

МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ
БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ
КАФЕДРА ПАТОЛОГИЧЕСКОЙ ФИЗИОЛОГИИ

ПАТОЛОГИЧЕСКАЯ ФИЗИОЛОГИЯ

PATHOLOGICAL PHYSIOLOGY

Практикум



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Содержит описания и протоколы оформления лабораторных работ по основным разделам курса патофизиологии.

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

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Section I General Nosology

LESSON 1. Introductory lesson. the subject, tasks, methods of pathological physiology

Date: « ____ » _____ 200__.

The purpose of the Lesson: to consider the subject of studying, the essence and tasks 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.

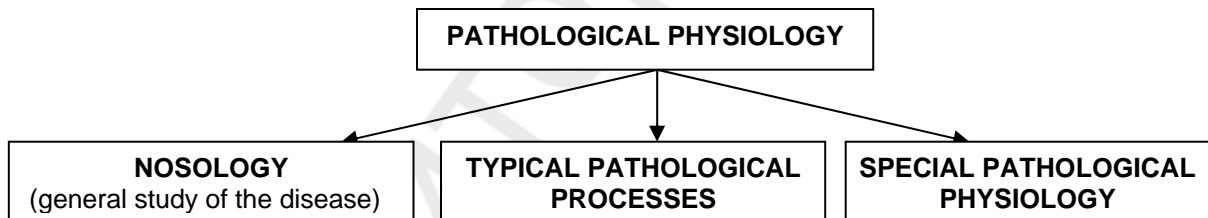
The subject of studying includes the most common basic laws and the mechanisms underlying on basis of resistance of the organism, origin, development and outcomes of pathological processes — diseases.

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 pathophysiologicalist 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 biologic 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 pathophysiological 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 safety precautions instructing for doing practical works at laboratories of the department.

Work 1. DEMONSTRATION OF TEACHING VIDEOS

1. Practical recommendations for dealing with laboratory animals.
2. Modelling 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. Pavlov).
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: « ____ » _____ 200__.

The purpose of the Lesson:

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

Tasks:

- To get acquainted with methodic 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.

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

Accomplishment

To carry out the experiment 8–10 frogs are connected to each other with their fore paws. «The alive chain» of frogs is suspended to a wooden stand. Needle electrodes are stuck into the paws 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 defined.

Results of the experiment

№	The reflex time by Turki (in seconds)			Note
	Initial data	Electric current exposure		
		2 sec.	60 sec.	
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

Accomplishment

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. What 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 monocausenalism, 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 non-specific) and their characteristic.

5. The reasons of lethal electrocutions and their mechanisms. «Imaginary death».

6. Rules of giving the first-aid on electrocution.

The teacher's signature:

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

Date: « ____ » _____ **200** ____.

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
SISTEM (experiments № 1, 2)**

Experiment № 1

Research is performed on white mongrel mice of identical weight. Hexenal (i/p, 100 mg/kg) is injected to one of them, then the mouse falls asleep 7–10 min. The occurrence of narcosis is determined by disappearance of a corneal reflex. The sleep lasts for 1,5–2 h.

Both mice — an intact, innarcotized 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. Further on, immediately after death of animals we determine the contents of O₂ and CO₂ 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
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 shows anxiety. It stands up on hind paws more often. The breath has accelerated and 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	–

Min	The control		The Experiment	
	RR/min	General state	RR/min	General state
35	–		12	–
45	–		6	–
48	–			Respiratory arrest
	Gas mixture content in the flask: O₂ = 7,1 %; CO₂ = 11,8 %		Gas mixture content in the flask: O₂ = 3,4 %; CO₂ = 14,6 %	

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



2. Construct a graph of changing [c] O₂ and [c] CO₂ 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 equal 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 innarcotized ones — are placed into one flask with the capacity of 200 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	–
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 breath. Respiratory arrest	50	The mouse is asleep. Sharp cyanosis
32	–	Spasms. Agonal breath	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: « ____ » _____ 200__.

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.

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 shout». Causes of development, karyotype, semiology.

12. Hereditarily determined diseases and abnormalities the 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; harm 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 labour protection for prophylaxis of hereditary congenital diseases for the faculty of medical prevention).

The teacher's signature:

LESSON 5. TOPICAL PROBLEMS OF GENERAL NOSOLOGY

Date: « ____ » _____ **200** __.

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 and 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. Incidence 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. Fill in table 1:

Table 1

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		

6. The characteristic of pathological reactions:

1 –

2 –

3 –

4 –

7. The characteristic compensatory reactions:

1 –

2 –

3 –

4 –

5 –

8. Fill in table 3:

Table 3

Periods of a terminal condition

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

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.

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: « ____ » _____ 200__.

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 sharp radiation sickness on the basis of materials presented in the teaching video «Sharp radiation sickness».
- Solving situational tasks.
- Test control over the topic of the Lesson.

**Work 1. STUDYING MATERIALS OF THE TEACHING VIDEO
«ACUTE RADIATION SICKNESS»**

- While viewing the video pay attention to the fragments presenting:
- Peculiarities of ionizing radiation action as a damaging factor;
 - The dependence of damaging action of radiation on the doze and kind of radiation;
 - Peculiarities of radiosensitivity of various organs and systems;

- The notion of biological dosimetry, its significance and bases (dose-effect, dose-time effect dependence); criteria (bone marrow state, cellular content of peripheral blood, quantitative and qualitative changes of cellular chromosomal structure);
- Local and general manifestations of sharp radiation sickness, dynamics of their development.

Answer the questions:

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

1. What form of acute radiation sickness has developed in this patient?

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).

Fill in table 2.

Table 2

Dynamics of changing the leukocyte count in peripheral blood in various dozes of radiation

Doze Gy (gray)	Period duration of primary leukocyte count reduction in blood (days)	Term of appearing an abortive rise of leukocytes in blood (+/-), (days)	Approach time and period duration of agranulocytosis (days)	Survival rate (+/-) stem elements in bone marrow	Survival rate (+/-) of intermediate forms of leukocytes in bone marrow
Up to 5 Gy					
Over 5 Gy					

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 dose of irradiation?

7. Why does the abortive rise of leukocyte count in blood develop in irradiation doses up to 5 Gy?

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

Fill in table 3.

Table 3

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		

Conclusion: (on the basis of case history data draw a presumable conclusion regarding the severity of ARS for the given patient).

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. Master 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. THE ISCHEMIA

Date: « ____ » _____ 200__.

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 of 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 and we 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 questions:

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

Vasodilators —

Vasoconstrictors —

2. List master 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: an 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. Syndromes of robbing the brain and myocardium.

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. EMBOLIA. HEMOSTASIS

Date: « ____ » _____ 200__.

The purpose of the Lesson:

– To study the reasons, incidence conditions, development mechanisms, basic manifestations and consequences for the organism of thrombosis, embolia, 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 embolia 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 fragments 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 Virkhov's triads**			Outcome		
			1 (+/-)	2 (+/-)	3 (+/-)	sludge (+/-)	hemostasis (kind) (+/-)	thrombosis (like a blood clot) (+/-)
Cooling								
Overheating								
Trauma								
Histamines								
Catecholamine								
Irritation n. sympathicus								
Ergotamine								
Nicotinic acid								

** **The note:** components of Virkhov's triad: 1 — imbalance between coagulation and anticoagulation systems, the activity of the coagulating system prevailing; 2 — damage of endothelium; 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. ACQUAINTANCE WITH 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 adjoined 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. ACQUAINTANCE WITH MODELING FATTY EMBOLIA OF THE FROG'S TONGUE VESSELS

An immobilized frog is placed on a plate with its abdomen up. 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 embolia of the frog's tongue vessels

Fill in the table:

BASIC LOCALIZATION AND SIGNS OF THROMBOEMBOLISM

Vascular region-source of thromboemboli	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			

Embolia result			
The basic embolia signs in vessels of the given localization			

Control questions

1. The definition of the notions: «thrombosis», «embolia», «hemostasis». General characteristic.

2. The reasons and incidence conditions of thrombosis. Master factors of thrombosis.

3. Stages and mechanisms of thrombosis. Types of blood clots and thrombosis outcomes. Thrombosis consequences for the organism. Prophylaxis of thromboses.

4. The reasons and mechanisms of embolus formation.

5. Types of embolia. The significance, outcomes and consequences of embolia for the organism. Prophylaxis of embolia.

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: « ___ » _____ **200** __.

The purpose of the Lesson:

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

Tasks:

– To study the reasons, development 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 fragments 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, embolia and inflammation.

