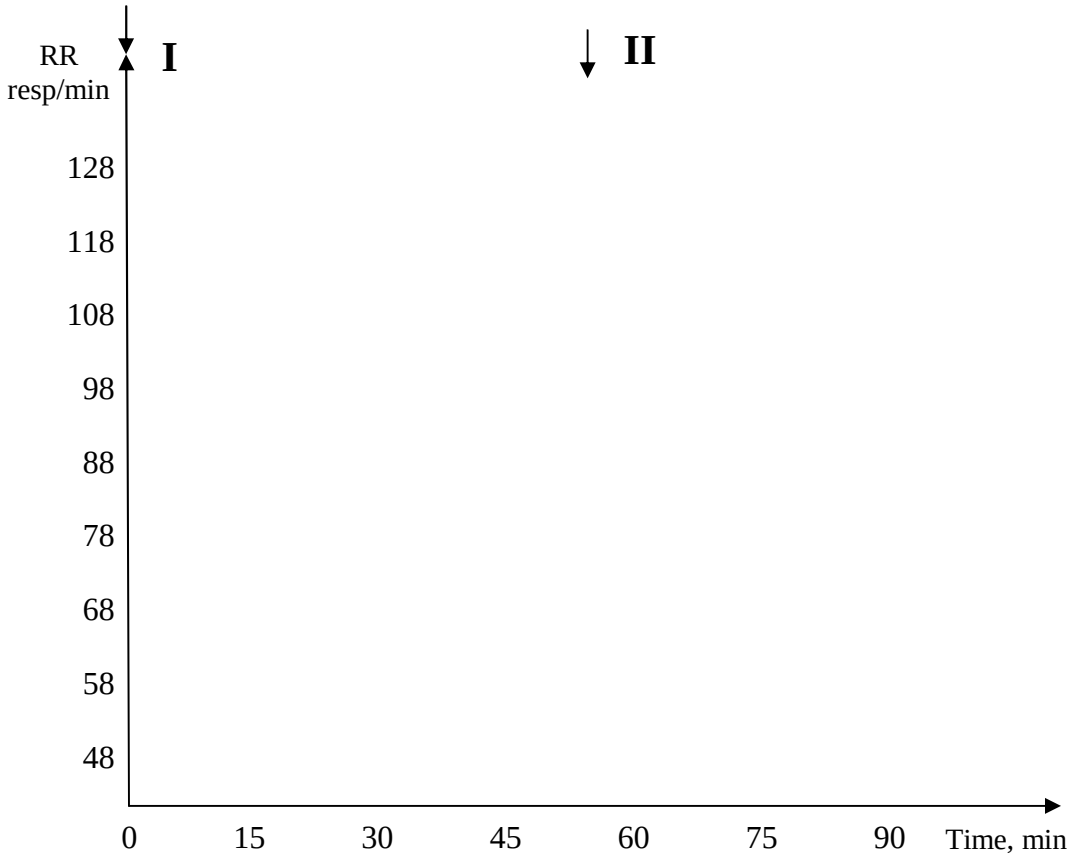
Construct the graphs allowing to compare changing the body temperature, temperature of the auricle, respiratory rate and heart rate of the intact and tested rabbits in dynamics of experiment.





I — at the moment of injecting pyrogenal (0,5 mcg/kg) or 0,9 % sol. of NaCl in T 40–42 °C;
II — at the moment of placing the animals in thermo-neutral conditions at T 20–21 °C





I — at the moment of IV injecting pyrogenal (0,5 mcg/kg) or 0,9 % sol. of NaCl in T 40–42 °C; **II —** at the moment of placing the animals in thermo-neutral conditions at T 20–21 °C

Answer the questions:

1. How does overheating affect the character of the first stage of fever?

2. Is the ability of thermoregulation preserved during fever?

3. What is the distinction of fever from hyperthermia observed in overheating?

Work 3. CONSTRUCTION AND THE CHARACTERISTIC OF VARIOUS TYPES TEMPERATURE CURVES

Construct temperature curves in the following kinds of fevers (specify the Latin name):

- sustained →
- remittent →
- hectic→
- intermittent→
- relapsing →
- atypical →
- pervert →

Temperature curve																					Fever name	Daily temperature variations	Occurs in diseases
	1		2		3		4		5		6		7		8		9		10				
	m	e	m	e	m	E	m	e	m	e	m	e	m	e	m	e	m	e	m	e			
41																							
40																							
39																							
38																							
37																							
36																							
35																							

Temperature curve																					Fever name	Daily temperature variations	Occurs in diseases
	1		2		3		4		5		6		7		8		9		10				
	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e			
41																							
40																							
39																							
38																							
37																							
36																							
35																							

Temperature curve																					Fever name	Daily temperature variations	Occurs in diseases
	1		2		3		4		5		6		7		8		9		10				
	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e			
41																							
40																							
39																							
38																							
37																							
36																							
35																							

Temperature curve																					Fever name	Daily temperature variations	Occurs in diseases
	1		2		3		4		5		6		7		8		9		10				
	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e			
41																							
40																							
39																							
38																							
37																							
36																							
35																							

Temperature curve																					Fever name	Daily temperature variations	Occurs in diseases
	1		2		3		4		5		6		7		8		9		10				
	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e			
41																							
40																							
39																							
38																							
37																							
36																							
35																							

Temperature curve																					Fever name	Daily temperature variations	Occurs in diseases
	1		2		3		4		5		6		7		8		9		10				
	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e			
41																							
40																							
39																							
38																							
37																							
36																							
35																							

Temperature curve																					Fever name	Daily temperature variations	Occurs in diseases
	1		2		3		4		5		6		7		8		9		10				
	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e	m	e			
41																							
40																							
39																							
38																							
37																							
36																							
35																							

* m — morning, e — evening.

Control questions:

1. The definition of the notion «fever». Fever as a typical pathological process.
2. Etiology of fevers. Pyrogenic substances.
3. Pathogenesis of fevers. Action mechanisms of pyrogens.
4. Fever stages. Mechanisms of body temperature elevation in fever. The relationship between heat production and heat emission during various stages of fever.
5. Varieties of fever (by the level of elevation of body temperature). Types of temperature curves in fever.
6. Changes of metabolism, functions of systems and organs in fever.
7. The role of functional condition of the nervous, endocrine and immune systems in formation of a fever response.
8. General biological significance of fever.
9. Basic distinction of fever from hyperthermia (overheating).
10. Pyrotherapy. Definitions of the notion, general characteristic.

The teacher's signature:

LESSON 10. TYPICAL IMPAIRMENTS OF METABOLISM. ACID-BASE STATE IMPAIRMENTS OF THE INTERNAL ENVIRONMENT OF THE ORGANISM

Date: «____» _____ 20___

The purpose of the Lesson:

– To study typical forms of acid-base impairments of the internal environment of the organism, their kinds, reasons, development mechanisms, manifestations and compensatory mechanisms, basic laboratory parameters, correction principles of the acid-base state.

Tasks:

– To get acquainted with laboratory parameters of the acid-base state.

– To study: 1) the parameters of primary impairments and mechanisms of expected impairments of the acid-base state (ABS); 2) interrelation of mechanisms of ABS regulation and water-electrolyte balance.

– Computer control of the topic of the Lesson using special teaching maps with light indication.

– Solving situational tasks.

– Test control.

ABS parameters in norm

Blood parameters	Values in SI units
pH	7,35–7,45
p_aCO_2	35–45 mm Hg
HCO_3^-	22–26 mmol/l
SB (standard bicarbonate)	22–27 mmol/l
BB (buffer bases)	44–53 mmol/l
BE (excess/deficiency of buffer bases)	± 2,3 mmol/l
Lactic acid (lactate)	0,5–2,2 mmol/l
Ketonic bodies	0,43–1,033 mmol/l
Electrolytes of blood plasma (mmol/l)	
Na^+	135–145
K^+	3,5–5,0
Ca^{2+}	2,23–2,57
Mg^{2+}	0,65–1,1
Cl^-	96–108
HCO_3^-	22–26
Phosphates	0,81–1,45

Additional ABS parameters

Titrated acidity (TA) of diurnal diuresis of 20–40 mmol/l.

Ammonia of diurnal diuresis of 10–107 mmol/24 h (20–50 mmol/l).

Fill in the table.

Parametric changes of a respiratory and metabolic component in typical ABS impairments

Type of ABS impairment	Primary impairment	Expected compensation
1. Respiratory acidosis		
2. Non-respiratory acidosis		
3. Respiratory alkalosis		
4. Non-respiratory alkalosis		

The analysis plan of a type of ABS impairments

1. Acidosis/alkalosis.
2. Compensated/decompensated acidosis/alkalosis (on pH change).
3. Type of acidosis/alkalosis on the origin (according to clinical anamnesis).
4. Evaluate triggering of compensatory mechanisms (criteria: laboratory parameters testifying to **hyperfunction** of the respiratory or metabolic system of pH regulation).

SITUATIONAL TASKS

№ 1

A group of tourists from the middle region of the European part of the CIS is transported by air to a tourist camp on Pamir, 2500 m about the sea level. Several people began complaining of tiredness, weakness, early fatigue. While examining one of them on the 2nd day of staying at the camp the following parameters of the acid-base state were seen:

$pH_{arterial\ blood}$ = 7.46;
p_aCO_2 = 32 mm Hg;
HCO_3^- = 22 mmol/l;
BE = -1 mmol/l;
pH_{urine} = 6.0;
TA of urine = 20 mmol/day.

In a week the patient's condition improved. ABS parameters were the following:

$pH_{arterial\ blood}$ = 7.41;
p_aCO_2 = 30 mm Hg;
HCO_3^- = 17 mmol/l;
BE = -6 mmol/l;

pH_{urine} = 7.2;
Bicarbonates in the urine;
TA of urine = 0.
Make a conclusion regarding the character of ABS impairments.

№ 2

The patient aged 56 years old suffers from pulmonary emphysema and respiratory insufficiency.

ABS parameters and of electrolyte balance:
$pH_{arterial\ blood}$ = 7.37;
p_aCO_2 =56 mm Hg;
HCO_3^- = 32 mmol/l;
BE = 7.5 mmol/l;
Na^+ = 142 mmol/l;
K^+ = 4 mmol/l;
Cl^- = 88 mmol/l.
Make the conclusion regarding the character of ABS impairments.

№ 3

The patient suffering for many years from diabetes was admitted to hospital in a coma. Parameters of ABS and electrolyte balance on admission:

$pH_{arterial\ blood}$ = 6.95;
p_aCO_2 = 20 mm Hg;
HCO_3^-= 5.5 mmol/l;
BE =-20 mmol/l;
SB = 4 mmol/l;
Ketonic bodies in blood plasma = 10 mmol/l;
K^+ = 7.5 mmol/l;
TA of urine = 60 mmol/l;
Ketonic bodies present in urine.
Make the conclusion regarding ABS and possible approaches for its correction.

№ 4

The patient suffers from diffuse glomerulonephritis for 10 years. He was admitted to hospital due to expressed renal insufficiency. Oliguria.

ABS parameters and electrolyte balance:
$pH_{arterial\ blood}$ = 7.27;
p_aCO_2 = 27 mm Hg;
HCO_3 = 15.5 mmol/l;
BE = -10 mmol/l;
SB = 15 mmol/l;

Concentration of trace anions in plasma = 21 mmol/l;
K^+ = 5.8 mmol/l.
Make the conclusion regarding the character of ABS impairments.

№ 5

The patient was admitted to the first aid hospital in the condition of asphyxia. The blood test revealed:
$pH_{art.\ blood}$ = 7.0;
p_aCO_2 = 80 mm Hg;
HCO_3^- = 19 mmol/l;
BE = -8 mmol/l;
SB = 18 mmol/l;
BB = 37 mmol/l;
Lactate = 4.5 mmol/l.
Make the conclusion regarding the character of ABS impairments.

№ 6

The patient was admitted to clinic in a severe condition. Extensive infarction of anterior lateral walls of the left ventricle, acute left-ventricular cardiac insufficiency, pulmonary edema was diagnosed. While estimating ABS parameters the following data were received:
$pH_{art.\ blood}$ = 7.22;
p_aCO_2 = 55 mm Hg;
HCO_3^- = 20 mmol /l;
BE = -5 mmol/l;
Lactate = 4.76 mmol/l.
Make the conclusion regarding the character of ABS impairments.

№ 7

The patient, 46 years old, was admitted to clinic with an extensive trauma (multiple fractures of bones, damage of soft tissues), accompanied by massive blood loss. On admission the consciousness is inhibited, the skin is pale, cold and damp with sweat. BP is 95/60 mm Hg. Pulse — 120 beats/min. Marked breathlessness, thirst. Oliguria.

On ABS investigation the following data are received:
$pH_{art.blood}$ = 7.26;
p_aCO_2 = 28 mm Hg;
HCO_3^- = 14.5 mmol/l;
BE =-12 mmol/l;
SB = 14 mmol/l;
Lactate = 6.8 mmol/l.
Make the conclusion regarding the character of ABS impairments.

№ 8

The patient has peritonitis, paralytic intestinal obstruction, fever. Loss of liquid is 6 l. Oliguria. On investigation of ABS parameters and electrloyte balance the following data are received:

$pH_{art.\ blood}$ = 7.15;

p_aCO_2 = 25 mm Hg;

HCO_3^- = 12 mmol/l;

BE = -20 mmol/l;

SB = 15 mmol/l;

Lactate = 6.2 mmol/l;

Ketonic bodies in blood plasma = 3.7 mmol/l;

Potassium = 6.5 mmol/l;

Concentration of trace anions in plasma = 26 mmol/l;

Reduced content of K^+ in erythrocytes.

Characterize the type of ABS impairment.

№ 9

Patient B., 13 years old, with acute poliomyelitis on the 4th day of the disease noted the difficulty of respiration, due to which he was administered artificial pulmonary ventilation (APV).

Investigation results of ABS are presented in the table:

Parameters	Before APV	In 2 h after APV was started
$pH_{art.\ blood}$	7.26	7.46
$paCO_2$	62 mm Hg	30 mm Hg
HCO_3^-	26 mmol/l	18 mmol/l
BB	43 mmol/l	40 mmol/l
SB	22 mmol/l	20 mmol/l
BE	1 mmol/l	-2.2 mmol/l

1. What form of ABS impairment took place in the child before artificial pulmonary ventilation?

2. Draw the conclusion regarding the character of ABS impairment in 2 h after APV.

3. Is the volume of pulmonary ventilation during APV established correctly?

№ 10

Patient Z., 16 years old, was admitted to clinic with acute pneumonia. The condition is severity. The body temperature is 39.8 °C. Expressed breathlessness.

The anamnesis revealed no pulmonary pathology.

The investigation of ABS parameters revealed:

$pH_{art.blood}$ = 7.47;

$paCO_2$ = 29 mm Hg;

HCO_3^- = 22 mmol/l;

BE = -1.8 mmol/l.

1. What ABS impairment is present in the patient?

2. What is the cause?

№ 11

Patient K., 38 years old, was admitted to the hospital with an attack of tetanic spasms.

Questioning of the patient revealed that about half a year ago he got into a car accident. He had an open fracture of the right humeral bone. Fracture healing occurred in usual terms. But since then he had been suffering from strong heartburn and to relive it he constantly takes baking soda.

The investigation of ABS parameters revealed:

$pH_{art.\ blood}$ = 7.50;

$paCO_2$ = 43 mm Hg;

HCO_3^- = 32 mmol/l;

BE = +12 mmol/l.

1. What kind of ABS impairment developed in the patient?

2. What is the direct cause of the impairment of the acid-base balance in this case?

3. Can these changes of the acid-base state result in the development of tetania?

№ 12

Patient M., 37 years old, was admitted to the intensive care department with acute poisoning with sleeping draught.

The investigation of ABS parameters revealed:

$pH_{art.\ blood}$ = 7.29;

$paCO_2$ = 56 mm Hg;

HCO_3^- = 25 mmol/l;

BE = +1 mmol/l.

1. What form of ABS impairment is present in the patient?

2. Is there a necessity to administer sodium bicarbonate in this case to correct the impaired acid-base state?

№ 13

ABS shifts were studied in the group of sportsmen under the conditions of growing loadings on the veloergometer. The loading in decathlonist B., 24 years old, was started from 150 Wt and it was increased by 50 Wt every 2 min till the individual maximum. Immediately after the loading the acid-base state was investigated. Meanwhile it was revealed that:

$pH_{art.\ blood}$ = 7.29;

$paCO_2$ = 30 mm Hg;
HCO_3^- = 18 mmol/l;
BE = -11 mmol/l.

1. In what way did ABS change in the sportsman as a result of significant physical loading?
2. What is the probable cause of ABS impairment in this case?
3. How can the decrease of paCO2 parameter be explained?

№ 14

Patient M., 54 years old, was admitted to hospital in a grave condition. He complained of general weakness, heavy loss of weight. For the last 5–6 days almost after each meal he feels pain in the epigastric area accompanied by vomiting.

The investigation of ABS parameters revealed:
$pH_{art.\ blood}$ = 7.55;
$paCO_2$ = 60 mm Hg;
HCO_3^- = 50 mmol/l;
BE = 18 mmol/l.

1. Make a conclusion regarding the character of ABS impairment.
2. What is the possible cause of ABS impairment in this patient?

№ 15

Child D., 4 years old, was admitted to hospital due to elevation of the body temperature and frequent loose stool (8–10 times a day). On examination moderate dehydration and breathlessness were noted.

The investigation of ABS parameters revealed:
$pH_{art.\ blood}$ = 7.39;
$paCO_2$ = 27 mm Hg;
HCO_3^- = 17 mmol/l;
BE = -8 mmol/l.

1. Make a conclusion regarding the character of ABS impairment.
2. What is the possible cause of ABS impairment in the child?

№ 16

Patient L., 48 years old, with diabetes was admitted to hospital in a heavy pre-comatose condition. The patient was administered a complex therapy, including, insulin intramuscularly and solution of sodium bicarbonate intravenously. The results of КОС investigation are presented in the table:

Parameter	Before treatment	On the 2nd day of treatment	On the 3rd day of treatment
pH	7.28	7.34	7.44
pCO_2	20 mm Hg	36 mm Hg	49 mm Hg
BB	31 mmol/l	39 mmol/l	51 mmol/l
HCO_3^-	12 mmol/l	18 mmol/l	29 mmol/l
BE	-18 mmol/l	-9 mmol/l	6 mmol/l

1. Specify the type of KOC impairment on admission and on the 2nd and 3rd day of treatment.

2. Is there a necessity for further introduction of sodium bicarbonate to the patient?

Control questions:

1. Mechanisms of maintaining pH of extracellular liquids of the organism.
2. Classification of ABS impairments.
3. Basic laboratory estimation criteria of ABS impairments.
4. Etiology and pathogenesis of respiratory acidosis and alkalosis.
5. Etiology and pathogenesis of non-respiratory acidosis and alkalosis.
6. Major pathogenic development mechanisms of primary acidosis.
7. Interrelation of ABS mechanisms and water-electrolyte balance.
8. Compensatory mechanisms in ABS impairments, laboratory criteria of their estimation.
9. Basic clinical manifestations in non-compensated acidosis and alkalosis.
10. Correction principles of ABS impairments.

The teacher's signature:

LESSON 11. TYPICAL IMPAIRMENTS OF TISSUE GROWTH. TUMORS. BIOLOGICAL PECULIARITIES. REPRODUCTION OF EXPERIMENTAL METHODS. ETIOLOGY OF TUMOURS

Date: «____» _____ 20___

The purpose of the Lesson:

– To study the laws of tumor distribution in phylo- and ontogenesis, biological peculiarities of malignant and benign formations, etiology of tumors, to get acquainted with methods of experimental reproduction of tumor growth.

Tasks:

– Studying the methods of experimental oncology, issues of epidemiology and etiology of malignant neoplasms, biological peculiarities of a tumor cell on the basis of the illustrated Atlas «Pathophysiology of Tumor Growth» (sections 1–6).

– Studying manifestations of cellular atypism of tumors on micropreparations of Ehrlich's ascite carcinoma and cellular line of human gastric cancer CaVe (carcinoma ventricular).

– Solving situational tasks.

Work 1. STUDYING THE MATERIALS OF THE ILLUSTRATED ATLAS «PATHOPHYSIOLOGY OF TUMOR GROWTH» (SECTIONS 1–6)

On the basis of the textbook materials, the lesson and illustrated atlas answer the questions:

1. Name the principal causes of growth incidence of malignant neoplasms for the last 50 years:

2. List experimental methods used for studying tumors:

3. Fill in the table:

Biological peculiarities of malignant tumors

Biological peculiarity	**Main factors; its manifestations in an integral organism**
1. Independent and uncontrollable growth	
2. Morphological atypism: – tissue – cellular	
3. Functional atypism: – hypo- – hyper- – dysfunction	
4. Biochemical atypism	
5. Energetic atypism	
6. Antigenic (AG) atypism: – AG simplification – AG divergence – AG reversion *Specify specific tumor AG markers*	
7. Invasive destructive growth	
8. Metastasis *List the stages of metastasis*	
9. Inhering changes	
10. Tumor progression	

Biological peculiarity	Main factors; its manifestations in an integral organism
11. Recurrent ability	
12. Systemic effect of the tumor on the organism	

4. List the basic **exogenic** chemical carcinogens:

5. List the basic **endogenic** chemical carcinogens:

6. List the basic cancerogenic effects of physical origin:

7. List the basic biological carcinogens:

8. Specify the types of human malignant neoplasms, the viral etiology of which is recognized:

9. Specify the types of human malignant neoplasms, the dyshormonal etiology of which is recognized:

Work 2. STUDYING MANIFESTATIONS OF MORPHOLOGICAL (CELLULAR) ATYPISM OF TUMORAL CELL S IN EHRLICH'S ASCITE CARCINOMA AND CELLULAR CULTURE OF GASTRIC CANCER CaVe

Studying a micropreparation of Ehrlich's ascite carcinoma

Take out some ascite liquid with a thin needle of a 5 mm syringe from a narcotized mouse with an intertwisted ascite tumor of Ehrlich. Prepare a culture smear, fix it for 2–3 min in methyl spirit, stain it according to Romanowsky–Giemsa, wash it, dry it and examine under the microscope: at first with small, and then with large magnification (10 × 90).

During microscopic investigation observe cellular atypism (dwarfish and gigantic cells of various form), prevalence of round cells with an extremely

hypochromous nucleus and sharp basophilic cytoplasm (so-called dark cells), the presence of large cells with clearly outlined chromatin structure and pale-stained cytoplasm («light» tumor cells); frequent mitosis and amitosis, pathologic mitosis, division of nuclei without division of cytoplasm.

Sketch morphological peculiarities of tumor cells:

Fig. 1. Cells of Ehrlich's ascite carcinoma:
1 — dwarfish cells; 2 — gigantic cells, *2a* — gigantic multinuclear cells; 3 — irregular-shaped cells; *3a* — cell with spherical cytoplasmatic processes; 4 — dark cells with hyperchromous nuclei and sharp basophilic cytoplasm; 5 — large light cell with a clearly marked structure of nuclear chromatin; 6 — cellular mitosis; 7 — pathological mitosis; 8 — nuclear division without division of cytoplasm

Studying a micropreparation of the cellular line of gastric cancer CaVe

The cellular line CaVe was discovered by J. V. Dobrynin and R. P. Dirlugjanom in 1959 from a solid cancer of the antral department of the stomach.

The cellular line is presented by large polygonal or slightly elongated epithelium-like cells with light transparent cytoplasm. The cell borders are clearly visible. The nuclei are round, with 3–7 nucleoli of irregular shape. Overgrown cultures look as a continuous epithelial layer or as fusing cellular membranes with narrow slits. Sometimes the tubular formations remaining iron elements are observed among a continuous layer of cells.

Examining the preparation fixed and stained in hematoxilin-eosin under large magnification (10 × 90), observe and sketch morphological peculiarities of tumor cells:

Fig. 2. Cells of the CaVe line:
1 — gigantic multinuclear cells; 2 — cell with 3–4 polar pathologic mitosis; 3 — cells with stuck chromosomes in pathologic mitosis; 4 — cells with chromosomal bridges in pathologic mitosis

Answer the questions:

1. What manifestations of cellular atypism are characteristic of cells of Ehrlich's ascite carcinoma and the CaVe cellular line of gastric cancer?

2. What division abnormalities are characteristic of tumor cells?

Control questions:

1. The definition of the notion «tumour». Characteristic of tumor growth as a typical pathologic process.
2. The distribution of tumors in phylo- and ontogenesis.
3. Basic biological features of malignant tumors.
4. Experimental methods of tumor reproduction.
5. The role of chemical carcinogens in tumor development; main factors of carcinogenicity of chemical compounds.
6. The role of physical carcinogens in tumor development. Types of physical carcinogens.
7. Oncogenic viruses, their kinds and the action mechanisms.
8. The notion of syn-carcinogenesis and co-carcinogenesis.
9. Transplantation carcinogenesis.
10. The role of nutrition, harmful habits, heredity in the development of tumors.

The teacher's signature:

LESSON 12. TYPICAL IMPAIRMENTS OF TISSUE GROWTH. PATHOGENESIS OF TUMORS. SYSTEMIC EFFECT OF A TUMOR ON THE ORGANISM

Date: «____» _____ 20___

The purpose of the Lesson:

– To get acquainted with evolution of the nature oncogenesis theories; to study modern conceptions of molecular-genetic mechanisms of the initial link of carcinogenesis — tumoral transformation of a cell, mechanisms of antitumor resistance, interrelation of the tumour and the organism, principles of prophylaxis and treatment of tumors.

Tasks:

– To study mutational, epigenomic, viral-genetic theories of tumor pathogenesis, modern conceptions of tumoral transformation mechanisms (the theory of an oncogen); interaction problems of a tumor and major regulatory systems of the organism — neuro-endocrine and immune; mechanisms of systemic tumor effect of on the organism on the basis of the illustrated Atlas« Pathological physiology of tumor growth» (sections 7–9).

– Studying cytogenetic peculiarities of cells in ascite hepatoma 22 A.

– Solving situational tasks.

– Final computer control of the section: «Typical impairments of tissue growth. Etiology and pathogenesis of tumors».

Work 1. STUDYING THE MATERIALS OF THE ILLUSTRATED ATLAS «PATHOLOGICAL PHYSIOLOGY TUMOR GROWTH » (SECTIONS 7–9)

On the basis of materials of the textbook, the lesson and the illustrated atlas answer the questions:

1. What DNA structure is a target for the action of carcinogenic factors resulting in tumoral transformation of a cell?

2. What is a proto-oncogen?

3. What functions do proteins, products of a proto-oncogen, perform?

4. List the transformation mechanisms of a proto-oncogen into an oncogen:

1 –

2 –

3 –

4 –

5 –

6 –

7 –

5. List the basic functions of oncogen — oncoproteins:

6. List the basic kinds and functions of cellular anti-oncogens:

7. List the basic carcinogenesis stages:

8. What is the inefficiency of immune reactions to a tumour due to:

1 –

2 –

3 –

4 –

9. List the basic mechanisms of immunosuppression in cancer:

1 –

2 –

3 –

4 –

5 –

Fill in the table:

Basic manifestations of systemic tumour effect on the organism (paraneoplastic syndrome)

Syndrome	Development mechanism	Basic manifestations
Cachexia		
Immunopathological		
Psychoneurological		
Paraneo-endocrine		
Thrombo-hemorrhagic		
Anemic		

Specify principal developmental causes of a pain syndrome in malignant tumors:

List the diseases which are a facultative precancerous condition:

List the diseases which are an obligate precancerous condition:

List the basic ways of malignant neoplasms prophylaxis:

Work 2. STUDYING CYTOGENETIC PECULIARITIES OF A CELL IN ASCITE HEPATOMA 22A

The cellular karyotype is examined by studying metaphasal plates under the light microscope. For this purpose cells of ascite hepatoma 22 A is processed with colchicine resulting in the arrest of cellular division at a metaphase stage by suppressing the formation of spindles. Then, the cells are applied to the cover glass, and are exposed to hypotonic solution of sodium chloride that results in breaking cellular and cytoplasmatic membranes and favorable distribution of chromosomes over the preparation. After that, the preparation is covered with the object glass under pressure. As a result, metaphase chromosomes stay on the object glass (it is one of the methods for receiving isolated chromosomes).

Further on the preparation is fixed and stained by special methods (according to Romanowsky, Felgen or with aceto-orcein).

The karyotype of tumor cells differs from the karyotype of a normal, homologic tumor, tissue. The number of chromosomes in tumor cells can increase in multiple (polyploidy) or not multiple (aneuploidy) times as compared to a normal diploid chromosomal complement. The cells of one and the same tumor sometimes contain a different number of chromosomes.

In the inhomogenous population of tumor cells, the cells of the stem line are differentiated, they possess identical properties. Somatic cells of healthy mice contain 40 chromosomes (a diploid complement). The stem line of ascite hepatoma 22a contains the cells with 39 chromosomes (a paradiploid complement). ***Three marker chromosomes*** are present in all tumor cells: an acrocentric one with a despiralized paracentrameral area 1–2 subcentrameral ones.

Fig. 1. Abnormal chromosomes in ascite hepatoma 22a cell

Control questions:

1. The pathogenesis of tumors. Modern views on the molecular and genetic mechanisms of neoplastic transformation. The modern interpretation of the concept of oncogene. The role of mutations, viruses and epigenomic violations in the mechanism of transformation in proto oncogene.
2. Types and functions of cellular oncogenes, the role of onconcoproteins in violation of the functions of the transformed cells. The concept of antionkogen.
3. The relationship of disorders of the nervous system with the emergence and growth of tumors.
4. Interrelation of the endocrine system with the emergence and growth of tumors. Hormone-dependent tumors.
5. The relationship of disorders of the immune system with the emergence and growth of tumors. Features of antitumor immunity. The main causes and manifestations of immunosuppression in cancer.
6. Systemic effect of the tumor on the body. Paraneoplastic syndrome, its pathogenesis, the main manifestations. The pathogenesis of cancer cachexia.
7. The concept of pre-cancerous conditions. Obligate and facultative precancer. Stage of development of malignant tumors (L. M. Shabad). Basic principles of treatment and prevention of tumors.

The teacher's signature:

SECTION III
SPECIFIC PATHOLOGICAL PHYSIOLOGY

LESSON 1. HEMOPOIESIS AND GENERAL LAWS OF BLOOD FORMATION. ERYTHROPOIESIS, ITS IMPAIRMENTS. MORPHOFUNCTIONAL PECULIARITIES OF ERYTHROCYTES AND HEMOGLOBIN IN PATHOLOGY

Date: «____» _____ 20___

The purpose of the Lesson:

– To discuss the types of erythropoiesis and peculiarities of its impairment, to study basic morphofunctional peculiarities of erythrocytes and hemoglobin in pathology.

Tasks:

– To get acquainted with general laws, types and basic impairments of blood formation (hemopoiesis).

– To study morphofunctional peculiarities of erythrocytes, hemoglobin and the character of peripheral blood in various pathology.

– To draw the cells of a megaloblastic and normoblastic type of blood formation.

– To draw regenerative and degenerative forms of erythrocytes.

Work 1. STUDYING MORPHOFUNCTIONAL PECULIARITIES OF CELLS IN NORMO- AND MEGALOBLASTIC TYPES OF BLOOD FORMATION

Examine blood smears of human embryos (3^{rd}–4^{th} weeks) under the microscope with 10 × 90 magnification. Pay attention to various sizes of cells, sizes and staining of nuclei, staining of the cytoplasm of megaloblasts and normoblasts, the presence of cellular inclusions.

Fig. 1. Cells of embryonic blood

Answer the questions:

1. List the basic cellular morphofunctional peculiarities of blood formation of a megaloblastic type in comparison with cells of a normoblastic type of blood formation:

2. The deficiency of what factors in the organism is revealed by the occurrence of blood cells of a megalloblastic type of blood formation in the postnatal period?

Work 2. STUDYING MORPHOFUNCTIONAL PECULIARITIES OF REGENERATIVE AND DEGENERATIVE FORMS OF ERYTHROCYTES

Examine a blood smear under the microscope with 10 × 90 magnification, that is supravitally stained with ***brilliant cresyl blue*** for revealing reticulocytes.

1. _____ 2. _____

Fig. 3. The blood smear at supravital staining with brilliant cresyl blue:
1 — erythrocytes; 2 — reticulocytes

Degenerative forms of erythrocytes:

1. ***Abnormalities of cell sizes***. Draw and specify the size of erythrocytes abnormal in form and size:

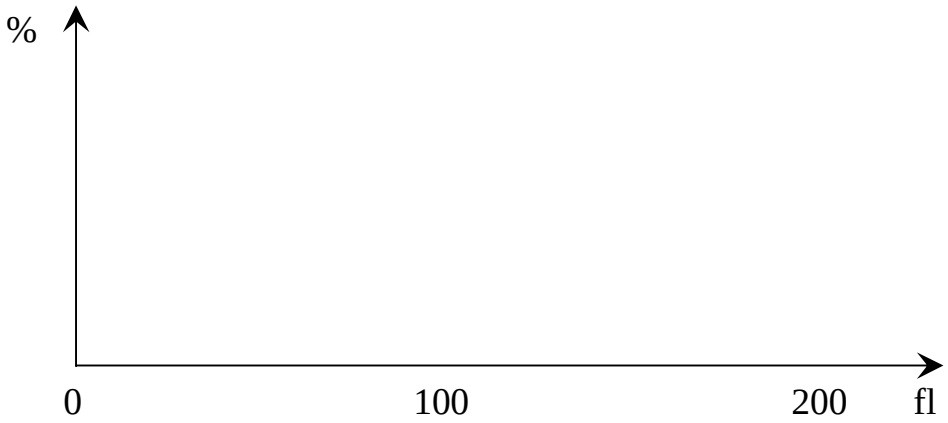
a) a normocyte (_____ micron), or _____ fl;

b) microcytes (_____ micron), or less _____ fl;

c) macrocytes (_____ micron), or more _____ fl;

d) megalocytes (_____ micron).

Draw a normal curve of erythrocytes distribution in volume and its changes (shifts) in microcytosis and macrocytosis; mark the **RDW*** parameter (**a parameter of anisocytosis**) on the curve: for this purpose draw a line parallel to the abscissa axis at the level of 20 % from the peak amplitude. RDW* (red cell distribution width) — distribution width of erythrocytes in volume. The normal value of RDW: _____ %.



The curve of erythrocytes distribution in volume in norm (1), in macro- (2) and microcytosis (3)

2. ***Abnormalities of the cell form.*** Draw and designate the basic pathological forms of erythrocytes: ovalocytes (1), microspherocytes (2), torocytes or codocytes (3), acanthocytes (4), sickle erythrocytes (5), echinocytes (6), degmacytes (a nibbled erythrocyte) (7), schistocyte (8).

1. _____ 2. _____

3. _____ 4. _____

5. _____ 6. _____

7. _____ 8. _____

3. ***Abnormality of cell staining***. Sketch in comparison with normocytes (1) and designate hypochromous erythrocytes (anulocytes) (2) and hyperchromous erythrocytes (3). Pay attention to the correlation between the intensity of staining and sizes of cells.

1. _____ 2. _____ 3. _____

4. ***The presence of pathological inclusions***. Draw and designate erythrocytes with the basic pathological inclusions: Howell-Jolly bodies (1), Cabot's rings (2), basophilic puncture (3), Heinz bodies (4).

1. _____ 2. _____

3. _____ 4. _____

Answer the question:

1. What does the occurrence of degenerative forms of erythrocytes in peripheral blood mean?

Control questions:

1. The blood system, the definition of the notion, general characteristic.
2. Hemopoesis. General laws of blood formation. Periods and types of blood formation in ontogenesis.
3. The characteristic of the basic classes of blood cells according to the structure of blood formation (according to A. I. Vorobjevu and I. P. Tchertkov).
4. Hemopoietic cells-progenitors: colony-forming units or colony-forming cells (CFC).
5. The development scheme of hemopoietic cells-progenitors and colony-stimulating factors regulating them.
6. Erythropoiesis. Cells-progenitors of erythropoiesis: BFU-E (burst-forming mature and immature units) and CFU-E (colony-forming erythrocyte unit).
7. Morphofunctional cellular characteristic of normoblastic and megaloblastic types of blood formation.
8. Morphofunctional peculiarities of erythrocytes in pathology. Regenerative and degenerative forms of erythrocytes.
9. Types and pathological forms of hemoglobin.
10. Neurohumoral regulation of erythropoiesis, its impairments.

The teacher's signature:

LESSON 2. ANEMIAS AND ERYTHROCYTOSIS

Date: «____» _____ 20___

The purpose of the Lesson:

– To study etiology and pathogenesis of the most common anemias and erythrocytosis, blood pattern in this pathology.

Tasks:

– To study under the microscope and draw the pattern of peripheral blood:
  a) after acute blood loss (on the fifth day);
  b) in iron-deficiency anemia;
  c) in B_{12}-deficiency anemia;
  d) in microspherocytosis (disease of Minkovsky–Shoffar).

– Test control of the topic «Anemias and erythrocytosis»;

– Analyzing complete blood count (CBC) (№ 1–11, 20) and solving situational tasks (2–15) on the topic of the Lesson (see the collection of situational tasks on pathological physiology).

Work 1. STUDYING THE BLOOD SMEAR IN ACUTE POSTHEMORRHAGIC ANEMIA (5th day after acute blood loss)

A. Smear staining according to **Romanowsky-Giemsa**.

Examine a blood smear under the microscope with 10 × 90 magnification. Find in the smear immature (regenerative) forms of erythrocytes — polychromatophils (1–2 and more in the field of vision). Pay attention to moderately expressed poikilocytosis and anisocytosis of erythrocytes.

Fig. 1A. Blood smear at the acute posthemorrhagic anemia (5th day after the blood loss):
1 — erythrocytes; 2 — polychromatophils; 3 — poikilocytes

B. Supravital staining of the smear with **brilliant cresyl blue**. Examine the smear under the microscope. Find 2–4 reticulocytes with characteristic cytoplasmatic inclusions of dark blue color as a small net in the field of vision. Draw the cells.

Fig. 1B. Blood smear at the acute posthemorrhagic anemia (5th day after blood loss):
1 — erythrocytes; 2 — reticulocytes

Answer the questions:

1. What changes in the pattern of red blood are observed on the 5^{th} day after acute blood loss?

2. To what processes in the erythron system do the revealed changes mean?

3. List regenerative forms of erythrocytes revealed in peripheral blood, in acute posthemorrhagic anemia:

4. Explain the origin of basophile net substance in reticulocytes:

Work 2. STUDYING THE BLOOD SMEAR IN IRON-DEFICIENCY ANEMIA

Examine a peripheral blood smear of a patient with iron-deficiency anemia under the microscope with 10 × 90 magnification. Observe the presence of hypochromous erythrocytes; slight aniso- and poikilocytosis.

Fig. 2. Blood smear at the iron-deficiency anemia:
1 — hypochromous erythrocytes (anulocytes); 2 — poikilocytes

Answer the questions:

1. What quantitative changes on the part of red blood (erythrocyte and hemoglobin content) and erythrocyte indices (MCV, MCH, RDW) are characteristic of iron-deficiency anemia?

2. What pathological forms of erythrocytes appear in peripheral blood in iron-deficiency anemia?

Work 3. STUDYING BLOOD SMEAR AT THE B_{12}-(FOLIC ACID) DEFICIENCY ANEMIA

Examine a blood smear of the patient with the B_{12}-deficiency anemia under the microscope with 10 × 90 magnification. Pay attention to expressed anisocytosis, poikilocytosis (round, pear-shaped, oval erythrocytes); anisochromia and hyperchromia, the presence of megalocytes, erythrocytes with Howell-Jolly bodies, Cabot's rings, basophile puncture; and also single megaloblasts and giant polysegmentonuclear leukocytes. Draw these cells.

Fig. 3. Blood smear at the B_{12}-(folic acid) deficiency anemia:
1 — a megaloblast (*1a* — basophilic; *1b* — polychromatophilic; *1c* — oxifilic); 2 — megalocytes; 3 — poikilocytes; 4 — erythrocytes with pathological inclusions (*4a* — with Howell–Jolly bodies; *4b* — with Cabot's rings; *4c* — with basophilic puncture); 5 — a giant polysegmentonuclear neutrophile

Answer the questions:

1. What type of hemopoiesis is characteristic of B_{12}-(folic acid)-deficiency anemia?

2. What quantitative changes on the part of red blood (erythrocytes and hemoglobin content) and erythrocyte indices (MCV, MCH, RDW) are characteristic of B_{12}-deficiency anemia?

3. Explain the origin of pathological inclusions in erythrocytes in the given type of hemopoiesis:

– Howell-Jolly bodies are …

– Cabot's rings are …

– Basophilic puncture is …

Characterize various kinds of anemia by morphofunctional features:

I. By the type of hemopoiesis:

– megaloblastic:

– normoblastic:

II. By the color parameter:

– hypochromous:

– hyperchromous:
– normochromous:

III. By cell sizes:

– microcytic:
– macrocytic:
– normocytic:

IV. By the ability of the bone marrow for regeneration:

– hypo-and aregenerative:
– generative and hyperregenerative:

Table 2

Morphology of peripheral blood erythrocytes in anemias

Degenerative forms of erythrocytes	**Is most common in the pathology of**
Microcytes	
Macro (megalo-)cytes	
Microspherocytes	
Sickle erythrocytes	
Torocytes (codocytes)	
Hypochromous erythrocytes (anulocytes)	
Hyperchromous erythrocytes	
Megaloblasts	
Erythrocytes with Howell–Jolly bodies, Cabot's rings	
Erythrocytes with Heinz bodies	
Anisocytosis, poikilocytosis	
Degmacyte («nibbled erythrocyte»)	
Echinocyte	
Schistocyte	

Control questions:

1. The definition of the notions «anemia» and «erythrocytosis».
2. Classification principles of anemia:
   a) by etiopathogenesis;
   b) by the color parameter;
   c) by the hemopoiesis type;
   d) by the abilities of the bone marrow for regeneration;
   e) by erythrocytes sizes.
3. Etiology, pathogenesis, general characteristic, blood pattern in anemia due to blood loss:
   a) acute posthemorrhagic anemia;
   b) chronic posthemorrhagic anemia.
4. Etiology, pathogenesis, general characteristic, blood pattern in anemia due to impaired hemopoiesis (dyserythropoietic):
   a) iron-deficiency;
   b) sideroachrestic;
   c) B_{12}-(folic acid) deficiency;
   d) B_{12}-(folic acid)-achrestic;
   e) hypo- and aplastic, metaplastic.
5. Etiology, pathogenesis, general characteristic, blood pattern in anemia due to enhanced hemopoiesis:
   a) membranopathies (hereditary microspherocytosis);
   b) enzymopathies (deficiency of glucose-6-phosphatedehydrogenase of erythrocytes);
   c) hemoglobinopathies (sickle-cellular anemia; thalassemia);
   d) anemia due to exposure of antibodies and other damaging factors.
6. Impairments and compensatory-adaptive processes in the organism in anemia.
7. Erythrocytosis. The definition of the notion. Types (primary and secondary, absolute and relative). Etiology and pathogenesis, blood pattern in erythremia (Vaquez disease).

The teacher's signature:

** blood smears are kindly given by Prof. E. D. Buglov, Dr. of Medical Sciences and are selected by V. J. Peretjatko from the archives of the Institute of pediatric oncohematology of the Health Ministry of the Republic of Belarus.

LESSON 3. LEUKOPOIESIS, ITS IMPAIRMENTS. LEUKOCYTOSIS, LEUKOPENIAS

Date: «____» _____ 20___

The purpose of the Lesson:

– To study quantitative and qualitative changes in leukocyte system; typical forms of their impairments, types of blood leukocyte profile in pathology.

Tasks:

– To get acquainted with general laws and basic typical pathological forms and reactive changes in leukocyte system on the basis of materials presented on tables for the given topic and the blood pattern in peripheral blood smears of patients.

– To draw the cells of IV–VI classes of granulocyto-, lympho- and monocytopoiesis using materials of the textbook, hematologic atlas, album, slides and tables.

– To draw pathological forms of leukocytes showing some impairments in the leukocyte system using materials of the textbook, hematologic atlas and tables.

– To study under the microscope and draw the blood pattern in neutrophilic and eosinophilic leukocytosis.

– To discuss some complete blood count (CBC) including typical pathological forms and reactive changes in leukocyte system (№ 12–20), to acquire skills of solving situational tasks (№ 16–18) on the topic of the Lesson (see the collection of situational tasks on pathological physiology).

– To get acquainted with clinical characteristic of impairments in the leukocyte system.

Work 1. ACQUAINTANCE WITH MORPHOFUNCTIONAL CHARACTERISTIC OF PATHOLOGICAL FORMS OF LEUKOCYTES

Draw pathological (degenerative) forms of leukocytes using presented tables, materials of the textbook, hematological atlas and album.

1. _____ 2. _____

3. _____ 4. _____

5. _____ 6. _____

Fig. 4. Degenerative forms of leukocytes:
1 — neutrophilic leukocytes with toxic granularity; 2 — with vacuolization of the nucleus and cytoplasm; 3 — with hyper- and hyposegmentation of the nucleus; 4 — with bodies of Khiazkov–Dele; 5 — with chromatolysis; 6 — rod nuclear with thorns

Answer the question:

What does the occurrence of degenerate forms of leukocytes in peripheral blood mean?

Work 2. STUDYING A BLOOD SMEAR IN NEUTROPHILIC LEUKOCYTOSIS (large eosinophilia)**

Examine a blood smear of the patient with neutrophilic leukocytosis under the microscope with 10 × 90 magnification. Pay attention to the great number of neutrophilic leukocytes of various degree of maturity in the field of vision. Draw them.

Fig. 5. Blood smear at the neutrophilic leukocytosis:
1 — a metamyelocyte; 2 — a rod neuclear neutrophil; 3 — a segmented neutrophil; 4 — a neutrophil with toxic granularity

Work 3. STUDYING A BLOOD SMEAR IN EOSINOPHILIC LEUKOCYTOSIS (large eosinophilia)**

Examine a blood smear of the patient with large eosinophilia under the microscope with 10 × 90 magnification. Pay attention to the great number of eosinophilic leukocytes of various degree of maturity in the field of vision. Draw them.

Fig. 6. Blood smear at the eosinophilic leukocytosis (large eosinophilia):
1 — a rod shaped eosinophil; 2 — a segmented eosinophil; 3 — a segmented neutrophil

Answer the questions:

1. What does it mean the large eosinophilia?

2. What pathological states the large eosinophilia is characteristic for?

Work 4. STUDYING TYPICAL CHANGES OF THE LEUKOCYTE FORMULA

Define the notions:

Relative «-cytosis (-philia)» or «-penia» is …

Absolute «-cytosis (-philia)» or «-penia» is …

The formula for expressing ***relative*** parameters (i.e. %) of the leukocyte formula in ***absolute*** *ones***:**

$$\textbf{Abs. value} = \frac{\textbf{\%}}{\textbf{100}} \times \boldsymbol{L},$$

where *L* — the quantity of leukocytes in 1 unit of blood volume (in l or mm^3).

Specify *the quantitative* (in 1 unit of blood volume) **range of the following complete blood count changes**:

Absolute neutrophilia —

Absolute neutropenia —

Agranulocytosis —

Absolute lymphocytosis —

Absolute lymphopenia —

Specify the basic developmental mechanisms of leukocytosis:

1 –

2 –

3 –

Specify the basic developmental mechanisms of leukopenias:

1 –

2 –

3 –

4 –

Using the material of the textbook and other sources, fill in the tables:

Table 1

Types of leukocytosis and leukopenias

Character of the leukocyte formula changes (in absolute figures)	**Most common conditions, for which the given leukocyte formula change is characteristic of**
Neutrophilia (neutrophilic leukocytosis)	
Neutropenia	
Eosinophilia	
Eosinopenia or aneosinophilia	
Lymphocytosis	
Lymphopenia	
Monocytosis	
Monocytopenia	
Agranulocytosis	
Panmyelophthisis	

Table 3

Leukocyte formula changes in some pathological conditions

Pathological condition		Characteristic changes of the leukocyte formula	
Acute bacterial (coccal) infection	Peak of the disease		
	Recovery period		
	Proceeding as a sepsis type		
Acute viral (flu, measles, German measles) infection, peak of the disease			
Chronic specific infection			
Allergic conditions, helminthic invasion			
Agranulocytosis			

Answer the questions:

1. Give the formula for calculation of NSI (nucleo shift index):

The normal value of NSI (nucleo shift index):

2. What does the presence of regenerative and hyperregenerative shifts of the leukocyte formula to the left mean?

3. What does the presence of degenerative shifts of the leukocyte formula to the right mean?

Control questions:

1. Leukopoiesis, its impairments.
2. Pathological forms of leukocytes, their morphofunctional peculiarities.
3. Leukopenia, the definition of the notion, the cause and developmental mechanisms, its types.
4. Agranulocytosis, the definition of the notion. Types of agranulocytosis, the causes and their developmental mechanisms. Peripheral blood pattern in various types of agranulocytosis.
5. Panmyelophthisis. The causes and its developmental mechanisms, the pattern of peripheral blood and bone marrow in panmyelophthisis.
6. Leukocytosis, the definition of the notion, types, the causes and developmental mechanisms.
7. Leukocyte formula changes, absolute and relative changes of some types of leukocytes, pathogenetic and prognostic characteristic.
8. The characteristic, pathogenetic and prognostic characteristic of various types of the leukocyte formula shifts.

The teacher's signature:

** blood smears are kindly given by Prof. E. D. Buglov, Dr. of Medicine and are selected by V. J. Peretjatko from the archives of the Institute of pediatric oncohematology of the Health Ministry of the Republic of Belarus.

LESSON 4. HEMOBLASTOSIS. LEUKEMOID REACTIONS

Date: «____» _____ 20___

The purpose of the Lesson:

– To study the reasons, developmental mechanisms and hematologic reactions of leukosis (basic types of leukograms in leukosis).

Tasks:

– To get acquainted with morphofunctional peculiarities of cells observed in the patients' blood with some types of leukosis.

– To study under the microscope and to draw the blood pattern in some types of leukosis (acute and chronic myelo- and lympholeukoses). To draw the blood pattern in acute myeloleukosis using the hematological atlas and tables.

– To analyze a series of complete blood count (№ 21–29) of patients with leukosis and to define the presence, type and form of leukosis, to acquire skills of solving situational tasks (№ 15, 19–26) on the topic of the Lesson (see the collection of situational tasks on pathological physiology).