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

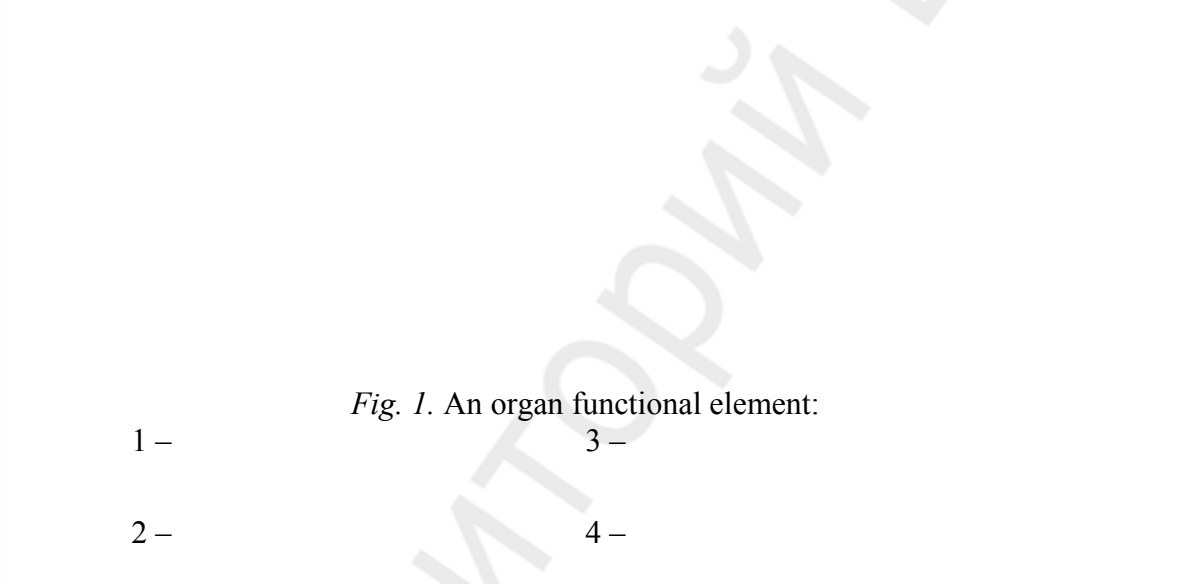


Fig. 1. An organ functional element:

- 1 –
- 2 –
- 3 –
- 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 (1)	Transmural (2)	Extravascular (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. Development mechanisms of intravascular impairments of microcirculation.
4. The reasons, development mechanisms, manifestations of transmural impairments of microcirculation.
5. The reasons, development mechanisms, manifestations of extravascular impairments of microcirculation.

6. The definition of the notion «sludge»; the reasons, development mechanisms; manifestations, consequences, the significance for the organism.

7. The definition of the notion «capillarotrophic insufficiency», development 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. DAMAGE OF THE CELL

Date: « ____ » _____ 200__.

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 CELLULAR DAMAGE BY SLIDES AND TABLES

Answer the questions:

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

2. List basic manifestations of cell 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 organellas 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: « ____ » _____ 200__.

The purpose of the Lesson:

– To study principal causes of occurrence, development 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 development 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 a teaching video, the textbook and the lecture **answer the following questions:**

1. The 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 kallikrein-kinin 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 leukopoiesis	Fever
Histamines									
Serotonin									
Prostaglandines of group E									
Leukotriens (LTC ₄ , D ₁ , E ₄)									
Prostacyclin (PGI ₂)									
Throboxans (TxA ₂)									
NO									
Lyzosomal enzymes									
Cytokines (IL-1β, TNF-α)									
Bradykinine									
Components of the complement system (C3a, C5a, C5, C9)									

6. Specify general manifestations of inflammation:

7. List basic enzymes of an acute phase:

8. Specify major factors regulating reparation and proliferation processes:

**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, 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 minutes with small breaks. We pay attention to changing of a 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, 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 cause inflammation of the frog's intestinal mesentry 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: « ____ » _____ **200**__.

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 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 a phagocyte (protective) reaction of the organism in inflammation on the basis of materials presented in the teaching video «The role of the granulocyte colonistimulating factor (Γ -KC Φ) 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 °C (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 porpoise 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 Romanovsky–Gimze 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 exsudate?

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 incomplet 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 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 bichromate crystal with a droplet of mercury

Conclusion:

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

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 theories pathogenesis of inflammation. Modern conceptions of inflammation mechanisms.

The teacher's signature:

LESSON 7. HYPOXIA

Date: « ____ » _____ **200**__.

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 development 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 are observing 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 «DYSBARRIC» PHENOMENA (MODELING EXPERIMENT)

Under the bell connected to the Komovsky's pump, place a tied up rubber glove and a glass with water, $t\ 37\ ^\circ\text{C}$ (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 embolia 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?

б) «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 (\uparrow or \downarrow in comparison with the norm)

Type of hypoxia	P_AO_2	P_aO_2	P_vO_2	$\Delta a-v\text{O}_2$	HbO_2 content	PaCO_2	P_vCO_2
1. Hyperbarric							
2. Normobarric							

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: « ____ » _____ 200__.

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 development mechanisms of pulmonary edema in experimental acute cardiac insufficiency induced by injection of adrenaline.
- Studying development 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. ADRENALIN PULMONARY EDEMA IN A RAT

For the experiment take two white rats with the body mass of 200 g, and take 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 take the respiratory rate ever 1–2 min to the moment of death. Euthanasia of the control rat is performed by stretching cervical vertebrae. 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 0,1 % Adrenaline solution			
– Initial	120	Normal	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
– 1 min	160	General excitation, impairment of motor coordination	
– 2 min	Rare deep respiration	Foamy discharge from the mouth	
– 3 min	Terminal respiration	– // – // – // –	
– 4 min	Respiratory arrest	Death of the animal	
i/p injection of 0,9 % solution of NaCl			
– Initial	130	General state without visible changes.	Weight of the lungs — 1,2 g, pulmonary weight factor — 0,006. The trachea is freely passable. Lungs are collapsed, of light pink color
– 1 min	–		
– 2 min	–		
– 4 min	–		

Conclusion:

Explain the development mechanism of adrenalin-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. **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 innarcotized 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 vertebra. 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 ₄ Cl			
– Initial	128	Normal	
– 15 min	150	Impairment of movements coordination	Lungs weight — 6,0 g, pulmonary weight factor — 0,03. Foamy liquid in the trachea.
– 30 min	200	The rat is motionless, is lying on one side	Lungs volume is enlarged, they remind marble, foamy discharge on dissection
– 45 min	Rare deep respiration	Neck and mouth muscles take part in respiration	
– 55 min	Terminal respiration Respiratory arrest	Foamy discharge from the mouth Death of the animal	
Hexenal narcosis + injection of NH ₄ Cl			
– Initial	100		
– 15 min	103	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
– 30 min	102		
– 55 min	102		
	Quiet, rhythmic respiration		

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 –
- dropsy –
- ascites –
- hydrothorax –
- hydropericardium –
- hydrocele –
- hydrocephaly –

Fill in the tables. Specify (\uparrow \downarrow or $=$) character of changes in the corresponding parameter.

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, cachectic, 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: « ____ » _____ 200__.

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 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 electrocardiogram. 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.

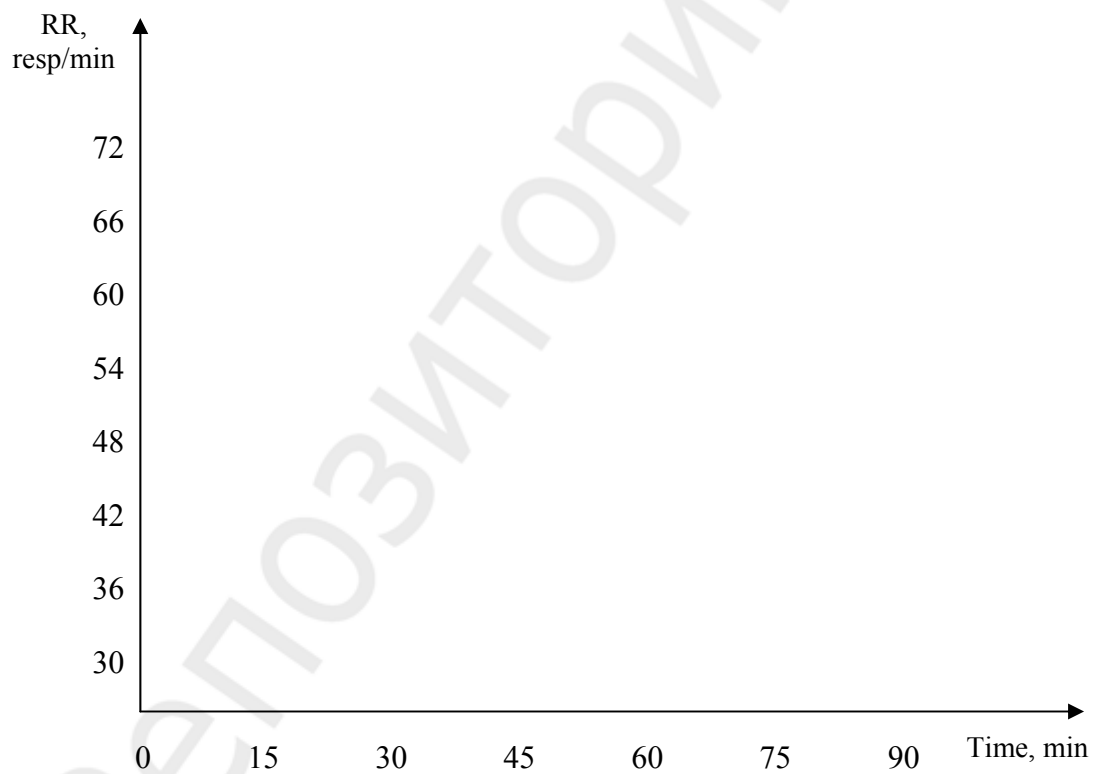
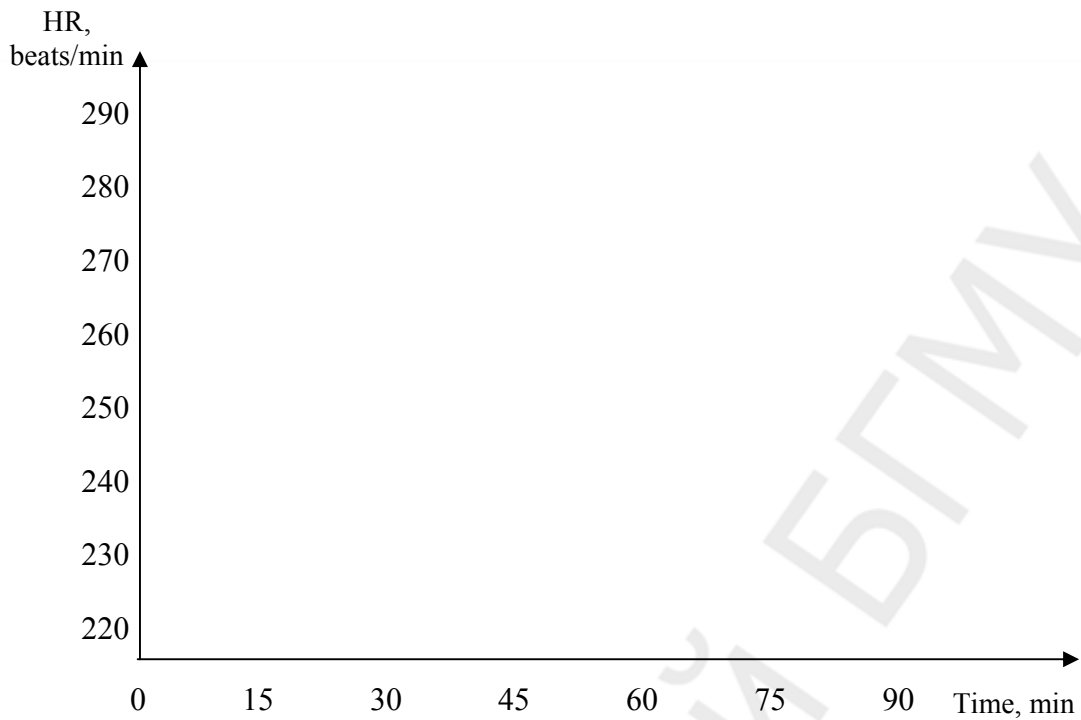
Results of the experiment

№	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					
	– 15 min	38,8	33	60	220	Ears are pale, cold, vessels are narrowed
	– 30 min	39,2	24,0	72	260	
	– 45 min	39,6	24,0	30	270	
	– 60 min	39,9	27,0	46	280	
	– 75 min	40,2	28,0	58	280	
	– 90 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					
	– 15 min	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	

1. 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.

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Draw conclusions, answer the following questions:

1. What evidences 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?

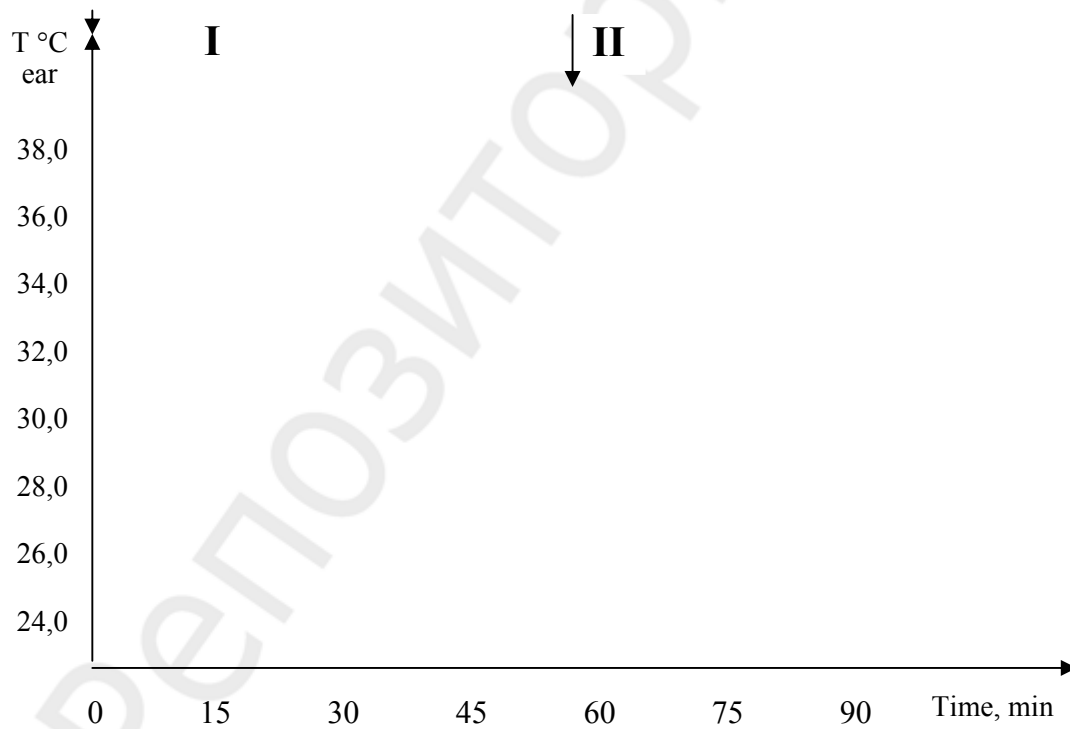
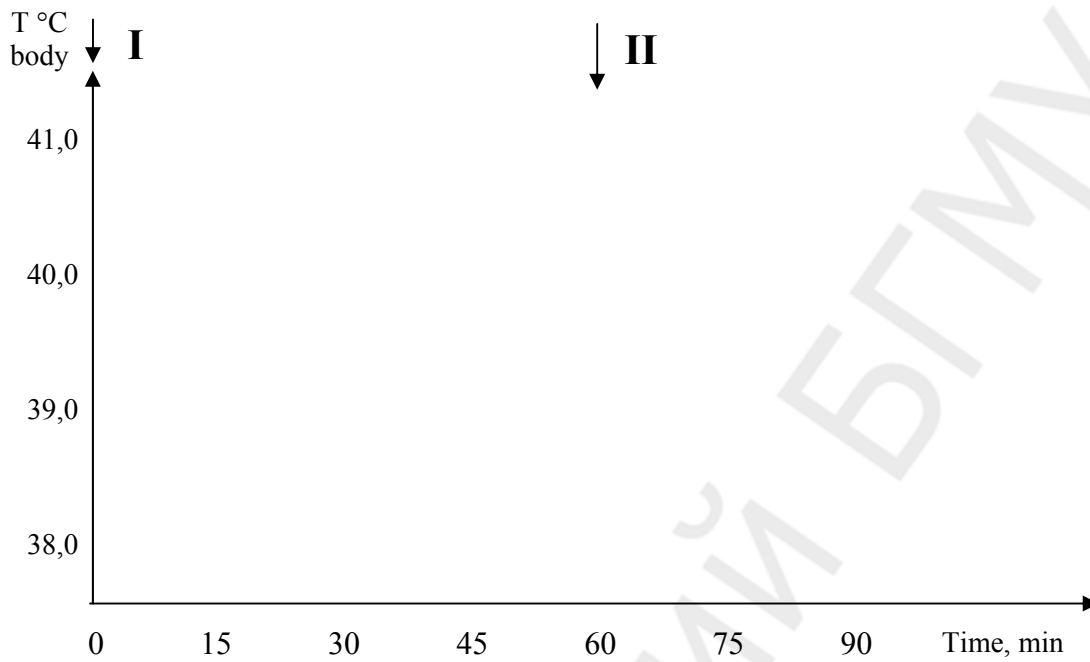
Work 2. STUDYING THE PECULIARITIES OF THERMOREGULATORY REACTIONS IN RABBITS WITH ENDOTOXIC FEVER UNDER OVERHEATING

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 ear, 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 taking of body temperature, respiratory rate and heart rate are continued everyone 15 min during their staying in **thermo-neutral conditions (20–21 C).**