– Test control of the topic «Leukocytosis, leukopenias and leukosis».

Work 1. EXAMINATION OF MORPHOFUNCTIONAL CHARACTERISTIC LEUKEMIC CELLS, BLOOD SMEAR AND SOME SYNDROMES AT THE LEUKEMIAS

Whereas the names of various types of leukemia come from the names of parental cells — progenitor cells of normal hemopoiesis, with which leukemic cells have a number of common features, study morphological peculiarities of proliferating and mature cells of granulocytopoiesis, lymphocytopoiesis and monocytopoiesis in leukemia using the text-books (albums, tables, slides) and under the microscope.

Using materials of the textbook, hematological atlas, tables and slides fill in the tables:

Table 1

Comparative characteristic of the blood pattern in acute and chronic myeloleukosis (during the advanced stage)

Myeloid leukemia type	Presence (1) and (or) prevalence (2) of blast cells	Presence of all maturing cells of class V (+/-)	Leukemic failure (+/-)	Eosinic-basophilic association (+/-)	Ph¢-chromosome in cells of the myeloid series (+/-)	Pancytopenia (+/-)
Acute						
Chronic						

Answer questions:

1. What basic criterion is used for division of leukemia into acute and chronic?

2. What does it mean a «leukemic failure»? For what leukemia it is characteristic?

3. The prevalence of what cells in peripheral blood (blastic or maturing (mature) is typical of acute and chronic leukemia?

4. Of what leukemia (acute or chronic) is pancytopenia more characteristic? Specify the principal cause of its development.

Table 2

Basic syndromes in acute leukemia

Syndrome	Developmental mechanisms	Basic manifestations
Anemic		
Hemorrhagic		
Infectious		
Intoxication		
Leukemic infiltration of organs and tissues (metastatic)		
Osteoarthropathic		

Study blood smears of patients with a leukemic form of acute myeloid leukemia under the microscope with 10 × 90 magnification. ** While examining the blood smear pay attention to the number, morphology of blood cells, cellular polymorphism.

In particular, pay attention, that blood smears of patients with **acute myeloid leukemia** (sub-or leukemic forms) reveal a great number of blastic cell alongside with increase of the number of leukocytes in the field of vision; the absence of intermediate forms and the presence of single segmented neutrophiles (*hiatus leukaemicus*).

Fig. 1. Peripheral blood smear at the subleukemic or leukemic form of acute myeloid leukemia: 1 — a blastic cell; 2 — a segmented neutrophile

Conclusion:

Characterize basic changes of cellular composition of peripheral blood in acute myeloid leukemia.

Work 2. STUDYING QUANTITATIVE AND QUALITATIVE CHANGES OF LEUKOCYTES IN BLOOD SMEARS OF PATIENTS WITH SOME TYPES OF CHRONIC LEUKEMIA**

Study blood smears of patients with leukemic forms of chronic leukemia under the microscope with 10 × 90 magnification. While examining blood smears pay attention to the quantity, morphology of blood cells, cellular polymorphism.

In particular, pay attention, that blood smears of patients with **chronic myeloid leukemia** (sub- or leukemic forms), alongside with increase of the number of leukocytes reveal also:

– all morphologically revealed cells of granulocytopoiesis: myeloblasts, promyelocytes; neutrophilic, eosinophilic and basophilic myelocytes, metamyelocytes, rod nuclear and segmented cells;

– the content of eosinophils and basophils (eosinophilic-basophilic association) in the field of vision is increased.

Fig. 2. Blood smear at the chronic myeloid leukemia:
1 — a myeloblast; 2 — promyelocytes; 3 — a myelocyte: neutrophilic *(a)*, eosinophilic *(b)* and basophilic *(c)*; 4 — a metamyelocyte (young) (*a, b, c)*; 5 — rod-nuclear (*a*, *b, c*); 6 — segmented (*a*, *b, c*)

Blood smears of patients with **chronic lymphoid leukemia** (sub- or leukemic forms) reveal the presence of all morphologically defined cells of lymphocytopoiesis: lymphoblasts, prolymphocytes, lymphocytes (the last ones prevail in the field of vision) alongside with a great number of leukocytes in the field of vision. There are also revealed cells — shadows of lymphocytes (cells of Botkin–Humbprecht).

Fig. 3. Blood pattern in chronic lymphoid leukemia:
1 — a lymphoblast; 2 — a prolymphocyte; 3 — lymphocytes; 4 — cells (shadows) of Botkin–Humprecht

Conclusions:

Characterize the basic changes of cellular content of peripheral blood in chronic leucosis by filling the table.

Table 3

Comparative characteristic of the peripheral blood pattern in the advanced stage of chronic leukemia

Leukemia type	Prevalence of blasts or maturing and mature forms in blood	Cells of a tumoral process occurring in blood	Specific hematologic «markers» of leukemia	Red blood status	Number of thrombocytes in blood
Myeloid leukemia					
Lymphoid leukemia					

Control questions:

1. Leukemia, the definition of the notion. General characteristic and principles of classification.
2. Etiology and pathogenesis of leukemia. Modern theories of the origin of leukemia. The tumoral nature of leukemia.
3. Peculiarities of leukemic cells, their morphological, cytochemical and cytogenetic characteristic.
4. Peculiarities of hemopoiesis and cellular content of the blood in various types of leukemia.
5. Basic impairments in the organism in leukemia, their mechanisms.
6. Leukemoid reactions. Basic types, causes, blood pattern, differentiation from leukemia.
7. Principles of diagnosis and therapy of leukemia.

The teacher's signature:

** blood smears are kindly given by Prof. E. D. Buglov. Dr of Medicine and are selected by V. J. Peretjatko from the archives of the Institute of pediatric oncohematology of the Health Ministry of the Republic of Belarus.

LESSON 5. IMPAIRMENTS OF THE TOTAL BLOOD VOLUME. BLOOD LOSS

Date: «____» _____ 20___

The purposes of the Lesson:

– To consider typical impairment forms of the total blood volume, their causes and consequences, factors affecting their severity. To study semiology, pathogenesis of posthemorrhagic conditions, forms and mechanisms of compensatory reactions in blood loss.

– To get acquainted with principles of treating acute blood losses.

The task:

- To analyze available experimental protocols (see work 1) of studying:

– effects of acute blood loss of various volume and bleeding velocity on severity of arising impairments by the parameters of blood pressure, heart rate, respiration;

– manifestations of urgent compensatory reactions of the organism in acute blood losses of various severity;

– effects on the parameters of hemodynamics and respiration after acute massive blood loss in transfusions of: a) physiological solution; b) blood.

- To acquire skills of solving situational tasks (№ 1–2) (see the collection of situational tasks on pathological physiology).

Work 1. STUDYING THE EFFECTS OF BLOOD LOSS AND SUBSEQUENT INTRAVENOUS TRANSFUSIONS OF PHYSIOLOGICAL SOLUTION AND BLOOD ON THE DOG'S ORGANISM

Both femoral arteries and a femoral vein of a narcotized dog are prepared. A cannula is inserted into the one of the arteries and is connected to the manometer, for registration of arterial pressure. Then cannulas are inserted into the other femoral artery and the vein for bloodletting and subsequent transfusion of blood or isotonic solution of sodium chloride.

For graphic registration of respiratory excursions a special cuff is fixed on the thorax of the animal, being connected by a rubber tube with Marey's.

The animal's circulating blood volume (CBV) is calculated on the basis of its body mass.

After the initial parameters have been recorded, 5 % of CBV is ***slowly*** let out from the artery into a glass vessel, meanwhile registering changes of heart rate (HR), arterial pressure (BP) and respiration rate (RR).

In 5 min ***stream*** bloodletting is repeated, the same volume of blood being taken out (total blood loss makes up 10 % of the animal's blood mass). Pay attention to distinctions of the registered parameters; analyze the causes and also the mechanisms of fast normalization of BP and HR. For revealing compensatory mechanisms of the organism carry out the third ***(stream)*** bloodletting in the volume of 10 % of blood; all parameters are being recorded.

In 5 min ***massive stream*** bloodletting is performed, about $^1/_3$ of the total blood mass being taken out. Observe the persistent significant decrease of BP, significant amplitude reduction of pulse waves of the 1st order, tachycardia, inspiratory breathlessness. Analyze the received results.

To decide, which of the factors (decrease of blood pressure or loss of erythrocytes) plays a leading part in the development of hypoxia and death of the organism in acute massive blood loss, make sure that BP persists at a critically low level, the animal is IV injected 100–150 ml of warmed up physiological solution and 5 min later — autogenic blood (60 % of the lost volume), BP and RR being registered.

Table 1

Experimental results

Type of exposure	Blood pressure (mm Hg)	Pulse (beats/min)	Respiration (resp./min)
The initial data	130/100	86	12
Bloodletting(5 % of blood, slowly)	125/100	90	14
In 5 min	130/95	90	14
Bloodletting (5 % of blood, fast)	115/95	106	15
In 5 min	125/95	105	16
Bloodletting (10 % of blood, fast)	65/60	120	14
In 5 min	120/110	95	14
Bloodletting(30 % of blood, fast)	30/25	60	0
In 5 min	60/50	100	Inspiratory breathlessness
Intravenous injection of physiological solution (150 ml)	85/65	80	4
In 5 min	80/65	90	8
Intravenous injection of 60 % of the lost blood	130/110	108	32
In 5 min	135/110	80	16

Using the data of the table, construct the graphs showing, in dynamics of experiment, changing of systolic (1) and diastolic (2) blood pressure, heart rate (3) and respiration rate (4), marking with a vertical arrow the effect and character of this or that exposure.

Fig. 1. Changes of some parameters of the dog's cardio-respiratory system in dynamics of acute blood loss and in various ways of its correction:
1 — systolic pressure; 2 — diastolic pressure; 3 — HR; 4 — RR

Answer the questions:

1. What causes the absence of essential changes on the part of BP, HR and respiration in slow blood loss comprising 5 % of the blood volume of the animal?

2. Why can you observe a visible (in blood loss of 5 % of the blood volume) and significant (in additional loss of 10 % of blood) decrease of BP?

3. Due to what compensatory mechanisms is the BP normalization achieved 5 min later under the above mentioned variants of experiment?

4. Taking into account the changes of analyzed parameters, estimate the condition of the organism 5 min later after the last stream massive bloodletting exceeding in total 50 % of blood volume?

5. What caused some elevation of BP after transfusion of 150 ml of physiological solution to the dog that has lost a half of its blood volume during 25–30 min?

Conclusions:

1. Give the pathogenic background of performing stage-by-stage transfusion therapy to correct the state of vital functions in acute massive blood loss.

Control questions:

1. Typical forms of pathology and reactive changes of the total blood volume. Normo-, hypo- and hypervolemias and their types depending on the relationship of corpuscular elements and blood plasma. The causes of their incidence, clinical manifestations.
2. Blood loss: acute and chronic. Their causes, characteristic.
3. Master factors of the course and outcomes of posthemorrhagic conditions.
4. Basic components of pathogenesis of posthemorrhagic conditions.
5. Types and mechanisms of compensatory reactions (urgent and long-term) in blood loss.
6. Centralization of blood circulation in acute blood loss; its essence, mechanisms, pathogenetic assessment.
7. The causes of death in acute blood loss.
8. Principles and methods of blood loss treatment.

The teacher's signature:

LESSON 6. IMPAIRMENTS OF HEMOSTASIS

Date: «____» _____ 20___

The purpose of the Lesson:

– To study the basic forms of hemostasis impairments, their incidence causes, development mechanisms, clinical and hematological manifestations.

Tasks:

– To acquire skills of solving situational tasks (№ 27–34) of the topic of the Lesson (see the collection of situational tasks on pathological physiology) with formulation of a prospective diagnosis.

– To get acquainted with some methods of diagnosing hereditary coagulopathies, to analyze the presented results of correcting hemostasis impairments, to determine their type using the collection of situational tasks on pathological physiology.

Work 1. DIFFERENTIAL DIAGNOSTICS OF HEREDITARY COAGULOPATHIES BY THE TEST OF MIXING

The given test is based on the principle of correcting impairments of plasma coagulation using specially prepared samples of plasma being aware of the deficiency of this or that coagulation factor.

If the added plasma (standard) corrects the impairments of coagulation parameters, then there is a deficiency of different coagulation factors in it and in the tested plasma; if it does not — then one and the same defect is present in them.

Method of investigation

Mix 0.2 ml of beforehand prepared plasma which is a standard with obviously known deficiency (the factor content **is 1 % less than the norm**) of factors VIII, IX, XI, XII and 0.8 ml of the tested plasma. Then its activated partial platelet time (APPT), thrombin time (TT) and prothrombin time (PT) are determined.

Table 1

Results of correcting hemostasis parameters of tested plasma samples

Plasma samples with hereditary coagulopathy		Added standards of plasma with obviously known plasma factor deficiency				Diagnostic conclusion regarding this or that factor deficiency in tested plasma
		Factor deficiency				
		VIII	IX	XI	XII	
Tested plasma	1	APPT – 80s TT – 14 s PT – 13 s	APPT – 54s TT – 16 s PT – 12 s	APPT – 55s TT – 15 s PT – 14 s	APPT – 54s TT – 14 s PT – 16 s	
	2	APPT – 55s TT – 16 s PT – 12 s	APPT – 56s TT – 16 s PT – 13 s	APPT – 54s TT – 14 s PT – 15 s	APPT – 102s TT – 15 s PT – 12 s	
	3	APPT – 56s TT – 15 s PT – 15 s	APPT – 55s TT – 15 s PT – 12 s	APPT – 98s TT – 16 s PT – 13 s	APPT – 55s TT – 16 s PT – 15 s	
	4	APPT – 57s TT – 13 s PT – 14 s	APPT – 100s TT – 14 s PT – 14 s	APPT – 55s TT – 15 s PT – 16 s	APPT – 54s TT – 15 s PT – 14 s	
	5	APPT – 87s TT – 14 s PT – 12 s	APPT – 93s TT – 15 s PT – 12 s	APPT – 57s TT – 16 s PT – 14 s	APPT – 55s TT – 15 s PT – 16 s	
	6	APPT – 56s TT – 16 s PT – 16 s	APPT – 91s TT – 16 s PT – 13 s	APPT – 96s TT – 15 s PT – 15 s	APPT – 55s TT – 14 s PT – 14 s	

Answer the question:

1. What phase and what activation mechanism of blood coagulation are impaired in the presented tests of blood plasma with hereditary coagulopathy?

Work 2. STUDYING A BLOOD SMEAR IN ESSENTIAL THROMBOCYTHAEMIA

Examine a blood smear under the microscope with 10 × 90 magnification. Pay attention to the great number of thrombocytes in the field of vision. Draw them.

Fig. 1. Blood smear in essential thrombocythaemia:
1 — erythrocytes; 2 — neutrophilic leukocytes; 3 — thrombocytes

Control questions:

1. The hemostasis system. The definition of the notion, functional purpose. The moderm scheme of blood coagulation, regulation mechanisms.
2. Hemostasiopathies. The definition of the notion. Classification of the hemostasis system impairments.
3. The impairment of vasculo-thrombocyte hemostasis. The causes, development mechanisms, clinical manifestations.
4. Incidence causes, development mechanisms, clinical and hematological manifestations of thrombocytopathies (hereditary and acquired); thrombocytopenias; thrombocytoses (reactive and primary).
5. Coagulation hemostasis impairments caused by hereditary and (or) acquired deficiency of the blood coagulation factor (hemophilia A, B, C, mixed hemophilias, parahemophilias, etc.), their pathogenesis, clinical manifestations, laboratory diagnostics, principles of treatment.
6. The anti-coagulation system. Factors, regulation mechanisms. The causes, development mechanisms, consequences of regulation impairments of the blood coagulation system.
7. The impairments of vascular hemostasis (vasopathy) and mixed genesis, development mechanisms, basic clinical manifestations, laboratory diagnostics, principles of treatment.
8. Purpura and other hemorrhagic conditions (immune and non-immune thrombocytopenic purpuras). Classification, basic clinical manifestations, laboratory diagnostics, principles of treatment.
9. Fibrinolysis and its impairments. Etiology, pathogenesis and clinical manifestations.
10. Thrombotic syndrome. Etiology and pathogenesis.
11. Hemorrhagic syndrome. Etiology and pathogenesis.
12. Thrombohemorrhagic syndrome (DIC-syndrome (disseminated intravascular coagulation)) or a syndrome of intravascular microcoagulation of blood. Etiologic and pathogenetic factors of development, clinical manifestations, laboratory diagnostics, principles of treatment.
13. Basic tests characterizing vascular-thrombocytic and coagulation hemostasis, their diagnostic value.

The teacher’s signature:

LESSON 7. THE FINAL LESSON IN THE SECTION «PATHOLOGICAL PHYSIOLOGY OF THE BLOOD SYSTEM»

Date: «____» _____ 20___

The purpose of the Lesson:

– To consolidate and evaluate knowledge received at six previous laboratory works and while studying the corresponding section of the textbook on the problems concerning pathophysiological aspects of various pathologies of the blood system.

Control questions:

1. Hemopoiesis and its impairments. General characteristic.
2. Anemias. The definition of the notion. Principles of classification. Anemia as a syndrome and as a nosological form. Qualitative and quantitative changes of an erythron in anemias.
3. Etiology, pathogenesis, general characteristic of anemias caused by blood loss. Blood pattern.
4. Etiology, pathogenesis, general characteristic of anemias caused by the impairment of hemopoiesis (dyserythropoietic). Blood pattern.
5. Etiology, pathogenesis, general characteristic of anemias caused by intense blood destruction. Blood pattern.
6. Impairments and compensatory-adaptive processes in the organism in anemias.
7. Erythrocytoses, their types (primary and secondary, absolute and relative). Etiology and pathogenesis of erythremia (Vaquez disease), Blood pattern.
8. Leukocytosis and leukopenias, their types, causes and developmental mechanisms, pathogenetic characteristic.
9. Agranulocytosis. The definition of the notion, its types, etiology, pathogenesis. Blood pattern in various types of agranulocytosis.
10. Panmyelophthisis. Its causes, development mechanism and consequences. The pattern of peripheral blood and bone marrow in panmyelophthisis.
11. Leukemia. The definition of the notion. Etiology and pathogenesis. Modern theories of the origin of leukemia. Principles of classification. Blood pattern.
12. Leukemoid reactions, their types. Etiology and pathogenesis, distinctions from leukocytosis and leukemia. Blood pattern.
13. Hemostasis. The definition of the notion, types of hemostasis, general characteristic.
14. Hemostasiopathies. The definition of the notion. Impairments classification of the hemostasis system.

15. Coagulation hemostasis impairments caused by hereditary or acquired deficiency of blood coagulation factors, their pathogenesis, clinical manifestations. Hemophilias.

16. Quantitative and qualitative changes of thrombocytes. Thrombocytoses, thrombocytopenias and thrombocytopathies, their types and differentiative peculiarities.

17. Hemostasis impairments of vascular and mixed genesis (vasopathy), their mechanisms, basic clinical manifestations.

18. Thrombotic syndrome. Etiology and pathogenesis.

19. Hemorrhagic syndrome. Etiology and pathogenesis.

20. Thrombohemorrhagic syndrome (DIC-syndrome) or a syndrome of intravascular blood microcoagulation. Etiology and pathogenesis.

21. Typical forms of the blood volume changes. Normo-, hypo- and hypervolemias and their types depending on the relationship of corpuscular elements and blood plasma; their causes and manifestations.

22. Blood loss and its types.

23. Main factors of blood loss consequences.

24. Basic components of pathogenesis of posthemorrhagic conditions.

25. Types and compensation mechanisms of impaired functions in blood loss.

26. Centralization of the blood flow in acute blood loss and its mechanisms, pathogenetic characteristic.

27. The causes of death in acute blood loss.

28. Principles and methods of blood loss treatment.

The final Lesson also includes:

1. The ability to analyze complete blood count and to solve situational tasks in details analyzing the state of both red and white blood and backgrounding the conclusion regarding a possible pathology, for which the given blood pattern is characteristic.

2. The ability to identify the morphology, pathological changes of single cells and blood patterns as a whole, as well as to determine a type of possible pathology by microphotos.

The teacher's signature:

Complete blood count (CBC)

№ 1

RBC (erythrocytes))	**3.79 × 10^{12}/l**	
HGB (hemoglobin)	83 g/l	
HCT	27.8 %	
MCV	73.3 %	
MCH	21.9 pg/cell	
MCHC	29.9 g/dcl	
RDW	20.8 %	
WBC (leukocytes)	**6.4 × 10^{9}/l**	
basophiles	1 %	
eosinophiles	3 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	4 %	
– segmentated	62 %	
lymphocytes	20 %	
monocytes	10 %	
PLT (thrombocytes)	**415.0 × 10^{9}/l**	
ESR	12 mm per h	
Blood serum iron — 6,85 micromol/l.		
Conclusion:		

№ 2

RBC (erythrocytes)	**3.5 × 10^{12}/l**	
Hemoglobin	72 g/l	
HCT	25 %	
MCV	calculate	
MCH	calculate	
RDW	15.5 %	
WBC (leukocytes)	**3.6 × 10^{9}/l**	
basophiles	0 %	
eosinophiles	3 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	5 %	
– segmentated	64 %	
lymphocytes	23 %	
monocytes	5 %	
PLT (thrombocytes)	**180.0 × 10^{9}/l**	
ESR	8 mm per h	
Blood serum iron — 58,3 micromol/l.		
Conclusion:		

№ 3

RBC (erythrocytes)	**3.36×10^{12}/l**	
Hemoglobin	67 g/l	
Color index	calculate	
WBC (leukocytes)	**5.1×10^{9}/l**	
basophiles	0 %	
eosinophiles	2 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	5 %	
– segmentated	51 %	
lymphocytes	38 %	
monocytes	4 %	
PLT (thrombocytes)	**180.0×10^{9}/l**	
ESR	15 mm per h	
In the smear: poikilocytosis, microcytosis.		
Conclusion:		

№ 3a

RBC (erythrocytes)	**3.36×10^{12}/l**	
Hemoglobin	67 g/l	
Color index	calculate	
HCT	22 %	
MCV	calculate	
MCH	calculate	
RDW	16.9 %	
WBC (leukocytes)	**5.1×10^{9}/l**	
basophiles	0 %	
eosinophiles	2 %	
neutrophiles:		
– myelocytes	0 %	
– young	70 %	
– band	5 %	
– segmentated	51 %	
lymphocytes	38 %	
monocytes	4 %	
PLT (thrombocytes)	**180.0×10^{9}/l**	
ESR	15 mm per h	
Conclusion:		

№ 4

RBC (erythrocytes)	**1.58×10^{12}/l**	
Hemoglobin	68 g/l	
Reticulocytes	0 %	
Color index	calculate	
WBC (leukocytes)	**2.8×10^{9}/l**	
basophiles	0 %	
eosinophiles	0 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	1 %	
– segmented	42 %	
lymphocytes	55 %	
monocytes	2 %	
PLT (thrombocytes)	**85.0×10^{9}/l**	
ESR	28 mm per h	
In the smear: megalocytes, megaloblasts, macrocytosis, anisocytosis, poikilocytosis, RBC (erythrocytes) with Howell–Jolly bodies, Cabot's rings, polysegmentated neutrophiles.		
Conclusion:		

№ 4a

RBC (erythrocytes)	**1.58×10^{12}/l**	
Hemoglobin	68 g/l	
Reticulocytes	0 %	
HCT	18 %	
Color index	calculate	
MCV	calculate	
MCH	calculate	
RDW	18.7 %	
WBC (leukocytes)	**2.8×10^{9}/l**	
Basophiles	0 %	
eosinophiles	0 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	1 %	
– segmentated	42 %	
lymphocytes	55 %	
monocytes	2 %	
PLT (thrombocytes)	**85.0×10^{9}/l**	
ESR 28 mm per h. In the smear: megalocytes, megaloblasts, RBC (erythrocytes) with Howell–Jolly bodies bodies, Cabot's rings, polysegmentated neutrophiles.		
Conclusion:		

№ 5

RBC (erythrocytes)	**$2{,}0 \times 10^{12}$ /l**	
Hemoglobin	70 g/l	
Reticulocytes	0,05 %	
HCT	20,5 %	
Color index	calculate	
MCV	102,5 fl.	
MCH	35 pg/cell	
MCHC	calculate	
RDW	15,2 %	
WBC (leukocytes)	**$2{,}5 \times 10^{9}$ /l**	
Basophiles	1 %	
eosinophiles	2 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	2 %	
– segmentated	52 %	
lymphocytes	41 %	
monocytes	2 %	
PLT (thrombocytes)	**$80{,}0 \times 10^{9}$ /l**	
ESR	30 mm per h.	
In the smear: anisocytosis, toxic granularity of neutrophils.		
Conclusion:		

№ 6

RBC (erythrocytes)	**$2{,}7 \times 10^{12}$/l**	
Hemoglobin	68 g/l	
Reticulocytes	5 %	
Color index	calculate	
WBC (leukocytes)	**12.0×10^{9}/l**	
Basophiles	0 %	
eosinophiles	2 %	
neutrophiles:		
– myelocytes	0 %	
– young	7 %	
– band	17 %	
– segmentated	53 %	
lymphocytes	17 %	
monocytes	4 %	
PLT (thrombocytes)	**150.0×10^{9}/l**	
ESR	18 mm per h	
In the smear: polychromatophiles, single normoblast.		
Conclusion:		

№ 6a

RBC (erythrocytes)	**$2{,}7 \times 10^{12}$/l**	
Hemoglobin	68 g/l	
Reticulocytes	5.0 %	
HCT	24 %	
Color index	calculate	
MCV	calculate	
MCH	calculate	
RDW	13.8 %	
WBC (leukocytes)	**12.0×10^{9}/l**	
basophiles	0 %	
eosinophiles	2 %	
neutrophiles:		
– myelocytes	0 %	
– young	7 %	
– band	17 %	
– segmentated	53 %	
lymphocytes	17 %	
monocytes	4 %	
PLT (thrombocytes)	**150.0×10^{9}/l**	
ESR	18 mm per h	
In the smear: polychromatophiles, single normoblast.		
Conclusion:		

№ 7

RBC (erythrocytes)	**1.9×10^{12}/l**	
Hemoglobin	45 g/l	
Color index	calculate	
Reticulocytes	12 %	
WBC (leukocytes)	**7.8×10^{9}/l**	
basophiles	0.5 %	
eosinophiles	1.5 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	4 %	
– segmentated	60 %	
lymphocytes	28 %	
monocytes	6 %	
PLT (thrombocytes)	**350.0×10^{9}/l**	
ESR is 1 mm per h. In the smear: crescent RBC (erythrocytes), meniscocytes.		
Conclusion:		

№ 8

RBC (erythrocytes)	**3.32×10^{12}/l**	
Hemoglobin	72 g/l	
Color index	calculate	
Reticulocytes	10 %	
WBC (leukocytes)	**4.4×10^{9}/l**	
basophiles	0.5 %	
eosinophiles	2 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	3 %	
– segmentated	54.5 %	
lymphocytes	35 %	
monocytes	5 %	
PLT (thrombocytes)	**180.0×10^{9}/l**	
ESR is 20 mm per h. In the smear: anisocytosis, poikilocytosis, basophil punctuation of RBC (erythrocytes), target-like RBC (erythrocytes), microcytosis. Blood serum iron — 64 mcmol/l. Osmotic resistance of RBC (erythrocytes) is increased.		
The presumable **conclusion:** What additional examination is necessary for making the diagnosis more precise?		