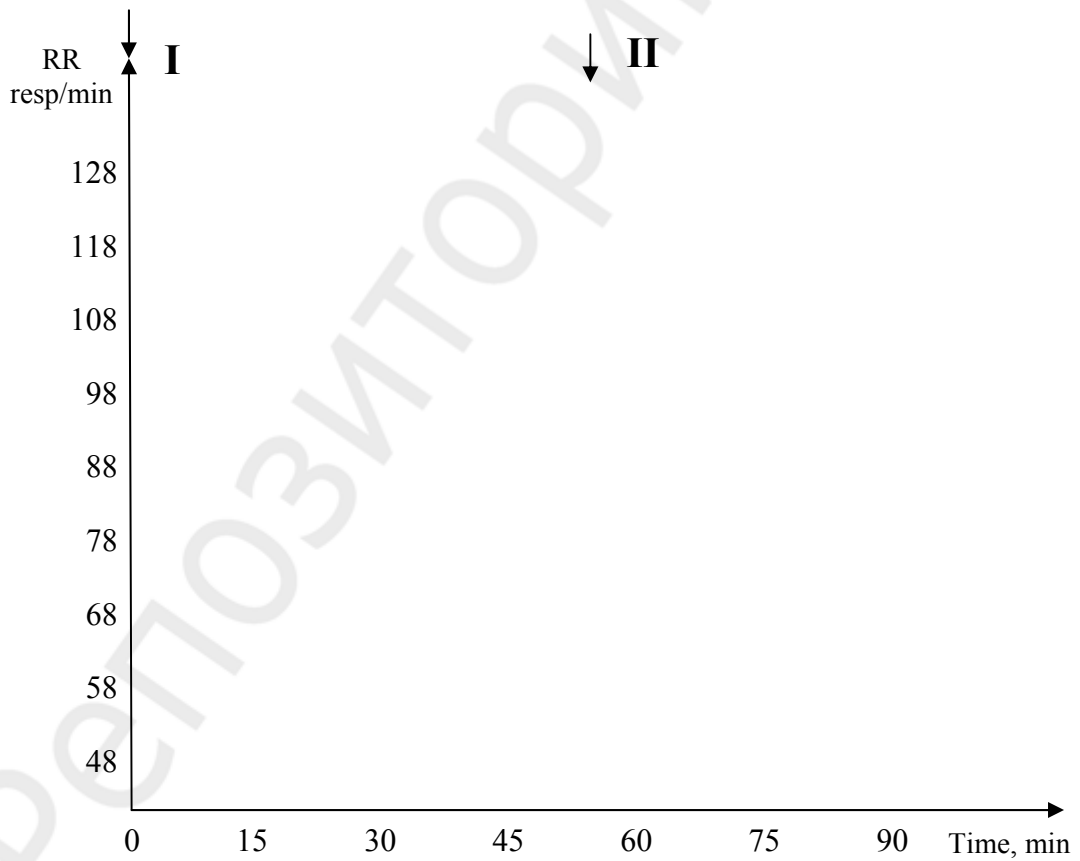
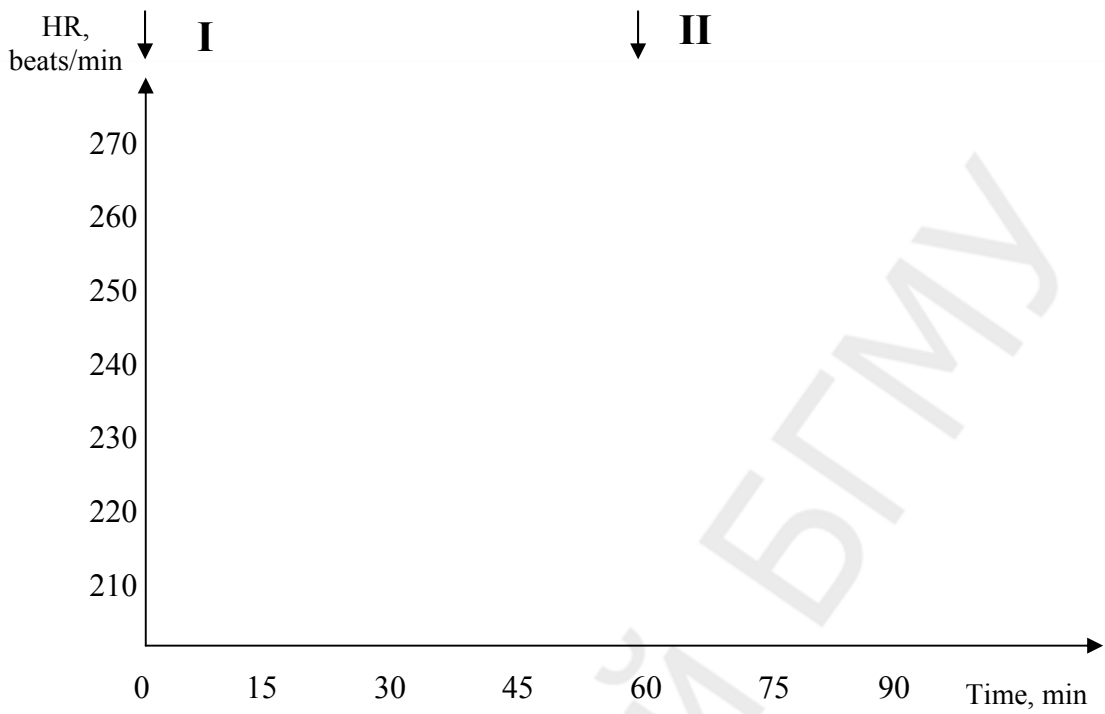
The received data are recorded into the table.

№	Group of animals. Time since the beginning of the experiment	Temperature, °C		Heart rate (beats/min)	Notes
		rectal	ear skin		
1.	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	
2.	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):

- constant →
- laxative →
- intermittent →
- exhausting →
- recurrent →
- wrong →
- perverted →

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	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	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
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
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
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
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			
41															
40															
39															
38															
37															
36															
35															

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 body temperature elevation in fever. The relationship between heat production and heat emission on 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: « ____ » _____ 200__.

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 _a CO 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
Electrolits 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 changing of a respiratory and metabolic component in typical ABS impairments

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

Control questions

1. Mechanisms maintaining pH of environmental liquids of the organism.
2. Classification of ABS impairments.
3. Basic laboratory estimation criteria of ABS impairments.
4. Etiology and pathogenesis of respiratory acidoses and alkaloses.
5. Etiology and pathogenesis of non-respiratory acidoses and alkaloses.
6. Major pathogenic development mechanisms of primary acidoses.
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 acidoses and alkaloses.
10. Correction principles of ABS impairments.

The teacher's signature:

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

Date: « ____ » _____ 200__.

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.

– 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	Major master 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 <i>Specify specific tumor AG markers</i>	
7. Invasive destructive growth	

Biological peculiarity	Major master factors; its manifestations in an integral organism
8. Metastasis <i>List the stages of metastasis</i>	
9. Inhering changes	
10. Tumor progression	
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 CELLS 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 intertwined ascite tumor of Erlich. Prepare a culture smear, fix it for 2-3 min in methyl spirit, stain it according to Romanovsky-Gimze, wash it out, dry up and examine under the microscope: at first with small, and then with large magnification (10×90).

During microscopic investigation observe cellular atypism (dwarfish and gygantic cells of various form), prevalence of round cells with an extremely hypochromous nucleus and sharp basophil cytoplasm (so-called dark cells), the presence of large cells with clearly outlined chromatin structure and pale-stained cytoplasm («light» tumor cells); frequent mitoses and amitoses, pathologic mitoses, 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 basophil 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 received 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 mem-

branes with narrow slits. Sometimes the tubular formations reminding iron elements are observed among a continuous layer of cells.

Examining the preparation fixed and stained in hematoxylin-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 mitoses; 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; master factors of carcinogenicity of chemical compounds.
6. The role of physical carcinogens in tumor development. Types of physical carcinogens.
7. Onkogenic 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: « ____ » _____ **200**__.

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 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 22A.
- 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 protooncogen?

3. What functions do proteins, products of a protooncogen, perform?

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

- 1 –
- 2 –
- 3 –
- 4 –
- 5 –
- 6 –

5. List the basic functions of oncogen — oncoproteins:

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

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		

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

Specify principal development 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 22A is processed with kolchicine resulting in the arrest of cellular division at a metaphase stage by suppressing the formation of spindles. Then the cells applied to the cover glass, 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 Romanovsky, Felgen or with aceto-orceine).

The karyotype of tumor cells differs from the karyotype of a normal, homologous 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 an 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 paracentromeral area 1–2 subcentromeral ones.

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: « ____ » _____ 200__.

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 degenerate forms of erythrocytes.

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

Examine blood smears of human embryos (3rd–4th 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 megaloblastic type of blood formation in the post-natal period?

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

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

1. _____ 2. _____

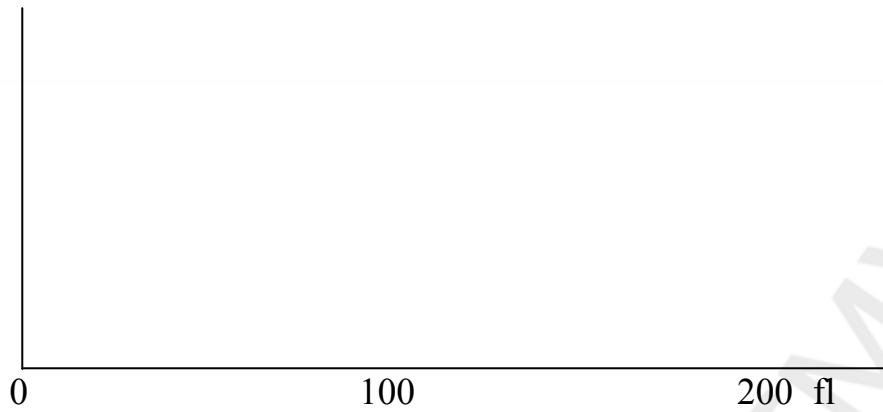
*Fig. 3. The blood smear in supravital staining with brilliant cresyl blue:
1 — erythrocytes; 2 — reticulocytes*

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;
- б) microcytes (_____ micron), or less _____ fl;
- в) macrocytes (_____ micron), or more _____ fl;
- г) 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 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: Jolli's (1), Kabo's rings (2), basophilic puncture (3), Heinz's bodies (4).

1. _____

2. _____

3. _____

4. _____

Answer the question:

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

Control questions

1. The blood system, the definition of the notion, general characteristic.
2. Hemopoiesis. 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 ERYTHROCYTOSES

Date: « ____ » _____ 200__.

The purpose of the Lesson:

– To study etiology and pathogenesis of the most common anemias and erythrocytoses, 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 B₁₂-deficiency anemia;
 - d) in microspherocytosis (disease of Minkovsky–Shoffar).
- Test control of the topic «Anemias and erythrocytoses»;
- Analyzing hemograms (№ 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 **Romanovsky-Gimza**.

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 pattern in 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 pattern in 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 5th day after acute blood loss?
2. To what processes in the erythrone system do the revealed changes testify?
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 the 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 pattern in 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 IN B₁₂-(FOLIC ACID) DEFICIENCY ANEMIA

Examine a blood smear of the patient with the B₁₂-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 Jolli's bodies, Kabo's rings, basophile puncture; and also single megaloblasts and giant polysegmentonuclear leukocytes. Draw these cells.

Fig. 3. Blood pattern in B₁₂-(folic acid) deficiency anemia:

1 — a megaloblast (*1a* — basophilic; *1б* — polychromatophilic; *1в* — oxifilic); 2 — megalocytes; 3 — poikilocytes; 4 — erythrocytes with pathological inclusions (*4a* — with Jolli's bodies; *4b* — with Kabo's rings; *4c* — with basophilic puncture); 5 — a giant polysegmentonuclear neutrophile

Answer the questions:

1. What type of hemopoiesis is characteristic of B₁₂-(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 B₁₂-deficiency anemia?

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

- Jolli's bodies are
- Kabo'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
Microcytes	
Macro (megallo-)cytes	
Microspherocytes	
Sickle erythrocytes	
Torocytes (codocytes)	
Hypochromous erythrocytes (anulocytes)	
Hyperchromous erythrocytes	
Megalloblasts	
Erythrocytes with Jolli's bodies, Kabo'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 hemorrhagic 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) B₁₂-(folic acid) deficiency;
 - d) B₁₂-(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-phosphatedehydrogenasa of erythrocytes);
 - c) hemoglobinopathies (sickle-cellular anemia; tallasemia);
 - d) anemia due to exposure of antibodies and other damaging factors.
6. Impairments and compensatory-adaptive processes in the organism in anemia.
7. Erythrocytoses. 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.
LEUKOCYTOSES, LEUKOPENIAS**

Date: « ___ » _____ 200__.

The purpose of the Lesson:

– To study quantitative and qualitative changes in leukocyte system; typical forms of their impairments, types of leukograms 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 leukocytoses.

– To discuss some hemograms 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, hematologic 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 Khiaskov–Dele; 5 — with chromatolysis; 6 — rod nuclear with thorns

Answer the question:

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

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 a great number of neutrophilic leukocytes of various degree of maturity in the field of vision. Draw them.

Fig. 5. Blood pattern in neutrophilic leukocytosis:

1 — a metamyelocyte; 2 — a rod nuclear neutrophile; 3 — a segmented neutrophile;
4 — a neutrophile 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 a great number of eosinophilic leukocytes of various degree of maturity in the field of vision. Draw them.

Fig. 6. Blood pattern in eosinophilic leukocytosis (large eosinophilia):

1 — a rod shaped eosinophile; 2 — a segmented eosinophile; 3 — a segmented neutrophile

Answer the questions:

1. What is meant for large eosiniphilia?
2. For what pathological conditions is large eosinophilia characteristic of?

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*:

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

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 hemogram changes:

Absolute neutrophilia —

Absolute neutropenia —

Agranulocytosis —

Absolute lymphocytosis —

Absolute lymphopenia —

Specify the basic development mechanisms of leukocytoses:

1 –

2 –

3 –

Specify the basic development mechanisms of leukopenias:

1 –

2 –

3 –

4 –

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

Table 1

Types of leukocytoses 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	

Character of the leukocyte formula changes (in absolute figures)	Most common conditions, for which the given leukocyte formula change is characteristic of
Monocytopenia	
Agranulocytosis	
Panmyelophthisis	

Table 3

Leukocyte formula changes in some pathological conditions

Pathological condition		Characteristic changes of the leukocyte formula
Acute bacterial (coccal) an infection	Peak of the disease	
	Recovery period	
	Proceeding as a sepsis type	
Acute viral (the 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 testify to?

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

Control questions

1. Leukopoiesis, its impairments.
2. Pathological forms of leukocytes, their morphofunctional peculiarities.
3. Leukopenia, the definition of the notion, the cause and development mechanisms, its types.
4. Agranulocytosis, the definition of the notion. Types of agranulocytosis, the causes and their development mechanisms. Peripheral blood pattern in various types of agranulocytosis.
5. Panmyelophthisis. The causes and its development mechanisms, the pattern of peripheral blood and bone marrow in panmyelophthisis.
6. Leukocytosis, the definition of the notion, types, the causes and development 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. HEMOBLASTOSES. LEUKOMOID REACTIONS

Date: « _____ » _____ **200**__.

The purpose of the Lesson:

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

Tasks:

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

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

– To analyze a series of hemograms (№ 21–29) of patients with leukoses 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 «Leukocytoses, leukopenias and leukoses».

Work 1. ACQUAINTANCE WITH MORPHOFUNCTIONAL CHARACTERISTIC LEUKOSAL CELLS, BLOOD PATTERN AND SOME SYNDROMES IN LEUKOSES

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

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

Table 1

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

Myelo-leukosis 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 leukoses into acute and chronic?

2. What is meant for «a leukemic failure»? For what leukoses is it characteristic of?

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

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

Table 2

Basic syndromes in acute leukoses

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

Study blood smears of patients with a leukemic form of acute myeloleukosis 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 myeloleukosis** (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 neutrophils (*hiatus leukaemicus*).

Fig. 1. Peripheral blood pattern in subleukemic or leukemic form of acute myeloleukosis:
1 — a blastic cell; 2 — a segmented neutrophile

Conclusion:

Characterize basic changes of cellular composition of peripheral blood in acute myeloleukosis.

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

Study blood smears of patients with leukemic forms of chronic leukoses 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 myeloleukosis** (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 pattern in chronic myeloleukosis:

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 lympholeukosis** (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–Humbrecht).

Fig. 3. Blood pattern in chronic lympholeukosis:

1 — a lymphoblast; 2 — a prolymphocyte; 3 — lymphocytes; 4 — cells (shadows) of Botkin–Humbrecht

Conclusions:

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

Table 3

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

Leukosis type	Prevalence of blasts or maturing and mature forms in blood	Cells of a tumoral process occurring in blood	Specific hematologic «markers» of leukosis	Red blood status	Number of thrombocytes in blood
Myelo-leukosis					
Lym-pholeu-kosis					

Control questions

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

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: « ____ » _____ 200 ____.

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. One of the arteries is introduced a cannula connected with the manometer to register arterial pressure on the kymograph tape. 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, it 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 opportunities of the organism carry out the third (*stream*) bloodletting in the volume of 10 % of blood; all parameters are being registered.

In 5 min *massive stream* bloodletting is performed, about $\frac{1}{3}$ 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. Changing 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: « ____ » _____ 200__.

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.

Accomplishment

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	
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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 THROMBOCYTOPENIA

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

Fig. 1. Blood pattern in thrombocytopenia:

1 — erythrocytes; 2 — neutrophilic leukocytes; 3 — thrombocytes

Control questions

1. The hemostasis system. The definition of the notion, functional purpose. The modern 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: « ____ » _____ **200**__.