№ 9

RBC	**$2{,}4 \times 10^{12}$ /l**	
Hemoglobin	85 g/l	
Reticulocytes	35 %	
HCT	20 %	
Color index	calculate	
MCV	69 fl.	
MCH	35,4	
MCHC	42,4	
WBC (leukocytes)	**$6{,}1 \times 10^{9}$ /l**	
basophiles	0 %	
eosinophiles	0 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	3 %	
– segmentated	60 %	
lymphocytes	32 %	
monocytes	5 %	
PLT (thrombocytes)	**$200{,}0 \times 10^{9}$ /l**	
ESR	19 мм в час	
In the smear: microspherocytosis, normoblasts. Osmotic resistance of erythrocytes is lowered.		
Conclusion:		

№ 10

RBC (erythrocytes)	**6.6×10^{12}/l**	
Hemoglobin	174 g/l	
Color index	calculate	
Reticulocytes	5 %	
WBC (leukocytes)	**8.7×10^{9}/l**	
basophiles	0 %	
eosinophiles	1 %	
neutrophiles:		
– myelocytes	0 %	
– young	1 %	
– band	5 %	
– segmentated	65 %	
lymphocytes	24 %	
Monocytes	54 %	
PLT (thrombocytes)	**280.0×10^{9}/l**	
ESR	8 mm per h	
Conclusion:		

№ 11

RBC (erythrocytes)	**7.32×10^{12}/l**	
Hemoglobin	180 g/l	
HCT	57 %	
Color index	calculate	
Reticulocytes	3 %	
WBC (leukocytes)	**16.4×10^{9}/l**	
basophiles	0.5 %	
eosinophiles	7.5 %	
neutrophiles:		
– myelocytes	0 %	
– young	3 %	
– band	10 %	
– segmentated	59 %	
lymphocytes	17 %	
monocytes	3 %	
PLT (thrombocytes)	**628.0×10^{9}/l**	
ESR is 1 mm per h. In the smear: polychromatophiles, single normoblasts.		
Conclusion:		

№ 12

RBC (erythrocytes)	**4.2×10^{12}/l**	
Hemoglobin	125 g/l	
Color index	calculate	
WBC (leukocytes)	**17.4×10^{9}/l**	
basophiles	0 %	
eosinophiles	0.5 %	
neutrophiles:		
– myelocytes	0 %	
– young	5 %	
– band	12 %	
– segmentated	64 %	
lymphocytes	14 %	
monocytes	4.5 %	
PLT (thrombocytes)	**290.0×10^{9}/l**	
ESR	25 mm per h	
Conclusion:		

№ 13

RBC (erythrocytes)	**3.22×10^{12}/l**	
Hemoglobin	75 g/l	
Color index	calculate	
WBC (leukocytes)	**30.0×10^{9}/l**	
basophiles	0 %	
eosinophiles	0 %	
neutrophiles:		
– myelocytes	6 %	
– young	17 %	
– band	30 %	
– segmentated	42 %	
lymphocytes	4 %	
monocytes	1 %	
PLT (thrombocytes)	**220.0×10^{9}/l**	
ESR	45 mm per h	
In the smear: toxic granularity of neutrophiles.		
Conclusion:		

№ 14

RBC (erythrocytes)	**3.8×10^{12}/l**	
Hemoglobin	116 g/l	
Color index	calculate	
WBC (leukocytes)	**14.8×10^{9}/l**	
basophiles	0 %	
eosinophiles	2 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	5 %	
– segmentated	21 %	
lymphocytes	60 %	
monocytes	12 %	
PLT (thrombocytes)	**185.0×10^{9}/l**	
ESR	17 mm per h	
Conclusion:		

№ 15

RBC (erythrocytes)	**4.4×10^{12}/l**	
Hemoglobin	130 g/l	
Color index	calculate	
WBC (leukocytes)	**8.8×10^{9}/l**	
basophiles	1 %	
eosinophiles	11 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	5 %	
– segmentated	54 %	
lymphocytes	24 %	
monocytes	5 %	
PLT (thrombocytes)	**200.0×10^{9}/l**	
ESR	10 mm per h	
Conclusion:		

№ 16

RBC (erythrocytes)	**4.28×10^{12}/l**	
Hemoglobin	142 g/l	
Color index	calculate	
WBC (leukocytes)	**3.2×10^{9}/l**	
eosinophiles	1 %	
basophiles	0 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	12 %	
– segmentated	23 %	
lymphocytes	57 %	
monocytes	7 %	
PLT (thrombocytes)	**285.0×10^{9}/l**	
ESR is 18 mm per h. Patient B., 28 years old with high temperature.		
Conclusion:		

№ 17

RBC (erythrocytes)	**3.84×10^{12}/l**	
Hemoglobin	120 g/l	
Color index	calculate	
WBC (leukocytes)	**1.0×10^{9}/l**	
basophiles	0 %	
eosinophiles	0.5 %	
neutrophiles:	0 %	
– lymphocytes	82 %	
– monocytes	17.5 %	
PLT (thrombocytes)	**182.0×10^{9}/l**	
ESR	17 mm per h	
Conclusion:		

№ 18

RBC (erythrocytes)	**2.96×10^{12}/l**	
Hemoglobin	97 g/l	
Color index	calculate	
WBC (leukocytes)	**1.0×10^{9}/l**	
basophiles	0 %	
eosinophiles	0 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	0 %	
– segmentated	15 %	
lymphocytes	68 %	
monocytes	17 %	
PLT (thrombocytes)	**85.0×10^{9}/l**	
ESR	49 mm per h	
In the smear: toxic granularity of neutrophiles. Note: quinsy with necrotic coating.		
Conclusion:		

№ 19

RBC (erythrocytes)	**0.56×10^{12}/l**	
Hemoglobin	17 g/l	
Color index	calculate	
WBC (leukocytes)	**0.9×10^{9}/l**	
basophiles	0 %	
eosinophiles	0 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	0 %	
– segmentated	12 %	
lymphocytes	86 %	
monocytes	2 %	
PLT (thrombocytes)	**25.0×10^{9}/l**	
ESR — 40 mm per h. In the smear: anisocytosis, poikilocytosis, toxic granularity of neutrophiles.		
Conclusion:		

№ 20

RBC (erythrocytes)	**4.36×10^{12}/l**	
Hemoglobin	118 g/l	
Color index	calculate	
WBC (leukocytes)	**18.2×10^{9}/l**	
eosinophiles	3 %	
basophiles	0 %	
neutrophiles:		
– myelocytes	0 %	
– young	1 %	
– band	5 %	
– segmentated	10 %	
lymphocytes («lymphomonocytes»)	67 %	
monocytes	13 %	
PLT (thrombocytes)	**350.0×10^{9}/l**	
Single lymphoblast in the field of vision. Plasmatic cells — 4 per 100 WBC (leukocytes). Toxic granularity of neutrophiles.		
Conclusion:		

№ 21

RBC (erythrocytes)	**2.4×10^{12}/l**	
Hemoglobin	75 g/l	
Color index	calculate	
WBC (leukocytes)	**3.2×10^{9}/l**	
basophiles	0 %	
eosinophiles	0 %	
myeloblasts	30 %	
promyelocytes	1 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	4 %	
– segmentated	30 %	
lymphocytes	30 %	
monocytes	5 %	
PLT (thrombocytes)	**75.0×10^{9}/l**	
ESR	55 mm per h	
Conclusion:		

№ 22

RBC (erythrocytes)	**2.5×10^{12}/l**	
Hemoglobin	78 g/l	
Color index	calculate	
WBC (leukocytes)	**200.0×10^{9}/l**	
myeloblasts	97 %	
promyelocytes	0.5 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	0 %	
– segmentated	2.5 %	
lymphocytes	0 %	
monocytes	0 %	
PLT (thrombocytes)	**48.0×10^{9}/l**	
ESR	60 mm per h	
Conclusion:		

№ 23

RBC (erythrocytes)	**1.1×10^{12}/l**	
Hemoglobin	37 g/l	
Color index	calculate	
WBC (leukocytes)	**8.4×10^{9}/l**	
Basophiles	0 %	
eosinophiles	0 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	2 %	
– segmentated	10 %	
lymphoblasts	62 %	
lymphocytes	20 %	
monocytes	6 %	
PLT (thrombocytes)	**28.0×10^{9}/l**	
ESR	52 mm per h	
Conclusion:		

№ 24

RBC (erythrocytes)	**2.0×10^{12}/l**	
Hemoglobin	64 g/l	
Color index	calculate	
WBC (leukocytes)	**8.4×10^{9}/l**	
basophiles	0 %	
eosinophiles	0 %	
neutrophiles:		
– segmentated	4.5 %	
– lymphocytes	4 %	
– monocytes	1 %	
– blast cells	90.5 %	
PLT (thrombocytes)	**32.0×10^{9}/l**	
Peroxidase reaction is positive		
Conclusion:		

№ 25

RBC (erythrocytes)	**2.3×10^{12}/l**	
Hemoglobin	58 g/l	
Color index	calculate	
WBC (leukocytes)	**2.7×10^{9}/l**	
basophiles	0.5 %	
eosinophiles	0 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	1.5 %	
– segmentated	8.5 %	
lymphocytes	7.0 %	
monocytes	4.5 %	
blast cells (cytochemical reactions are negative)	78 %	
PLT (thrombocytes)	**93×10^{9}/l**	
Conclusion:		

№ 26

RBC (erythrocytes)	**3.5×10^{12}/l**	
Hemoglobin	110 g/l	
Color index	calculate	
WBC (leukocytes)	**150.0×10^{9}/l**	
basophiles	6 %	
eosinophiles	7.5 %	
myeloblasts	1 %	
promyelocytes	2 %	
neutrophiles:		
– myelocytes	25 %	
– young	22.5 %	
– band	18 %	
– segmentated	14 %	
lymphocytes	3 %	
monocytes	1 %	
PLT (thrombocytes)	**522.0×10^{9}/l**	
ESR	35 mm per h	
Conclusion:		

№ 27

RBC (erythrocytes)	**3.2×10^{12}/l**	
Hemoglobin	87 g/l	
Color index	calculate	
WBC (leukocytes)	**38.0×10^{9}/l**	
basophiles	8 %	
eosinophiles	3 %	
myeloblasts	1 %	
promyelocytes	1 %	
neutrophiles:		
– myelocytes	5 %	
– young	4.5 %	
– band	5.5 %	
– segmentated	45 %	
lymphocytes	24 %	
monocytes	3 %	
PLT (thrombocytes)	**380.0×10^{9}/l**	
ESR	35 mm per h	
Conclusion:		

№ 28

RBC (erythrocytes)	**2.8×10^{12}/l**	
Hemoglobin	68 g/l	
Color index	calculate	
WBC (leukocytes)	**300.0×10^{9}/l**	
basophiles	0 %	
eosinophiles	1 %	
neutrophiles:		
– myelocytes	0 %	
– young	0 %	
– band	1 %	
– segmentated	2 %	
Lymphoblasts	1 %	
lymphocytes	94 %	
monocytes	1 %	
PLT (thrombocytes)	**87.0×10^{9}/l**	
ESR	40 mm per h	
In the smear: in a plenty of a cell (shadow) of Botkin–Gumprecht.		
Conclusion:		

№ 29

RBC (erythrocytes)	**$2{,}1 \times 10^{12}$ /l**	
Hemoglobin	61,1 g/l	
HCT	16 %	
Color index	calculate	
WBC (leukocytes)	**$176{,}5 \times 10^{9}$ /l**	
basophiles	10 %	
eosinophiles	3 %	
Myeloblast	10 %	
Pro	12 %	
neutrophiles:		
– мyelo	16 %	
– мeta	17 %	
– band	9 %	
– segmentated	19 %	
lymphocytes	3 %	
monocytes	1 %	
PLT (thrombocytes)	**$93{,}6 \times 10^{9}$ /l**	
ESR	50 mm per h	
Cytogenetic characteristic of blood cells: 95,5 % of cells content Ph' t(9; 22) (q34; q11) chromosome.		
Conclusion:		

BLOOD PARAMETERS IN NORM

Parameter name	SI system	Non-systemic units
RBC (erythrocytes) (RBC) in women in men	 $3.9–4.7\times10^{12}$/l $4.0–5.0\times10^{12}$/l	 3.9–4.7 million/1 micrl 4.0–5.0 million/1 micrl
Hemoglobin (HGB) in women in men	 120.0–140.0 g/l 130.0–1600 g/l	 12.0–14.0 g % 13.0–16.0 g %
Hematocrit (HCT) in women in men	 0.36–0.42 0.40–0.48	 36–42 % 40–48 %
Mean erythrocyte volume (mean corpuscular volume — **MCV**) MCV = HCT: RBC	80–100 phl (10^{-15} l)	80–100 mcm^3
Mean hemoglobin content in an erythrocyte (mean corpuscular hemoglobin — **MCH**) MCH = HGB: RBC	$25.4–34.6\times10^{-15}$ kg/cell	**25.4–34.6 pg/cell***
Mean hemoglobin concentration in an erythrocyte (mean corpuscular hemoglobin concentration — **MCHC**) MCHC = HGB: HCT	0.30–0.38 kg/l	**30–38 g/dl *** 30–38 %
Distribution width of RBC (erythrocytes) by the volume (red cell distribution width — **RDW**) — anisocytosisindex	11.5–14.5 %	1.5–14.5 %
Color index (**CI**)	0.8–1.0	0.8–1.0
Reticulocytes	0.2–1.0 %	2.0–10.0 %
ESR in women in men	 1–15 mm/hour 1–10 mm/hour	 1–15 mm/hour 1–10 mm/hour

***** The most common dimension of the parameter

Calculation of erythrocyte indices:

Mean erythrocyte volume (MCV) is calculated by division of the hematocrit value of $1mm^3$ of blood by the erythrocyte count in $1mm^3$ by the formula:

$$\mathbf{MCV} = \frac{\textbf{Hematocrit in 1 mm}^3}{\textbf{Erythrocyte count in 1 mm}^3};$$

Normal MCV value — 80–100;
MCV < 79 — microcytosis;
MCV > 100 — macrocytosis;

In practice the mean erythrocyte volume is calculated by multiplication of the hematocrit (%) by 10 and divisions of the received product by the erythrocyte count per 1 L of blood:

$$\textbf{MCV} = \frac{\textbf{Hematocrit (\%)} \times \textbf{1}}{\textbf{Erythrocyte count in 1 } \textbf{\textit{l}} \times \textbf{10}^{\textbf{-12}}}.$$

The Mean content of hemoglobin in an erythrocyte (MCH) is established by the formula:

$$\textbf{MCV} = \frac{\textbf{Hematocrit (g/l)}}{\textbf{Erythrocyte count in 1 } \textbf{\textit{l}} \times \textbf{10}^{\textbf{-12}}}.$$

Mean concentration of hemoglobin in an erythrocyte (MCHC):

$$\textbf{MCHC} = \frac{\textbf{Hemoglobin (g/100 } \textbf{\textit{ml}}\textbf{)} \times \textbf{100}}{\textbf{Hematocrit (\%)}}.$$

BLOOD PARAMETERS IN NORM (continuation)

WBC (leukocytes)	**4.0–9.0×10^9/l**	**4.0–9.0 thousand in 1 micl**
Neutrophiles: band Segmentated	 1–6 % 0.040–0.300 × 10^9/l 47–72 % 2.000–5.500 × 10^9/l	 1–6 % 40–300 in 1 mcl 47–72 % 2000–5500 in 1 мcl
Eosinophiles	1.0–5 % 0.020–0.300 × 10^9/l	1.0–5 % 20–300 in 1 мcl
Basophiles	0–1 % 0–0.065 × 10^9/l	0–1 % 0–65 in 1 мcl
Lymphocytes	19–37 % 1.200–3.000 × 10^9/l	19–37 % 1200–3000 in 1 мcl
Monocytes	3–11 % 0.09–0.6 × 10^9/l	3–11 % 90–600
PLT (thrombocytes)	150.0–450.0 × 10^9/l	150–450.0 thousand in 1 mcl

MORPHOLOGICAL CHARACTERISTIC OF BASIC TYPES OF ANEMIA WITH THE ACCOUNT OF ERYTHROCYTE INDICES

Anemia type	CI	Erythrocyte-diameter (micron)	MCV phl	MCH pg	RDW (%)	Characteristic
Acute post-hemorrhagic	0.8–1.05	7.2–7.5	80–90	27–33	norm	normochromous, normocytic
Fe-deficient	< 0.8	< 6.5	<79	<27	> 14,5	hypochromous, microcytic
B_{12}-deficient	> 1.1	> 8	> 100	> 34	> 14,5	hyperchromous, macrocytic
Hemolytic	0.8–1.05	< 6.5 or norm	< 79 or norm	> 34 or norm	> 14,5	normochromous, normocytic or hyperchromous, microspherocytic
Aplastic	0.8–1.05	7.2–7.5	80–90	27–33	norm	normochromous, normocytic

SEVERITY CHARACTERISTIC OF ANEMIA (according to E. D. Goldberg)

Severity degree	Hemoglobin (g/l)	RBC (erythrocytes) × 10^{12}/l
Light	> 100	> 3
Moderate	100–66	3–2
Severe	< 66	< 2

CYTOCHEMICAL CHARACTERISTIC OF VARIOUS FORMS OF LEUKEMIA

FAB	Acute leukemia form	Reaction to nutrients			Reactions to enzymes			
		Glycogen schiff (PAS)-reaction)	*GAG	Lipids (black sudan)	Perox-idase	Acid phospha-tase	α-naphtyl-esterase	Chlor-acetate-esterase
M0	Non-differentiated	–	–	–	–	–	–	–
M1 M2	Myeloblastic	+	–	+	+	+	slightly+	+
M3	Promyelocytic	sharply+	+	+	sharply+	slightly+	slightly+	sharply+
M4	Myelomonoblastic	+(diffuse)	–	–	highly+	+	+	slightly+
M5	Monoblastic	slightly+	–	slightly+	slightly+	highly+	+	-
M6	Erythromyeloblastic	+	–	Reactions depend on blast elements belonging to this or that row (myeloblasts, monoblasts, non-differentiated blasts)				
M7	Megakarioblastic	Is defined by characteristic morphology of cells						
	Lymphoblastic	+(as lumps)	–	–	–	sometimes+	–	–
	Plasmoblastic	Is defined by characteristic morphology of cells & the presence of paraprotein in the blood serum						

* GAG — glycosaminoglycans.

LESSON 8. INSUFFICIENCY OF BLOOD CIRCULATION. ACUTE CARDIAC INSUFFICIENCY. CORONARY INSUFFICIENCY

Date: «____» _____ 20___

The purpose of the Lesson:

– To discuss basic types of blood circulation insufficiency, to study the causes, forms and developmental mechanisms of acute blood circulation insufficiency of cardiac genesis.

Tasks:

– To study the causes, developmental mechanisms and manifestations of acute right ventricular insufficiency in experiment on the basis of teaching video materials.

– To get acquainted with modeling experimental myocardial necrosis, to analyze some formation mechanisms of electrocardiogram impairments in the given pathology.

– Solving situational tasks (see the collection of situational tasks on pathological physiology).

Work 1. STUDYING MATERIALS OF THE TEACHING VIDEO «ACUTE INSUFFICIENCY OF BLOOD CIRCULATION OF A RIGHT VENTRICULAR TYPE » (A. A. Krivchik et al., MSMI, 1978)

Analyze the presented material and answer the following **questions:**

1. What is the essence of the methodical approach used for modeling acute insufficiency of blood circulation?

2. What technique provided the possibility to register the values of arterial, venous and portal pressure, the degree of oxygen saturation of the blood, etc. under the conditions of experiment on an unnarcotized animal without serious consequences?

3. Underline with blue color the changes that show the development of pathological reactions in response to acute impairments of blood circulation in the posterior vena cava:

1) sharp decrease of BP, the collaptoid state with loss of consciousness;
2) pressure increase in veins under the occlusion site;
3) pressure increase in the portal venous system;

4) increase of arterial-venous difference by oxygen;
5) expressed hypoxia of the brain;
6) hypoxia of the respiratory and vascular-motor centers;
7) tachycardia;
8) breathlessness;
9) hypoxia of the myocardium;
10) decrease of the blood velocity;
11) intermittent type of respiration.

Which of them reflects changes of compensatory-adaptive character (underline with red)?

4. Why should the named changes be regarded as compensatory-adaptive? The achievement of what are they directed to? In what cases does tachycardia not improve, but aggravates the situation and why?

5. The reactions of what type (pathological or compensatory-adaptive) prevailed in the modeled form of acute insufficiency of blood circulation?

6. Could the organism independently, without being rendered medical aid, overcome such condition?

Work 2. ACQUAINTANCE WITH MODELING EXPERIMENTAL MYOCARDIAL NECROSIS. THE ANALYSIS OF SOME FORMATION MECHANISMS OF ELECTROCARDIOGRAM IMPAIRMENTS IN THE DEVELOPMENT OF MYOCARDIAL NECROSIS

An immobilized frog is fixed to a wooden plate in a supine position on the back. Needle electrodes from the electrocardiograph are stuck into both fore extremities and a left hind one. Expose the heart (opening the pericardium). Record the initial electrocardiogram in I and III standard outlets. A crystal of silver nitrate is applied on the frontal surface (the left half) of the ventricle causing myocardial necrosis. Register again the electrocardiogram and observe the elevation of ST segment (the so-called «coronary wave»). Register electro-cardiogram changes, mark the ST segment with a red pencil:

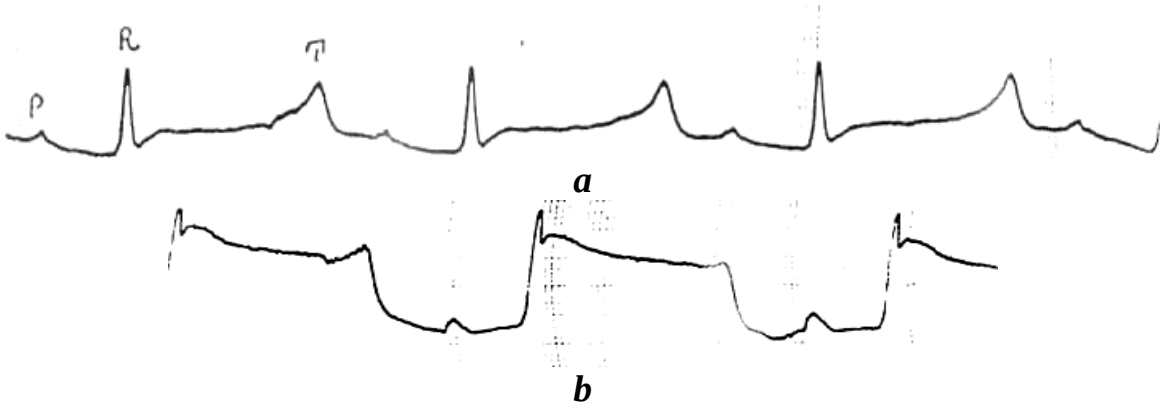


Fig. 1. Electrocardiogram changes in experimental myocardial necrosis of the frog, induced by the silver nitrate crystal action:
a — an electrocardiogram of the frog in norm; *b* — an electrocardiogram after application of a silver nitrate crystal to the myocardium surface

To explain the elevation mechanism of the ST interval in necrosis, perform the comparison of ECG changes in the following experiments. The second immobilized frog is fixed, its heart is exposed (opening the pericardium), electrodes from the cardiograph are stuck into corresponding extremities. An electrocardiogram is recorded in the same outlets. Further on the frontal surface of the heart is applied:

1. A slice of necrotized cardiac muscle of the first frog. At subsequent registration of the electrocardiogram the elevation of the ST interval is marked, then the heart is washed with Ringer's solution for the cold-blooded and normalization of ECG is noted.