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 nosologic 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. Leukocytoses and leukopenias, their types, causes and development 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. Leukoses. The definition of the notion. Etiology and pathogenesis. Modern theories of the origin of leukoses. Principles of classification. Blood pattern.
12. Leukemoid reactions, their types. Etiology and pathogenesis, distinctions from leukocytoses and leukoses. 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. Master 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 hemograms and to solve situational tasks with detailed 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:

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

Date: « ____ » _____ **200** __.

The purpose of the Lesson:

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

Tasks:

– To study the causes, development 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 innarcotized 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 condition 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? To the achievement of what are they directed? In what cases does not tachycardia improve, but aggravates the position 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 electrocardiogram changes, mark the ST segment with a red pencil:

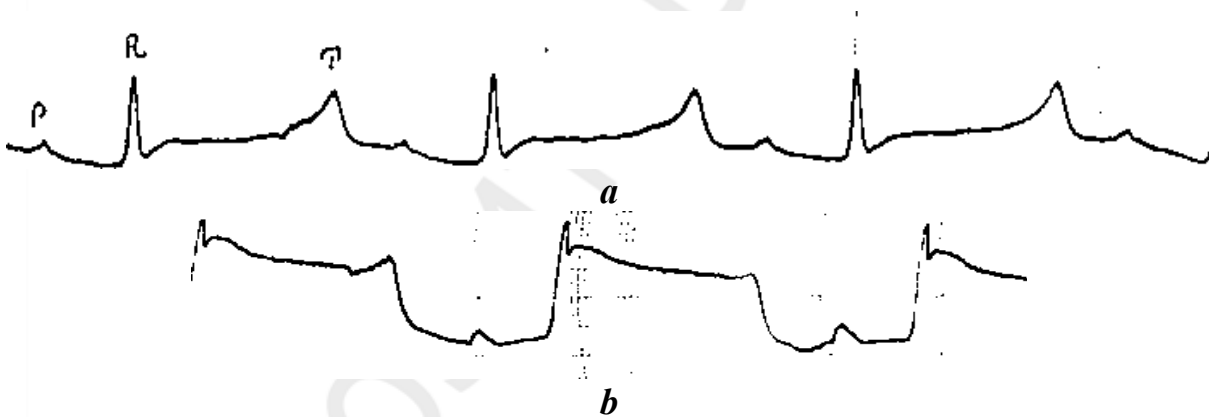


Fig. 1. Electrocardiogram changes in experimental myocardial necrosis of the frog, induced by the silver nitrate crystal action:

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.

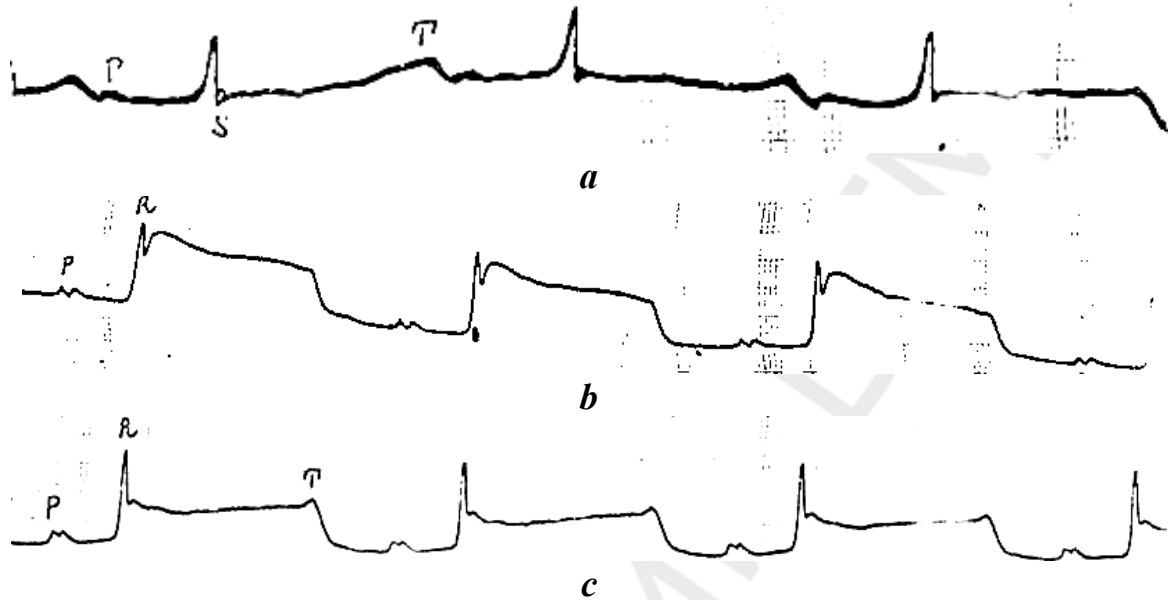


Fig. 2. Changing of the frog's ECG under the effect of local application of a necrotized slice of the cardiac muscle with subsequent washing up of the heart by Ringer's solution: *a* — the electrocardiogram in norm; *b* — the electrocardiogram after application of a necrotized slice of the cardiac muscle;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

2. A cotton wool, moistened with 1 % solution of potassium chloride. Register 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.

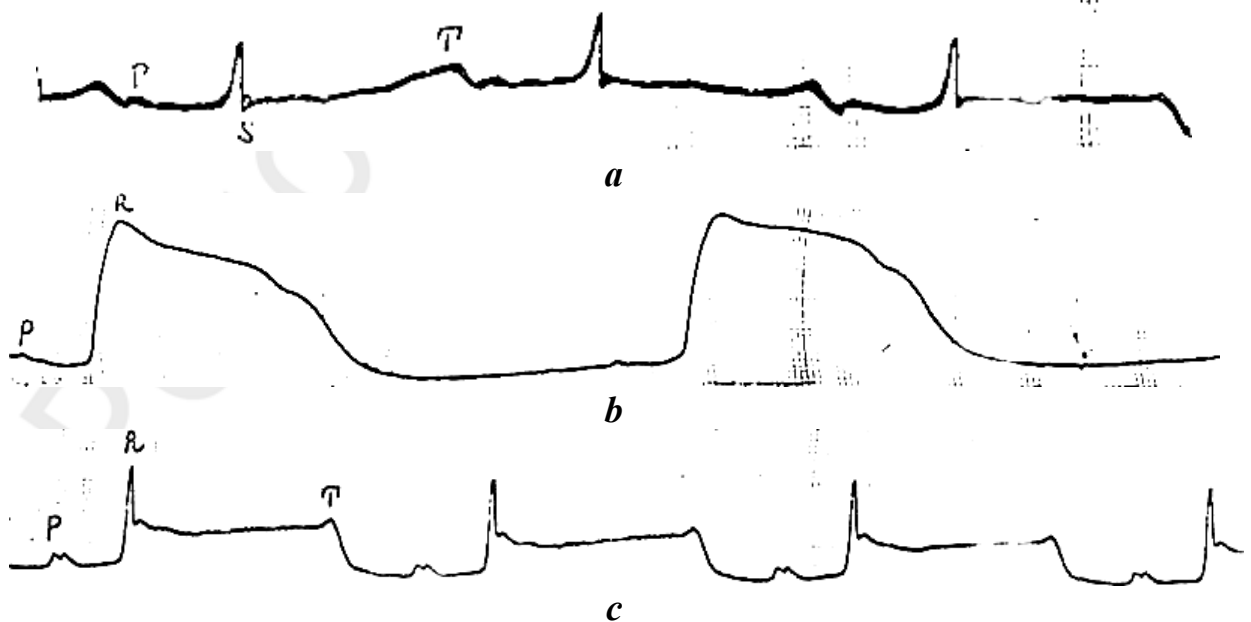


Fig. 3. Changing of the frog's electrocardiogram under the effect of local application of a cotton wool, moistened with 1 % solution of KCl with subsequent washing up of the heart with Ringer's solution:

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 ICD. Relative and absolute coronary insufficiency.
7. Etiologic risk factors of ischemic cardiac disease (ICD). Experimental methods of its inducing. Principal causes of non-coronary necroses 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: « ____ » _____ 200__.

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 O₂, 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 compensation 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 (B) 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 $\Delta A-V O_2$;
- 6) increasing signs of cerebral and cardiac hypoxia;
- 7) decrease of a pumping function of the heart;
- 8) deepening and accelerating 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), 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), 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.

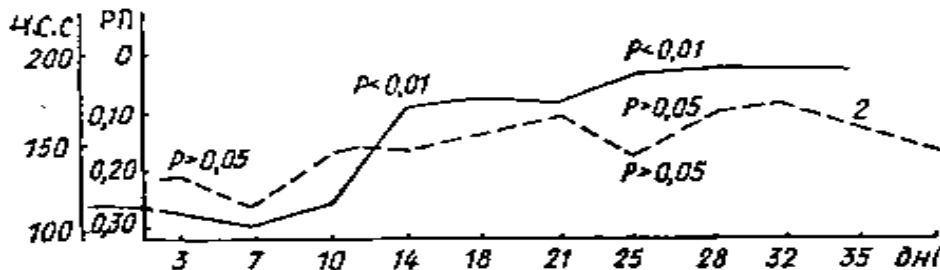


Fig. 1. RR interval duration, the average HR and pulse rhythm factor (PR) in CBCI

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 mechanisms of intracardiac compensation in cardiac overstrain.
4. 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.
5. 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.