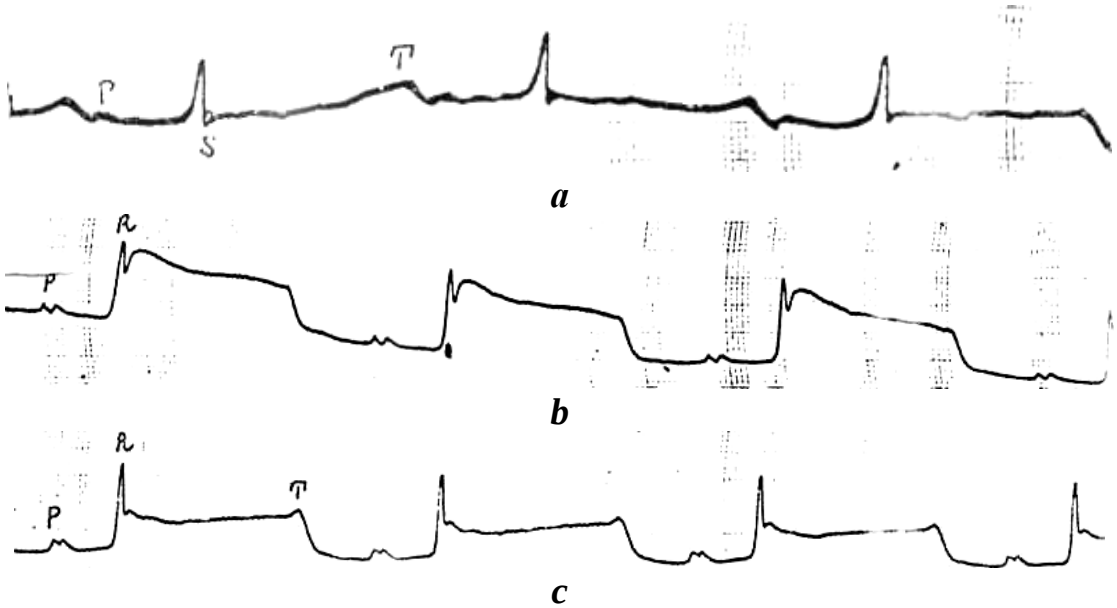


Fig. 2. Changing of the frog's ECG under the effect of local application of a necrotized slice of the cardiac muscle with subsequent washing up of the heart by Ringer's solution:
a — the electrocardiogram in norm; *b* — the electrocardiogram after application of a necrotized slice of the cardiac muscle; *c* — the electrocardiogram after washing up of the heart by Ringer's solution

2. A cotton wool, moistened with 1 % solution of potassium chloride. Record the electrocardiogram, also mark the elevation of segment ST, which disappears in repeated washing up of the heart by solution Ringer's for the cold-blooded.

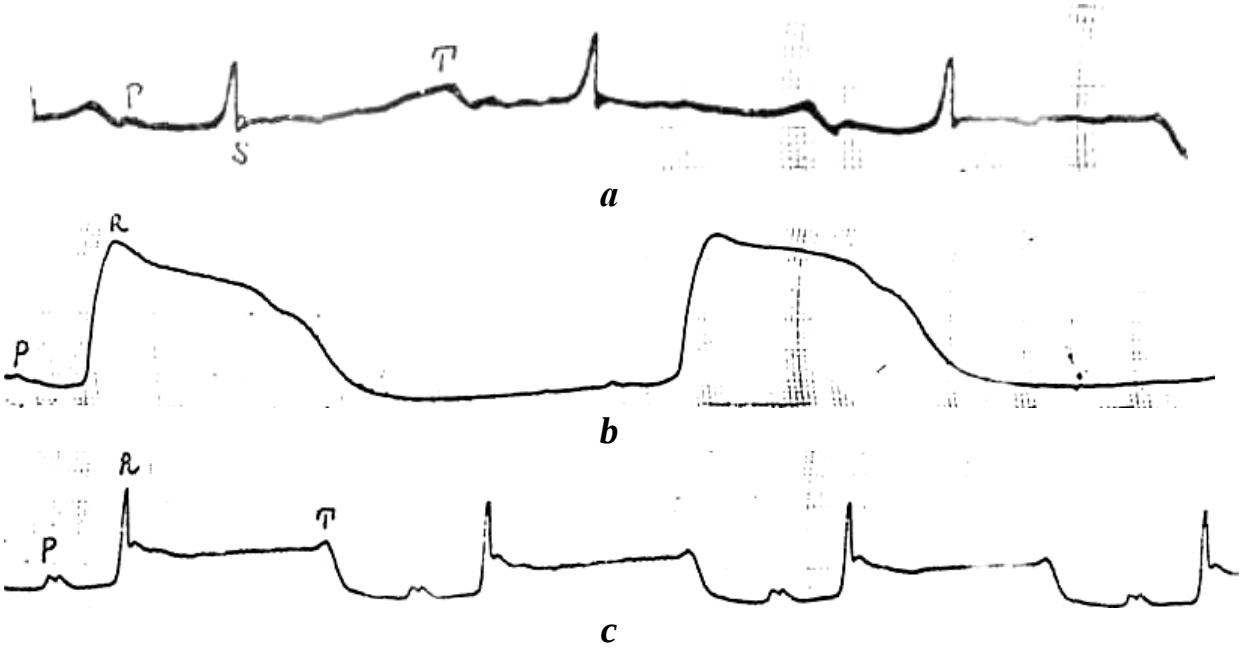


Fig. 3. Changing of the frog's electrocardiogram under the effect of local application of a cotton wool, moistened with 1 % solution of KCl with subsequent washing up of the heart with Ringer's solution:
a — the electrocardiogram in norm; *b* — the electrocardiogram after application of KCl; *c* — the electrocardiogram after washing up of the heart by Ringer's solution

Draw a conclusion on a possible formation mechanism of the ST segment elevation in myocardial necrosis:

Present as a scheme and describe in short electrocardiogram changes characteristic of:

a) ischemia

b) ischemic damage

c) myocardial necrosis

Control questions:

1. Blood circulation insufficiency. The definition of the notion, its types.
2. Cardiac insufficiency. The definition of the notion. Principal incidence causes of cardiac insufficiency. Classification of cardiac insufficiency by pathogenesis, localization, course, severity degree. The notion of primary and secondary cardiac insufficiency.
3. Hemodynamic classification of cardiac insufficiency. The notion of systolic and diastolic dysfunctions. Etiology, pathogenesis, hemodynamics impairments and clinical manifestations of systolic and diastolic dysfunctions.

4. Basic parameters of changes in intracardiac and systemic hemodynamics in all forms of cardiac insufficiency.

5. Etiology, pathogenesis and manifestations of acute left- and right-side ventricular cardiac insufficiency.

6. Coronary insufficiency. The definition of the notion, clinical forms of IHD (ischemic heart diseases). Relative and absolute coronary insufficiency.

7. Etiological risk factors of IHD. Experimental methods of its inducing. Principal causes of non-coronary necrosis of the myocardium.

8. Pathogenesis of ischemic and reperfusion syndromes in coronary insufficiency, their manifestations.

9. Myocardial infarction. Pathogenesis and manifestations of basic clinical-laboratory syndromes: pain, acute left-ventricular insufficiency (cardiac asthma, cardiogenic shock), resorption-necrotic syndrome. The impairments of metabolism, bioelectric and contractive properties of the myocardium.

The teacher's signature:

LESSON 9. CHRONIC BLOOD CIRCULATION INSUFFICIENCY OF CARDIAC GENESIS

Date: «____» _____ 20___

The purpose of the Lesson:

– To study the forms and development mechanisms of chronic blood circulation insufficiency of cardiac genesis, to give pathogenetic characteristic of urgent and long-term compensatory reactions in the given form of blood circulation insufficiency.

Tasks:

– To study the causes, forms and development mechanisms of chronic cardio-genetic blood circulation insufficiency on the basis of the teaching scientific video «Chronic blood circulation insufficiency of the right-ventricular type»; to analyze the materials of the video and to answer the questions; to formulate conclusions using the analysis of experimental results.

– To study the dynamics of changing the pulse rhythm (PR) and heart rate (HR) in the development of chronic blood circulation insufficiency of the right-ventricular type.

– Solving situational tasks (see the collection of situational tasks on pathological physiology).

Work 1. STUDYING THE MATERIALS OF THE TEACHING-SCIENTIFIC VIDEO «CHRONIC BLOOD CIRCULATION INSUFFICIENCY OF THE RIGHT-VENTRICULAR TYPE» (A. A. Krivchik et al., 1979)

While viewing the video pay attention to the following information:

– Peculiarities and advantages of the used technique of modeling chronic blood circulation insufficiency (CBCI) of the right-ventricular type (RVT);

– The approach providing the opportunity to monitor the expressiveness degree of the compensation phenomena at various stages of CBCI RVT;

– The character and dynamics of changing arterial, venous and portal pressure, blood flow velocity, arterial-venous difference by O_2, contractive abilities of the myocardium, ECG and EEG findings in the process of CBCI development;

– The effect of increasing CBCI RVT on the condition of vessels, blood filling, the structure and functional status of the liver;

– Symptoms that occurred in a number of organs and systems showing predominantly the events of «breakage», damage;

– Reactions of compensatory-adaptive character;

– Manifestations of decompensation;

– The role of training compensatory mechanisms in the achievement of adaptive effect in CBCI development.

Answer the questions:

1. What are the peculiarities of the used technique of modeling chronic blood circulation insufficiency (CBCI) of the right-ventricular type (RVT)? What are its advantages as compared to applying a constricting ligature to a vessel that is usually used for these purposes?

2. Draw a scheme showing the dynamics of blood pressure changing in the posterior vena cava (PVC) (a), portal (b) veins and in the aorta (в) in CBCI RVT process.

3. Underline with **dark blue color** the signs that show mainly the phenomena of «breakage», damage, i. e. proper pathological reactions of the organism in the process of CBCI RVT:

1) substantial increase of pressure in the posterior (inferior) vena cava;
2) progressive elevation of pressure in the portal venous system;
3) increasing of the blood flow velocity deceleration;
4) moderate tachycardia;
5) saturation decrease of blood oxygenation and increase of ΔA-V O_2;
6) increasing signs of cerebral and cardiac hypoxia;
7) decrease pumping function of the heart;
8) deepening and acceleration of respiration;
9) ↓ of the number of functioning hepatic vessels due to their obliteration;
10) development of collateral blood circulation (caput medusae);
11) congestion events in the liver with atrophy of parenchyma and fibrosis;
12) development of hepatic-cellular insufficiency;
13) edemas of extremities, ascites, hydrothorax.

Which of the shown in the video changes in CBCI RVT should be considered as primary manifestations of compensatory reactions (mark with **red color**), see above?

4. Mark with **red color** the signs that suggest the conclusion about gradual increase and about the achieved expressiveness of compensatory reactions?

1) sharp edematization of tissues;

2) expressed breathlessness at rest;

3) relative stabilization of hemodynamics and hepatic function;

4) relative normalization of general status of the animal in compressing its posterior vena cava (PVC);

5) ↑ of the PVC compressing period safe for life (till 2 hours);

6) repeated sharp increasing of the blood flow deceleration.

Which of the shown in the video shifts of registered parameters should be regarded as decompensation manifestations (mark with **dark blue color**), see above?

Work 2. STUDYING THE DYNAMICS OF PULSE RHYTHM (PR) CHANGES AND HEART RATE (HR) IN THE DEVELOPMENT OF CHRONIC BLOOD CIRCULATION INSUFFICIENCY OF THE RIGHT-VENTRICULAR TYPE

The video demonstrates modeling of chronic blood circulation insufficiency of the right-ventricular type. To form the notion of the character of vegetative extracardiac effects on the heart in the development of chronic blood circulation insufficiency of the right-ventricular type an electrocardiogram was registered (with complexes by 50 cardiocycles), the duration of R-R intervals was taken as well as the average HR and the width of distribution of R-R intervals, reflecting the pulse rhythm (PR) with the precision of 0.01.

The results are presented in the figure.

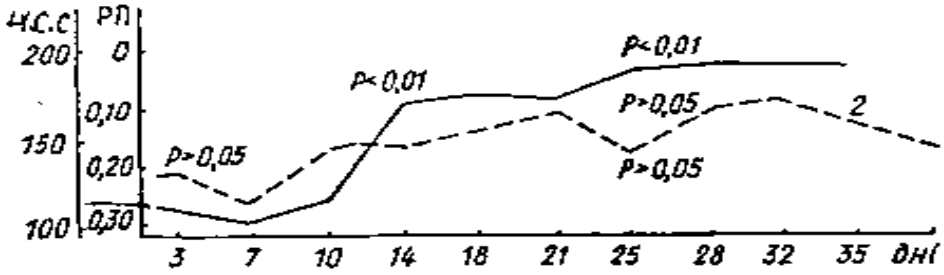


Fig. 1. RR interval duration, the average HR and pulse rhythm factor (PR) in CBCI

Answer the questions:

1. Strengthening of what vegetative effects on the heart does the given graph show?

2. What is the pathogenetic characteristic of such effects on the sinoatrial node of the heart in the development of CBCI of cardiac genesis?

Control questions:

1. Classification of chronic blood circulation insufficiency of cardiac genesis by the severity degree (Vasilenko–Strazhenko).
2. Compensation mechanisms of cardiac insufficiency. Their types, manifestations and pathogenetic characteristic.
3. Comparative characteristic of heterometric and homeometric
4. mechanisms of intracardiac compensation in cardiac overstrain.
5. The notion of remodeling of the myocardium. Outcomes of myocardial remodeling depending on the type of hemodynamic overstrain and in the damage of the myocardium.
6. Etiology, pathogenesis, mechanisms of urgent and long-term intracardiac compensation in chronic overstrain of the myocardium by volume and pressure, outcomes, character of hemodynamics impairment, clinical manifestations.
7. Pathogenesis and clinical manifestations of syndromes of small output and congestion on the ways of inflow to the weakened department of the heart. Manifestations of congestion in pulmonary and general blood circulation.
8. Extracardial compensatory mechanisms of cardiac insufficiency, their pathogenetic characteristic. The role of vegetative nervous system in compensating chronic cardiac insufficiency. The notion of hormonal-neuromediator dissociation. Its pathogenetic characteristic.
9. Main effects of hyperactivation of sympato-adrenaline and renin-angiotensin-aldosterone system in chronic cardiac insufficiency. Mechanisms of cardiotoxic action of cachetolamines. Pathogenic characteristic of tachycardia in cardiac overstrain.
10. Reactions of the respiration and hemopoietic system in the development of cardiac insufficiency, trigger mechanisms of these systems.
11. Etiology, pathogenesis and manifestations of chronic left- and right-ventricular cardiac insufficiency.
12. Characteristic of the cardiac compensatory hyperfunction (CCH) in acute experimental overstrain of the left ventricle with resistance (according to F.Z.Meerson). Developmental stages of the cardiac compensatory hyperfunction.
13. Hypertrophy of the myocardium, causes and mechanisms of its development. Functional and metabolic peculiarities of a hypertrophied myocardium. Developmental mechanisms of decompensation in pathological hypertrophy of the myocardium.
14. Pathogenetic therapeutic principles of cardiac insufficiency.

The teacher's signature:

LESSON 10. ARRHYTHMIAS. IMPAIRMENTS OF EXCITABILITY, AUTOMATISM AND CONDUCTION OF THE HEART

Date: «____» _____ 20___

The purpose of the Lesson:

– To study cardiac rhythm impairments: impairments of excitability, automatism and conduction of the heart, their types, causes, and development mechanisms, electrocardiographic and hemodynamic manifestations.

Tasks:

– To study electrocardiographic manifestations of cardiac rhythm changes in irritation of the frog's stomach.

– To study electrocardiographic manifestations of cardiac rhythm changes of the rabbit in intravenous injection of the solution of barium chloride and inhalation of NH_4OH.

– To study the sequence of electrocardiographic impairments while conducting a stimulus by the conduction system of the rat's heart in the development of hypothermia.

– To get acquainted with typical impairments of automatism, excitability and conduction of the cardiac muscle in experimental animals and humans on the basis of a set of electrocardiograms.

Work 1. ELECTROCARDIOGRAPHIC MANIFESTATIONS OF CARDIAC RHYTHM CHANGES IN IRRITATION OF THE FROG'S STOMACH (GASTROCARDIAC REFLEX)

An immobilized frog is fixed with pins to a wooden plate with its stomach upward. The heart is exposed by cutting the breastbone and soft tissues. The electrocardiograph electrodes are stuck into both fore and a left hind extremities. The initial electrocardiogram in the II standard outlet is recorded. The abdominal cavity is opened and the stomach is taken out. The stomach is irritated by induction current and ECG is recorded again.

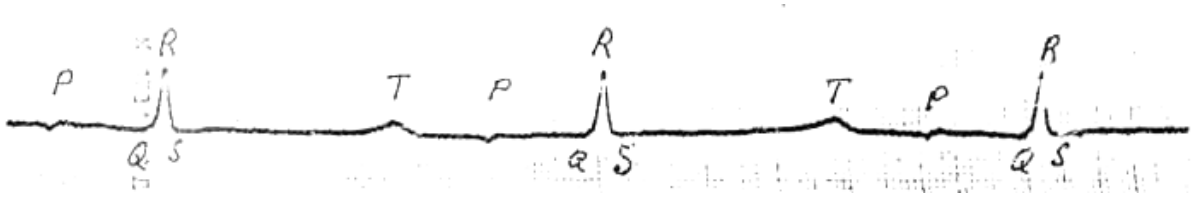


Fig. 1. Electrocardiograms of the frog in norm.
R-R = 1,2". HR = 60 c. RR =

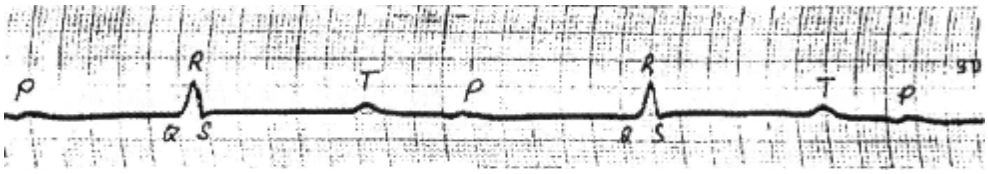


Fig. 2. Electrocardiograms of the frog after irritation of the stomach by induction current
R-R " = 1,5". HR =

Answer the questions:

1. What ECG changes were observed in experiment?

2. What type of rhythm impairments are they referred to?

3. What is the mechanism of these impairments?

Work 2. ELECTROCARDIOGRAPHIC MANIFESTATIONS OF CARDIAC RHYTHM IMPAIRMENTS INDUCED BY INTRAVENOUS INJECTION OF BARIUM CHLORIDE AND AT INHALATION OF NH_4OH

For the experiment an adult rabbit is taken and fixed in a special arrangement. Then needle electrodes of the electrocardiograph are stuck into both fore and a left hind extremities of the animal. The initial electrocardiogram in the first standard outlet is recorded, then 1 ml of 1 % solution of barium chloride is injected into a marginal vein of the rabbit's ear and in 20–30 sec the ECG is repeatedly recorded. ECG changes are registered and analyzed. After normalization of the electrocardiogram, a cotton wool moistened with NH_4OH is brought to the rabbit's nose. An electrocardiogram is recorded again and the rhythm impairment is marked.

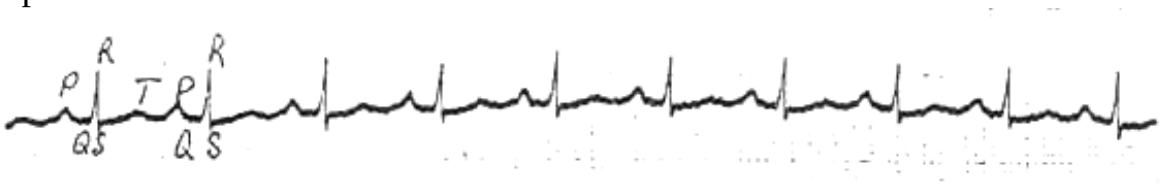


Fig. 3. Electrocardiograms of the rabbit in norm

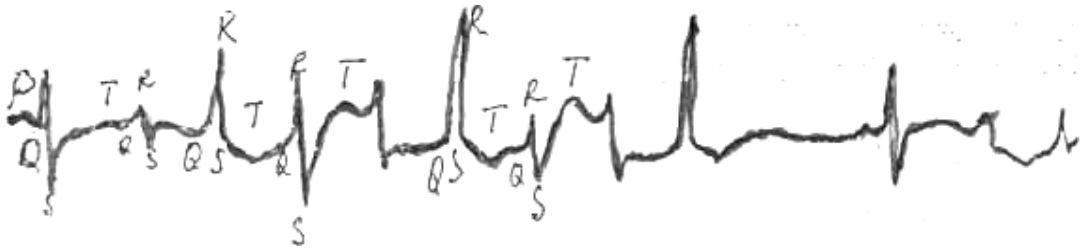


Fig. 4. Electrocardiograms of the rabbit immediately after injections of barium chloride

Specify the type of impairment of the cardiac rhythm:

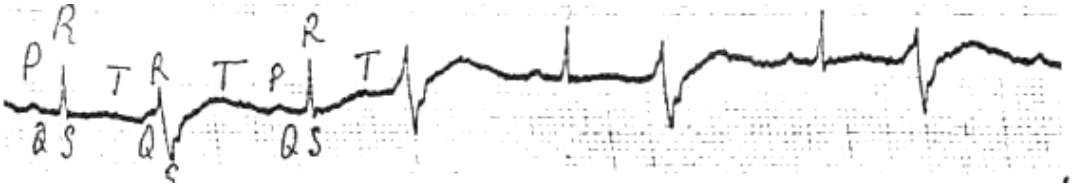


Fig. 5. Electrocardiograms of the rabbit at 1 minute after injection of barium chloride

Specify the type of impairment of the cardiac rhythm:

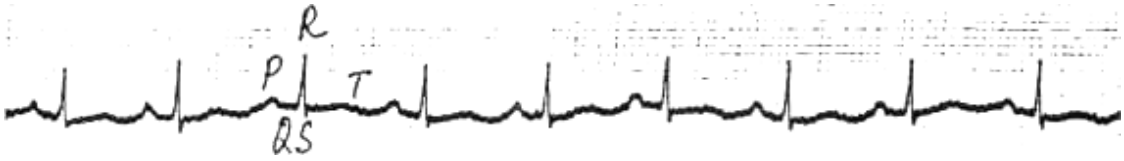


Fig. 6. Electrocardiograms of the rabbit at 15 minutes after injection of barium chloride

Specify the type of impairment of the cardiac rhythm:

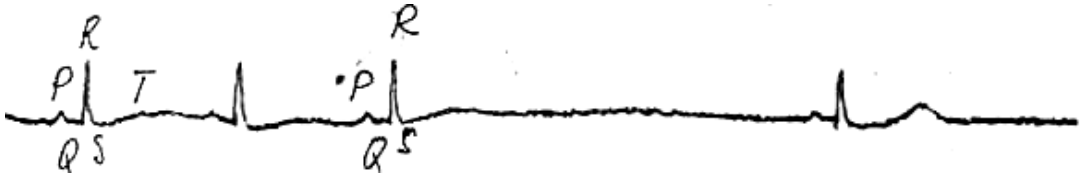


Fig. 7. Electrocardiograms of the rabbit immediately after inhalation of NH_4OH

Specify the type of impairment of the cardiac rhythm:

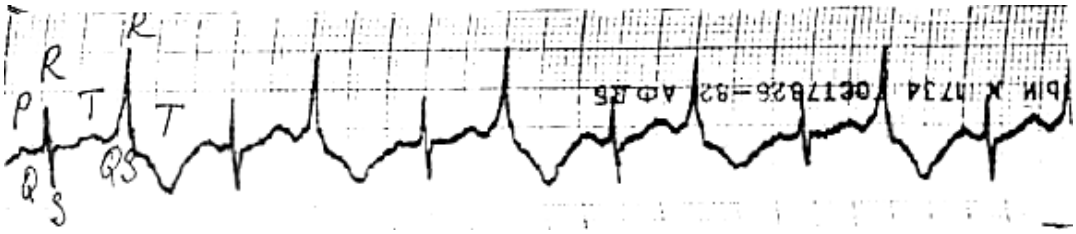
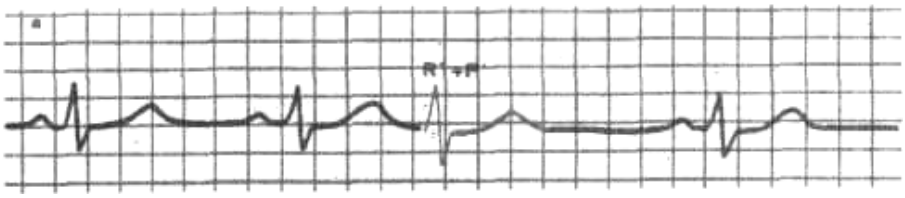