6. 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.

7. Extracardial compensatory mechanisms of cardiac insufficiency, their pathogenetic characteristic. The role of vegetative nervous system in compensating chronic cardiac insufficiency. The notion of hormonal-neromediator dissociation. Its pathogenetic characteristic.

8. Main effects of hyperactivation of sympato-adrenaline and rennin-angiotensin-aldosterone system in chronic cardiac insufficiency. Mechanisms of cardiotoxic action of catecholamines. Pathogenetic characteristic of tachycardia in cardiac overstrain.

9. Reactions of the respiration and hemopoietic system in the development of cardiac insufficiency, trigger mechanisms of these systems.

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

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

12. 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.

13. Pathogenetic therapeutic principles of cardiac insufficiency.

The teacher's signature:

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

Date: « ____ » _____ 200__.

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.

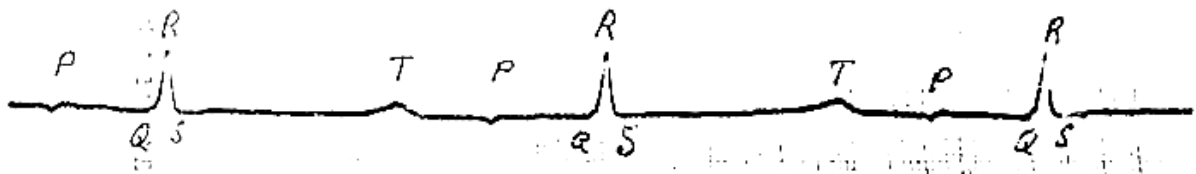


Fig. 1. Electrocardiograms of the frog in norm. R-R = 1,2". HR = 60 c/RR =

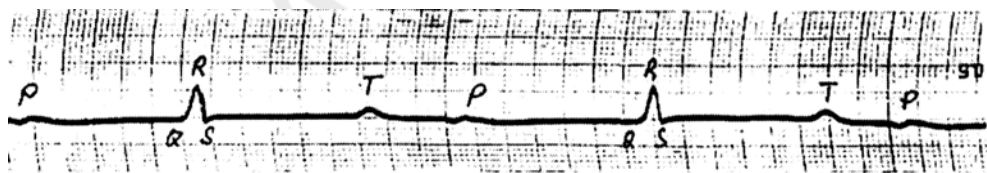


Fig. 2. Electrocardiograms of the frog after irritation of the stomach by induction current R-R " = 1,5". HR =

Answer the questions:

1. What ECG changes were observed in experiment?
2. To what type of rhythm impairments are they referred?
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.

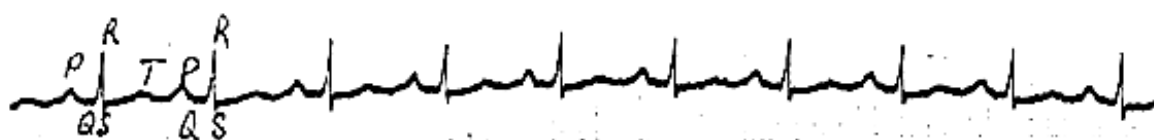


Fig. 3. Electrocardiograms of the rabbit in norm

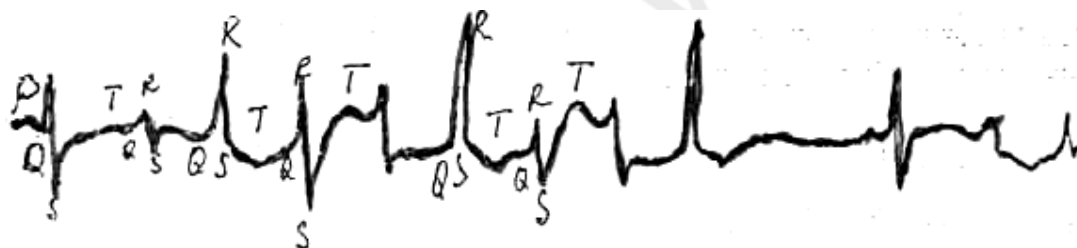


Fig. 4. Electrocardiograms of the rabbit immediately after injections of barium chloride
Specify the type of impairment of the cardiac rhythm:

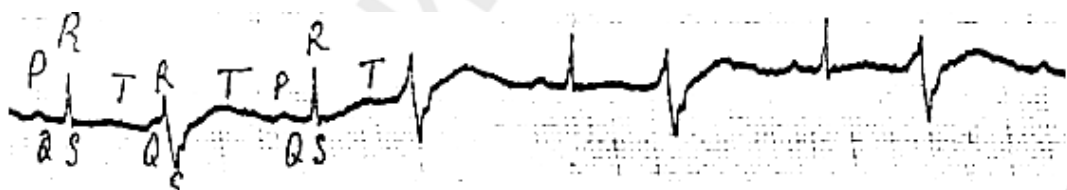


Fig. 5. Electrocardiograms of the rabbit in 1 minute after injection of barium chloride

Specify the type of impairment of the cardiac rhythm:

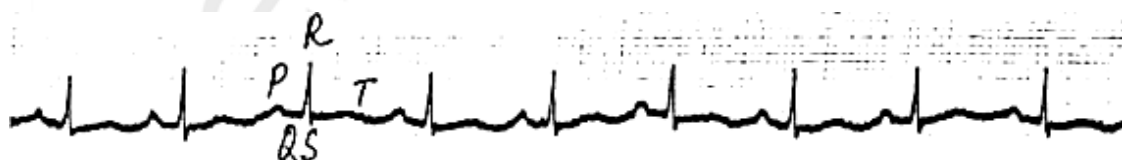


Fig. 6. Electrocardiograms of the rabbit in 15 minutes after injection of barium chloride

Specify the type of impairment of the cardiac rhythm:

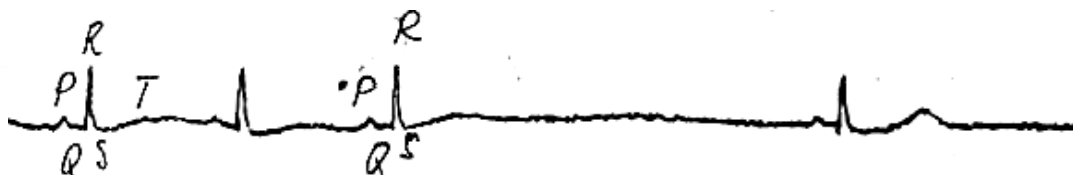


Fig. 7. Electrocardiograms of the rabbit immediately after inhalation of NH_4OH

Specify the type of impairment of the cardiac rhythm:



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

Specify the type of impairment of the cardiac rhythm:

Control questions

1. The definition of the notion «cardiac arrhythmias». Classification of arrhythmias.
2. Impairments of cardiac excitability: extrasystoly (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 palpitation and flickering (causes, characteristic, ECG-manifestations, hemodynamic impairments);
 - б) 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: « ____ » _____ 200 ____.

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, crises, 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 ICD (ischemic cardiac diseases). Relative and absolute coronary insufficiency.

7. Etiological risk factors of ICD. Experimental methods of induction. Principal causes of non-coronarogenic necroses 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 intracardiac 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 renine-angiotensine-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) palpitation and flickering of atria (causes, characteristic, ECG-manifestations, hemodynamic impairments);

б) 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 renine-angiotensine-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 «robbing the brain», «Robin Good», excessive cerebral perfusions; their characteristic, pathogenetic characteristic.

34. Cerebrovascular insufficiency, its types. Paroxysms, crises, strokes. Pathogenetic therapeutic principles of cerebrovascular insufficiency.

35. Atherosclerosis, its 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.

36. Participation of the vascular wall cells in interaction with modified lipoproteins and the formation mechanism of atherosclerotic deposits. Basic experimental models of atherosclerosis.

The teacher's signature:

LESSON 12. PATHOLOGICAL PHYSIOLOGY OF THE EXTERNAL RESPIRATION SYSTEM. TYPICAL IMPAIRMENTS OF PULMONARY FUNCTIONS

Date: « ____ » _____ **200** __.

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	1 stage	2 stage	3 stage
CNS (central nervous system)			

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 allocated in a narcotized dog and a cannula is introduced into it, then using the tubes filled with the solution of magnesia sulphate it is connected to the mercury manometer for arterial pressure registration.

The trachea is allocated and a tracheal cannula is introduced 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 registration 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).