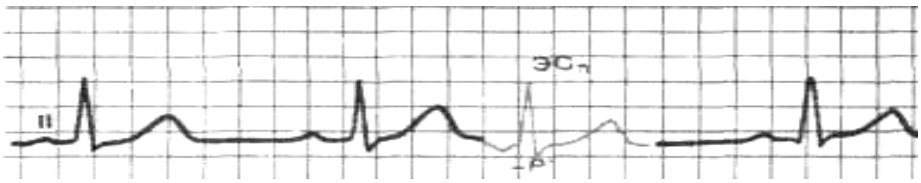
Fig. 8. Electrocardiograms of the rabbit at 1 minute after inhalation of NH_4OH

Work 3. SPECIFY THE TYPE OF IMPAIRMENT OF THE CARDIAC RHYTHM:

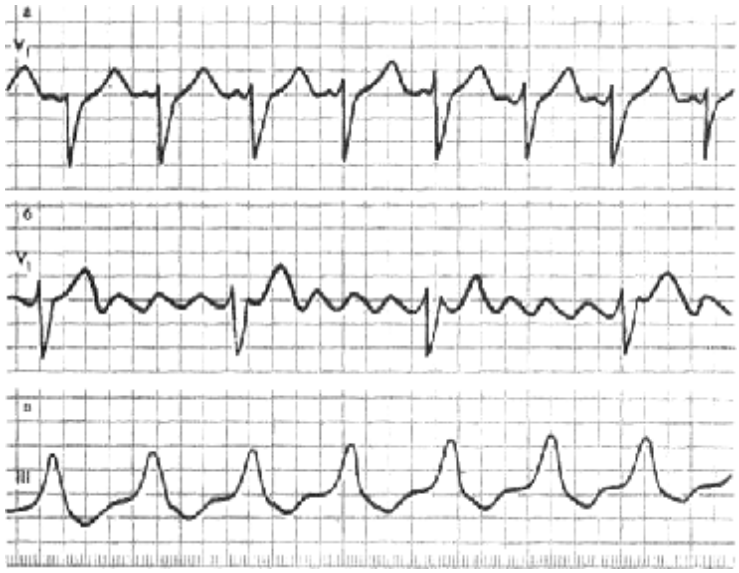
№ 1



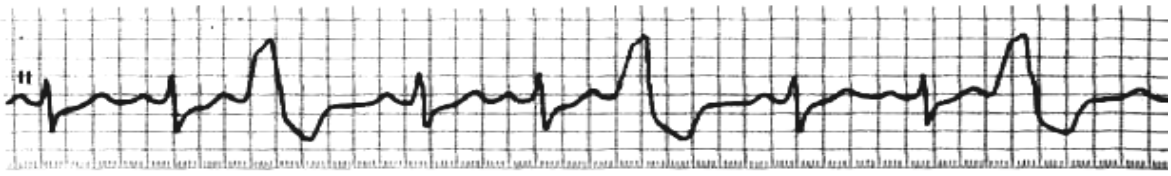
№ 2



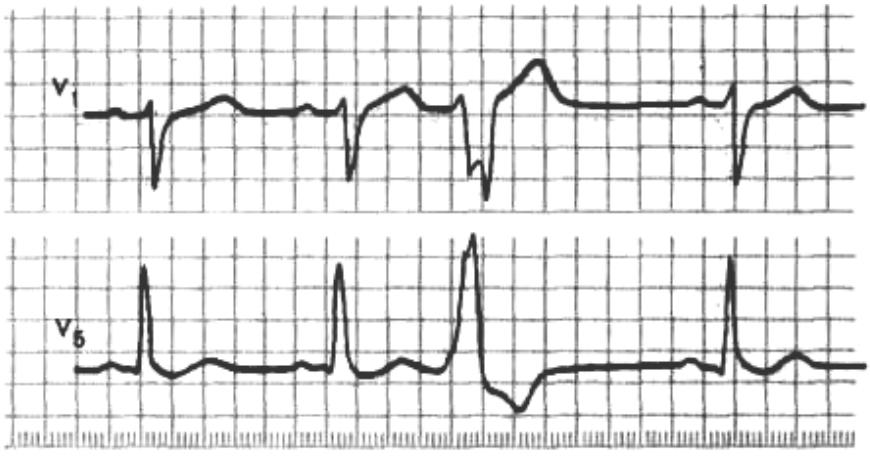
№ 3



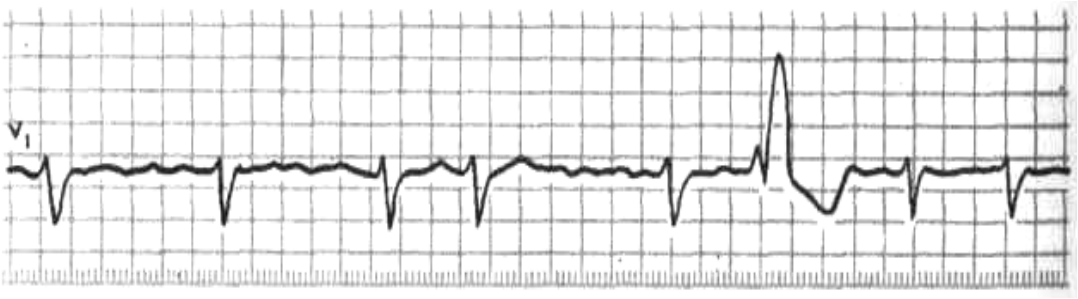
№ 4



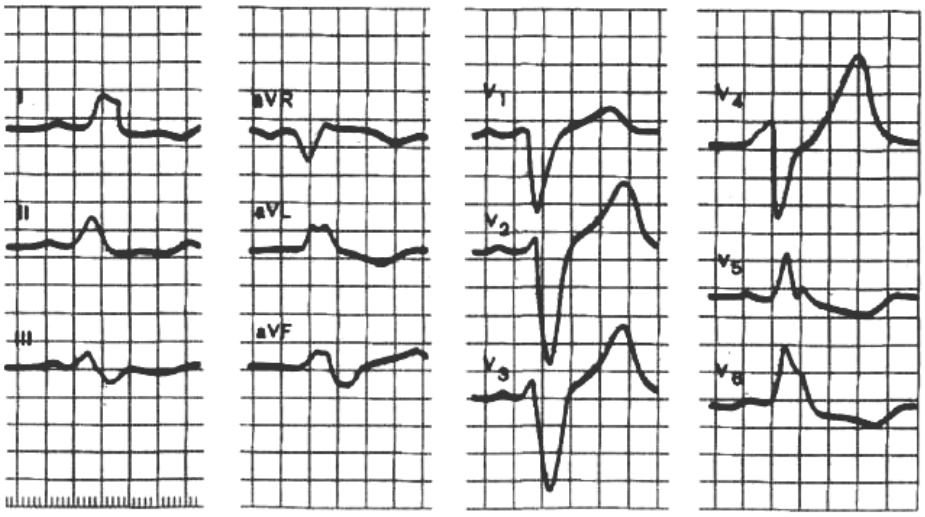
№ 5



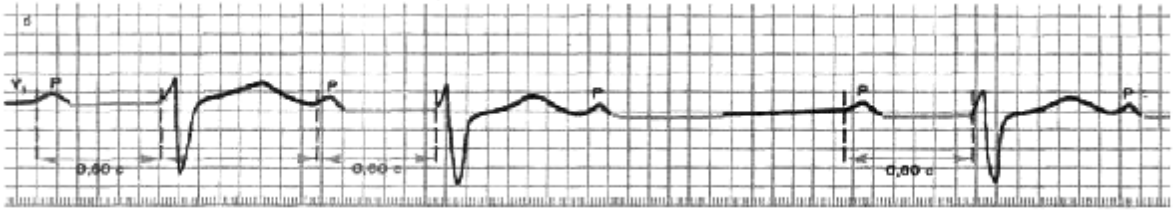
№ 6


V1

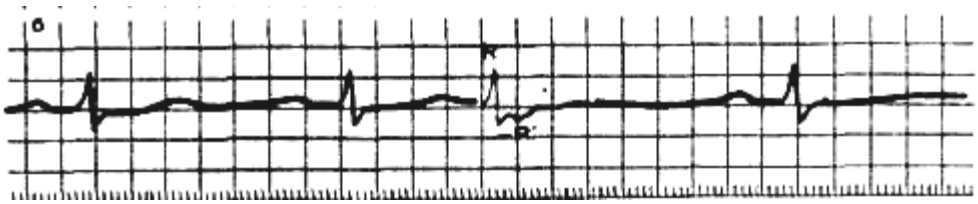
№ 7


I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

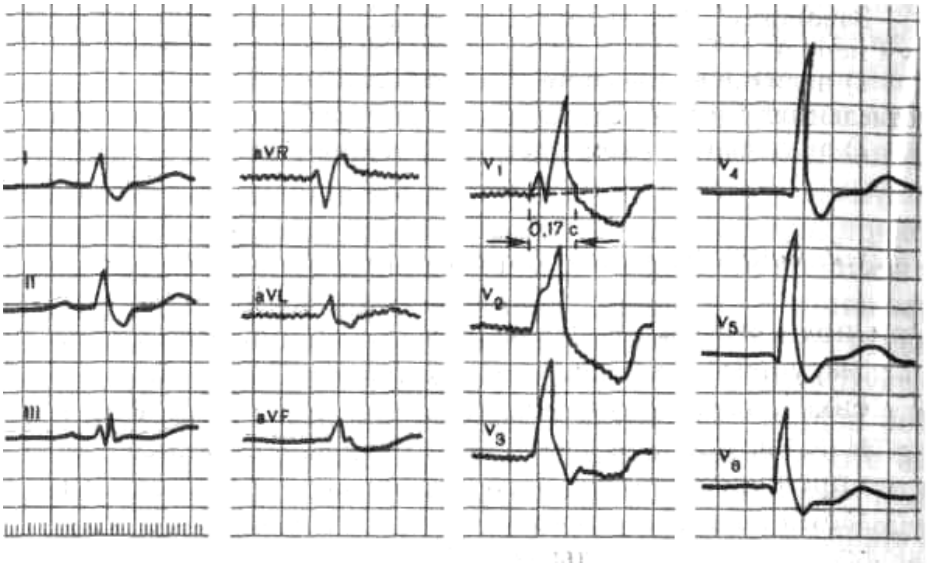
№ 8


V1
P
P
P
P
P
0,60 с
0,60 с
0,60 с

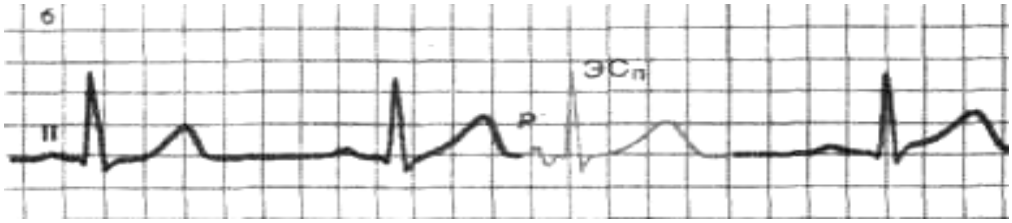
№ 9


R
S

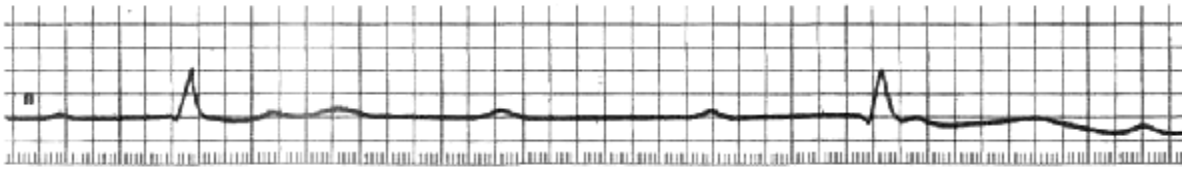
№ 10



№ 11



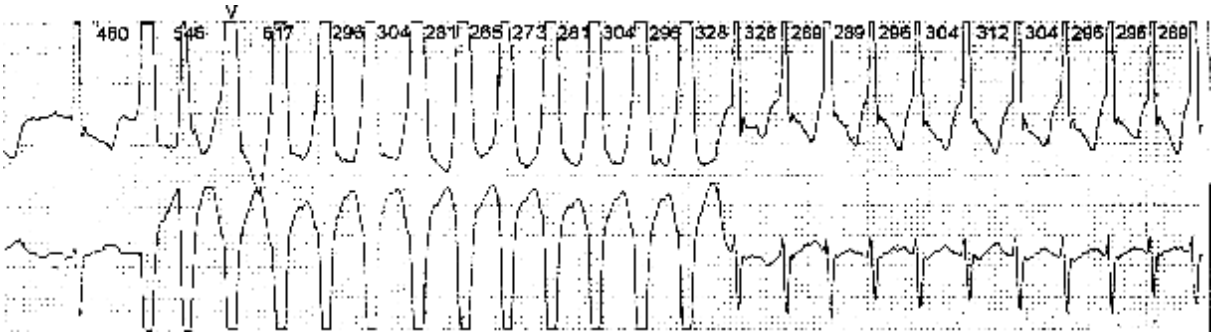
№ 12



TEST-TASK FOR SELF-CHECK OF LEARNING THE TOPIC

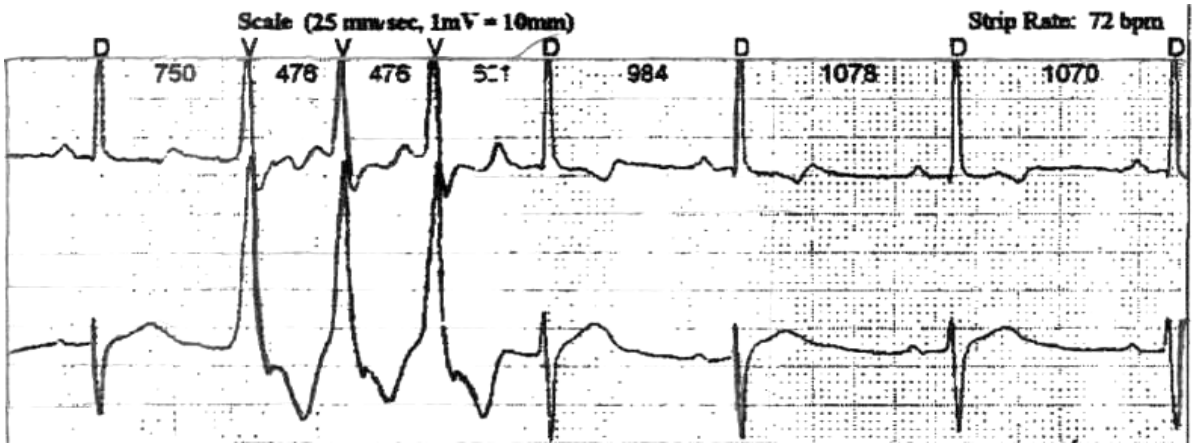
Choose correct answers of the given variants to every task, specifying, which types of arrhythmia and other forms of cardiac pathology are registered on ECG № 1–11.

№ 1



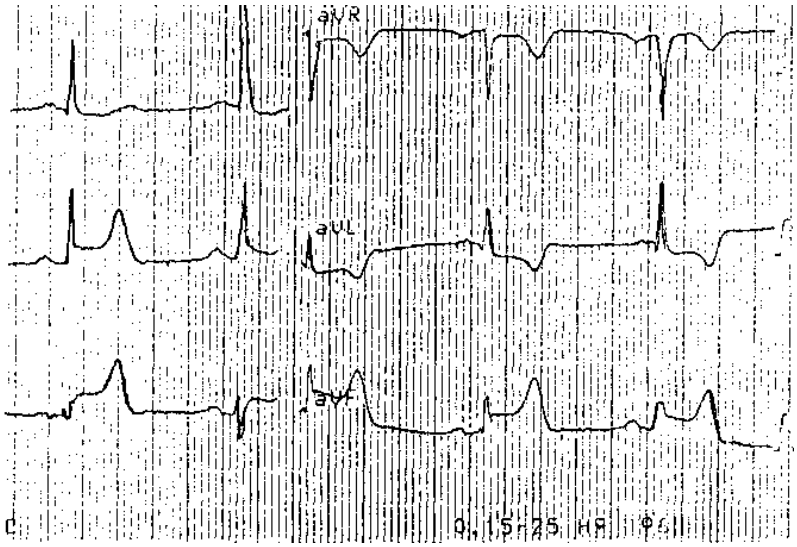
a) paroxysmal ventricular tachycardia;
b) ventricular palpitation;
c) sinus tachycardia;
d) polytopic extrasystole.

№ 2



a) polytopic ventricular extrasystole;
b) group monotopic ventricular extrasystole.

№ 3

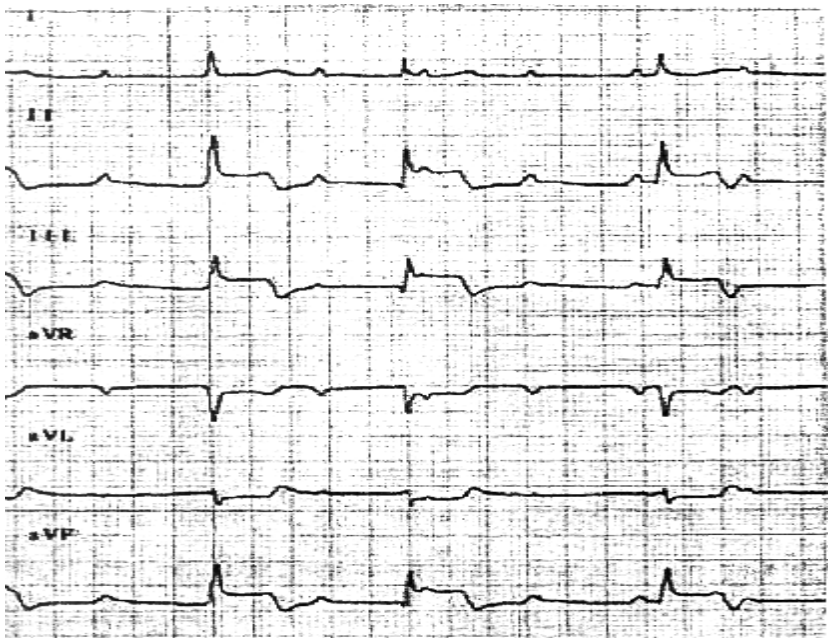


a) acute large-focal myocardial infarction of the posterior wall of the left ventricle (LV);

b) acute large-focal myocardial infarction of the anterior wall of LV;

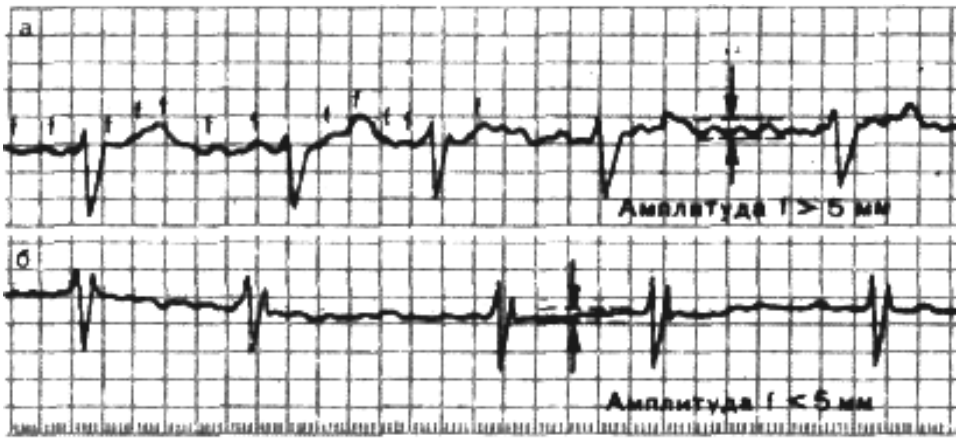
c) acute large-focal myocardial infarction of the apical part involving the lateral wall of LV.

№ 4



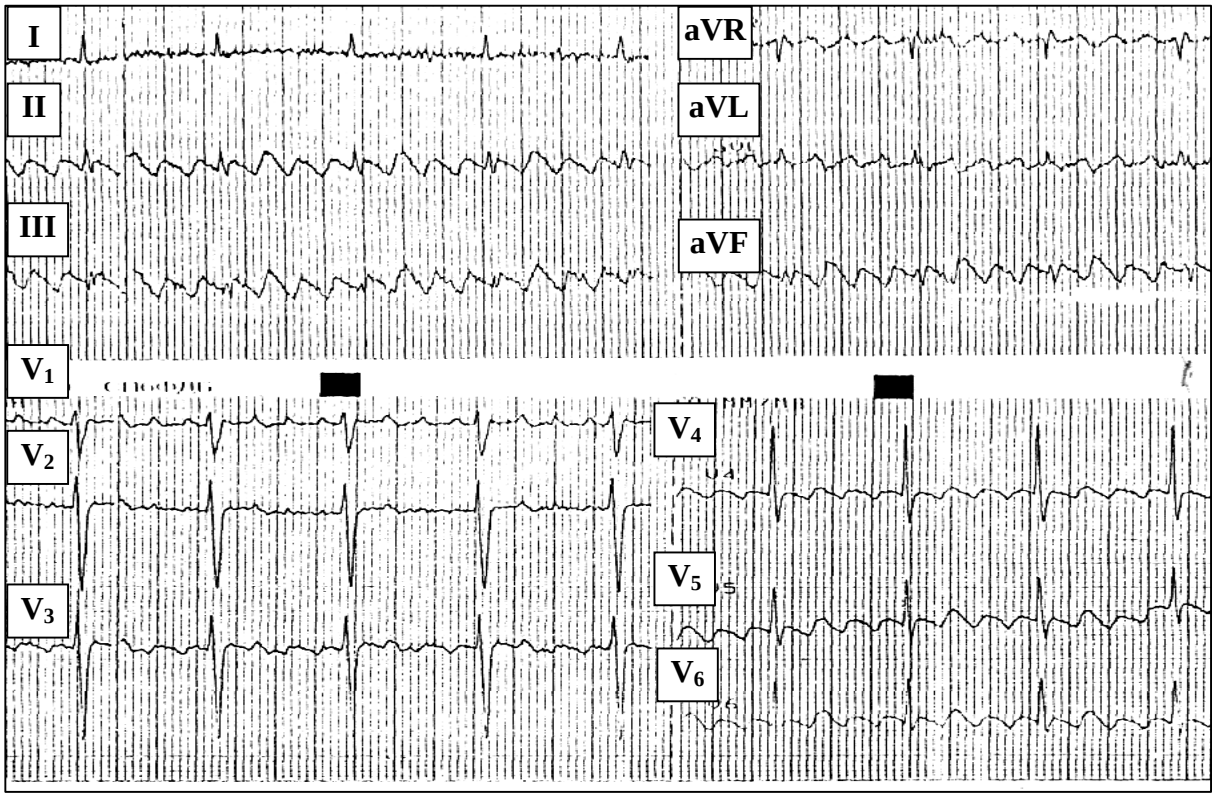
a) large-focal myocardial infarction of the inferior wall of LV;
b) incomplete A-V blockade of the II degree, Mobits I;
c) incomplete A-V blockade of the II degree, Mobits II;
d) right-ventricular ES (extrasystole) on bigeminy type;
e) complete A-V blockade.

№ 5



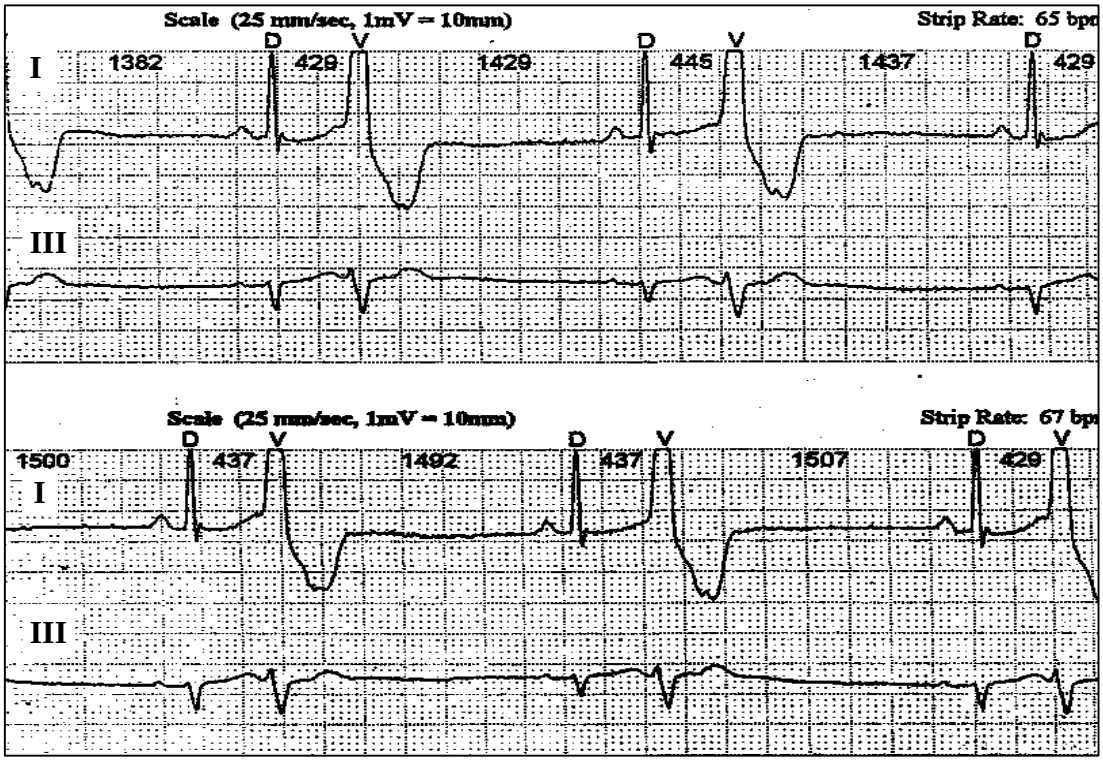
a) flutter of atria;
b) fibrillation of atria.