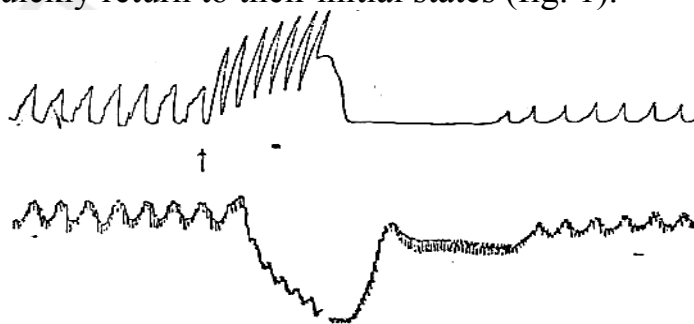


Fig. 1. Changes of respiration (the top curve) and arterial pressure (the bottom curve) in elevating the intraalveolar pressure of the dog. The arrows correspond to the moment of inflating air into the lungs

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 a possible mechanism of these changes?
3. In what pathological processes, diseases can 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).

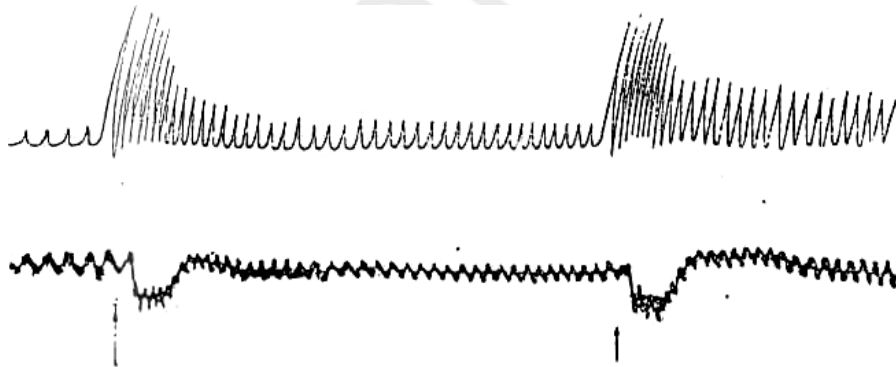


Fig. 2. Changes of respiration (the top curve) and arterial pressure (the bottom curve) in the development of acidosis in the dog. The first arrow corresponds to injection of the acetic acid solution into the blood, the second arrow — to injection of the sodium dihydrophosphate solution

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 similar phenomena arise?

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 (polipnea)			
Stenosed			
Breathlessness	Inspirat.		
	Expirat.		
Periodic	Chain–Stocks		
	Wavy		
	Biot		
Terminal	Hasping		
	Apnesis		
	Kussmaul		

Control questions

1. Insufficiency of the external respiration system. The definition of the notion, classification. Causes and development 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: « ____ » _____ 200__.

The purpose of the Lesson:

– To study the causes, development 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	Hyperchlorohydrria with pepsin hypersecretion	Hypo- and achlorohydrria 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 (+/-)		
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 character
--------------	-----------------	----------------

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

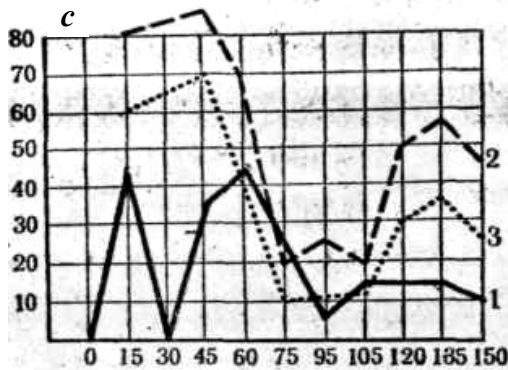
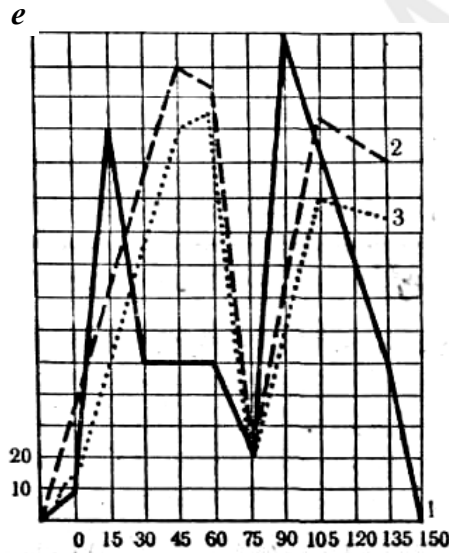
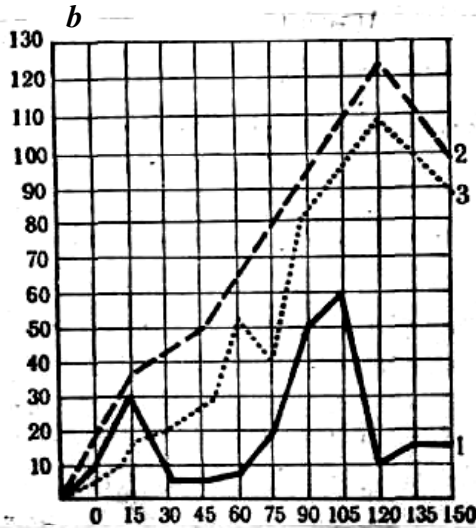
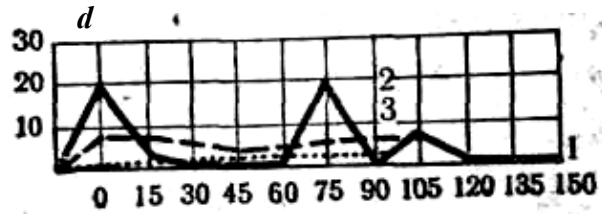
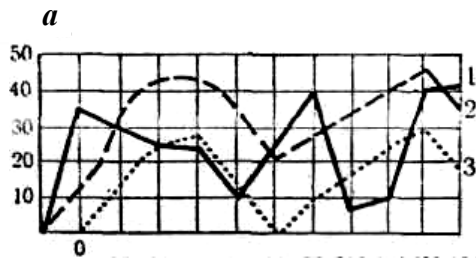
ULCER

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 (see tab. 3).

Table 3

Fractional test parameters of gastric juice

	Conditions of taking and studying gastric contents	Gastric contents volume, ml	Titration units			Pepsin, МГ %
			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 hypochlorohydrria. 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: « ____ » _____ **200**__.

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		Feces	
		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 4

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 tab. 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 frog jumps, bumping at the chamber walls. The muscular tone is elevated, muscular

Time	Control	Experiment
	The muscular tone is not changed. Coordination of movements is not impaired. HR — 42 per min	contractions are periodically observed. Lying on the back, the frog cannot take the former position at once. HR — 30 per min
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, answer the following questions:

1. What syndrome arises in the animal being injected bile parenterally? By what bile components is it caused?
2. On the part of what systems are the impairments noted? Give their characteristic, possible development 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 bag, 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 effect 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 portocaval 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: « ____ » _____ 200__.

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 departments 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 found. 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 °C) 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 let 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 °C), 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 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 ischemized kidney						+++
Time of appearing the stained urine			intact kidney			2 min
			ischemized 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 development mechanism of anuria in stretching of the bladder.

4. Explain the development mechanism of pain oliguria.

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, hemoglobinuria, proteinuria, cylindruria, anuria, oliguria, 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: « ____ » _____ **200**__.

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, development 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 paralyzes;
- б) 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 departments (structures) of the nervous system results in central (spastic) and peripheral (flaccid) paralyzes?

3. In what way does the muscular tone, tendon and periosteal reflexes, muscular trophic condition change in spastic and flaccid paralyzes?

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 to pinch the right hind paw, no reaction occurs (fig. 1). If to pinch a hind paw on the side with cut anterior processes (fig. 2), there will be no reaction due to switching off the locomotor processes, however contraction of the right paw is revealed.

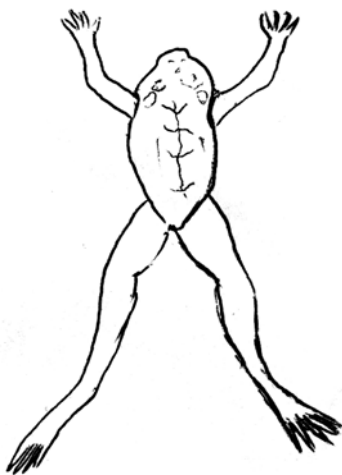


Fig. 1. Absence of reaction to irritation



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. Syndrome of Brown-Sekar. The mechanism of its origin and manifestation.
5. Neurogenic disturbances of the locomotor function. Hypokinetic conditions: pareses and paralyzes, 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, neuroses. The significance of the types of higher nervous activity in the development of neuroses. The causes of neuroses, their characteristic, principles of therapy.
10. Experimental models of neuroses (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 nervous trophics 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: « ____ » _____ 200__.

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–Kushing, 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-Kushing, primary and secondary hyperaldosteronism, adreno-genital syndrome, pheochromocytoma, clinical manifestations.

10. Diabetes of the Ist and IInd type, their etiology, pathogenesis, clinical manifestations. Mechanisms of hyperglycemia and glycozuria. Manifestations of the impairment of organs — targets in diabetes.

The teacher's signature:

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