№ 6



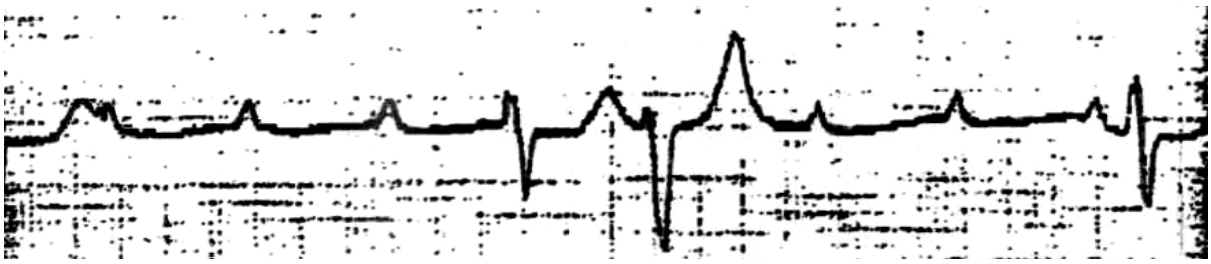
a) flutter of ventricles;
b) flutter of atria;
c) fibrillation of atria.

№ 7



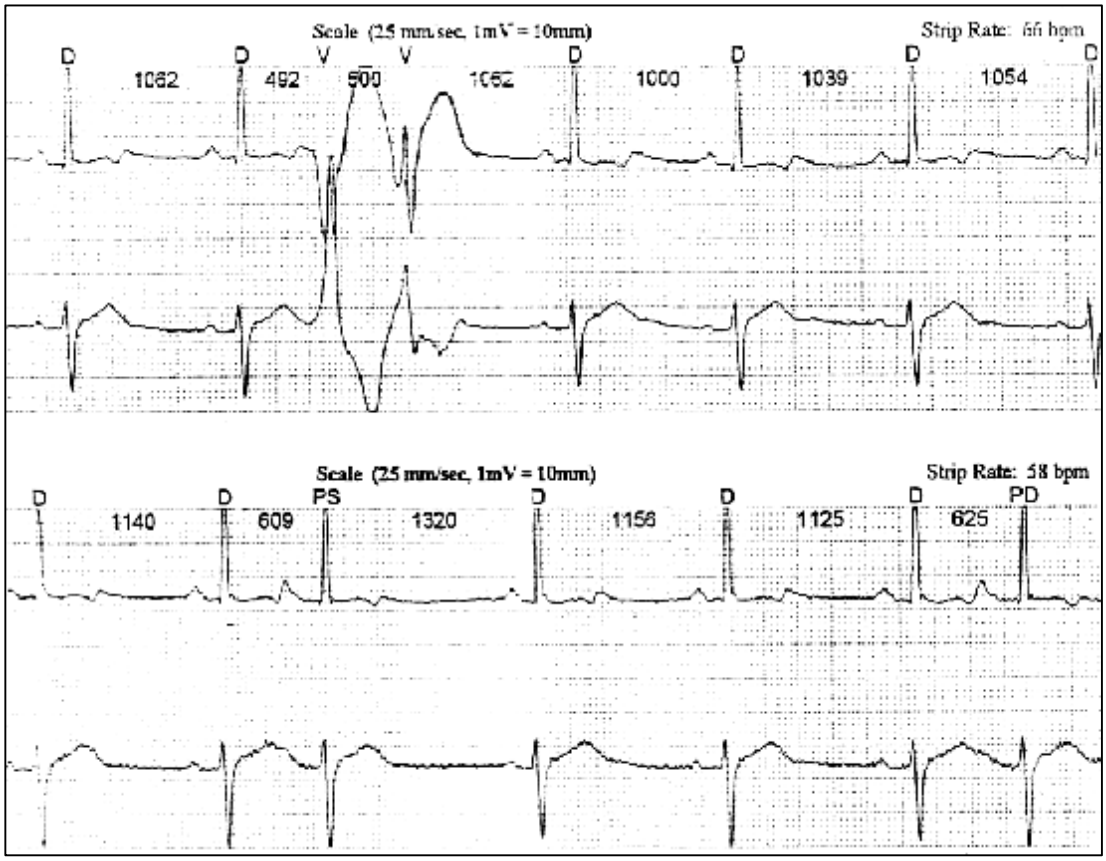
a) right-ventricular ES, bigeminaltype;
b) left-ventricular ES, bigeminaltype;
c) sub-ventricular bigeminy.

№ 8



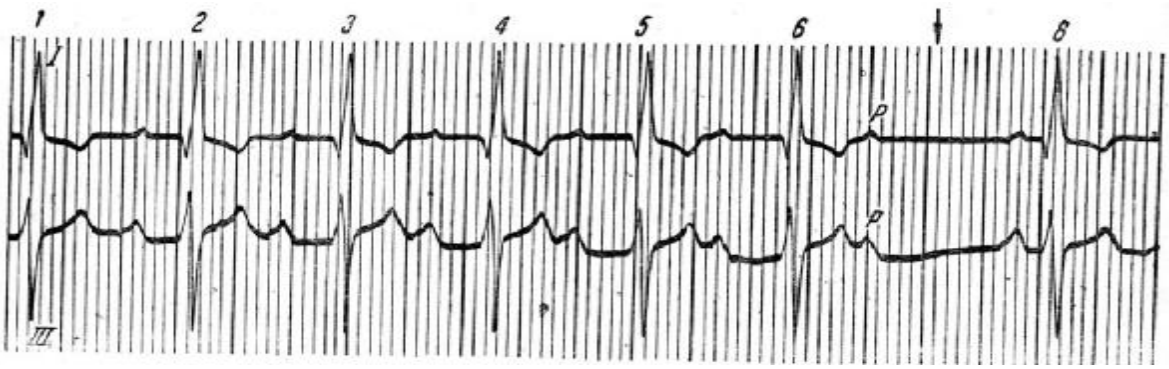
a) complete A-V blockade;
b) single ventricular extrasystole;
c) incomplete A-V blockade of the II degree, Mobits II;
d) right-ventricular ES on bigeminy type.

№ 9



a) polytopic ventricular extrasystole;
b) monotopic ventricular extrasystole;
c) sub-ventricular extrasystole.

№ 10



a) complete A-V blockade;
b) incomplete A-V blockade of the II degree, Miobits I;
c) incomplete A-V blockade of the II degree, Mobits II.

CORRECT ANSWERS TO THE QUESTIONS:

№ 1 – a; № 2 – b; № 3 – a; № 4 – a, e; № 5 – b; № 6 – b; № 7 – a; № 8 – a, b; № 9 – a, c; № 10 – c.

Control questions:

1. The definition of the notion «cardiac arrhythmias». Classification of arrhythmias.
2. Impairments of cardiac excitability: extrasystole (definition of the notion, causes, types, characteristic, ECG-manifestations, hemodynamic impairments).
3. Cardiac automatism impairment (types, causes, characteristic, development mechanisms, ECG-manifestations, hemodynamic impairments).
4. Cardiac conduction impairments: blockade of the heart (definition of the notion, causes, types, characteristic, ECG-manifestations, hemodynamic impairments).
5. Excitability and conduction impairments of the heart:

a) atrial flutter and fibrillation (causes, characteristic, ECG-manifestations, hemodynamic impairments);

b) ventricular fibrillation (causes, characteristic, ECG manifestations, hemodynamic impairments).

6. The notion of defibrillation of the heart.

The teacher's signature:

LESSON 11. PATHOLOGICAL PHYSIOLOGY OF THE BLOOD CIRCULATION SYSTEM (final seminar lesson)

Date: «____» _____ 20___

The purpose of the Lesson:

– To consolidate and estimate knowledge received at the lessons and practical classes on the blood circulation system impairments (etiology, pathogenesis, basic clinical manifestations and hemodynamic impairments).

Tasks:

1. To study:

– regulation disturbances of the vascular tone (arterial hyper- and hypotension), etiology, pathogenesis, mechanisms of hemodynamic impairments and manifestations;

– types, development mechanisms and manifestations of cerebrovascular insufficiency: paroxysms, crisis, strokes;

– etiology and pathogenesis of atherosclerosis.

2. Computer control of the topics:

– «Arrhythmias. Typical impairments of excitability, automatism and conduction of the heart»;

– «Pathological physiology of the blood circulation system».

3. Solving situational tasks (see the collection of situational tasks on pathological physiology).

Control questions:

1. Insufficiency of blood circulation. The definition of the notion, types.

2. Cardiac insufficiency. The definition of the notion. Principal incidence causes of cardiac insufficiency. Classification of cardiac insufficiency by pathogenesis, localization, course, severity degree. Primary and secondary cardiac insufficiency.

3. Hemodynamic classification of cardiac insufficiency. Systolic and diastolic dysfunctions. Etiology, pathogenesis, hemodynamic impairments and clinical manifestations of systolic and diastolic dysfunctions.

4. The basic parameters of intracardiac and systemic hemodynamic changes in all forms of cardiac insufficiency.

5. Etiology, pathogenesis and manifestations of acute left- and right-ventricular cardiac insufficiency.

6. Coronary insufficiency. The definition of the notion, clinical forms of IHD (ischemic heart diseases). Relative and absolute coronary insufficiency.

7. Etiological risk factors of IHD. Experimental methods of induction. Principal causes of non-coronarogenic necrosis of the myocardium.

8. Pathogenesis of ischemic and reperfusion syndromes in coronary insufficiency, their manifestations.

9. Myocardial infarction. Pathogenesis and manifestations of basic clinical-laboratory syndromes: pain, a syndrome of acute left-ventricular insufficiency (cardiac asthma, cardiogenic shock), a resorption-necrotic syndrome. Metabolism impairment, bioelectric and contractile properties of the myocardium.

10. Classification of chronic blood circulation insufficiency of cardiac genesis by a severity degree (Vasilenko–Strazhenko).

11. Compensation mechanisms of cardiac insufficiency. Their types, manifestations and pathogenetic characteristic.

12. Comparative characteristic of heterometric and homeometric mechanisms of intracardiac compensation in cardiac overstrain.

13. Remodeling of the myocardium. Outcomes of the myocardium remodeling depending on the type of hemodynamic overstrain and in damage of the myocardium.

14. Etiology, pathogenesis, mechanisms of urgent and long-term intracardial compensation in chronic myocardial overstrain by volume and pressure, outcomes, character of hemodynamic impairments, clinical manifestations.

15. Pathogenesis and clinical manifestations of syndromes of small output and congestion on the inflow ways to the weakened department of the heart. Signs of congestion in pulmonary and general blood circulation.

16. Extracardiac compensation mechanisms of cardiac insufficiency, their pathogenetic characteristic. The role of the vegetative nervous system in compensating chronic cardiac insufficiency. Hormono-neuro-mediator dissociation. Its pathogenetic characteristic.

17. Main effects of hyperactivation of sympato-adrenaline and renin-angiotensin-aldosterone systems in chronic cardiac insufficiency. Mechanisms of cardio-toxic effect of catecholamines. Pathogenetic characteristic of tachycardia in cardiac overstrain.

18. Reactions of respiratory and hemopoietic systems in the development of cardiac insufficiency trigger mechanisms of these systems.

19. Etiology, pathogenesis and manifestations of chronic left- and right-ventricular cardiac insufficiency.

20. Characteristic of compensatory cardiac hyperfunctions (CCH) in acute experimental overstrain of the left ventricle by resistance (according to F. Z. Meerson). Development stages of compensatory cardiac hyperfunction.

21. Hypertrophy of the myocardium, causes and mechanisms of its development. Functional and metabolic peculiarities of a hypertrophied myocardium. Development mechanisms of decompensation in pathological hypertrophy of the myocardium.

22. Pathogenetic therapeutic principles of cardiac insufficiency.

23. The definition of the notion «cardiac arrhythmias». Classification of arrhythmias.

24. Impairments of cardiac excitability: extrasystoly (the definition of the notion, causes, types, characteristic, ECG-manifestations, hemodynamic impairments).

25. Impairments of cardiac automatism (types, causes, characteristic, development mechanisms, ECG-manifestations, hemodynamic impairments).

26. Impairments of cardiac conduction: blockade of the heart (definition of the notion, causes, types, characteristic, ECG-manifestations, hemodynamic impairments).

27. Impairments of cardiac excitability and conduction:

a) flutter and fibrillation of atria (causes, characteristic, ECG-manifestations, hemodynamic impairments);

b) fibrillation of ventricles (causes, characteristic, ECG-manifestations, hemodynamic impairments).

28. Arterial hypertension, classification. Experimental forms of induction. Symptomatic arterial hypertension.

29. Etiology and basic pathogenesis theories of hypertonic disease.

30. The role of hyperactivation of renin-angiotensin-aldosterone systems in dysfunction development of organs-targets and stabilization of arterial hypertension. Clinical manifestations of the impairment of organs-targets in arterial hypertension.

31. Arterial hypotensions. Classification. Vascular insufficiency of blood circulation: fainting, collapse. Etiology, pathogenesis, manifestations.

32. Regulation impairments of cerebral blood circulation. Etiology, pathogenesis, manifestations. Pathological reactions of cerebral arteries, their types, characteristic.

33. Syndromes of «steal of the brain», «Robin Hood», excessive cerebral perfusions; their characteristic, pathogenetic characteristic.

34. Cerebrovascular insufficiency, its types. Paroxysms, crisis, strokes. Pathogenetic therapeutic principles of cerebrovascular insufficiency.

35. Atherosclerosis, it etiology and pathogenesis. The role of LPLD-impairments of receptor interaction in atherogenesis. Pathological and modified lipoproteins, their elimination from the organism by scavenger-receptors.

The teacher’s signature:

LESSON 12. PATHOLOGICAL PHYSIOLOGY OF THE EXTERNAL RESPIRATION SYSTEM. TYPICAL IMPAIRMENTS OF PULMONARY FUNCTIONS

Date: «____» _____ 20___

The purpose of the Lesson:

– To study etiology, pathogenesis, basic impairment forms of the external respiration system caused by the impairment of alveolar ventilation and perfusion; ventilation-perfusion relationships, diffusion in the lungs; development mechanisms of respiratory insufficiency, its stages.

Tasks:

– To study the elevation effect of intraalveolar pressure on parameters of respiration and blood circulation in the dog;

– To study the acidosis effect on parameters of pulmonary ventilation in experiment;

– To draw schematically and give a brief characteristic of pneumogram changes in typical impairments of pulmonary ventilation;

– Solving situational tasks (see the collection of situational tasks on pathological physiology);

– Test control of the topic of the Lesson.

Fill in the table.

Table 1

Clinical forms and manifestations of respiratory insufficiency

№	Respiratory insufficiency form	Basic development causes	Arterial blood gas structure	Clinical manifestations
1.				
2.				
3.				

3. Fill in the table.

Table 2

Functional impairments of organs and systems in acute mechanical asphyxia

Functions of organs and systems	1st stage	2nd stage	3rd stage
CNS (central nervous system)			

Functions of organs and systems	1st stage	2nd stage	3rd stage
Vegetative nervous system			
System of blood circulation (HR, BP)			
Respiratory system (type of respiratory impairment)			

Work 1. THE EFFECT OF INTRAARTERIAL PRESSURE ELEVATION ON PARAMETERS OF RESPIRATION AND BLOOD CIRCULATION IN THE DOG

A femoral artery is chosen in a narcotized dog and a cannula is inserted into it, then using the tubes filled with the solution of magnesium sulphate it is connected to the mercury manometer for arterial pressure recording.

The trachea is opened and a tracheal cannula is inserted into it; the last one (the lateral aperture being open) is connected with the artificial respiration apparatus.

The pneumograph cuff is fixed on the thorax and by means of a tube it is connected with Marey's capsule for recording of a pneumogram.

Having fixed the initial level of blood pressure and respiration rate, the intraalveolar pressure is elevated by closing the aperture in the tracheal cannula and blowing the air by means of the artificial respiration apparatus (5–6 inflations). Mark respiration changes and arterial pressure caused by these manipulations.

In subsequent opening of the lateral aperture of the tracheal cannula and letting out the excess of air from the lungs the pneumogram and the curve of blood pressure quickly return to their initial states (fig. 1).

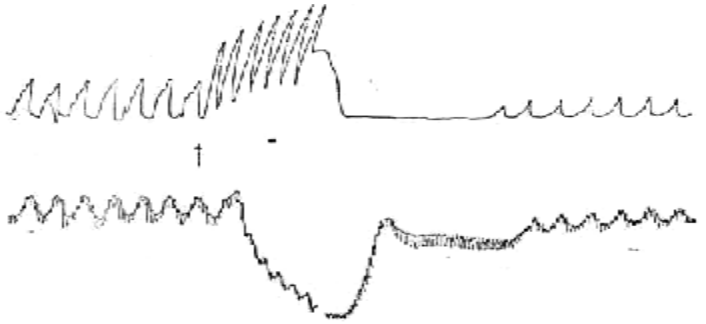


Fig. 1. Changes of respiration (the top curve) and arterial pressure (the bottom curve) in elevating the intraalveolar pressure of the dog. The arrows correspond to the moment of inflating air into the lungs

Answer the questions:

1. What changes of respiration and arterial pressure are noted in the dog after inflation of air into the lungs?

2. What is the possible mechanism of these changes?

3. In what pathological processes, diseases can a similar phenomena occur?

Work 2. RESPIRATION CHANGES IN THE DOG IN ACIDOSIS

The initial parameters of respiration (pneumogram) and arterial pressure of the dog are recorded, then 5 ml of 10 % solution of acetic acid are injected into its vein. Changes of registered parameters and their subsequent normalization are noted. After the establishment of the initial pneumogram and arterial pressure value, 10 ml of 25 % solutions of sodium dihydrophosphate (NaH_2PO_4) are injected into the vein (fig. 2).

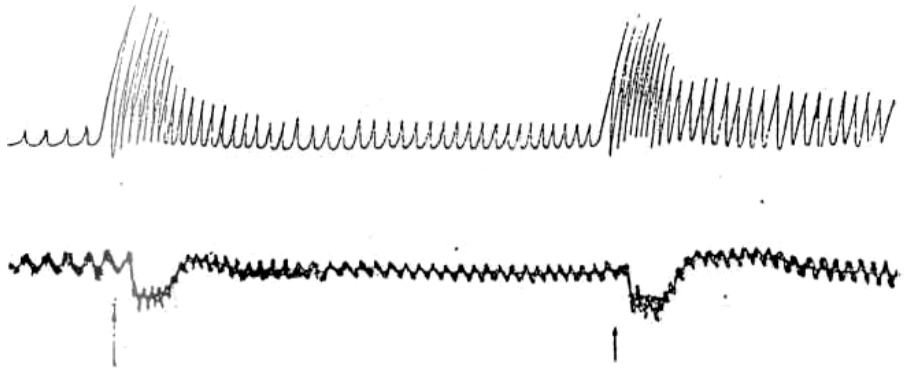


Fig. 2. Changes of respiration (the top curve) and arterial pressure (the bottom curve) in the development of acidosis in the dog. The first arrow corresponds to injection of the acetic acid solution into the blood, the second arrow — to injection of the sodium dihydrophosphate solution

Answer the questions:

1. What changes of respiration and arterial pressure are observed in the dog in injecting of the acetic acid and sodium dihydrophosphate solutions into the vein?

2. What are the mechanisms of these changes?

3. In what diseases, pathological processes can a similar phenomena occur?

Work 3. CHARACTERISTIC OF TYPICAL IMPAIRMENTS OF PULMONARY VENTILATION

Fill in the table:

Table 5

Pathological types of respiration

Respiration forms	Types	Occurs in pathological conditions	Pneumogram
Normal (eupnea)	Is not present	Is not present	
Deep accelerated (hyperpnea)			
Hurried superficial (polypnea)			
Stenosed			
Breathlessness	Inspirat.		
	Expirat.		
Periodic	Chain–Stocks		
	Wavy		
	Biot		
Terminal	Gasping		
	Apnesis		
	Kussmaul		

Control questions:

1. Insufficiency of the external respiration system. The definition of the notion, classification. Causes and developmental mechanisms. Stages of chronic respiratory insufficiency, its clinical manifestations.

2. Impairments of pulmonary ventilation: obstructive, restrictive and mixed, principal causes and manifestations. Changes of alveolar air gas content and arterial blood in the impairment of ventilation.

3. Impairments of gas diffusion through the lung membrane, principal causes and manifestations. Changes of gas content of alveolar air and arterial blood in the impairment of diffusion of gases. Etiology and pathogenesis of respiratory distress-syndrome of adults.

4. Principal causes of the impairment of pulmonary perfusion. Forms and causes of pulmonary hypertension. Chronic pulmonary-cardiac insufficiency: pulmonary heart, etiology, pathogenesis, clinical manifestations.

5. Regulation impairments of respiration. Breathlessness, periodic and terminal respiration. Their forms, pathogenetic characteristic, development mechanisms.

6. Asphyxia. Etiology, pathogenesis, development stages.

The teacher's signature:

LESSON 13. PATHOLOGICAL PHYSIOLOGY OF DIGESTION SYSTEMS

Date: «____» _____ 20___

The purpose of the Lesson:

– To study the causes, developmental mechanisms and manifestation of impairment forms of secretory, motor and absorption functions of the gastrointestinal tract.

Tasks:

– To determine the types of gastric secretion of patients by the presented graphs and tables, to get acquainted with clinical assessment of the secretory activity impairment of the stomach;

– Solving situational tasks (see the collection of situational tasks on pathological physiology);

– Test control of the topic of the Lesson.

1. Fill in the table.

Table 1

Interrelation of motor and secretory functional impairments of the stomach

Clinical manifestations	Hyperchlorohydria with pepsin hypersecretion	Hypo- and achlorohydria with pepsin hyposecretion
Acidity and volume of gastric contents		
Velocity of chyme evacuation and its neutralizations in the duodenum		
Pyloric sphincter, is mainly spasmodic/gaping		
Pain syndrome (+/-)		
Muscular tone of the stomach (↑↓)		
Antiperistaltics (+/-)		

Clinical manifestations	Hyperchlorohydria with pepsin hypersecretion	Hypo- and achlorohydria with pepsin hyposecretion
Heartburn (+/-)		
Belching (+/-), its character		
Vomiting (+/-), its character, whether it relieves the pain or not		
Impairment of intestinal motility (+/-), its form (diarrhea /constipation)		

2. Fill in the table.

Table 2

Pain syndrome in gastrointestinal diseases

Type of pain	Its main causes	Pain characteristics
Visceral		
Somatic		

3. Factors of gastric mucus protection:

–

–

–

–

–

4. Factors of aggression towards gastric mucus:

–

–

–

–

5. Draw schematically Shey's balance (the ratio of protection factors (1) and aggressions factors (2) of gastric mucus)

NORM ULCER

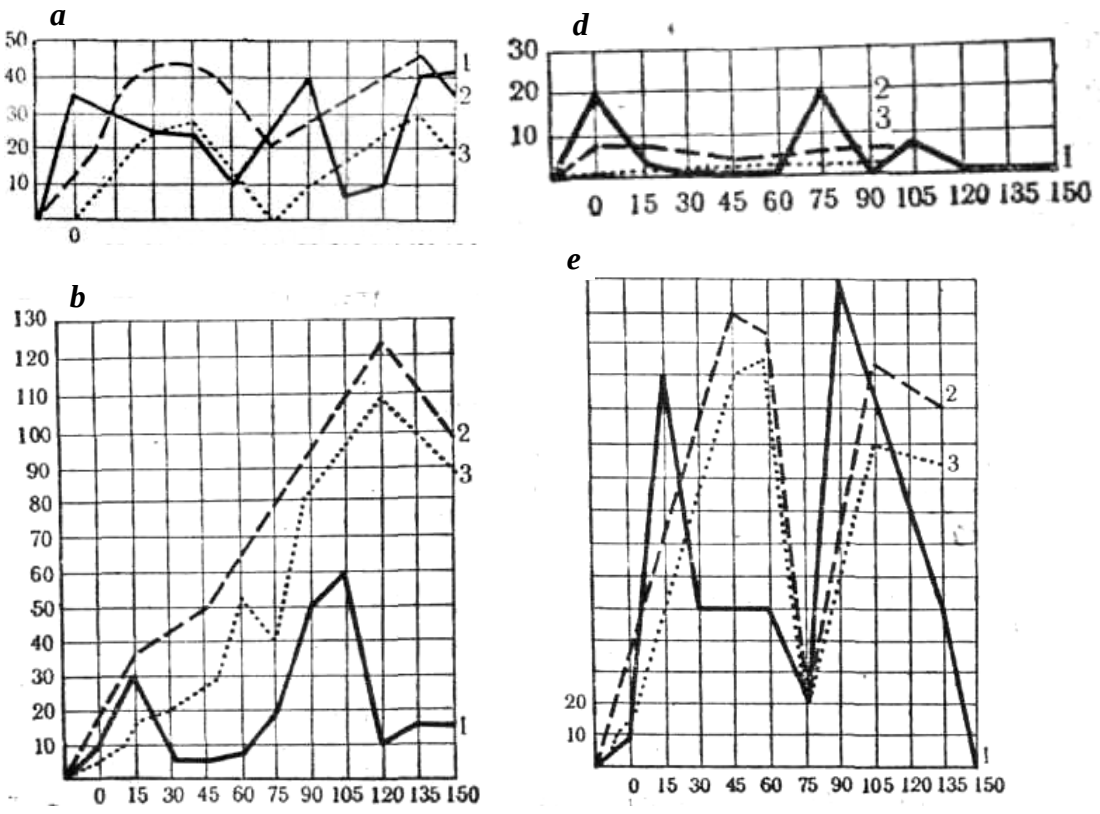
8. Imbalance forms between factors of aggression and protection, their role in pathogenesis of gastric ulcer in young and advanced age:

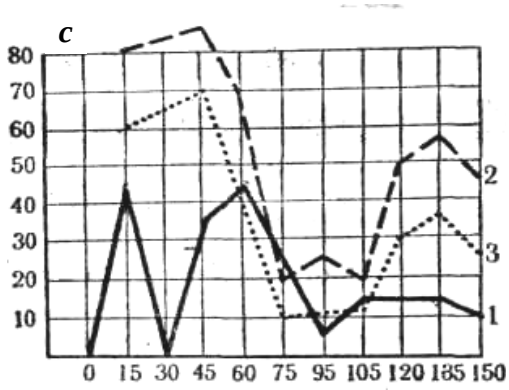
–

–

Work 1. DETERMINING THE TYPES OF GASTRIC SECRETION

Determine the types of gastric secretion of various patients using graphs





a –

b –

c –

d –

e –

Ordinate axis — the amount of juice in milliliters, acidity in titration units. Abscissa axis — the time in minutes: under 10 min — on an empty stomach, 10–60 min — in mechanical irritation of the stomach, 60–150 min — in chemical irritation of the stomach.

Conclusion: specify the types of gastric secretion (graphs a, b, c, d, e).

Work 2. DETERMINING TYPICAL IMPAIRMENT FORMS OF GASTRIC SECRETORY ACTIVITY IN PATIENTS

Determine the type of secretion and the state of gastric secretory activity of patients A, B, C using digital data of laboratory findings (table 3).

Table 3

Fractional test parameters of gastric juice

	Conditions of taking and studying gastric contents	**Gastric contents volume, ml**	Titration units			**Pepsin, мg %**
			total acidity	**free HCl**	**bound HCl**	
Norm	On an empty stomach	No more than 50	Up to 40	Up to 20	–	0–21
	Basal secretion	50–100	40–60	20–40	10–15	20–40
	Stimulated secretion	50–110	40–60	20–40	10–15	21–45
Patient A	On an empty stomach	10	30	–	10	10
	Basal secretion	–	–	–	–	–
	Stimulated secretion	20	35	10	10	5
Patient B	On an empty stomach	100	60	30	20	15
	Basal secretion	120	80	60	10	30
	Stimulated secretion	140	100	50	30	50
Patient C	On an empty stomach	70	50	30	10	–
	Basal secretion	120	60	30	15	–
	Stimulated secretion	10	10	–	5	–

Conclusions: Type of gastric secretion and functional state of gastric secretory activity:

Patient A –

Patient B –

Patient C –

Control questions:

1. Experimental methods of studying the digestive system activity in norm and in pathology (I. N. Basov, I. P. Pavlov).
2. The impairment causes of the digestive system activity and basic signs of these impairments.
3. Digestion impairment in the oral cavity: principal causes and consequences of hypo- and hypersalivation, mastication impairments. Principal causes of dysphagia.
4. Basic manifestations of gastric dyspepsia: the impairment of appetite, nausea, belching, vomiting, pain syndrome. Causes of their development.
5. Interrelation of secretory and motor functional impairments of the stomach. Manifestations of hyper- and hypochlorohydria. Pathology of a pyloric reflex.
6. Gastric ulcer and duodenal ulcer. Development theories of ulcer. Modern conceptions of etiology and pathogenesis of gastric ulcer. The role of *H. pylori* in pathogenesis of the diseases.
7. Impairments of intestinal secretory activity and absorption processes. Etiology, pathogenesis and clinical manifestations of syndromes of maldigestion and malabsorption.
8. Impairment mechanisms of motor intestinal function (diarrhea, constipation). Etiology, pathogenesis.
9. Intestinal autointoxication. Etiology, pathogenesis, manifestations.

The teacher's signature:

LESSON 14. PATHOLOGICAL PHYSIOLOGY OF THE LIVER

Date: «____» _____ 20___

The purpose of the Lesson:

– To study the causes and mechanisms of basic syndromes occurring in liver pathology. To characterize typical forms of functional impairments of the liver.

Tasks:

– To study mechanisms and manifestations of general toxic action of bile, its effect on the nervous system and cardiac muscle;

– Solving situational tasks (see the collection of situational tasks on pathological physiology).

1. Fill in the table:

Table 1

Contents (↑, ↓ or N) of bile pigments in biological environments in various forms of jaundice

Jaundice form	Blood	Urine		Faeces	
		Color	Pigment	Color	Pigment
Mechanical					
Parenchymatous					
Hemolytic					

2. Fill in the table:

Table 2

Basic syndromes in jaundice

Syndrome	Characteristic of jaundices	Pathogenesis	Manifestations
Cholemias			
Acholias			
Hyper-cholias			

3. List the basic pathogenetic factors of hepatic coma:

4. List the manifestations of portal hypertension:

5. Positive consequences of the development of collateral blood circulation in portal hypertension:

6. Negative consequences of the development of collateral blood circulation in portal hypertension:

7. Draw schemes of operations:

Table 3

N. V. Ekka's fistulas	N. V. Ekka, I. P. Pavlova's fistulas

Work 1. STUDYING GENERAL TOXIC ACTION OF BILE ON THE ORGANISM

1.5–2.0 ml of bile are injected into the frog's lymphatic bag located under the skin. A healthy animal serves as control. The observation data are given in table 4.

Table 4

Bile effect on the state of the nervous and cardiovascular system

Time	Control	Experiment
1'	Spontaneous twitching of extremity muscles is not marked. Coordination of movements is preserved. HR — 40 per min	Periodic twitching of paws is observed. The muscular tone is not changed. Coordination of movements is preserved. The frog, being turned over on the back, returns to a normal position. HR — 43 per min
3'	The frog is sitting, under external stimuli its motor activity increases. The muscular tone is not changed. Coordination of movements is not impaired. HR — 42 per min	The frog jumps, bumping against the chamber walls. The muscular tone is elevated, muscular contractions are periodically observed. Lying on the back, the frog cannot take the previous original position at once. HR — 30 per min

Time	Control	Experiment
5'	The same condition. HR — 42 per min	Motor activity is reduced due to significant decrease of the muscular tone. The frog is motionless, listless, is lying, it cannot return to the initial position from the position on the back. HR — 35 per min
7'	The same condition. A pain stimulus action is accompanied by squeak and increase of motor activity. HR — 43 per min	The frog has not changed the position given to its body. It does not response to the action of a pain stimulus. HR — 30 per min

Draw the conclusions and answer the following questions:

1. What syndrome arises in the animal being injected with bile parenterally? By what bile components is it caused by?

2. On the part of what systems are the impairments noted? Give their characteristic, possible developmental mechanisms.

Work 2. STUDYING THE EFFECT OF BILE ON THE TIME OF A MOTOR REFLEX IN THE FROG

A decapitated frog is suspended by the mandible on the stand. In 5–10 min a paw of the frog is dipped into 0,2 % solution of hydrochloric acid. Using the metronome the time of the frog's motor response to irritation by the acid (it jerks the paw back) is taken. After several repeated irritations the average latent period of response (the number of metronome beats) is determined. After each immersion into the acid it is necessary to wash the paw with water carefully. Then 0,5–1,0 ml of bile are injected into the frog's lymphatic sac, in 15–20 min the experiment with irritating the paw with hydrochloric acid is repeated.

Table 5

Bile effect on the time of a motor reflex in the frog

Reflex time by Turk, sec	
Before bile injection	**After bile injection**
2	7
1	9
3	8
2	10
average latent period	average latent period
2	8,5

Draw the conclusions, answer the following questions:

1. What are the manifestations of bile effects on the nervous system?

2. What are possible mechanisms of this action?

Work 3. STUDYING THE BILE EFFECT ON HEART RATE OF THE FROG

An immobilized frog is attached to a plate with its abdomen upward, the thorax and the pericardium are open and the heart is exposed. The heart rate is counted. Then some drops of bile are applied to the frog's heart with the pipette in various concentrations: 1 : 10, 1 : 5, 1 : 2 and whole bile. After every application and repeated HR registration the heart is carefully washed with physiological solution.

Table 7

Effect of bile in various concentrations on the frog's HR

Effect	**Heart rate, beats/min**
Reference value (before the effect)	43
Bile, dilution 1:10	40
Bile, dilution 1:5	30
Bile, dilution 1:2	5
Whole bile	Cardiac arrest

Analyze the results, draw the conclusions and answer the following questions:

1. What is the character of the cardiac muscle response to application of bile?

2. What is the action mechanism of bile on the cardiac muscle?

Control questions:

1. Experimental methods of studying functions of the liver (N. V. Ekk, E. S. London, I. P. Pavlov). Changes in the organism in the given interventions.
2. Basic etiologic factors of hepatic damage. Basic syndromes in pathology of the liver and bile ducts.
3. The definition of the notion, etiology and pathogenesis of mechanical, parenchymatous and hemolytic forms of jaundice. Bilirubin exchange in various forms of jaundice.

4. The definition of the notion and basic syndromes manifestations of cholemia, acholia and hypercholia in jaundice of various forms.

5. The syndrome of portal hypertension. The definition, forms, clinical symptoms.

6. Pathogenetic characteristic of collateral and portocoval blood circulation in portal hypertension.

7. Pathogenesis of ascites in portal hypertension.

8. Hepatic insufficiency. The definition, etiology, pathogenesis, laboratory and clinical manifestations.

9. Hepatic coma. The definition, forms (bypass, hepatic-cellular). Pathogenesis.

The teacher's signature:

LESSON 15. PATHOLOGICAL PHYSIOLOGY OF KIDNEYS

Date: «____» _____ 20___

The purpose of the Lesson:

– To study the causes, development mechanisms and basic clinical manifestations of renal functional impairments. To characterize typical forms of renal functional impairments.

Tasks:

– To study some typical functional impairments of kidneys in experiment;

– Solving situational tasks (see the collection of situational tasks on pathological physiology).

Work 1. STUDYING SOME MECHANISMS OF DIURESIS IMPAIRMENT IN EXPERIMENT

The abdominal cavity of the dog narcotized with morphine-ether is opened and the ureters are carefully taken out. Cuts are made in the upper third of them, and glass cannulas are inserted into the proximal part connected with two glass tubules letting out the urine. A cannula with a rubber tube and a clamp is inserted into a distal part of one of the ureters, the other distal part is tied up with a ligature. The ligature is applied to the mouth of the ureter. The common vena cava, femoral artery and jugular vein are located. A cannula is inserted into vena cava connected with the manometer to register arterial blood pressure. The cannula with a rubber tube and a clamp is inserted into the femoral artery. Insert a cannula connected to the graduated cylinder with a tube and fill in the system with physiological solution. Bring ligatures under the allocated sciatic nerve and one of renal arteries and veins.

Experiment 1. Changing of diuresis in hydremia

Determine the initial level of diuresis, counting the drops of urine excreted for 3 minutes by each kidney. 300–400 ml of a physiological solution (38–40 ºC) are introduced into the jugular vein and diuresis is determined again. Simultaneously arterial blood pressure is taken.

Experiment 2. Changing of diuresis in hyperglycemia

Having determined the initial level of diuresis, 40 % solution of glucose (1 ml/kg of the body weight) is injected into the jugular vein. In 5 minutes diuresis is evaluated by the number of drops of urine.

Experiment 3. Changing of diuresis in acute blood loss

After preliminary evaluation of diuresis 50–100 ml of blood are taken out from the femoral artery. Diuresis is determined and arterial pressure is taken.

Experiment 4. Hormonal effects on diuresis

0.1 % solution of adrenaline (0.02 ml/kg of body weight) is injected into the jugular vein. In 3-5 minutes diuresis is determined and arterial pressure is taken.

Experiment 5. Reflex anuria in stretching of the bladder

The walls of the bladder are stretched with the air injected by a syringe through a cannula with a rubber tube and a clamp, inserted into a distal part of one ureter. Diuresis is determined before and after stretching of the bladder.

Experiment 6. Reflex oliguria in pain irritations of a sciatic nerve

Electrodes are applied to a sciatic nerve and it is irritated with electric pulses from the electrostimulator. Diuresis change is examined, arterial pressure is registered.

Experiment 7. Changing of diuresis in renal ischemia

One of renal arteries is occluded with a ligature for 1–2 minutes. Having collected a small amount of urine from an ischemized kidney a test is made for the presence of protein in the urine. Then 200 ml of physiological solution (38–40 ºC), which is stained with 2 ml of 5 % solution of indigocarmine, are introduced into the jugular vein. Register the time, when the color appears in the urine excreted by an intact kidney and an inschemized one.

Experimental results are presented in the table.

Table 1

Changes of the amount of diuresis and arterial blood pressure in a number of typical renal functional impairments

Pathological effect	Diuresis, drops/min				BP, mm Hg	
	Left kidney		Right kidney			
	before	after	before	after	before	after
Hydremia	6	8	5	9	130/60	145/65
Hyperglycemia	5	9	6	10	125/65	130/75
Acute blood loss	6	2	6	2	130/60	95/75
IV injection of 0.1 % of adrenaline	5	2	5	3	120/65	150/80
Stretching of the bladder	6	1	7	0	125/60	140/65
Irritation of a sciatic nerve	7	3	6	3	130/60	150/85
Renal ischemia	6	2	5	6	125/60	140/80
Test on the presence of protein in the urine from an ischemic kidney						+++
Time of appearing the stained urine			intact kidney			2 min
			ischemic kidney			5 min

Answer the questions:

1. Explain the mechanism of diuresis changing in hydremia, hyperglycemia.

2. Explain the mechanism of diuresis changing in acute blood loss, in IV injection of adrenaline.

3. Explain the developmental mechanism of anuria in stretching of the bladder.

4. Explain the developmental mechanism of pain olyguria.

5. Explain the mechanism of diuresis changing in renal ischemia.

6. Why does the test sample from an ischemized kidney reveal protein? What kind of proteinuria develops in this case?

7. Why does the time of appearing the color in the urine is different for an intact and ischemized kidney?

Control questions:

1. General etiology and pathogenesis of renal functional impairments.
2. Impairment mechanisms of glomerular filtrations, proximal and distal reabsorption, canaliculi secretion and excretion.
3. Clinical manifestations of renal functional impairments. Changes of diuresis and urine content. A uric syndrome: hematuria, hemoglobinonuria, proteinuria, cylidruria, anuria, olyguria, polyuria, hypostenuria, isostenuria. Causes and mechanisms of their development. Pathological components of urine of renal and extrarenal origin.
4. General symptoms in renal diseases.
5. Glomerulopathies. Diffused glomerulonephritis (etiology, pathogenesis and clinical manifestations).
6. Nephrotic syndrome.
7. Acute renal insufficiency. Its forms, etiology, pathogenesis, course stages, clinical manifestations, outcomes. Changes of the volume and content of blood and urine.
8. Chronic renal insufficiency. Etiology, pathogenesis, stages, clinical manifestations. Azotemias and uraemias. Basic clinical manifestations of uraemia.
9. Causes and mechanisms of formation of renal stones, urolithiasis.
10. Changes in dentition tissues in chronic renal insufficiency.

The teacher's signature:

LESSON 16. PATHOLOGICAL PHYSIOLOGY OF THE NERVOUS SYSTEM. SENSOR AND LOCOMOTOR FUNCTIONAL IMPAIRMENTS

Date: «____» _____ 20___

The purpose of the Lesson:

– To study the causes, mechanisms and the basic clinical manifestations of sensor and locomotor functional impairments of the organism in damaging various departments of the nervous system.

Tasks:

– To study the causes, developmental mechanisms and clinical manifestations of locomotor functional impairments in the damage of the pyramidal and extrapyramidal systems on the basis of materials presented in teaching videos.

– To study manifestations of the impairments of sensor and locomotor functions of the organism in the damage of anterior and posterior processes of the spinal cord in experiment.

– Solving situational tasks (see the collection of situational tasks on pathological physiology).

– Test control of the topic of the Lesson.

Work 1. STUDYING THE ETIOLOGY, PATHOGENESIS AND CLINICAL MANIFESTATIONS OF FUNCTIONAL IMPAIRMENTS OF THE NERVOUS SYSTEM ON THE BASIS OF MATERIALS OF TEACHING VIDEOS

a) Mechanisms and clinical manifestation forms of spastic and flaccid paralyses;

b) Pathogenetic treatment of some hereditary extrapyramidal diseases.

On the basis of videos draw the conclusions, answer the questions:

1. What are the signs of the impairment of locomotor functions of the organism in damage of the nervous system?

2. The damage of what structures of the nervous system results in central (spastic) and peripheral (flaccid) paralyses?

3. In what way does the muscular tone, tendon and periosteal reflexes, muscular trophic condition change in spastic and flaccid paralyses?

4. Why do the tendon and periosteal reflexes increase in spastic paralysis, while in flaccid paralysis they are absent?

5. For what kind of paralysis is the presence of pathological reflexes typical?

Conclusions (define the symptomocomplex, characteristic for spastic (central) and flaccid (peripheral) paralysis):

Work 2. STUDYING THE IMPAIRMENTS OF LOCOMOTOR REACTIONS IN CUTTING ANTERIOR AND POSTERIOR PROCESSES OF THE SPINAL CORD IN THE FROG

The frog is fixed to a plate with its back upwards. We cut the skin of the back from the fourth vertebra to the caudal part and deepen the cut up to osseous processes of the vertebrae. Detach the adjoining muscles to expose the vertebral arches. The arches are removed by the scissors from the third up to the fifth vertebra. Now the spinal cord with its membranes, which are carefully cut, is seen, and the processes of the spinal cord are revealed. Cut the posterior (sensitive) processes on the right and anterior (locomotor) processes on the left.

If the right hind paw is pinches, no reaction occurs (fig. 1). If a hind paw on the side with cut anterior processes is pinched (fig. 2), there will be no reaction due to switching off the locomotor processes, however contraction of the right paw is observed.

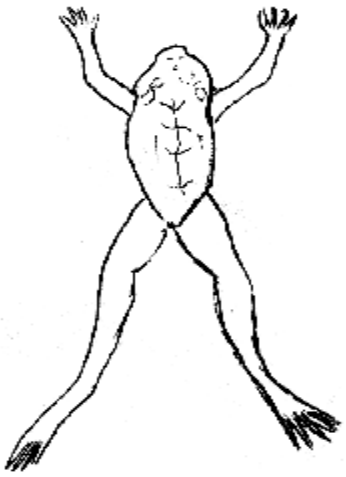


Fig. 1. Absence of reaction to irritation

Fig. 2. Contraction of the right paw

Answer the questions:

1. The impairment of what forms of sensitivity are marked in cutting posterior processes of the spinal cord and why was the motor reaction to irritation of this paw absent in the frog (fig. 1)?

2. Why is the motor reaction to irritation of the paw absent in the frog with cut anterior processes of the spinal cord on this side, but there is a motor reaction of the paw on the side with cut posterior processes of the spinal cord (fig. 2)?

3. What kind of a paralysis is marked in cutting anterior processes of the spinal cord?

Control questions:

1. General etiology and pathogenesis of the nervous system impairments.
2. Protective, regenerative and compensatory processes in the nervous system. «Protective inhibition », its role for pathology.
3. Neurogenic impairments of sensitivity, their types, mechanisms and clinical manifestations.
4. Brown-Sequard syndrome. The mechanism of its origin and manifest-tation.
5. Neurogenic disturbances of the locomotor function. Hypokinetic conditions: paresis and paralysis, their mechanisms and characteristic.

6. Hyperkinesis. The definition of the notion. Types of hyperkinesis.

7. Convulsive conditions, types of spasms and their pathogenesis.

8. Functional impairments of the vegetative nervous system, their types and mechanisms.

9. The impairments of higher nervous activity, neurosis. The significance of the types of higher nervous activity in the development of neuroses. The causes of neurosis, their characteristic, principles of therapy.

10. Experimental models of neurosis (I. P. Pavlov, M. K. Petrov). Therapeutic principles of neuroses.

11. Pain. The definition of the notion, its biological significance. Pathogenesis of a pain syndrome. The antinociceptive system and its characteristic.

12. The study of the trophic function of the nervous system and neurogenic dystrophies. The standard form of neurogenic dystrophies (A. D. Speransky). The role of neurogenic dystrophies in pathogenesis of diseases.

13. Modern conceptions of the mechanisms of trophic effect of the nervous system on tissues and organs and the development of neurogenic dystrophies. Trophogens and pathotrophogens.

The teacher's signature:

LESSON 17. PATHOLOGICAL PHYSIOLOGY OF THE ENDOCRINE SYSTEM

Date: «____» _____ 20___

The purpose of the Lesson:

– To study general etiology and pathogenesis of endocrinopathies; typical impairment forms of some endocrine glands.

Tasks:

– To get acquainted with typical impairment forms of some endocrine glands on the basis of materials presented on slides, tables and figures to the topic.

– Solving situational tasks (see the collection of situational tasks on pathological physiology).

– Test control of the topic of the Lesson.

1. Specify the basic pathogenetic ways of developing endocrine pathology:

1)

2)

3)

2. Fill in the table:

Table 1

Classification of endocrinopathies

Classification principle	Endocrinopathy type
Functional condition of the gland	1 2 3
Secretion impairment of one or all hormones of the gland	1 2
Prevalence of the process in the endocrine system	1 2
Changing of the hormone production by the gland or the impairment of peripheral effect	1 2 3
Damage level	1 2 3

3. List the possible peripheral (extraglandular) mechanisms of endocrine functional impairments:

1)

2)

3)

4)

4. List the major factors resulting in the development of pathological processes in the gland itself:

5. Specify the basic manifestations of the endocrine gland dysfunction:

1)

2)

3)

6. Name the treatment principles of endocrine disturbances:

1)

2)

3)

Control questions:

1. Etiology and pathogenesis of endocrinopathies. Principles of their classification. Main principles of treatment.
2. The notion of intra-uterine endocrinopathy. Peculiarities of functional integration of homologous endocrine organs of the maternal organism and the fetus.
3. Total (Simmonds disease) and partial hypofunction of adenohypophysis (Hypophyseal nannism, infantilism), clinical manifestations.
4. Hyperfunction of the adenohypophysis: hypophyseal giantism, acromegally, disease of Itsenko–Cushing, clinical manifestations.
5. The pathology of a posterior lobe of the hypophysis: signs of hypo- and hypersecretions of vasopressin.
6. The thyroid gland pathology, its forms, pathogenesis, clinical manifestations.

7. The parathyroid glands pathology, its forms, pathogenesis, clinical manifestations.

8. Hypofunction of the cortical substance of adrenal glands. Acute and chronic insufficiency of adrenal glands, etiology, pathogenesis, clinical manifestations.

9. Hyper- and dysfunction of the cortical and medulla substance of adrenal glands. Syndrome of Itsenko-Cushing, primary and secondary hyperaldosteronism, adreno-genital syndrome, pheochromocytoma, clinical manifestations.

10. Diabetes of the I and II type, their etiology, pathogenesis, clinical manifestations. Mechanisms of hyperglycemia and glycozuria. Manifestations of the impairment of organs — targets in diabetes.

The teacher's signature:

CONTENT

Учебное издание

Висмонт Франтишек Иванович
Касап Валентина Александровна
Жадан Светлана Анатольевна и др.

ПАТОЛОГИЧЕСКАЯ ФИЗИОЛОГИЯ

PATHOLOGICAL PHYSIOLOGY

Практикум
На английском языке

4-е издание

Ответственный за выпуск Ф. И. Висмонт
Переводчики: Т. Ф. Данилова, С. А. Жадан
Компьютерный набор С. А. Жадан, Е. В. Меленчук
Компьютерная верстка Н. М. Федорцовой

Подписано в печать 17.09.15. Формат 60×84/16. Бумага офсетная.
Ризография. Гарнитура «Times».
Усл. печ. л. 11,16. Уч.-изд. л. 6,1. Тираж 120 экз. Заказ 544.

Издатель и полиграфическое исполнение: учреждение образования
«Белорусский государственный медицинский университет».
Свидетельство о государственной регистрации издателя, изготовителя,
распространителя печатных изданий № 1/187 от 18.02.2014.
Ул. Ленинградская, 6, 220006, Минск.